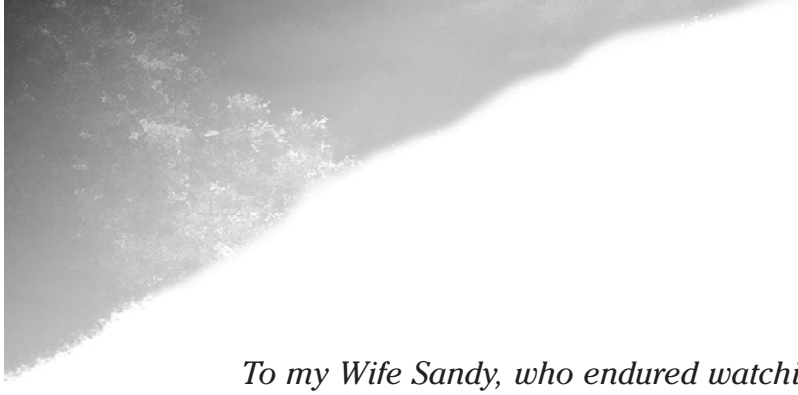
4th Edition

ALFRED A. BOVE

Bove and Davis' DIVING MEDICINE

SAUNDERS



To my Wife Sandy, who endured watching long enough and then became my partner in both diving and diving education.

Memorial

HUGH GREER, M.D.

Hugh D. Greer was a former contributor to *Diving Medicine* and a recognized authority in the neurologic aspects of diving. He died suddenly while swimming on October 2, 2001. Dr. Greer was born in Madison, Wisconsin in 1932 and joined the Navy Reserve as a midshipman in 1949. After completing college at Dartmouth, he was commissioned as a Lieutenant Junior Grade in the Navy and was a member of Underwater Demolition Team 22 until his discharge in 1956.

He attended Medical School at the University of Kansas and received his medical degree in 1960. He interned at the Mary Hitchcock Hospital from 1960 to 1961 and continued his training in neurology at the Mayo Clinic. Dr. Greer published several papers in clinical neurology and was board certified in neurology and psychiatry. He joined the Santa Barbara Clinic as a clinical neurologist in 1964, where he remained until his death. Over his many years at the Clinic, he developed expertise in the neurologic aspects of diving and became recognized as an international expert. Dr. Greer was formally trained in diving medicine through the National Oceanic and Atmospheric Administration program in 1978. He served as an adjunct scientist to the USC Institute of Marine and Coastal Studies and was a diving medicine consultant to the Catalina Hyperbaric Chamber, Santa Barbara City College, and a number of commercial diving companies. He was a fellow of the Explorers Club of New York. Along with Dr. Paul Linaweaver, he directed the southwestern division of the Divers Alert Network. Throughout his career, he continually published in the literature of diving medicine and contributed the chapter on the neurologic aspects of diving in the second and third editions of this text.

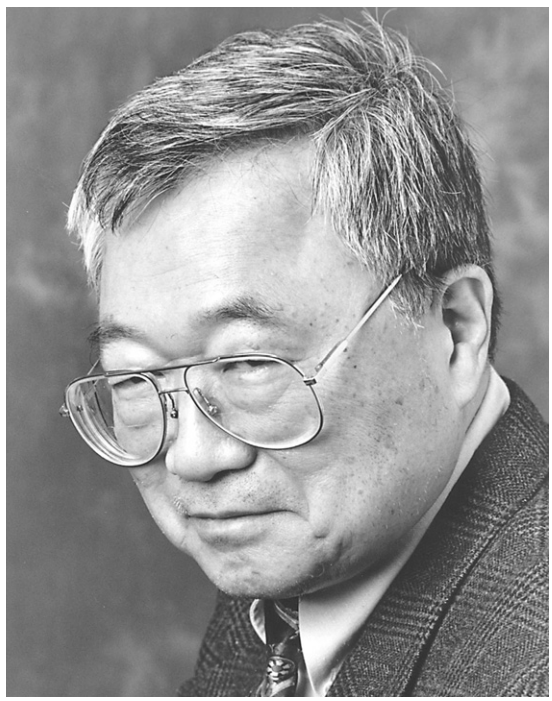
Dr. Greer contributed to the governance of the Santa Barbara Clinic by serving at various times as the President of the Board of Trustees and as President of the Board of Directors.

Dr. Greer was a fellow of the American Academy of Neurology, a fellow of the American Academy of Electromyography and Electrodiagnosis, and a member of the Undersea and Hyperbaric Medical Society. He is survived by his wife, son, two daughters, and six grandchildren.

Paul G. Linaweaver, M.D.

SUK-KI HONG, M.D., Ph.D.

Suk-Ki Hong, author of the chapter on breath-hold diving in the second and third editions of *Diving Medicine,* died on October 4, 1999. Dr. Hong's studies in diving physiology encompassed both human breath-hold diving and saturation diving and were performed in collaboration with colleagues in Korea, Japan, Europe, and the United States. His publications concerning breath-hold diving covered 35 years and constitute the most thorough record in the literature on all aspects of breath-hold diving. Throughout his career, Dr. Hong received numerous awards for his work in diving medicine. His scientific legacy is not only the impressive volume and quality of his research publications but also the many students and fellows who now follow in his path and have gone on to productive scientific careers in many parts of the world.



Dr. Hong's generosity with his ideas, his comprehensive knowledge, and his unselfish good nature earned him the enduring respect and genuine affection of all who had the good fortune to know him. Even though he was a famously hard worker, setting high standards for himself and his associates, he was always constructive and truly interested in bringing out the best in people. His desire to excel was always tempered by his humanity, sense of fairness, and lively sense of humor. His family, friends, and colleagues sorely miss him.

Charles V. Paganelli

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Foreword

Advances in diving medicine have intermittently followed and led the past 100 years of astounding engineering developments in practical operational diving. Two milestones in treating the triad of decompression sickness, nitrogen narcosis, and oxygen poisoning were Haldane's increase in helmet ventilation to avoid the effect of CO_2 compounding nitrogen narcosis and the permanently sensible concept of multiple exponential uptake and elimination of inert gas in albeit indefinable body microtissues during compression and decompression.

In the 1920s and 1930s, dedicated diving medical giants related to the U.S. and British Navies laboriously established improved tables for limited air diving, derived in part from Haldane's concepts of staged decompression to "avoid formation of gas bubbles." These groups then responded to the suggestions of Hildebrand in 1924 and to civilian open-water diving trials concerning the use of helium to avoid the narcosis induced by nitrogen in deep air diving. These groups developed equipment and procedures to facilitate decompression by using helium with high levels of inspired oxygen in both working and decompression phases (the tables were baptized in the severe challenges of the salvage of the U.S.S. *Squalus*). In laboratory experiments on human divers, these groups explored the degrees of hyperoxic exposure that would avoid the drastic diving hazard of oxygen convulsions.

These early advances in suited hardhat diving and in the prevention and therapy of decompression sickness were refined in Navy laboratories by trial and error in large numbers of practical tests. These allowed empirical adjustment around a theoretical base. The rules were established and the equipment designed to encase the diver, provide security and stability at the work site, and provide for safe passive extraction back to the surface when necessary. Before 1940, diving that required backup and developments by diving medicine was essentially limited to naval services.

The extensive damage resulting from World War II turned Navy salvage diving methods development back to shallow air diving for clearing harbors alongside Army Engineer diving. However, before and during that war, a new form of diving evolved in Italy, the United States, and the United Kingdom: pure oxygen diving with rebreathing and carbon dioxide absorption in closed-system "pendulum" and "circuit rebreathing" designs. The resulting Self-Contained Underwater Breathing Apparatus—scuba—provided complete independence from the surface. The requirement now was to closely match detailed engineering design with the human physiologic demands of covert, long-duration submergence astride an underwater "chariot" or, for neutral-buoyancy underwater swimming, with "fins" over long distances at variable depths. The specific stresses were temperature, the toxicity of oxygen, and carbon dioxide accumulation, none of which was solvable by medical guidance alone.

These military operational advances were generally not well known because of their initial highly secret status, but the neutral-buoyancy shallow diving method using pure oxygen opened wide new areas of basic physiologic research interest important to oxygen therapy, respiratory and circulatory regulation, blood gas transport, the concept of damage by free radicals, improved therapy of all decompression sickness, and expanded recognition of the usefulness of oxygen in diving gas mixtures to limit inert gas uptake and accelerate its elimination.

After World War II, wide civilian use of a demand valve for self-contained, open-system air breathing underwater swung the cycle of diving medical interest back to the classic naval guidelines for air diving. The relative safety of the open-system method for shallow air diving allowed many millions of individuals to begin diving for

sport. The result was a parallel expansion of interest by civilian physicians in diving and diving medicine while military interest was low.

This book on diving medicine has provided a window on the continually expanding scope of operational and scientific accomplishment related to all forms of diving, from their beginnings to the extreme range of present activity. The book is generally concerned with the effects of self-imposed exposures to stresses by otherwise healthy persons rather than with spontaneous disease occurring in working divers. Stresses may be small or severe. Today, most sport diving involves the relaxed, harmless, and pleasurable activity of air breathing and seeing during submerged swimming in conditions of neutral buoyancy in clean, warm, shallow water. This hardly requires the attention of diving medicine. In the usual properly controlled circumstances of current open-circuit diving, stress and its effects are inconsequential; problems relate to the potential for accident rather than to intolerance of stress.

However, diving is not just breathing underwater, and all divers are not normal. With increased degree and durations of exposure to hydrostatic pressure, respiration of inert and chemically active gases, and severe thermal environments, the varied forms of physiologic stresses inherent to all types of diving may be intrinsically harmless but can lead to personal hazard or death in the unnatural underwater situation. The commercial working diver or the military combat diver continues to encounter the most severe combination of stresses and physiologic trauma of any form of human activity. At the extremes of practical forms of working diving, the individual is exposed to resistance to breathing, toxic effects of increased oxygen pressures, mental dulling by nitrogen, neurologic derangement due to the effects of physical ambient pressure, incapacitating loss or excess of body heat, and damage due to failure to avoid free gas phase development in body tissues. Because each of these stresses is a consequence of exposure to the pressure or temperature of water, or both, disease is always possible. The composite result of multiple added stresses is unpredictable and conducive to accident or failure.

From my vantage point as an equipment designer, operational diver, investigator, and diving physician, I am impressed by the collective breadth of scientific competence represented by the many contributors to this text. Such detailed expertise was hard to come by. How did it develop?

The evolution of clinical or technical close communion has played a special large role in accelerating research and development in diving and diving medicine. The present state of instantaneous voice or graphic communication should be contrasted with the previous limitations of worldwide direct personal communication by mail and ship prior to World War II.

The expansion of interest and activity following World War II was directly aided by the U.S. Office of Naval Research's interest in sustaining international medical research in aviation and diving and other forms of physiologic environmental stress. This effort stimulated development of a National Science Foundation and the National Institutes of Health, with each new agency actively supporting undersea biomedicine for several decades. All of this individual and agency initiative, communication, and national support gave rise to spontaneous and wide activity in university laboratories, including development of new laboratory systems for pressure and thermal environmental research. The composite of university, industry, and naval interest investment and work was worldwide.

Two large steps were responsible for the special worldwide influence on the course of international communication and the advance of undersea activity and medicine. One was the 30-year triennial series of International Underwater Physiology Symposia. The other was establishment of an Undersea Medical Society, which in turn spawned a European Underwater Biomedical Society and satellites. All participants enjoyed the new practicality of international travel and continuous direct scientific communication. Interest in diving medical research expanded concurrently with the initiation and gigantic growth of an offshore petroleum industry, diving for recreation, and military clandestine diving equipment. Inevitably, hyperoxygenation therapy research and application became important for clinical disorders beyond the scope of diving decompression incidents.

With all of the varied forms and purposes of human underwater activity and the expanding ranges of interacting stresses, modern diving medicine must continue to

assume clear responsibilities. It has a "need to know" in all areas of physical and physiologic stress. It has a primary role in aiding and providing operational guidelines, which prevent pathophysiologic failure or a pathologic event. It serves to provide a rational basis for effective therapy of diving-induced damage. It must conduct new research to further advance diving activity and the therapy of diving-related disorders.

As I said in the prior edition of this book, personal gratification afforded by the advancement of the scientific bases for operational roles is enlarged by a close awareness of why it all took so long.

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Preface

This edition of *Diving Medicine* continues our effort to provide physicians who care for divers, or who may encounter diving-related questions in their practice, a compendium of diving medicine that can be used as a daily practice aid and as a general reference for patient care related to diving.

To this end, we have added a chapter on diabetes and diving by Drs. Scott and Marks that is applicable to the recreational diving community (but that does not apply to commercial or military diving). We have also expanded several of the clinical chapters to cover topics that have appeared since the publication of the third edition. Medical evaluation for sport diving is covered in a specific chapter and is separated from commercial and military diving. Drs. Smith and Butler provided an insightful chapter that reflects their extensive experience in Navy diving and can be used by Diving Medical Officers in many navies of the world. Dr. Flynn's chapter is also pertinent to military diving. Dr. Elliott provided an update on assessment for commercial diving.

Most chapters have been significantly revised. Chapter 2, *Diving Physics,* and Chapter 5, *Breath-Hold Diving,* have new authors who provide expanded insight into these two areas of diving medicine. Some chapters describe slowly changing areas of diving medicine and have undergone minimal modification. Dr. Hamilton demonstrates his considerable expertise in mixed-gas diving with an excellent review of the topic in Chapter 6. Dr. Vann has considerably revised the chapter on mechanisms of decompression sickness, and Dr. Moon's chapter on treatment of decompression sickness is an excellent summary of the recent changes in approaches to treatment of diving-related disorders. Dr. Neuman provides updates to the chapters on barotrauma, near drowning, and pulmonary disorders. These chapters bring the most recent information and clinical opinion to these topics. In particular, the revision of clinical thinking on asthma and diving is addressed in Chapter 24. The cardiovascular chapter has been updated to reflect the accumulating information on patent foramen ovale, the exercise workloads required for diving, concerns with cardiac arrhythmias, and the application of newer coronary interventional procedures. Drs. Tipton, Mekjavic, and Golden have contributed a new chapter on hypothermia. Dr. Taylor provides an excellent updated review of issues related to women and diving. Her review of sports medicine, exercise in women, and exercise during pregnancy offers a practical approach to understanding the unique situations of women who dive. Previous material on marine intoxication is now a separate chapter that complements Dr. Edmonds' chapter on hazardous marine life. We have added an appendix on diabetic protocols for diving that supplements Chapter 26. Special appreciation goes to Dr. Massey, who agreed to complete the work of Dr. Greer on the neurologic aspects of diving.

We continue to use the standard nomenclature for diving-related disorders rather than one of several proposed changes in the description of diving disorders. In particular, the use of the term *decompression illness* is used when addressing the totality of disorders related to decompression (i.e., decompression sickness and lung barotrauma with arterial gas embolism). *Decompression sickness* in this text describes disorders caused by evolution of bubbles in gas-supersaturated tissues; *pulmonary barotrauma* and *arterial gas embolism* indicate disorders due to physical expansion of gas and mechanical injury to lungs with subsequent embolization of air in the vascular system. Neither term includes the other, and overlapping clinical syndromes are mentioned where appropriate. We understand the difficulty in some cases of ascribing the symptoms or signs to one disorder or the other; however, this system of nomenclature reflects the current understanding of diving pathophysiology and follows the usual

method of categorizing diseases by their pathophysiology rather than by their symptoms or signs. In particular, the medical - evaluation of a diver with a diving-related disorder and prognostic advice demand that the pathophysiology be elucidated to the extent possible.

Since the publication of the third edition of *Diving Medicine*, two of our chapter authors, well-respected physicians and scientists in diving medicine and physiology, have died. A short memorial is provided to honor Drs. Hong and Greer for their important contributions to this text and to the field of diving medicine.

This edition follows the tradition of previous editions in that chapters are written by physicians and scientists who are expert in their fields. We are grateful for the time and energy committed by all of the contributors to this text who share their extensive knowledge with the world's diving community.

Our goal, and that of this text, is to improve the health and safety of all divers.

Alfred A. Bove, M.D., Ph.D.

CHAPTER 1 A Short History of Diving and Diving Medicine

Eric P. Kindwall

Man's first entry into the sea was through breath-hold diving, undoubtedly to harvest shellfish and to retrieve lost tools or utensils. From early history we find that breath-hold divers accomplished such prodigious amounts of work that they became economically important. In many areas of the world, commercial pearl and pearl-shell diving still relies on the breath-hold diver to a great extent. Depths of 60 to 80 ft are common, and commercial breath-hold diving has reached depths of 100 ft.

Even treasure has been salvaged using the free diver. In 1680, Sir William Phipps recovered some £200,000 in sterling silver from a wrecked Spanish galleon in the Caribbean, and the "fishing up of the wrecked plate ships at Vigo Bay" cited by Stevenson in *Treasure Island* was accomplished by naked divers.

The depths that can be reached by the breath-hold diver depend on two factors. The first is how long divers can hold their breath without the CO_2 level in the blood forcing them to breathe *(breath-hold breaking point).* The second is the relationship between total lung capacity and residual volume. As pressure is increased on the lung, its volume is decreased, and even with a thoracic blood shift to fill some of the space, lung squeeze occurs somewhere in excess of 150 to 200 ft. However, certain exceptional persons with a high tolerance for CO_2 who have practiced breath-hold diving have set extraordinary depth records. A record of 247 ft was set in 1967 by Robert Croft, a U.S. Navy submarine engineman and escape-training tower instructor. Jacques Mayol, a Frenchman, set a record of 282 ft in 1973, surfacing fully conscious without the help of a positive buoyancy aid on ascent. In January 2000, Francisco Farreras set the current world breath-hold depth record of 531.5 ft off Cozumel, but his ascent was aided by an inflated buoy. (See Chapter 5.)

BELL DIVING

The use of the diving bell, which consists of trapped air in an inverted container, was the next method employed to extend working time on the bottom.

The diving bell is first mentioned in a French manuscript of 1250 AD, which has a fanciful illustration of Alexander the Great descending in the diving bell at the Siege of Tyre in 332 BC. It is highly unlikely that Alexander ever did go down in a diving bell, but he was shrewd enough to use military divers (free swimmers) for destroying enemy vessels.

The first modern records of diving bells used in practical salvage start in the 1640s, when Von Treileben used a primitive bell in the salvage of 42 cannons from the sunken Swedish ship of the line *Vasa,* which lay in 132 ft of water in Stockholm Harbor. The bell, shaped like a truncated cone, had no air supply other than that contained within the bell. Divers would descend to the bottom in the bell, swimming from the bell to the wreck to attach lines to the objects to be salvaged and returning to the bell for a breath of fresh air between excursions. Bell divers soon learned that the air at the top of the bell was more breathable than that at the bottom after they had been working for some period under water. CO_2 is slightly heavier than air, and as it accumulated, the CO_2 became more concentrated along the surface of the water toward the bottom of the bell. There is no report of decompression sickness among Von Treileben's submarine workers, but it is extremely possible that by working at those depths, especially if several dives a day were made, they could have absorbed enough nitrogen into their systems to have caused decompression sickness. The amount of work that was accomplished by those early bell divers is amazing; in 1960, a single remaining

bronze cannon was recovered from the same wreck by a helmeted deep-sea diver. Even with all of the advantages of modern equipment and a 150-ton floating crane, it took the diver 1½ days to remove the gun.

The next recorded note of a diving bell dates to 1690, when Halley (discoverer of the comet) devised a successful bell with the first system for renewing air within the bell while it was on the bottom. Lead-weighted barrels carried fresh air down to the occupants of the bell. Halley's bell was somewhat cumbersome and heavy, but we have records that it was used to depths of 60 ft. It is unlikely that it was used to perform any practical salvage.

The first modern practical diving bell was invented by Smeaton in 1790 with a workable force pump to continuously refresh the air in the bell. This bell, or *caisson,* was the forerunner of all modern types. It was first used in Ramsgate Harbor, England, for breakwater construction. Caissons are still used for the construction of bridge piers in much the same manner that Smeaton used his.

SURFACE-SUPPLIED DIVING GEAR

The object of having a man free to walk around the bottom without having to hold his breath or return to the safety of a diving bell was first realized when Augustus Siebe invented his diving dress. Siebe was a German coppersmith working in London. In 1819, he devised a diving rig that consisted of a copper helmet riveted to a leather jacket. The diver entered the dress through the open waist and then thrust his arms into the sleeves with his head protruding into the helmet. There was no control over the amount of air entering the helmet, and the excess air bubbled out around the diver's waist. Other inventors had tried their luck at similar designs, but apparently Siebe's diving dress was accepted because of his extremely reliable and successful force pump that produced the necessary compressed air. Siebe's original rig was used for successful salvage work on the sunken British warship, *The Royal George,* and was used by divers on many other important projects. It had one disadvantage in that if the diver lay down or turned upside down, the dress quickly filled with water and he was likely to drown. Nevertheless, this primitive apparatus accomplished much useful salvage.

Siebe was a constant innovator, and by 1837 he had improved his design. This device consisted of a full suit that was waterproofed and could be bolted to a breastplate and helmet. Because the suit covered the diver's entire body, divers could work in any position. Valves were provided for admitting varying amounts of air to the diving suit as needed, and an air exhaust valve was provided in the helmet. The 1837 Siebe closed-dress design proved itself so successful that it has remained essentially unchanged to the present day for classic deep-sea diving. The United States Navy Mark V deep-sea diving suit, which was used by the Navy until the mid-1980s, is almost an exact copy of Siebe's original 1837 design, except for some refinements in materials and improvements in the valves. Navy instruction with the Mark V ceased in 1982, and it was officially replaced by the Mark XII in 1986. A number of commercial harbor divers still use this device, however.

The classic deep-sea diving suit remained unchallenged until approximately 1945, when a lightweight diving mask for work down to depths of 90 to 100 ft was introduced. This was designed by a Milwaukee diver, Jack Browne, and was manufactured for the U.S. Navy. It subsequently became widely used among commercial divers, especially in the Gulf of Mexico.

It was also at the end of World War II that the self-contained underwater breathing apparatus (scuba) first made its appearance outside of occupied France. It had been invented in 1943 by Emile Gagnon and Jacques Cousteau. The Cousteau-Gagnon patent had at its heart a demand regulator that automatically delivered only the amount of air the diver needed at any depth to which he dived. This simple but ingenious device presaged the current boom in sport diving and was adapted for a number of commercial applications. Since 1960, there have been many advances made in deep-sea diving equipment, with the use of more modern helmets made of space-age materials, hot-water–heated suits for thermal protection, and combinations of diving bells and diving suits.

DECOMPRESSION SICKNESS

Sir Robert Boyle provided the first hint as to the cause of decompression sickness in 1670

when he produced symptoms of decompression sickness in a snake that had been placed in a vacuum chamber. He was prompted to write: "I once observed a Viper furiously tortured in our Exhausted Receiver … that had manifestly a conspicuous Bubble moving to and fro in the waterish humour of one of its eyes." Thus, Boyle noted that rapid reduction of ambient pressure may result in the production of bubbles in the tissues of the body.

The first description of the symptoms of decompression sickness in humans was provided by Triger in 1841. The victims in this case were coal miners who worked in mines pressurized to keep out the water. Triger noticed that some men suffered cramps and pains in their muscles after leaving compressed air, and apparently their symptoms were treated vigorously with cognac ("spirits of wine") given both internally and rubbed on externally. We have no report as to how they later fared.

In 1854, Pol and Watelle began to study the phenomenon of decompression sickness. They noticed that this disease was always associated with leaving the compressed air environment. "One pays only on leaving," they wrote. They also noted that a return to compressed air alleviated the symptoms. They pointed out that young men of 18 who had "not reached their greatest mature physical strength" suffered less from decompression sickness symptoms than those in their mid-30s "who were in their prime." The first scientific approach to the problem of decompression sickness was begun by the French physiologist Paul Bert, when he published his monumental book, *Barometric Pressure,* in 1878. Bert was able to demonstrate that bubbles associated with symptoms of decompression sickness were formed during rapid decompression and, furthermore, that these bubbles consisted mainly of nitrogen. Bert also discovered that oxygen is toxic when breathed under pressure; the convulsions that occur when oxygen is breathed for any period of time at pressures greater than 33 ft have been called the "Paul Bert effect."

The word *bends* as a synonym for decompression sickness came into being during the construction of the piers for the Brooklyn Bridge. The fashionable ladies of the era had an affected posture for walking called "the Grecian bend." Workers emerging from the caisson, limping with symptoms of decompression sickness, were chided by their fellows for "doing the Grecian bend." This was later shortened to simply "the bends" and subsequently became legitimized by use.

Although Pol and Watelle had recognized that reexposure to compressed air ameliorated symptoms of decompression sickness, there is no recorded evidence that they used it as a treatment. Andrew Smith, a throat specialist at the Manhattan Eye and Ear Hospital, who was engaged as medical advisor for the Brooklyn Bridge caisson work, observed the same thing but called such treatment "the heroic mode" and never applied it either. The reason for this is that putting a bends victim back into compressed air seemed to be homeopathic treatment. Because compressed air was known to cause the disorder, physicians were loath to recommend more of it for cure.

It remained for E. W. Moir, a British engineer, to first utilize purposeful recompression for treatment of bends. In 1889, efforts were being made to drive railroad tunnels underneath the Hudson River. At the time Moir took over as project superintendent, the death rate from decompression sickness among the workers was 25% per year. Moir erected a recompression chamber at the job site and promptly recompressed any worker with symptoms—followed by a slower decompression. Although in his own description of his work he admitted his treatment was homeopathic, he reduced the mortality rate to 1.6%.

By the turn of the century, even though the cause of decompression sickness was known to be nitrogen bubbles evolving within the body and the symptoms could be relieved by returning to increased pressure, there were no decompression schedules that could be followed to minimize the possibility of decompression sickness occurring. The Royal Navy consistently used divers in its routine operations, and so it commissioned J. S. Haldane to work out a set of decompression schedules that could be written down in tabular form and followed by its fleet divers. In 1908, Haldane published the first set of practical, though empirical, decompression schedules. In his work, Haldane demonstrated that the body could tolerate a two-to-one reduction in ambient pressure without symptoms. All common decompression schedules in use since have been based on Haldane's method.

The Haldanian schedules were found to be quite realistic over their middle range, but

divers soon found that it was possible to "cut corners" on short, shallow dives without risking bends and that on long, deep dives, the Haldane tables were not conservative enough. Haldane's tables were modified empirically over the years to solve these problems. Haldane must also receive the credit for developing the concept of half-time tissues; he realized that all of the tissues of the body absorb nitrogen at varying rates, depending on their vascularity and the types of tissue involved. Recognizing that this was a spectrum that probably went from seconds to hours, he arbitrarily chose to recognize the existence of 5-, 10-, 20-, 40-, and 75-min half-time tissues for mathematical convenience in calculating nitrogen uptake and elimination. He assumed that nitrogen uptake and elimination occurred at equal rates and that the longest half-time tissue in the body was probably 60 min. He therefore assumed that the body would essentially achieve total saturation in 6 hours. However, he made his longest tissue 75 min just to be on the safe side. Since that time, the U.S. Navy standard air decompression tables have been based on a 12-hour period for total saturation, and the exceptional exposure air tables have been based on a 24-hour time period for total saturation. Even longer tissue half-times have been developed for saturation diving.

INCREASING DEPTHS AND EXPERIMENTS WITH HELIUM-OXYGEN BREATHING

In 1915, the United States Submarine F-4 sank in 306 ft of water off Honolulu. The U.S. Navy was anxious to recover the submarine and bodies of its crew, and thus diving operations were commenced. In that year, Frank Crilley set a world depth record of 306 ft by descending to the submarine and attaching a large hawser to it. The pressures at such depths are enormous, having been enough to completely crush the sides of the submarine and to reveal the outlines of the diesel engines beneath. The fact that Crilley was able to dive to this depth and return to the surface alive, using the primitive decompression schedules then employed, was astounding. Perhaps Crilley's size had something to do with it: He weighed only 127 pounds. Three hundred feet is still about the extreme limit for compressed air diving; the nitrogen narcosis at that depth renders all but the most experienced divers incapable of any kind of useful work. The current U.S. Navy maximum operating depth for compressed air diving is 190 ft.

Because air seems to have a limit of approximately 300 ft, the physiologist Elihu Thompson wrote a letter to the Bureau of Mines in 1919 suggesting that helium mixed with oxygen might be used as a diving gas. Because helium is so much lighter than nitrogen, he thought that, with the decreased breathing resistance, permissible diving depth might be doubled. Nitrogen narcosis was still not understood in 1919. The British Admiralty, along with the United States Bureau of Mines, began experimenting with helium-oxygen mixtures and thought that bends might be avoided because nitrogen was no longer in the breathing mixture. However, because Royal Navy divers experienced severe decompression sickness after breathing helium, even when decompressed on conservative air decompression schedules, they concluded that it was unsafe as a diving gas and ceased further experiments. The U.S. Navy Experimental Diving Unit, which had worked with the Bureau of Mines on helium, also abandoned its studies of helium in 1924 because helium seemed to produce decompression sickness more quickly than when compressed air was breathed. In Admiral Momsen's words, experimentation with helium diving was "put very much on the back burner." Because of the necessity to dive to great depths on occasion for military operations, Damant extended the original Haldane air schedules to 320 ft in 1930.

NEW DEVELOPMENTS

Occasionally, divers returning to the surface from trivial depths (<33 ft) suffered sudden incapacitation. This was attributed to a capricious form of decompression sickness, and indeed the U.S. Navy reported two cases of "unusual decompression sickness in 16 ft of water" in the mid-1930s. Both of these cases proved fatal, but the mechanism of death was not understood. Submarine escape training began in the U.S. Navy at the beginning of the 1930s, and occasionally even with the use of the Momsen lung, trainees would experience severe distress or

die quickly after surfacing. Further investigation revealed that death in these cases was due to overdistention of the lungs, with subsequent rupture and escape of air into the pulmonary veins. From the pulmonary veins, the air bubbles were directed to the left heart and thence to the brain. Cerebral air embolism became recognized for the first time. When it was understood that air bubbles in the brain were the cause of the symptoms and that nitrogen alone was not involved, immediate recompression to 165 ft became the standard treatment, and the victims of air embolism were treated as though they had severe decompression sickness. Most of them survived when immediately recompressed, and eventually recompression chambers were installed at the top of the submarine escape training towers in New London, Connecticut, and in Honolulu to handle such cases.

Meanwhile, Albert R. Behnke, a U.S. Navy Submarine Medical Officer and an outstanding scientist, became interested in the problem of mental deterioration when the divers exceeded depths of 150 ft. Using mixtures of gases other than nitrogen, he demonstrated that heavier inert gases produce more narcosis and that nitrogen produces mental deterioration in air diving. Behnke also demonstrated that high levels of CO_2 contribute to nitrogen narcosis but that nitrogen itself is the culprit. He showed that the narcotic potency of any inert gas is predicated on its oil-water solubility ratio and, like the inhalation anesthetics, followed the Meyer-Overton hypothesis for predicting anesthetic effect.

HELIUM REVISITED AND NEW DEPTH RECORDS SET

In 1937, Edgar End, a 26-year-old intern at the Milwaukee County General Hospital, thought that helium could be used successfully to avoid nitrogen narcosis. He was undeterred by the fact that both the British Admiralty and the U.S. Navy Experimental Diving Unit had been unable to successfully adapt helium for diving. By performing some original calculations, he developed a set of helium decompression schedules that he thought would be compatible with this rapidly diffusing gas. Together with Max Gene Nohl, a friend and a Milwaukee diver, End breathed helium-oxygen in an old recompression chamber located at the Milwaukee County Emergency Hospital. The two men found that they could surface safely from depths of 100 ft after various exposures breathing helium. Using Nohl's self-contained suit, they conducted a series of open-water dives in Lake Michigan to increasing depths until finally they surpassed Frank Crilley's record and set a new world depth record of 420 ft in December of 1937, diving from a Coast Guard cutter off Port Washington, Wisconsin. Nohl surfaced safely without signs of decompression sickness. After End and Nohl proved that helium could be used successfully for deep diving, the Navy stepped up its own interest in helium/oxygen experimentation. By 1939, a series of helium/oxygen decompression schedules that had been developed by Behnke were ready. The helium/oxygen equipment had been sent to a warehouse at Kittery, Maine, for field-testing in the summer of 1939 when the submarine *U.S.S. Squalus,* operating out of Portsmouth, New Hampshire, sank off the Isles of Shoals in 240 ft of water.

The submarine was quickly located, and the first dive was made on compressed air. The downhaul cable to the torpedo room hatch had parted, and a compressed air diver was too confused to replace it. A diver breathing helium then went down and accomplished the task with ease. Some 36 men were rescued from the submarine using the McCann Rescue Bell, and then the actual salvage of the submarine was carried out using the new helium/oxygen schedules and equipment. Over 100 helium dives were made on the *Squalus,* and it is remarkable that with this first venture in deep water with a new gas, not a single diver was killed or seriously injured. For the next 20 years, the U.S. Navy was to be the only user of helium/oxygen diving (the United States had the only readily available sources of helium), and all Navy submarine rescue vessels were equipped with helium/oxygen diving gear.

In April 1945, the previously mentioned Jack Browne, son of a Milwaukee automobile dealer, had become interested in diving and thought that a practical diving mask could be more useful than the heavy and cumbersome standard deep-sea dress. He devised a triangular mask, and in a wet test tank at the Diving Equipment and Supply Company in Milwaukee, Wisconsin, descended to a new world depth record of

550 ft. The decompression schedules for this dive were worked out by Edgar End with some modifications by Behnke, who was also present.

It was also in 1945 that the Swedish engineer Arne Zetterström investigated the possibilities of using a mixture of hydrogen and oxygen for diving. Hydrogen-oxygen is nonexplosive when the oxygen percentage is less than 4%. Zetterström reached a depth of 526 ft in the Baltic Sea in August 1945, and the hydrogen-oxygen mixture was perfectly satisfactory as a breathing mix. Unfortunately, he was killed on ascent because of a winch accident that had nothing to do with his breathing mixture. Hydrogen diving was not attempted again until the 1970s, when Peter Edel in New Orleans began experimenting with gas on a contract from the U.S. Navy.

DEVELOPMENT OF TREATMENT TABLES FOR DECOMPRESSION SICKNESS

Since E. W. Moir first introduced recompression as treatment for bends in 1889, there have been many schools of thought as to what the best treatment schedule should be. Some thought that divers should be returned to their original working pressure; others held that divers should be taken to the depth of relief; still others thought that the treatment pressure should be the depth of relief plus 1 atm. Then there were many schemes for gradually reducing the pressure on divers so that they would not sustain decompression sickness during ascent in the treatment chamber.

In 1944 and 1945, the U.S. Navy studied all of these methods and soon promulgated the U.S. Navy Air Recompression Tables 1 through 4 (see Chapter 10 for further discussion). These tables represented a ninefold improvement over previous recompression procedures and became the world standard of treatment for the next 20 years. They embodied the concept that the diver should be taken to depth of relief plus 1 atm as a minimum, with a 6 atm maximum, as a trade-off between maximally compressing any offending bubbles and causing too much nitrogen narcosis and too much extension of subsequent decompression time. For serious symptoms, they provided a "12-hour soak," sometimes known as the "overnight soak," at the 30 ft stop on return to the surface so that all tissues could theoretically be equilibrated to 30 ft. In line with Haldanian theory, decompression to the surface could then be safely made without exceeding a 2:1 ratio for any tissue. However, to be cautious, several more hours were taken to allow decompression from 30 ft. Tables 1 through 4 proved themselves fairly successful when used to treat decompression sickness stemming from dives carried out on standard Navy schedules. Air was used as the breathing medium throughout the tables, but oxygen was later introduced for use in the shallower stops. The shortest of the air tables, Table 1A, took 6 hours and 13 min, and Table 4 took 38 hours. The length of these schedules did not make them popular with divers but represented the only escape from unbearable pain, paralysis, or both. The reason for the length of the tables was the addition of iatrogenic nitrogen to the patients' tissues as a consequence of treatment.

In 1947, Edgar End, still active in the diving field, began treating caisson workers in Milwaukee with oxygen, using the rationale that gaseous nitrogen was the cause of the patients' symptoms and that the addition of more nitrogen to the patients' tissues, when taken to great depth, only prolonged treatment time. He generally treated his patients for 1 to 2 hours at 30 lbs (67 ft) and then decompressed them. His experience with some 250 cases was excellent, but his data using this method remained unpublished.

Since 1947, no diver or compressed air worker has been treated for bends in Milwaukee with compressed air treatment; only oxygen has been used.

SATURATION DIVING

When a diver goes to depth under water, the inert gas or gases breathed—nitrogen, helium, or even hydrogen—begin going into solution in the tissues. After many hours at a given depth, probably in excess of 24 hours, no more gas enters the diver's tissues and a state of equilibrium is reached. The tissues are then totally saturated. After that time, the decompression obligation is the same whether the diver stays under water for 2 days or 2 weeks. This is commercially

useful because the diver does not waste time every day decompressing.

The first intentional saturation dive was carried out by Edgar End and Max Nohl in Milwaukee at the County Emergency Hospital recompression chamber on December 22, 1938, when they spent 27 hours breathing air at 101 ft. They underwent decompression fairly successfully in about 5 hours, with only Nohl experiencing decompression sickness. These bends symptoms were treated with moderate pressures of air with complete relief. The reason for this experiment was that horses and mules used for hauling muck cars in compressed air tunnels were often kept in the tunnels for the full length of the contract, which might last many weeks or months. Attempts at decompressing the animals without them experiencing severe and disabling decompression sickness had been unsuccessful, so that animals were usually killed before decompression. End reasoned that, given enough time, decompression from saturation could be successful—hence the experiment on himself.

Practical saturation diving was first conceived in 1957 by the late Captain George Bond of the U.S. Navy when working in the Submarine Medical Research Laboratory in New London, Connecticut. Captain Bond (then Commander Bond) envisioned undersea laboratories located at various depths down to 600 ft on the continental shelf. He calculated that by breathing helium, scientists could work at full sea pressure in these laboratories studying physiology, submarine geology, and marine biology for prolonged periods. They could then be transferred under pressure by submarine vehicle to a shallower habitat, where they could continue their studies while undergoing decompression. Several habitats would be used, each one at a shallower depth, so that finally scientists could emerge with minimal decompression after completing their tour of study, which might last weeks.

It was first necessary to demonstrate that animals could tolerate saturation exposures. These research efforts were called Project Genesis and, after further work at the Experimental Diving Unit in Washington under the direction of R. D. Workman, saturation decompression schedules were devised for humans. These were later tested in the open sea on Projects Sealab 1 and Sealab 2. In 1962, Ed Link saturated a diver for 24 hours at a depth of 200 ft in the Mediterranean. Captain Jacques Cousteau also established saturation habitats in his "Con Shelf" series.

In 1965 commercial saturation diving began when Westinghouse, using their Cachelot diving system, worked at 200 ft on the Smith Mountain Dam in Virginia to replace faulty trash racks. Divers were saturated for periods of up to 5 days on this job. Since then, saturation has become commonplace, especially in oil field work, where periods of saturation up to 2 weeks are routine and 1-month saturations have occurred.

COMMERCIAL HELIUM DIVING

With the advent of offshore oil production, diving services were required in deep water, especially on the West Coast of the United States. Diving companies usually hired local abalone divers to handle various odd jobs associated with drilling rigs, but when pressures of 250 ft were reached, the compressed air equipment used by the commercial divers caused nearly prohibitive nitrogen narcosis. Dan Wilson, an abalone diver from California, decided that helium-oxygen was necessary. In 1962, using a Japanese abalone deep-sea diving dress and a special oronasal mask, he made the first modern civilian helium dive to a depth of 420 ft. Within a year, he was contracting helium-oxygen diving services to oil companies in the Santa Barbara area.

On the Gulf Coast, the oil rigs were also moving into deeper water, and Edel calculated the first helium-oxygen schedules for use in the Gulf in 1963. With the demand for deep-sea commercial diving accelerating rapidly, civilians developed new helium equipment, and commercial helium diving capabilities soon outstripped those of the U.S. Navy. Bell diving also came into vogue as a means of delivering the commercial diver to the work site.

In all fairness to the U.S. Navy, it must be stated that in the early 1960s, those responsible for Navy budgeting could not identify the operational necessity for deeper helium diving or improved helium diving equipment. It was only in the early 1970s that the U.S. Navy again became active in doing frontline research in this area.

LOW-PRESSURE OXYGEN TREATMENT OF DECOMPRESSION SICKNESS

By 1964, the Navy noted that the failure rate for bends treatment using the schedules in Tables 1 through 4 began to rise sharply. This was because the Navy was called on to treat more civilian scuba divers who had failed to observe any kind of standard decompression schedules. In 1964, the failure rate on the initial recompression for serious symptoms had risen to 47.1%. Workman and Goodman of the U.S. Navy Experimental Diving Unit reinvestigated the use of oxygen under low pressure as the primary treatment modality for decompression sickness. Oxygen had been suggested by Behnke in 1939 as a promising treatment method after starting with a brief excursion to 6 atmospheres absolute (ata). After 3 years of work, the U.S. Navy promulgated the low-pressure oxygen Tables 5 and 6 on August 22, 1967. At the same time, Tables 5A and 6A for treatment of air embolism were published (see Chapter 10). The treatment times required for decompression sickness were drastically reduced, and the maximum depth of treatment was only 60 ft (26.7 psig). Table 5 took only 135 min and had a failure rate on the initial recompression of only 1%. For serious symptoms and recurrences, Table 6 took only 285 min, and the failure rate on the initial recompression fell to only 3.6%. The use of Tables 1 through 4 has now been nearly abandoned. Continued experience with the low-pressure oxygen tables revealed frequent recurrences of decompression sickness with the shorter Tables 5 and 5A, and these also have been abandoned or have seen limited use.

More recent animal research by Leitch and others has shown that little or no advantage is gained by going to 165 ft (6 ata) while breathing air (per Table 6A) in the treatment of embolism, and in fact it may do more harm than good. For this reason, most facilities now use mixed gas containing 50% oxygen-nitrogen or helium-oxygen instead of air with the 6 ata treatment depth. The importance of using high partial pressures of oxygen in treatment of bubble-related diving disorders has been well established. The use of helium-oxygen mixed gas at pressures greater than 2.8 ata was beginning to supplant nitrogen-oxygen mixtures by 1995. Also, pressures greater than 2.8 ata using high concentrations of oxygen had been shown to produce better results than Table 6 in some very complicated or delayed cases.

NEW PRESSURE RECORDS

In the mid-1960s, Hannes Keller, a Swiss experimental diver, reached a depth of 1000 ft in the open sea using a proprietary blend of gases, and the race for increasing depth was on. In 1970, the British reached a depth of 1500 ft in a dry chamber at the Royal Navy Physiological Laboratory at Alverstoke, England, using helium-oxygen as the breathing mixture. A new phenomenon appeared called the *high-pressure nervous syndrome* or (HPNS). It was discovered that rapid compressions to depths in excess of 500 ft could bring on uncontrollable shaking and nausea in the divers breathing helium. Bennett found that slow compressions could be used to minimize this problem, and the 1970 British dive was accomplished using several days to reach maximum depth.

The French were in strong competition with the British. Using slow compression, they set a record of 1700 ft in the dry chamber in 1971. Again in 1972, the French set a new record of 2001 ft in the dry chamber at the Comex facility in Marseille. The U.S. Navy, using its Mark 1 deep-sea diving system, set an open sea depth record of 1010 ft off Catalina Island in June of 1972. Commercial work has been performed at depths exceeding 1300 ft. The current pressure record is 2300 ft (701 m), set in 1992 at Comex in the dry chamber. They used a mixture of hydrogen, helium, and oxygen to minimize HPNS and lower breathing resistance. At Duke University, Bennett had discovered that adding 10% nitrogen back to the gas mixture could alleviate most of the clinical symptoms of HPNS. This finding enabled compression to 1000 ft in less than half an hour without symptoms. Later it was found that hydrogen has a narcotizing or anti-HPNS capability slightly less than nitrogen, but being much lighter and less dense, considerably eased the work of breathing when mixed about half and half with helium. At depths over 2000 ft breathing helium-nitrogen-oxygen, half of the diver's energy was expended on the work of breathing.

RECENT DEVELOPMENTS

Universities with oceanography programs took an interest in diving, and civilian saturation diving for research purposes gained prominence in the late 1960s and the early 1970s. The U.S. Navy, along with other agencies of the government, sponsored the Tektite series of saturation dives to depths of 50 ft in the Caribbean. The Tektite divers breathed normoxic nitrogen-oxygen mixtures. Hydrolab was established by the Perry Submarine Company off Freeport, Grand Bahama, at depths of 42 and 60 ft. Dozens of scientists have been saturated for periods of up to 2 weeks in this habitat breathing compressed air. Hydrolab now resides in the Smithsonian Institution. The Puerto Rican International Underwater Laboratory was built with a saturation capability to 100 ft. The Tektite 2 series saw the first all-woman team of aquanauts carry out scientific research while saturated. Saturation on air deeper than about 60 ft cannot be carried out because of primary oxygen toxicity considerations. Deeper than that, mixed gas with a lesser partial pressure of oxygen must be used.

Tri-gas mixtures became of interest commercially in the 1960's, and André Galèrne of International Underwater Contractors pioneered their use. These mixes consist of helium, nitrogen, and oxygen and are being used commercially more and more. Neon and helium have been used experimentally. The French, successfully experimenting with mixtures of hydrogen and helium, reached depths in the open sea greater than 1750 ft in March 1988.

Commercial contracts for deep diving have become more sophisticated, and by 1974 contracts called for diving services to depths of 1500 ft in support of offshore oil production, if needed.

Studies to define limits and protect divers exposed to increasing hydrostatic pressure continue, but another development has been the use of "1 atm diving systems" for deep diving. These are basically submarines with manipulators to allow the operator to work at great depth while the interior is maintained at 1 ata and environmental control systems maintain safe physiologic parameters. They range from armored one-person 1 ata suits (e.g., Jim, WASP, and so forth) to submersibles, which allow for more than one occupant.

FUTURE RESEARCH

Diving depths to 3000 ft are now being considered with tri-gas mixes. Hydrogen as a diving gas, under active investigation by Comex in France, is showing great promise, and the blood changes in decompression sickness are beginning to be quantified. The first symposium on blood changes in bends was conducted in Toronto in 1973.

Today, more attention is being paid to the study of the actual elimination curves of inert gas during decompression, but empiricism (using computer data) is relied on more than mathematical models for devising decompression tables. Future research will undoubtedly provide answers to the exact mechanism of inert gas elimination from the body and what the tolerable limits of tissue trauma may be during this process.

CHAPTER 2 Diving Physics

Larry "Harris" Taylor

If one is to function normally and minimize risk while exposed to the underwater environment, where breathing requires a support system, one must understand the physical aspects of that environment. This chapter defines physical concepts and presents methods for solving problems related to diving and exposure to the underwater domain.

The physical environment is understood through the interactions of five fundamental properties: length, time, mass, force, and energy (Table 2–1).

Measurements are generally based on one of two systems. *English system units* were derived from human anatomy or arbitrary measurements: the *foot* (standard of length since the Romans), the *yard* (girth around a tenth-century Saxon king), the *digit* (width of a finger), the *palm* (four digits), the *span* (distance between the outstretched thumb and the little finger, equal to 3 palms), the *cubit* (distance between the elbow and the tip of the middle finger, equal to 2 spans or 6 palms), the *pace* (one step, equal to 10 palms), the *fathom* (distance between two outstretched hands, equal to 6 palms), and the *rod* (allegedly the length of a line in front of a medieval English pub, approximately 16½ ft). Volume measurements in many areas were based on the amount of water from the Scottish river Tay. For example, the *boil* (equal to 12 English gallons) was the amount of clear water from the river Tay that weighed 164 pounds. The English system was formally "defined" in the reissue of the Magna Charta in 1225, when Henry III agreed to have one measure throughout the realm for wine, ale, and corn. In 1324, the *inch* was defined as the length of "three round and dry barley corns, laid end-to-end." Because the English system represents a collection of measurements evolved from the merging of many cultures over thousands of years, it has a multitude of possible measurements and no apparent logical system of conversions.

The *metric system,* on the other hand, was specifically developed to make conversions between units simple. In the metric system, all units are related by factors of 10. The originator is considered to be Gabriel Mouton, who proposed a decimal system of units in the year 1670. In 1790, the French Academy developed a system of measures based on astronomic (believed to be invariant), instead of human anatomic, measures. Their standard, the *meter,* was chosen from the Greek word *metron,* meaning "measure." The metric system was propelled into reality during the time of the French revolution. The English system is still used in the United States; the metric system is used nearly everywhere else.[1]

Table 2–1. Fundamental Measures Used in Physics

Length	Distance between two points
Time	Measurement of duration
Mass	Property of matter which resists a change in movement
Force	Push or pull that tends to produce a change in movement
Energy	Ability to do work

LENGTH

The standard unit of length in the English system is the foot. The unit of length in the metric system, the meter, was historically defined as 1/10,000,000 of the distance from the earth's equator to the north pole. Unfortunately, there was a slight error in this approximation. This error, coupled with a need for a scientifically precise definition that would be unaffected by changes in temperature, humidity, or pressure or be subject to chemical corrosion over long periods of time, led the scientific community to adopt a measurement based on atomic spectroscopy.

Table 2–2. Some Useful Conversions

6 feet	=	1 fathom
5280 feet	=	1 statute mile
6076 feet	=	1 nautical mile
1.0 inch	=	2.54 centimeters
3.28 feet	=	1 meter
14 pounds	=	1 stone
1 kilogram	=	2.2 pounds
453.6 grams	=	1 pound
1 gallon	=	0.134 ft^3
1 ft^3	=	28.316 liter
1 liter	=	1.06 quarts
1 liter	=	0.0353 ft^3
1 joule	=	1 newton-meter = 0.7376 foot-pound
1 erg	=	1 dyne-centimeter
1 joule	=	107 ergs
1 watt	=	1 joule/second
1 kilowatt	=	1.34 horsepower
1 horsepower	=	550 foot-pounds/second
1 horsepower	=	746 watts

The nature of the task at hand determines the level of precision required for the measurement. A diver estimating the in-water distance to the charter boat, a nautical archeologist wishing to return to the same location at sea, and an astronaut wishing to return to the earth from outer space all need estimates of distance. In general, the greater the consequence of error, the more precise the measurement must be. Table 2–2 provides several length relationships.

TIME

In both the English and metric systems, time is based on the *second.* A second, historically determined by astronomic measurements, is based on the rotation of the earth—specifically, the duration of 1/86,400 of a mean solar day. Unfortunately, this period varies. Although this variance is insignificant to divers, it does affect navigation. Longitude (distance east or west of the Greenwich Meridian) is determined by measuring the time difference between the observer and the Meridian. Historically, ocean navigation and exploration were hindered by the lack of precise clocks. The imprecision in the determination of time meant a corresponding uncertainty in position at sea. Much of the success of modern navigation is due to the development of precise time standards. The current state-of-the-art standard is the "atomic clock." With this device, a second is the duration of 9,192,631,770 periods of the radiation corresponding to the transition between the two hyperfine levels of the ground state of the cesium-133 atom. This clock is accurate to within 1 second per 300,000 years. A newer version, which uses a high-precision laser to excite the cesium atoms, is accurate to within 1 second every 3 million years. This is contrasted with a typical dive watch, which may be accurate to within a few seconds per month.

MASS AND WEIGHT

Mass and weight are often confused. Physics makes a rigorous distinction between *mass,* an intrinsic property of matter that is a reflection of the total number of atoms present in a substance, and *weight,* the result of force operating on that mass. Mass refers to the property of matter that resists change in movement. For example, a moving boat continues to move in the water after the motor has been turned off. This tendency of the boat to resist change in movement is called *inertia.* The property of matter that provides this resistance to change in motion is called *mass.* In the metric system, the unit of mass is the *kilogram.* The corresponding English unit of mass is the *slug.* Weight is the result of some force (i.e., gravity) acting on mass. The unit of weight in the metric system is the *newton;* the unit of weight in the English system is the *pound.* Historically, these units have been confused and, aside from the scientific community, slugs and newtons are seldom used. Additionally, some in the scientific/technical community use *poundsf (lbf)* and *poundsm (lbm)* to distinguish between a unit of force (weight) and a unit of mass.[2] For example, an astronaut weighs less on the moon than on the earth because the gravitational force on the moon is less than on the earth. Mass (number of atoms represented as kilograms or slugs) does not change even though weight (in newtons or pounds) is less.

In practice, divers do not make the scientifically rigorous distinction between mass and weight and they refer to both kilograms and pounds as units of weight. If unsure whether the scientific use requires "mass" or "weight," consider the following: Weight is a force; it has a magnitude (mass) and a direction. If direction, as in a buoyancy calculation, is an important consideration in

understanding the nature of the problem, then the appropriate term is *weight.* If direction is not a factor, as in a gas law problem, then *mass* is the proper term.

VOLUME

Volume is the term used to describe capacity. It is measured in units of length cubed.

UNITS IN SOLVING PROBLEMS

The calculations used in diving physics are similar in either English or metric units. However, because the numeric values will differ, it is important to use the same measurement system during the entire calculation. For example, the formula relating depth in feet and pressure in atmospheres:

$$\text{Absolute Pressure} = \frac{\text{Depth}}{33} + 1$$

is valid only in seawater at sea level with depth measured in feet of seawater. Because some divers dive in other conditions, or with gauges calibrated in standards other than feet in seawater (one major American manufacturer has a series of depth gauges calibrated in feet of fresh water), or in locations other than sea level, this text uses a more general approach that emphasizes an understanding of hydrostatic and absolute pressure in all environments.

Water

Water is present in air as a gas. The amount of water that air can hold is proportional to the temperature; the higher the temperature, the more water vapor the air can hold. The amount of water in the air expressed as mass of water per unit volume is called the *absolute humidity*. The amount of water vapor present divided by the maximum possible water vapor concentration at a given temperature is called the *relative humidity.* Relative humidity is expressed as a percentage. Another measure of the total water vapor present is the *dew point,* the temperature at which the air can no longer hold the amount of water vapor present in the air and at which condensation begins. For example, as the temperature lowers during a humid evening, the dew point is reached and fog forms. As long as the temperature is above the dew point, fog does not occur because the water remains in the gaseous state.

With diving, it is desirable to have the humidity of the breathing gas as low as possible to prevent the formation of ice within the first stage of the regulator. Whenever air passes from a high pressure (the scuba cylinder) to a low pressure (the hose between the first and second stages) through an orifice, the air expands and the temperature drops. This is known as the *Joule-Thompson effect* (discussed later). During rapid gas flow (as with pressing the purge button or when sharing air via a common first stage), this temperature drop can be substantial (–80°F or more). If the humidity of the gas is high, then water vapor can condense and freeze. It is possible to freeze the regulator in either an open or a shut position. If the valve freezes open, a free flow results; if the valve freezes closed, the air supply is shut off.

Water vapor in expired air passing through a cold regulator can condense and freeze to mechanical parts inside the second stage of a demand regulator. This can result in second stage free-flow problems. When one is diving in extreme cold conditions, it is best not to exhale through a regulator until after it is beneath the surface of the water and allowed to equilibrate to ambient temperature.

Air

Air is a mixture of gases that primarily includes nitrogen and oxygen. It also contains water vapor, varying concentrations of trace gases (e.g., argon, neon, xenon), carbon dioxide, industrial pollutants, hydrocarbons and nitrous oxides from internal combustion engines, and particulate matter (Table 2–3).

Table 2–3. Typical Concentration of Dry Air

Component	% by Volume
Nitrogen	78.084
Oxygen	20.946
Argon	0.934
Carbon dioxide	0.033
Other gases	0.003

For most diving applications, divers may assume that air is 78% nitrogen, 21% oxygen, and 1% other gases.

DENSITY

Density is defined as the mass per unit volume. Expressed as a formula:

$$\text{Density} = \text{Mass} \div \text{Volume}$$

Density, particularly of liquids and gases, changes with temperature. Specific gravity relates the mass of material to an equal volume of water. Water has a density of 1.000 g/cc at 4°C. The density is slightly less than 1.000 g/cc at all other temperatures. Density has units of mass/volume; specific gravity is a ratio and has no units.

$$\text{Specific gravity} = \frac{\text{Mass of the Object}}{\text{Mass of an Equal Volume of Water at } 4^\circ\text{C}}$$

Divers do not operate at temperatures at which density and specific gravity are significantly different; thus, for most applications, these terms may be interchanged. The specific gravity of pure fresh water may be assumed to be 1.00; the specific gravity of seawater is 1.03.

BUOYANCY

Archimedes, the Greek mathematician, first stated what has become known as Archimedes' principle:

An object partially or wholly immersed in a fluid is buoyed up by a force equal to the weight of the fluid displaced by the object.

Thus, objects more dense than water will sink; objects less dense than water will float; objects of the same density will remain at the same level and neither sink nor float. Objects that sink are frequently called *negatively buoyant.* Objects that float are called *positively buoyant.* Objects that remain stationary at depth are said to be *neutrally buoyant.*

Buoyancy is best understood by the application of force vectors. Vectors are mathematical constructs that have magnitude and direction. Weight is a downward force (gravity acting on mass); buoyancy is an upward force. If these two forces are balanced, then neutral buoyancy is achieved. If they are not balanced, then the object immersed either sinks or floats.

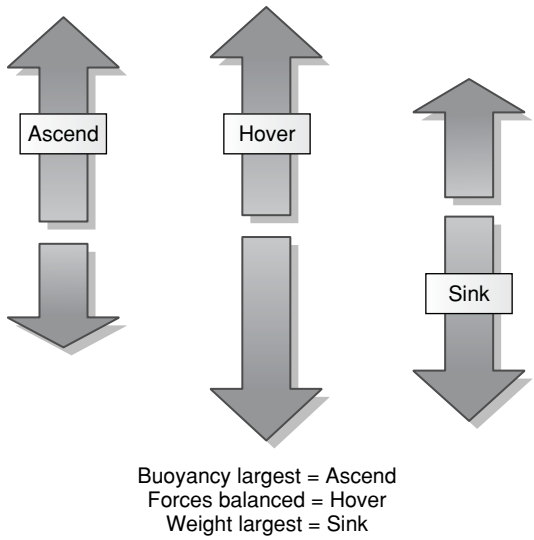

Figure 2–1. Relation of buoyancy *(upward arrows)* to weight *(downward arrows).*

Divers commonly are imprecise in the use of the term buoyancy. Rigorously, buoyancy is defined as an upward force directed against the force of weight. Although commonly used in the diving community, the terms *neutrally buoyant* and *negatively buoyant* are rigorously improper; the term *positively buoyant* is redundant. Buoyancy is much easier to understand if one considers only the balancing of an upward force (buoyancy) and a downward force (weight). This scheme allows for no positive or negative. We use the term *hover* to refer to the so-called neutrally buoyant state. Thus, objects either float, hover, or sink. If weight is greater than buoyancy, the object sinks (Fig. 2–1).

Buoyancy calculations involve three factors: the weight of the object being submerged, the volume of the object submerged, and the density of the liquid. Any two of these factors can be used to determine the third. This is best illustrated by example:

Example

What is the buoyancy in seawater of a piece of wood that weighs 2000 lb and measures 6 ft × 2 ft × 3 ft?

Determine forces involved:

a. The weight of wood = 2000 lb

b. The volume of wood = 6 ft × 2 ft × 3 ft = 36 ft^3

c. The corresponding weight of an equal volume of seawater

$$36\ ft^3 \times \frac{64\ lbs}{ft^3} = 2304\ lbs$$

Weight of wood = Downward force	=	2000 lbs ↓
Weight of water = Upward force displaced	=	2304 lbs ↑
Net force	=	304 lbs ↑

The object will float with a buoyant force of 304 lb. To sink, the object would have to weigh more than an additional 304 lb (without changing volume). Although the object is buoyant (i.e., a net force of 304 lb is pushing up on this log), it will not be completely out of the water. The density of the log can then be used to determine how much of the log will be submerged:

$$\text{Density of log} = \frac{2000\ lbs}{36\ ft^3} = 55.6\ \text{lbs/cubic foot}$$

Since this log is less dense than seawater, it will float. The amount of the volume that is submerged will be determined by the ratio between the density of the log and the density of the seawater. In general:

$$\text{Ratio: Volume submerged} = \frac{\text{Density of object}}{\text{Density of liquid}}$$

Substituting the value of this log above and seawater:

$$\text{Ratio} = \frac{55.6\ lbs/ft^3}{64\ lbs/ft^3} = 0.869$$

About 87% of the log's volume will be submerged.

Example

A fully suited diver weighs 200 lb. This diver displaces a volume of 3.0 ft^3 of seawater. Will the diver float or sink?

Determine forces involved:

$$3.0\ ft^3 \times \frac{64\ lbs}{ft^3} = 192\ lbs$$

Weight of diver	=	200 lbs ↓
Displaced weight	=	192 lbs ↑
Net force	=	8 lbs ↓

The diver will sink. This diver weighs 8 lb in the water and is overweighted.

Example

A fully geared diver in a wet suit weighs 210 lb. In fresh water, this diver with a scuba cylinder containing 500 psig needs 18 lb of lead to hover. How much lead will this diver need when diving in a wet suit in seawater?

Weight of diver	=	210 lbs ↓
Weight of lead	=	18 lbs ↓
Total weight acting on the water	=	228 lbs ↓

To hover, the volume of water displaced by the diver must exert a buoyant force upward equal to the total weight of the diver plus gear (downward force). This is the buoyant force exerted by a volume of fresh water (density = 62.4 lbs/ft^3) that weighs 228 lb.

$$\text{Volume} = \text{Mass} \div \text{Density}$$

Substituting:

$$\text{Volume} = \frac{228\ lbs}{62.4\ lbs/ft^3} = 3.65\ ft^3$$

With the volume of the diver known, determine (with the assumption the volume of the weight belt is not significant) the buoyant force from the seawater (density = 64 lb/ft^3) the diver would displace:

$$3.65\ ft^3 \times \frac{64\ lbs}{ft^3} = 233.6\ lbs$$

Apply force arrows:

Buoyant force of seawater	=	234 lbs ↑
Weight of diver and gear	=	210 lbs ↓
Net force	=	24 lbs ↑

The diver that was comfortable with 18 lb of lead on the weight belt in fresh water must add 6 more pounds (for a total of 24 lb) on the weight belt to dive in seawater.

Example

A fully geared diver in a wet suit weighs 210 lb. In seawater, this diver needs 18 lb of lead to hover. How much lead will this diver need when diving in a wet suit in fresh water?

Weight of diver	=	210 lbs ↓
Weight of lead	=	18 lbs ↓
Total weight acting on the water	=	228 lbs ↓

To hover, the volume of water displaced by the diver must exert an upward buoyant force equal to the total weight of the diver plus gear (downward force). This is the upward buoyant force exerted by the displaced volume of seawater (density = 64 lb/ft^3) that weighs 228 lb.

Determine volume of diver:

$$\text{Volume} = \frac{228 \text{ lbs}}{64 \text{ lbs/ft}^3} = 3.56 \text{ ft}^3$$

Now that we know the volume of the diver, we can determine the upward buoyant force from fresh water (density 62.4 lb/ft^3) the diver would displace:

$$3.56 \text{ ft}^3 \times \frac{62.4 \text{ lbs}}{\text{ft}^3} = 222.1 \text{ lbs}$$

Apply force arrows:

Buoyant force of sea water	=	222 lbs ↑
Weight of diver and gear	=	210 lbs ↓
Net force	=	12 lbs ↑

The diver that was comfortable with 18 lb of lead on the weight belt in seawater must remove 6 lb (for a total of 12 lb) from the weight belt to dive in fresh water. The difference in density between fresh and seawater is the reason why different amounts of weight must be used when diving in different environments. When moving from fresh to seawater (with the same equipment configuration), divers must add weight. When moving from seawater to less dense fresh water, divers should remove weight.

Divers wearing wet or dry suits have an additional factor to consider. Within the wet suit are trapped bubbles of gas; a dry suit diver has air spaces between the diver and the suit. This gas is subject to changes in volume as a result of changes in pressure (see Boyle's Law). This means that as the diver moves up or down in the water column, the volume of these gas spaces changes. This change in gas volume affects the diver's buoyancy. As a diver descends, the volume decreases as ambient pressure increases, less water is displaced, and the diver becomes less buoyant and sinks. On ascent, the gas expands and occupies a larger volume, more water is displaced, and the buoyant (upward) force increases.

LIFTING

The lift associated with air spaces can be used to raise objects from the bottom. Because air weighs very little compared with the weight of the displaced water, it can be assumed that the lifting capacity is equal to the weight of the volume of water that is displaced by the air volume of the lifting device.

Example

Lift a 300 lb anchor from the bottom of a lake bed. The bottom is hard and flat (no excess lift is needed to overcome the suction associated with being immersed in the bottom muck). You have access to

55 gal drums (weighing 20 lb each) that have been fitted with overexpansion vents. How many 55 gal drums will it take to lift the anchor?

Determine forces involved:

Determine weight of water displaced:

$$\text{Weight} = \text{Density} \times \text{Volume}$$

Lake implies fresh water: Density = 62.4 lbs/ft^3

$$\text{Weight} = 55 \text{ gal} \times \frac{0.134 \text{ ft}^3}{\text{gal}} \times \frac{62.4 \text{ lbs}}{\text{ft}^3} = 459.9 \text{ lbs}$$

Weight of displaced water	=	460 lbs ↑
Weight of drum	=	20 lbs ↓
Net force	=	440 lbs ↑

Because the object to be lifted weighs less than the 440 lb lifting capacity of a 55 gal drum, a single 55 gal drum should be sufficient to lift the 300 lb anchor. In practice, large lifting objects (like a 55 gal drum) have a large surface area and generate considerable drag, which decreases lifting capacity. A rule of thumb is to assume that the lifting device has about 75% of the calculated lifting capacity in an actual lifting operation.

TRIM

As a diver moves in the water column, the diver is subject to a number of forces. In the vertical plane, gravity (weight) tends to make the diver descend and buoyancy (from too little weight or too much air in the buoyancy compensator) makes the diver ascend. In the horizontal plane, the diver moves forward, propelled by the force of the kick. The thrust, or forward motion, must overcome drag (or friction) that the diver and equipment present to the water. The overweighted diver must continually expend energy to overcome gravity and remain at constant depth, and the underweighted diver must continually expend energy in an attempt to overcome buoyancy with leg power. A more horizontal position presents a smaller area to the path of movement and thus lessens resistance.

ENERGY

Energy is the ability to do work. Energy that can be derived by a future change in position is called *potential energy;* energy that is due to moving mass is called *kinetic energy.* Consider a pile driver. This giant hammer device utilizes the kinetic energy of a large mass to drive construction supports into the earth. Energy is used to lift the "driver" to the top of the device. Here, while motionless, it possesses only potential energy. As the mass falls toward its target, the potential energy is transformed into kinetic energy. During the entire movement, the large falling mass has different portions of potential and kinetic energy, but the sum of these two types of energy remains constant. The six forms of energy are shown in Table 2–4.

Under ordinary conditions, energy can neither be created nor destroyed. This is known as the *Conservation of Energy* principle. Energy can be changed in *form,* however. For example, the potential energy of water at a high level is converted to kinetic energy as it falls to a lower level within a hydroelectric dam. The kinetic energy of the falling water turns a turbine (mechanical energy) that drives a generator, producing electricity (electric energy). The electricity lights a light bulb (radiant energy) and heats a small space heater (heat energy). During this entire process, energy was transformed from one form to another.

FORCE

Force is a push or a pull. Weight is the most commonly encountered force. It has a *magnitude* (how much push) and a *direction*

Table 2–4. The Six Forms of Energy

Mechanical	The sum of potential and kinetic energies derived from the movement of a body
Heat	Energy derived from molecular motion
Radiant	Energy in the form of electromagnetic waves such as light, x-rays, or radio waves
Chemical	Energy released from chemical reactions
Electrical	Energy derived from moving electrons
Nuclear	Energy derived from atomic forces within the atom

(the direction from which the push is coming). In 1687, Isaac Newton defined three principles that are known as *Newton's Laws of Motion:*

1. *A body will remain at rest or in a state of uniform motion along a straight line unless acted upon by some outside force.*
2. *Force acting on a mass produces an acceleration.* Mathematically:

$$F = ma$$

where F = force, m = mass, and a = acceleration.

If mass is in kilograms and acceleration is expressed as m/sec^2, then force is in newtons, i.e., 1 newton is the force that results from a mass of 1 kg being accelerated at a rate of 1 m/sec/sec. If mass is in grams and the acceleration is in cm/sec^2, then force is in dynes. If mass is in slugs and the force in ft/sec^2, then force is in pounds.

3. *For every action, there is an equal and opposite reaction.*

WORK

Work is the application of a force over a distance. Work requires energy. If no movement occurs, no work is accomplished. Pushing against a rigid wall that does not move produces no work. Work is expressed as length × force. Examples include foot-pounds (work done when a force of 1 lb moves an object 1 ft), newton-meters (work done when 1 newton of force moves an object 1 m), and ergs (work done by a force of 1 dyne moving an object 1 cm).

Consider two divers of the same size in the water; both are hovering ("weightless"). One has achieved this state by balancing the forces of weight and buoyancy. The other, overweighted, has compensated for this extra weight by inflating a buoyancy compensator. Even though "weightless," the overweighted diver does more work because more mass (the extra weight) has to be moved. In addition, overweighted divers generally are not horizontal in the water. This means they have a larger cross-sectional area, creating more drag. More drag means that more work is necessary for forward movement. Units of work are provided in Table 2–2.

POWER

Power is the measure of work over time. Mathematically:

$$\text{Power} = \frac{\text{Work done}}{\text{Time taken to do the work}}$$

HEAT

Heat is thermal energy: the sum of the kinetic energies for all the random movements of all molecules contained within a substance. It is convenient to measure the amount of heat as if heat were independent of the particular substance whose molecular motion determines the magnitude of heat energy present. The amount of heat necessary to raise 1 g of pure water from 14.5° to 15.5°C is defined as 1 *calorie.* One thousand calories is a kilocalorie (kcal). The corresponding English measurement is the amount of heat necessary to raise a pound of pure water from 63.0° to 64.0°F. This unit is called the *British Thermal Unit (BTU).* One BTU is equivalent to 252 calories.

Matter may be thought of as a heat reservoir. Because of their molecular makeup, different substances are capable of holding different amounts of heat. The amount of heat required to raise 1 g of a substance 1°C is called the *specific heat* (thus, water has a specific heat of 1.0 cal/g C). The *heat capacity* of a particular substance is defined as the specific heat of the material multiplied by its mass. The higher the heat capacity, the more heat a substance can absorb and store. Substances like water or helium have high specific heats compared with air (Table 2–5), and thus divers in contact with water or helium lose more heat than they would in air. Heat capacities of gases are commonly listed at a specific temperature and pressure (usually 25°C at 1 atm pressure). Because adding heat to a system can affect either the

Table 2–5. Gas Heat Capacities at 25°C and 1 ata Pressure

Substance	Cp (cal/g°C)	Cv (cal/g°C)
Air	0.3439	0.2943
Argon	0.1252	0.0750
Helium	1.2420	0.7620
Nitrogen	0.2477	0.1765
Oxygen	0.2200	0.1554

volume or the pressure of a gas (depending on the nature of the container), it is customary to measure thermal properties of a gas at constant pressure (C_p) or constant volume (C_v). Values for thermal properties of materials can be found in a number of standard references.

Heat capacity is the amount of heat required to raise the temperature of the substance by 1°C.

$$\text{Heat Capacity} = \text{Mass of body} \times \text{Specific heat of body}$$

The amount of heat necessary to change the temperature of a body is:

$$\text{Heat Required} = \text{Mass} \times \text{Specific heat} \times \text{Change in temperature}$$

Example

While holding pressure constant, how much heat is necessary to raise the temperature of 100 g of air 10°C? —of 100 g of helium 10°C? —of 100 g water 10°C:

a. For air:

$$\text{Heat needed} = 100\ \text{g} \times \frac{0.3439\ \text{cal}}{\text{g°C}} \times 10°\text{C}$$

$$= 344\ \text{cal}$$

b. For water:

$$\text{Heat needed} = 100\ \text{g} \times \frac{1.000\ \text{cal}}{\text{g°C}} \times 10°\text{C}$$

$$= 1000\ \text{cal}$$

c. For He:

$$\text{Heat needed} = 100\ \text{g} \times \frac{1.2420\ \text{cal}}{\text{g°C}} \times 10°\text{C}$$

$$= 1242\ \text{cal}$$

The amount of heat required to raise the temperature of 100 g of helium is larger than for an equal mass of air. This might suggest that the respiratory heat loss from breathing helium, as compared with air, would be enormous. However, the specific heat equation is based on mass. Helium is much less dense than air, so an equal volume of helium has a significantly lower mass. This is why respiratory heat loss while one is breathing helium is much less than would be expected solely according to heat capacity. (Respiratory heat loss at depth from breathing heliox is estimated at about 30% of the heat loss from breathing air.) This relative difference is demonstrated by comparing the amount of heat needed to warm a given mass (e.g., 100 g) to the relative amount needed to warm a unit volume of 1 L 10°C. Gas density values 1.296 g/L (air) and 0.178 g/L (He) values[3]:

a. For dry air:

$$\frac{0.3439\ \text{cal}}{\text{g°C}} \times 10°\text{C} \times \frac{1.296\ \text{g}}{\text{L}} = 4.5\ \text{cal/L}$$

b. For He:

$$\frac{1.2420\ \text{cal}}{\text{g°C}} \times 10°\text{C} \times \frac{0.178\ \text{g}}{\text{L}} = 2.2\ \text{cal/L}$$

Thus, although the heat capacity of helium is much greater than that of air, the difference in densities explains the lower-than-expected respiratory heat loss that occurs during the breathing of mixes containing helium.

To understand respiratory heat transfer, one must consider the thermal conductivity of the breathing gas and environment in which the diver is operating. *Thermal conductivity* (Table 2–6) is the rate at which heat is transferred between objects of different temperatures. Thermal conductivity is expressed as the amount of heat that can be transmitted from a fixed area across a known distance in a fixed amount of time. The higher the thermal conductivity, the quicker a warm object cools. As a corollary, the lower the thermal conductivity, the better the material acts as a heat retainer or insulator. Thermal

Table 2–6. Thermal Conductivity (kcal/hr per cm°C)

Substance	Conductivity
Air	2.3
Foam neoprene	4.6
Wool	8.0
Helium	12.2
Seawater	52.0

conductivity of an object varies with pressure and temperature of the surroundings. Table 2–6 provides some common thermal conductivity values expressed as heat conducted (kcal/h) through a 1-cm-thick slab of 1 m^2 of the material evaluated, per degree of temperature gradient.[4, 5]

The increased heat loss due to the high specific heat and thermal conductivity of helium and water as compared with air is responsible for hypothermia problems associated with working in an aqueous or heliox environment. This heat loss occurs primarily via direct contact with the environment (immersion in water or a heliox atmosphere contained within a diving habitat).

A physically large diver has more heat than a smaller diver. Thus, in general, the smaller diver, regardless of sex, is at higher risk for hypothermia. Physical size, however, is not the only factor. Variables such as age, physiologic condition (particularly if affected by drugs or alcohol), physical fitness, amount of in-water exercise, thermal protection system employed, temperature of the water, and duration of immersion can also influence the severity of hypothermia (see Chapter 13).

Temperature is a measurement of the intensity of heat energy. When two materials possessing different heat energies (different temperatures) come together, heat always moves from the higher to the lower temperature and continues to be transferred until the two bodies have the same temperature. This means that any time a diver is in water cooler than body temperature, the diver loses heat.

Problem

Given that dry air has a density of 0.0012 g/mL and water has a density of 1.0 g/mL, estimate the approximate ratio in heat capacity between water and air.

Determine ratio of mass from density:

$$\text{Ratio} = \frac{\text{mass 1 mL water}}{\text{mass 1 mL air}} = \frac{1.000\ \text{g}}{0.0012\ \text{g}} = 833.3$$

Determine ratio of specific heats:

$$\text{Ratio} = \frac{\text{specific heat water}}{\text{specific heat air}} = \frac{1.00\ \text{cal/g°C}}{0.2943\ \text{cal/g°C}} = 3.39$$

Finally, estimate the ratio: Heat capacity = Mass × Specific heat

Approximate heat capacity ratio
= 833.3 × 3.39
= 2824

Water has about 2800 times the heat capacity of air. The precise ratio depends on temperature, pressure, amount of particulate matter present, and humidity of the air, as well as the temperature and purity of the water. The water need not be frigid for hypothermia to occur. As has been stated, any time water is cooler than body temperature, the diver loses heat. Repeated exposure, even in tropical water, can lead to hypothermia.

ADIABATIC EXPANSION

An *adiabatic system* is one in which no heat is added or removed.[6] For an ideal gas (see Real and Ideal Gases), the following relationship holds:

$$C_v \ln (T_2 \div T_1) = -R \ln (V_2 \div V_1)$$

where C_v = heat capacity at constant volume, ln = natural logarithm, T = absolute temperatures at condition 1 or 2, R = universal gas constant (see later), and V = volume at condition 1 or 2.

This equation can be used to calculate the temperature change following an adiabatic change in volume. The equation indicates that temperature will increase during compression and decrease on expansion of an ideal gas. When air is compressed during the filling of a compressed gas cylinder, the temperature rises and the cylinder becomes hot. This increase in temperature can be as much as 1500°F during rapid compressions. In the presence of hydrocarbon contaminants, this heat can serve as an energy source for fire or explosion in an oxygen-enriched atmosphere. When air is rapidly released from a scuba cylinder, either through the direct opening of the valve to release its contents or via the purge valve, the volume of the gas increases and temperature falls. (The adiabatic cooling associated with gas movement driven by high pressure through a tiny orifice

is called the Joule-Thompson effect.) When a hyperbaric chamber is compressed, temperature increases; when pressure is reduced, temperature falls. Most hyperbaric chambers used for clinical therapy require heating and cooling systems to maintain constant temperature during changes in pressure.

TEMPERATURE

Daniel Fahrenheit introduced the first reliable calibration of temperature in 1724. He picked the lowest temperature he could obtain with a mixture of ice, salt, and water and called that his zero point. He next picked the temperature of a healthy man's blood and arbitrarily gave it a value of 96. Using mercury as the expanding fluid that would mark his thermometer, he found that water would freeze at a temperature of 32 and boil at a temperature of 212 on his scale. His system, the Fahrenheit temperature scale, is still used in the United States. About 12 years later, Anders Celsius proposed a scale that would be based on 100 units between the freezing point and boiling point of water.

The two systems of measurement can be converted using the following expressions:

$$°F = (1.8 \times °C) + 32 \text{ or } °F = 9/5°C + 32$$

$$°C = (°F - 32) / 1.8°C = 5/9\ (°F - 32)$$

Two other temperature scales are important to divers. They are the *Rankine* (absolute Fahrenheit) and the *Kelvin* (absolute Celsius). The significance of these absolute temperature scales is discussed later (see Charles' Law). By international convention, the definition of absolute temperature is in degrees Kelvin; thus, no degree symbol is used for Kelvin temperatures.

$$°R \text{ (Rankine)} = °F + 460$$

$$K \text{ (Kelvin)} = °C + 273$$

Although these formulas can be used to convert one temperature scale to another, in diving this is rarely done. Divers accustomed to the Fahrenheit scale use °R (°F + 460), and divers familiar with the Celsius scale use K (°C + 273) for problems that require the use of absolute temperature.

LIGHT

Light is a form of energy. It provides the illumination that we use to visually perceive and characterize our surroundings. White light, as first discussed by Isaac Newton, is composed of a number of components, each perceived as a different color. If white light passes through a prism, then these colors, known as the *light spectrum,* can be seen. The colors from the prism always have the same order: red, orange, yellow, green, blue, indigo, and violet.

The perception of color depends on which components of the light have been reflected or absorbed by the object being observed. If an object reflects all the colors, it is observed to be white; if no colors are reflected, then the object observed will be black. Other colors result from combinations of reflection and absorption of the various components of light. The propagation of light is influenced by a number of factors, including absorption, diffusion, refraction, and reflection.

Absorption

Each of the colors in the light spectrum possesses a different energy and wavelength. Red is the least energetic color, whereas blue is the most energetic form of visible light.

As light moves through water, the water absorbs the components of light. Because red is the least energetic, it is absorbed first. Each of the colors, in turn, is absorbed as light passes through any appreciable distance in water. In shallow water, only the red colors disappear, and as depth increases, the environment takes on a bluish cast. Eventually everything visible becomes deep blue, then black. Application of artificial white either from a dive light or a photographic strobe light allows the diver to observe and record true color.

Diffusion

As light moves through water, it interacts at the molecular level with all substances in the water. The result is that light is scattered and moves in random directions. This process is called *diffusion.* Divers see less light at depth because the total amount of

light available at the surface has been scattered by diffusion.

Turbidity refers to the amount of particulate material in the water. If turbidity is high, then the abundance of suspended material increases the amount of both diffusion and absorption that occurs. The diver sees less light in turbid water.

Refraction

Light travels at different speeds in different substances. Light slows about 25% when it enters water from air. This change in velocity results in a bending of the light path as it changes from air to water. This bending affects light as if it had moved through an optical lens. The alteration in the path of light as a result of changing media is called *refraction.* The diver's mask is an air/water interface; thus, the mask also acts as a lens. One reason why a diver needs a mask is that our eyes have adapted to focus in air. Objects appear blurred underwater because the eyes cannot adjust enough to bring objects into focus in water. One function for the dive mask is to provide an air/eye interface so that the eyes can focus the light. The result of the air/water interface of the mask is that divers perceive objects to be larger (by four thirds) and closer (by one fourth) than they really are. An object 4 ft away from the diver appears as to be only 3 ft away (see Chapter 3).

Reflection

When light waves strike a smooth polished surface, they bounce off the surface much like a billiard ball bounces off the side cushions of a billiard table. The angle formed by the light leaving the polished surface is the same angle as the light striking the surface when measured from a line perpendicular to the surface. In the same fashion, a portion of the light striking water is reflected away from the surface. Near sundown, this effect can significantly reduce the amount of ambient light at depth.

SOUND

Sound is a longitudinal pressure wave that moves through a fluid. Mechanical vibrations caused by the pressure waves produce sound. The ear converts the vibrations to electrical signals that the brain interprets as sound. In air, we can perceive the direction of a sound source by sensing the time delay between the sound energy striking one ear and then the other. The brain processes this time delay to give a direction. Underwater, the velocity of sound is about four times faster than in air, and the time delay between sound energy striking each ear is too small to be perceived.[7] Localization of a sound underwater by humans is possible, particularly with low-frequency signals, but it is extremely difficult. Divers should consider sound an unreliable directional cue.

PRESSURE

Pressure is defined as a force that acts on a unit area. The force most often encountered by divers is weight. Thus, pressure is measured in terms of a weight per unit area. The pressure divers must cope with is a result of the weight of the water and atmosphere above the diver.

The Greek philosopher Empedocles first expressed the belief that air had weight in the fifth century BC. Even Aristotle said, "Nature abhors a vacuum." In 1645, Guericke used his newly developed air pump to remove the air from the space defined by two hollow steel hemispheres that had been placed together. Horses pulling on his hemispheres could not separate them. Yet, when the air was replaced in the sphere, the hemispheres could easily be separated. The implication was that some force (later demonstrated to be atmospheric pressure) in the air was capable of holding the spheres together. The first scientific explanation of the weight of air was by the Italian mathematician, Evangesta Torricelli (a student of Galileo), in 1643. His experiment was the basis of the modern barometer. Torricelli filled a tube closed on one end with mercury and, after inverting the tube, placed the tube in a dish of mercury. He noted that the mercury did not drain from the tube into the dish. Instead, it remained within the tube. His explanation was that air had weight. The weight of the air pushing down on the mercury in the dish was equal to the weight of the mercury in the tube. The height of the mercury (760 mm) in the tube was then defined as *atmospheric pressure.* Equivalent measurements of pressure can be made with

different fluids; mercury was originally chosen because of its high density (specific gravity of 13.6). An equivalent instrument using water (specific gravity of 1.00) would be over 30 ft high.

Problem

What is the approximate height of a seawater column that corresponds to 760 mm Hg?

Water is less dense; thus, the height will be greater. The heights of liquids in a vertical column are inversely proportional to specific gravity (the specific gravity of seawater is 1.0256, the specific gravity of mercury is 13.546).

$$\frac{760 \text{ mm Hg}}{x \text{ mm } H_2O} = \frac{1.0256}{13.546}$$

$x = 10{,}037.99$ mm H_2O
10,037.99 mm = 10.04 m = 32.9 ft

Thus, 760 mm Hg (1 atm) corresponds to 33 ft, or 10 m, of seawater (feet of seawater = fsw; meters of seawater = msw). Units of pressure and conversion factors can be found in Appendix 1.

Pressure due to the water surroundings is called *hydrostatic* or *gauge pressure*. This is equal to 1 atm of pressure for every 33 ft (10 m) of depth in seawater (34 ft, or 10.3 m, in fresh water). Open bodies of water are also subjected to the weight of the atmosphere, so the total (absolute) pressure at depth is the sum of the hydrostatic and atmospheric pressures.

Example

Determine hydrostatic and absolute pressure at a depth of 78 fsw (23.8 msw) using the definition of hydrostatic pressure:

$$\text{Hydrostatic pressure} = \frac{\text{Depth of water}}{\text{Definition of atm}}$$

Note: Water depth is in units of length; atm should be expressed in the same units. If depth is in fsw, then 1 atm = 33 fsw; if depth is in ffw, then 1 atm = 34 ffw; if depth is in msw, then 1 atm = 10.1 msw, or 10 bar.

Substitute:

		English	Metric	
Hydrostastic pressure	=	$\frac{78 \text{ fsw}}{33 \text{ fsw/atm}}$	$\frac{23.8 \text{ msw}}{10.1 \text{ m/atm}}$	$\frac{23.8 \text{ msw}}{10 \text{ m/bar}}$
Hydrostatic pressure	=	2.36 atm	2.36 atm	2.38 bar
Absolute pressure	=	Hydrostatic + Atmospheric		
	=	2.36 atm + 1 atm		
	=	3.36 ata (ata = Atmospheres absolute)		
Absolute pressure	=	2.38 bar + 1.01 bar = 3.39 bar		

Gas pressure in cylinders is measured in gauge pressure, which reads zero at 1 atm. To determine absolute pressure, 1 atm (in the same units as the gauge) must be added to the gauge pressure.

Example

An 80 ft^3 cylinder contains gas at a pressure of 3000 psig (pounds per square inch gauge).

Determine absolute pressure using absolute pressure = gauge pressure + atmospheric pressure:

3000 psi + 14.7 psi = 3014.7 psia
($lbs/inch^2$ absolute)

Example

A scuba cylinder contains 2400 L at a gauge pressure of 200 bar.

Determine absolute pressure, which corresponds to an absolute pressure of:

200 bar + 1.01 bar = 201.01 bar

DEPTH GAUGES AND ALTITUDE DIVING

Depth gauges measure pressure, they do not measure water depth. A printed scale on the face of the instrument converts the measured pressure into an equivalent scale reading for water depth. The gauge will be accurate only if it is used in the environment for which it has been calibrated. When the device is taken to a different environment,

such as high altitude, the reading of water depth on the gauge may be substantially different from the actual measured water depth.[8] This is most often a problem when depth gauges calibrated at sea level are taken to altitude, as in the following example.[9]

Example

At a mountain lake, the barometer reads 24.61 inches (625 mm) Hg. Thus, at this altitude, 24.61 inches (625 mm) Hg is the atmospheric pressure. Consider also that high mountain lakes usually are filled with fresh water (density ≈ 62.4 lbs/ft^3; 1.00 g/cc), not salt water (density 64 lb/ft^3; 1.03 g/cc). What will a depth gauge designed for use in seawater read at an actual depth of 60 ffw (18.29 m) in this lake?

The use of actual pressure units makes this problem easier to understand:

Determine the pressure equivalent of 1 atm at this altitude:

$$\frac{24.61 \text{ in Hg}}{29.27 \text{ in Hg}} \times 14.7 \text{ psi} = 12.36 \text{ psi}$$

At 60 ffw, the hydrostatic pressure is:

$$\frac{60 \text{ ft}}{34 \text{ ft/atm}} \times 14.7 \text{ psi} = 25.94 \text{ psi}$$

This is an absolute pressure of:

$$12.36 \text{ psi} + 25.94 \text{ psi} = 38.3 \text{ psia}$$

This corresponds to a sea level pressure of:

$$\frac{38.3}{14.7 \text{ psi at sea level}} = 2.6 \text{ sea level ata}$$

This would then correspond to a hydrostatic sea level pressure of:

$$2.6 \text{ ata} - 1 \text{ atm} = 1.6 \text{ atm}$$

Which would be read on the sea level calibrated scale as:

$$1.6 \text{ atm} \times \frac{33 \text{ ft}}{\text{atm}} = 53 \text{ feet}$$

So, the measured depth was 60 ft; the sea level depth gauge at this altitude would read 53 ft.

OCEAN EQUIVALENT DEPTH (FOR DECOMPRESSION OBLIGATION)

Decompression tables are based on pressure ratios. Safe decompression usually depends on not exceeding certain pressure ratios that can be tolerated within the tissue compartments without injury to the diver (see Chapter 7). Thus, altitude decompression adjustments must be based on calculated actual pressures that account for the barometric pressure at altitude and the density difference between fresh and saltwater. Decompression schedules must account for the lower atmospheric pressure at the surface when determining safe surfacing ratios.

Equivalent Ascent Rates

Ascent rates are part of the decompression calculations. U.S. Navy sea level tables assume a rate of 60 fsw/min.[10] This ascent rate is part of the calculations used to derive the decompression schedules. Because, at altitude, the actual amount of water column that "defines" 1 atmosphere is less than 33 fsw (10.1 msw), an ascent in a high-altitude mountain lake must be slower than an ascent from the corresponding depth at sea level to maintain the same rate of pressure change with time. For this example:

At sea level, the recommended ascent rate is:

$$\frac{60 \text{ fsw}}{\text{min}} \times \frac{1 \text{ atm}}{33 \text{ fsw}} = \frac{1.82 \text{ atm}}{\text{min}}$$

At this altitude, corresponding at-altitude ascent rate:

$$\frac{1.82 \text{ atm}}{\text{min}} \times \frac{27.9 \text{ ffw}}{\text{atm}} = \frac{50.8 \text{ ffw}}{\text{min}}$$

Sea level–based dive procedures (tables or computers) are inadequate for determining decompression obligations at high-altitude dive sites. Divers at high altitudes (above 1000 ft, or 300 m) should consider high-altitude conversion tables or altitude-compensating dive computers.

KINETIC THEORY OF GASES

All gases, regardless of chemical composition, behave similarly in response to physical changes of composition, temperature, and pressure. It is one of the dogmas of science that the behavior of a material is a reflection of the particles that make up the substance. The differences among solids, liquids, and gases reflect the movement of the small particles (atoms) that compose all matter. This assumption is part of the Kinetic Theory of Gases. This theory is based on six fundamental assumptions:

1. Gases are composed of molecules in constant motion.
2. Gases mix to uniformity and fill all portions of the containment vessel.
3. Molecules of a gas collide frequently with each other and with the walls of the containment vessel.
4. Under ordinary circumstances, the distance between gas molecules is far greater than the size of the individual molecules. This is why gases can be compressed.
5. The molecules of a gas move in all directions with an average velocity at a given temperature. At a given temperature, the average energy of molecules in the gaseous state is the same for all substances.
6. Molecules are perfectly elastic; thus, they lose no energy when they collide with another molecule.

These assumptions are the basis for understanding gas behavior. For example, the measurement of the intensity of the collisions of the gas molecules with the walls of the containment vessel (force per unit area) is called pressure. As the kinetic energy is increased by raising temperature, the molecules gain more velocity and collide with more force more often on the vessel walls; pressure in a closed container increases as temperature is raised.

As gas molecules move about in containment, they strike the walls of the container. In Figure 2–2, the gas molecules are randomly moving inside a cylinder. At the right end of the cylinder is a piston, held in place by some spring mechanism within the box. As the gas molecules strike the flat plate of the piston, the combined force of all the impacts moves the piston backward until the force of the gas molecules striking the flat plate of the piston

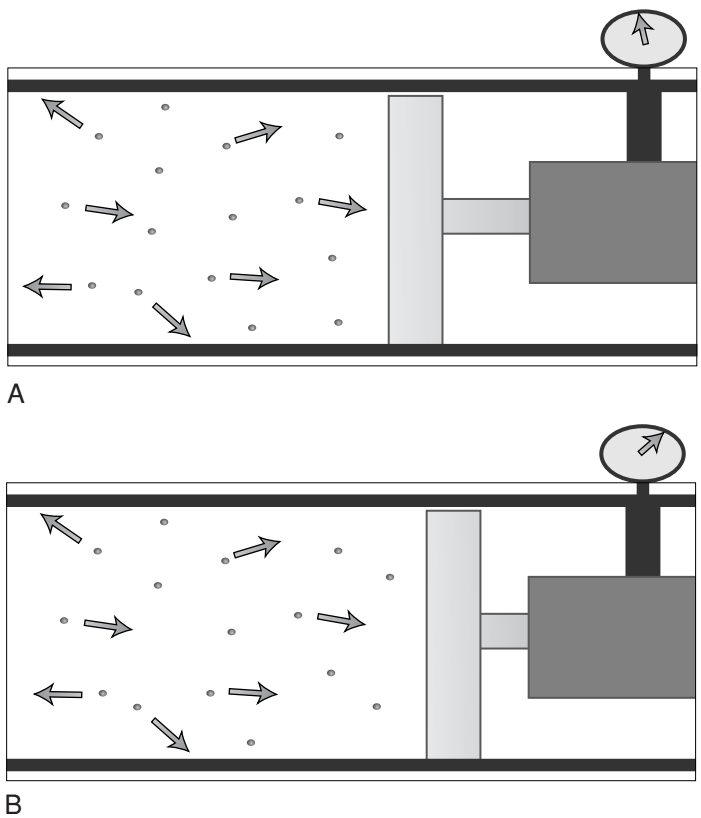

Figure 2–2. *A,* Pressure results from impact of gas molecules on the flat piston. *B,* Increased energy of gas molecules causes more impacts at higher energy against the flat piston, causing the pressure to increase.

balances a spring device contained within the brown box. A measurement of this impact is displayed on a mechanical gauge (Fig. 2–2*A*). If the temperature increases, the average velocity of the gas molecules increases; they will strike the flat plate with more force, and the plate will move within the cylinder to indicate a higher pressure (Fig. 2–2*B*).

Gas Law Fundamentals

Historically, the behavior of gases was evaluated by measuring the temperature, pressure, and volume of the gas under study. Because of the complexity of attempting to simultaneously measure and predict all values, one of the values typically was held constant and one of the other values was changed to determine the effect on the third parameter. These relationships have been named for the scientists who established the validity of the particular relationship that is now called a gas law.

CHARLES' LAW

In 1787, the French scientist Jacques Charles studied the relationship between temperature and volume at constant pressure. He noted that in the vicinity of 0°C, the volume of a gas decreased by a factor of 1/273 for each degree Celsius decrease.[11] If one theoretically continued this decrease in temperature, then a gas would have zero volume at –273°C. This value is called *absolute zero*. If a gas has zero volume, then there will be no molecular motion (velocity = zero in the kinetic energy equation) and thus no kinetic energy. Measurements of temperature based on this absolute zero point are called *absolute temperature* (zero volume is not obtainable because gases will liquefy before absolute zero is reached). Because this 1/273 change in volume corresponds to a 1-degree change on the absolute temperature scale, absolute temperatures are used when the gas laws are used to predict variations in pressure, temperature, and volume. Charles' observations have been formalized into *Charles' law:*

At constant pressure, the volume of a gas is directly proportional to the absolute temperature:

$$\frac{V_1}{T_1} = \frac{V_2}{T_2}$$

A practical example of Charles' law involves the effect on the volume (size) of any flexible container with change in temperature. The volume of gas in a buoyancy compensator declines when passing through thermoclines into colder water. The loss of buoyancy from increased pressure when descending through a water column with cold thermoclines is exacerbated by this temperature effect on volume. Thus, as the gas chills after entering a thermocline, buoyancy continues to decrease until the temperature of the gas in the buoyancy compensator is the same temperature as the ambient water. On ascent out of the thermocline, the warmer water causes an increase in buoyancy from the expansion of the gas related to this temperature effect.

The magnitude or behavior of gases is best illustrated by looking at some numeric examples. It is important to remember that in all gas law problems, relationships are only valid when absolute values are used.

Example

If a scuba cylinder is capable of delivering 40 ft^3 of air to a diver at 78°F, how much air is available at 55°F?

Using Charles' law:
Determine absolute temperature:

$$T_1 = 78°F + 460 = 538°R$$

$$T_2 = 55°F + 460 = 515°R$$

Charles' law:

$$V_1 / T_1 = V_2 / T_2$$

Substituting:

$$40\ ft^3 / 538°R = V_2 / 515°R$$

Solving:

$$V_2 = 38.3\ ft^3$$

Comment: The temperature 55°F is typically the temperature of the first thermocline of a fresh water lake. Charles' law explains why divers have less air available to them in colder water.

GAY-LUSSAC'S LAW

The relationship between pressure and temperature has been associated with the French scientist, Joseph Gay-Lussac. Because Gay-Lussac collaborated with Jacques Charles, some have associated this principle with Charles. However, Charles, the mentor, gave credit for this relationship to his student, Gay-Lussac, because Gay-Lussac was the first to build an apparatus to demonstrate the validity of the linear relationship between pressure and temperature. He performed his measurements using a fixed-volume, gas-filled sphere. He measured the temperature and pressure of the gas in the sphere while ascending in a hot air balloon.[12] His published observation (known as Gay-Lussac's law) states:

At constant volume, the pressure of a gas is directly proportional to the absolute temperature:

$$\frac{P_1}{T_1} = \frac{P_2}{T_2}$$

Example

A cylinder at 25°C (298 K) contains gas at a gauge pressure of 200 bar (201.01 bar absolute). Determine the pressure at 42°C (315 K).

Using Gay-Lussac's law:

$$P_1 / T_1 = P_2 / T_2$$

Substituting:

$$201 \text{ bar} / 298 \text{ K} = P_2 / 315 \text{ K}$$

Solving:

$$P_2 = 212.5 \text{ bar}$$

Converting to gauge pressure:

$$P_2 = 212.5 \text{ bar} - 1.01 \text{ bar}$$

$$P_2 = 211.5 \text{ bar}$$

Thus, a scuba cylinder with a gauge pressure of 200 bar at 25°C heated to 42°C will show a gauge pressure of about 212 bar.

BOYLE'S LAW

In 1662, Sir Robert Boyle published the classic *The Spring of Air and Its Effects,*[6] in which he measured the relationship between pressure and volume at constant temperature.[13] He measured the volume of air trapped at the small end of a J-shaped tube. The tube was filled with mercury, and the volume of the air space was measured. Adding mercury (increasing the height of mercury in the J-tube) decreased the volume of air trapped at the small end of the J-shaped tube. He noted that the product of the pressure (as determined by the height of the mercury column) and the volume was constant. Expressed mathematically:

$$PV = k$$

where P = the pressure (height of mercury in tube), V = volume (of air space in tube), k = a constant.

This relationship, PV = k, held for a variety of P, V combinations. In mathematics, products equal to the same value can be set equal to each other.

Boyle's law states:

At constant temperature, the volume is inversely proportional to the absolute pressure:

$$P_1 V_1 = P_2 V_2$$

A corollary to this law states that density (mass/volume) increases directly with the pressure.

Example

What is the physical volume (in cubic feet) of an aluminum "80" scuba cylinder?

An aluminum 80 cylinder delivers 80 ft^3 of air at 1 atm (14.7 psia) when filled to a pressure of 3000 psig (3014.7 psia). Thus, the physical volume of the tank is the volume at 3000 psig (3014.7 psia).

Substituting into Boyle's law:

$$P_1 V_1 = P_2 V_2$$

$$(14.7 \text{ psia})\ (80 \text{ ft}^3) = (3014.7 \text{ psia})\ V_2$$

Solving for V_2:

$$V_2 = 0.39 \text{ ft}^3$$

This physical volume represents how much water the cylinder would hold if the valve were removed and the cylinder filled with water. This is the value known as the *water capacity* of a gas cylinder.

Example

A scuba cylinder is rated at 2400 L with a pressure of 200 bar. What is the physical volume (water capacity) of the cylinder? The cylinder delivers 2400 L if all the air is released at 1 bar. The physical volume (water capacity) of the cylinder is the volume of gas compressed to 200 bar. The volume can be found by using Boyle's law:

$$P_1 V_1 = P_2 V_2$$

Determine absolute pressure: gauge + atmospheric:

$$200 \text{ bar} + 1 \text{ bar} = 201 \text{ bar}$$

Substituting into Boyle's law:

$$P_1 V_1 = P_2 V_2$$

$$(1 \text{ bar}) \times (2400 \text{ L}) = (201 \text{ bar}) \times V_2$$

Solving:

$$V_2 = 11.9 \text{ L}$$

Example

A scuba cylinder has a rated capacity of 80 ft^3 on the surface. Determine the volume of air from this cylinder available to the diver at 33, 66, 99, and 132 fsw.

Answer: Boyle's law allows calculation of decreasing volume of air with increasing depth.

Determine absolute pressure (Hydrostatic pressure + Atmospheric pressure):

For 33 ft

$$33 \text{ fsw} / 33 \text{ fsw} / \text{atm} = 1 \text{ atm}$$

$$1 \text{ atm} + 1 \text{ atm} = 2 \text{ ata (ata = Absolute pressure in units of atmospheres)}$$

For 66 ft:

$$66 \text{ fsw} / 33 \text{ fsw} / \text{atm} = 2 \text{ atm}$$

$$2 \text{ atm} + 1 \text{ atm} = 3 \text{ ata}$$

For 99 ft:

$$99 \text{ fsw} / 33 \text{ fsw} / \text{atm} = 3 \text{ atm}$$

$$3 \text{ atm} + 1 \text{ atm} = 4 \text{ ata}$$

For 132 ft:

$$132 \text{ fsw} / 33 \text{ fsw} / \text{atm} = 4 \text{ atm}$$

$$4 \text{ atm} + 1 \text{ atm} = 5 \text{ ata}$$

Substituting into Boyle's Law:

$$P_1 V_1 = P_2 V_2$$

For 33 fsw:

$$(1 \text{ ata}) (80 \text{ ft}^3) = (2 \text{ ata}) V_2$$

$$V_2 = 40 \text{ ft}^3$$

For 66 fsw:

$$(1 \text{ ata}) (80 \text{ ft}^3) = (3 \text{ ata}) V_2$$

$$V_2 = 26.7 \text{ ft}^3$$

For 99 fsw:

$$(1 \text{ ata}) (80 \text{ ft}^3) = (4 \text{ ata}) V_2$$

$$V_2 = 20 \text{ ft}^3$$

For 132 fsw:

$$(1 \text{ ata}) (80 \text{ ft}^3) = (5 \text{ ata}) V_2$$

$$V_2 = 16 \text{ ft}^3$$

The answers are summarized in Table 2–7.

The volume shown is the volume calculated for an 80 ft^3 cylinder. The fraction represents the proportional amount of the surface volume at that absolute pressure available from any size gas cylinder. The percentage change represents the difference in volume between each successive 1 ata pressure change.

As pressure increases, volume decreases. Because breathing is a constant-volume

Table 2–7. Change in Volume with Pressure

Depth (fsw)	Absolute Pressure (ata)	Volume (ft^3)	Fraction	% Change
0	1	80	1	0
33	2	40	1/2	50
66	3	27	1/3	33
99	4	20	1/4	25
132	5	16	1/5	20

process, the deeper the dive, the less breathing gas is available.

Likewise, as pressure decreases, the volume of gas in all flexible containers (lungs and other air spaces) increases. Because the physical size of the body cavity (e.g., lungs, ears, sinus) containing the air is limited, the expanding gas volume either properly vents through unobstructed passages or increases until tissues are injured. Figure 2–3 shows that the greatest volume change per unit of pressure is in the vicinity of the surface. This means that the greatest risk of injury due to barotrauma occurs near the surface.

GENERAL GAS LAW

Any two of the three gas laws of Boyle, Charles, or Gay-Lussac can be combined into a relationship called the *General* or *Combined Gas law:*

$$\frac{P_1V_1}{T_1} = \frac{P_2V_2}{T_2}$$

This relationship can be used to predict pressure, volume, and temperature relationships when any five of the six variables are known.

Example

The gas in a scuba cylinder occupies a volume of 72 ft^3 at 78°F on the surface. What volume of gas is available to the diver at a depth of 126 ffw and a temperature of 40°F?

Determine pressure:
On the surface:

$$P_1 = 1 \text{ ata}$$

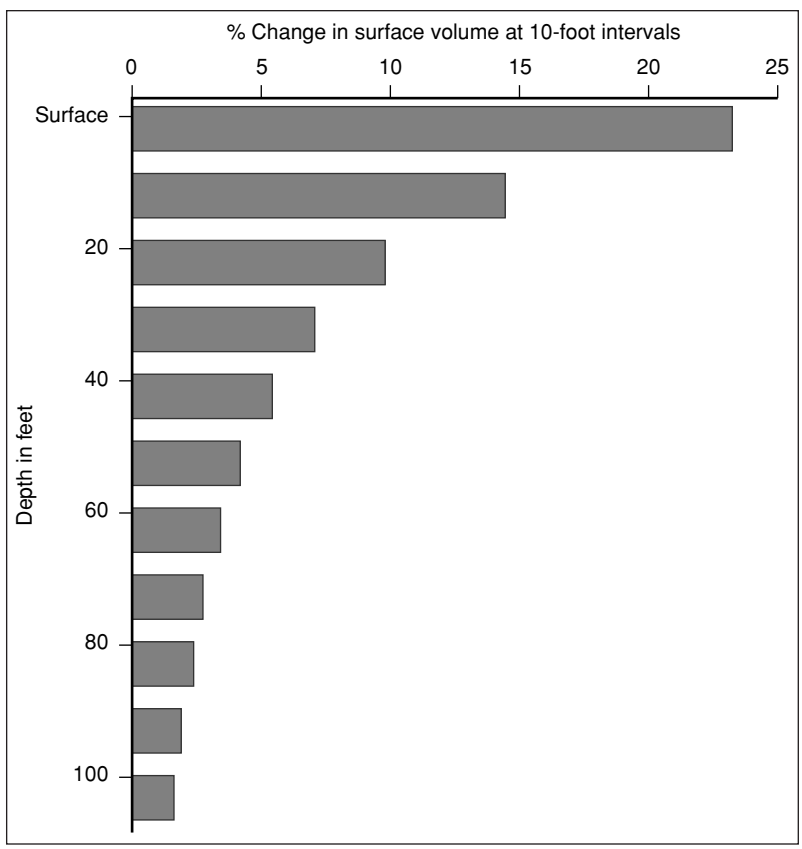

Figure 2–3. Volume changes as a function of depth, determined from Boyle's law. Data are shown as percentages of initial volume in 10-ft increments. The greatest percentage change occurs near the surface.

At 126 ffw:

$$P_2 = 126 \text{ ffw} / 34 \text{ ffw} = 3.7 \text{ atm}$$

(In fresh water; 34 ffw = 1 atm)

$$3.7 \text{ atm} + 1 \text{ atm} = 4.7 \text{ ata}$$

$$P_2 = 4.7 \text{ ata}$$

Determine absolute temperature:

$$T_1 = 78°F + 460 = 538°R$$

$$T_2 = 40°F + 460 = 500°R$$

General Gas law:

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

Substituting:

$$(1 \text{ ata})(72 \text{ ft}^3) / 538°R = (4.7 \text{ ata}) V_2 / 500°R$$

Solving:

$$V_2 = 14.2 \text{ ft}^3$$

Because it is most likely that both temperature and pressure will vary between the filling of a compressed gas cylinder and its use, the General Gas law will give a slightly more realistic evaluation of volume available at depth than a relationship that only examines two of the three pressure-temperature-volume variables.

DALTON'S LAW

In 1810, the English chemist John Dalton, along with collaborator William Henry (of Henry's Law), observed the pressures obtained when gases were mixed in the same container. He concluded that when gases were mixed in a container, each gas behaved as if it were the only gas present.[11] Thus, the total pressure in a closed system can be obtained by summing the pressures of each component. The pressure of each component is called the *partial pressure.* Expressed mathematically:

$$p_{(total)} = p_1 + p_2 + p_3 + \cdots p_n$$

where n = number of components in the gas mixture.

The total pressure of a gas mixture is the sum of the partial pressures of all the components.

Because the distances between gas molecules are so vast, each gas molecule behaves as if it were alone. So, even though components being mixed have pressures of their own, when they are combined in a container at near-atmospheric pressures the total pressure is simply the sum of the individual components. Under high pressure, the volume of gas molecules (compared with the total volume of space available) becomes significant, and the simple summing of pressures no longer applies.

Example

A 1 ft^3 (28.3 L) container contains 500 psig (34 bar) of nitrogen gas. Into the container, an additional 346 psig (23.8 bar) of oxygen gas is introduced. Determine the final pressure.

Using Dalton's law:

$$P_{(total)} = p_1 + p_2$$

Substituting:

$$P_{(total)} = 34 \text{ bar} + 23.8 \text{ bar}$$

Solving:

$$P_{(total)} = 57.8 \text{ bar}$$

Another way of viewing Dalton's law:

$$p_n = P_{(total)} \times \text{fraction gas}_{(n)} \text{ by volume}$$

Example

Determine the partial pressure of oxygen in compressed air at a depth of 88 fsw (26.8 msw).

Using Dalton's law:

$$p_n = P_{(total)} \times \text{fraction gas}_{(n)} \text{ by volume}$$

Remembering that air = 21% O_2:
Determine absolute pressure:

$$P \text{ (hydrostatic)} = 88 \text{ fsw} / 33 \text{ fsw} = 2.7 \text{ atm}$$

$$P \text{ (absolute)} = 2.7 \text{ atm} + 1 \text{ atm} = 3.7 \text{ ata}$$

Substituting:

$$p\ O_2 = 3.7\ ata \times 0.21$$

$$p\ O_2 = 0.77\ ata$$

Example

Determine the partial pressure of nitrogen (78% of air) at the same depth:

$$p\ N_2 = 3.7\ ata \times 0.78$$

$$p\ N_2 = 2.89\ ata$$

HENRY'S LAW

Whenever a gas is in contact with a liquid, gas dissolves in the liquid. Gas molecules simultaneously move out of solution into the gas phase and move from the gas phase into solution within the liquid phase. Although it is impossible to predict the behavior of an individual gas molecule, the net movement of gas equilibrates such that the partial pressure of the gas going into solution is the same as the partial pressure of the gas coming out of solution. When the gas reaches the state at which the amount of gas going into solution is the same as the amount of gas molecules coming out of solution, the solution is said to be saturated with gas. This state is called *equilibrium*. At this point, although individual gas molecules move at random into and out of solution, there is no net change in gas concentration within the solution.

Henry's law states:

The amount of gas that will dissolve into a solution is directly proportional to the partial pressure of that gas and inversely proportional to the absolute temperature.

The greater the partial pressure of the gas, the greater the driving force for solution and the greater the amount of gas that will dissolve into solution. As the temperature decreases, more gas will dissolve into solution. It is important to realize that Henry's law is concerned with the *amount* of gas in solution when equilibrium is reached. It specifically does not address *how rapidly* that state is reached.

Henry's law approximates the dissolution of nitrogen within body tissues. The deeper one dives, the greater the partial pressure of nitrogen (and any gas in the gas mix) and the greater the gas load each tissue must bear. Upon ascent, the partial pressure in the gas phase decreases. The gas in solution then escapes from solution in an attempt to obtain equilibrium. If this escape from tissue is too rapid for the body to handle, decompression sickness is the result (see Chapters 7 and 8).

UNIVERSAL GAS LAW

Boyle measured the product of pressure and volume and always found the same number:

$$PV = k$$

Scientists wanted a single equation that would—without requiring the measurement of multiple volumes, temperatures, and pressures—give reliable pressure, temperature, volume, and quantity measurements on gases. This led to an investigation of the constant k. The resulting generalized gas equation takes the form:

$$PV = nRT$$

where P = absolute pressure, V = volume, n = number of moles, R = universal gas constant, and T = absolute temperature.

One mole contains 6.024×10^{23} *(Avogadro's number)* molecules. One mole of a gas at standard temperature and pressure (STP: 0°C, 1 atm absolute) has a volume of 22.414 L. Thus, this equation allows one to derive not only pressure, temperature, and volume relationships but quantities of a substance as well.

R is the universal gas constant equal to the value of PV/nT.

Example

How many liters would 5 moles of any gas occupy at 25°C (298 K) and 2 atm absolute pressure?

Using:

$$PV = nRT;\ R = 0.082\ L\text{–}ata\ /\ K\text{–}mole$$

Substituting:

$$(2\ ata)\ V = (5\ moles)\ (0.082\ L\text{–}ata\ )\ (298\ K)\ /\ K\text{–}mole$$

Solving:

$$V = 61.1 \text{ L}$$

Thus 5 moles of gas would correspond to:

$$5 \text{ moles} \times 32 \text{ g / mole} = 160 \text{ g } O_2$$
(oxygen, MW = 32)

$$5 \text{ moles} \times 28 \text{ g / mole} = 140 \text{ g } N_2$$
(nitrogen, MW = 28)

$$5 \text{ moles} \times 4 \text{ g / mole} = 20 \text{ g He}$$
(helium, MW = 4)

Note: 20 g of helium occupies the same volume as 160 g of O_2. This is because 20 g of helium (5 moles) contains the same number of molecules as 160 g (5 moles) of oxygen.

Example

Determine the volume of one mole of gas at 5.1 ata and 3°C (276 K).

Using the Ideal Gas Law:

$$PV = nRT$$

Rearranging:

$$V = nRT / P$$

Substituting:

$$V = \frac{(1 \text{ mole}) (0.082 \text{ L-ata / K-mole}) 279 \text{ K}}{5.1 \text{ ata}}$$

$$V = 4.441$$

REAL AND IDEAL GASES

The equation PV = nRT is called the *Ideal Gas Law.* It is used to predict the behavior of so-called ideal gases. An ideal gas is a gas that exactly behaves according to the laws of Charles and Boyle. In other words, for an ideal gas, the product of PV is always constant. In reality, no gas is ideal. Most gases, at conditions near STP behave according to Boyle's and Charles' Law. As temperature and pressure move away from STP, values calculated by the ideal gas laws, including Boyle's and Charles', are different from the values measured experimentally. The ideal gas situation is best suited to high temperatures and low pressure (when the distances between individual gas molecules are greatest, so molecular volume is insignificant compared with total container volume). At the pressures in a compressed gas cylinder, gases no longer are ideal; thus, ideal equations no longer accurately predict volume and pressure measurements. This deviation from ideal behavior has been explained by the fact that molecules do occupy space. Because moving molecules cannot move unhindered in all directions, the volume appears larger than predicted by ideal behavior. Also, slight forces of attraction (van der Waals forces) exist between molecules so that individual molecules do not truly act totally independently of each other. This makes the volume appear smaller than predicted for ideal behavior.

The proximity of molecules to each other depends on both temperature and pressure. Low pressures and high temperatures keep molecules apart and allow gas behavior to be close to that predicted by the ideal equations. However, low temperatures and high pressures tend to decrease molecular distance and lead to a significant difference from ideal behavior. Such behavior is called *real behavior*, and equations that predict gas behavior in regions in which the simple ideal laws are inadequate are called *real equations.*

Under ordinary conditions, the deviation between real and ideal gas behavior is of little concern to divers. However, at compressed gas cylinder pressures, the difference between real and ideal calculations can be substantial. This difference is particularly important when one is calculating components for a breathing mixture other than air.

van der Waals Equation

Because measurement of the pressure and volume of a number of gases at different conditions clearly demonstrated that the simple ideal gas law was inadequate to predict observed behavior, it became necessary to modify the ideal gas equation. Near the end of the nineteenth century, the Dutch chemist Johanns van der Waals examined

the ideal gas equation and made the following assumptions:

- At low pressures, the intermolecular attractive forces act to cause a decrease in pressure. This causes the product PV in the ideal gas equation to be lower than expected.
- At high (compressed gas cylinder) pressures, the volume occupied by individual molecules is significant with respect to the total volume occupied by the gas. At high pressures, the density of the gas is greater. Thus, there will be more molecules per unit volume and the percentage volume occupied by gas molecules will increase. Because the term V in the ideal gas equation should represent only free space available for gas movement, a correction would be needed to account for the volume of space occupied by gas molecules. Because this correction factor is not present in the ideal gas law, values calculated for PV at high pressures are larger than measured.

In order to make the ideal gas law more closely conform to observed parameters, van der Waals introduced the following modifications:

1. The ideal pressure could be represented as:

$$P\ (\text{ideal}) = P + (an^2 / V^2)$$

where P = pressure measured, V = the volume, a = a constant characteristic of each gas, and n = number of moles present.

The constant a represents the attraction between molecules; it is different for each gas and has been determined from empirical observations.

2. The ideal volume could be represented as:

$$V\ (\text{ideal}) = V - nb$$

where V = volume measured, b = a constant characteristic of each gas, and n = number of moles.

The constant b represents the excluded volume of the molecules that make up the gas; it is different for each gas and has been determined from numerous measurements. Tables of a and b values for various gases are available. One of the most utilized sources of such data is the CRC *Handbook of Chemistry and Physics*.[14] The constants a and b are for pure compounds only; values for mixtures, except air, are commonly not available.

Adding these new P and V terms to the ideal gas law gives rise to the van der Waals equation for real gases. This equation has also been called the *real gas law:*

$$(P + an^2/V^2)\ (V - nb) = nRT$$

This equation can be used to derive pressure, temperature, volume, and composition predictions for conditions away from STP. At STP, a/V^2 approaches zero and b becomes very small compared with V; thus, the van der Waals equation (by substituting 0 for constants a and b) reduces to the ideal gas equation.

Real Versus Ideal Calculations

The difference between real and ideal is best illustrated by numeric example.

Example

Determine the pressure in a compressed gas cylinder filled with air using both real and ideal gas laws.

Assume a compressed gas cylinder has a volume of 0.4 ft^3 (11.3 L).

Assume that this cylinder contains 80 ft^3 (about 2266 L) of gas at atmospheric pressure. Because 1 mole of gas occupies 22.4 L at STP, we can approximate the number of moles at STP:

$$2266\ \text{L} / 22.4\ \text{L/mole} = 101.2\ \text{moles}$$

To simplify, assume the cylinder contains about 100 moles of air.
Use 25°C (298 K) as the temperature.
R is 0.0821 L–ata/K mole).
IDEAL GAS LAW:

Using the ideal equation:

$$PV = nRT$$

Rearranging:

$$P = nRT / V$$

Substituting:

$$P = \frac{(100\ \text{moles})\ (0.0821\ \text{L-ata/deg K moles})\ (298\ \text{K})}{11.3\ \text{L}}$$

Solving:

P = 216.5 ata (This corresponds to 3182.5 psia or 3168 psig)

Compare this to the pressure predicted from van der Waals real gas equation. The values for a and b are from Himmelbau (1982), with R listed in units of L–ata/K mole. This means pressure must be in ata and volume must be in liters for the obtained solution to be correct. The units for constants a and b in the van der Waals equation must be consistent with the units chosen for R, pressure, and temperature. Tables listing values for R, a, and b with a variety of units are available (see reference 3).

REAL (VAN DER WAALS EQUATION):
Using van der Waals equation of state:

$$(P + an^2/V^2)\,(V - nb) = nRT$$

Rearranging:

$$P = \frac{nRT}{(V - nb)} - \frac{n^2\,a}{V^2}$$

Substituting:

$$P = \frac{(100\text{ mole})\,(0.0821\text{ L-ata/K mole})\,(298\text{ K})}{(11.3\text{ L} - 0.036\text{ L/mole}\,(100\text{ mole}))} - \frac{(100\text{ mole})^2\,(1.33\text{ L}^2\text{-atm/mole}^2)}{(11.3\text{ L})^2}$$

Solving:

P = 317.738 ata – 104.159 ata
P = 213.58 ata (this corresponds to 3139 psia or 3124 psig)

There is a difference between the pressure obtained from the real relationship (3124 psig) and the ideal equation (3168 psig) for the pressure exerted by the same physical quantity (100 moles) in the same volume gas cylinder. For most diving, this difference (about 1.4% for air) is insignificant. However, when pressures of gases are used to determine the composition of breathing gases other than air, then the difference between the real gas composition and the gas composition predicted from the simpler, ideal gas behavior can be life-threatening. This difference is particularly true for helium-containing mixtures.

Compressibility

Another approach to resolving the dilemma between ideal and real behavior is the concept of compressibility. In this scheme, the formula for predicting gas behavior is:

$$PV = znRT$$

where z = compressibility factor.

The value z is different for each gas and varies with the pressure and temperature. Tables and graphs to find the appropriate "z-factor" at needed conditions are available. Note that for an ideal gas, z = 1 and the real "compressibility" equation reduces to the ideal gas law.

Beyond Real

The ideal gas equations adequately predict gas behavior at conditions near STP. As conditions move away from STP, more terms have to be added to the equations so that the predictions are close to observed values of pressure, temperature, volume, and composition. These new terms gave rise to the real gas law. The real gas equation of van der Waals correlates well enough with observed values to be used at pressures used in compressed gas cylinder. However, as the pressure continues to increase, the so-called real gas law begins to deviate from observed values and additional terms must be added to this real equation in order for calculated values to correlate with observed gas behavior.

CONCLUSIONS

Understanding the physical principles that govern the underwater environment not only enhances the enjoyment and safety of those who dive in any of the world's waters; it also clarifies the consequences of this activity and the treatments necessary to manage problems that may occur in diving.

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CHAPTER 3 Diving Equipment

Glen H. Egstrom

Diving equipment has evolved dramatically since the 1950s. The increased use of specialized materials has spurred engineering design advances and manufacturing programs. Proliferating full-service dive operations throughout the world are marketing sophisticated products and services to meet the needs of a larger diving population. Divers in the 21st century have access to a wide range of equipment needed to work effectively in widely differing diving environments (Fig. 3–1). This chapter provides practical insight into some important considerations of diving equipment and its effective use. It is important that each diver be comfortable and safe on every dive. It is equally important for divers to be aware of their own limitations when using the wide array of available diving equipment. Adequate preparation for safe, effective diving includes proper training in the use of the equipment within the specific requirements of the diving environment. This training, coupled with knowledge of personal limitations, minimizes the risk of loss of control, which can lead to injury or death.

MASKS

The purpose of the mask is to provide an air pocket over the eye that permits the eye to focus and thereby allows the diver to see clearly under water. The size of the air pocket can vary from that within a special contact lens to that confined within goggles, masks, and even helmets. Problems with masks are related to visual distortion, a restricted visual field, pressure, volume changes with attendant discomfort, and occasional irritation from chemical or bacteriologic sources.

Visual distortion is the result of variations in the distance from the mask lens to the eye. Air has an index of refraction of 1.0, whereas the index of refraction of water is 1.333. This difference results in refraction of the light rays at the air–water interface, causing the diver to perceive objects to be closer and larger than they really are. For example, an object 4 ft away appears to be 3 ft away if it is viewed directly forward with the mask lens perpendicular to the line of vision. However, distortion increases as the line of vision deviates from the perpendicular to the lens, and the object appears to grow larger. Divers adapt readily to this problem and, with experience, learn to adjust their hand-eye coordination and spatial visual judgments accurately.

Restrictions of the visual field through the mask are annoying and are largely a function of the distance of the lens from the eye, the size of the nose, and the dimensions of the lens and the skirt of the mask. Placing the lens close to the eye widens the visual field. The size of the nose and the nose pocket found on many masks creates an obstruction in the medial portion of the visual field. Masks with side lenses at corrected angles are popular, but there is always a distorted area where the planes of the lenses change, which can lead to distorted visual images. For example, a fish swimming across a diver's line of vision may be seen out of the side panel, but as it gets closer it may disappear from view or may appear to bend as it comes into view on the front panel. Additionally, some of the newer clear skirts and side windows permit light to come into the mask from the side and reflect off the inside of the lens and back into the diver's eyes, causing some loss of acuity. Generally, lower-volume masks that place the lens closer to the eye are favored by knowledgeable divers, especially if they enjoy free diving.

Hypoallergenic silicone skirts and straps are more comfortable, cause less irritation of the skin and eyes, and are significantly longer-lasting than natural rubber products. Periodic cleaning, particularly of the inside of the mask, is especially important in climates

Figure 3–1. Fully dressed scuba diver wearing a wet suit, buoyancy compensator with alternative air source, independent air supply, dive computer integrated with tank pressure gauge, mask, fins, and snorkel. (Photograph courtesy of Mike Steidley.)

where black algae and other organisms can grow easily. Cleaning products for the lenses and skirts should be handled with care. On occasion, some of the cleaning products leave behind a residue that may cause severe eye irritation and potential injury. Thorough rinsing of the mask prior to use is a fundamental precaution.

The fit of the mask to facial contours is very important and should be considered carefully before purchase because tightening the mask strap on a poorly fitting mask in order to create a seal results in discomfort and potential leaking. Proper placement of mask straps and wider straps minimizes the angle of pull on the mask and reduces the likelihood of a poor seal. Leaky masks usually result from poor fit, trapped hair, or catching of the edge of the hood under the mask skirt. Ensuring smooth contact of the mask with the skin is a much more effective way of making a seal than is tightening the strap excessively. Periodic checks of the mask skirt will reveal any small tears that may cause small leaks.

Figure 3–2. Several types of diving fins.

Contact lens wearers should use care when diving because these can easily be washed out of the eye should the mask flood suddenly. The practice of inserting eyeglasses into the mask cavity does not provide satisfactory vision and is not recommended. Lenses with the appropriate corrections can be placed in masks quite easily and offer an alternative to contact lenses and eyeglasses.

FINS

Fins (Fig. 3–2) provide a greater resistive surface to improve propulsion. Fins can now meet the needs of almost any diver. The development of long flexible fins for competitive fin swimming and the use of new lightweight materials for better thrust and durability have added a new dimension to diving efficiency. The split-fin technology has achieved great popularity, and tests have shown them to be more efficient with less noticeable leg strain. There is a learning curve for the split fins, especially with any kick other than the flutter-type kick, but most users are pleased with the results.

One criterion for evaluating fins involves comfort, both in the foot pocket and in the stress on the leg muscles under diving conditions. Leg length and strength are also important because a diver with weak muscles on long legs may not be able to effectively use an otherwise excellent fin configuration. For example, weak hip rotational muscles may permit the hip to rotate during the thrust phase of the kick, resulting in the solid blade fin turning on its edge and slicing through instead of flexing and providing thrust. The split-fin technology does not appear to have this problem because each side of the fin directs water flow out through the slot in the middle of each fin, effectively reducing the torque on the hip joint.

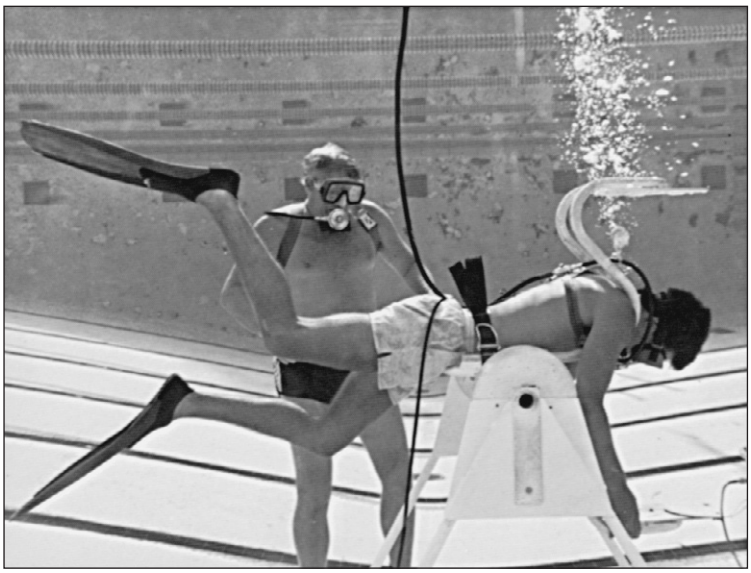


Figure 3–3. Diver swimming while monitored by an underwater ergometer. Note the nearly perpendicular angle of the left knee as the diver prepares to execute the power stroke of the kick.

Fin studies conducted at the University of California, Los Angeles, and elsewhere have consistently demonstrated that individual variations in the ability to use fins effectively for a particular type of diving dictate which fin may be superior for an individual at a given level of conditioning. In an early UCLA study of nine popular solid-blade fins, nine subjects were asked to use each fin in random order, under three workloads, on two separate occasions in a blind test. The subjects were experienced divers, ranging in height from 5 ft 5 inches to 6 ft 4 inches. The data revealed that the longer, narrower fins tended to be slightly more efficient than the shorter, wider fins and that fins with vents, regardless of their direction, were not superior to those without vents. The longer, less flexible fins required stronger leg muscles and delivered higher levels of thrust, without causing rapid fatigue.

Divers should condition themselves to the fins they intend to use in order to use fins effectively. This may require working with fins of increasing rigidity over time in order to develop the necessary strength and endurance to support the workload imposed by the more rigid fins. Cramping and discomfort may be the result of poor adaptation to a particular fin. This logic is appropriate for solid-blade fins, but it is clearly not the same issue for the newer split-blade fins. Hardy and associates (personal communication) conducted extensive tests on all currently available diving fins. They demonstrated a 27% advantage of the split-blade over the solid-blade fins. Their data can be found on the Internet.[1] The Hardy studies found that the split fins did require a flutter kick to give the best results. The split-fin technology is superior while the diver swims straight ahead using the shallower flutter kick and results in lower air consumption for experienced users. Unique swimming techniques must be mastered to enable the diver to use alternate kick styles such as sculling and maneuvers requiring rapid turns. These alternate propulsive maneuvers are somewhat easier to perform with solid-blade fins.

Kicking style is important when evaluating fins because force must be applied in the direction opposite to the intended path. With a drag-dominant kick, in which the fin works primarily as a paddle, the vector of force at 90 degrees of flexion of the knee is primarily to the rear (Fig. 3–3). When the knee is fully extended, the vector of force is perpendicular to the path of travel. A wider, slower kick is more efficient than the rapid, shallow kick often used by novice divers. With a lift-dominant kick, such as a sculling-type kick, the fins respond like propeller blades or wings, directing the resistance to the rear when the leg is nearly straight The power from this type of kick comes from the powerful rotator muscles of the hip joint; the fins sweep through the water rather than paddle against it. Because these two kicks require the use of different functional muscle groups, it helps to become proficient in both kicking styles in order to prevent fatigue. One of the characteristics of the split-fin design is that

the split blade permits the development of strong lift forces on both sides of the split on both the up and down stroke without a strong feeling of strain on the thigh musculature.

Although the modern, lightweight, durable plastic fins provide excellent thrust characteristics and work well with a variety of kick patterns, the buckles and straps are usually large and offer significant drag. Full-foot fins constructed of plastic materials are somewhat more efficient. However, they are lost in surf more readily than open-heel fins with neoprene booties. With booties with open-heel fins, the configuration of the foot pocket should be evaluated for comfort with the foot covering (booties) that will be worn. Many newer booties have thick soles for walking on land, and those attached to or worn with dry suits are often larger than normal. Discomfort from blisters or a tight fit can be avoided with proper fin selection. Each new pair of fins requires a period of "breaking in" while the diver is adapting the leg and hip musculature to the new workload. It is not wise for a diver to use a new, higher-resistance fin on a strenuous dive without preconditioning with the new fin. Comfort and efficiency with new fins develop with progressive increases in the workload.

SNORKELS

Snorkel tubes, used for easier breathing while swimming on the surface, have evolved from simple tubes that are open at both ends to devices that offer purge valves, swivel mouthpieces, advanced materials, and mouthpieces of improved design (Fig. 3–4). An adequate snorkel should permit the diver to swim at high workloads on the surface without encountering excessive breathing resistance that would significantly impair the snorkeler's ability to breathe comfortably. Longer, smaller-diameter tubes with unnecessary bends, internal corrugations, and any unnecessary airway obstructions are undesirable and may lead to intolerable levels of respiratory distress under moderate to heavy workloads.

Self-draining snorkels have reduced the amount of water the diver must move in order to clear the snorkel. These devices contain an exhaust valve below the waterline that permits water trapped in the tube to drop to the level of the surrounding water. A sharp pulse of exhalation pressure is then directed against a smaller water column, and water is purged out of the tube with the momentum generated in the water column. The diver must understand that doubling the flow rate of air through the tube results in the need to overcome the square of the resistance to breathing and that the energy cost of this extra effort greatly increases. The snorkel must be considered an extension of the airway and as such should provide minimal resistance to breathing. If the diver experiences exceptional respiratory distress, he or she should consider swimming on the back with the snorkel removed and, if necessary, also the mask, but only after the buoyancy compensation device has been inflated. Long snorkels increase physiologic dead space and can lead to CO_2 retention and hypercarbia. Excessively long snorkels should be avoided.

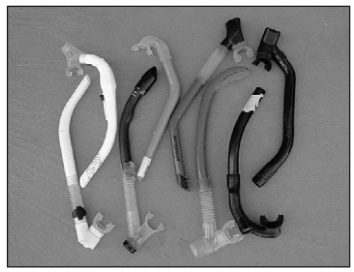


Figure 3–4. Examples of several snorkels. Note that some snorkels are designed with alternative purge valves.

The snorkel mouthpiece should be able to rotate on the snorkel tube so that the lip flange of the mouthpiece can be placed parallel to the teeth and gums. Blisters of the oral mucosa and temporomandibular joint problems can result from poor alignment. Some innovative mouthpieces come in several sizes, and care should be taken to obtain a proper fit. Persons who tend to bite through the tabs on the mouthpiece can often be well served with a customized mouthpiece.

BREATHING APPARATUS

The continuous evolution of breathing apparatus has resulted in a variety of devices with minor differences in construction and function. This discussion covers the generic types of life-support equipment and provides some guidelines for their effective use. Every diver should understand the basic operation

of breathing equipment and should be able to maintain it properly for safe, effective operation. Up-to-date information on most diving equipment for virtually all of the manufacturers can be found on the Internet. In addition, specialized user groups provide alternative sources of equipment information on the Internet.

By far, the most widely used life-support equipment is the scuba, or self-contained underwater breathing apparatus, used by recreational, scientific, commercial, and military divers. This apparatus permits divers to move independently under water while carrying the entire life-support system on their body. Umbilical diving, on the other hand, uses a hose connected to the surface or to a submerged bell or habitat, which limits the diver's mobility. The tradeoffs between the systems generally involve consideration of the need for communication, heating, increased gas supply, and increased workloads.

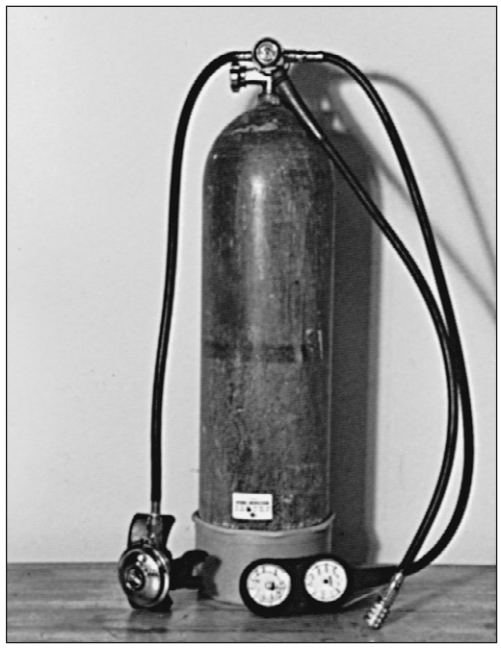


Figure 3–5. Typical open-circuit scuba apparatus consisting of a pressure cylinder, single-stage regulator, underwater tank pressure gauge, depth gauge, and spare hose for connecting to the buoyancy device.

Scuba

OPEN-CIRCUIT SCUBA

The most common form of scuba is open-circuit scuba, which consists of a "tank" or high-pressure cylinder of compressed air and a regulator, which reduces the compressed gas to ambient pressure so that the diver can breathe without difficulty (Fig. 3–5). Breathing gas is inhaled from the regulator and exhaled into the water. These high-pressure cylinders are usually constructed of steel or aluminum, but stainless steel and titanium are also used. Cylinders should be inspected annually by a trained inspector in addition to the required hydrostatic test procedure that is required every 5 years in the United States (but less frequently in other countries). In recent years, the appreciation for the damage that can be caused by exploding, improperly maintained high-pressure cylinders has been promoted by most training agencies. The fact that an 80 ft^3 aluminum cylinder at 3000 psi can release approximately a million foot-pounds of energy—capable of blowing out cement walls and killing people—should be good reason to welcome regular inspections. More information about high-pressure cylinder safety can be found on the Internet.

One Web site provides a list of the high-pressure cylinders that are more likely to develop the neck cracks that often precede catastrophic failure.[2] Although newer materials are less likely to crack, the potential does exist. One particular aluminum alloy identified as 6351 has been largely removed from high-pressure cylinders in the United States for this reason. Sustained load cracking, particularly of the threaded area and necks of aluminum cylinders, is generally easy to detect by a trained inspector using appropriate tools. Although most scuba cylinders are 71.2 or 80 ft^3, tank volumes can range from a few cubic feet in small accessory air bottles to 120 ft^3 in tanks used for deeper or longer exposures. The wide variety of tanks makes it important to ensure that the tank being used is filled to an appropriate pressure and that the proper over pressure "burst disk" is installed. A thin coat of a nongalling dielectric material should also be used on cylinder valves to further control corrosion. Maintaining a positive pressure of 100+ psi within the cylinder at all times can prevent backflow through the regulator when moving up and down in the water column at the end of a dive. Do not breathe the cylinder pressure below 200 psi except in an emergency.

The compressed air in the tank moves through a first stage of the regulator, where its pressure is reduced to an intermediate pressure of 130 to 150 psi. The air then passes through an intermediate pressure hose to a second stage, located at the mouth, where the air pressure is further reduced to the pressure of the surrounding environment and the diver's lungs. The diver exerts a slight negative pressure on a mouthpiece connected to the second stage and causes the opening of a nonreturn valve between the intermediate pressure hose and mouthpiece, allowing air to flow into the mouthpiece and then the airways. The diver then exhales back through the mouthpiece, and the exhaled air is discharged to the open water through a nonreturn exhaust valve. Failure to maintain and inspect the regulator prior to use can result in leaks that can cause water aspiration. This condition can result in coughing and aspiration of contaminated water with subsequent pneumonia.

The basic scuba system can be configured in a variety of ways; generally, the tank, backpack, regulator and accessories, and buoyancy compensators are considered as the basic life support unit. Each manufacturer offers variations on the basic design and competes on the basis of cost, enhanced performance, and design appeal. This equipment is a tool for diving under the water, and skill in the use of this tool must include a basic understanding of the effective and safe operation of the selected components.

The open-circuit systems are designed to provide easy breathing with inhalation and exhalation resistance of less than 3 inches (7.5 cm) of water. Actual resistance is usually about 1.5 inches (3.7 cm) of differential water pressure during normal respiration at sea level. Regulators with inhalation and exhalation resistances above 3 to 4 inches (7.5 to 10 cm) of differential pressure need maintenance or repair. An exception may be the alternative air source regulator: It is sometimes set at a slightly higher resistance in order to reduce the tendency toward air losses induced by "free flow" caused by negative pressure on the mouthpiece while the diver is swimming in currents or making entries from boats. The breathing effort of regulators can be expected to change as a function of several variables, such as respiration rate, water depth, lack of maintenance, and temperature.

Different regulator designs influence breathing resistance characteristics, and the diver should be encouraged to obtain the most efficient regulator for the type of diving planned. For example, most of the higher-quality regulators have balanced first stages that compensate for changing tank pressures, thus providing the diver with a consistent breathing resistance regardless of the tank delivery pressure. This gives the diver an advantage in terms of breathing work but may pose a disadvantage for the diver who fails to heed the tank pressure gauge. Breathing resistance will not increase when the tank pressure becomes low, and the diver may not feel the breathing becoming more difficult. As a result, a careless diver at depth may suddenly find that there is insufficient air to make a normal ascent to the surface.

Although divers should be thoroughly familiar with the capabilities of each piece of equipment, including tank pressure gauges, special attention should be directed to the regulator because its operation is critical. Although regulator failure is extremely rare, it is possible, and divers should be prepared for such an event. A fundamental of good diving practice is the reinforcement of emergency skills. It is important to review the emergency procedures that are appropriate for the equipment currently used by the buddy pair for a given dive. The tank pressure gauge and, in most cases, a dive computer, a depth gauge, and low-pressure hoses are all integral parts of the regulator assembly (see Fig. 3–5). These devices are usually worn together, sometimes with compasses attached in a console arrangement. Frequently, this somewhat weighty console is left unattached at the distal end. This configuration allows the console to swing free and injure a diver in its path. It is also common for the dive computer to be included in the console, either as a stand-alone instrument or integrated with the high-pressure hose. Dive computers with data on remaining airtime frequently replace the tank pressure gauge and depth gauge because information on depth and tank pressure is part of the computer display. If this type of configuration is used, attention must be directed to battery life and minimization of impact.

It is important to arrange a stable position for the location of gauges as well as the alternative air source in order to minimize trauma to the equipment and the diver. For example, the high-pressure hose can, in the absence of

dedicated attachment, be directed under the left arm and under the waist strap of the backpack, so that the console hangs down along the left thigh with sufficient length to permit the console to be held up for easy viewing of the instruments. As an important safety feature, high-pressure hoses normally have a pinhole orifice at the proximal end that restricts flow, thus preventing injury from a flailing hose in the event of a rupture of the high-pressure hose. Periodic assessment of the condition of the hoses and the accuracy of the instruments adds a significant margin of safety to diving performance.

BUDDY BREATHING

Sport scuba diving currently requires an alternative air source as a solution to the out-of-air emergency (which is usually precipitated by a poorly managed air supply). Unfortunately, the proliferation of these devices raises an important equipment-related issue. A variety of alternative air-source configurations are available—octopus, pony bottle, combination oral inflation, breathing devices for the buoyancy compensator, and small independent air systems; buddy breathing from a single air source is also a traditional and acceptable alternative (Fig. 3–6). Although there has been little acceptance of a standardized procedure for any of the alternatives for an out-of-air emergency other than buddy breathing, in many programs buddy breathing is no longer being taught. The large number of potential solutions for an out-of-air emergency causes confusion. The dive team must be comfortable in the use of whatever alternate air source configurations are being used. For buddy breathing and for devices that attach to the oral inflation hose on the buoyancy compensator, the location of the alternative air source is fixed. The octopus system and independent systems, such as pony bottles, are usually configured to the whims of the individual. Frequently, hoses connected to mouthpieces permit different locations depending on the position of the diver's body in the water at any given moment. In short, the configurations for solving an out-of-air emergency are limited only by the imagination of the diver.

With this variety of configurations, it is possible and perhaps necessary to standardize the donor response to the standard out-of-air signal. The source of the air that will be given to the recipient should therefore be located in a consistent position on the front of the donor's body, where a single move by the donor would enable the air source to be presented to the mouth of the person who gave the out-of-air signal. The use of colorful mouthpiece protectors and second stages mounted on the front of the buoyancy compensator can draw attention to the location of the alternative air source.

Figure 3–6. Clockwise from lower left: independent air supply, octopus, primary regulator, Air II, and pony bottle.

To simplify the problem, all divers can agree beforehand that the signal for an out-of-air emergency is a hand drawn sharply across the throat followed by an "I want to buddy breathe" signal with the hand and fingers motioning toward the mouth. After this signal, the donor and recipient would link; the donor would grasp the recipient's shoulder strap with the left hand and the recipient would grasp the donor's shoulder strap with the right hand. At this point, the buddy team would be facing each other as the donor would immediately pass an air source toward the recipient's mouth and the recipient would use the left hand to guide the donor-controlled air source to the recipient's mouth. Such an agreement by the buddies should be established before the dive, when the procedure can be reinforced by careful

rehearsal under nonstressful conditions. A reasonable solution would involve a procedure using a single, simple, standardized device, but it appears that variations in the equipment and techniques promoted in the field require a more generalized response, such as the one just suggested. Regardless of the procedure selected, divers must establish a comfort level for its execution with a given partner. There is no procedure for an out-of-air emergency that does not require repeated rehearsal for its ultimate effectiveness. In any event, the predive buddy check should include the clarification and rehearsal of the emergency procedures, particularly when one is diving with a new buddy.

CLOSED-CIRCUIT SCUBA

A traditional closed-circuit breathing apparatus consists of a mouthpiece and hoses connected to a breathing bag, a carbon dioxide–absorbent canister, and a high-pressure breathing gas supply (Fig. 3–7; also see Chapter 29). The diver inhales from the breathing bag, and upon exhalation, expired gas containing carbon dioxide is routed through a nonreturn valve on the mouthpiece into a canister containing a carbon dioxide absorbent. From the absorbent canister, the remaining breathing gas returns to the breathing bag, where breathing gas is added from the high-pressure supply, and a full bag of breathing gas is once again available for inhalation. The flow of breathing from the high-pressure source may be controlled manually, by fixed flow in simple models, or by automatic sensors that monitor the bag volume to keep the bag full at the end of each exhalation. More advanced mixed-gas rebreather devices contain sensors that monitor the partial pressures of the gases and keep them within the safe ranges for the depth (see Chapter 29). These devices typically require additional training and care in their use. Historically, 1.6 ata (25 fsw) has been the depth limitation for the use of pure oxygen, but recent standards consider 1.4 ata to be an upper limit (see Chapter 6). Anyone using closed-circuit breathing equipment must consider the combination of oxygen partial pressure and exposure time. Sensor-controlled rebreather devices are typically set to hold the oxygen partial pressure between 0.5 and 0.7 atm. Their greatest appeal is the absence of bubbles and a significant reduction in the volume of gas used. Depth limitations for oxygen toxicity and the need for expensive monitoring devices for greater depths have generally restricted the use of these devices to special applications other than recreational diving.

Figure 3–7. Closed-circuit rebreathing device showing counterlung and gas supply. (Photograph courtesy of Lee Somers.)

Although rebreathers of moderate cost are making closed-circuit scuba more affordable, each specific type of device requires a high level of training. Several new closed-circuit devices are being marketed for technical diving applications, and in some areas the technology is being offered to recreational divers. These devices are designed to minimize the technical support and advanced training that has traditionally accompanied closed-circuit rebreathing operations. The use of rebreathers generally requires special training in concert with specialized technical and logistical support. For example, some manufacturers require that a manufacturer's course of instruction

on the use and maintenance of the devices be completed before the unit can be used in the field. Several of these devices are computer-controlled and include a decompression status function that permits divers to monitor their status for depth, bottom time, time remaining for the gas supply, and decompression status. This is in addition to their maintaining the oxygen partial pressure between 0.5 and 0.7 atm. Acceptance of these sophisticated devices has been limited in the general diving population, and any widespread use appears to be several years away.

Increased breathing resistance and large dead spaces are common to most of these systems, and these factors interfere with a diver's ability to perform heavy work at depth. Carbon dioxide buildup is also a significant threat because the absorbent materials tend to lose efficiency because of channeling of the expired gas through the absorbent material, accumulation of moisture in the canister, decreases in temperature, and carbon dioxide saturation of the absorbent. Technologic advances are currently correcting many of these problems, and the rebreathers are becoming easier to use.

Surface-Supplied Diving

The use of hoses and lines from the surface to the diver permits the diver to maintain active communication with the surface and to have almost unlimited supplies of gas, power, and heat. Surface-supplied diving requires highly specialized training and surface support because the danger of fouled lines and entanglement is always present. A detailed treatment of this topic can be found in the *United States Navy Diving Manual* and the *NOAA Diving Manual,* among others.[3,4] Surface-supplied diving is also becoming more popular with public safety divers and others who are concerned with diving in polluted waters. Special methods for isolating the divers from the environment and rinsing the diver following exposure are widely used.

Recreational surface-supplied devices are found in some shallow reef areas, where the divers tow small compressors or tanks of compressed gas on the surface. Although such devices confer some degree of added control over the diver, the basic rules of diving behavior and physics cannot be understated or ignored.

PERSONAL FLOTATION DEVICES

Personal flotation devices have evolved from small front-mounted bladders that could be inflated only orally to large jacket-type flotation bladders with up to 80 lb of positive buoyancy (Fig. 3–8). Tradeoffs in the selection, operation, and training needed for personal buoyancy control are highly controversial. The controversy arises from the consideration of the amount of buoyancy needed for adequate control versus the amount needed in an emergency, with concern regarding rapid ascent rates and restricted movements.

It is important to understand that the buoyancy control device is not a life jacket in the traditional sense. Life jackets have the primary function of floating the victim face up, head out of the water during a water emergency. The buoyancy compensator is used as a tool whose primary function is to maintain the diver in a near-neutral state at any depth while maintaining a desired position, usually face down. To accomplish this, the device should concentrate much of the flotation near the center of mass of the body. This location permits movement around the center of mass for purposes of trim as well as neutral buoyancy. Surface flotation with the head out of the water is easily accomplished by the conscious diver who can inflate and maneuver at will. The unconscious diver, on the other hand, often requires a buddy to help with achieving a position of head out, face up because the buoyancy compensator may not automatically float divers with their face out of the water. Part of the problem relates to the air in the bladder that ultimately seeks a position as near to the surface as possible. In a head-down position, for example, the bubble may be in the highest portion of the device, which tends to hold the diver in that position until the diver's position can be inverted.

A state-of-the-art buoyancy device has a large bladder arranged in a jacket-like configuration so that substantial areas of the device rest under the arms and on the front of the chest. Front-mounted "horse collar" vests and back-mounted, horseshoe-shaped bladders are still preferred by a relatively

Figure 3–8. Low-profile buoyancy compensator with an integrated "quick draw" weight system and combined autoinflation air source. (Photograph courtesy of Mike Steidley.)

small number of divers, but the trend is toward jacket configurations that localize the buoyancy near the center of mass of the body. This location permits smaller and more streamlined configurations. The exception is with technical diving, wherein the buoyancy control device also acts as a load-bearing jacket used for conveying extra tanks and tools for specialized dives. Adequate training in the use of these complex systems is crucial. Such customized arrangements require a lengthy training program before the diver can be in complete control of the devices.

All buoyancy devices have an oral inflation hose with an option for an automatic inflation device designed to deliver air directly from the tank to the bladder. These auto-inflation devices are not standardized in terms of design or placement of the controls, and many are designed to incorporate an alternative air source. The use of these multifunction features in an emergency requires that each member of the buddy team be familiar with the strengths, limitations, and operational control of their own and their partner's equipment. Because emergency use of the buoyancy compensator and the attached alternative air sources is not standardized, there is a risk of confusion and a delayed response to an emergency.

In most buoyancy compensators, the oral inflation hose is located on the left side and should be long enough to permit easy inflation by the user or the buddy. A Velcro collar or other attaching device located on the hose near the mouthpiece with a corresponding attachment surface on the body of the flotation bladder is useful to keep the location of the mouthpiece stable during the dive and in case of an emergency. Familiarity with the location and function of the inflator mechanism and deflator mechanism is fundamental.

The diver should be properly weighted to minimize the need for adjustments of the buoyancy device. Improper weighting can result in the need to add air to the bladder in amounts that can lead to loss of control when the air expands rapidly in the bag during the latter portion of the ascent. Purging excess air during ascent can become difficult if the diver waits too long before starting the process. Recall that the greatest volume changes take place near the surface. The buoyancy device should be used as a tool rather than as a crutch. Most expert divers rarely find it necessary to make major adjustments in buoyancy. Relying on the device to accommodate for overweighting is unnecessary and potentially dangerous. Proper weighting techniques reduce the need for inflating the buoyancy device as depth changes or for surface inflation to avoid becoming submerged.

The inflator valves should be maintained regularly because seawater left in the bladder and the oral inflation hose causes salt deposits and corrosion that often leads to inflator valve malfunction. Rinsing thoroughly, externally and internally, with fresh water after each use and checking for leaks should be fundamental tasks. Single-bladder configurations are usually smaller and produce less form drag than do the double-bag types. If speed remains constant, increasing the frontal surface area of the diver increases the swimming resistance considerably. This increase in resistance

requires a corresponding increase in energy production if the swimming speed is to be maintained. Form drag reductions are important considerations for diver efficiency, particularly in currents or when a diver is moving rapidly through the water. The ease with which a diver explores a reef is deceptive because movement is slow. The reality is that the resistance the diver must overcome is increased four times when the speed is doubled.

The rate of ascent under varying degrees of buoyancy may become a significant factor with larger buoyancy bladders. One liter of air displaces 1.03 kg of seawater (2.3 lb); thus, a buoyancy bladder exerts 2.3 lb of lifting force for every liter of water displaced. Smaller buoyancy compensators have a capacity of approximately 10 L, whereas larger devices often have a capacity of 20 L or more. The increased potential for a loss of control and rapid ascent with greater water displacement requires that the diver adjust the air in the bladder to a safe level before control becomes a problem. Divers should follow the rule of equalizing early and often on ascent as well as on descent, and they should keep in mind the exponential nature of gas expansion, especially as they near the surface. Neutral buoyancy, the state at which a diver neither rises nor sinks, is obviously desirable at the depth the diver desires to hold stable. This is especially true at the end of a dive, when the bottom time is close to the decompression limit and the diver wishes to take an optional safety stop during ascent. The ability to achieve neutral buoyancy at 15 ft requires that the diver consider the problem of increasing buoyancy resulting from air consumption and suit expansion before the dive begins in order to avoid serious over- or underweighting.

HYDRODYNAMIC DRAG

Drag develops in three basic ways during diving: by frontal resistance, by skin friction, and by turbulent or eddy resistance. Drag is the sum of these three types of resistance.

Frontal resistance is the force that develops when an object presents a surface to a fluid and attempts either to move through the fluid or to have the fluid move past it. In either case, the resistive force is a function of the frontal surface area and the shape and speed of the object. If the frontal surface area of a diver is increased and the speed remains constant, the resistive force increases linearly, with a shape function that can be expected to increase the drag further as a result of any protuberances in the configuration. If the frontal surface area is constant and the speed is doubled, the resistive force is quadrupled. Reducing the speed or the surface area reduces frontal resistance dramatically. These relationships emphasize the importance of maintaining a body position aligned with the intended travel path in a head-to-toe direction. Over- or underweighting results in an angle of the body that is upward or downward to the intended travel path and dramatically increases frontal surface exposure, with significant increases in resistance. Inflating buoyancy compensating devices or adding equipment to the body also results in an increased frontal surface area. Streamlining efforts can effectively reduce this factor.

Surface friction is the force that develops as fluid particles pass over the body and exert frictional drag on the body. Viscosity, speed, and the shape of the body are important considerations. A laminar flow of water over the surface of the diver is nearly impossible, but flaps, straps, and other protuberances can be reduced by proper attention to rigging and smoothing the body surface as much as possible.

Eddy resistance, or turbulent flow, usually results when the smooth flow of water passing over the body is disrupted by an irregularity. Where water passes over sharp bends or corners such as the end of the tank or the back of the head when the neck is hyperextended, the turbulence creates resistance that slows the diver's forward progress. Divers being towed by boats or diver propulsion vehicles are faced with the prospect of losing their masks if they position their heads inappropriately and permit turbulent flow to develop on the edge of the mask. Eddy resistance can accentuate the displacement of any loose pieces of gear that are not secured properly.

These drag-producing factors become more important as the speed of water flowing over the body increases. The exponential nature of the increase is frequently not well understood. Divers who go downstream of the boat and then attempt to swim up against the current to return to the boat at the end of the dive may fail simply because they cannot produce the force

necessary to overcome the additional resistance caused by the current.

THERMAL PROTECTION

Chapter 13 provides a detailed treatment of physiology of hypothermia. When a diver enters water with a heat conduction capacity 25 times that of air, heat is conducted from the body and adaptive changes occur to protect the body from a fall in core temperature. Because the comfort range for humans is approximately ±1°C of core temperature, and because a gain or loss of 3° to 4°C in the core temperature can result in significant physiologic impairment, additional thermal protection is necessary under most prolonged diving conditions. Protective garments have been developed to increase the length of time that a diver can remain within the safe range of core temperature.

Many divers use so-called skin suits made of nylon, Lycra, or thin laminated materials. These suits are worn for protection from the sun, for warm-water thermal protection, and as undergarments for wet or dry suits. Whereas the lightest of these suits have only limited thermal insulation, other, more substantial models add comfort and protection in water above 75°F (24°C).

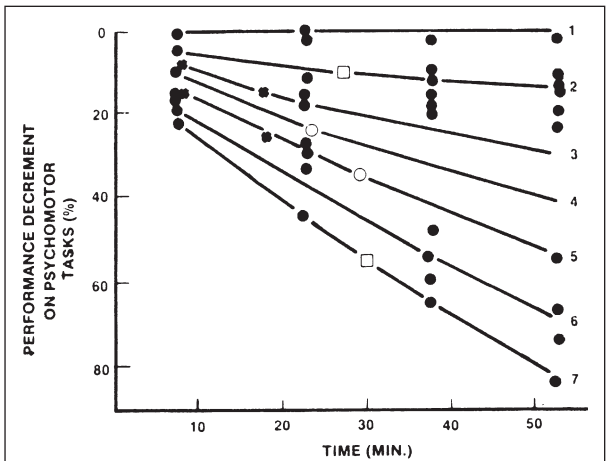
The most common protective garment is the wet suit (Fig. 3–9), which uses a layer of air-impregnated neoprene rubber as the insulating boundary to trap water next to the diver's skin as an insulating layer. A well-fitted wet suit holds the water in place so that heat is not exchanged by water displacement. The wet suit compresses as the diver descends, and insulation is reduced with greater depths, where lower water temperature is usually found. The graph shown in Figure 3–10 can be used as a guideline for anticipating the effects of cold water on a diver's performance. The data on the graph reflect the temperature effects on a diver wearing a 0.25 inch (6.35 mm) thick neoprene wet suit with hood, booties, and gloves. The numbers under the temperature readings reflect the appropriate decrement curve for listed motor skills. For example, after diving in 60°F water for 50 min, fine digital manipulation skills would be expected to be reduced by more than 50% (see Fig. 3–10).

The improvement in wet suit materials has led to improvements in the wet suit's insulating value and comfort (see Chapter 13). Less-compressible but still flexible materials

Figure 3–9. Wet-suited divers ready for a dive. Suit thickness is based on the degree of thermal protection needed. (Photograph courtesy of Mark Lonsdale.)

reduce the loss of thermal protection at depth. More-flexible materials such as Lycra, used on the inner and outer surfaces of the wet suit, permit a snug fit that eliminates internal water pockets that may result in flushing water through the suit with a resultant increase in heat loss. Neoprene rubber has also been used to develop a dry suit that fits much like a wet suit but contains seals at the neck, wrist, and ankles that prevent water from entering the suit. These suits offer better thermal protection but usually provide less mobility for the diver.

Dry suits have become more popular in recent years because of improvements in fit and mobility. Comfortable insulating undergarments, effective valve mechanisms, and better seals have also been added. Training in the proper use of dry suits is required. Newer dry suits are sometimes called shell suits because they provide a waterproof outside covering over an inner insulating garment (Fig. 3–11). These suits provide considerably improved thermal protection over the other two types of thermal protection garments but restrict range of motion



PROPER DECREMENT CURVE

TYPE TASK	WATER TEMPERATURE 70	60	50	40
FINE DIGITAL MANIPULATION	1	5	6	7
SIMPLE ASSEMBLY	1	2	4	6
GROSS BODY & POWER MOVE	1	2	2	3

● STANG * WIENER (1970)
● BOWEN (1968)
○ WELTMAN & EGSTROM, ET AL. (1970)
□ WELTMAN & EGSTROM, ET AL. (1971)

Figure 3–10. Upper graph shows the percentage of reduction in performance in cold water. Numbers to the right indicate the conditions of measurement described in the lower graph. (See text for interpretation.)

Figure 3–11. A trilaminate "shell" dry suit that permits several types of undergarments to be used for thermal insulation. (Photograph courtesy of Diving Unlimited International, San Diego.)

and may impose difficulties in buoyancy control. It is recommended that such suits be used with an independent buoyancy control system after the diver has been thoroughly trained and has become skilled in the operation of the entire diving system. Using the shell suit as a buoyancy control system may result in difficulties with an internal air bubble, which will move to the portion of the suit closest to the surface of the water. Such a bubble can be large enough to cause serious control problems. Divers must pay strict attention to controlling suit volume to avoid loss of mobility while under water. Specific training and development of adequate levels of strength and endurance to meet the demands of the environment should enable the diver to concentrate on the dive and the tasks involved rather than on equipment function.

DIVE COMPUTERS

Because decompression tables are based on predictable mathematical models of physiologic parameters, decompression can be computed with portable dedicated microprocessor computers. These dive computers monitor pressure and time; they then provide information that can guide the diver through a proper decompression. Divers have many choices of designs and algorithms with this technology. These choices are not unlike those available to the personal computer owner, who is faced with the fact that devices that are bought today are likely to be soon

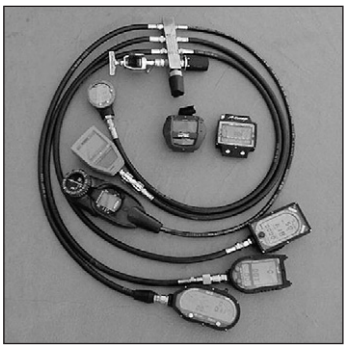


Figure 3–12. Device used for comparative testing of dive computers.

outmoded. Device characteristics vary widely, and many of these computers have far more functions than most divers need. Dive computers may be independently mounted as stand-alone devices, or they may be integrated into consoles with tank pressure, remaining airtime, and other functions (Fig. 3–12).

The choice of a personal dive computer should be based on a careful review of functions and specifications. Reading the owner's manual should provide a better understanding of the nature of the calculated risks and the design assumptions that are inherent in each device. This understanding will minimize, but not guarantee, diver safety. Significant databases of comparative information are available on the Internet and in diving-related consumer reports.[5]

Because the dive computer constantly monitors depth and time, it provides information (based on the assumptions made in its design) that enable the diver to make immediate informed decisions regarding the conduct of the dive. Computers are not risk-free. They are tools that provide the diver with information but do not guarantee that following the advice will result in a safe dive. A review of several manuals for decompression computers revealed multiple warnings and 40 to 100 pages of instructions. Warnings include avoiding decompression dives, carrying a set of printed dive tables as a back-up source for decompression schedules, and maintaining the prescribed ascent rate. Other decompression computer manuals suggest avoiding high-altitude diving without special training, avoiding air flights for 24 hours after diving or until the computer indicates it is safe, diving with a partner, and limiting sport diving to 60 ft. Manuals warn that the risk of decompression sickness is increased if the recommended decompression schedule is ignored.

These examples, which are representative of statements found in most current instruction manuals, indicate the concern and uncertainty associated with the current trend toward the widespread use of dive computers. Current dive tables and dive computers are not perfect, but they have demonstrated safety and efficacy in millions of uneventful dives. Responsible divers use all of the diving safety tools at hand to minimize the hazards associated with diving and rarely have problems they cannot resolve with their tools. Adequate knowledge and training can result in a realistic calculated risk. This "informed consent" is fundamental to the acceptance of the calculated risks involved in any inherently risky activity. Because there are no "safe" tables or "safe" dive computers, every diver must accept some risk when diving for sport or occupation. Experience to date with the dive computers is somewhat mixed. The largest single cause of decompression sickness while using computers appears to be diver error associated with a lack of understanding of the limitations of the devices. The causes of the problems associated with the misuse of the dive computer are probably little different than those associated with the misuse of printed dive tables. Carelessness, lack of understanding regarding the limitations of the logic, and failure to recognize personal limitations account for the greatest number of decompression incidents.

Fundamental advice on the use of dive computers and tables remains as it has been since they were developed. Dive computers are tools that can help divers understand their dive profiles. Dive computers cannot guarantee that their use will prevent decompression sickness. Properly used, these devices should reduce the risk under normal circumstances.

The proliferation of diving equipment has complicated the diver's quest for comparative information, which can be used to make informed choices. Fortunately, the Internet has opened information channels that allow for up-to-date information. Virtually all of the training agencies, equipment manufacturers, dive magazines, and special-interest groups associated with diving offer information and links to issues involving diving equipment.

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CHAPTER 4 Inert Gas Exchange and Bubbles

Richard D. Vann

Decompression sickness (DCS) is a disease that occurs when the body is exposed to a decrease in ambient pressure sufficient to cause dissolved gases to leave solution and form bubbles. It affects divers, aviators, astronauts, and compressed-air workers, but understanding and avoiding DCS consistently have been elusive goals. There have been various obstacles:

- The disease differs widely in severity, has no definitive test, and is uncertain in diagnosis.
- Experimental trials are hazardous and costly.
- Epidemiologic observations are time-consuming and difficult to document.
- Detecting bubbles and measuring inert gases in tissue are challenging.
- Many of those affected by DCS are less interested in understanding it than in pursuing their occupational or recreational activities.

Nonetheless, steady progress has provided decompression procedures that have virtually eliminated death and made paralysis much less common than at the end of the nineteenth century.[1–5] Progress can be expected to continue as the evidence-based approach that has been effective elsewhere in medicine is applied to decompression science.[6]

The scientific understanding of DCS began in 1878 with Paul Bert's observations that the adverse reactions suffered by animals and humans after decompression from high pressure were often associated with bubbles in blood and tissue.[1] Bert concluded that bubbles were the cause of DCS, and DCS pathophysiology was studied throughout the latter part of the nineteenth century,[4] but there was little work on how to avoid it. Bert had recommended slow linear ascent at 3 fsw/min, and linear ascent was widely adopted but without consensus as to the safest rate: the *Royal Navy Diving Manual* specified 5 fsw/min, whereas in Germany, Heller[4] recommended 1.5 fsw/min.[3] None of these prescriptions successfully avoided serious or fatal injury.

In Britain, the government's official indifference to DCS ended in the early twentieth century when the submarine *A1* sank with the loss of all hands. The British government recognized that successful submarine development required diving support and that decompression safety, in particular, needed improvement.[7] John Scott Haldane, a well-known respiratory physiologist, had a theory that offered an alternative to slow linear ascent, and the Royal Navy agreed to test its validity.

HALDANE DECOMPRESSION THEORY: STAGE DECOMPRESSION

Paul Bert's hypothesis that bubbles caused DCS was central to Haldane's theory, and he argued that DCS would not occur if bubbles could be avoided.[3,5] Having noted that caisson workers were free from DCS if they decompressed to 1 ata from not more than 2 ata, he proposed that decompression would be bubble-free so long as the difference between the dissolved nitrogen tension in tissue and the absolute pressure, the *supersaturation*, did not exceed a critical value. Haldane expressed supersaturation as the ratio of tissue nitrogen tension to absolute pressure and claimed that bubble-free decompression was possible as long as the supersaturation ratio did not exceed 2:1. He tested this hypothesis by exposing goats to high pressure followed by immediate decompression to a lower pressure. Within biologic variability, he convinced himself that decompression was safe from 2 to 1 atm, from 4 to 2 atm, and from 6 to 3 atm (Fig. 4–1).

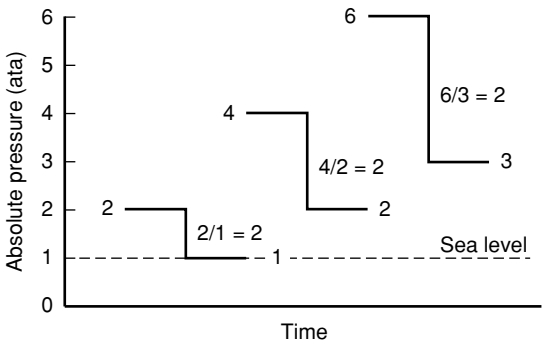

Figure 4–1. Derivation of Haldane's 2:1 supersaturation ratio rule. Goats were exposed for 4 hrs at various pressures before ascent to a lower pressure. Decompression sickness did not occur if the initial pressure was less than two times the final pressure.

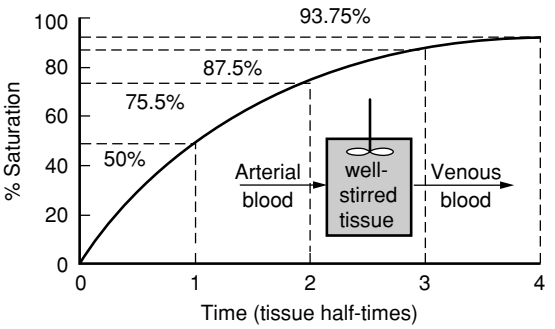

Figure 4–2. Absorption of nitrogen as a function of time as measured in tissue half-times. The half-time defines the rate of nitrogen exchange in well-stirred tissue (see *inset*).

Haldane had been impressed by the strong effect that dive duration had on DCS risk. Alexander Lambert, a famous Siebe-Gorman diver, had safely salvaged £70,000 in gold from the wreck of the *Alfonso* during 33 dives at 162 fsw (48.6 msw) with bottom times of 25 min,[3,8] but on extending his dive to 45 min, he experienced paralysis from which he never fully recovered. According to the current U.S. Navy tables,[9] Lambert's 25 min dives needed 30 min of decompression while the 45 min dive needed 100 min of decompression.

Haldane thought that Lambert's short dives were safe because he had absorbed insufficient nitrogen to exceed the 2:1 supersaturation ratio at which bubbles would form. This suggested to him that a diver would absorb nitrogen progressively while at depth as the circulation carried dissolved nitrogen from lungs to tissue, and he reasoned theoretically that nitrogen was absorbed rapidly at the start of a dive but more slowly as the tissue nitrogen tension approached the alveolar partial pressure. When these were equal, the diver was said to be *saturated* with nitrogen at his current pressure.

Because diffusion distances between tissue capillaries are very small, Haldane thought that arterial nitrogen would diffuse into and completely equilibrate with nitrogen in tissue and venous blood. Today, such tissue is described as *well-stirred* or *perfusion-limited* (Fig. 4–2, *inset*), with effectively instantaneous diffusion of nitrogen between blood and tissue.[10] Blood flow is the sole determinant of inert gas exchange in a perfusion-limited tissue. Without formal mathematics, Haldane showed that perfusion-limited tissue could be characterized by a half-time that defined the tissue's rate of saturation (or desaturation) such that the difference between the arterial tension and the tissue (or venous) nitrogen tension was reduced by half with each passing half-time (see Fig. 4–2). Thus, a tissue would be 50% saturated (or desaturated) in one half-time, 75% saturated in two half-times, 87.5% saturated in three half-times, and so on until saturation or desaturation was effectively complete (98%) after about six half-times.

The Mathematics of Nitrogen Exchange in Perfusion-Limited Tissue.

In describing nitrogen exchange in perfusion-limited tissue, the venous (P_vN_2) and tissues (P_tN_2) nitrogen tensions are assumed equal to represent rapid diffusion between closely spaced capillaries. A mass balance for nitrogen is given by

$$(N_2)_{stored} = (N_2)_{in} - (N_2)_{out}$$

The mass balance is illustrated in Figure 4–3 in which nitrogen enters with the arterial blood at a tension equal to the alveolar nitrogen partial pressure (P_AN_2) and leaves with the venous blood where α_b and α_t are the nitrogen solubilities in blood and tissue, Q is blood flow and V_t is the tissue volume. In this example, P_aN_2 is assumed to change instantaneously to a constant value, P_a, at a time, t, equal to zero.

The Mathematics of Nitrogen Exchange in Perfusion-Limited Tissue—cont'd.

The rate of change of P_tN_2 defines the rate at which nitrogen is stored in the tissue. Thus,

$$\alpha_t * V_t * dP_t/dt = \alpha_b * \dot{Q} * P_aN_2 - \alpha_b * \dot{Q} * P_VN_2$$

and

$$dP_t/dt + k * P_t = k * P_a \quad (4\text{-}1)$$

where

$$k = \alpha_b * \dot{Q}/\alpha_t * V_t$$

The solution to Equation 4–1 is

$$P_t(t) = P_a * [1 - \exp(-k * t)] + P_0 * \exp(-k * t) \quad (4\text{-}2)$$

where P_0 is the initial N_2 tension and the tissue half-time is

$$T_{1/2} = 0.693/k = 0.693/(a_b * \dot{Q}/a_t * V_t)$$

$P_t(t)$ in Equation 4–2 is the sum of the decay in the initial nitrogen tension and the response to a step change in P_AN_2 as illustrated in Figure 4–3.

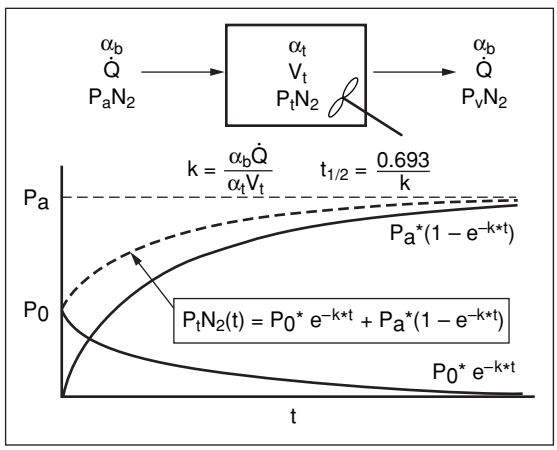

Figure 4–3. The mathematics of nitrogen exchange in a well-stirred tissue (see text).

Haldane postulated that the tissues of the body have different perfusion rates that he represented by half-times of 5, 10, 20, 40, and 75 min (Fig. 4–4, *inset*). Tissues with shorter half-times saturated (or desaturated) faster than those with longer half-times (see Fig. 4–4). The longest tissue half-time determined the exposure for which the entire body reached equilibrium (saturated) with atmospheric nitrogen after a change in pressure.

The behavior of Haldane's five-tissue model is illustrated in Figure 4–5 for a 4 min dive on air to 168 fsw (50.4 msw), with descent and ascent at 5 fsw/min.[3] To simplify his calculations, Haldane assumed air to be 100% nitrogen. Tissue with a 5 min half-time is nearly saturated by the end of the bottom time and begins to desaturate immediately on ascent. Slower tissues continue to absorb nitrogen during initial ascent.

These ideas led Haldane to conclude that the accepted method of slow linear ascent was both unsafe and unnecessarily long. He called his alternative method *stage decompression* in which a rapid initial ascent at 30 fsw/min (9 msw/min) was followed by increasingly longer stages or stops as the diver approached the surface. Figure 4–6 compares stage decompression with uniform ascent at 3.5 fsw/min for a 16 min dive to 168 fsw (50.4 msw). Nitrogen exchange in

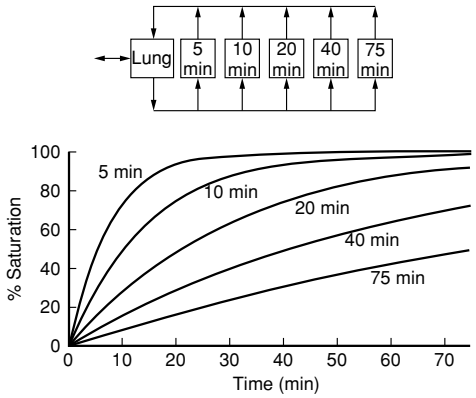

Figure 4–4. Nitrogen exchange in the human body as defined by Haldane's five parallel well-stirred tissues (see *inset*). Tissue half-times are indicated in minutes.

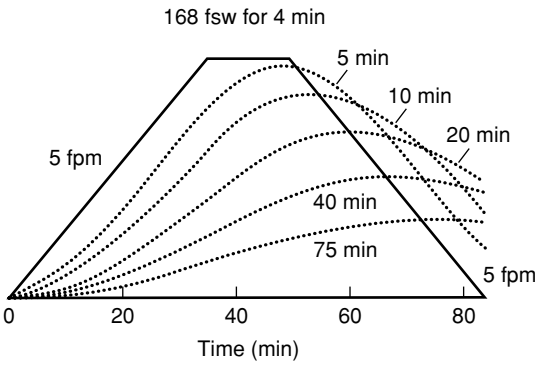

Figure 4–5. Nitrogen uptake and elimination from the five Haldane tissues during a 4-min dive to 168 fsw (51.4 msw). Ascent and descent are at 5 fsw/min.

What Is the Half-time of the Slowest Tissue in the Body?

If tissues are 98% saturated (equilibrated with alveolar nitrogen) in six half-times, a 5 min tissue is nearly saturated in 30 min and 75 min tissue is nearly saturated in 7.5 hrs. The slowest tissue used to calculate the U.S. Navy dive tables was 120 min, and these tables consider a diver to be "clean" (free of excess nitrogen) at 12 hrs after a previous dive[11,12]. As 24–48 hrs is believed to be long enough to saturate the body with inert gas during a saturation dive (Chapter 6), this would imply that the slowest tissue half-times are on the order of 240–480 min. Thus, Neo-Haldanian decompression theories with tissue half-times as long as 1,440 min[13] would not appear to represent perfusion-limited inert gas exchange and may suggest other physiologic mechanisms.

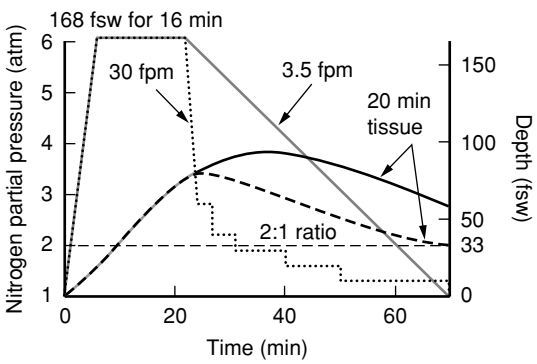

Figure 4–6. Comparison of slow uniform ascent and stage decompression. The nitrogen tension in the tissue with the 20-min half-time is higher after uniform ascent than after stage decompression.

tissue with a 20 min half-time is shown for both methods of ascent. (The other tissues are omitted for clarity.) With stage decompression, rapid initial ascent avoids the additional nitrogen uptake that occurs with slow linear ascent. The stages were chosen so that the 2:1 pressure ratio was never exceeded in any tissue. Stage decompression allowed the diver to surface with a 2:1 pressure ratio in a 20 min tissue, whereas with linear ascent, the pressure ratio was 3:1.

Haldane published two tables of stage decompression schedules.[3,5] The first was for short dives as deep as 204 fsw (62.5 msw) with decompression times of up to 30 min. This table proved very successful for the short, deep dives that were typical for the unpredictable waters of the British Isles and virtually eliminated DCS, but with experience, the deeper decompression stages were judged to be unnecessary. This is illustrated in Figure 4–8*A* for a 40 min dive to 100 fsw (30 msw) with decompression according to the Haldane and U.S. Navy schedules.[9] The first stop of the Haldane schedule is at 30 fsw (9 msw), whereas that of the U.S. Navy schedule is at 10 fsw (3 msw). The total stop times are 15 min for the U.S. Navy schedule and 30 min for the Haldane schedule.

Linear Ascent and Stage Decompression a Century Later.

A modern experiment by Broome in 1996 appears consistent with Haldane's stage decompression theory[14]. Broome dived two groups of 20 pigs to 200 fsw (60 msw) for 25 min (Figure 4–7). One group decompressed at a linear ascent rate of 20 fsw/min (6 msw/min) while the other group ascended in two phases, at 60 fsw/min (18 msw/min) until reaching 110 fsw (33 msw) and at 12.9 fsw/min (4 msw/min) to the surface. Both groups reached the surface in 10 min, but with uniform ascent, the DCS incidence was 55% while with the bi-phasic ascent, the incidence was 25%. The difference was nearly significant at p=0.053. These results (Figure 4–7) are consistent with the Haldane theory illustrated in Figure 4–6.

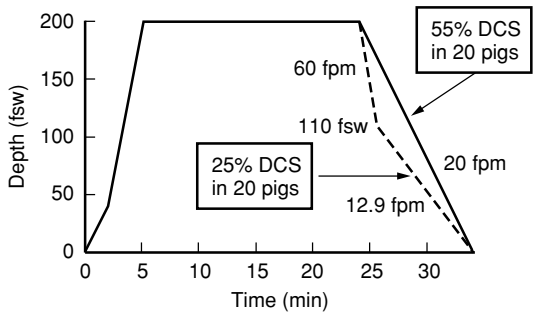

Figure 4–7. A comparison of the DCS incidences in pigs for uniform ascent at 20 fsw/min (fpm) with biphasic ascent at 60 fsw/min to 110 fsw (33 msw) and 12.9 fsw/min to sea level.

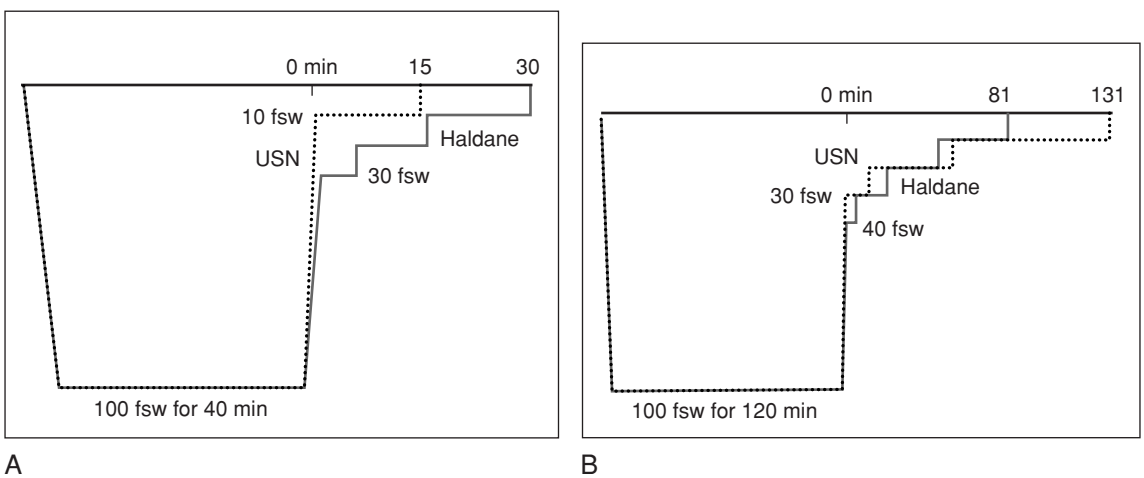

Figure 4–8. *A.* A decompression schedule from Haldane's first table[5]. Schedules from this table have deeper first stops and more decompression time than corresponding U.S. Navy schedule[9]. *B.* A decompression schedule from Haldane's second table. Schedules from this table have deeper first stops but less decompression than corresponding U.S. Navy schedules.

Haldane's second table was for dives with bottom times longer than 1 hour and with more than 30 min of decompression. Figure 4–8*B* shows the Haldane and U.S. Navy schedules for a 120 min dive at 100 fsw (30 msw). The first Haldane stop is at 40 fsw (12 msw), whereas the first U.S. Navy stop is at 30 fsw (9 msw). The Haldane schedule is 81 min long, whereas the U.S. Navy schedule is 131 min long. The decompression schedules of Haldane's second table proved too short to prevent DCS.

The Decade of Decompression, 1897–1907: J.S. Haldane and L. Hill.[7,15]

Robert Davis, manager of the Siebe Gorman diving company, concluded in a 1898 article, "…for the man who succeeds in overcoming the difficulties which now present themselves, there lies at the bottom of the sea a wealth compared with which the combined forces of our great millionaires are infinitesimal." Leonard Hill, a young professor on the rise in London academic medicine, saw that whoever solved the problems of deep diving would gain the glory of scientific reputation, and he arranged with Davis to conduct decompression experiments based on Paul Bert's theory of uniform decompresssion. Diving suddenly became more than an academic pursuit in 1904, however, when the submarine, *A1*, was lost with all hands. In response, the Vickers armaments company, exclusive manufacturer of submarines for the Navy, bought Siebe Gorman but kept Davis on as Managing Director. As diving grew in national importance, Davis and Hill, both egomaniacs, feuded publicly in *The London Times* over the primacy of their ideas. The spat alerted John Scott Haldane and motivated his concept of stage decompression, but rather than *The London Times*, Haldane took his proposal to the Admiralty. The Admiralty was surely influenced by Haldane's considerable scientific reputation, but his older brother, Richard, soon to become Minister of War, may have played a less obvious role. Davis recognized the merit of Haldane's ideas as well as his political connections, and Siebe Gorman supported his research. Haldane's well-financed and organized program was galling to Leonard Hill, and he did his best to interfere, leading Haldane to test uniform decompression before his own stage decompression. After a few days and several dead goats, Hill's theory was demolished, and he went away seething. He and Haldane were to lock horns many times over the next 30 years.

Haldane and Hill lived in the golden Edwardian era when British economic and military power, not to mention self-esteem, were at a peak, and good ideas were encouraged to reach fruition. Although Hill was proved wrong about uniform decompression at the time, his strongly held belief has since been shown appropriate for saturation diving, and his concept of a submersible decompression chamber that he and Davis had wrangled over, was prophetic. Born into a tradition of leadership within the Scottish aristocracy and the world's greatest expert of his day on breathing, Haldane also was not without limitations. On an expedition to Pike's Peak in Colorado in 1911, he demonstrated the role of hypoxia in ventilatory control and showed that acclimatization to high altitude represented the body's defense of its oxygen level. But he had also convinced himself that acclimatization to high altitude led the lungs to actively "secrete" oxygen from alveolar air into the blood, and he was undeterred by mounting evidence to the contrary. This firmness of mind ultimately cost him the sought-after Professorship of Physiology at Oxford and excluded him from research into poison gases during the war of 1914–1918.

Why did Haldane adhere so tenaciously to the idea of oxygen secretion in the face of so much contrary evidence? Perhaps his strong self-confidence and habit of being right overcame his good judgment. History often rewards hubris with disappointment.

DIFFUSION BETWEEN BLOOD AND TISSUE

The primary factor controlling the exchange of dissolved inert gas between blood and tissue is blood flow, but the effects of diffusion can slow down inert gas exchange in tissue. These effects include diffusion between heterogeneous regions of tissue, within capillary domains, or between adjacent arterial and venous vessels. Inert gas tension gradients and diffusion between adjacent regions of tissue may occur in tissues of heterogeneous solubility or perfusion.[16,17] Simulations indicate that gradients can develop between 1 cm thick tissue regions when regional blood flow differences exceed 3:1.[18] Diffusion between heterogeneous tissue regions was a possible explanation for the continued absorption of a nitrogen isotope by the human knee after the isotope source had been removed from the inspired gas.[19]

Gases diffuse between blood and tissue within capillary domains. Blood in adjacent capillaries may flow in the same direction (concurrent) or in opposite directions (countercurrent). Diffusion distances are small in most tissues,[20,21] and diffusivities are reasonably

large.[22,23] For concurrent capillary flow with accepted diffusivities and diffusion distances in vascular tissues, calculations indicate that diffusion is rapid and inert gas concentration gradients are minimal.[10,24–30] Diffusion might be more important in tissues such as bone, articular cartilage,[31] and the eye,[32] where diffusion distances are on the order of millimeters.

With countercurrent flow in adjacent capillaries or between arterial and venous vessels, diffusion effects are more complex and the interpretation of inert gas exchange measurements is less certain.[33] Gas molecules in venous vessels can diffuse into adjacent arterial vessels and be retained in tissue,[34–37] an effect that is more pronounced for rapidly diffusing gases.[38,39] Novotny and colleagues found that when inert gas exchange and blood flow distribution to muscle were measured simultaneously, gas exchange was slower than predicted on the basis of the measured flow distribution.[40] The retention of gas in tissue by countercurrent diffusion was a possible explanation for this observation and might be one reason that tissue half-times in decompression models are longer than would be expected on the basis of physiologically reasonable blood flow.

DECOMPRESSION THEORIES BASED ON SUPERSATURATION

The tissue half-times and pressure reduction ratios of Haldane's original model evolved with experience and a search for safe and efficient decompression.[41] Some of these changes are summarized in Figure 4–9. The tissue half-times are shown on the x-axis and the corresponding pressure ratios on the y-axis. Haldane's original 2:1 supersaturation ratio in five tissues with 5 to 75 min half-times appears as a straight line. As a result of human decompression trials for submarine escape, later workers concluded that higher ratios could be tolerated in the tissues with 5-, 10-, and 20-min half-times but lower ratios were needed in the tissues with 40 and 75 min half-times.[42] The higher ratios in the faster tissues eliminated the deeper stops and reduced the total stop time for short dives. For long dives, a 120 min half-time tissue was added to provide additional decompression time. Such changes eventually led to the present U.S. Navy schedules shown in Figure 4–8.

Haldane's supersaturation ratio was a measure of the supersaturated nitrogen that could be tolerated without bubble formation after ascent to the surface or next decompression stop. Haldane had treated air as 100% nitrogen, but later workers redefined Haldane's supersaturation ratio as the ratio of the nitrogen tension at the present depth to the ambient pressure at next shallower stop. This made it possible to use nitrogen-oxygen mixtures other than air. The *M-value* is an equivalent measure of supersaturation that defines the nitrogen tension (measured in fsw) from which it was safe to ascend to the next shallower decompression stop. M-values allow the tissue ratio to change with depth and introduce additional flexibility. The Haldane tissue ratio, the tissue ratio, and the M-value are equivalent measures of the ascent criteria that ostensibly define bubble-free decompression. Their definitions and equivalent values are illustrated in Table 4–1 for the tissue half-times used in the decompression model for the U.S. Navy decompression tables.[9] Table 4–2 shows a table of M-values for decompression diving.

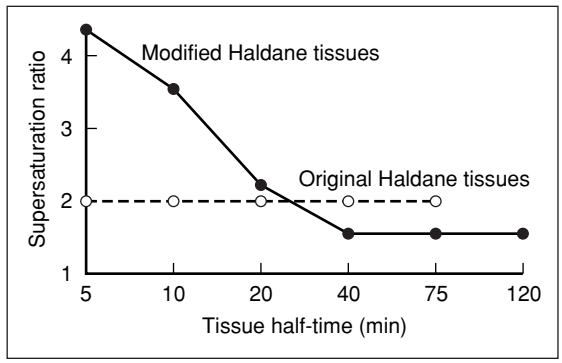

Figure 4–9. Allowable pressure ratios for the original and modified Haldane tissues[41].

A Haldane decompression model with M-value ascent criteria was adopted for use in 1983 by the first commercially successful digital dive computer, the Orca EDGE, that tracked a diver's decompression obligation in real time.[43] The EDGE had a display (Fig. 4–10*A*) that showed the M-values in each of 12 theoretical tissue compartments with half-times ranging from 5 to 480 min. Figures 4–10*B* and *C* show how the theoretical nitrogen tensions in the tissue compartments absorb and eliminate inert gas during a dive.

Tissue ratios and M-values were not the only modifications to the Haldane decompression model. Hempleman replaced Haldane's series of perfusion-limited tissues with a single slab of avascular tissue (suggesting cartilage) into which nitrogen diffused from

Table 4–1. Definitions and examples of safe-ascent criteria based on supersaturation*

	Tissue Half-Time (min)					
	5	*10*	*20*	*40*	*80*	*120*
Haldane ratio = $P_{N2}/(0.79 \times P_B)$	4	3.4	2.75	2.22	2	2
Tissue ratio = P_{N2}/P_B	3.15	2.67	2.18	1.76	1.58	1.55
M-value (fsw) = ratio × (D_{next} + 33 fsw)	104	88	72	58	52	51

*The M-value shown is for ascent directly to the surface from depth. See text for further discussion.

Table 4–2. M-values for ascent in increments of 10 fsw

Safe Ascent Depth (fsw)	M-Values: Tissue Half-Time (min)								
	5	*10*	*20*	*40*	*80*	*120*	*160*	*200*	*240*
0	104	88	68	46	38	35	34	34	33
10	120	98	78	56	48	45	44	44	43
20	130	108	88	66	58	55	54	54	53
30	140	118	98	76	68	65	64	64	63
40	150	128	108	86	78	75	74	74	73
50	160	138	118	96	88	85	84	84	83

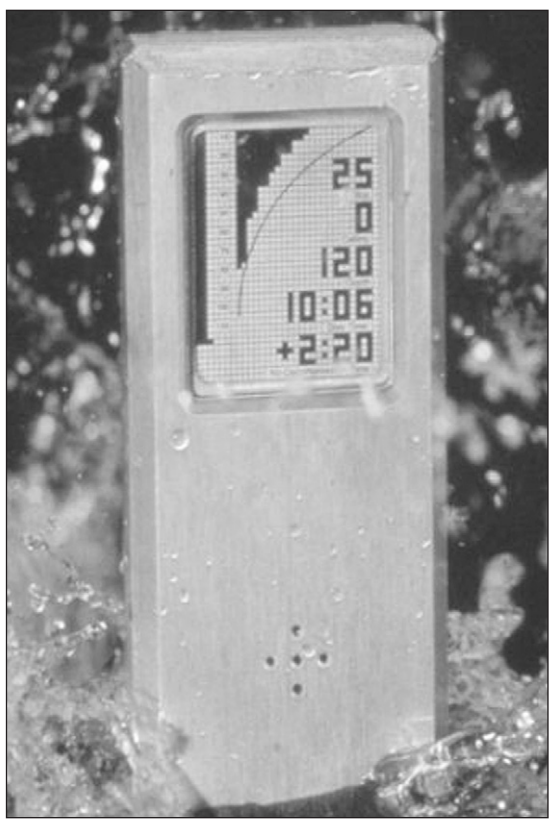

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Figure 4–10. *A.* The Orca EDGE (Electronic Dive Guide Experience), the first commercially successful digital dive computer. The display of the EDGE had a bar graph that represented the computed nitrogen tension (in fsw) in each of 12 Haldane tissues.

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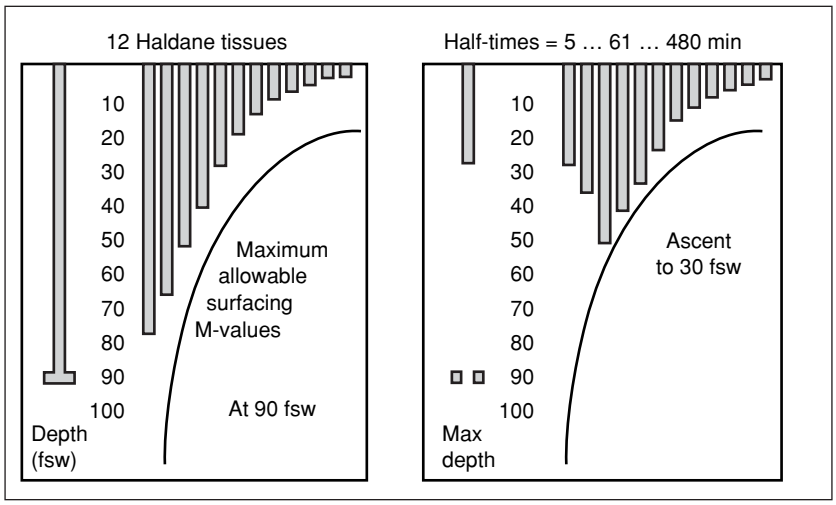

Figure 4–10—cont'd. *B.* The EDGE display during a dive to 90 fsw (27 msw). *C.* The display after ascent to 30 fsw (9 msw).

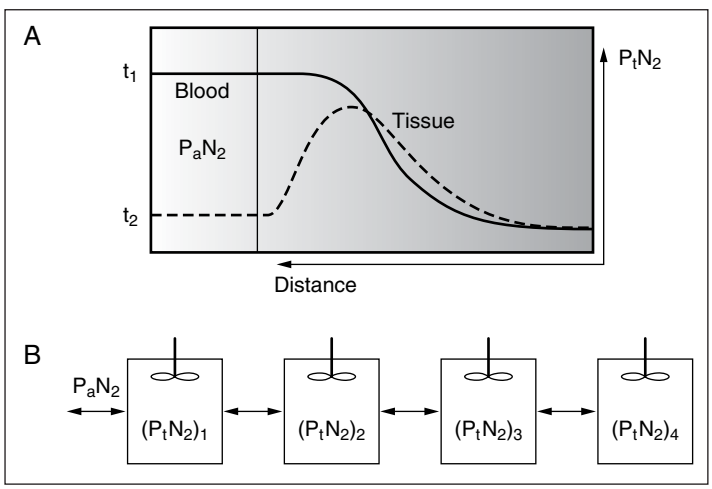

Figure 4–11. *A.* The decompression model developed by Hempleman[44] and used to compute the British Navy air decompression tables[45]. Nitrogen diffuses from the arterial blood into a slab of avascular tissue. Time, t_1, indicates the nitrogen gradient as gas diffuses into the tissue during a dive. Time, t_2, indicates the nitrogen gradient after decompression. *B.* The decompression model developed by Kidd and Stubbs[47] for a pneumatic dive computer but implemented mathematically by Nishi[48] for a digital computer and used to compute the DCIEM air decompression tables[49]. Nitrogen in the arterial blood diffuses between a series of well-stirred tissue compartments.

arterial blood on one face.[40] Figure 4–11*A* shows the nitrogen tension in this tissue slab after a time t_1 at depth during which a nitrogen gradient has developed in tissue. After decompression at time t_2 (see Fig. 4–11*A*), nitrogen nearest the blood has diffused out of tissue while nitrogen deeper in the tissue remains elevated. The British Navy Air Decompression Tables[45] were based on Hempleman's diffusion model, in which the ascent criteria were defined by the difference of the maximum nitrogen tissue tension and the barometric pressure.[46]

Another approach to inert gas exchange introduced by Kidd and Stubbs (see Fig. 4–11*B*) transferred arterial nitrogen through well-stirred tissue compartments connected in series by diffusion barriers with ascent determined by the tissue compartment having the greatest supersaturation.[47] This configuration, which could be viewed as a mathematically simpler representation of diffusion in Hempleman's tissue slab (see Fig. 4–11*A*), was first implemented as a pneumatic analog computer and tested in human trials. A later version solved the applicable equations numerically and became the basis of the well-regarded Canadian Defense and Civil Institute of Environmental Medicine (DCIEM) decompression tables.[48,49]

This discussion of decompression models was not comprehensive and was meant only to illustrate that many approaches to inert gas exchange and ascent criteria have been

successful in preventing catastrophic DCS that was so common before Haldane's pioneering work of a century ago. Tikuisis and Gerth should be consulted for a more complete review of decompression theory[51]. Success in improving decompression safety and the dictum "what works, works" are of obvious practical importance,[50] but success is by no means sufficient to prove a model is based on valid theory. Very different decompression models can produce relatively safe decompression schedules for which a variety of physical, physiologic, and pathophysiologic mechanisms can be postulated. The challenge is to design experiments that are capable of discovering the relevant mechanisms. Knowledge of these mechanisms should help to make decompression procedures even safer and more efficient.

THE OXYGEN WINDOW

The discussion so far has assumed that inert gases remain dissolved in tissue until a critical level of supersaturation is exceeded and bubbles form. The nature of inert gas exchange changes when bubbles are present, however, and oxygen metabolism and diffusion become as important as perfusion. Haldane pointed out that in vivo bubbles are absorbed because their nitrogen partial pressure is greater than the nitrogen partial pressure in the lungs.[52] This difference is the driving force for the elimination of bubbles and has been called the *partial pressure vacancy,*[53] the *inherent unsaturation,*[54] and the *oxygen window*.[55,56] The oxygen window is a consequence of the metabolic conversion of oxygen into carbon dioxide and to the nonlinear nature of the oxyhemoglobin dissociation curve.

Metabolism exchanges a relatively insoluble gas, oxygen, for carbon dioxide, which is some 21 times more soluble. This exchange is illustrated in Table 4–3 for a diver equilibrated with air at sea level. The sum of the alveolar partial pressures is 760 mm Hg (1 ata) by Dalton's law of partial pressures, while the total arterial gas tension is slightly less because of inequalities of ventilation/perfusion ratio.[57] The alveolar, arterial, and venous nitrogen tensions are equal because the diver's body is equilibrated with air. The arterial oxygen tension falls from 95 mm Hg (0.125 atm) to a venous level of 40 mm Hg (0.053 atm); the arterial carbon dioxide tension rises from 40 mm Hg (0.053 atm) to a venous level of 45 mm Hg (0.059 atm). The total gas tension in venous blood is 701 mm Hg (0.922 atm), or 59 mm Hg (0.078 atm) less than the absolute pressure. This difference in gas tension is the oxygen window.

Figure 4–12 illustrates how bubble formation affects the levels of nitrogen, oxygen, and carbon dioxide. Figure 4–12*A* represents the gases listed in Table 4–3 for an air-equilibrated diver at sea level. If the diver descends to 60 fsw (18 msw; 2.82 ata; see Fig. 4–12*B*), the alveolar oxygen and nitrogen partial pressures increase but the tissue nitrogen tension remains unchanged until the circulation transports nitrogen from the lungs. The tissue oxygen tension is metabolically controlled to a relatively constant level depending on the local metabolic rate, as discussed later. After 2 hours at 60 fsw (Fig. 4–12*C*), the 20 min tissue is nearly saturated with nitrogen.

When the diver ascends to sea level and a bubble forms (Fig. 4–12*D*), several important things happen. During a short interval, metabolism and diffusion return the partial pressures of oxygen and carbon dioxide in

Table 4–3. Alveolar partial pressures and arterial and venous gas tensions of carbon dioxide, oxygen, water, and nitrogen at 1 ata

	Partial Pressure or Tension (mm Hg)		
	Alveolar	*Arterial*	*Venous*
Carbon dioxide	40	40	45
Oxygen	104	95	40
Water vapor	46	46	46
Nitrogen	570	570	570
Total	760	751	701

the bubble to their tissue levels. Because the bubble obeys Dalton's law, the sum of all gas tensions in the bubble equals the absolute pressure of 1 ata. (Surface tension and tissue elasticity may increase the pressure in the bubble but are omitted in Figure 4–12 for clarity.) Consequently, the partial pressure of nitrogen in the bubble is greater than the nitrogen tension in tissue, which remains elevated after the dive.

The excess nitrogen in tissue can leave by two pathways: by perfusion and removal to the lungs in dissolved form or by diffusion into the bubble causing bubble growth. Eventually (Fig. 4–12*E*), all excess tissue nitrogen is transported to the lungs or has diffused into the bubble, and the nitrogen level in the bubble is greater than in the blood, causing nitrogen to diffuse slowly back into tissue from where it is removed by the circulation. The oxygen window, the difference between the nitrogen partial pressures in the bubble and lung, is small in this situation.

If the bubble should cause DCS symptoms at sea level (Fig. 4–12*F*), the diver is given 100% oxygen to breathe and there is a large increase in the oxygen window, which means the bubble resolves more rapidly than during air breathing. Therapeutic recompression to 60 fsw (18 msw; 2.82 ata) on air (Fig. 4–12*G*) compresses the bubble to 37% of its initial volume and raises its nitrogen partial pressure in accordance with Dalton's law after readjustment of the metabolic gases to tissue levels. Initially, a large nitrogen gradient between the bubble and tissue causes the bubble to shrink rapidly, but with time, nitrogen is carried to tissue by the circulation and the rate of bubble resolution decreases. This is why divers with DCS are given 100% oxygen to breathe at 60 fsw (Fig. 4–12*H*). The oxygen window is maximized by recompression on 100% oxygen. Nitrogen elimination from tissue increases, and the bubble resolves rapidly by direct removal of nitrogen from bubble to tissue to lungs. This is the physiologic rationale for using oxygen and pressure to treat bubbles that cause DCS. Oxygen has additional beneficial effects on physical or biochemical damage caused by the bubbles.

Figure 4–12 assumed that the tissue oxygen tension and oxygen partial pressure in the bubble remained constant at all levels of inspired oxygen (PIO_2). This is not always true. At high PIO_2, the metabolic requirements of tissue may be met entirely by dissolved oxygen and the venous oxygen tension will rise. This is illustrated on the hemoglobin dissociation curve in Figure 4–13 with data

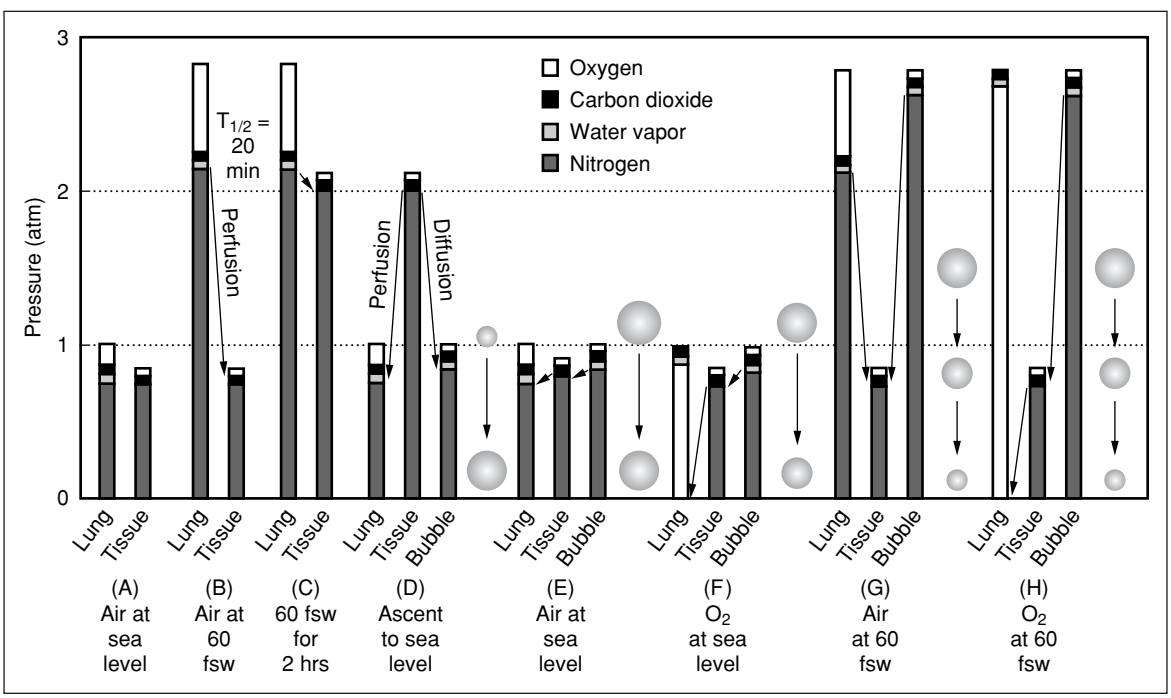

Figure 4–12. The effects of metabolism and pressure on gases in the body and on bubble absorption (see discussion in text).

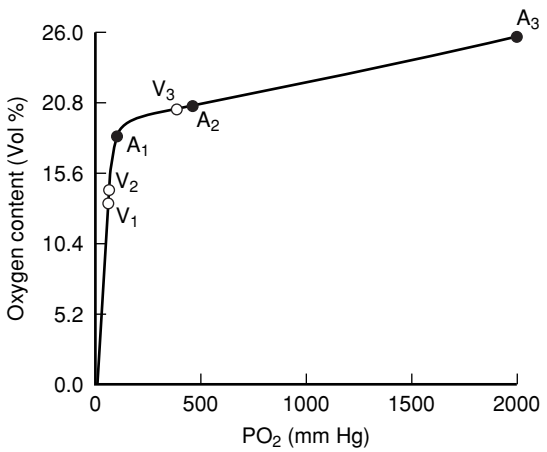

Figure 4–13. The total blood oxygen content in vol% (ml/O_2/100 ml blood) as a function of blood oxygen tension.[59] Total content is the physically dissolved oxygen plus the oxygen chemically bound to hemoglobin. The points marked A_1, A_2, A_3, and V_1, V_2, V_3 are approximate arterial and venous oxygen tensions during air breathing at sea level, during air breathing at 3.5 ata, and during oxygen breathing at 3.5 ata. The oxygen extraction from blood is taken as 5 vol%.

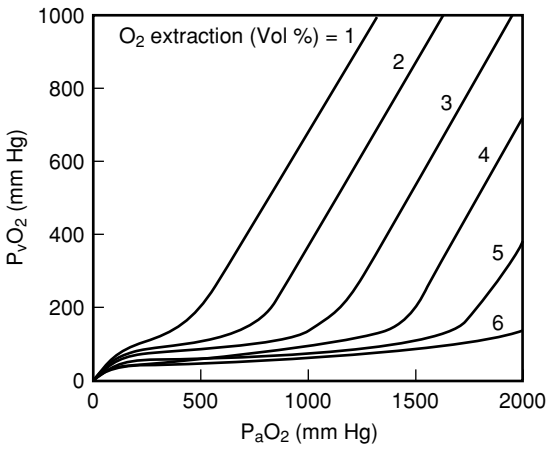

Figure 4–14. The effect of oxygen extraction on venous oxygen tension (P_vO_2) as a function of arterial oxygen tension (P_aO_2).[59] At higher oxygen extractions, P_vO_2 remains relatively constant as Pa_{O_2} rises. In tissues with lower extraction, P_vO_2 rises steeply at high $P_{a_{O_2}}$. This increase begins sooner at lower extractions.

collected by Lambertsen and coworkers.[58] Under normal conditions, blood transports most of its oxygen bound chemically to hemoglobin, and only a small fraction is dissolved. Hemoglobin is nearly 100% saturated with oxygen in the arterial blood of a diver breathing air at sea level (point A_1). As the arterial blood passes through tissue, 5 vol% of oxygen are removed and converted to carbon dioxide. This causes the venous oxygen tension (point V_1) to fall to 46 mm Hg (0.061 atm).

Now consider divers breathing air at 3.5 ata (83 fsw; 24.9 msw). Their alveolar oxygen partial pressure is 504 mm Hg (0.663 atm), but the arterial tension (point A_2) is only about 450 mm Hg (0.592 atm) as a result of ventilation-perfusion inequalities.[58] When 5 vol% of oxygen are extracted by tissue, the venous tension (point V_2) falls to 53 mm Hg (0.070 atm). If divers switch to 100% oxygen at 3.5 ata, their alveolar partial pressure rises to 2570 mm Hg (3.38 atm) but ventilation-perfusion inequalities reduce the oxygen tension in the arterial blood (point A_3) to approximately 2000 mm Hg (2.63 atm). The venous oxygen tension (point V_3), however, rises to 380 mm Hg (0.5 atm), far above the previous venous values. This abnormally high venous oxygen tension occurs because the metabolic requirements of tissue are met entirely by dissolved oxygen. The venous hemoglobin remains saturated and on the flat rather than on the steep part of the hemoglobin dissociation curve. The extra oxygen in venous blood and tissue participates in bubble formation and growth just as does inert gas.

Figure 4–13 assumed that tissues extract 5 vol% of oxygen from the arterial blood. In actuality, tissue oxygen extraction ranges from 1.3 to 10 vol%, depending on perfusion and metabolic rate.[60] The effect of oxygen extraction on venous oxygen tension as a function of arterial oxygen tension is shown in Figure 4–14. The lowest curve, for an extraction of 6 vol%, shows that the venous tension rises gradually at arterial tensions of up to 2000 mm Hg (2.632 atm). For extractions of 5 vol% and less, the venous tension increases steeply. At the lowest extractions, the venous oxygen tension can contribute more than 760 mm Hg (1 atm) to the dissolved gas tension.

In experiments with subcutaneous gas pockets in rats, Van Liew[61] demonstrated that

$$\text{Oxygen window } (O_2W) = P_AO_2 - P_bO_2 + P_ACO_2 - P_bCO_2 \qquad (4\text{–}1)$$

where the letters *A* and *b* refer to the alveolar and bubble partial pressures, and the three terms on the right side of equation are essentially constant at low inspired oxygen partial pressures (P_IO_2). If P_bO_2 and P_bCO_2 are approximated by their venous values from Table 4–3 and

$$P_AO_2 = P_IO_2 - P_ACO_2 - PH_2O \qquad (4\text{–}2)$$

then

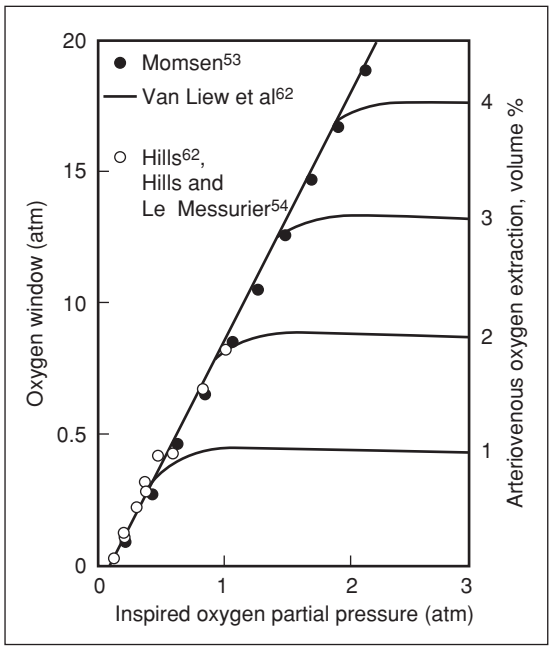

Figure 4–15. The oxygen window as a function of inspired oxygen partial pressure. The values from Momsen[53] are predictions, while the values from Hills[62] and Hills and LeMessurier[54] are measurements. The oxygen window in tissue does not increase indefinitely but reaches a maximum value, which is determined by the arteriovenous oxygen extraction. (Redrawn from Van Liew.[61])

$$\text{Oxygen window} = P_IO_2 - 0.172 \text{ atm} \quad (4\text{–}3)$$

The venous oxygen tension does not remain constant, however, but rises as illustrated in Figure 4–14. As a result, the oxygen window reaches a plateau that is determined by the oxygen extraction of the tissue. Figure 4–15 shows the effect of oxygen extraction on the oxygen window according to estimates by Van Liew[61] with independent estimates by Momsen[53] as well as measurements in rabbits by Hills and LeMessurier.[54] Skin, for example, has an oxygen extraction of about 2.5 vol%,[60] and the oxygen window reaches its maximum value at a P_IO_2 of about 1.3 atm. In most tissues, the oxygen extraction is 5 vol% or greater, so the oxygen window would not appear to achieve its maximum plateau until a P_IO_2 of about 3 atm.

OXYGEN BENDS

Increased tissue oxygen tension contributes to supersaturation, limits the oxygen window, and might raise DCS risk. Weathersby and colleagues tested this hypothesis with oxygen partial pressures of 0.21 atm and 1.3 atm after human dives with the same nitrogen partial pressures but found no significant difference in DCS.[63] In experiments with goats, Donald compared oxygen partial pressures of 0.53 atm and 3.53 atm in similar experiments and saw no DCS at 0.53 atm but serious symptoms in six of seven animals at 3.53 atm.[64] Donald called this effect *oxygen bends* to indicate that oxygen can cause DCS at a partial pressure of 3.53 atm. These limited data are consistent with the conclusion that a PIo_2 of up to 1.3 atm will not increase DCS risk but a PIo_2 of 3.53 atm will do so. If humans and goats are similar, Figure 4–15 suggests that the relevant tissues for DCS have an oxygen extraction of between 3 and 5 vol%.

EFFECTS OF BUBBLES ON INERT GAS EXCHANGE

Bubbles reduce the rate at which nitrogen is eliminated from tissue because nitrogen in a bubble must diffuse back into tissue before it can be transported by the circulation to the lungs (see Fig. 4–12*E*). Thus, the elimination of nitrogen from a bubble is slower than the elimination of nitrogen dissolved in tissue. This has been demonstrated in both animal[65–67] and human studies.[68–70]

Most decompression models assume that bubbles do not form, but when bubbles are present, diffusion between bubble and tissue cannot be ignored. Diffusion is a simple physical process but difficult to describe mathematically. Figure 4–16 is a schematic representation of diffusion from a bubble filled with either oxygen, nitrogen, or helium into an adjacent perfusion-limited tissue.[71] Because oxygen is metabolized in tissue, its tension falls rapidly with increasing distance from the bubble. Helium and nitrogen, on the other hand, are metabolically inert and are removed only by perfusion, so their concentration gradients extend deeper into tissue. Helium penetrates further into tissue than nitrogen does because its diffusivity is greater.

The diffusion process is often simplified to make it more manageable mathematically. Figure 4–17 shows three representations of diffusion in decreasing order of complexity. Figure 4–17*A* illustrates the situation depicted in Figure 4–16 in which diffusion is a continuous process throughout tissue.

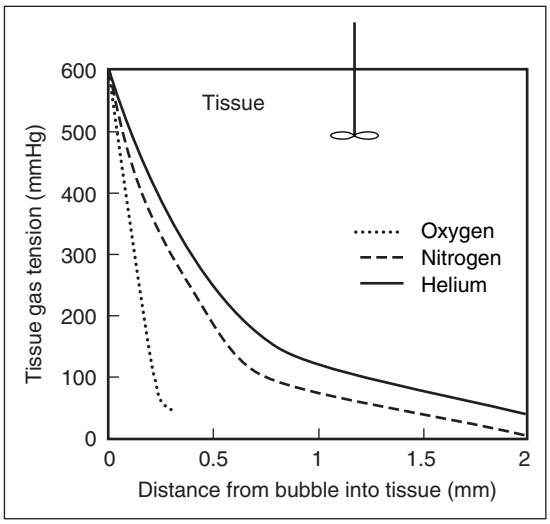

Figure 4–16. Tissue tension gradients around a gas cavity (redrawn from Hlastala[71]). The oxygen gradient is steepest because oxygen is removed both by the circulation and by tissue metabolism, whereas nitrogen and helium are removed only by the circulation. The helium gradient extends further into tissue than the nitrogen gradient because helium diffuses faster than nitrogen.

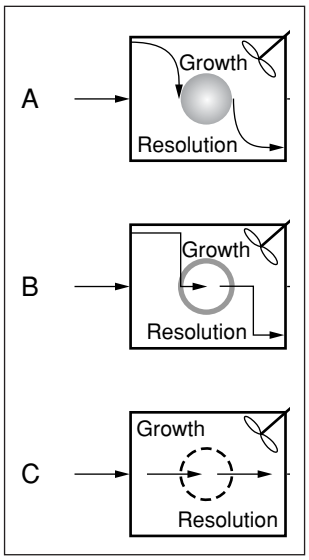

Figure 4–17. Representations of diffusion for mathematical modeling. (*A*) Bulk diffusion through tissue as in Figure 4-16. (*B*) All diffusion resistance at a barrier around the bubble. (*C*) No diffusion resistance and the tissue and bubble are in equilibrium.

Diffusion into and out of the bubble is represented by curvilinear concentration gradients indicating bubble growth or resolution. The difference between the inert gas partial pressure in the bubble and the dissolved inert gas tension in tissue is a consequence of the oxygen window.

In Figure 4–17*B*, the entire tissue region around the bubble is considered to be well-stirred, and all diffusion resistance is concentrated in a barrier around the bubble. This is the basis of Gernhardt's decompression algorithm and commercial diving decompression schedules.[72,73]

A further mathematical simplification in Figure 4–17*C* omits the diffusion barrier around the bubble such that the inert gas partial pressure in the bubble and the dissolved inert gas tension in tissue are equal. Hills offered the first analysis of this problem,[62] which was later refined by Hennessy and Hempleman.[74] In this circumstance of diffusion equilibrium between bubble and tissue, when a nitrogen molecule enters tissue from the arterial blood, another molecule moves from the tissue to the bubble. The reverse is also true, and the bubble shrinks by one molecule when there is a net loss of one inert gas molecule from tissue to venous blood.

As illustrated in Figure 4–18*A*, suppose a bubble forms in a diffusion-equilibrium tissue (see Fig. 4–17*C*) upon decompression from 60 fsw (18 msw; 2.8 ata) to sea level (1 ata). Because the bubble and tissue are in diffusion equilibrium, all supersaturated nitrogen dissolved in tissue immediately diffuses into the bubble. Figure 4–18*B* shows how the nitrogen tension in tissue changes with time when a diffusion-equilibrium bubble is (or is not) present. If no bubble forms, nitrogen uptake and elimination follow the exponential kinetics expected of a well-stirred tissue, but the presence of a bubble causes the tissue nitrogen tension to fall to a level defined by the oxygen window (equation 4–3) and to remain constant as long as the bubble is present. Although the nitrogen tension in tissue and the partial pressure in the bubble are equal and constant, perfusion removes nitrogen from tissue and the bubble volume resolves at a linear rate (Fig. 4–18*C*). When the bubble is gone, nitrogen kinetics revert to an exponential function. Vann described the mathematics of inert gas exchange in a diffusion-equilibrium bubble.[75]

Diffusion-equilibrium bubbles are the basis of Thalmann's *exponential-linear (E-L)* decompression model.[76,77] The nitrogen

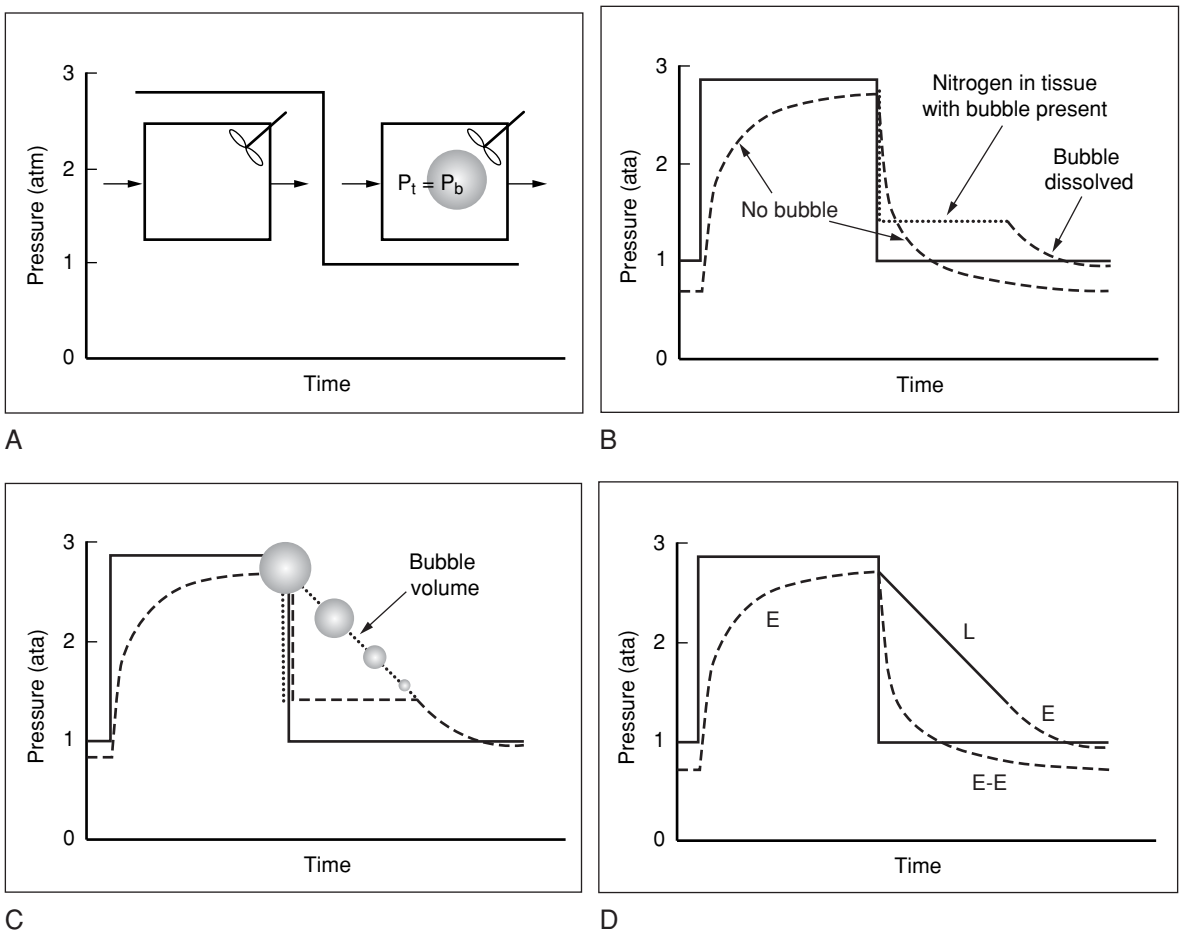

Figure 4–18. Nitrogen exchange in response to bubble formation in a diffusion equilibrium tissue after decompression from 60 fsw (18 msw; 2.82 ata) to sea level. *A.* Formation of a diffusion equilibrium bubble upon decompression. *B.* While a diffusion equilibrium bubble is present, the tissue nitrogen tension remains constant and equal to the nitrogen partial pressure in the bubble. *C.* The bubble volume decreases linearly until it dissolves. *D.* The exponential-linear ("E-L") model in which the bubble is replaced by an "equivalent" dissolved gas tension that washes out at a linear rate so long as a bubble is present.[76,77] When the bubble dissolves, washout becomes exponential.

kinetics of the E-L model are illustrated in Figure 4–18*D*. The E-L model uses a conventional M-value matrix as in Table 4–1 (called *V-value* to indicate E-L kinetics) that allows critical levels of supersaturation to exist in the tissues. (This excess supersaturation might be interpreted as surface tension and tissue elasticity.) When a critical supersaturation is exceeded, however, the nitrogen exchange kinetics change from exponential to linear, which is equivalent to the supposition that a bubble has formed in that tissue. After the supersaturation has resolved, the kinetics return to exponential, which is equivalent to the supposition that the bubble has dissolved. The effect of bubble formation is to reduce the rate at which nitrogen is eliminated from tissue as indicated earlier. The E-L model provided a biophysical explanation for the asymmetry between nitrogen uptake and elimination that others had observed experimentally and was the basis for the U.S. Navy 0.7 atm oxygen partial pressure nitrogen-oxygen and helium-oxygen decompression tables.[78,79] The E-L model has been implemented in the recently approved U.S. Navy dive computer.[80]

Return of the Deep Stop.

For air diving, a deep decompression stop might be defined as a first stop that is deeper than for a corresponding dive on the U.S. Navy tables[9], the *de facto* air diving standard. The deep decompression stops of the Haldane tables (Figure 4–8) went out of fashion as decompression theory evolved[41] but never disappeared entirely, and deep stops have returned with anecdotal reports of their effectiveness for Australian pearl divers[81] and for sport and technical divers.[50,82,83] Hills' theory of "zero supersaturation" provided an explanation for the beneficial effect of deep stops: supersaturation caused immediate bubble formation, which could be avoided by deep decompression stops.[81] Controlling the formation and growth of bubbles has been the rationale of modern decompression models[72,73,84–86] that tend to have deeper decompression stops than the U.S. Navy tables. After helium-oxygen diving, Momsen[53] and Cabarrou[87] reported that unexpectedly deep decompression stops were required to limit DCS risk. According to Momsen, deep stops accommodated the "initial out-rush" of helium that was exchanged more rapidly than nitrogen (Figure 7–16).[88] Haldanian decompression models, that are based on dissolved inert gas exchange, provide deep stops if low supersaturation ratios are used in the fast tissues (Figure 4–8), but deeper stops also cause slower tissues to absorb additional inert gas (Figures 4–6 and 4–7) and, theoretically, prolong the shallower stages. Bubble models, on the other hand, purport to require less shallow decompression because deeper stops avoid bubbles that become problematic at shallow depths. Resolution of this question awaits a better understanding of inert gas exchange, and proof of the effectiveness of deep decompression stops awaits the collection of reliable depth-time and medical outcome data.

PHYSICS OF BUBBLE FORMATION AND STABILITY

A bubble is a volume of undissolved gas irrespective of shape. The tendency for bubbles to form increases with the supersaturation, the difference between the absolute pressure and the sum of the dissolved gas tensions plus the water vapor pressure (equation 4–4).

$$\text{Supersaturation} = (PO_2 + PCO_2 + PH_2O + PN_2 + PH_e + \ldots.) - P_B \qquad (4\text{–}4)$$

Equation 4–4 indicates that supersaturation can result from:

- Excess dissolved gas and water vapor (P_{H2O})
- A reduction in local pressure (P_B)

If no dissolved gas is present, very pure water will not form (water vapor) bubbles until P_B is reduced to about –1400 atm.[89] This is known as *de novo* ("from nothing") bubble formation and represents the tensile strength of water. When dissolved helium is present, pure water can withstand a supersaturation of only 240 atm, and with dissolved nitrogen, the supersaturation threshold is only 120 atm.[90] In most aqueous solutions, bubble formation occurs at less than several atmospheres of gaseous supersaturation. Harvey proposed that bubble formation at low supersaturations indicated the presence of preexisting gas cavities called *gas nuclei*.[91]

Decreases in the local absolute pressure (P_B) have a number of mechanical causes. One mechanism involves a flowing liquid that accelerates upon entering a constriction or passing an obstruction. The resulting bubble formation is known as *Reynolds* or *Bernoulli cavitation*.[92] In another hydrodynamic mechanism, closely opposed surfaces are pulled apart in a viscous liquid. Viscosity retards the flow of liquid into the widening gap and decreases the pressure between the surfaces causing them to stick together by *viscous adhesion*. (This is why tape sticks to surfaces.) The resulting supersaturation can exceed 1000 atm,[93] leading to bubble formation in a process known as *tribonucleation*.[94] Figure 4–19 shows tribonucleation between two rapidly separating surfaces.[95]

Spherical bubbles are inherently unstable and have short lifetimes because surface tension (γ) raises the internal pressure in the bubble (P_b) as described by LaPlace's law (equation 4–5; Fig. 4–20*A*).

$$P_b = P_B + 2 \times \gamma / r \qquad (4\text{–}5)$$

If the surrounding liquid is in diffusion equilibrium with the ambient atmosphere, the bubble pressure exceeds the dissolved gas tension and the bubble shrinks because of

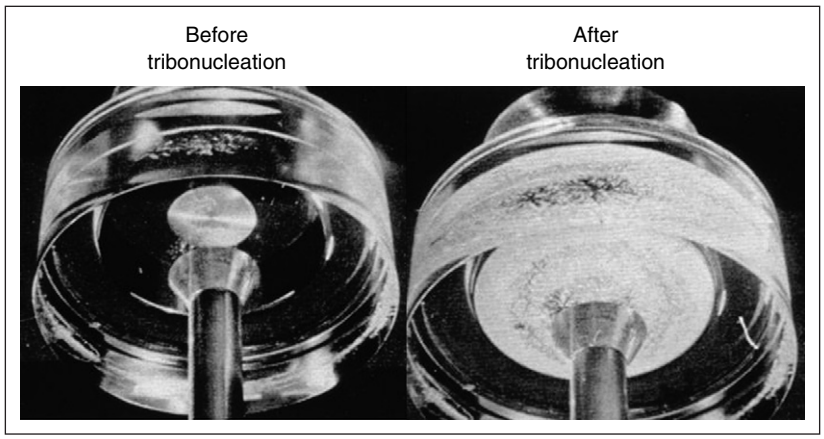

Figure 4–19. *In vitro* bubble formation due to tribonucleation[95]. Before tribonucleation, two surfaces separated by a viscous liquid are pressed close together. When the surfaces are pulled rapidly apart, a large negative pressure develops between them causing the liquid to fracture into vaporous bubbles.

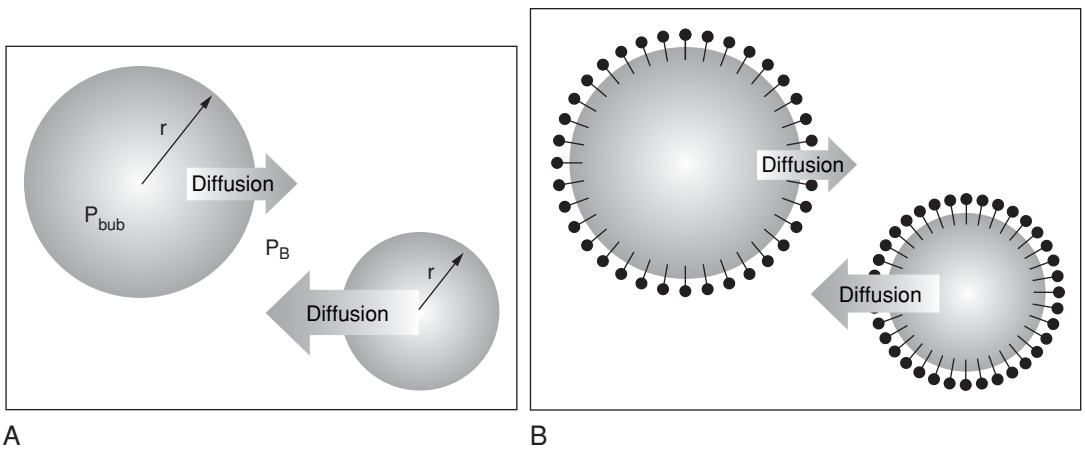

Figure 4–20. *A.* As defined by Laplace's law (equation 4.5), the surface tension at a convex gas-liquid interface causes a spherical bubble to dissolve by the outward diffusion of gas. *B.* Surface tension and diffusion are reduced by surface-active molecules at the gas-liquid interface (equation 4.6). This stabilizes the bubble and increases its lifetime. (P_{bub}, gas pressure in the bubble; P_B, absolute pressure; γ, surface tension; π, surface pressure; r, bubble radius)

outward diffusion of gas. Because the excess pressure due to surface tension increases with decreasing radius (equation 4–5), the bubble shrinks at an ever-increasing rate until finally dissolving. Adding surfactant to the water reduces the effect of surface tension (equation 4–6) by an amount known as the surface pressure (π), a characteristic of each surfactant (Fig. 4–20*B*). The surface tension of pure water is 72 dynes/cm at 37°C. The surface pressures of common surfactants are on the order of 35 to 40 dynes/cm[96] but can be large enough to reduce the effect of surface tension to less than 5 dynes/cm for pulmonary surfactant.[97]

$$P_{bubble} = P_B + 2(\gamma - \pi)/r \qquad (4–6)$$

Bubbles can act as gas nuclei, but their lifetimes are relatively short unless they are stabilized against the effects of surface tension. Harvey proposed that in vitro gas nuclei would be stabilized against the dissolving pressure of surface tension in hydrophobic crevices,[92] but no such crevices have been identified in vivo. Yount suggested that the surface pressure of surfactants might stabilize spherical bubbles by counteracting surface tension as in Figure 4–20*B*,[98,99] but no such surfactants have been identified. Moreover, bubble formation experiments that varied the surfactant concentration found results contrary to this theory.[100]

Epstein and Plesset derived equations for calculating the solution time of spherical bubbles as a function of surface tension and dissolved gas partial pressure.[101] Table 4–4

Table 4–4. Lifetimes of spherical bubbles as a function of diameter, surface tension (γ, dyne/cm), and the oxygen window according to the model of Epstein and Plesset[101]

O_2W		Bubble Lifetime (s, m, h)					
		O_2W = 0.0 atm		*O_2W = 0.08 atm*		*O_2W = 0.83 atm*	
μ	mm	γ = 0	γ = 72	γ = 0	γ = 72	γ = 0	γ = 72
10	0.01	∞	5 s	6 s	1 s	1 s	0 s
50	0.05	∞	1 m	2 m	1 m	14 s	13 s
100	0.1	∞	23 m	10 m	6 m	1 m	1 m
250	0.25	∞	5 h	1 h	1 h	6 m	6 m
500	0.5	∞	40 h	4 h	4 h	23 m	23 m
1000	1	∞	322 h	16 h	15 h	92 m	91 m

O_2W, oxygen window.

shows the estimated lifetimes for bubbles with diameters of 10 to 1000 μm (columns 1 and 2) for pure water (γ = 72 dynes/cm) and for water in which a hypothetical surfactant has eliminated the effect of surface tension (γ – π = 0). The Epstein-Plesset equations were solved for dissolved gas tensions representing:

- In vitro bubbles in equilibrium with a 1 ata environment
- An air-breathing animal at 1 ata
- An oxygen-breathing animal at 1 ata after elimination of all tissue nitrogen

As indicated by Figure 4–12, the oxygen window provides the driving force for eliminating bubbles from tissue, and the magnitude of the oxygen window depends on the oxygen partial pressure in the inspired gas. For in vitro bubbles, the dissolved gas tension was equivalent to an oxygen window (O_2W) of 0.0 atm. During air breathing (see Fig. 4–12*E*), the dissolved nitrogen tension was equivalent to an oxygen window of 0.08 atm and 0.83 atm when breathing 100% oxygen (see Fig. 4–12*F*). Columns 3 and 4 of Table 4–4 show the lifetimes of in vitro bubbles. Bubbles unaffected by surface tension persist indefinitely (column 3). With a surface tension of 72 dynes/cm (column 4), large bubbles persist for many hours but small bubbles last only minutes to seconds before dissolving. Surfactants are important determinants of the lifetimes of bubbles in nonliving systems.

The situation is quite different in living tissue, wherein the oxygen window (O_2W) causes the inert gas partial pressure in the bubble to exceed the dissolved inert gas tension in tissue. Columns 5 and 6 of Table 4–4 show the estimated bubble lifetimes for an air-breathing animal (O_2W = 0.08 atm), and columns 7 and 8 show bubble lifetimes for an animal breathing 100% oxygen (O_2W = 0.83 atm). The oxygen window exerts such a powerful effect on bubble resolution that even without surface tension, small bubbles persist for only seconds to minutes and larger bubbles for only minutes to hours. Because diffusion was the only transport mechanism involved in these estimates, the medium surrounding the bubble did not lose gas due to perfusion as in the tissue model of Figure 4–16. If the Epstein-Plesset model had included perfusion as well as diffusion, the estimated bubble persistence times would be shorter than indicated in Table 4–4.

Spherical bubbles stabilized by surfactants can persist as gas nuclei for long periods in nonliving systems, but the estimates of bubble lifetimes in Table 4–4 suggest that this would not be so in living tissue, which is undersaturated because of the oxygen window. The message of Table 4–4 is that surface tension effects are less important than the effect of the oxygen window on in vivo bubble resolution. In vivo bubbles would be rapidly eliminated by the oxygen window even if surface tension were absent.

NONINVASIVE METHODS FOR DETECTING BUBBLES

Much of what is known about the physics of bubbles comes from in vitro studies of visible bubbles. Except for a few transparent marine animals, knowledge of bubbles in living systems has relied on a few low-resolution, noninvasive imaging or detection methods.

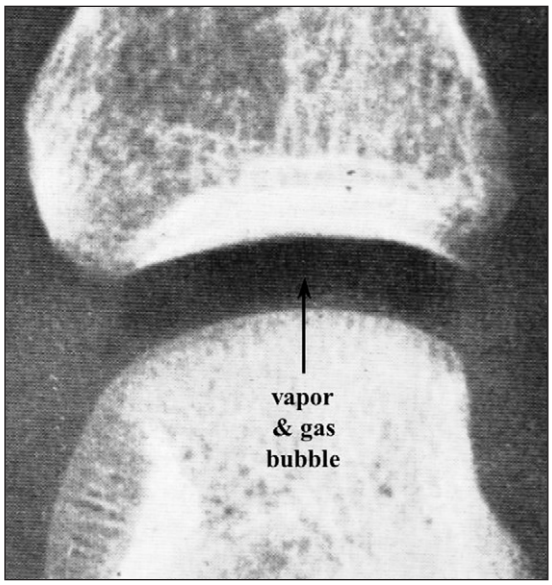

Figure 4–21. Radiograph showing bubble formation during "knuckle-cracking"[103].

Radiography

Bubbles were first detected in the human body by radiography in 1910 and are described extensively in the clinical literature.[102] Known today as *vacuum phenomena*, these bubbles often appear in synovial joints placed under tension and form as a result of viscous adhesion (see section Physics of Bubble Formation and Stability). Vacuum phenomena that are filled with water vapor collapse noisily as in the cracking knuckle joint of Figure 4–21[93,103]; those filled with gas remain stable, such as the bubble in the spinal canal of a 52-year-old man with a history of chronic low back pain (Fig. 4–22).[104] Gas collections in the spinal canal can persist for at least 10 weeks[104] and appear to be associated with vacuum phenomena in intervertebral discs or apophyseal joints.[105]

Although vacuum phenomena are usually not associated with symptoms, this is not necessarily the case for bubbles detected after decompression. Figure 4–23 is a radiograph of a large bubble behind the knee of an experimental subject at an altitude in excess of 30,000 feet (9144 m; Dr. A. A. Pilmanis, personal communication). U.S. Army Air Force aircrew members were routinely exposed to such altitudes during World War II. Many experienced pain (as illustrated in Fig. 4–23 by the notation "muscle dissection, intense pain") associated with the vacuum phenomenon behind the knee. Thomas and

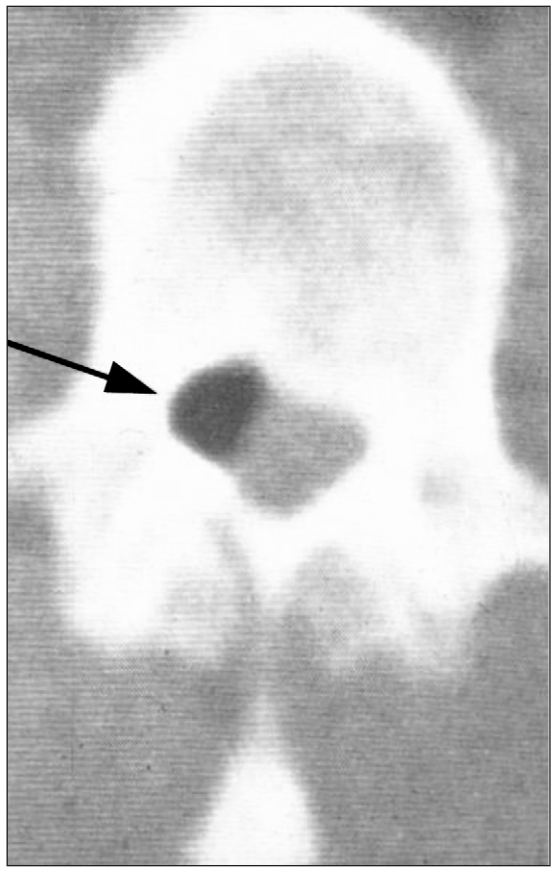

Figure 4–22. Radiograph showing bubble in the spinal canal of a 52-year-old man with a history of chronic low back pain[104].

Williams obtained radiographs of both knees of 35 subjects at altitude when pain occurred in one knee and found that all subjects had gas in the knee joints: 62% with pain had bubbles behind the knee, and 76% with pain had streaking along tendons and facial planes.[106,107] These examples do not prove a causal association of vacuum phenomena and DCS, but supersaturation in the vicinity of a vacuum phenomenon would promote bubble growth by diffusion and the consequences of decompressing a bubble in the spinal canal can be postulated (see Fig. 4–22).

Doppler Ultrasonography

The most common method for detecting vascular bubbles, Doppler ultrasonography, operates on the principle that moving bubbles change the frequency of reflected sound waves. The frequency shift is converted electronically to an audible signal that a trained operator can interpret as gas emboli

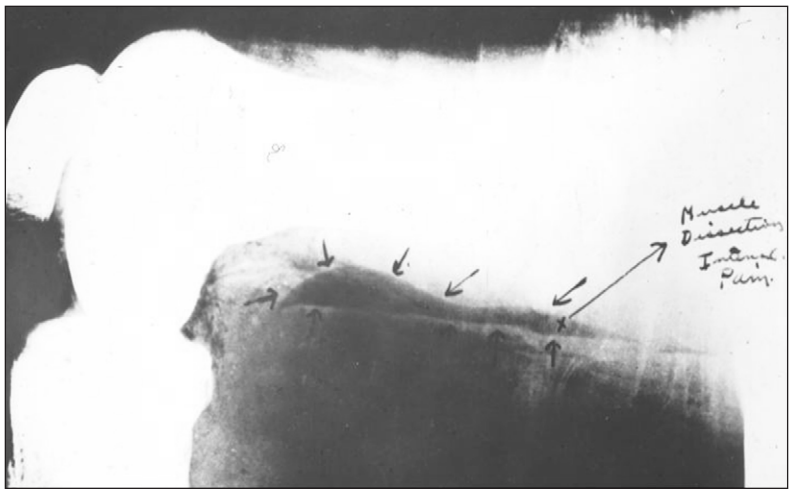

Figure 4–23. Radiograph of the leg of a U.S. Army Air Force volunteer at an altitude in excess of 30,000 ft (9,144 m) (Courtesy of Dr. A. A. Pilmanis). A large bubble is visible behind the knee with the notation, "Muscle Dissection, Intense Pain."

Figure 4–24. Doppler bubble monitor showing transmitting and receiving probe.

(Fig. 4–24). The interpretation is subjective and commonly scored according to the five-point Spencer scale[108] (Table 4–5) or the 16-point Kisman-Masurel scale, which collapses into the Spencer scale.[109] Doppler bubble signal scales are nonlinear and cannot be averaged unless linearized by one of several suggested transformations.[109,110] Typical Doppler monitoring sites are the precordium, the pulmonary artery, the subclavian or femoral veins, and the cerebral arteries.

Doppler bubble detection was introduced into a diving world dominated by Haldane decompression theory.[111] Because the theory held that DCS did not occur until bubbles formed, Doppler seemed to hold the promise of bubble detection as an early warning of DCS. When Doppler-detected venous gas emboli (VGE) were found to be common in the absence of DCS and DCS occasionally occurred with no detectable VGE, some workers rejected Doppler as too imprecise to

Table 4–5. Doppler bubble signal scoring system according to Spencer[108]

Bubble Grade	Definition
0	No bubble signals
1	Occasional signal
2	Signals in less than half the cardiac cycles
3	Signals in all cardiac cycles
4	Signals override cardiac signals

Table 4–6. Relationship of Doppler bubble scores and decompression sickness

Bubble Grade	Air Diving* (% DCS)	30,000 ft Altitude† (% DCS)
0	0	10
1	1	11
2	1	50
3	6	60
4	10	78

* 35 DCS in 1761 dives[113]
† 64 DCS in 275 flights[114]

be of value.[112] Table 4–6 indicates that Doppler scores and DCS were significantly associated in diving[113] and high-altitude populations,[114] however, and the Canadians used Doppler extensively in developing the DCIEM decompression tables.[49,115,116]

Doppler has demonstrated VGE in humans after decompression to an altitude of only 12,000 ft (3658 m)[117] and after ascent from a 12-hour dive to only 12 fsw (3.6 msw).[118] These are pressure changes of 0.4 atm or supersaturation ratios of 1.6:1 and 1.4:1, respectively. Doppler-detected VGE are also common during routine recreational diving in the absence of DCS (see Chapter 7, Figs. 7–17, 7–18, 7–24).[119] VGE are certainly abnormal, but further study is needed to determine whether they can be pathologic (see the discussion "Possible Roles of Venous Gas Emboli in Neurologic Decompression Sickness" in Chapter 7).

Echocardiography

The echocardiograph is another ultrasonic instrument used in decompression research, but one used less frequently than Doppler because of the high cost (although less expensive portable systems are now available). Two-dimensional echocardiography is based on the same principles as computed tomography but uses ultrasound instead of x-rays. Bubble images are relatively easy to locate within the four chambers of the heart (see Chapter 25, Fig. 25–9).

The principal use of echocardiography in decompression research has been to investigate the question of whether a patent foramen ovale (PFO) predisposes to neurologic DCS. The hypothesis is that the PFO provides an anatomic pathway through the right side of the heart by which VGE may bypass the filtering action of the lungs and reach the brain or spinal cord through the arterial circulation (see Chapter 8). To test for the presence of a PFO, a mixture of saline and microbubbles is injected into a peripheral vein. The appearance of bubbles in the left side of the heart is evidence of a functional PFO. Several workers have found that PFO was more prevalent in divers who had suffered neurologic DCS than in controls.

The U.S. Air Force routinely uses echocardiography during experimental altitude exposures to search for arterial bubbles in the left side of the heart.[108] If any are detected, the exposure is immediately terminated because of the potential risk of cerebral arterial gas embolism. To date, left-ventricular bubbles have been observed in 8 of 2587 subject exposures. All 8 had grade 4 VGE; of these, 7 experienced limb-pain DCS[120] (Dr. J. Webb, personal communication). Of 4 who were evaluated for PFO by saline bubble contrast injection, 3 had a PFO and 1 had a functionally similar abnormality known as a sinus venosus defect. The Air Force experience suggested that, for altitude exposure at least, arterial bubbles were rare, and those that did occur did not predispose to cerebral DCS during a short interval before recompression. Neurologic DCS at altitude is unusual compared with diving, however, possibly because oxygen breathing before decompression reduces or eliminates nitrogen supersaturation of the brain and spinal cord.

CONCLUSIONS

Stage decompression, introduced in 1908 by John Scott Haldane, was the most significant achievement of the 20th century in reducing severe or fatal decompression sickness and was the first model of decompression to be

based on a physiologic explanation of DCS. Later workers refined Haldane's method empirically and further improved decompression safety and efficiency. The effect of bubble formation on retarding inert gas elimination was not appreciated until the latter half of the century, however, and has only recently been incorporated into decompression models. To a large extent, this was because of limited techniques for detecting bubbles and measuring inert gas exchange. Although these techniques have improved, the situation remains less than satisfactory.

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CHAPTER 5 Breath-Hold Diving

Massimo Ferrigno

Since the beginning of human history, men and women must have used breath-hold diving to gather seafood or recover things accidentally lost underwater. There are several historical accounts of divers used in salvage and military operations in the ancient world and throughout the centuries,[1] including a detailed report describing breath-hold dives down to 80 m (about 262 ft) performed by a Greek sponge diver to recover the lost anchor of an Italian battleship in 1913.[2] Also very old is the history of diving women (called Ama in Japan), who started gathering food underwater around 2000 years ago along the coasts of Japan and Korea.[3] A few thousands of these women are still breath-hold diving for the same purpose with basically the same techniques, except for the introduction of wet suits in the 1970s. Typically, the Ama perform a large number of breath-hold dives, either assisted or unassisted (Table 5–1), of about a minute in duration[4] and generally shallower than 25 m (82 ft).[3] Another group of divers that perform many repetitive dives consists of competitive spear-fishermen, who may reach 30 to 40 m of depth while holding their breath for 1 to 2 min (L. Magno, personal communication). Repetitive, but shorter and shallower dives, are also performed by underwater hockey players. In the United States, breath-hold diving probably started in Southern California in the early 1930s with the famous "San Diego Bottom Scratchers" and from there it spread to the rest of the country.

Since the mid-twentieth century, deep breath-hold diving has become a new sport worldwide, from its beginning in 1949 when the Italian Raimondo Bucher made and won a wager: holding his breath, he was able to snatch a waterproof envelope containing 20,000 Italian Lira from the hands of an astonished hard-hat diver at 30 m of depth (about 98 ft).[5] Since then, new depth records have been set almost every year until the present world record established by Tanya Streeter, who reached the depth of 160 m (almost 525 ft) in 2002 (Fig. 5–1), during an assisted breath-hold dive lasting 3 min 26 sec, in which she was pulled down by a weight and then pulled back to the surface by an inflated balloon (see Table 5–1).

Breath-hold diving includes three different conditions: breath holding (also known as apnea), immersion (frequently in cool or cold water), and exposure to increased pressure under water. However, most physiologic studies of breath-hold diving have involved subjects holding their breath at the surface, with their face exposed to cold water (called "simulated diving"); this means that some of the results of these laboratory studies may not be applicable to real breath-hold diving.

EFFECTS OF IMMERSION

Because immersion is integral to breath-hold diving, a brief description of its effects on the respiratory and cardiovascular systems of the diver follows.[6] Actually, breath-hold divers spend more time snorkeling at the surface than diving underwater. When a diver is immersed in water up to the neck, the body is exposed to the atmospheric pressure plus the hydrostatic pressure, the latter being proportional to depth, while the lungs are exposed to atmospheric pressure (Fig. 5–2). In the vertical head-out position, the average pressure resulting from the different hydrostatic forces on the diver's chest is about 20 cm H_2O; in the horizontal position, as during snorkeling, this pressure is probably less than 10 cm H_2O.[7] These pressures represent the degrees of negative pressure breathing to which the diver is exposed at the surface. The maximal inspiratory pressure a diver can generate is about 150 cm H_2O.[8] This represents the theoretical maximal limit for snorkeling; however, most snorkels are shorter than 30 cm. Trying to inspire from a very long snorkel may lead to severe cardiac dilatation and failure, as happened to Stigler, who unsuccessfully tried to

Table 5-1. Types of breath-hold diving

Type	Technique	Used By
Unassisted (free) diving	Swimming during descent and ascent	Competitive spear fishermen, record divers, Ama-Cachido, underwater hockey players
Assisted-descent diving	Pulled by weight during descent, swimming during ascent	Pearl divers Sponge divers Record divers
Assisted diving	Pulled by a weight during descent; during ascent, lifted by air-balloon or by surface assistant with a rope; electrically powered scooter used during descent and ascent	Ama-Funado Spear fishermen Record divers

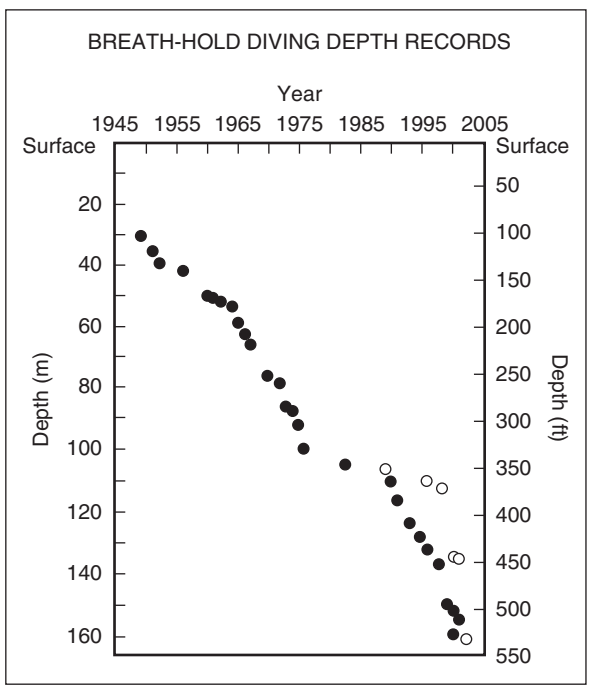

Figure 5–1. Depth records (mostly with assisted techniques—see Table 5–1) established by elite breath-hold divers since 1949, when this type of competition started (see text). The information presented in this figure is not complete and is derived from nonscientific sources. *Circles* represent records set by female divers. (Modified from Ferrigno M, Lundgren C: Human breath-hold diving. *In* Lundgren CEG, Miller JN [eds]: The Lung at Depth. New York, Marcel Dekker, 1999.)

breathe through a tube at 2.5 m (about 8 ft) of depth in 1911.[9]

Immersion to the neck pushes the abdominal wall inward and the diaphragm upward; it also causes redistribution of blood into the chest, as the water pressure counteracts the pooling of blood in the dependent regions of the body (which happens in air because of gravity). Contributing to this intrathoracic blood pooling may be the increased cardiac afterload induced by water pressure, which makes vascular pressures outside the chest higher than intrathoracic pressure. Arborelius and colleagues[10] showed that about 700 mL of blood moved into the chest during head-out immersion in thermoneutral water, leading to a 30% or more increase in cardiac output. Conformational changes of the chest probably account for the 69% reduction in expiratory reserve volume and the 16% reduction in residual volume (RV) described by Agostoni and associates[11] during head-out immersion; blood redistribution is responsible for the 9% reduction in vital capacity (VC) observed by the same authors. In fact, the VC reduction during head-out immersion is affected by the water temperature, which influences vasomotor tone and, therefore, the degree of blood shift: the cooler the water, the larger the reduction, with no difference between the dry and immersed VC at 40°C and a reduction of VC to 91.9% in 20°C water compared with the dry condition.[12] Immersion in the horizontal position, as

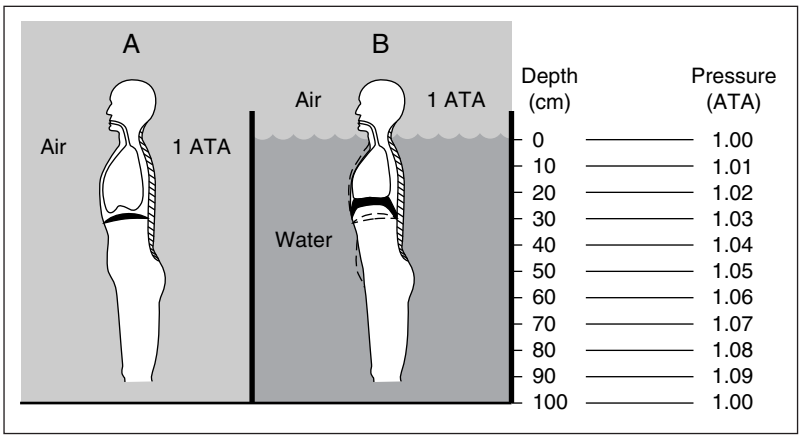

Figure 5–2. Distribution of pressure surrounding a man standing in air *(A)* and immersed in water to the neck *(B).* The density of dots reflects the magnitude of pressure. The *broken curves* over the chest and below the diaphragm indicate the position of the chest wall and the diaphragm standing in air. (From Hong SK: Breath-hold diving. *In* Bove AA, Davis JC [eds]: Diving Medicine. Philadelphia, WB Saunders, 1990, pp 59–68.)

during snorkeling, reduces VC in proportion to the chest depth, down to 68% of dry value when the sternum is 40 cm below surface.[7]

According to Agostoni and coworkers,[11] immersion also causes a 58% increase in airway resistance because of narrowing of airways due to a reduction in functional residual capacity, and possibly also because of a decrease in lung recoil secondary to vascular engorgement (causing a reduction in airway flow at low lung volumes): These changes result in a 12% reduction in maximal voluntary ventilation.[13] Pulmonary vascular congestion during immersion is also thought to be responsible for most of the increase in closing volume[14] and in the volume of trapped gas (up to 290 mL)[15] and for a 30% decrease in pulmonary compliance.[16] Interestingly, pulmonary compliance appears to decrease even more with time, suffering from an additional reduction of 13.2% over 30 min.[17] This phenomenon may be due to an increase in intravascular or interstitial fluid in the lungs over time (see the later discussion, Clinical Aspects of Breath-Hold Diving).

PHYSIOLOGY OF BREATH-HOLD DIVING

Cardiovascular System

The French physiologist Paul Bert first described "diving bradycardia" in 1870, when he reported an impressive slowing of the heart rate in ducks forced to stay underwater.[18] This vagally mediated reflex bradycardia is part of the diving response, which in diving animals also includes peripheral vasoconstriction, a reduction in cardiac output, maintenance of arterial blood pressure, and, in forced or longer dives, reliance on anaerobic metabolism and possibly a reduction in metabolic rate.[19] In 1935, Irving and colleagues[20] suggested that this response may conserve the limited oxygen available during a dive for the organs that are particularly sensitive to ischemia, such as the heart and brain. According to this concept, these important organs are perfused by blood at the expense of organs more resistant to ischemia (e.g., muscles, skin, and viscera), wherein the vasoconstriction leads to anaerobic metabolism with accumulation of lactic acid. The lactic acid is washed out after the dive as perfusion of the ischemic tissues is resumed.[21] The diving response is elicited by breath-hold diving, but it can also be triggered to certain extent by apnea alone,[22] immersion of the face in cold water,[23] or even simply by cooling the face.[24] Although the diving response was originally described in diving animals, humans were found to experience a similar response to breath-hold diving.[25] However, as explained in Clinical Aspects of Breath-Hold Diving (see later), the human diving response may include dramatic increases in arterial blood pressure and arrhythmias,[26] which appear to be rare in diving animals and which may be dangerous in breath-hold divers.

Breath-hold divers often experience tachycardia before diving, which may be due to stimulation of pulmonary stretch receptors[27] from the fact they start their dives with a deep inspiration, unlike diving animals who dive after expiration.[28] Predive hyperventilation and sometimes anxiety (i.e., during depth record attempts) may contribute to

Figure 5–3. Enzio Maiorca and his two daughters, Patrizia *(right)* and Rossana *(left),* preparing to perform deep breath-hold dives. Each of them has, at one time or another, held a world depth record. As explained in the text, they performed several dives to 55 m (~180 ft) in a research chamber, providing precious physiologic information.

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Figure 5–4. Electrocardiogram (ECG, *top trace*), invasively recorded arterial blood pressure *(middle trace)* and depth profile *(bottom trace)* during a chamber dive to 50 m (~164 ft) lasting 175 sec as performed by an experienced breath-hold diver. (From Ferrigno M, Ferretti G, Ellis A, et al: Cardiovascular changes during deep breath-hold dives in a pressure chamber. J Appl Physiol 83:1282–1290, 1997.)

this initial tachycardia. Ferrigno and coworkers[26] took electrocardiographic recordings on three elite breath-hold divers (Fig. 5–3) during wet dives down to 55 m (~180 ft) in cool water in a pressure chamber; initial tachycardia was followed by a sharp drop in heart rate to 20 to 30 beats/min near the "bottom" (Fig. 5–4). The divers' heart rate returned to normal predive levels within 15 sec after "surfacing." The bradycardia observed in these chamber dives, and in some breath-hold dives to 65 m (~213 ft) performed by the same elite divers at sea,[29] was quite irregular because of the presence of many cardiac arrhythmias. Arrhythmias have been described frequently in human breath-hold divers[30]; possible causes and clinical implications are discussed later under Clinical Aspects of Breath-Hold Diving.

The drop in heart rate appears to cause a decrease in cardiac output during breath-hold diving, as is observed during chamber dives to 55 m (about 180 ft) wherein cardiac output fell (to less than 3 L/min in two of the three elite divers) because of the bradycardia.[26] On the other hand, other studies of cardiac output performed at the surface have shown different results (either no change, a decrease, or an increase), probably because of the different experimental conditions.[31] As mentioned before, peripheral vasoconstriction is also part of the diving response, and in fact finger, forearm, and calf blood flow are reduced during breath-holding with face immersion.[32] However, blood flow in the carotid artery has been shown to increase by 36.6% when subjects were holding their breath while underwater

at a depth of 4 m.[33] Similarly, up to a 100% increase has been shown in cerebral blood flow (measured with magnetic resonance imaging, or MRI) during nonimmersed breath holds.[34] Although the reduced limb perfusion was attributed to an increased sympathetic activity in arterial limb vasculature, the increased cerebral perfusion was attributed to cerebral vasodilation secondary to the increase in CO_2 concentration in the blood during breath holding. Similar to what happens in diving animals, peripheral vasoconstriction may lead to anaerobic metabolism with lactate accumulation in peripheral tissues in human divers also. Such an increase in lactate has been described following breath-hold dives,[35,36] even in some deep ocean dives for which the authors calculated a low metabolic cost normally not associated with anaerobic metabolism.[37]

Unlike diving animals, in which arterial blood pressure remains constant or increases slightly,[19] human divers experience very large increases in blood pressure during diving: Ferrigno and colleagues[26] invasively recorded pressures up to 280/200 mm Hg (37.3/26.7 kPa) and 290/150 mm Hg (38.7/20.0 kPa), with a few systolic peaks reaching 345 mm Hg (46.0 kPa), in two elite divers in the early part of breath-hold dives to 50 m (~164 ft) in a chamber (see Fig. 5–4). Then, the blood pressure started to fall, probably because of bradycardia that may have developed because of baroreceptor stimulation. Anxiety about these chamber dives and a pronounced vasoconstrictor response to apnea and face immersion in cool water (probably secondary to their training) may have contributed to these high values of blood pressure in these two elite divers. Bjertnæs and associates[38] also noted very large increases in blood pressure, recording a mean arterial pressure as high as 25.33 kPa (~190 mm Hg) at the end of experiments involving apneic face immersion in ice water and exercise. However, other studies performed at the surface have shown no or only a small increase in arterial blood pressure.[31]

Another aspect of the diving response that human beings may share with diving animals is splenic contraction: Qvist and coworkers[39] described release of red blood cells from the spleen into the circulation in Weddel seals. In those animals, the hematocrit increased by 44% in the first 10 to 12 min of diving, providing an increase in both O_2 and CO_2 stores for the following dives. A 9.5% increase in hematocrit was found in Korean diving women after 115 dives to 6 m over a 2.5 hours: This increase was ascribed to splenic contraction because splenic volume was reduced by almost 20%.[40] In another study of the same professional divers, the hematocrit underwent cyclical increases of 8.9% with each of a series of breath-holds.[41] More recently, Schagatay and colleagues[42] observed a 6.4% increase in hematocrit in 10 normal subjects (but not in 10 splenectomized subjects) during a series of five breath holds; the hematocrit returned to the baseline value 10 min after the last breath hold.

Respiratory System

Gas exchange in the alveoli is influenced by the environmental pressure; therefore, its course during breath holding at the surface is very different from its course during breath-hold diving (Fig. 5–5),[43] as is discussed later. During a breath hold, O_2 uptake from the lung continues but CO_2 cannot be eliminated, and therefore it is retained in the diver's body to be stored first in the lungs and the blood and, after about a minute, in muscles and viscera.[44] In this condition, the Pa_{CO_2} first increases rapidly and then more slowly, with CO_2 net transfer from the blood into the alveoli nearly stopping at about 30 sec while O_2 uptake from the lungs, with its concentrating effect on alveolar CO_2, falls markedly (see Fig. 5–5).[45]

In the course of a voluntary breath hold, two phases can be observed: During the first one, called the "easy-going" phase, the glottis is closed and the intrathoracic pressure remains stable; this phase ends with the onset of involuntary inspiratory muscle contractions (with the glottis still closed) and is followed by the "struggle" phase.[46] During this latter phase, the involuntary contractions increase in frequency and intensity until the airway is opened, when the diver can no longer resist the urge to breathe. The end of the easy-going phase depends on physiologic factors, such as arterial P_{CO_2} and lung volume,[47] and therefore is called the "physiologic breaking point." Both psychological and physiologic factors influence the highly variable duration of the second phase, which ends with the "conventional breaking point" (coinciding with the end of the voluntary breath hold).[48] Mean alveolar

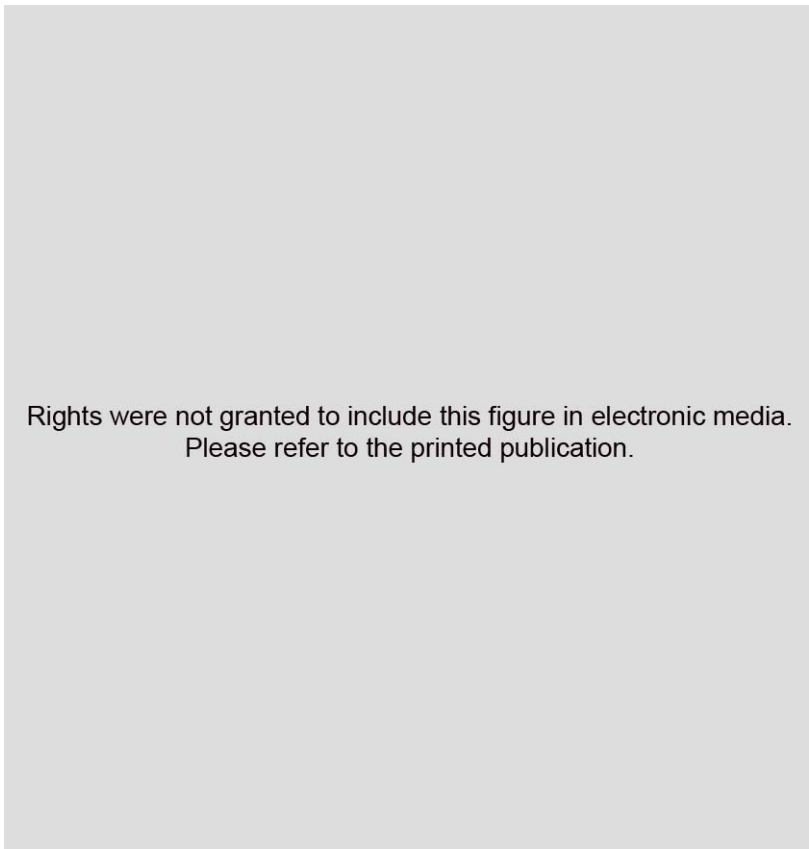

Figure 5–5. Exchange of O_2 *(top)* and CO_2 *(bottom)* during immersed breath holds at the surface and breath-hold dives to 20 m (simulated in a chamber). Gas transfer occurs from (positive values) or into the lungs (negative values). *Bars* show values of cardiac index obtained under identical conditions in other studies. STPD, standard temperature and pressure, dry (0°C, 760 mm Hg). (From Linér MH, Ferrigno M, Lundgren CEG: Alveolar gas exchange during simulated breath-hold diving to 20 m. Undersea Hyperbar Med 20:27–38, 1993.)

gas tensions at the conventional breaking point range from 43.3 to 53.5 mm Hg for CO_2 and 46 to 80 mm Hg for O_2, with the duration of maximal breath holds at rest ranging between 93 and 150 sec.[49]

Of course, there is an inverse relationship between breath-hold duration and oxygen consumption. For example, in a study by Lin and coworkers,[46] the average breath-hold duration was 162 sec at rest, whereas it was only 66 sec when the five subjects exercised at 167 kg-m/min. Diving techniques that reduce physical effort during a dive (as in the case of assisted diving, wherein the diver is pulled down by a weight and is pulled up by a flotation device—see Table 5–1) allow longer breath holds by decreasing the rate of oxygen consumption and carbon dioxide production. With a longer breath hold, the diver can reach greater depths. The metabolic rate is also increased by immersion in cold water, which can cause a great increase in O_2 uptake (256% higher than dry control)[50] and a 25% to 55% reduction in breath-holding times.[51] The 20% to 25% extension in breath-holding time caused by immersion in thermoneutral water[50] could be due to the increase in acute CO_2 storage capacity in this condition as described by Chang and Lundgren.[52] On the other hand, sudden immersion in 0°C water reduces breath-holding time by as much as 75% compared with the dry condition because of the very strong respiratory drive elicited by cold stimulation.[51]

During a breath-hold dive, as the pressure exerted by the water surrounding the diver's chest increases, so does the pressure of O_2 in the alveoli (see Fig. 5–5), allowing the diver to use more of the pulmonary oxygen store (a phenomenon sometimes called "burrowed oxygen").[53] Actually, even more important than the increased Pa_{O_2} to explain the higher O_2 uptake at depth may be an increased

cardiac output at depth, compared with breath-holding at the surface,[54] as pointed out by Linér and colleagues[55] (see Fig. 5–5.) This means that the breath-holding time can be longer at depth than at the surface. However, during ascent, as the ambient pressure drops, so does the Pa_{O_2}, and the diver now faces the risk of "hypoxia of ascent." Measurements taken at the end of breath-hold dives attest to this risk, with Pa_{O_2} values as low as 28 mm Hg (3.7 kPa) at end of 45 sec working dives to 11 m (36 ft) by Korean Ama[56] and Pa_{O_2} as low as 30.6 mm Hg (4.1 kPa) at the end of assisted dives to 70 m (almost 230 ft) in the ocean lasting about 150 sec.[37] In fact, during the last part of ascent, reversal of the oxygen flow (i.e., oxygen moving from the venous blood into the alveoli) has been observed.[43] Interestingly, this phenomenon may have the beneficial effect of slowing the fall in PA_{O_2} (and therefore in Pa_{O_2}), possibly offering some protection against hypoxia in the last part of deep dives.[57]

With regard to CO_2 exchange in the alveoli during a dive, its transfer is reversed during descent as the Pa_{CO_2} increases (because of chest compression) above the mixed venous level; during ascent, the falling ambient pressure reduces alveolar Pa_{CO_2}, thus re-establishing CO_2 transfer into the alveoli.[43] Because the volume of CO_2 transferred into the alveoli during ascent may not be enough to compensate for all the CO_2 accumulated during the dive, some CO_2 may be retained at the end of a breath-hold dive.

Hyperventilation is an effective method to extend breath-hold duration because it decreases the CO_2 stores in the diver's body at the beginning of a breath hold: it will therefore take longer for CO_2 accumulation during the breath hold to reach a breaking point level. However, vigorous predive hyperventilation is very dangerous because it does not appreciably increase the oxygen stores (it minimally increases lung stores and causes almost no change in blood stores, because the blood is normally already saturated with oxygen), whereas predive hyperventilation is very effective in delaying the diver's urge to breathe. Breath-hold durations of up to 5 min have been recorded in three nonimmersed elite divers[37]; longer breath-holds (up to 7 min) have been achieved during immersion in a pool.[58]

Unfortunately, the diver's hypercapnic respiratory drive can be delayed past the point of hypoxic loss of consciousness, which, underwater, would lead to drowning.[59] When hyperventilation is combined with oxygen inhalation during the final breaths, breath-hold duration appears to depend mostly on the pulmonary oxygen store, with breath-holding times up to 14 min.[60] These prolonged breath-hold durations with O_2 may also be due to the fact that the level of alveolar ventilation from a certain Pa_{CO_2} is modulated by the Pa_{O_2}, and vice versa. Klocke and Rahn recorded alveolar CO_2 tensions as high as 91 mm Hg (12.1 kPa) at the end of breath holds lasting up to 8.5 min after oxygen breathing[60]; therefore, this maneuver may lead to dangerously high CO_2 tensions.

During diving, the volume of the gas in the lungs is reduced according to Boyle's law; divers who go deep enough could damage their chest. Until the mid-1960s, researchers thought that the maximal depth that a diver could safely reach could be calculated from the ratio of the maximal initial lung volume (total lung capacity, or TLC), and the minimal natural volume (RV) of the lungs. According to this concept, most divers could not have safely gone any deeper than about 30 m (<100 ft) without risking "chest squeeze." However, the scientists were proven wrong by the record-setting divers who have reached greater and greater depths, diving repeatedly to more than 100 m (328 ft) down to the present record of 160 m (almost 525 ft). At this depth, the diver's lung volume should have been reduced to about 1/17 of its volume at the surface, well below RV.

The fact that these deep dives have been performed without evident damage to the chest is due to translocation of blood from the periphery into the heart and vascular bed in the diver's chest; blood is practically incompressible, and therefore it makes up for the loss in gas volume in the diver's lungs, thus opposing chest compression below its natural (minimal) volume. According to this concept, blood moves into the chest during descent along a transthoracic pressure gradient caused by a drop in intrathoracic pressure (relative to the pressure outside the diver's chest) as the chest is compressed and it starts recoiling outwards.[31] Craig first demonstrated this redistribution of blood in 1968 by calculating that 600 mL of blood had entered a subject's chest during a dive to 4.5 m (almost 15 ft), performed after expiration to RV, without significant change in transthoracic pressure.[61]

Schaefer and coworkers[62] provided additional evidence for this phenomenon when they measured intrathoracic translocation of up to 1047 mL of blood during experimental dives to 130 ft; more recently, Warkander and colleagues[49] reported estimates of intrathoracic blood pooling of up to 1.7 L during chamber dives to 55 m (~180 ft). Finally, it is also possible that the diver's chest may be safely compressed during descent to a smaller volume than the one arrived at during forced exhalation at the surface. Unfortunately, although translocation of blood allows deep dives, it may also cause damage to the blood-containing structure of the diver's chest, as discussed later under Clinical Aspects of Breath-Hold Diving.

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Some respiratory techniques may help divers to start a dive with the largest possible volume of air in the lungs, potentially helping them to reach greater depths. For example, some record divers take the last maximal inspiration (prior to diving) either outside the water or partially immersed to the waist line, minimizing the intrathoracic pooling of blood due to immersion.[5] Similarly, whistling sounds performed by the Ama divers may allow inspiration of a larger volume of air prior to diving, as the resulting increase in intrathoracic pressure is likely to expel blood out of the diver's chest.[63,64]

FACTORS AFFECTING THE DIVING RESPONSE AND THE HUMAN ABILITY TO DIVE

Several factors can affect the diving response, including lung volume, intrathoracic pressure, hypoxia, hypercapnia, exercise, diving experience, age, and psychological factors. With regard to lung volume, bradycardia and reduction in peripheral flow appear to be more pronounced at lower lung volumes.[65] The increase in intrathoracic pressure usually present during a breath hold at the surface (due to the inward recoil of both the lungs and the chest wall at large lung volumes[31]) reduces venous return into the chest, thus decreasing the cardiac output and, through pressor-receptor unloading, contributes to the tachycardia frequently observed at the beginning of a dive (see Fig. 5–4). However, during descent, the intrathoracic pressure relative to the pressure outside the diver's chest drops as the chest wall recoils outward at low lung volume,[31] possibly contributing to the diving bradycardia. Lin and associates[66] experimentally separated the effects of apnea, hypoxia, and hypercapnia on diving bradycardia: Apnea by itself decreased heart rate by 18%, whereas hypoxia contributed an additional 18% reduction and hypercapnia actually caused a 6% acceleration, resulting in a net heart rate reduction of 30%. Physical exercise, particularly the dynamic type, appears to potentiate diving bradycardia.[67] A similar effect has also been described by some authors for physical conditioning[68] and for diving experience,[22] whereas other studies have not confirmed such an effect of conditioning[69] or diving experience.[70] Finally, the diving response appears to be strengthened by anxiety and fear,[71] whereas it is diminished by increasing age.[72]

The diving response appears to have an oxygen-conserving role in habitually diving animals by slowing depletion of central O_2 stores (in the blood and lungs) during prolonged dives.[19,28] This occurs because bradycardia reduces O_2 consumption in the heart and peripheral vasoconstriction reduces it in splanchnic organs; in addition, a reduced perfusion of the muscles makes them rely first on O_2 derived from myoglobin rather than blood, and then on lactic acid production. Some experimental evidence indicates a similar role for the diving response in trained human breath-hold divers. In 1965, Wolf and colleagues[72] described a slower oxygen desaturation in arterial blood during a pronounced diving response, compared with a situation in which bradycardia or peripheral vasoconstriction (or both) were less intense; these findings were later confirmed by other authors.[73,74] A pronounced diving response has also been associated with prolongation of maximal breath-holding times: In 1985, Mukhtar and Patrick observed a 15% increase in apneic time during breath holds with face immersion in cold water compared with breath holds without face immersion.[75] These authors ascribed this phenomenon to a reduction in ventilatory drive.[76]

Splenic contraction, similar to what occurs in Weddel seals,[39] may also lead to prolongation of breath holding in divers: Its associated increase in hematocrit would augment both O_2 and CO_2 storage capacity, thus postponing the physiologic breaking point.[42] Another aspect of the diving response that may aid oxygen conservation is reliance on anaerobic metabolism, first

described in divers by Schaefer in 1965.[77] More recently, Ferretti and coworkers[37] observed increased anaerobic energy production (at levels of oxygen consumption normally not associated with such increases) during breath-hold dives down to 70 m (almost 230 ft) in the ocean as performed by three elite breath-hold divers. The same authors found that the ability to rely on anaerobic metabolism appears better developed in trained divers than in nondiver controls. Finally, prolongation of breath holding also results from a short-term training effect: A more than 200% increase in the time to the physiologic breaking point (with a much more modest postponement of the conventional breaking point) during a series of five breath holds, with 3 min recovery periods, may be due to involuntary hyperventilation between breath holds.[78]

Other physiologic adaptations have been described in experienced breath-hold divers. A blunted ventilatory response to hypoxia and hypercapnia in divers compared with nondivers was ascribed to training in the case of U.S. Navy divers,[79] Japanese Ama,[80] and elite deep divers.[81] Training is also responsible for some changes in respiratory mechanics likely to improve diving performance, such as a greater VC and the ability to generate a higher inspiratory pressure in the diving women of Korea and Japan compared with nondiving controls[82,83] as well as a larger VC and a smaller RV in U.S. Navy divers resulting from training.[84] Besides strengthening of the respiratory muscles in divers, these changes may be due to increased compliance of the respiratory system in Japanese Ama,[85] which would provide a larger pulmonary gas store (prolonging breath holding) and a higher TLC/RV ratio (allowing deeper dives before chest squeeze ensues). Finally, a higher CO_2 storage capacity has been described in three highly trained breath-hold divers compared with untrained controls: It took longer for the divers to reach their breaking-point Pa_{CO_2}, with twice the amount of CO_2 stored in the tissues of the divers compared than in controls.[37]

CLINICAL ASPECTS OF BREATH-HOLD DIVING

Physicians should consider the conditions described in the following paragraphs when examining or treating breath-hold divers. The pathophysiologic mechanisms of these conditions are described under Physiology of Breath-Hold Diving. It is also important to remember that there are forms of involuntary breath holding, such as a person falling in water or the diver using an underwater breathing apparatus that suddenly malfunctions. Finally, some of the cardiovascular and respiratory physiology discussed earlier for breath holding at the surface may apply to the clinical situation in which a patient in acute respiratory failure (secondary, for example, to either an upper airway obstruction or the use of neuromuscular blocking agents, commonly known as muscle relaxants) cannot be adequately ventilated by the assisting physician or other medical personnel.

Cardiovascular Problems

Extreme levels of bradycardia have been reported during both simulated and actual breath-hold diving. In 1985, Arnold described R-R intervals as long as 10.8 sec, corresponding to a heart rate of 5.6 beats/min, induced by apneic face immersion in cold water[86]; Ferrigno and associates[26] recorded R-R intervals corresponding to heart rates of 8, 13, and 24 beats/min in three elite divers, respectively, during chamber dives to 55 m (180 ft) in cool water (Fig. 5–6). Heart rates as low as 20 to 24 beats/min were also recorded in the same three divers during ocean dives to 65 m (~213 ft).[29] Interestingly, these divers reported no symptoms during these episodes of accentuated bradycardia, probably because the intense peripheral vasoconstriction of the diving response helped to maintain cerebral perfusion pressure during the prolonged diastolic periods. A large number and variety of arrhythmias have been described in breath-hold divers[36]; these rhythm disturbances are more frequent during dives in cold water[30] and while the diver is at depth,[26] and they are not only of the inhibitory type (to be expected from an increased vagal tone) but also include premature contractions (Fig. 5–7).[26] Despite the frequent arrhythmias recorded in three elite breath-hold divers during chamber dives to 55 m (180 ft) in cool water, these divers did not report any symptoms even during prolonged periods without any sinus beats (up to 45 sec; see Fig. 5–7), probably because many of the arrhythmic beats were hemodynamically effective.[26] The following

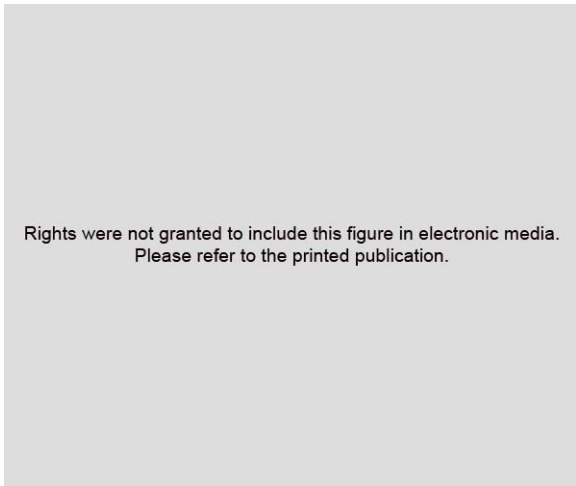

Figure 5–6. Electrocardiographic recordings in three experienced breath-hold divers during chamber dives to 50 m (*A* and *C*) and 40 m *(B)* in 25°C water. (From Ferrigno M, Ferretti G, Ellis A, et al: Cardiovascular changes during deep breath-hold dives in a pressure chamber. J Appl Physiol 83:1282–1290, 1997.)

factors may contribute to the development of arrhythmias during diving:

- High vagal tone
- Distention of the heart from blood redistribution into the chest secondary to both immersion, particularly in cold water,[12] and to a drop in intrathoracic pressure during diving[31]
- Apneic face immersion in cold water[87]
- Possible subendocardial ischemia[88] from a large increase in blood pressure[26]

In fact, as already described under Physiology of Breath-Hold Diving, arterial hypertension has been observed in breath-hold divers, with systolic values of approximately 300 mm Hg and diastolic values of approximately 200 mm Hg.[26]

Arrhythmias and arterial hypertension appear to be rare in diving animals (P. Ponganis, personal communication), and they may represent maladaptations in human divers. These phenomena appear to be well tolerated by young and fit divers but may have more ominous consequences in older persons or in divers with preexisting cardiac disease. There is also the danger that the large intrathoracic blood pooling (more than 1.5 L of blood is redistributed from peripheral tissues into the heart and vessels in the chest during deep breath-hold dives)[49] that protects the diver from chest squeeze may cause rupture of pulmonary vessels and overdistention of the heart. In this regard, there are several anecdotal accounts of divers coughing up blood-tinged sputum after repetitive breath-hold dives to 30 m or more (L. Magno, personal communication). Better documented is the case of an unfortunate French diver who, after a series of dives to 25 m (82 ft) over 2 hours, experienced hemoptysis and died shortly thereafter.[89] He had taken aspirin before diving, and he was found to have intraalveolar hemorrhage by radiography, bronchoscopy, and bronchoalveolar lavage.

Actually, even while a diver is swimming at the surface, approximately 700 mL of blood[10] is already redistributed from the periphery into the chest. Cardiac diastolic filling may increase by 180 to 250 mL, and pulmonary capillary blood volume may increase by 51 to 200 mL.[90–93] These hemodynamic changes, which are enhanced by immersion in cold water, may contribute to pulmonary edema in swimmers and divers (see Chapter 25).[94–96] Typically, the symptoms, including shortness of breath and coughing, resolve as soon as the diver gets out of water; symptoms may become more frequent with advanced age[97] and in swimmers with subnormal baseline spirometry values.[98] Snorkeling between dives, allowing the chest to be submerged more deeply, results in a lower intrathoracic pressure, further increasing intrathoracic blood pooling and possibly contributing to deaths in elderly divers due to increased cardiac pre- and afterload or arrhythmias.[87,99]

Problems in the Respiratory System

As mentioned before, immersion induces air trapping in the lungs; this phenomenon appears to be more pronounced in

asthmatics, in whom reductions in pulmonary airflow have been observed during immersion after exercise.[100] Physicians should remember this phenomenon when evaluating divers with asthma. Pulmonary maneuvers, such as "buccal pumping" or "lung packing," are sometimes used by breath-hold divers to increase TLC and therefore the TLC/RV ratio at the beginning of a dive, potentially increasing the reachable depth. These techniques consist of rapidly taking in mouthfuls of air after a maximal inhalation while performing maneuvers similar to swallowing, which direct the additional air it into the lungs. By doing so, the divers can increase VC by up to about 40%,[101] probably because blood is expelled out of the chest due to the increased inward recoil of the overexpanded chest and lungs. In fact, the large increase in airway pressure resulting from these maneuvers could lead to lung rupture.[102] These dangerous techniques can also cause substantial reductions in blood pressure and even fainting secondary to a decrease in venous return and, consequently, in cardiac output.[103]

Although pulmonary barotrauma during ascent is typically a danger for divers who breathe a compressed gas underwater, this condition may affect a breath-hold diver, even though the total gas volume in the lungs at the end of a dive cannot be larger than the one present at the beginning of the dive. What could happen in a breath-hold diver is that something would prevent escape of the expanding gas from one or more regions of the diver's lungs during ascent, causing localized overdistention, rupture, and its clinical consequences, including pneumothorax, pneumomediastinum, and arterial gas embolism. Some cases of neurologic problems and even death in breath-hold divers may have been caused by emboli secondary to pulmonary barotrauma of ascent.[104–106] Several mechanisms have been suggested for this condition. One possibility is related to very rapid ascents: Blood that had redistributed into the pulmonary circulation during the descent may drain out of the pulmonary vessels more slowly than the rate at which alveolar air is expanding, causing blood engorgement of these vessels (L. Magno, personal communication). This would lead to a decrease in lung compliance and an increase in airway closure,[107] with the possibility that some regions of the diver's lungs may not be able to safely accommodate the expanding gas during ascent. Another possibility is related to differences in compliance between lung regions, particularly in divers with preexisting lung disease or surgical scarring, causing tears in the lungs. Finally, as mentioned earlier, lung-packing maneuvers or simply a very forceful inspiration could lead to lung rupture before the dive,[108] making the diver more susceptible to pulmonary barotrauma during ascent.

Neurologic Problems

Breath-hold divers can experience decreased levels or even loss of consciousness from hypoxia as a consequence of hyperventilation or hypoxia of ascent; when the diver is alone, this may lead to drowning. Craig[109] clearly explained the danger of a forceful hyperventilation when he cited 58 cases of loss of consciousness during underwater swimming. Spear fishermen face a similar danger,[110] although loss of consciousness appears to be rare among Ama divers, who do not practice forceful hyperventilation.[56] As explained earlier, hypoxia of ascent results from the fall in alveolar P_{O_2} that is particularly rapid during the final part of ascent. At this dangerous time, there may be a paradoxical relief from air hunger due to expansion of the chest wall[111] and the concomitant fall in alveolar P_{CO_2},[43] giving the diver a false sense of security.

Another condition that could lead to decreased levels of consciousness in breath-hold divers is CO_2 accumulation, which could occur if surface intervals between dives were very short in the absence of vigorous hyperventilation. Paulev and Neraa described enough CO_2 retention to cause narcosis following a series of seven dives to 18.5 m (about 60 ft), separated by surface interval of only 1 to 2 min.[112] Linér and Linnarsson recommended surface intervals of at least 3 min between dives to avoid CO_2 accumulation.[113]

In 1965, Cross[64] suggested the possibility that repetitive breath-hold diving could cause decompression sickness: He described several neurologic symptoms, including partial or complete paralysis, vertigo, loss of consciousness, and even death, in pearl divers from the Tuamotu Archipelago, where these problems were called *taravana* (*tara,* "to fall"; *vana,* "crazily"). These divers performed frequent dives to 100 fsw or more,

with bottom times of 30 to 60 sec, staying underwater for about a minute and a half; they dived for about 6 hours a day with brief intervals between dives. In the same year, Paulev[114] described similar neurologic problems in four divers of the Danish Navy after repeated breath-hold dives to 15 to 20 m (49 to 65 ft); fortunately, these divers were successfully treated with recompression in a hyperbaric chamber. Theoretical calculations by Lanphier indicated that enough nitrogen could be accumulated after repeated deep breath-hold dives separated by short surface intervals to cause decompression sickness.[115] In fact, nitrogen accumulation with repetitive breath-hold diving has been described in venous blood of Korean female divers.[116] A considerable amount of nitrogen can also accumulate during the course of a single deep breath-hold dive: In 1987, Olszowka[117] calculated that an extra 700 mL of nitrogen would accumulate in the body of a diver after a single 220 sec dive to 90 m (295 ft).

Serious neurologic problems, including sensory, motor, visual, and speech disturbances, have been reported more recently in breath-hold divers from Australia,[118] Italy,[119] Spain,[106,120] France,[121] and Japan.[122,123] Fortunately, most of these neurologic problems either resolved spontaneously or were successfully treated with recompression. Some changes in diving techniques may have contributed to the reappearance of decompression sickness among breath-hold divers: In the case of the Spanish divers, all of them had repeatedly dived to 40 m (131 ft) or more using electrically operated underwater scooters; in the case of the Ama divers from Japan, in whom decompression sickness was not a problem in the past,[124] the relatively recent introduction of wet suits has allowed longer daily diving sessions in recent decades. This new practice may be responsible for the appearance of decompression sickness among the Amas,[125] as confirmed by focal cerebral injuries detected with MRI in some Japanese divers.[122,126] MRI presented a similar picture in a French diver[121]; the possibility that emboli may be responsible for these lesions has been corroborated by detection of venous gas emboli with ultrasound Doppler technique after repetitive breath-hold diving.[127,128]

Neurologic problems suggestive of decompression sickness have also been reported in at least two cases of single deep breath-hold dives.[106,119] In one case, the diver was using a new and faster buoyancy device to ascend from about 120 m (almost 394 ft); his rate of ascent was about 4 m/sec (13 ft/sec) and, shortly after surfacing, he experienced paresthesias, quickly followed by right-sided hemiplegia.[119] Fortunately, his symptoms resolved within about 30 min during recompression treatment. A possible explanation for these symptoms is bubble formation in the arterial blood during an extremely rapid ascent: In this situation, blood saturated with nitrogen at a given depth would reach the brain (and release bubbles) when the diver has arrived at a much shallower depth.[49] Another possibility is that the diver suffered from a form of pulmonary barotrauma leading to arterial gas embolism (see the earlier discussion, Problems in the Respiratory System).

Finally, even at depths at which scuba divers suffer from nitrogen narcosis, this condition does not appear to be a practical problem in deep breath-hold diving, probably because exposure to high nitrogen pressures is very brief. It is also possible that nitrogen uptake is greatly reduced during a deep breath-hold dive because the alveolar area available for gas exchange is reduced by the extreme compression of the lungs at great depths.

Ear and Sinus Problems

Breath-hold divers may be particularly prone to ear and sinus barotrauma and related problems because of repeated exposures to rapid pressure changes, particularly at shallower depths. For a detailed discussion of these conditions, see Chapter 22.

CONCLUSIONS

Breath-hold diving to modest depths is a wonderful sport that can be done safely as long as divers understand the physiologic changes this activity produces and take appropriate precautions, such as limiting predive hyperventilation and never diving alone. On the other hand, deep breath-hold diving is much more dangerous, as demonstrated by the accidents involving spear fishermen and record divers as described under Clinical Aspects of Breath-Hold Diving.

Recently, a terrible accident claimed the young life of Audrey Mestre during her attempt to establish a new world depth record with totally assisted technique (also known as "no limit").[129] This unfortunate event is a reminder of how dangerous this type of extreme breath-hold diving is and should lead to its abandonment.

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CHAPTER 6 Mixed-Gas Diving

R.W. Bill Hamilton

Atmospheric air has always been the standard gas for diving. Air has the advantages of being familiar and safe to breathe for shallow-water diving, and with proper handling and perhaps filtration, it can be available for diving purposes anywhere on earth. Air is compressed into cylinders or used directly from compressors with surface-supplied equipment. For practical purposes, the limit of air diving is a depth of about 50 m, at which depth the pressure is approximately 6 atm (approximately 600 kPa); some air diving is performed up to the range of 60 msw (meters of seawater) depth. Beyond this depth, and within it for special purposes, mixtures other than air are used. These may be mixtures of helium and oxygen; various mixtures of nitrogen and oxygen; pure oxygen; trimixes of oxygen, helium, and nitrogen; or more exotic gas mixtures based on neon, hydrogen, or sometimes even argon. All of these mixtures may provide advantages over air under specific conditions.

Overwhelmingly, the reason for using a nonair mixture is to avoid narcosis in deep diving, but other advantages of nonair mixtures might include better characteristics for decompression, lower density, lower thermal capacity, better control of oxygen toxicity, reduced support of combustion, and operational demands. Cost and availability are also important considerations. The categories of diving that use special gas mixes include commercial, military, scientific (which includes diving from undersea habitats), and now also recreational and the extended-range recreational diving category known as technical diving. To a large extent, diving can be categorized as saturation or nonsaturation; these categories are mainly related to decompression methods, but gas properties play a big role.

TERMINOLOGY

The term *mixed gas* has a specific meaning in commercial and military diving, but it may also refer to any breathing mixture other than air. *Mixed-gas diving* traditionally refers to diving with mixtures of helium and oxygen, also called *heliox* or just "gas," which for many years was the only widely used alternative to air. *Special mix diving* is a more general term for nonair diving.

Another category of nonair gas mixes not containing helium is now extensively used in recreational, scientific, and some military diving; this is oxygen-enriched air, colloquially called *nitrox,* which involves mixtures of oxygen and nitrogen, most often air with extra oxygen. Yet another mixture category increasingly in use is that of a *trimix* of oxygen, helium, and nitrogen. Pure oxygen might also be regarded as an exotic gas in diving; as a diving gas it is limited to shallow water, but it is widely used for decompression both in and out of the water. Oxygen is always present in diving mixes, but for use in deep diving it may be a very small fraction of the mix. For example, the terms *diving on gas* or *diving on neon* may be used, but oxygen is always present in the mixture. It is imperative that the inspired oxygen partial pressure (Po_2) of a gas mixture be appropriate for the situation because large deviations can be disastrous. Oxygen management is a major element of diving with mixtures; its operational aspects are covered briefly in this chapter.

The new practice of "technical" diving gives a fresh perspective to the basic concept of diving with gas mixtures other than air. Technical diving is untethered diving (with scuba or rebreathers) beyond the traditional air range, made possible

because of extra experience, training, and discipline—which lead to competence—and special equipment, breathing mixtures, decompression tables, support, organization, and procedures. Technical dives involve the use of more than one gas mix during a dive or the use of a rebreather. With technical diving being an established form of diving, medical personnel need to understand it and be equipped to deal with the special problems it may generate.

The term *gas* usually refers to an elemental gas or compound such as oxygen, nitrogen, helium, neon, hydrogen, or argon; gases make up the components of a gas *mix* or *mixture*. The proportion of a gas in a mixture (by volume) is its *fraction* and is usually expressed as a decimal; components may also be expressed as percentages (the fraction multiplied by 100). If one or more gases are specified, the remainder may be called the *balance*.

The concept of *partial pressure* is essential to the understanding of gas physiology. The partial pressure of a gas component of a mixture is the fraction of the component gas multiplied by the total pressure. For example, for the oxygen in air at a pressure of 1 atm, the inspired oxygen partial pressure (PIO_2) equals the total pressure (P_T) times the fraction of inspired oxygen (FIO_2):

$$PIO_2 = P_T \times FIO_2$$

$$PIO_2 = 1.0 \text{ atm} \times 0.21$$

$$PIO_2 = 0.21 \text{ atm}$$

Physiologists represent partial pressure with a capital P often followed by a letter indicating the source or location and a small cap letter with subscript indicating the gas symbol. Engineers tend to use the abbreviation *PP* for partial pressure, which also is unambiguous. Using a single lower-case *p* is confusing and should be avoided because that has another meaning in chemistry, as in *pH*. Fraction is abbreviated with a capital F.

Partial pressures are expressed in units of pressure, generally atmospheres (atm), or in bars or pascals (Pa) in SI units (System Internationale or "metric"); sometimes depth units are used. This chapter uses atmospheres (atm) for partial pressures. A metric unit, the *bar* (defined as 100 kPa), is physiologically equivalent to 1 atm (the atm is 101.325 kPa). A meter of seawater (msw) is defined as 0.100 bar or 10 kPa, and the Imperial depth unit is the foot of sea water, defined such that 1/33 atm = 1 fsw and therefore 3.2568 fsw = 1 msw. The megapascal (1 MPa = 10 bar) is becoming more commonly used for pressure measurements. This chapter uses SI units or metric units. For the record, to express partial pressures in "ata" or "ATA," intending to mean atmospheres absolute, is inappropriate because these gas partial pressures represent chemical potentials and the distinction of absolute or differential pressure has no relevance in that context. This chapter uses *atm*. Appendix 1 provides a table of pressure units and conversions.

GASES AND GAS PROPERTIES

The two main purposes for using a special nonair diving gas mixture are (1) to be able to change the makeup of the inert gas components and (2) to be able to control the oxygen level. Usually, both purposes are served by the diving mix. These are related to the properties of the gases, reviewed here in general terms. The attributes of an inert gas that are of greatest interest in diving are its potential to cause narcosis, decompression properties, density, thermal properties, and effect on speech, with narcosis clearly the dominant factor. Cost and availability may take precedence over some of these physical properties.

Because oxygen can be toxic at higher concentrations, metabolically inert gas is used in the mix to lower the oxygen level; as breathed, the inert gas itself is not metabolized by the body. The inert gas is called a *diluent gas* because it dilutes the oxygen.

The selection of inert gases is limited. Whether these gases are useful as diving gases depends largely on their density and fat solubility, which tend to be correlated with narcotic potency. Some of the useful inert gases are diatomic (they have two atoms per gas molecule), including nitrogen (N_2) and hydrogen (H_2). Oxygen (O_2) is also diatomic but is by no means inert. The next series of potentially useful gases is that of monatomic noble gases, which are inherently inert and which do not normally combine chemically with other gases: helium, neon, argon, krypton, xenon, and radon. Other hydrocarbon and combined hydrocarbon gases that are inert enough to be breathed include methane, acetylene,

carbon tetrafluoride, and sulfur hexafluoride. Nitrous oxide (N_2O, "laughing gas") and other gases of larger molecular weight, such as halothane, cyclopropane, and xenon, act as anesthetic gases. Nitrous oxide is used to stimulate nitrogen narcosis without having to increase pressure above that of the atmosphere. At sea level, the noble gas xenon is a mild anesthetic of about the same potency as nitrous oxide; it is too expensive to be used routinely for anesthesia.

Inert Gases

Nitrogen is the familiar inert diluent gas, being the inert gas in air. Although nitrogen can be oxidized at high temperatures and is frequently found in biologically important compounds such as proteins and their amino-acid building blocks, higher animals cannot convert molecular nitrogen to compounds that can be metabolized (this process can be performed by certain bacteria), and nitrogen remains available as an inert gas. It has recently been discovered that at the cell level, nitric oxide (NO) acts as a hormone that, among other functions, regulates dilatation of small blood vessels. The narcotic action of a gas does not result from chemical combination in the traditional sense but is the result of a physical process analogous to that occurring during gaseous anesthesia; all of the gas taken up by the organism being anesthetized or narcotized is eventually excreted without being changed.

Hydrogen in its diatomic form (H_2) behaves in the respiratory system as if it were totally inert. In addition to being a component of water, hydrogen ions formed from the breakdown of water cause acidity. Also, hydrogen, when added catalytically to unsaturated fats such as vegetable oil, causes the fats to harden, making them more saturated. Early experimenters with hydrogen as a diving gas were concerned about the possibility that large amounts of hydrogen in the body at relatively high pressures might cause either of these reactions—a disturbance of the acid-base balance or hydrogenation of the lipid in nerve cells. So far, neither of these concerns appears to be valid. The removal of hydrogen from the body during decompression by certain bacterial enzymes that can metabolize molecular hydrogen is being investigated; this is examined further in the discussion of diving gases. It is fairly certain that hydrogen is not oxidized by mammalian tissue.[1]

Other hydrocarbon gases such as methane and acetylene are inert as breathing gases, but most of these are too narcotic and flammable to be useful; they are sometimes used as tracers in laboratory work. Fuel gases must have a certain fraction of both the fuel gas and oxygen available in order to burn, and therefore mixtures containing only a small percentage of the gas with oxygen or air will not burn. At the other end of these mix ratios, it is possible to have mixtures with the oxygen fraction low enough (<~5%) that they will not burn, but that at increased pressures can carry enough oxygen to provide a normoxic P_{O_2} and meet respiratory requirements. The "chipmunk" speech resulting after inhalation of helium can be a serious limitation to the use of this gas, but helium speech "unscramblers" can render a satisfactory level of intelligibility (99% is claimed for modern units). Hydrogen inhalation also leads to unclear speech and may confuse an unscrambler tuned to helium. Neon causes much less speech distortion than helium, as is the case with oxygen-helium-nitrogen trimixes having a significant amount of nitrogen (Table 6–1).

Properties of Diving Gases

Viable choices of component gases for diving are discussed in this part of the chapter. (Densities given are those at 37°C and 1 atm of pressure.) The common physical and chemical properties are summarized in Table 6–1. Diving mixtures consist of one or more inert gases in combination with a fraction of oxygen that will give a suitable P_{O_2} at the depth of the dive.

AIR (MOLECULAR WEIGHT, 29; DENSITY, 1.1 g/L)

Despite the ubiquity and general suitability of air for breathing, for diving purposes there are good reasons for wanting an alternative to air and the nitrogen it contains. Overwhelmingly, the problem with air as a diving gas is narcosis, but its density and the toxicity of its oxygen component can also be detrimental factors; also, in some cases, air's decompression properties are unfavorable.

Pls. confirm this style.

Table 6–1. Physical and chemical properties of common diving gases

	Air	Hydrogen	Helium	Neon	Nitrogen	Oxygen	Argon
Molecular weight	28.8	2.016	4.003	20.183	28.016	31.999	39.944
Density at 37°C (g/L 1 atm)	1.139	0.0792	0.1572	0.7926	1.1017	1.2584	1.571
Viscosity at 20°C, 1 atm (μpoise)	182.7	87.6	194.1	311.1	175	201.8	221.7
Solubility in water at 38°C (mL/mL 1 atm)	—	0.017	0.0086	0.0097	0.013	0.095	0.026
Solubility in olive oil at 38°C (mL/mL 1 atm)	—	0.05	0.015	0.019	0.061	0.012	0.14
Diffusivity in water at 37°C ($cm^2/sec \times 10^{-6}$)*	—	112.6	63.2	34.8	30.1	28.2	25.2
Diffusivity in olive oil at 37°C ($cm^2/sec \times 10^{-6}$)	—	26.3	18.6	8.34	7.04	6.59	5.29
Thermal conductivity ($cal/sec/cm/°C \times 10^{-5}$)	6.42	45.9	36.9	11.8	6.4	6.6	4.4
Specific volume gas/liquid 21°C, 1 atm (m^3/kg)	0.83	11.99	6.06	1.20	0.87	0.75	0.6
Thermal capacity or specific heat (Cp) cal/g/°C	0.24	3.39	1.25	0.25	0.25	0.22	0.13
Thermal capacity or specific heat (Cv) cal/g/°C	0.17	2.40	0.75	0.15	0.18	0.16	0.08
Ratio of specific heats (Cp/Cv)	1.40	1.41	1.66	1.64	1.40	1.40	1.67

*Diffusion coefficients are calculated by Graham's law from nitrogen data.[114]
Data from Compressed Gas Association: Handbook of Compressed Gases, 3rd ed. New York, Chapman & Hall, 1990; Flynn ET, Catron PW, Bayne GL: Diving Medical Officer Student Guide. Course A-6A-001O. Panama City, Fl., Naval Diving and Salvage Training Center, 1981; Weast RC (ed): Handbook of Chemistry and Physics. Cleveland, Chemical Rubber, 1969.

At the limit of its depth range, air becomes significantly dense, which can limit a diver's ability to perform work and may contribute to a buildup of carbon dioxide.

OXYGEN (MOLECULAR WEIGHT, 32; DENSITY, 1.3 g/L)

Oxygen is, of course, the critical component of any breathing gas mixture. It is quite soluble in both water and fat and is thought to be as narcotic or slightly more narcotic than nitrogen. Because oxygen is metabolized in tissue, the exact amount present in a given tissue under different conditions is hard to determine. When present in excess, oxygen can contribute to bubble formation during decompression. Its thermal properties are similar to those of nitrogen, and it is an inexpensive gas. Great care is required when handling oxygen at high pressures because of its strong oxidizing properties.

NITROGEN (MOLECULAR WEIGHT, 28; DENSITY, 1.1 g/L)

Nitrogen is the major constituent of air, and many of its properties are similar to those described for air. The narcotic potency of nitrogen is the dominant reason to find an alternative. Nitrogen can also be difficult to unload during decompression, but it may be preferred over helium for short dives, in which smaller amounts of the gas are taken up. Under pressure, nitrogen's density can be significant.

HELIUM (MOLECULAR WEIGHT, 4; DENSITY, 0.16 g/L)

Helium is not narcotic at any pressure. Its low density makes it relatively easy to breathe at high pressures, and it is beneficial for therapy in respiration-compromised patients. Helium is relatively insoluble and is therefore favorable for decompression, except in short exposures, during which it appears to be taken up faster than nitrogen by body tissues.

NEON (MOLECULAR WEIGHT, 20; DENSITY, 0.8 g/L)

Neon is not narcotic, but its density approaches that of nitrogen and can limit its use. Its solubility is low, being close to that of helium. Its expense limits neon to use for diving only in exceptional circumstances. Because it is extracted from atmospheric air, it can be obtained in places where helium is not available. Because of its physical properties, neon is expected to have favorable decompression properties, and this is supported by limited data. Neon would be the ideal inert gas for use in a space station atmosphere, where cost may not be a major factor.

HYDROGEN (MOLECULAR WEIGHT, 2; DENSITY, 0.08 g/L)

Hydrogen behaves as an inert gas when breathed, and it is being promoted as a diving gas for three main reasons. Its low density makes it favored over helium in very deep exposures, it counteracts the effects of the high-pressure nervous syndrome (HPNS) better than does nitrogen, and there is the possibility of using bacterial enzymes to metabolize the hydrogen in the body. An earlier, but currently less important, advantage was that hydrogen is more readily available than helium, but in most locations today the handling expenses more than offset the initial cost differential. Hydrogen is narcotic, and because of its solubility it does not move out of tissues as fast as helium does during decompression.

ARGON (MOLECULAR WEIGHT, 40; DENSITY, 1.6 g/L)

Argon is even denser than nitrogen and is more narcotic. It is more soluble and hence not favorable for decompression. It is found in air and in some gas mixtures made from air separation, and it is used in underwater welding. Argon's thermal properties make it a better insulator than air and especially better than helium, and it is therefore used in dry suits by technical divers.

ELIMINATION OF NARCOSIS

Narcosis is discussed in Chapter 11. Although helium was first suggested as a diving gas because of its lower solubility and hence presumed benefit to decompression,[2] it was soon realized that helium was not

narcotic, and this became the primary reason for its use.

Until the mid-1930s, operational air diving had been limited to depths shallower than 50 to 60 m because of the effects of narcosis.[3] The first significant dive with helium was in 1937 by Max Gene Nohl under the physiologic guidance of Edgar End.[4] Helium came into its own during the salvage of the submarine *Squalus,* which sank in 1939 in 75 m of water.[5] Navy divers rescued the surviving crew and recovered the submarine using a surface-supplied helium-oxygen mix; it is universally accepted that this job could not have been accomplished using air alone.

Since this early experience with helium, human exposures to simulated depths of nearly 700 msw (approximately 2280 fsw) have been accomplished with heliox mixtures,[6] and in the deepest exposure so far, a single diver attained a depth of 701 msw in a chamber with mixtures including some hydrogen.[7] Extensive research on the effects of these exposures has been conducted (for examples, see Chapters 7, 11, and 18).

The absence of a narcotic effect from helium has unmasked a new diving disorder —HPNS—that appears to result from the direct effects of pressure on excitable nerve cells. HPNS is manifested most likely as a breakdown of inhibitory functions revealed by a complete lack of a narcotic effect from helium (see Chapter 11 for a further discussion of HPNS). It can be somewhat alleviated by including a narcotic gas (nitrogen or hydrogen) in the breathing mixture.[8] Hydrogen appears especially useful in this regard, providing more effective relief than does nitrogen at pressures in the range of 45 atm,[9,10] and it appears to relieve the "no joint juice," or stiff joints, of hyperbaric arthralgia to some extent.

It is well established that helium does not lead to narcosis at any pressure. Neon also does not induce narcosis up to pressures as high as 37 atm, and therefore, as a diving gas, it can be considered to be non-narcotic.[11] Argon is considered to be about twice as narcotic as nitrogen,[12] but this may be an overestimate.[13] Hydrogen has been found to exhibit a distinctive narcotic potential at high pressures, at possibly one fourth or one fifth the potency of nitrogen; this is a definite limitation to its use in very deep diving. The threshold for operational exposure to hydrogen-oxygen mixtures is about 20 atm,[14] but these mixtures are not usable at partial pressures less than about 7 atm because of the need to keep the oxygen fraction low enough to stay below flammability limits. With no mention of physiologic mechanisms, performance tests and descriptions by divers suggest that the narcosis of hydrogen is different from that of nitrogen. Whereas nitrogen affects the rapidity and precision of movement, with the narcosis resembling alcohol intoxication, hydrogen acts on the intellect and is more like hallucinatory drugs.[9,14]

Oxygen probably has about the same narcotic potency as does nitrogen, as deduced from the properties of these gases, but the amount of oxygen that may be present at the cellular site of the narcosis cannot easily be determined. The two gases seem to behave about the same.[15,16] Technical trimix divers, who generally finance their own dives, are conscious of the cost of the helium component of their bottom gas mix and therefore dilute it with nitrogen. They normally make mixes that contain just the amount of helium necessary to relieve narcosis. Because the intensity of nitrogen narcosis varies widely among individuals, the optimal composition of these mixtures is usually determined empirically. A myth that has evolved among divers and promoters of oxygen-enriched air is that, in analogy to decompression, only the nitrogen partial pressure of a mix needs to be considered in planning the expected level of narcosis. A more conservative and probably more reliable method is to predict the narcosis of a mix by comparing the partial pressures of the combined nitrogen and oxygen components of the mix with the equivalent air depth or pressure, thus accounting for the possible narcotic effects of oxygen. It was to avoid the dangers of narcosis during stressful dives to caves and wrecks that some safety-conscious divers began to add helium to their breathing mixtures, leading to the development of technical diving.

DECOMPRESSION

Before discussing the role of gases in decompression, it is pertinent to review, from an operational perspective, the two major diving categories that are related to decompression patterns, namely, saturation diving and short-duration nonsaturation diving. The second type of diving is also called "bounce," "stage decompression," or "subsaturation" diving. These are discussed more

specifically in relation to gas properties later in this chapter.

Decompression Patterns

SATURATION DIVING

"Saturation," when referring to decompression, means that a diver has taken up enough gas so that more time at pressure does not add to the decompression obligation. In other words, saturation decompression is independent of the bottom time and therefore is only a function of the bottom depth and the gas mixture. In practice, a diver is saturated in less than 24 hours, but certain procedures can detect a difference between 24 and 48 hours of exposure to pressure. Divers in saturation live in a chamber or sea floor habitat and excurse to the worksite, usually without a significant pressure change. Decompression from saturation may take from a third to nearly a full day per 10 msw of ascent, depending on the gas mix and the starting depth, and the pattern may be a linear "slow bleed" or may take place in small steps or stages.

NONSATURATION DIVING

The most common decompression pattern is direct ascent to surface pressure without stops using a profile designed not to require stops. Most recreational and many commercial dives are carried out this way. These are called "no-decompression" or "no-stop" dives; the latter term is used because all dives involve decompression even if they might not require stops. Other nonsaturation dive patterns include "stage" decompression, in which the diver makes one or more stops on the way to the surface; these stops may be done in the water or in a chamber and may involve various techniques. One of the more common types of stage decompression is surface decompression, which involves a transfer from the water into a deck decompression chamber at the surface, where the decompression is completed.

Role of Gases in Decompression

The current practice for preparing decompression tables by most practitioners is to calculate decompression on the basis of the inert gas partial pressure and more or less to ignore the oxygen, but others feel it is sometimes necessary to account for the oxygen. The role of oxygen also depends on the decompression profile. The role of inert gases in decompression has been analyzed by Weathersby and colleagues.[17,18]

For bounce diving, the predominant gas effect on decompression is the oxygen level, with inert gas properties being of secondary importance. In saturation decompression, the nature of the inert gas is the primary factor dictating the speed of the decompression, but ascent rate is also proportional to the starting depth and the P_{O_2} in the breathing mix.[19] The reason inert gas plays a greater role in saturation diving is that the exposures are so long that oxygen toxicity sets upper limits on the P_{O_2} level that can be used. These effects are well established empirically and clearly demonstrable. Saturation decompression with helium as the inert gas takes about one third the time that it does with nitrogen.

The inert gas effects are different in short-duration diving. Helium tends to be taken up more rapidly than nitrogen and there is then a lot of it to unload, and thus short dives with nitrogen as the inert component have shorter decompressions. An inert gas switch from helium to nitrogen can improve the decompression from a heliox dive. The oxygen level in both the bottom gas and the intermediate decompression gases is important in achieving optimal decompression. High oxygen content results in faster decompression but must be kept within tolerance limits, of course. Interestingly, when calculated with the same risk factors, decompressions that are made shorter by the use of oxygen also tend to be more reliable. The probable explanation for this is that the shorter exposure time involves less chance for bubble formation, but less time during decompression also allows less gas to build up in the tissue.

Isobaric Counterdiffusion

In an experiment by scientists from Duke University conducted at the United States Navy Experimental Diving Unit in Washington, D. C., divers saturated with and immersed in normoxic helium-oxygen at 7 atm began to breathe a normoxic oxygen-nitrogen mixture. In a few minutes, they began to itch and

experienced a rash similar to skin bends. This was at first thought to be due to gas osmosis.[20] In a subsequent experiment at the University of Pennsylvania, gas-containing skin lesions developed in divers exposed to pressures up to 37 atm when breathing mixtures containing nitrogen or neon while saturated in a helium-oxygen environment.[11] These skin lesions did not form when divers were placed in a sealed suit and surrounded by the same gas as the one being breathed or when the skin was covered with foil to exclude the external helium.

Graves and colleagues[21] reported further on this phenomenon and hypothesized the diffusion kinetics responsible for the effect on the skin, leading to the now-established term *counterdiffusion.* This effect occurs with two gases having different diffusion and solubility coefficients; the rapidly diffusing gas moves into the tissue, whereas the more slowly diffusing (or more soluble) gas does not move out as fast, resulting in a local supersaturation. This "superficial inert gas counterdiffusion" depends on gas diffusion through the skin and occurs when a subject immersed in a lighter, more rapidly moving gas breathes a heavier, more slowly diffusing gas. This leads to lesions in the skin and possibly vestibular lesions. Similar counterdiffusion apparently occurs in the inner ear, but this has not been demonstrated experimentally.[22]

Another category of counterdiffusion, deep tissue counterdiffusion, occurs in tissues that may not be exposed to external gas and depends on tissue perfusion to supply and remove inert gas. It results from switching of the breathing gas from a heavier or more soluble gas to a lighter one. Some gas switches that cause supersaturation lead to formation of gas phases while the diver is under pressure; examples are air to helium, hydrogen to helium, and, in experimental animals at 1 atm, nitrous oxide to helium.[23] The subject has to be relatively loaded with the heavier gas. This can lead to a condition that is essentially the same as clinical decompression sickness.

The Hydra V experiment by Comex showed that the narcotic properties of hydrogen would counteract many HPNS symptoms during compression to 46 atm. During decompression from "hydreliox" (a trimix of oxygen, helium, and 55% hydrogen), divers switched to a helium background gas and promptly developed Doppler-detected bubbles and "niggles" and therefore had to undergo recompression treatment. Subsequent decompression involved slower switching to allow equilibration, thereby avoiding this problem.[10]

In operational diving, gas switches suspected of causing a counterdiffusion problem have to be accompanied by a small pressure increase to avoid supersaturation.[24–26] The same measure applies to treatment of gas lesions; in treating a diver soaked with nitrogen, it is advisable when switching to helium to compensate for counterdiffusion by compressing at the same time.

Despite these well-established results, many experienced divers are still suspicious that any gas switch may predispose to decompression disorders. Figure 6–1 shows the theoretical buildup of supersaturation after a switch, offering a possible mechanism for gas-phase formation in tissues during switching of gases at fixed ambient pressure.

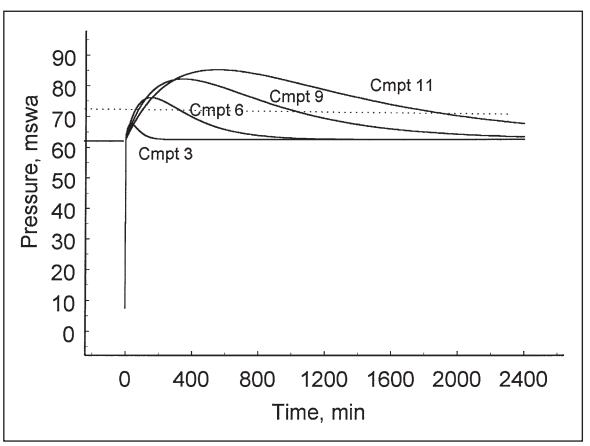

Figure 6–1. Demonstration of counterdiffusion. The sum of calculated tissue gas supersaturation after switching the breathing gas from saturation with 90% nitrogen at 60 msw (70 mswa) to 90% helium at time zero. Compartment half-times used for nitrogen and helium, respectively, are as follows: compartment 3–25, 20; compartment 6–145, 80; compartment 9–385, 160; compartment 11–670, 240. The *dotted line* shows the M-value (maximum tolerable supersaturation) for compartment 6 at 60 msw; slower compartments are slightly lower. This predicts that bubble formation is likely within 2 or 3 hours after the switch. The partial pressure is expressed in meters seawater absolute (mswa). Cmpt, compartment.

OXYGEN IN MIXED-GAS DIVING

The ability to control oxygen in a breathing mixture is a major incentive for using mixed gas. This requires a consideration of the physiologic actions as well as the toxicity of oxygen.

Benefits of Oxygen

More than half a century ago, Lambertsen[27] showed the benefits of oxygen in mixed-gas diving, these benefits are still under study. Oxygen can replace the inert gas in a diver's breathing mixture, and because the evolution of inert gas is the cause of decompression problems, judicious use of oxygen can improve decompression.

Behnke[28] coined the term *oxygen window.* The gradient tending to remove inert gas is caused by the metabolism of oxygen in body tissues and thus allows "isobaric" decompression. This was called the "partial pressure vacancy" by Momsen[29] and "inherent unsaturation" by Hills.[30]

The fundamental concern about mixed-gas diving, whether saturation or bounce, is to have the right oxygen concentration. Too much or too little oxygen can be fatal. A striking number of fatal accidents among divers using mixed gases are due to improper oxygen concentration. During one period of several years of intense commercial diving activity in the North Sea, more than half the fatalities were caused, in some way, by a diver's breathing a gas mix with the wrong amount of oxygen for the situation—often too little oxygen or none at all. A predominant cause of fatalities in technical diving is too much oxygen in the breathing gas for the current pressure. In both commercial and technical diving situations, it appears that the overwhelming problem is using the wrong mixture rather than defining, mixing, or analyzing it incorrectly, but these factors are important as well.

Although the basic role of oxygen is the same in both bounce and saturation diving, oxygen is effective in different ways. In short-duration bounce diving, almost all efforts to improve decompression involve manipulating the oxygen, with only modest benefits being derived from changing the inert gas. In saturation diving, the inert gas is usually fixed and not changed during a given operation but the specific inert gas used has a profound effect on the dive. The oxygen level must range within rather narrow limits, but the level chosen does have a strong effect that is well correlated with the efficiency of the decompression.[19]

In air diving, the fixed oxygen fraction (F_{O_2}) limits the possibility for manipulating the oxygen. However, air diving can be improved by the diver's breathing oxygen during decompression. This has been in practice for some time—long before computers made it relatively easy to generate custom decompression tables—by the use of tables such as the United States Navy Exceptional Exposure tables for air,[31] with the substitution of oxygen for air during the later, shallower stops (this is best done at 6 and 3 msw because at 9 msw oxygen is too toxic). This tactic can make these otherwise risky tables quite reliable. More recently, specific tables have been prepared for use of in-water oxygen.[32,33] Yet another way that oxygen can be used in air diving is to breathe it during the interdive surface interval to improve decompression during a repetitive dive.[34]

For open-circuit diving with fixed mixes, wherein the F_{O_2} can be controlled, the most prevalent and efficient method is to use an optimal level of oxygen in a bottom mix otherwise designed to minimize narcosis, to switch to one or more intermediate mixtures higher in oxygen (air may be one of these), and then to switch again to pure oxygen. Switches during ascent are beneficial because with a constant fraction of oxygen in a mixture, the P_{O_2} decreases as the diver ascends to lower pressures. Intermediate mixes may also involve a change of inert gas followed by in-water oxygen or oxygen in a deck chamber as surface decompression. For commercial operations, practical matters tend to dictate the mixtures used, because having a consistent mix may be much more important operationally than a small shortening of decompression time. For example, a widely used but still proprietary set of commercial decompression tables designated *Oceaneering Alpha* is designed to use a single mix—10% oxygen in helium—over a wide range of bottom depths (30 to 120 msw or 100 to 400 fsw) and a single intermediate mix of 50% oxygen and 50% nitrogen. Bottom mixes higher in oxygen can be used as long as they do not exceed the

company's oxygen exposure limits, and with the same tables these mixes provide greater conservatism but not necessarily faster decompression.

Practices leading toward more complex operations include those for which specific custom decompression tables are generated. These tables normally would involve mixtures chosen to be optimal, and tables would be generated specifically for the particular dive or operation. The decompression pattern may be similar to the one described in the previous paragraph, with a bottom mix, one or more intermediate mixes, and then oxygen, but by using more mixes, greater oxygen efficiency may be realized. The intermediate mixtures are selected to keep the oxygen level maximal but within the tolerance limits. This technique, which has its roots in commercial diving, has been used in operations such as deep cave exploration in which many tanks of gas are needed; because several tanks are needed anyway, the cost of varying the mixtures becomes less burdensome.

Another innovation is the oxygen-controlled rebreather. Several contemporary rebreathers, including those used by the United States Navy, control the oxygen to a set partial pressure, thus making it possible to provide a near-optimal oxygen level throughout the dive. Some rebreathers are especially efficient because they monitor the oxygen level and compute the optimal decompression with a built-in dedicated dive computer.

One other application of high-oxygen exposures with mixed gases is for the treatment of decompression disorders; these are covered in detail in Chapter 10.

Hypoxia

Overwhelmingly, the greatest hazard pertaining to oxygen in mixed-gas diving is not having enough of it. Although hypoxia is not a concern for most divers or a major problem in air diving, mixed-gas diving inevitably introduces the possibility of a diver's getting a mixture without adequate oxygen. This can result from breathing the wrong mix or from breathing the right mix at the wrong pressure. Rebreathers are notorious for allowing hypoxia to develop without warning.

As an example of hypoxia, a 10% mix might make some divers dizzy if breathed at surface pressure, but its partial pressure is adequate at pressures beyond 2 atm. An hypoxic situation can arise when a saturation chamber is decompressed without the addition of extra oxygen, as might occur if a dive is aborted before the divers are saturated. For example, for a 300 msw dive (a pressure of 31 atm), in order to have a storage P_{O_2} of 35 kPa (0.35 atm, the equivalent of 35% oxygen at sea level), the fraction of oxygen in the chamber at maximum pressure would be a little over 1%. This would be an inadequate level of oxygen at any pressure less than about 10 atm. As a countermeasure, the practice among divers in the North Sea is to always have a small amount of oxygen in gases that are taken offshore—even 2.5% oxygen is enough to keep a diver alive and generally conscious at a pressure greater than 3 or 4 atm.

Hypoxia is a real threat. It can cause debilitation, unconsciousness, and even death if extreme enough. An insidious aspect of hypoxia is that it tends to make the victims euphoric and unconcerned about their welfare.

Central Nervous System Toxicity

The techniques for avoiding central nervous system (CNS) toxicity are straightforward in most cases of commercial and military diving: Keep the oxygen level low enough to prevent any reasonable possibility of convulsion. In general practice, this is a P_{O_2} level of less than 1.4 to 1.5 atm. Commercial divers and some military divers have a decent chance of surviving a convulsion because they wear full-face masks or helmets that remain in place during a convulsion and they have communications and lifelines and can therefore usually be rescued with little risk.

Untethered technical divers using scuba equipment are at much higher risk for CNS oxygen toxicity. For one thing, they think that they must do whatever they can to minimize decompression time because of the limited gas supply, and one way to accomplish this is to raise the oxygen level, thereby increasing the oxygen exposure.

Furthermore, these divers are untethered, they have no communications, and worst of all from the standpoint of CNS toxicity, they breathe through a mouthpiece. The first event occurring during a convulsion is an

Table 6–2. NOAA oxygen exposure limits

P_{O_2} (atm)	Maximum Single Exposure (min)	Maximum per 24 hr (min)
1.60	45	150
1.55	83	165
1.50	120	180
1.45	135	180
1.40	150	180
1.35	165	195
1.30	180	210
1.25	195	225
1.20	210	240
1.10	240	270
1.00	300	300
0.90	360	360
0.80	450	450
0.70	570	570
0.60	720	720

These limits are appropriate for managed diving situations, but the upper limit (1.6 atm) is not recommended for untethered divers breathing by mouthpiece (a maximum of 1.4 atm is preferred). A series of repetitive dives may be accumulated within a single limit. If the single exposure limit is exceeded, a 2-hour wait is recommended. If the daily limit is exceeded, a 12-hour wait is recommended.
Data from Joiner JT (ed): NOAA Diving Manual: Diving for Science and Technology, 4th ed. Silver Spring, Md., National Oceanic and Atmospheric Administration, 2001.

evulsive movement of the tongue and jaws, which invariably causes the diver to expel the mouthpiece. Even if another diver attempts rescue, it is generally impossible to reinsert the mouthpiece until the convulsing diver begins to breathe again—but in this case, the breathing medium will be water. There have been some dramatic rescues of convulsing divers, but there have also been many fatalities. Successful rescue is much more likely if the diver is wearing a full-face mask or is near the surface, or both.

The exposure guidelines technical divers use to manage oxygen toxicity are derived from those published in the *NOAA Diving Manual* (Table 6–2).[35,36] The upper range of these limits (e.g., an allowed exposure of 45 min at 1.6 atm P_{O_2}) is appropriate for tethered and helmeted divers or for divers doing very light work, but from recent experience, these limits pose too much risk for untethered divers breathing through a mouthpiece. Thee divers generally use a maximum P_{O_2} of 1.5 or, even better, 1.4 atm. Interestingly, the decompression penalty on most technical dives for such a reduction of P_{O_2} (from 1.6 atm) is only a few minutes. Because the limits are for specific times and P_{O_2} levels, divers have learned to interpolate depth, time, and P_{O_2}, keeping records of their "CNS %" or "oxygen limit fraction," the portion of the published limit that has been reached.[37] There is no research basis for the method of interpolating between limits, but it has apparently worked in extensive field experience. Despite these misgivings, a workshop representing a broad sector of the relevant diving community has endorsed the 1.6 atm limit.[38]

Another approach is that of Harabin and colleagues,[39] who used maximum likelihood statistics to predict CNS toxicity. Using data mostly from exposures to pure oxygen in the water, their analysis shows that risk increases nonlinearly as a function of oxygen level and time of exposure, with the risk increasing sharply with oxygen levels above threshold values and being significantly attenuated by intermittent exposure. In a report based on work with animals and some data from humans, Arieli and colleagues derived a simple relationship based on the square of time and a power function of P_{O_2}, of the following form:

$$K = t^2(P_{O_2})^c$$

where K is a cumulative oxygen toxicity index such that symptoms appear when the index reaches a certain threshold level, and c is a variable determined from the data.[40] This equation can be applied to both pulmonary and CNS toxicity.

Whole-Body or Pulmonary Toxicity

The other types of oxygen toxicity of concern to divers are all included here in a category known as *whole-body toxicity*. When an exposure to hyperoxia (here this means to a P_{O_2} level > ~50 kPa or 0.5 atm) is low enough not to cause convulsions, the exposed person will, in time, experience a variety of other symptoms. These occur mostly during multiday oxygen-intensive diving operations or saturation-type exposures. As described in Chapter 12, the main manifestation is in the lungs, and this affliction has traditionally been called *pulmonary oxygen toxicity*. There are other symptoms, with a syndrome of vague conditions developing after several days of exposure, sometimes in divers showing little or no pulmonary manifestation. Nonpulmonary symptoms include headache, nausea, lack of

aerobic capacity, paresthesias, and general malaise, leading to the term whole-body toxicity.

Pulmonary effects, specifically the reduction of vital capacity, nonetheless are the primary measure of whole-body toxicity. In the early 1970s, Dr. Lambertsen's laboratory developed a mechanism for monitoring lung toxicity. The method uses a unit derived from a curve fit to empirical data, which was labeled the *unit* or *cumulative pulmonary toxicity dose* (*UPTD* or *CPTD*).[41,42] Without the need to identify or distinguish between these, most current practitioners of oxygen tolerance techniques just call these *oxygen tolerance units (OTUs).* The equation for calculating OTUs is the same as for UPTDs:

$$\text{OTU} = t\left(\frac{PO_2 - 0.5}{0.5}\right)^{0.83}$$

where t is the duration of exposure in minutes, P_{O_2} is the oxygen partial pressure in atm, and 0.5 is the exposure threshold in atm, below which effects are negligible.

The Lambertsen method, as published, provides a "unit," but it functions within narrow operational limits. It does not explicitly provide for multiday exposures or for recovery. A more recent project sponsored by the National Oceanic and Atmospheric Administration (NOAA) to explore repetitive excursions from saturation produced a set of mission-related guidelines for monitoring

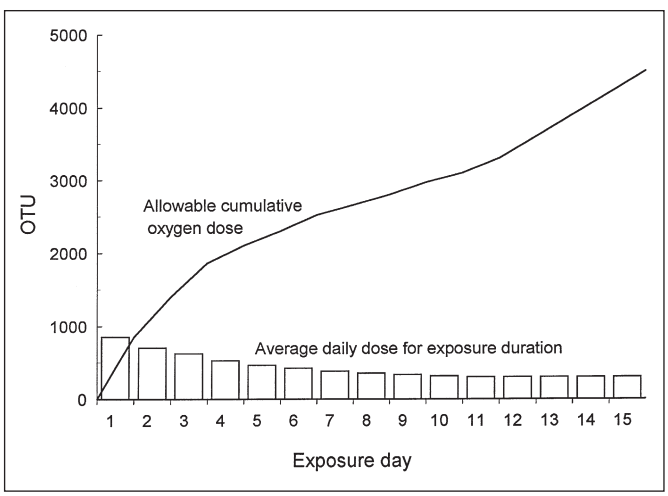

Figure 6–2. Repex whole-body operational exposure limits. For a diver starting fresh, the daily exposures in oxygen tolerance units (OTUs) are totaled and compared with the curve for allowable exposure.[43,44,46] Divers whose cumulative oxygen dose falls below the curve can normally avoid all but mild, operationally acceptable symptoms. Recovery takes place when exposure is to less than 0.5 bar P_{O_2}. Recovery rate is about 300 OTUs/day. See also Table 6–3. (Redrawn from Hamilton RW, Kenyon DJ, Peterson RE: Development of decompression procedures for undersea habitats: Repetitive no-stop and one-stop excursions, oxygen limits, and surfacing procedures. *In* Bove AA, Bachrach AJ, Greenbaum LJ Jr [eds]: Underwater and Hyperbaric Physiology IX. Bethesda, Md., Undersea and Hyperbaric Medicine Society, 1987.)

Table 6–3. Repex Operational Oxygen Exposure Limits

Duration of Exposure (Days)	Daily Exposure Limit (OTUs)*	Total Exposure Limit (OTUs)
1	850	850
2	700	1400
3	620	1860
4	525	2100
5	460	2300
6	420	2520
7	380	2660
8	350	2800
9	330	2970
10	310	3100
11	300	3300
12	300	3600
13	300	3900
14	300	4200
15–30	300	As required

*The daily limit is based on overall number of days of exposure. The center column shows the average daily limit for the number of days indicated, provided this does not exceed the stated total in column 3.[46] Also see Figure 6–2.
OTUS, oxygen-tolerance units.

oxygen tolerance. This method, designated by the project name *Repex,* showed that exposure could be monitored for a multiday mission and that empirical limits for each day of a multiday exposure to hyperoxia could be used to predict a trouble-free exposure.[43–45] Tables simplify the calculations,[46,47] and oxygen tolerance limits can be evaluated with a graph (Fig. 6–2) or numerically (Table 6–3). These limits do not directly consider CNS toxicity.

Using a larger database than that used by Lambertsen's team, Harabin and colleagues produced a useful linear algorithm for predicting the vital capacity decrement resulting from oxygen exposure.[48]

Oxygen as an "Inert" Gas

Although the beneficial role of oxygen in diving is as a replacement for inert gases, under certain circumstances it may act as an inert gas itself. Oxygen, no doubt, is a component of gas bubbles in the body, but this is usually at a level too low to be of major significance. However, when oxygen is in excess of the concentration that can be immediately consumed by the tissue, it may be necessary to plan for the oxygen component when calculating decompression tables. This is mentioned again later in the discussion of oxygen-enriched air.

SATURATION DIVING

As mentioned earlier, a saturation dive may require a lengthy decompression but one that is independent of "bottom time," the time spent at maximum pressure or depth. The practice of saturation diving is intimately tied to the types of gases and the pressures used. Saturation diving techniques are used for almost all commercial and military diving at depths beyond those easily accessible by bounce diving (which may routinely extend to 100 msw). Operationally, the saturation range depends on the specific task, the environment, the job duration, and other factors in addition to depth; in practice, for petroleum exploration and production, most offshore dives deeper than the air-diving range of 50 to 60 msw are normally done by saturation, but the technique may be used at depths as shallow as 20 msw if dictated by job conditions and the equipment is available. In the developmental stages of the offshore oil diving industry, dives to as deep as 200 msw were made as bounce dives with stage decompression, but the risks were high, even for the few companies that undertook such dives (the incidence of DCS was high); clients (the oil companies that hired the diving services) soon learned that they were better off paying for saturation dives and avoiding the operational uncertainties—and frequent lawsuits—brought on by subsaturation dives to these depths.[49]

Saturation divers live on the deck of a support ship in a chamber that has a sophisticated life-support system to maintain precise conditions of pressure, gas mixture (particularly P_{O_2}), temperature, and humidity and that also provides other essential amenities such as bunks, a shower, a toilet, and food. Meals and supplies are passed in and waste is passed out through a small transfer lock. The living chamber is usually maintained at a "storage depth" (i.e., at a pressure equivalent to a depth) that is almost identical to the depth of the worksite, and the divers therefore need little or no pressure change when they go to work. A pressurized transfer chamber—normally called a *bell* but more formally called a *submersible decompression chamber* or *personnel transfer capsule*—is mated to the living chamber and delivers divers to and from the worksite at constant pressure (Fig. 6–3). Many details of saturation and other commercial diving operations are covered in the monograph by Lettnin.[50]

Saturation Diving with Heliox

An important advance in diving technology resulted from the work of U.S. Navy doctor George Bond and coworkers, who conducted the earliest saturation experiments with helium-oxygen mixtures.[51,52] These investigators determined that oxygen was the toxic element in long exposures to compressed air, and they demonstrated that divers could spend prolonged periods (weeks) under pressure without serious physiologic changes if the P_{O_2} was maintained within the normal range. Currently, working saturation dives are routinely conducted at depths as shallow as about 30 msw and to depths routinely over 300 msw in the open sea; these may last for up to 3 to 4 weeks with the use of techniques developed initially by the U.S. Navy and expanded by commercial companies.

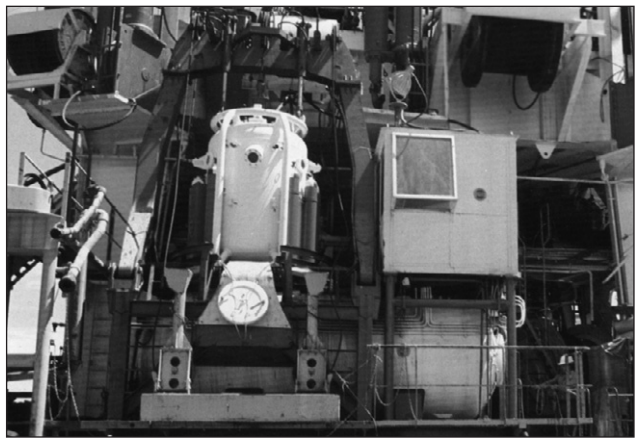

Figure 6–3. Saturation diving system, or deep-diving system. The bell is shown with the handling system that swings it over the side and lowers it on guide wires to the worksite. The living chamber with some of its piping is below the bell and to the right. (Courtesy of International Underwater Contractors, City Island, NY.)

Maintaining a normoxic P_{O_2} atmosphere (0.3 to 0.4 atm oxygen is normally used, which is actually a little higher than the sea level value) requires that the oxygen percentage be held to a tolerance of about ±0.10% to avoid hypoxia and oxygen toxicity. Figure 6–4 shows the typical percentage of oxygen in heliox for various depths. To get the desired P_{O_2} of 0.35 atm at 300 msw requires a mixture with an oxygen concentration slightly greater than 1%, with a tolerable range between 0.65% and 1.50%. Normally, the mixture is blended in the chamber during initial compression by selective addition of air and helium and is maintained by replacing metabolically consumed oxygen. In some early operations, it was considered necessary to minimize the nitrogen content, but the benefits of low levels of nitrogen are now generally accepted. Accurate analysis and constant surveillance of the oxygen level are necessary.

Sometimes vertical excursions are made to depths deeper or shallower than the habitat. In normal practice, divers travel only the vertical distance that can be managed on a no-stop basis; the United States Navy has developed "unlimited duration" no-stop excursion tables for saturation depths to 300 msw.[53,54] Timed excursions, which would combine the techniques of saturation and bounce diving, offer good prospects for improved operational efficiency, but their offshore use has not been extensive. More extensive excursions are in limited use because of lack of knowledge of effective techniques[55–57] and because excursions may consume extra gas to pressurize the diving bell. Once they are committed to a saturation dive, operators think that there is little benefit to maintaining a lower storage pressure, even though a lower pressure is inherently safer and the final decompression is shorter. Large excursions associated with shallow saturation storage depths are an attractive but relatively undeveloped option for shorter jobs. Vertical excursions are well developed for nitrox habitat diving (see later).

The Helium Environment

Several unique problems result from the helium component of the saturation gas. Because it is a small molecule, helium is highly diffusible and can penetrate pressure seals not affected by nitrogen. Electronic parts, cables, vacuum tubes, and pressure-proof watches are examples of equipment that has been damaged by penetration of helium (one of the Rolex watches made for deep diving even has a tiny valve to let helium out). Helium has been found to diffuse through glass into cathode ray (television) tubes. However, design modifications of the equipment to be used in heliox environments, which often effectively eliminate exposure of the item to helium, have solved most of the problems arising from helium diffusion.

A superficially amusing but actually quite troublesome problem with a helium atmosphere is the change in voice characteristics caused by the properties of helium.[58] The high-pitched cartoon-character quality of the voice causes voice communication with divers to be difficult at best and in many cases impossible. Electronic voice unscrambling has improved communications

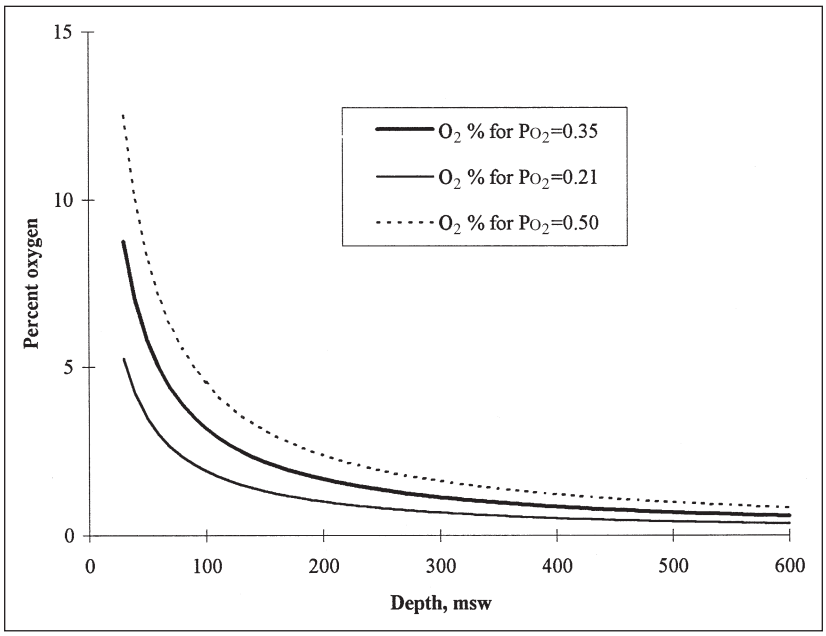

Figure 6–4. Range of oxygen concentrations for deep saturation diving. The *heavy solid line* represents the oxygen concentration needed to maintain 0.35 atm, a common choice for P_{O_2}. The *lower line* represents the oxygen concentration needed to maintain the normoxic level of 0.21 atm. The *dotted line* represents the upper limit of continuous exposure to avoid whole-body toxicity, 0.5 atm. The low oxygen concentrations needed are difficult to mix and analyze within acceptable tolerance limits, and they are therefore usually mixed as the chamber is being pressurized.

significantly. Unscramblers reconstruct normal voice characteristics by frequency filtering and spectral shifting. The resultant voice, although not ideal, makes it possible for divers to communicate with their support teams and each other.

Helium has a high thermal conductivity, generally characterized as being six times that of air. As a result, divers living in a heliox environment suffer an exceptionally high level of heat loss, especially at elevated pressure, because heat loss is increased in denser gas. Even when divers think they are comfortable they may be experiencing substantial heat loss. This leads to an increase in food consumption, yet despite increased intake, weight loss is quite common in saturation divers.[59] The comfort zone between feeling too cold or too hot is very narrow; a thermally neutral temperature (which is influenced by pressure) may be approximately 34°C. In the high-pressure helium atmosphere, food is nearly tasteless and it cools almost instantly, augmenting the problem of maintaining weight; tasty meals can be provided to divers in saturation at high pressures, but with considerable effort.[60]

Another prominent problem of deep diving with helium is HPNS, discussed in detail in Chapter 11. Helium is not responsible for HPNS; the condition is the direct result of the effect of hydrostatic pressure and begins to be noted in excess of approximately 15 atm. However, helium is implicated because it is the inert gas of choice for diving in the pressure ranges at which HPNS is prominent and because its total lack of narcotic properties allows HPNS to be unmasked. Taking advantage of this point, Bennett and colleagues found that adding a narcotic gas (e.g., ~5% nitrogen) to the diver's breathing mixture relieves the tremor of HPNS dramatically in dives to the range of 30 to 45 atm improves diver performance.[8,61] This group calls the resulting mixture *trimix,* but this is not to be confused with the trimix used in short-duration trimix technical diving, which is covered later in this chapter. Hydrogen, also, can ameliorate symptoms of HPNS.[10,62]

Although the condition is not limited to heliox dives, a major medical concern with saturation diving is the prevention of external otitis in divers. This condition, the same as swimmer's ear, can be tenacious and is

caused usually by strains of *Pseudomonas* (a gram-negative bacteria). An infection breaking out in a team of saturation divers can lead to the loss of one or more divers from the work crew for a few days or, at worst, to the dive being aborted. Infection can be prevented by rigorous hygiene, daily prophylactic treatment of the ears with Burow's solution (Domeboro), and monitoring for diagnosis if necessary. Treatment for established infection consists of topical use of strong antibiotics such as polymyxin and gentamicin.

Nitrox Saturation and Excursion Diving

Saturation diving is also done with mixtures of oxygen and nitrogen. As with helium mixtures, the P_{O_2} in the living chamber must be maintained in the tolerable range between hypoxia and toxicity, and as with helium mixes, this is usually a P_{O_2} of 0.3 to 0.4 atm. To achieve this P_{O_2}, oxygen-nitrogen mixtures with a reduced oxygen percentage are used. These mixtures are called *nitrox.* This was the original use of the term nitrox, which is now also sometimes used to describe oxygen-enriched air mixtures used in diving (these are covered later in this chapter).

For most nitrox saturation diving, the "lockout" or "excursion" breathing gas is air. Although some diving in the air range is done with these mixtures (nitrox in the chamber and air on the lockout) in the manner of traditional saturation diving with heliox, by far the most effective and widely used application for nitrox saturation is for vertical excursions. This concept is most developed in the practice known as *habitat diving.* Divers live in ambient-pressure habitats located on the sea floor. A habitat is filled with gas and open to the sea through hatches on the bottom. Aquanauts live inside the habitat and exit into the water for work. The work may be at a depth different from the habitat, usually deeper, in which case the divers travel to the worksite using procedures for vertical excursions, normally with air as the breathing gas.[63,64] Nitrox saturation-excursion diving is often called *NOAA OPS* (for NOAA operations) diving; the procedures for making no-stop excursions have been published.[65] The NOAA-sponsored project Repex, mentioned earlier, and a follow-up experiment extended the NOAA OPS technique to include deeper and longer excursions as well as excursions with stops.[66] NOAA currently operates the habitat *Aquarius,* which has been located in the United States Virgin Islands and now the Florida Keys for studies of coral reefs.

Interestingly, with air excursions, divers can get enough exposure to oxygen that whole-body or pulmonary oxygen toxicity becomes a limiting factor. Divers diving with air from sea level are not likely to reach oxygen tolerance limits because the need for decompression sets limits on the exposure, but tolerance limits can be reached by divers saturated near the pressure of the worksite. As described earlier, an algorithm for managing the resulting exposure to oxygen was also developed by Repex.

Numerous shallow-habitat saturation exposures have been carried out since the 1960s. Habitat depths for these dives have ranged from less than 10 to more than 40 msw (30 to 140 fsw) and have employed both normoxic nitrox mixes and air.[64] It has been found that air can be used as the atmosphere in the chamber for pressures lower than about 15 msw (NOAA's limit is 50 fsw), but at greater depths, the toxicity of the oxygen in air will, in time, be too high, exposing the divers to whole-body toxicity and thus limiting the tolerable duration of the exposure.[65]

Nitrox mixtures lead to problems with narcosis at habitat depths greater than about 35 msw, and helium saturation diving techniques are commonly used beyond that depth. NOAA has developed procedures whereby divers living in a nitrox habitat can make excursions using oxygen-helium mixes or oxygen-helium-nitrogen trimixes, increasing the effective excursion range to as great as 80 msw.

One of the methods of preparing the habitat mix for a nitrox saturation, the method of "breathing down" the oxygen, is shown in Figure 6–5. The objective with this method is to prepare a mixture of oxygen and nitrogen having a P_{O_2} of about 0.35 atm, starting with a chamber filled with air. The figure shows how the desired mix can be reached by pressurizing the chamber with air and having the divers consume the oxygen in the atmosphere until enough of the oxygen is removed. This example assumes a chamber fixed volume of 10 m^3 at a pressure of 3 atm absolute (20 msw), or 30 m^3 of air, which holds about 6300 L of oxygen. With four resting divers each consuming 0.3 L/min of oxygen for a total of 1.2 L/min, which is

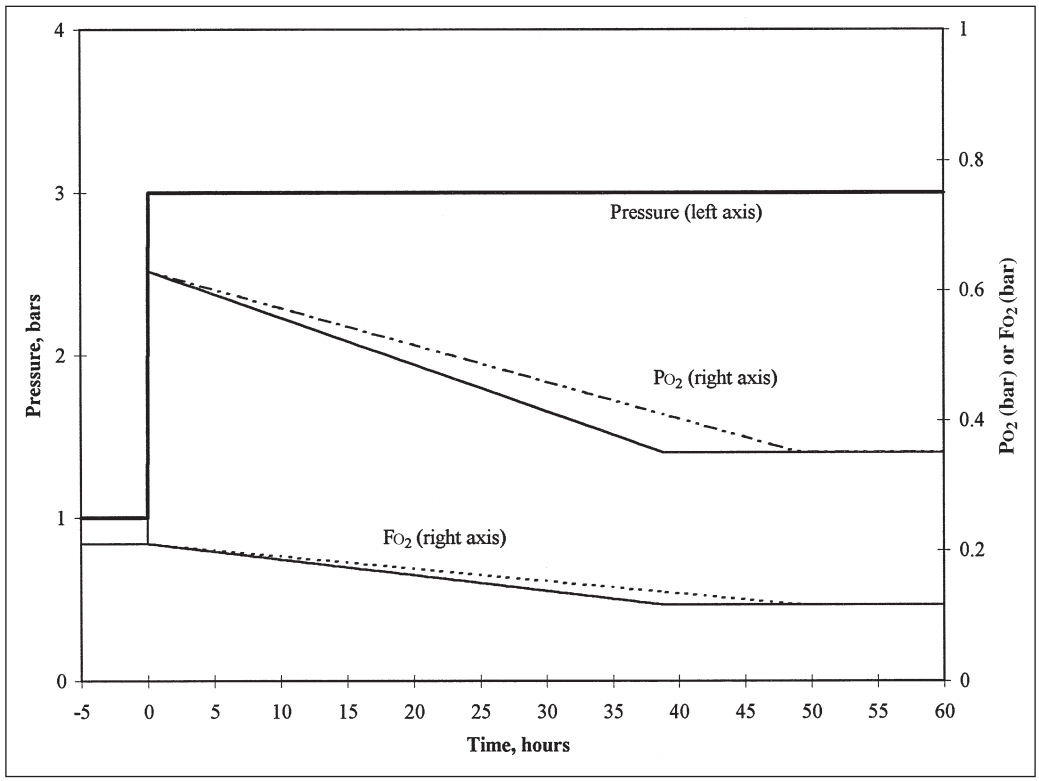

Figure 6–5. The "breathing down" method of preparing a nitrox habitat atmosphere. A chamber volume of 10 m^3 with four divers, each consuming oxygen at 0.3 L/min, is assumed. The *upper solid line* shows the pressure, which increases in a few minutes to 3 atm (20 msw). The *lower pair of curves* show the change in the fraction of oxygen (F_{O_2}) as oxygen is consumed, and the two *center curves* show the resulting P_{O_2}. The *dotted* and *solid lines* reflect replacement of the consumed oxygen with air and nitrogen, respectively.

replaced with either pure nitrogen *(solid lines)* or air *(dotted lines),* the chamber takes 38.9 hours or 49 hours, respectively, to reach 0.35 atm P_{O_2}, which is 11.7% oxygen. Either of these is a modest oxygen exposure. Once the P_{O_2} of 0.35 atm is established, this level of oxygen is maintained by replenishing the oxygen consumed.

A more typical procedure for preparing the nitrox chamber atmosphere is to compress the chamber initially with a small amount of air and to complete the compression with pure nitrogen; the same technique is used with helium. Using the same example, the chamber is pressurized with air to 1.67 atm absolute or about 7 msw. This is the target pressure that will result in a P_{O_2} of 0.35 atm (e.g., 1.67 atm [pressure of air] × 0.21 [F_{O_2} of air] = 0.35 atm [P_{O_2} desired]). The remainder of the compression is with 100% nitrogen so that the P_{O_2} does not change as the chamber is pressurized; the gases have to be well mixed, a matter that can be significant if helium is used for pressurization. One disadvantage of this method, in addition to the slightly greater cost for nitrogen instead of air, is that having a pure inert gas connected to the diving system allows the possibility of a mixture deficient in oxygen being given to a diver to breathe.

Decompression from Saturation Diving

Decompression from saturation is accomplished either with a gradual, more or less linear ascent by a slow "bleed" of the diving chamber or with very small (about 1 msw) stage steps, from the "storage" depth to the surface. The whole crew may undergo decompression from saturation after the work is over, but in some commercial "spreads" with multiple locks, crews are cycled in and out using locks; the living chamber is kept at pressure, and work continues in shifts around the clock or even around the calendar for an overall saturation mission that may last many

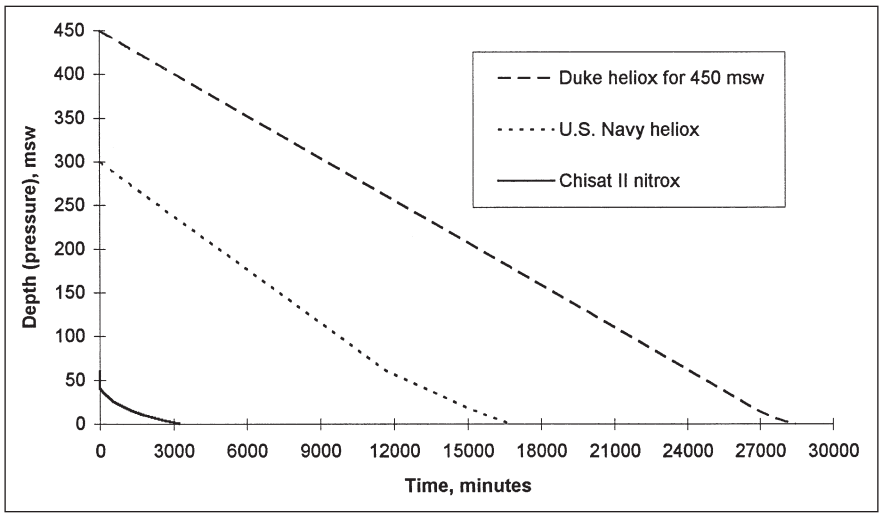

Figure 6–6. Decompression from heliox and nitrox saturation. The *upper curve* is for a heliox saturation dive at 450 msw using the Duke procedures used at GKSS[61] (German Nuclear Energy Research Facility) and requires about 19 days. The *center curve* is a recent U.S. Navy procedure[53] for decompression from 300 msw and requires about 11 days. The *lower curve* is from a Repex table used for the Chisat II decompression from nitrox saturation at 25 msw; it starts at 40 msw following excursions and requires a little more than 2 days[43]; it would start at 25 msw and be 9 hours shorter if excursions did not have to be accounted for.

weeks. During decompression, it is necessary to add oxygen to the chamber in order to maintain the P_{O_2} during the reduction in pressure as well as to compensate for metabolic consumption.

The rate of ascent depends on the inert gases, the P_{O_2} of the breathing gas, and the starting depth. Decompression is much faster if helium is the inert gas than if it is nitrogen; this is illustrated in Figure 6–6, which shows decompression rates of about 3 atm/day for heliox and about 1 atm/day for nitrox.

The deeper a saturation dive is, the slower the ascent rate has to be. This is an empirical observation that is not easily modeled, but the reason could be that gas is coming out of solution to form gas phase during the decompression, and during a longer decompression, more gas can accumulate. Or, the observation could be the result of the probability that in a longer decompression, decompression sickness from a given bubble load is more likely.

Because only low (slightly above normoxic) oxygen levels can be tolerated for the duration involved in a saturation decompression, the oxygen level has a relatively small effect compared with the inert gas. However, the rate of decompression, or "ascent rate," during saturation is quite sensitive to changes in inspired P_{O_2} and, in fact, has been found to be proportional to it. Vann,[19] by analyzing experimental and operational data, found that an acceptable ascent rate for a trouble-free decompression could be determined using a parameter based on the prevailing P_{O_2} (for a given inert gas and starting depth). This has been called the *Vann k factor* and is defined by the equation

$$\text{Rate of ascent (msw/h)} = k\ (\text{msw/hr/atm } P_{O_2}) \times P_{O_2}\ (\text{atm})$$

A conservative Vann k for a 300 msw heliox dive is an ascent rate of about 2.1 msw/h/atm P_{O_2} (7.0 fsw/h/atm).[19] For a typical P_{O_2} of 0.6 atm during ascent, the rate would be 1.26 msw/h. For a 30 msw saturation with nitrox, Repex procedures suggest a Vann k of 1.2 msw/h/atm (3.9 fsw/h/atm).[45]

A number of decompression patterns are available for returning from a habitat dive. The simplest is the one used commercially in which the habitat is actually part of a deep-diving system located at the surface, and the divers go to the worksite or seafloor habitat under pressure in a bell; decompression in the chamber ends with the divers stepping out to sea-level pressure. A variation on this for a sea floor habitat is for the divers to complete decompression to surface pressure in the habitat, transfer into the bell while still at surface pressure, and then go to the surface in the bell at that

pressure; or they transfer under pressure from the habitat to the surface chamber and complete the decompression there. The Hydro-Lab pattern (which lacked a bell), now used by the *Aquarius* habitat, and the Repex procedures have the divers decompressing to surface pressure in the seafloor habitat, then pressurizing back to habitat depth (pressure), at which time they lock out and swim to the surface. Another method usable for relatively shallow habitats is the FLARE (Florida Aquanaut Research Expedition) method, in which the divers leave the habitat, swim to the surface, and undergo recompression in a surface chamber for decompression; this is analogous to surface decompression in commercial diving.[63]

A further complication of decompressing from a habitat dive arises from having to account for recent excursions. If divers return from descending excursions and immediately begin saturation decompression, they are much more likely to experience decompression sickness than if they start from full saturation at storage depth. No good algorithm exists for how much delay is needed to start the saturation decompression in a bubble-free condition that would provide a smooth decompression; hold times at storage depth of 36 to 48 hours are recommended empirically. In one method, used by Repex, the saturation decompression is started at a point (a pressure) that is within the distance covered during the last excursion. This is intended to slow the decompression from the excursion before bubbles begin to form.

A new twist has been given to decompression from saturation dives by research by the U.S. Navy on methods of accelerating decompression from saturation at 3 to 4 atm for rescuing survivors of a sunken submarine. Attempts to speed up the decompression by giving massive amounts of oxygen breathing during the transit proved to be ineffective in eliminating decompression sickness, and the oxygen exposures became limiting. It was found that if the oxygen was breathed prior to the beginning of ascent, decompressions became much more efficient, and even though the oxygen was breathed at a greater depth, oxygen toxicity was less.[67,68] This "prebreathing" is a common practice of high-altitude aviators and balloonists and of astronauts prior to performing extravehicular activities.[69]

SHORT-DURATION MIXED-GAS DIVING METHODS

Short-duration, or bounce, diving includes a variety of methods of nonsaturation diving. It embraces different diving communities, such as commercial, military, scientific, recreational, and technical divers, and it uses different types of equipment, including scuba, surface-supplied, and bell bounce diving equipment and rebreathers.

Surface-Supplied Diving with Stage Decompression

The fundamental type of mixed-gas diving is traditional stage decompression diving, in which the diver—who is usually supplied with breathing gas through a hose from the surface—makes one or more stops on the way to the surface (see Chapter 3). Stops are most often done in the water, sometimes on a suspended platform or stage, but they may be in an "open" bell (open at the bottom) in which the diver is enclosed but at ambient water pressure. Stops are used instead of continuous ascent because stops are operationally easier to perform.

The diver may undergo surface decompression by ascending to the surface after a few in-water stops, then recompressing in a deck decompression chamber to breathe pure oxygen for the remainder of the decompression. Sometimes the diver transfers under pressure to a bell and then to the deck chamber for decompression without the period at surface pressure. Surface decompression has several advantages. The diver is in a controlled chamber environment that prevents loss of body heat, communications are improved, and oxygen toxicity poses minimal risk (also, shark attacks are not a threat). Other advantages are that other divers can be put into the water or the diving-support vessel can break its moor and get underway rather than remaining on station for the total decompression time. The main disadvantages of surface decompression are (1) that bubble growth may be initiated during the surface interval, thus making these decompressions less reliable unless this problem is accounted for in the design of the decompression table and (2) that the time-urgent transfer to the chamber after surfacing may predispose to accidents.

There is no acceptable algorithm for determining the exact penalty for the surface interval (or for determining how long the surface interval can be), but a number of methods account for bubble formation. Available surface decompression tables have not been reliable in North Sea work, partly because the more difficult dives tend to be done with surface decompression techniques; statutory limits on the allowed bottom time were found to dramatically reduce the incidence of decompression sickness and are in current practice, presumably until reliable surface decompression methods become available.[70]

Bell Bounce Diving

Deep nonsaturation commercial diving usually involves a rapid descent to the working depth in a diving bell (see Fig. 6–3), a quick work period, and then a "long pull" to the first decompression stop, which is followed by many other stops and possibly by gas switches performed with the divers in the bell; divers usually transfer under pressure from the bell to the deck chamber for the final stops. This technique is widespread and well developed, with the more technologically advanced diving companies performing such dives to as deep as 200 msw. However, saturation is used more frequently beyond a depth of about 50 msw.

In commercial and military diving, it is paradoxical but understandable that the saturation diver has acquired a higher status than the bell diver; this is probably because the senior divers take the more lucrative saturation jobs, even though the job of the bell diver is much more demanding, requires greater skill, and involves a higher risk. Or, it might be that the impressive equipment spreads and manpower loads required to do saturation diving, as well as its cost and operational effectiveness, cause it to assume greater importance, and diver status follows.

Technical Diving

A new category of diving with special gas mixes began in the late 1980s. Recreational diving was once limited to air, but now a community of advanced sport divers have become even more creative in the use of mixes than even the traditional military and commercial sectors.[71–73] The term *technical diving* describes a category of special-mix diving that, strictly speaking, is still recreational diving—it is considered recreational because the practitioners do it for enjoyment rather than for employment—but it is still a highly disciplined and professional undertaking that does not belong with traditional recreational diving. This is a method of self-contained or untethered diving (i.e., without a gas hose to the surface) that extends well beyond the traditional boundaries of recreational diving; an analogous comparison would be that of technical mountain climbing to hiking. To purists, a technical dive is one that includes at least one change of gas mix during the course of a dive (or it could be a dive with a rebreather apparatus). The term would therefore not normally be used to describe a dive in the air range with a single mix of enriched air, nor would a deep dive on air be considered a technical dive. Others use the term more loosely to describe any dive that is not entirely on air or, in some cases, any air dive that involves decompression stops; such dives are not necessarily technical dives. For the record, from the late 1940s in the United Kingdom, diving with a rebreather was called technical diving, using military or ex-military equipment from the World War II era.

Technical diving involves the use of special breathing mixtures and custom decompression tables, together with specialized technology (Fig. 6–7). Special tanks are employed that are larger than standard, take higher pressures, or both; lightweight titanium tanks that can bear high pressures are available. Special attention is also given to buoyancy control. Divers working in a current or exploring a cave often use battery-powered diver propulsion vehicles, or "scooters," to increase mobility. The lengthy decompression stops are often carried out in underwater decompression stations that may be made of inverted cattle watering troughs or well-anchored air-filled lift bags. Divers with a high oxygen exposure sometimes use full-face masks instead of mouthpieces to improve chances of survival in the event of a convulsion.

Dry suits offer improved thermal protection over traditional wet suits, and these are made even more efficient by filling them with argon, whose thermal conductivity is lower

Figure 6–7. A technical diver shown wearing the complete outfit. Visible are dual back-mounted tanks, dual side-mounted tanks (each of these has a separate regulator, and there is a spare regulator on a long hose), a smaller tank of argon for suit inflation, a reel for managing independent ascent, lights, and a dive computer (mainly for logging), all on a hooded dry suit. Special thermal underwear and an adult diaper are also included with this outfit. (Courtesy of Dan Burton.)

than that of air and especially lower than that of helium. Argon's conductivity is about two thirds that of air, which increases its insulating capacity by about 50%.[74] An argon tank should be rigged so that the gas cannot possibly be breathed, or it should include a low percentage of oxygen. When argon is used in this way, counterdiffusion is not a concern. Some other gases could provide better insulation than argon (sulfur hexafluoride or krypton), but these are too expensive to be practical; carbon dioxide insulates but causes skin irritation.

Open-sea technical divers carry all the gas they will need because they cannot depend on finding staged bottles or even the dive boat. In a current, they may send up a float and carry out decompression while drifting, with the dive boat following along. "Drift decompression" reduces the wind chill effect of current and requires less effort to fight the current (this avoids strenuous exercise during decompression, thus improving decompression reliability).[71,72]

It should be emphasized that some technical dives are high-risk ventures. They do not meet the criteria for occupational safety and are therefore basically unsuitable for commercial diving operations. It should also be stressed that divers contemplating technical diving should be adequately trained and should not even consider such diving unless they have self-discipline and are willing to acquire the necessary training, equipment, procedures, gases, tables, and support to do it correctly.

DIVING GAS MIXES

Divers use many gases and gas mixes, along with a variety of gas-management patterns. These involve the use of air, oxygen-enriched air, heliox, trimix, exotic mixes, or the mixes created by rebreathers.

Air

Air is the basic diving gas. It is not the ideal breathing mixture because of the narcotic effects of nitrogen at increased pressure, its density, the possibility of oxygen toxicity, and its generally unattractive decompression properties. However, air is overwhelmingly the gas of choice for commercial diving in the range of depths to 50 msw. It is most commonly supplied by hose from the surface, and air diving uses any of several decompression patterns, with staged ascent being the most common (perhaps followed by surface decompression). Air, usually with scuba and without decompression stops, is similarly the gas used for most recreational diving and much military and scientific diving. In some cases, the use of oxygen during decompression greatly improves the effectiveness of air as a breathing gas, either in the water or in a chamber. Enriching air with oxygen is another useful technique that is covered in the next discussion.

Oxygen-Enriched Air (Nitrox)

The simplest mixed gas is air enriched with added oxygen. An operational advantage (and some problems) are introduced with the

use of mixtures of oxygen and nitrogen that have an F_{O_2} greater than the 0.2095 normally found in atmospheric air. The advantage is solely one of a reduced decompression obligation. The price for this is the expenditure of special effort in mixing and handling the breathing gas and the increased probability of oxygen toxicity, both prob-lems introducing the need for appropriate training.

The introduction of enriched air to the recreational diving community during the late 1980s created some controversy, but the use of oxygen-nitrogen mixes has a long history. Toward the end of and after World War II, mine-clearance divers extensively used oxygen-rich mixtures of oxygen and nitrogen (via rebreathers). The United States Navy tested many such mixtures, with mixed results. Work by Lanphier[75] showed that the density of such mixes could exacerbate carbon dioxide retention in divers predisposed to retain it, and for that reason he recommended the use of lower-density heliox in rebreathers.[76] Oxygen-enriched air mixtures were used commercially from the 1960s, particularly by Andre Galerne's International Underwater Contractors, but at the time this was a proprietary technique.[77] Galerne's success stemmed from his realizing that a proper decompression table could be prepared by considering only the nitrogen component of the mix, but both International Underwater Contractors and other commercial companies were discouraged by the complexity of the operations with such mixes for normal surface-supplied diving. In one major and quite successful commercial enriched-air project, a Norwegian contractor used a commercial on-line mixer involving over 5000 working dives.[78]

The use of oxygen-enriched air, commonly called *nitrox,* in scuba operations has been highly developed by NOAA for its underwater scientists.[36] A major reason for this success was the continuous blending method developed by Wells and colleagues,[79] a method that prepares mixes accurately and safely; a major advantage of this method is that it minimizes the handling of high-pressure oxygen, which is necessary when mixes are prepared by partial-pressure blending and other methods.[80] More sophisticated methods of "enriching" air by removing some of the nitrogen use physical processes such as selective membranes or adsorption with a molecular sieve (a synthetic zeolite ion exchanger with high surface area).[81] Enriched-air techniques have become available to the recreational diving community, in part because of the publication of both mixing methods and decompression tables in the *NOAA Diving Manual.*[35,36,65]

The introduction of enriched-air diving into the recreational diving community was accompanied by controversy, largely because it was strongly promoted by those wanting to train divers to do it and because several unanswered questions underscored the fact that this was a new technique that the established recreational diving agencies did not understand well.[82,83] These issues have been sufficiently resolved so that all the major recreational diver-training organizations now teach enriched-air diving. Among the issues were:

- Development of commercially available mixing equipment
- Definition of an unofficial standard for the air that was to be mixed with oxygen (the level of condensable hydrocarbons or oil mist should be < 0.1 mg/m^3)
- Widespread availability of suitable enriched air mixes at dive shops
- Acceptance that mixtures with up to 40% oxygen can be used with ordinary scuba gear if the gear is kept scrupulously clean and free of hydrocarbons and silicone greases and is lubricated with oxygen-compatible lubricants[38]
- Availability of computer programs that allow computation of custom decompression tables and of dive computers that can be set to use enriched air
- Availability of training facilities and materials
- Recognition that normal treatment procedures for decompression disorders would work without modification for enriched-air divers (correcting an early misunderstanding)

A consensus community standard for proper operations remains elusive, however.

As mentioned earlier, the only reason for using enriched air is its benefit to decompression. The current practice is to calculate decompression on the basis of the inert gas partial pressure, essentially ignoring the oxygen component.[17,84,85] Some computational models consider the oxygen in the mixture to be an inert gas when it is assumed to be in excess,[86] but some evidence suggests it may not be in excess if the diver stays within reasonable oxygen tolerance limits[17,87]; wide field experience supports the

latter concept. The physiologic effects of oxygen (e.g., causing prominent vascular changes) make it difficult to assess its purely "inert" properties. At partial pressures greater than 2 to 2.5 atm, oxygen's benefits to decompression begin to diminish.

The decompression benefit of oxygen-enriched air can be manifested in two ways: first, breathing an oxygen-enriched mixture and decompressing as if for air makes the dive more conservative; second, the diver gets increased bottom time for no-stop dives or reduced decompression time if stops are used.

In order to exploit the decompression advantages of enriched air, decompression procedures must account for oxygen. The most efficient way to decompress is with tables or dive computers appropriate for the specific mixtures. This works well for those able and willing to prepare and use such tables, but many organizations are not so flexible, and therefore a more traditional method is used. An effective method is to determine the air decompression table that has the same nitrogen partial pressure as the enriched air being used for the dive and to decompress using that table. The procedure for selecting the right table is called the *equivalent air depth (EAD)* procedure. This method is conservative (and consequently not as efficient as it could be) and uses familiar and readily available tables with recognized performance records (the most popular tables are the U.S. Navy Standard Air tables, but other tables can be used). Figure 6–8 illustrates the EADs based on the nitrogen partial pressures of several oxygen-enriched nitrox mixtures at the actual depth. The following equation is used to calculate the EAD:

$$EAD = \left(\frac{(D + 10) \times F_I N_2}{0.79} \right) - 10$$

where $F_I N_2$ = fraction of nitrogen in the inspired mixture or $(1–F_I O_2)$, D = depth in msw, and 10 = number of msw in an atmosphere. For example, using 32% oxygen, 6% nitrogen, and a depth of 30 msw:

$$EAD = \left(\frac{(30 + 10) \times 0.68}{0.79} \right) - 10 = 24.4$$

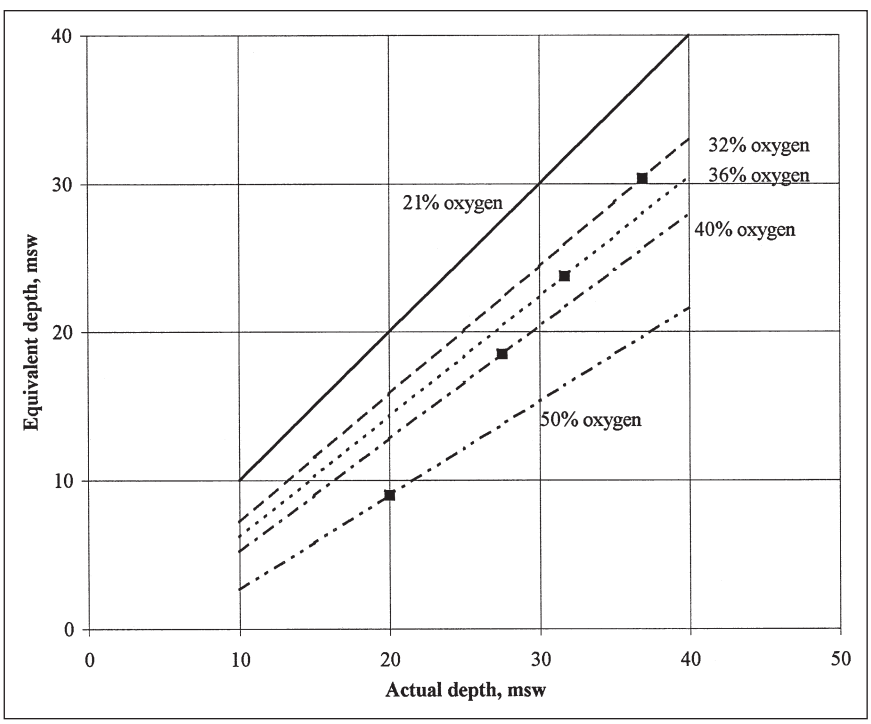

Figure 6–8. "Equivalent air depths" for decompression with enriched air mixtures. The curves show the depth of a dive with air that has the same P_{N_2} as the actual depth indicated. A decompression table for the equivalent depth can be used. The *square markers* indicate the point at which the P_{O_2} reaches 1.5 atm, a reasonable oxygen tolerance limit. Each gas should be used only for the range to the left of the marker; for example, with 50% oxygen, only dives at an actual depth of 20 msw or shallower should be done.

Thus, the appropriate air table for a dive to 24.4 msw should be used for decompression from this 30 msw dive. Using the Defense and Civil Institute of Environmental Medicine (DCIEM) tables (DCIEM, 1992), a 30 min dive with air at 30 msw requires 15 min of decompression; the equivalent 24.4 msw dive uses the 27 msw table, which requires only 11 min of decompression. However, using oxygen-enriched air with 36% oxygen (F_{N_2} = 0.64), one calculates an EAD of 22.4 msw, which allows the 24 msw table to be used for a required decompression time of 5 min, a greater saving.

Occasionally, enriched air is touted as being safer than atmospheric air. One can indeed make the case that the risk of decompression sickness is lower, but at the very low decompression sickness risk levels normally encountered in this type of diving, it is stretching a point to imply that a diver would be safer. The higher levels of oxygen pose added hazards. Also, as mentioned earlier, the implication that replacing some of the nitrogen with oxygen may reduce narcosis is not likely to be valid.

Because the mixes are richer in oxygen, the possibility of oxygen toxicity is greater, depending on the mixture being used. As shown in Figure 6–2, the *NOAA Diving Manual*[35,36] allows an exposure to a P_{O_2} of 1.6 atm for 45 min, but a wiser rule in recreational scuba diving—wherein a convulsion can easily lead to drowning—is not to exceed 1.4 atm. NOAA has adopted two standard enriched-air mixtures containing 32% and 36% oxygen to avoid the complication of having a variety of mixes on hand. Using the 1.4 atm maximum P_{O_2}, these mixtures can be used to 33 and 29 msw, respectively. The 1.4 atm limit is appropriate, and the recreational diver would be foolish to exceed it during the working phase of a dive. Paradoxically, for scuba diving, the greatest decompression efficiency with oxygen-enriched mixtures is in the depth range of about 20 to 25 msw, but here the allowable times are much longer than can be accomplished with scuba.[88] Enriched air diving is most effective in the range of approximately 15 to 35 msw (60 to 120 fsw).

Helium-Oxygen Mixes (Heliox)

Surface-supplied heliox is most effective for short working dives in which surface decompression can be used to shorten decompression time in the water, and this mixture is widely used for many jobs that do not justify or cannot easily be done with saturation because of the cost and complexity of the equipment. The heliox will have an oxygen content appropriate to the diving depth. This may be optimized for maximum decompression efficiency without oxygen toxicity or for operational effectiveness or simplicity of use.

The U.S. Navy helium-oxygen decompression tables[53] for many years recognized the fundamental principles of mixed-gas diving and oxygen decompression; they were based on the partial pressure of helium in the breathing gas at the depth of the dive and not just on depth alone. This allowed some flexibility in operations but made the tables somewhat difficult to use. More recently, the U.S. Navy heliox tables have been reformatted to be based on depth.[54]

The Navy tables called for oxygen to be supplied in the water starting at 50 fsw (15 msw), followed by surface decompression. They were modified by commercial companies to avoid the in-water oxygen. There is a rationale for employing in-water oxygen, but in these tables the concern was that it is used at too great a depth. The Navy has also modified its procedures on an interim basis to substitute an oxygen enriched air mix for the 100% oxygen breathed at 50 fsw in the water.

Commercial diving companies have developed proprietary heliox tables that consider, among other things, oxygen exposure, and effective heliox tables are now also in the public domain.[89]

Most heliox tables involve a switch to air or to an oxygen-enriched air mixture during decompression, and almost all tables end with oxygen being breathed in the shallow stops. The intermediate mix is selected so that nitrogen narcosis and oxygen toxicity are not limiting factors. The main benefit of a switch to an intermediate mix is to increase the oxygen because the bottom mix becomes relatively low in oxygen as decompression progresses. Another reason is that a switch to nitrogen as the inert gas also adds some efficiency. This situation appears paradoxical because nitrogen requires much longer for saturation decompression. It is, however, related to gas dynamics, because for a relatively short exposure, the slower diffusing nitrogen does not build up as fast as helium would. This advantage of nitrogen prevails over most of the range of short-duration bounce-type dives.

Helium has a high thermal conductivity, so it feels cold to breathe. In cold water, heat loss via the respiratory tract can be debilitating at depths below about 150 msw (500 fsw). This is blamed on helium because heliox is the breathing mix used at such depths and because helium feels cold.[90] However, because respiratory heat loss may be due more to convective than to conductive heat transfer, air or nitrogen-based mixes are likely to cause greater heat loss than does heliox.[91] Definitive experiments to resolve this question have yet to be undertaken. Small amounts of hydrogen in the breathing gas can be burned catalytically to add heat to the diver's inspired gas and perhaps replace or prevent some of this respiratory loss.[92]

Another instance in which the conductivity of helium is critical is in the case of a "lost" diving bell—the divers are trapped in a predominately helium environment, and the temperature in the bell soon approaches that of the sea (it can sometimes be as cold as 4°C). Until rescue, survival in this situation depends on heavy insulation to prevent skin heat loss and some means of preserving breathing-gas heat.[93]

Despite the existence of many exotic gases and gas mixtures, helium dominates the list of breathing gases, other than air; at the peak of its popularity in North Sea operations, helium may have been used more than air. A major deep-diving/saturation system may store as much as 50,000 m^3 of gas. The peak helium usage for oil operations in the North Sea for the year 1979 was almost 3 million m^3 (100 million cubic feet), most of it from the United States and Poland[94]; the peak annual usage in the Bay of Campeche, offshore in Mexico, was nearly 1 million m^3. In 1980, gas suppliers began delivering liquid helium, which occupies only 20% of the volume of compressed helium. In the mid-1980s, the petroleum economy changed, gas reclaim equipment became effective, and remotely operated vehicles began to do much of the work of divers, with the result that the total annual consumption of helium in North Sea operations now is about that of the single most active diving contractor in the 1970s.

Oxygen-Helium-Nitrogen Mixes (Trimix)

Technical diving originated when a group of cave divers wanted to reduce their level of narcosis for some dives in the range 70 to 75 msw. They had customized decompression tables prepared for mixes that reduced the narcosis to an acceptable level at the target depths. The use of enriched-air intermediate mixes and oxygen breathing at the end of the dive gave these decompression patterns significantly greater efficiency for these dives than was provided by commercial and navy tables available at the time. In commercial diving, multiple mixes for surface-supplied diving have been in use for decades, but in such operations, the complexity of the dives is managed by the topside support team. In deep, exploratory cave diving, a diver needs several tanks of gas and caches or stages them along the way, tied to the line. The mixes in these tanks can be varied to gain decompression efficiency. Dives as deep as 100 msw for times of more than 1 hour are not uncommon using these techniques.[95]

A major factor limiting the spread of this technique was the need for custom decompression tables. Trimix diving was originally developed used tables generated with a proprietary computational program, but Professor A. A. Bühlmann had published his method for calculating tables,[96] and creative divers soon learned to generate appropriate decompression tables with experience from their own dives. Computer programs that enable divers to prepare their own tables for trimix dives are available; however, their use without proper training is not recommended. Some of the organizations that train technical divers have prepared printed tables using such computer programs, but there are no recognized published tables for technical trimix dives. Many of those interested in technical diving are also qualified in mathematics and physics, and technical diving community is evaluating a number of relatively new algorithms or "models" for computing decompression tables.[97,98]

Zannini and colleagues[99] developed an earlier application of trimix using decompression tables; this application was used by divers collecting coral offshore Italy. The profiles were similar, except that the coral divers used surface decompression.

A typical technical trimix table profile is shown in Figure 6–9. This is a table commonly used for training: a dive to 75 msw for 25 min. It uses 17% oxygen and 50% helium as a bottom gas and requires a change to an intermediate enriched-air mix of 36% oxygen at 33 msw, the first stop depth and a change to pure oxygen at 6 msw. Decompression takes about 85 min.

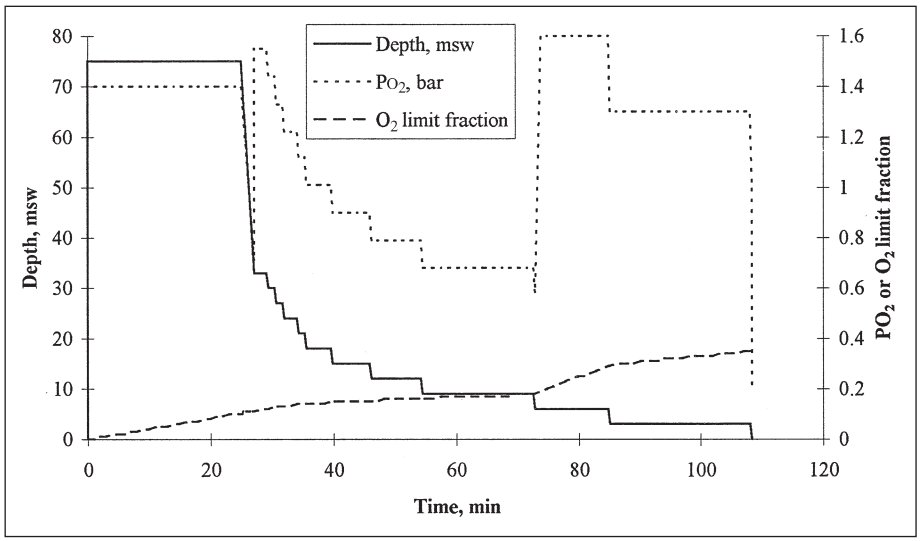

Figure 6–9. Profile of a technical trimix bounce dive to 75 msw for 25 min. This profile has been used many times without incident (the profile depicted is for display only and should not be used because it lacks some minor conservative modifications). The gas is switched to a 36% oxygen-enriched air mixture at the first stop at 33 msw and to pure oxygen at 6 msw. The *upper dotted line* shows the P_{O_2}, and the *lower dashed line* shows the buildup of the "oxygen limit fraction" (or *CNS %*), which reaches 0.35. Compare this profile with that in Figure 6–10.

Despite these being relatively stressful decompressions, the track record for technical trimix diving seems to be satisfactory from the point of view of decompression sickness. Technical diving has proven to be hazardous in other ways, however. Many divers have died because the wrong mixture was breathed at the wrong time. Decompression disorders that occur in divers surfacing without decompression from dives at more than approximately 50 to 70 msw are difficult to treat, and the diver may not survive even when treatment is prompt. Also, like air divers, technical divers tend to run out of breathing gas while underwater.

Rebreathers

A rebreather is a breathing apparatus that recirculates the diver's expired gas around a breathing loop, removing the carbon dioxide and replenishing the oxygen; a flexible "counterlung," or breathing bag, provides compliance to accommodate breathing. Rebreathers have existed for more than a century and have a long history in military use (see Chapter 29). There are many types of rebreathers, but those that provide the diver with a constant P_{O_2} are of particular interest here because they provide another special breathing gas. Constant oxygen rebreathers usually are controlled by electronic sensors, but mechanical methods also work reasonably well.[100,101]

The main objective of gas switching in a traditional heliox dive is to maintain as high a P_{O_2} as possible within tolerance limits. Figure 6–9 presents a simple example of this using gas switching. A constant oxygen rebreather allows the diver to breathe a mixture of high P_{O_2} throughout the dive. Maintaining a steady P_{O_2} of 1.4 atm provides almost as efficient an oxygen profile for a dive as possible and offers a tolerable oxygen exposure. A secondary benefit of switching to an intermediate nitrogen-based mix is to change the inert gas, but the benefit of this is secondary to that of the higher oxygen concentration. Rebreathers normally use only one diluent gas, but a built-in feature that would allow a switching of inert gas has been proposed. A 75 msw/25 min dive profile calculated for a constant P_{O_2} of 1.4 atm is shown in Figure 6–10. This dive profile has the same bottom exposure as the trimix dive involving two gas switches (see Fig. 6–9), and the two dives can therefore be compared. The rebreather decompression is 9 min shorter, and in neither dive is the oxygen exposure particularly stressful. At 6 msw, either the rebreather has to be purged to pure oxygen or the diver breathes oxygen supplied by an open-circuit apparatus from the surface by hose.

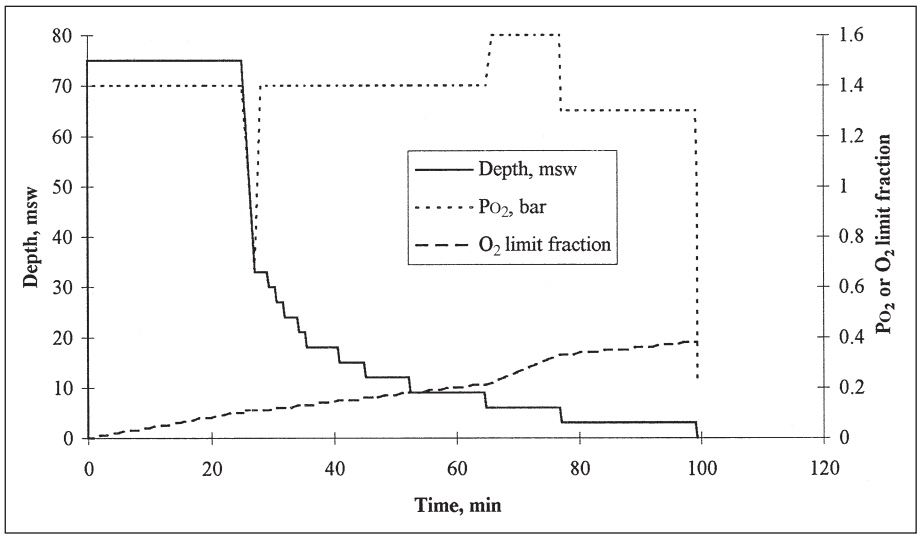

Figure 6–10. Profile of a constant Po_2 rebreather dive to 75 msw for 25 min. This dive does not involve gas switching as shown in Figure 6–9, but the composition of the mix changes with depth to maintain a constant Po_2. The diver breathes 100% oxygen at the 6 and 3 msw stops, which accounts for the deviations from 1.4 bar in the Po_2 curve. This profile assumes that the nominal gas is maintained at all times, but this is not normally the case in a real rebreather dive because it takes some time for the gas makeup to follow depth changes. The *upper dotted line* shows the Po_2, and the *lower dashed line* shows the oxygen limit fraction, which reaches 0.38.

Another type of rebreather, of which there are many variations, is the semiclosed rebreather. Like fully closed rebreathers, these have a breathing loop with a carbon dioxide absorbent canister and a counterlung, but these rebreathers use a constant inflow of a fixed mixture of oxygen and an inert gas, with the mixture oxygen fraction and flow calculated to provide a proper Fo_2 over the depth range of the dive. The diver consumes oxygen from the loop, so that the resulting Po_2 is affected by the diver's level of activity, the oxygen consumption rate. This causes the Po_2 to vary inversely with workload; this is favorable from an oxygen-tolerance perspective but makes decompression both inefficient and hard to calculate in advance. More sophisticated semiclosed units use a gas inflow system that is linked to the diver's respiratory minute volume; some even come close to providing a constant Po_2.

Alternative Inert Gases

NEON

Of the exotic breathing gases mentioned at the beginning of this chapter, neon and hydrogen are actually used as diving gases. Neon is a product of air distillation. Pure neon is very expensive, but an earlier cut in the air distillation process yields a mixture of neon and helium with about 75% neon and 25% helium that—where it is available—is priced similarly to helium; the "neon 75" mixture can be made available at prices remotely competitive with helium only in very large quantities. This mixture has been investigated in the laboratory[11,102] and has been used in commercial and technical diving. Neon is not narcotic but is about two thirds as dense as air, which somewhat limits its use; it is too dense for use beyond a depth of about 120 msw. Neon's advantages that led to its use in commercial diving are that it does not distort speech the way helium does, nor does it have such a high thermal conductivity[102]; however, the problems with helium have been resolved (with voice unscramblers, bell heaters, and wider availability), and commercial diving interest in neon has therefore waned. Neon is currently of interest to technical divers who think that the several minutes saved in decompression is worth the effort if the cost can be justified in some way. Neon's use in a rebreather is economically feasible, and this practice has been developed to a moderate extent.[103]

HYDROGEN

Hydrogen was first used as a diving gas by Zetterström in the early 1940s,[104] the motivation being the unavailability of helium in

Sweden and hydrogen's supposedly favorable decompression properties. Zetterström was killed on a hydrogen dive from an operational accident, but he did prove that the gas was usable.

An important operational limitation of hydrogen use, of course, is its extreme flammability. Mixtures of hydrogen and oxygen are explosive, except in situations where the percentage of oxygen is less than about 5%,[62,105] so beyond a depth of about 30 msw the P_{O_2} can be suitable for breathing. Extreme care must be used in handling the gas.[106,107]

Gardette and colleagues[10] and Rostain and colleagues[62] described several successful hydrogen-oxygen laboratory dives to 450 msw (46 atm). On one such dive, Hydra V, divers began with heliox and at 200 msw (650 fsw) switched to a nonexplosive mixture of oxygen, helium, and hydrogen, sometimes called *hydreliox*. Hydrogen alone at these depths is too narcotic for effective use. On decompression, the breathing gas was switched back to heliox at about 25 atm. The gas switch resulted in counterdiffusion sickness, which was treated similarly to decompression sickness. A slower transition in later dives eliminated the counterdiffusion problems. Interestingly, even though hydrogen and helium counterdiffuse and hydrogen is more soluble, experiments in hydrogen saturation diving have shown that the same decompression rate can be used for decompression from hydrogen saturation as is normally used for helium,[108] but for short-duration diving, hydrogen's decompression properties are somewhere between those of helium and nitrogen.

The advantage of hydrogen for very deep commercial (saturation) dives is that it is easier to breathe and thus allows divers to breathe through their noses, which improves their sleep and helps avoid respiratory infections; more important, in situations in which a diver's ability to work is limited by gas density, hydrogen allows heavier work to be performed. Hydrogen's narcotic potency is high enough that for use in the deepest depth range for human diving, about 50 atm or greater (500 msw or 1500 fsw), it is necessary to replace some hydrogen with helium. This narcosis can be somewhat helpful in combating HPNS during compressions.

Hydrogen gas can be obtained from electrolysis of water and is potentially more abundant than helium. Although most current hydrogen diving is experimental, the diminishing supply of helium may make mixes containing some hydrogen an attractive alternative to helium in the future.

The U.S. Navy has studied the possibility of breaking down the hydrogen in the body with bacterial enzymes.[109] Rats were fed bacteria that metabolize molecular hydrogen to methane, and when the animals were pressurized with hydrogen, large quantities of intestinal methane were generated. Upon decompression, these rats displayed fewer signs of DCS. Unfortunately, this program has been discontinued.

ARGON

Argon is much more soluble than nitrogen, much denser, and more narcotic, and therefore it offers little advantage over other diving gases. However, there are reasons why it might be breathed. It is used in underwater welding and may therefore be inhaled by the diver via the welding chamber atmosphere. It is also used to improve the insulation properties of dry suits. Further, some gas separation methods leave as much as 5% argon in the extracted oxygen. And finally, gas-manipulation techniques can be used to slightly improve decompression with an otherwise unfavorable gas, but the results are not likely to be worth the effort of dealing with another gas.

To assess the effects of the welding chamber environment in the Jason project, Comex has exposed diver subjects to an argon-oxygen atmosphere at 2.5 atm and has studied narcosis, counterdiffusion, and decompression in these divers.[13] Narcosis was definitive; it reduced performance scores and was regarded subjectively as being about the same as that induced by air at 40 msw. In these studies, counterdiffusion of the argon with helium was not a problem, and decompression, using helium ascent rates, was uneventful.

LIQUID BREATHING

Although it has been featured in movies and science fiction novels,[110] liquid breathing does not appear to be a likely prospect for real-world diving, the reason being the high density and viscosity of the liquid medium.

Kylstra and colleagues[111] showed that mice could survive while breathing normal saline solution saturated with oxygen at 3 atm. Subsequent studies with oxygenated fluorocarbon compounds showed that adequate oxygenation could be achieved without the need for inert gas and its accompanying narcosis and decompression problems.[112] It is possible to deliver enough oxygen using hyperoxygenated saline solution at pressures greater than approximately 3 atm and even at sea level using fluorocarbon fluids that readily dissolve large quantities of oxygen.[113] But the only way that carbon dioxide can be removed from the lungs is to flush it out (this is called *ventilation* when gas is used for flushing). The effective rate of carbon dioxide removal using a liquid medium is just enough to sustain normal basal metabolism at the flow rates possible, but hypercapnia develops with even low levels of exercise. If the right kind of low-density, low-viscosity, nontoxic solution could be found that would absorb the carbon dioxide and carry it out, perhaps with a nontoxic buffer or with stabilized microbubbles, liquid breathing might become feasible. At present, the viable breathing media are all gases.

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CHAPTER 7 Mechanisms and Risks of Decompression

Richard D. Vann

Knowledge of premorbid decompression physiology is essential to decompression procedures that avoid morbidity. Knowledge of the pathophysiology of decompression sickness (DCS) is a prerequisite for therapies that target underlying mechanisms. This chapter places bubble formation in a physiologic context and explores the interaction of bubbles with inert gas exchange at specific sites of injury. Individual, physiologic, and environmental factors that affect bubble formation and inert gas exchange are reviewed with an emphasis on the multivariate nature of DCS. Finally, the chapter addresses decompression safety from an epidemiologic perspective in which diagnosis, morbidity, and DCS probability are presented with the goal of refining the process for developing decompression procedures.

DECOMPRESSION SICKNESS AND THE THEORY OF BUBBLE FORMATION

Investigating bubbles and their relationship to DCS in intact animals and humans is technically difficult with available methods. In the absence of direct measurements, experimental manipulations of pressure, time, and environmental or physiologic conditions have been used to indirectly test hypotheses concerning underlying mechanisms. Together, these hypotheses support a theory about bubbles and DCS that originated in the 1940s.

As discussed in Chapter 4, Harvey[1] proposed that bubbles forming at low gaseous supersaturations originated from gas nuclei. He showed experimentally that short, rapid compressions to very high pressures reduced or eliminated bubble formation upon subsequent decompression (Fig. 7–1).

Evans and Walder[2] used hydrostatic compression as a specific test for gas nuclei in transparent shrimp decompressed to a pressure equivalent to 58,000 feet of altitude (17,679 m; 0.052 ata). Bubbles were visible under the shells of 96% of shrimp decompressed directly to 58,000 feet (left side of Fig. 7–2), but bubbles formed in only 8% of shrimp briefly compressed to 387 ata before altitude exposure (middle part of Fig. 7–2). This observation was consistent with the hypothesis that visible bubbles in transparent shrimp originated from gas nuclei that could be dissolved by hydrostatic compression.

Evans and Walder[2] compressed a third group of 50 shrimp to 387 ata followed by an electrical stimulation at altitude to induce physical activity, and bubbles formed in 32% of the shrimp (right side of Fig. 7–2). This observation was consistent with the hypothesis that physical activity caused the regeneration of gas nuclei.

Vann[3] conducted a similar study of DCS in rats (Fig. 7–3). A 2-hour control dive to 240 fsw (72 msw) followed by direct ascent to the surface resulted in 83% fatal DCS. With a brief compression to 600 fsw (180 msw; 19.1 ata) prior to the 240 fsw exposure, the DCS incidence was 74%; with compression to 1000 fsw (306 msw; 31.2 ata), the incidence was 64%. This observation was consistent with the hypothesis that the bubbles thought to cause DCS in the rat originated from gas nuclei that could be eliminated by rapid compression to pressures lower than those used by Evans and Walder.[2]

Daniels[4] investigated the effect of normal activity on the regeneration of the bubble formation capacity in hydrostatically compressed shrimp. Shrimp were exposed to a brief 200 ata hydrostatic compression prior to decompression to an altitude of 53,000 feet (16,155 m; 0.073 ata). Between the hydrostatic compression and the altitude exposure, there

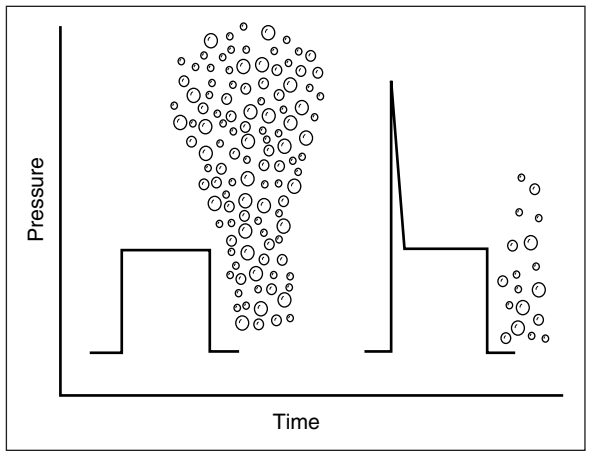

Figure 7–1. The hydrostatic pressure test for gas nuclei.[1] *Left,* Bubbles form after a dive. *Right,* Fewer bubbles form after a dive preceded by a short, rapid compression to a higher pressure. Gas nuclei are presumed to be eliminated during hydrostatic compression.

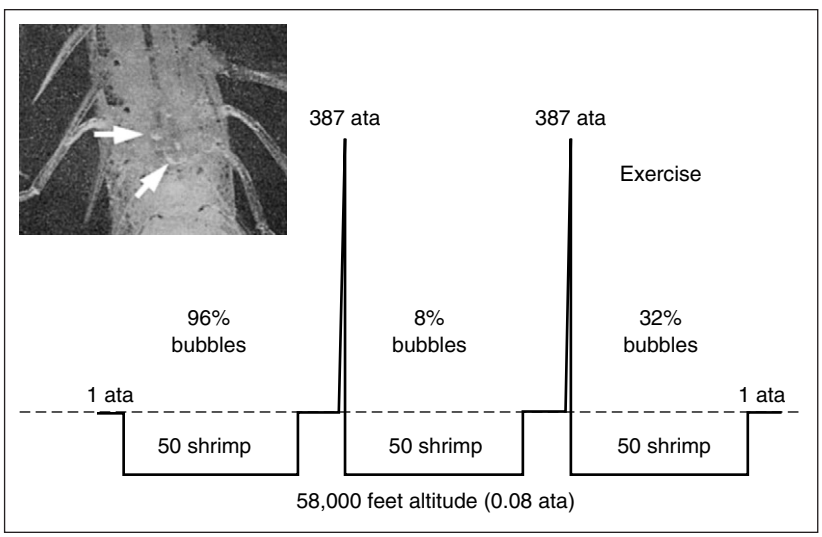

Figure 7–2. The hydrostatic pressure test applied to the formation of visible bubbles in transparent shrimp.[2] *Left,* After decompression to 58,000 ft of altitude (0.08 ata), bubbles form in 96% of shrimp. *Middle,* With a brief hydrostatic compression to 387 ata, bubbles form in only 8% of shrimp. *Right,* Exercise at altitude after hydrostatic compression increases bubble formation to 32%.

was a delay of 0 to 50 hours at 1 ata. There were few bubbles with no delay before decompression to altitude, and the number of bubbles increased as the delay lengthened (Fig. 7–4). With a 24-hour delay, bubble formation had returned to baseline levels. This observation was consistent with the hypothesis that the capacity for bubble formation in shrimp regenerated during 24 hours of normal activity.

Walder[5] observed that the DCS incidence in caisson workers was 10% to 12% when they began pressure exposure, but the incidence decreased to 1% to 2% after 1 to 2 weeks of daily exposure (Fig. 7–5). This phenomenon is known as *adaptation* or *acclimatization.* The higher incidence returned after 10 days without exposure. Acclimatization was specific for each pressure and recurred when the working pressure increased.[6] These observations were consistent with the hypotheses that (1) acclimatization occurred when the gas nuclei responsible for DCS were eliminated by daily exposure and (2) gas nuclei were regenerated during normal activity at 1 ata.

McDonough and Hemmingsen[7] studied the effect of physical activity on the formation of visible bubbles in crabs (Fig. 7–6). Resting crabs tolerated 150 atm of supersaturation without bubble formation; in active animals, bubbles formed at a supersaturation of only 2 atm. This observation supported the hypothesis of Evans and Walder[2] and Daniels[4] that physical activity promoted

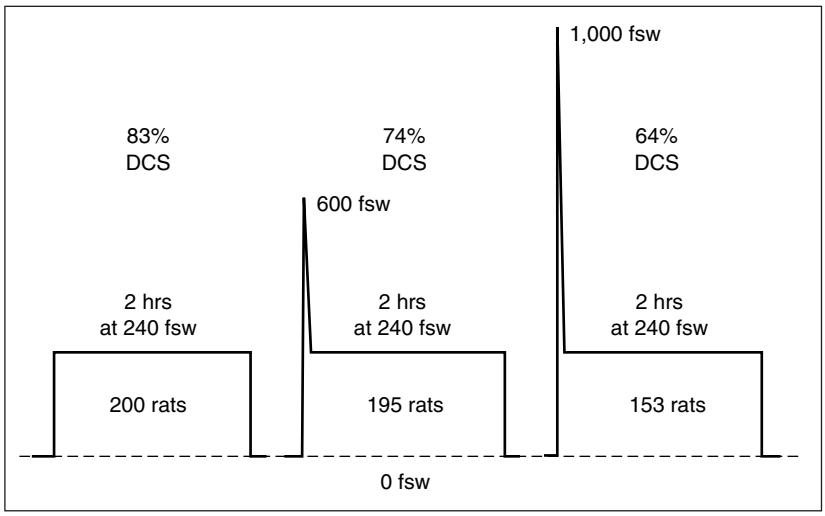

Figure 7–3. The hydrostatic pressure test applied to decompression sickness in rats.[3] *Left,* Fatal decompression sickness develops in 83% of rats exposed for 2 hours at 240 fsw (72 msw). *Middle,* With hydrostatic compression to 600 fsw (19.1 ata), 74% sustain decompression sickness. *Right,* Hydrostatic compression to 1000 fsw (31.2 ata) reduces the incidence of decompression sickness to 64%.

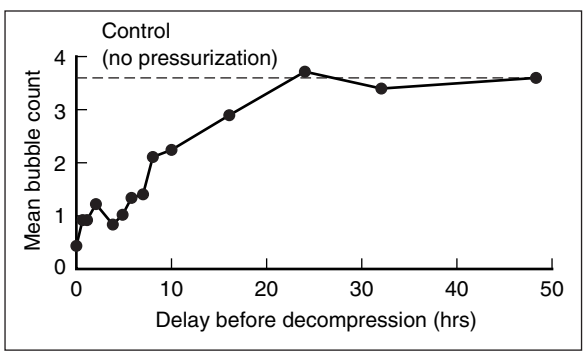

Figure 7–4. Recovery of the capability to form visible bubbles in shrimp after hydrostatic compression.[4] Shrimp were hydrostatically compressed to 282 ata, returned to 1 ata for a specified time, and decompressed to an altitude equivalent of 53,000 ft (16,155 m; 0.073 ata). Few bubbles formed when the delay to altitude exposure was short. As the delay increased to 24 hours, bubble formation returned to the control level without hydrostatic compression.

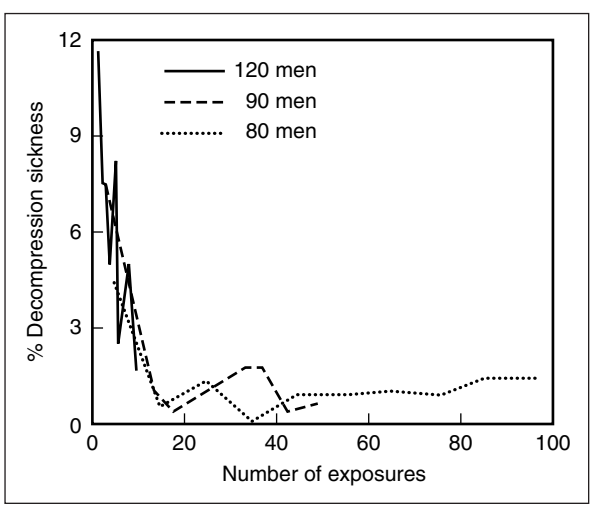

Figure 7–5. Acclimatization to decompression sickness in caisson workers during repeated days of exposure in three groups of men.[5] The incidence of decompression sickness decreased by five times during a period of 10 continuous exposures.

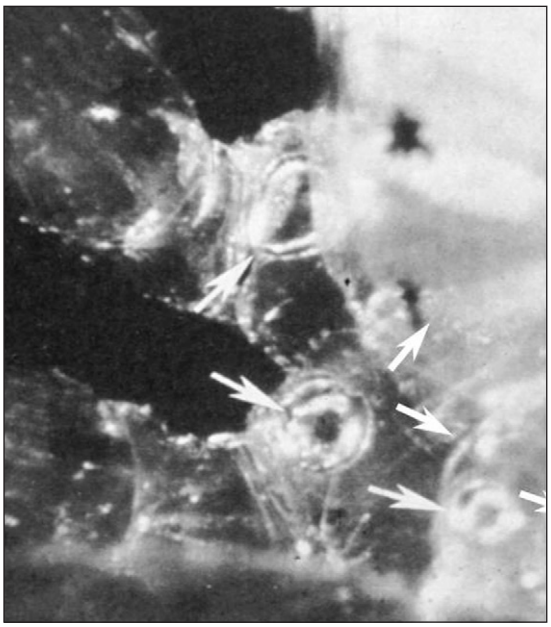

Figure 7–6. Immobilized crabs tolerated 150 ata of supersaturation without bubble formation, but bubbles formed in their leg joints at a supersaturation of only 2 ata with voluntary motion.[7]

bubble formation. Hydrostatic compression prior to supersaturation did not affect bubble formation, however, indicating that bubbles could form by tribonucleation in the absence of gas nuclei.[8]

McDonough and Hemmingsen[9] also investigated the persistence of bubbles that formed after decompression (Fig. 7–7). Freely moving crabs were exposed to 5 ata for 30 min. Upon decompression to 1 ata, approximately three bubbles, with diameters of 50 to 250 μm, formed in each crab. The crabs were immobilized after decompression, and the bubbles resolved in 10 to 47 min. Table 4–4 in Chapter 4 indicates that this is within the range of expected lifetimes for bubbles of these sizes in air-breathing animals (an oxygen window of 0.08 atm). After bubble resolution, the immobilized crabs were compressed to pressures of between 5 to 50 ata for 30 min, but no bubbles formed upon decompression. This observation was consistent with the hypothesis that bubbles in crabs were not stabilized against dissolution and that the same physics applied to bubbles in crabs as to in vitro bubbles (see Chapter 4 under Physics of Bubble Formation and Stability). The observation also reconfirmed physical activity as a promoter of bubble formation.

Dervay[10] studied the effect of pre-decompression exercise in humans on Doppler-detected venous gas emboli (VGE) after decompression to an altitude of 22,000 feet (6706 m) for 75 min (Fig. 7–8). Subjects performed 150 deep-knee bends at 1 ata with delays of 0, 1, or 2 hours before altitude exposure. The incidence of Doppler bubble grades 3 or 4 was 45% with immediate decompression, 24% with a 1-hour delay, and 9% with a 2-hour delay. These observations were consistent with the hypothesis that deep-knee bends

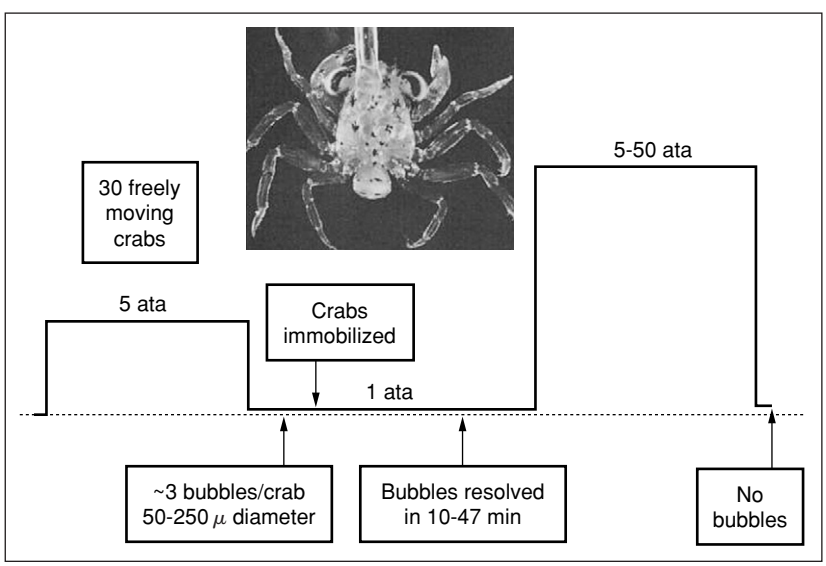

Figure 7–7. Bubbles formed in freely moving crabs after decompression from exposure to 5 ata.[9] The bubbles resolved in 10 to 47 min when the crabs were immobilized. With the crabs still immobilized, no bubbles formed after a second exposure to 5 to 50 ata.

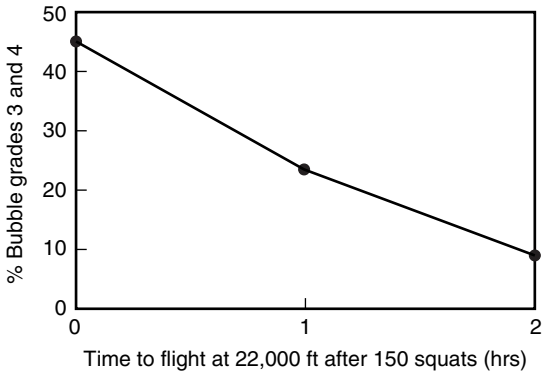

Figure 7–8. The effect of rest after heavy exercise on bubble formation at 22,000 ft (6706 m) in human subjects.[10] Fewer bubbles formed at altitude as the interval at 1 ata after 150 squats increased from 0 to 2 hours.

generated gas nuclei that decayed with a half-life of about 1 hour. Table 4–4 in Chapter 4 suggests that bubbles with diameters of at least 250 μm are consistent with this finding for air-breathing animals.

Vann[11] compared the incidences of Doppler VGE and DCS during altitude exposures at 30,300 feet (9236 m; 4.3 psia) in standing subjects and in reclining subjects. Both groups breathed 100% oxygen while seated at rest for 3.5 hours before ascent to altitude, and both groups performed the same upper-body exercises at altitude. There was no significant difference in the incidence of Doppler grades 3 and 4 detected precordially after arm movement, but the precordial Doppler incidence after leg movement was significantly higher for standing subjects (42%) than for reclining subjects (5%; $P = .0047$; Fig. 7–9*A*). In addition, there was no significant difference in the incidence of DCS pain in the arms, but the incidence of leg pain was significantly higher for standing subjects (50%) than for reclining subjects (5%; $P = .0011$) (Fig. 7–9*B*). These observations were consistent with the hypothesis that gas nuclei responsible for VGE and for the bubbles thought to cause DCS were generated during the physical activity of standing and walking.

A Theory of In Vivo Bubble Formation

Based on the studies reviewed above and the physics of bubble formation and stability discussed in Chapter 4, the following theory attempts to explain some aspects of in vivo bubble formation:

- Gas nuclei are the origin of some (1) visible bubbles in animals, (2) VGE, and (3) bubbles thought to cause DCS.
- Gas nuclei are bubbles generated by tribonucleation resulting from viscous adhesion during physical activity.
- In the absence of gas nuclei, bubbles may be generated directly by tribonucleation in supersaturated tissue.
- Gas nuclei are dissolved in minutes to hours (depending on size) by the oxygen window and surface tension.
- The creation and elimination of gas nuclei and bubbles are in dynamic equilibrium: Movement, exercise, or gravity promotes their creation; and rest, immersion, or microgravity favors their elimination.

Cracking Vertebrae and Spinal Decompression Sickness: A Circumstantial Anecdote or a Cautionary Tale?

"I clambered aboard the boat and hauled in my gear. The weather was mild and the sea mirror-flat. While breaking down my gear and stowing equipment, I looked down the reef where I recognized the dive guide in the 'Fin-n-Fins' dive boat as my friend Melvin. I waved to him, and he waved back shouting, 'How was your dive?' 'Great…just great,' I yelled. As I watched him help the tourists into the boat, pull anchor, and motor off towards the dive shop, I placed my fists behind me along my spine, one on top of the other, and simultaneously pressed inward and arched my back to 'crack' my vertebrae as I often did and sometimes still do. I remember being stunned at how 'complete' and 'robust' the crack was. Normally, I'd get two or three 'solid' cracks, with a few more 'soft' cracks, but this time, it seemed like every single vertebral joint from the small of my back to at least my shoulder blades yielded an extremely solid 'crack.' At the moment, it was extremely satisfying. I had never performed such a complete spinal 'crack' previously, nor have I ever since, and I've probably [cracked my back] a thousand times since I discovered the trick in high school. It couldn't have been more than about 30 to 90 seconds later

that Melvin's wake hit my boat, and the avalanche of symptoms began. I lost my balance slightly when my boat rocked, and I reached for the steering console, but my hand wouldn't go where I wanted. At first I didn't think much of it, but a few seconds later I realized that both of my hands and arms had lost coordination! I fought off panic as my brain scrambled to comprehend the situation. I looked out at Melvin's boat, but he was already too far away to hear my yelling over the roar of his engines. I moved my arms about, trying desperately to prove that they were really OK, but I was dizzy, and my legs were uncoordinated and numb. Losing control was horrifying."

The dives preceding these events had been severe. Within hours, the diver suffered from loss of bladder control and severe sensory and motor impairment of all limbs. After 28 recompressions, he could walk with a limp. Two years later, he could jog but had not regained normal sensation in his legs. In 2 more years, he was diving with a rebreather at 400 fsw.

—from *Confessions of a Mortal Diver—Learning the Hard Way (R. Pyle, personal communication)*

MECHANISMS AND MODELS OF SPECIFIC DECOMPRESSION INJURIES

This discussion focuses on putative mechanisms by which bubbles may initiate certain forms of DCS. Where possible, quantitative models of specific injuries are presented. See Chapter 8 for a full discussion of the pathophysiologic and pathohistologic aspects of injuries that follow the appearance of bubbles.

Sonophoresis, Cutaneous Decompression Sickness ("Skin Bends"), and Counterdiffusion

The mechanism of bubble formation in the skin appears to be different from the one described earlier. The skin is normally a barrier to the passage of externally applied substances; *sonophoresis* is a process by which ultrasound therapy can enhance transdermal drug delivery. Mitragotri and colleagues[12] found evidence that ultrasound energy caused in situ bubble formation in cadaver skin. The barrier property of the skin to drug permeability is attributed to its outermost layer, the stratum corneum,

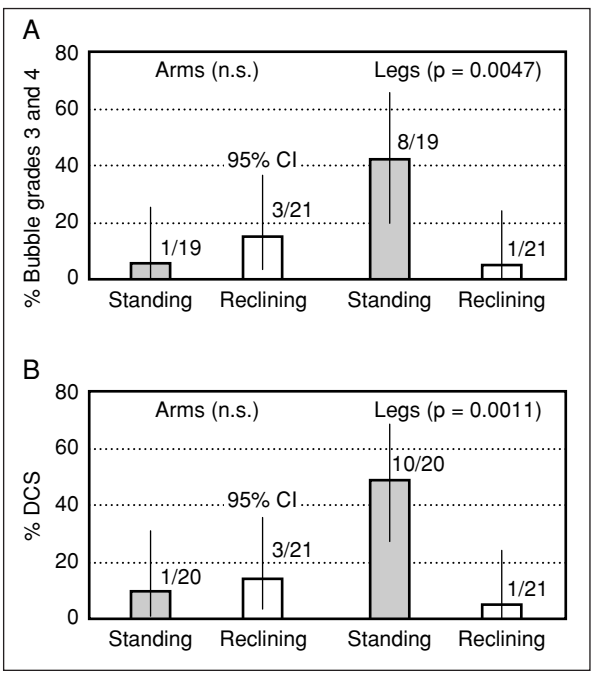

Figure 7–9. Precordial Doppler bubble scores and decompression sickness (DCS) in human subjects at 30,300 feet (9236 m; 4.3 psia) while standing or reclining.[11] The subjects performed the same arm exercises in all experiments, and neither Doppler bubbles *(A)* nor DCS *(B)* differed for standing or reclining subjects. Gravitational stresses were present in the legs of standing subjects but not in reclining subjects, and reclining subjects had significantly fewer Doppler bubbles *(A)* and DCS *(B)* than did standing subjects.

which is 15 μm thick and composed of keratinocytes surrounded by ordered lipid bilayers (Fig. 7–10). The cutaneous application of ultrasound energy induced cavitation at the interface of keratinocytes and surrounding lipid bilayers. Oscillating bubbles appeared to disorder lipid bilayers and enhance their permeability. The process was reversible, and the bilayers regained their ordering and impermeability when cavitation stopped. Whether this mechanism of bubble formation is active in skin bends and cutaneous counterdiffusion is uncertain, but sonophoresis demonstrates that in vivo bubbles can form at the modest levels of supersaturation induced by ultrasound therapy in sonophoresis.

Cutaneous DCS appears to have at least two distinct origins: in situ bubbles and arterial bubbles secondary to right-to-left shunting in the heart. The arterial bubble hypothesis is reviewed in Chapter 8. The following paragraphs discuss a model of in situ bubbles.

When humans or animals breathe slowly diffusing gases such as nitrogen or nitrous oxide while surrounded by rapidly diffusing helium, extensive bubble formation can occur through a process called *isobaric counterdiffusion*.[13] For example, bubbles dissected the subcutaneous tissue and caused severe bruising and capillary damage in pigs immersed in helium while breathing nitrous oxide (Fig. 7–11). Continuous counterdiffusion resulted in copious VGE and asphyxia when gas displaced blood from the heart. A similar phenomenon occurred during an experimental dive to 1200 fsw in which a human subject surrounded by helium-oxygen breathed 10 atm of nitrogen in a mixture of helium-nitrogen-oxygen. This subject experienced hard, raised, bloodless lesions of the skin with intense itching.[14]

The process by which isobaric counterdiffusion may generate supersaturation is represented in Figure 7–12 by a model of cutaneous inert gas exchange in which skin is treated as a diffusion barrier between the

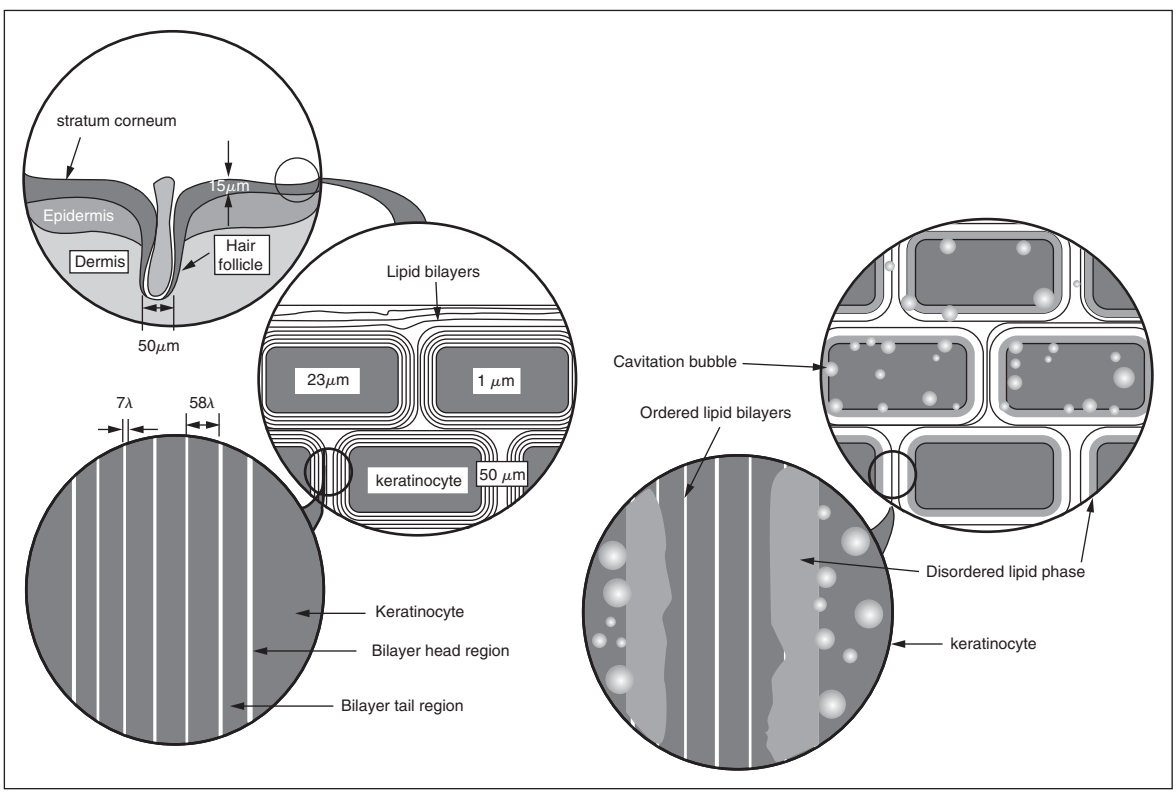

Figure 7–10. The fine structure of bubble formation in the skin during sonophoresis.[12] Externally applied ultrasound energy caused cavitation in the lipid bilayers adjacent to keratinocytes of the stratum corneum of the skin.

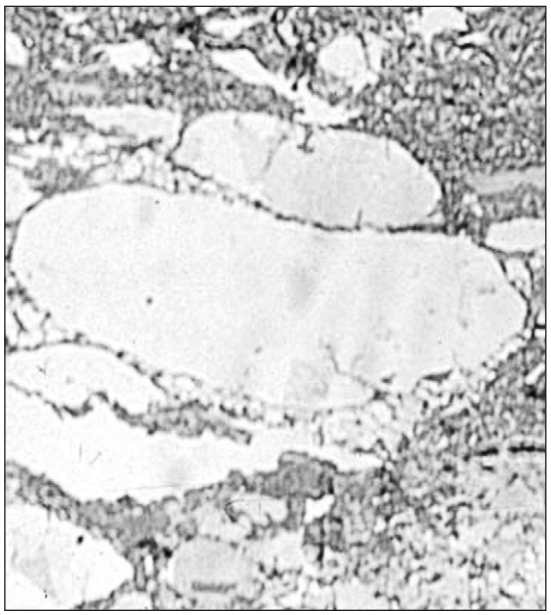

Figure 7–11. Gas spaces in a section of subcutaneous tissue from a pig breathing nitrous oxide and oxygen while surrounded by helium.[13,14]

environment and a well-stirred tissue compartment.[15] Helium diffuses from the environment through the skin into tissue more rapidly than nitrogen or nitrous oxide can diffuse out, and the resulting supersaturation causes bubbles to form without a pressure change.

The mechanism shown in Figure 7–12 can be applied to observations that postdive itching can be prevented by immersion in warm water[16,17] (see Chapter 8) and that in a dry environment, a cold arm may itch whereas a warm arm may not.[18] On the left side of Figure 7–13, poorly perfused cold skin is illustrated with its slow nitrogen elimination and thick diffusion barrier that impedes heat and nitrogen flux. On the right side of Figure 7–13, warm, well-perfused skin is shown as having rapid nitrogen elimination and a thin barrier to heat and nitrogen diffusion. Poor nitrogen exchange in the cold tissue would be expected to cause greater supersaturation (ΔP), increased bubble formation, and more intense itching.

Audiovestibular (Inner-Ear) Decompression Sickness

Inner-ear DCS can occur after long, rapid ascents or after a change in inspired inert gas from helium to nitrogen either with or without decompression.[19] Counterdiffusion of helium through the round window from gas in the middle ear space has been suggested as a contributor to inner-ear supersaturation,[14] but the mechanisms are poorly understood, particularly as to how the damage occurs. Chapters 8 and 22 review previous work in this area.

More recently, Doolette and Mitchell[20] proposed a physiologically plausible model of inert gas kinetics in the inner ear that predicts modest supersaturations and simulates the time course of reported signs and symptoms. The model, shown in Figure 7–14, is composed of three well-stirred compartments representing the vascular membranous labyrinth flanked on either side by avascular but well-stirred perilymph and endolymph compartments. The vascular compartment exchanges inert gas with its surroundings by perfusion with arterial blood and by diffusion from the perilymph and endolymph compartments. Inert gas also diffuses from the middle ear space through the round window. Diffusion barriers

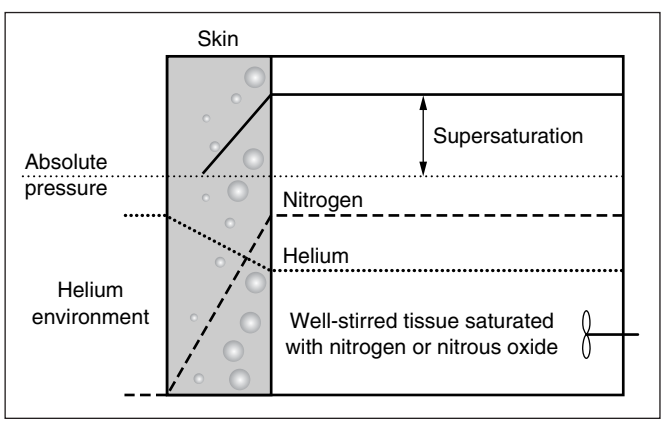

Figure 7–12. A model of inert gas exchange during isobaric cutaneous counterdiffusion based on the Hills model[15] in which well-stirred tissue is separated from the environment by a diffusion barrier. A rapidly diffusing gas (helium) surrounds the body while the inspired gas (nitrogen) is slowly diffusing. Helium diffuses into the body through the skin faster than nitrogen diffuses out, resulting in supersaturation and bubble formation in the diffusion barrier. As the bubbles grow, they cause the tissue damage shown in Figure 7–11.

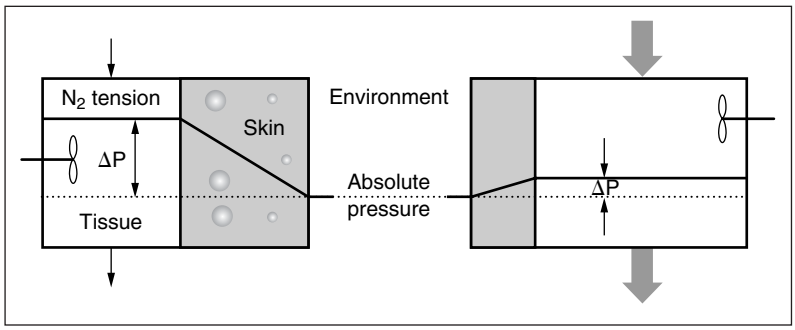

Figure 7–13. A model of "skin bends" (as shown in Fig. 7–12) after air diving in cool or warm water. *Left,* In cool water, subcutaneous tissue is poorly perfused and a large diffusion barrier reduces heat loss and restricts the outward diffusion of nitrogen through the skin. This results in a high level of supersaturation (ΔP) and significant bubble formation. *Right,* In warm water, subcutaneous tissue is well perfused, the diffusion barrier is small, and there is little supersaturation.

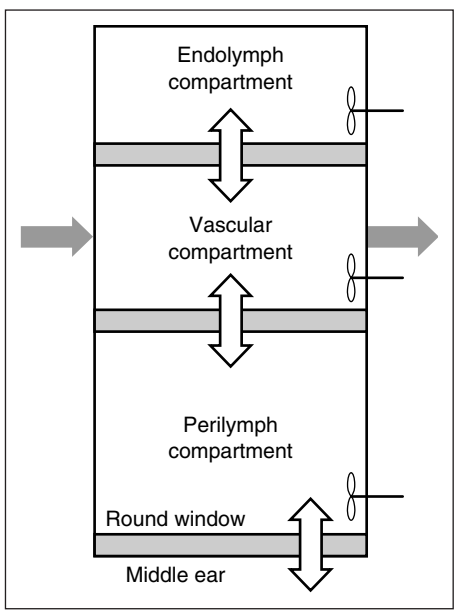

Figure 7–14. A model of inert gas exchange relative to decompression sickness in the inner ear according to Doolette and Mitchell.[20] A perfused vascular compartment exchanges inert gas through diffusion barriers with adjacent unperfused endolymph and perilymph compartments. All compartments are well stirred. Inert gas also diffuses from the middle-ear space through the round window. The fluxes of inert gases in these structures can lead to supersaturations during decompression from air diving or isobaric counterdiffusion as indicated in Figure 7–15.

at the compartmental interfaces simulate resistance to inert gas exchange between compartments. The diffusion and perfusion time constants were derived from published data for inner-ear anatomy and physiology. The resulting equations were solved for vascular, endolymph, and perilymph inert gas tensions and provided reasonable simulations of published measurements for endolymph and perilymph oxygen tension under anoxic conditions.

The approximate tissue half-time of the vascular compartment was 8.8 min. This is relatively fast, although slower than highly perfused brain, which has a half-time of about 1.7 min. Because the round window is small in area and the diffusion distance through perilymph to the vascular compartment is long, diffusion through the round window from the middle ear space had very little effect in the model on the overall inert gas exchange kinetics of the inner ear.

For a 367 fsw dive with rapid decompression followed by inner-ear DCS, the model predicted supersaturations of more than 1 atm. The isobaric change of breathing gas at 1200 fsw described earlier (see Sonophoresis Cutaneous Decompression Sickness ["Skin Bends"] and Counterdiffusion) also precipitated inner-ear DCS,[14] and the model predicted undersaturation in the perilymph and as much as 0.4 atm supersaturation in the endolymph and vascular compartments (Fig. 7–15). Although the dearth of well-defined cases of inner-ear DCS precludes calibration of model parameters, this is the first model to provide a credible simulation of processes likely to be involved in inner-ear inert gas exchange that could lead to supersaturation during decompression or isobaric counterdiffusion.

Limb Pain

One of the most common symptoms of DCS is pain in the joints and muscles, or *the bends.* Most of the evidence associating bubbles with limb pain is from altitude

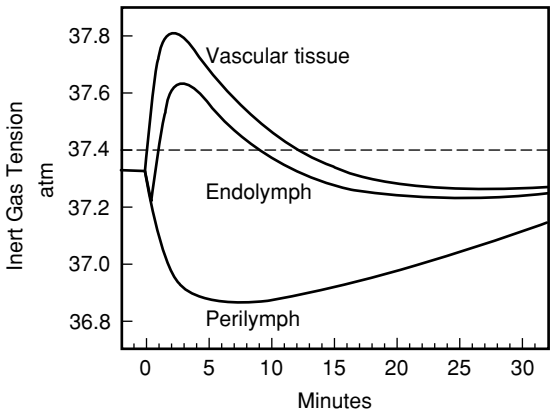

Figure 7–15. Compartmental inert gas tensions computed by the model of Doolette and Mitchell[20] for inner-ear decompression sickness on a dive to 1200 fsw (360 msw; 37.4 ata) with a subject surrounded by helium-oxygen and breathing 10 atm of nitrogen in a mixture of helium, nitrogen, and oxygen.[14] The subject experienced inner-ear decompression sickness as well as hard, raised, bloodless lesions of the skin with intense itching.

studies, but pain was similar among subjects exposed to both hypobaric and hyperbaric decompression, suggesting that the mechanisms and locations of altitude and diving DCS are similar.[21]

Radiographs of painful knees at altitude taken during World War II (see Chapter 4, Fig. 4–23) suggested an articular site for joint pain. The relationship of bubbles to pain was addressed in altitude exposures at 35,000 feet (10,668 m) in which both knees were radiographed when one knee became painful.[22–25] There was free gas in the knee joints of all subjects, with or without pain, but bubbles posterior to the femur in the upper posterior fossa and popliteal fat were statistically associated with pain, as were streaks of gas that appeared to be along fascial planes or tendons. The severity of pain and size of the gas lesion were associated with bubbles in the popliteal fat.

Acute altitude exposure also produced transient pains in the hands and feet accompanied by crepitus in the tendon sheaths.[22] Palpation of the tendon sheaths revealed bubbles that, when milked away, often relieved the pain. Ferris and Engle[22] argued that decompression pain was probably extravascular rather than intravascular because there was no local cyanosis, anoxic pain is usually maximal during the reactive hyperemia of recovery, local recompression sufficient to occlude blood flow relieves rather than intensifies pain, bubbles detected by radiograph that were associated with pain had an articular not a vascular distribution, and pain relieved by recompression recurred at the same site upon decompression 4 to 6 hours later.[22]

Bubbles associated with articular structures appeared to result from mechanical stresses in moving tissues (see Decompression Sickness and the Theory of Bubble Formation) and might reasonably be modeled by a diffusion barrier around a single bubble (see Fig. 4–17b) where the DCS risk increased with bubble size.[26,27] Delayed symptom onset after diving and symptom relief with recompression are consistent with bubble growth by diffusion, but bubble growth by diffusion is incompatible with symptoms that occur hours after descent from altitude when bubbles are resolving[28–30] or in cases refractory to recompression therapy.[31,32] Such cases may reflect secondary biochemical damage that accumulates as long as bubbles are present, with time required for healing.[33–37]

The diffusion barrier model might also be appropriate to describe autochthonous bubbles found in the spinal cord that are thought to be responsible for rapid-onset spinal DCS. A model of autochthonous spinal cord bubbles, however, would be expected to have different gas exchange kinetics than an articular bubble.

Possible Roles of Venous Gas Emboli in Neurologic Decompression Sickness

DCS involving the brain or spinal cord has the potential for causing permanent neurologic damage, which makes understanding the processes involved especially important. Chapter 8 concludes with a review of the pathophysiology of neurologic DCS and suggests that multiple mechanisms might be active, either alone or simultaneously, depending on the nature of the exposure and the physiology of the diver. Patent foramen ovale (PFO) is implicated among these mechanisms as a potential source of arterial bubbles that could seed the brain. The spinal cord appeared to be a less likely target, although Francis and coworkers[38] reported that delayed-onset DCS in dogs was histologically indistinguishable from gas embolism. The location of signs or symptoms does not

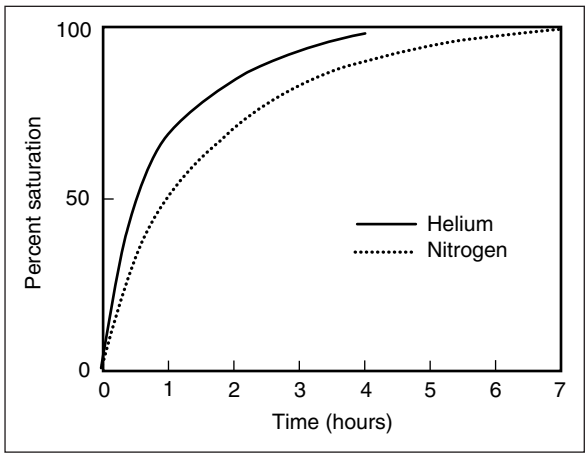

Figure 7–16. A comparison of respiratory helium and nitrogen exchange in humans as measured by Behnke and Willmon.[55] The apparent nitrogen half-time of is nearly double that of helium.

determine their cause, and DCS cases commonly described as spinal may be of cerebral origin.[39]

Bubbles in blood withdrawn from the sinus venarum of dogs after decompression were 19 to 700 μm in diameter.[40] In addition to passage through a PFO, these bubbles can enter the arterial blood through the pulmonary or bronchial circulation, which becomes more likely as larger gas volumes enter the lungs,[41–43] the pulmonary artery pressure increases,[44] or the bubble size decreases. In the absence of PFO, for example, ultrasound contrast agents containing bubbles with diameters of 2 to 10 μm[45] are visible by echocardiography in the left side of the heart after injection into a peripheral vein (unpublished observation).

Bubbles were cleared more effectively by the lungs when oxygen was breathed rather than air,[46] indicating that high bubble loads might be tolerated better at altitude with oxygen breathing than at sea level with air breathing. Indeed, more VGE were detected with air breathing in the surface intervals between repetitive dives than with oxygen breathing.[47]

Several studies have associated neurologic DCS with high Doppler bubble scores. In a series of 84 DCS cases for which Doppler data were available, 14 neurologic incidents were exclusively associated with Doppler grades 3 or 4.[48] Another study compared the effects of inert gas species on DCS and precordial Doppler bubble scores.[49] With statistical controls for differences in dive profile, Doppler grades 3 or 4 were present significantly more often ($P = .028$) after helium dives (grade 3 or 4 in 20% of 114 dives) than after nitrogen dives (grade 3 or 4 in 12% of 359 dives), and significantly more neurologic DCS ($P = .0014$) occurred after helium dives (80% neurologic cases in 5 DCS incidents) than after nitrogen dives (11% neurologic cases in 19 DCS incidents).[50] Neurologic DCS also was lighter with helium in a larger series of helium and nitrogen dives, in which the overall incidence of DCS was 3.7% for helium (64 cases in 1723 dives) and 5.2% for nitrogen (103 cases in 1976 dives), but serious symptoms accounted for 40.1% of all helium incidents (26 of 64) and 15.5% of all nitrogen incidents (16 of 103; $P < .001$).[49,51–54]

Figure 7–16 shows that helium is exchanged more rapidly than nitrogen.[55] The faster uptake of helium might explain why there were more VGE after helium dives than after nitrogen dives.[50] The deep decompression stops Momsen[56] found necessary after helium diving could have allowed excess helium to leave the body in the dissolved state rather than as VGE, thus reducing the potential for transpulmonary passage (see the side bar in Chapter 4, "Return of the Deep Stop").

Doppler studies have found VGE to be common after routine recreational air diving and to be predictively associated with the diver and dive profile,[57,58] but the frequency with which VGE might pass through the pulmonary circulation is unknown. Determining whether transpulmonary passage is significant in neurologic DCS should be a priority because a predictive model of VGE could control their incidence. VGE are easier to model than DCS because of their high incidence.[58,59] This would also address the added risk of neurologic DCS in divers with PFO that has been considered insufficient to justify PFO screening (see Chapter 8).

VGE may have a direct pathway to the epidural vertebral venous plexus of the spinal cord (Batson's plexus) through vessels that connect the systemic venous circulation to the epidural vertebral venous plexus at various locations.[60–62] These connections are a proposed conduit by which pathogens, tumor cells, and VGE might reach the epidural vertebral venous plexus from the systemic circulation. This is the basis for the venous infarction hypothesis of the spinal cord, although its active involvement in spinal DCS is uncertain.

FACTORS AFFECTING RISK OF DECOMPRESSION SICKNESS

Bubbles that initiate DCS are composed largely of inert gas, and factors that affect bubble nucleation or inert gas exchange might be expected to influence DCS risk. In addition, "host" factors such as age, gender, and weight, although not causes, can influence individual susceptibility.

Pressure Profile

DCS signs and symptoms differ with the pressure profile and breathing gas. Neurologic symptoms are most common after short deep dives[63] or altitude exposures with little or no preoxygenation.[28,30] Neurologic symptoms are rare for altitude exposures with long periods of preoxygenation,[64] after long shallow low-pressure caisson profiles,[32,65] or during slow decompression from saturation dives.[66] Chokes and pain are most common after long shallow dives or altitude exposure without preoxygenation.[63]

The association of spinal DCS with short, deep dives suggests that tissues responsible for spinal symptoms might exchange inert gas more rapidly than tissues responsible for pain. Figure 7–17 indicates that Doppler-detected VGE after open-water recreational diving increased with the dive depth and were more common after repetitive dives than after the first dive of the day.[57,58] In addition, VGE may originate from relatively fast tissues because the VGE incidence decreased with slow ascent rates,[67] deep decompression stops,[68] and "safety stops" after no-decompression (no-D) dives.[69,70] These techniques might decrease the risk of neurologic DCS by reducing both VGE and inert gas tension.

Acclimatization to Decompression

The phenomenon of acclimatization was discussed under Decompression Sickness and the Theory of Bubble Formation. Haldane had recognized acclimatization and recommended part-time duties for new caisson workers.[71] A Hong Kong tunnel project also provided evidence of acclimatization: The DCS incidence was 3.7 times greater for the first five exposures than for subsequent exposures.[32]

Acclimatization during air diving has proved difficult to demonstrate. Using Doppler-detected precordial bubbles as an index of acclimatization, Eckenhoff and Hughes[72] could find no evidence in 14 subjects during 12 single daily air dives for

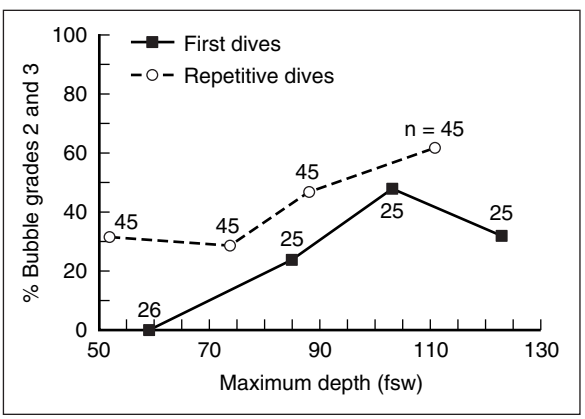

Figure 7–17. The incidence of Doppler bubbles for recreational divers after the first dive of the day and after repetitive dives as a function of the maximum dive depth.[57] The incidence of Doppler bubble grades 2 and 3 increased with maximum dive depth for first dives and repetitive dives.

30 min at 45 m (150 feet). A more recent Doppler study of multiday, repetitive, open-water recreational diving (Fig. 7–18), however, found that the incidence of grade 2 and 3 bubble signals decreased by 20% to 30% over 6 consecutive days of diving for the first dive of the day and for repetitive dives on the same day ($P < .001$).[54,75] The discrepancy between the chamber and open-water studies may reflect differences in the diving exposures or diving environment.

There is evidence of acclimatization to DCS in helium-oxygen diving. Tolerance was greater among divers making progressively deeper no-stop exposures than among divers first making deeper exposures.[73] In dives to 82 to 91 msw (270 to 300 fsw) for 15 to 20 min, 1 DCS incident occurred in 12 trials of "worked-up" divers and 6 incidents occurred in 6 trials without workup ($P < .001$).[74] In dives to 36 msw (120 fsw) for 40 min, no incidents occurred in 40 trials of worked-up divers and 6 incidents occurred in 17 trials of fresh divers ($P < .005$).[54,75] The workup effect seemed to persist for up to 4 days. Precordial Doppler bubble scores decreased progressively in seven divers who made three dives to 36 msw (120 fsw) for 20 min at 5-day intervals.[76] Because helium is exchanged more rapidly than nitrogen (see Fig. 7–16), helium dives would more closely approach the inert gas saturation levels of caisson exposures than would nitrogen dives. Thus, acclimatization to diving with helium might be more readily apparent than for diving with breathing gases that contain nitrogen.

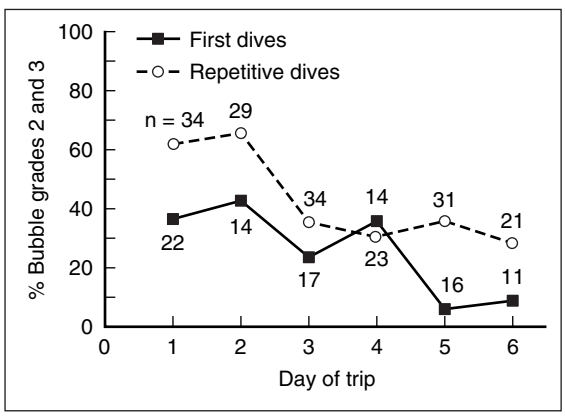

Figure 7–18. The incidence of Doppler bubbles for recreational divers for the first dive of the day and for repetitive dives as a function of the day of the trip during multiday diving.[57] The incidence of Doppler bubble grades 2 and 3 decreased during the course of the trip for first dives and repetitive dives.

Exercise

Exercise influences both bubble nucleation and inert gas exchange. The effect of exercise also depends on the phase of the pressure exposure in which the exercise occurs.

EXERCISE BEFORE PRESSURE EXPOSURE: BUBBLE NUCLEATION

Animal studies have demonstrated increased bubble formation due to exercise before decompression.[2,7–9,77–79] Anecdotal reports in humans have linked weight lifting and long-distance bicycle racing with increased DCS risk.[49,80] Other forms of pre-exposure exercise have been associated with unusual DCS after diving[81] and during altitude exposure.[82]

Dervay and colleagues[10] found that deep knee bends increased the incidence of Doppler-detected VGE at 22,000 feet of altitude but that this increase decayed with a half-time of about 60 min as the period between exercise and altitude exposure lengthened (see Fig. 7–8). Another study found that heavy weight lifting had no effect on DCS risk with a 24-hour delay between exercise and exposure at 30,300 feet of altitude.[83]

Studies that investigated the effects of endurance training in rats and pigs and found decreased bubble formation and DCS.[84–86] The benefit of physical conditioning was also suggested by the association of low VGE incidence with divers in whom maximal oxygen consumption was high.[87]

In summary, the effect of exercise before decompression appears to depend on the intensity of exercise, the interval between exercise and decompression, and physical conditioning. Studies of anaerobic exercise before altitude exposure in humans and animals found evidence of increased DCS risk if the exercise took place immediately or within several hours of decompression but that the risk was not increased after a 1-day interval. Studies of physical conditioning found that better conditioning reduced the risk of VGE in humans and of DCS in animals.

EXERCISE BEFORE PRESSURE EXPOSURE: NITROGEN ELIMINATION

To decrease the DCS risk due to altitude exposure, aviators and astronauts commonly

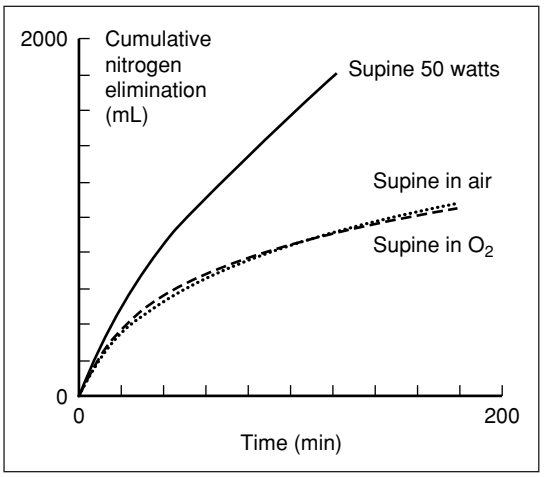

Figure 7–19. Respiratory nitrogen elimination from a subject breathing 100% oxygen at 1 ata while supine or during 50 watts of supine exercise with arms and legs.[89]

breathe 100% O_2 before decompression to eliminate tissue nitrogen.[64] The kinetics of nitrogen elimination are governed largely by tissue perfusion (see Haldane Decompression Theory: Stage Decompression in Chapter 4), which is strongly influenced by physical exercise. A number of studies have addressed the effect of exercise on respiratory nitrogen elimination, VGE incidence, and DCS risk. Balke[88] provided the first evidence that exercise might have a beneficial effect by showing that exercise during oxygen prebreathing delayed the onset of DCS symptoms.

Figure 7–19 shows the cumulative nitrogen eliminated at the mouth from a subject breathing 100% oxygen in a supine position at rest and during 50 watts of arm and leg exercise.[89] These and similar measurements demonstrated 25% to 38% increased respiratory nitrogen elimination during 25 watts of arm and leg exercise for 3 hours compared with resting controls. Exercise was significantly associated with increased respiratory nitrogen elimination in 122 measurements under various conditions ($P < .0001$).

In subjects who either rested or exercised at 25 watts during oxygen breathing before decompression to 30,300 feet (9236 m) of altitude, the DCS incidence was 39% with rest (32 cases of DCS in 82 trials) and 16.7% with 25 watts of exercise (7 cases of DCS in 42 trials; $P = .0003$).[89] Webb and coworkers (1996) found that a higher level of exercise for a shorter time interval was also effective at reducing DCS risk at 30,300 feet.[90] A 1-hour resting oxygen prebreathe was compared with a 1-hour prebreathe that began with 10 min of arm and leg exercise at 75% of maximum oxygen consumption. The incidence of DCS with this resting protocol was 77% (20 cases of DCS in 26 trials), whereas the incidence of DCS with the exercising protocol was 42% (11 cases of DCS in 26 trials; $P = .0.0109$).

EXERCISE AND DECOMPRESSION FROM THE SPACE STATION

The concepts described in the previous sections have been applied to the decompression of astronauts for extravehicular activity (EVA) from the International Space Station. EVA in the Space Shuttle had previously been conducted from a cabin pressure of 14.7 psia to a space-suit pressure of 4.3 psia using a 12- to 36-hour decompression stage at an intermediate pressure of 10.2 psia with 26.5% oxygen.[91] This procedure proved effective, and no DCS was reported during Shuttle EVA. The Space Station was designed for a fixed pressure of 14.7 psia, however, and staged decompression could be accomplished only with difficulty in a small lock. The excessive length of staged decompression would also make the increased frequency of EVA planned for Station operations impossible.

Operationally, a 2.5-hour pre-EVA protocol was preferred for the Space Station that would involve oxygen prebreathing at 14.7 psia followed by direct decompression to the suit pressure of 4.3 psia. Previous studies had observed DCS incidences of 20% to 40% at 4.3 psia after prebreathe times of 3.5 to 5 hours.[64,89] In these studies, DCS was 90 to 95% limb pain with the legs being the most frequent location. Figure 7–20 shows the location of limb pain among various groups exposed to decompression[89,92] and reveals that DCS in the legs occurred more than twice as often in people who were standing or walking (caisson workers, tunnel workers, saturation divers, altitude subjects) than in those who were seated (aviators) or immersed (bounce divers). Immersion is commonly used to simulate the microgravity of space,[93,94] which suggested the hypothesis that the antigravity stresses in the legs involved in standing were a source of gas nuclei responsible for bubbles that caused DCS (see Decompression Sickness and the

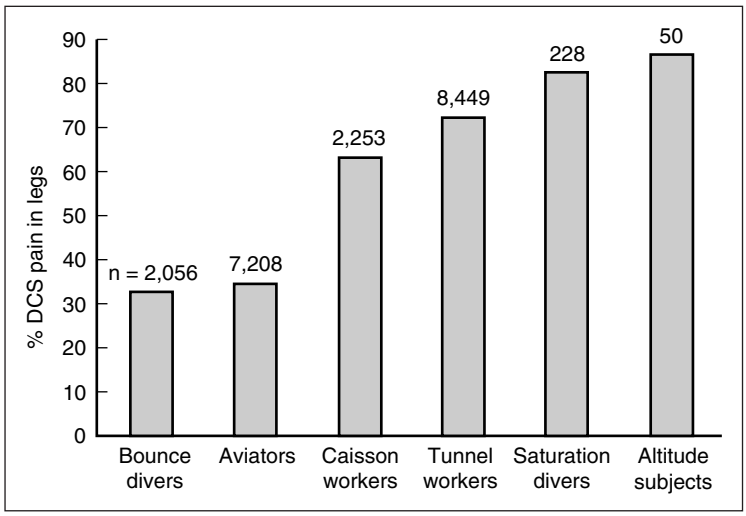

Figure 7–20. Distribution of leg pain in six populations affected by decompression sickness (DCS).[89,92]

Theory of Bubble Formation). Because ground-based EVA trials had traditionally used standing subjects, a study was conducted to find whether the incidence and location of DCS would change for reclining subjects. The results (see Fig. 7–9) showed significant reductions in DCS and VGE and were consistent with gravity as a DCS risk factor that is absent in astronauts, divers, and persons who are seated or reclining.

Dervay and associates[10] provided evidence that antigravity exercise generated VGE and showed that this effect resolved with a half-time of about 60 min (see Fig. 7–8). To improve the simulation accuracy of ground-based EVA trials, a new EVA protocol was developed in which subjects reclined for 3 hours to allow the effects of prior antigravity activity to dissipate before altitude decompression.[95] This protocol used arm and leg exercise to accelerate nitrogen elimination during oxygen prebreathing. Trials of several exercise regimens led to a 2-hour oxygen prebreathe with 10 min of heavy exercise as specified by Webb and colleagues,[90] followed by 40 min of light exercise. The protocol was tested 50 times without DCS[96] and has been used in 34 EVAs from the Space Station since installation of an air lock in 2001.

A recent study by Balldin and coworkers[97] has questioned the effect of simulated weightlessness on DCS incidence. During a 60-min oxygen prebreathe, subjects performed 10 min of arm and leg cycle exercise at 75% of maximum oxygen consumption followed by 50 min of rest. During the subsequent altitude exposure at 4.3 psia, 26 control subjects conducted EVA-simulation exercises while standing and walking while 39 reclining subjects performed the same exercises. The overall DCS incidence was the same, 42% in the control subjects and 44% in the reclining subjects, and the overall incidence of VGE (grades 1 to 4) was 81% in the controls and 51% in the test subjects, significant at $P = .0158$.

The lack of a significant difference in DCS incidence between standing and reclining subjects did not support gravity as a DCS risk factor and differed from the prior results (see Fig. 7–9).[11] Heavy exercise had been conducted only 50 min before altitude exposure, however, which may have been inadequate to dissipate the effects of exercise on bubble nucleation (see Fig. 7–8).[10] The interesting and opposing effects of exercise on DCS and VGE deserve further investigation.

EXERCISE AT DEPTH DURING DIVING

Just as exercise during oxygen breathing accelerates nitrogen elimination, exercise at depth during diving accelerates nitrogen uptake. Behnke and Willmon[55] demonstrated that exercise at sea level increased the rate of whole-body inert gas uptake. Nitrogen elimination measured at sea level after exercising no-decompression (no-D) air dives was also greater than after resting dives.[98] Figure 7–21 shows that a diver who exercised during 25 min exposures at 30 m (100 feet) eliminated 20% to 60% more nitrogen at 1 hour after decompression to sea level than did the same diver at rest.

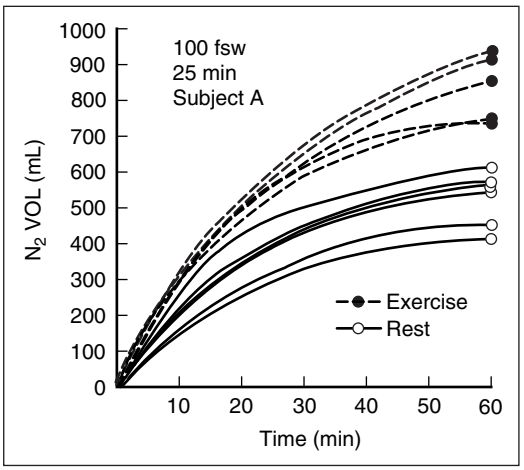

Figure 7–21. Respiratory nitrogen elimination measured at 1 ata after 25-min dives to 100 fsw (30 msw).[98] While at depth, the subject was at rest in six studies and exercised in five studies.

Van der Aue and associates[99] found that the incidence of DCS among resting divers was 11%, whereas among working divers it was 21% on the same schedules. DCS occurred most frequently in limbs exercised vigorously at depth. In other tests, Van der Aue and colleagues[100] reported that air-decompression schedules, which were safe for resting divers, led to a DCS incidence of 20% to 30% in working divers. Buehlmann[101] found that divers doing light work during helium-oxygen dives required 20% to 40% more decompression time than resting divers.

The effects of exercise on total decompression time were studied in 260 decompression dives in a hyperbaric chamber using a closed-circuit mixed-gas breathing apparatus at a constant oxygen partial pressure of 0.7 or 1.4 atm in nitrogen diluent gas.[49] The dive depths were 100 and 150 fsw with a 60-min bottom time, and the divers were dry and at rest or immersed in 20°C water while swimming at light or moderate exercise (oxygen consumption 1 L/min or 2 L/min, respectively). There were 13 DCS cases, for an overall incidence of 5%. Figure 7–22 shows the estimated effects of exercise on the total decompression time after a 60 min dive to 100 fsw. The experimental outcomes in DCS dives are shown as circles. The estimated total decompression stop time to achieve a 20% DCS incidence with light work was 10 min. With moderate work, the estimated total stop time was 100 min. Just as exercise during oxygen prebreathing reduced DCS risk during altitude exposure by accelerating nitrogen washout (see Exercise before Pressure Exposure: Nitrogen Elimination), so exercise at depth during diving accelerated nitrogen uptake and increased the postdive DCS risk.

EXERCISE DURING OR AFTER DECOMPRESSION

Exercise during decompression and exercise after decompression are different phenomena. In the first half of the twentieth century, U.S. and Royal Navy divers routinely exercised during decompression because exercise was thought to accelerate nitrogen elimination and reduce decompression risk.[102,103] Subsequent altitude and diving experiments, however, showed that exercise increased DCS incidence and severity and reduced the onset time. After decompression to 11,582 m (38,000 feet) of altitude, for example, the DCS incidence increased 32% in subjects who did five push-ups and five deep-knee bends every 15 min.[104] The increased incidence was equivalent to an additional 1524 m (5000 feet) of decompression. There was no evidence for increased DCS risk after

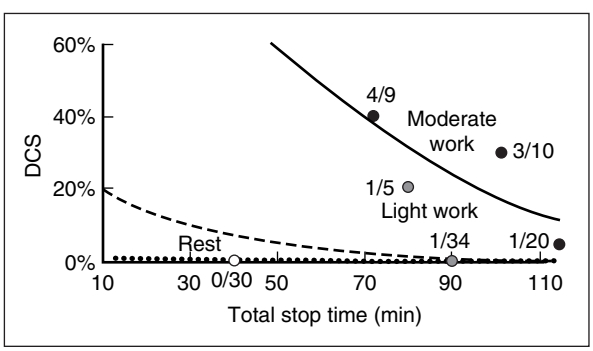

Figure 7–22. The effects of exercise and total decompression stop time on the incidence and estimated probability of decompression sickness (DCS) in humans after 60-min dives to 100 fsw (30 msw) while breathing 0.7 atm of oxygen in nitrogen.[49]

descent from altitude with moderate exercise at ground level.[105]

In diving experiments, Van der Aue and associates[106] found a 34% increase in DCS incidence in divers who lifted 25-lb weights for 2 hours after no-stop dives at 12, 30, and 46 m (40, 100, and 150 feet). Van der Aue titled this report "The Effect of Exercise During Decompression…," even though he had tested exercise only *after* decompression. He recommended that both forms of exercise be avoided. The prohibition on exercise endured for 30 years.

If exercise accelerates inert gas exchange, why would exercise during decompression *reduce* DCS risk and exercise after decompression *increase* risk? The question can be answered from the differences in inert gas exchange after bubbles form (see Effects of Bubbles on Inert Gas Exchange in Chapter 4). Decompression is designed to avoid or minimize bubble formation so that inert gas can be eliminated in the dissolved state as it was absorbed. If decompression progresses too far, inert gas can become isolated from the circulation in bubbles. This decreases the difference between the tissue and arterial inert gas tensions and reduces gas elimination rate. Thus, exercise during decompression can be beneficial if bubbles have not formed.

If bubble formation has not been excessive, exercise might be expected to accelerate inert gas elimination just as it did during oxygen prebreathing prior to altitude exposure. The data of Jankowski and colleagues[107] support this idea: They found that exercise during decompression reduced the incidence of VGE. Thirty-minute dives were conducted with immersed divers resting at 45 msw (150 fsw). Decompression took place according to the Defense and Civil Institute of Environmental Medicine (DCIEM) Standard Air Tables,[108] with 55 min of decompression during which 22 divers rested while 16 exercised intermittently with arms or legs at half their maximum aerobic capacity for 25 min. VGE were detected in 77.4% of resting divers but in only 42.7% of divers who exercised during decompression ($P = .019$).

The observation that exercise during oxygen prebreathing decreases DCS at altitude together with the finding that exercise during decompression from diving reduces VGE suggests that exercise during decompression might reduce the decompression time needed to limit DCS risk. With a 60-min dive to 100 fsw with light exercise at depth and resting decompression, one DCS incident occurred in 34 dives with 80 min of decompression time and no DCS occurred in 29 dives with 90 min of decompression (see Fig. 7–22).[49,109] When divers performed light exercise during 60 min of decompression, 26 dives were conducted without DCS. These data support the idea that exercise during decompression is beneficial, but further evidence is needed.

Immersion and Temperature

Immersion and temperature affect regional perfusion and thereby inert gas exchange, but few specific data are available to separate their effects on DCS risk. Moreover, exercise may exert part of its influence by warming an immersed diver and increasing inert gas uptake at depth or inert gas elimination during decompression. Some of the key studies are now summarized briefly.

Balldin[110] found that 2 of 10 subjects experienced DCS symptoms at altitude after breathing oxygen while immersed in 37°C water, whereas symptoms developed in 9 of 10 dry subjects ($P < .01$). Thalmann,[53] however, found no difference between decompression in warm (22°C) or cold (7° to 13°C) water. Weathersby and coworkers[111] estimated that immersion increased DCS risk by less than 30%, but this analysis was not controlled for exercise effects and immersed divers were generally exercising while dry divers were generally at rest.

Dunford and Hayward[112] studied divers wearing dry suits in 10°C water during no-D dives to 78 fsw (23.4 m) for 38 min and found that bubble scores increased by three times compared with scores for divers wearing $\frac{1}{8}$-inch wetsuits. The authors suggested that cold divers, who were peripherally vasoconstricted, absorbed less nitrogen than warm divers and thereby experienced fewer postdive bubbles.

Mekjavic and Kakitsuba[113] exposed four subjects to dry chamber dives at 30 fsw (9 m) for 12 hours followed by 3 hours of seated rest in a 10°C dry environment or, on a separate day, in a 40°C environment. Three of the four subjects had Doppler-detected VGE at 10°C; only one of four had VGE at 40°. (The difference was not significant.) After the 3-hour Doppler monitoring period, all subjects took hot showers, and three of four 10°C subjects

experienced mild shoulder pain 4 to 6 hours after surfacing whereas four of four experienced pruritus or shoulder pain. None of the 40°C subjects experienced symptoms. Compared with none of four, three of four was significant at $P = .029$ and four of four was significant. The authors speculated that cool subjects had more bubbles because decreased peripheral perfusion reduced the nitrogen elimination rate. The authors suggested that mild DCS symptoms developed in cool subjects after hot showers because the nitrogen solubility decreased, raising the local nitrogen gas tension. They concluded that a hot shower after diving might be a DCS risk factor in cold divers.

Leffler and White[114] discussed the salvage operations of TWA Flight 800 that also focused attention on the role of temperature in DCS. At the start of these operations, the divers used wet suits and experienced no DCS in 16 exposures with decompression according to the U.S. Navy Surface Decompression with Oxygen (SDO_2) Table.[115] When they switched to hot-water suits for better thermal protection, 5 DCS cases occurred in 14 dives, a significant increase in DCS incidence ($P = .036$). This problem appeared to be corrected by "jumping" schedules, i.e., decompressing according to schedules for longer or deeper dives. With the standard SDO_2 decompression time, 5 DCS cases occurred in 14 dives with hot-water suits. When a mean of 15.3 min extra SDO_2 decompression was added, 3 DCS cases occurred in 653 dives, a significant decrease in DCS incidence ($P < .0001$). Neurologic or respiratory signs or symptoms were present in seven of the eight TWA 800 DCS cases, raising the question of whether SDO_2 or hot-water suits predisposed to serious cases. Shields and Lee[116] had addressed this question in a study of commercial diving and concluded that hot-water suits contributed to the overall DCS incidence and the proportion of serious cases.

To further investigate the effects of water temperature on DCS risk and severity, Leffler[117] analyzed published data that had not been evaluated statistically, including 62 DCS cases in 11,662 dives[116] and 147 DCS cases in 1507 dives to develop and test the U.S. Navy SDO_2 tables.[100] Leffler concluded that the association of hot-water suits and DCS in the data of Shields and Lee was suggestive ($P = .07$) but recognized the uncertainties of an analysis without original data. With more DCS cases and greater detail from the Van der Aue data, Leffler found that the DCS risk increased by an odds ratio of 1.96 for each 10°C increase in ambient water temperature ($P = .0007$). The odds of DCS also increased by 88.6 for doubling the dive depth and by 10.3 for doubling the bottom time ($P < .0001$). Each hour of chamber decompression time reduced the DCS odds by 0.03 ($P < .0001$). When statistical controls were applied for differences in dive-profile characteristics, temperature was not associated with serious DCS but serious DCS was associated with shorter bottom times, fast average ascent rate in the water, and long chamber time.

Individual Factors

Although difficult to measure, individual susceptibility appears to significantly affect DCS risk. Caisson workers with a history of previous DCS were more likely to experience future DCS.[118] In a group of 376 compressed-air workers studied during 40,000 exposures, the mean DCS incidence was 0.87%. Fifty-five percent of the workers, however, had an incidence below the mean, 11% had an incidence equal to the mean, 6% had twice the mean incidence, and 10% had 5 times the mean incidence.[119] The remaining 18% experienced an incidence 28 times the mean but left work after only a few exposures.

Age and body fat are possible causes of individual variability. Age has been considered a contributing factor since the first study of DCS by Pol and Watelle.[120] Age was implicated as a risk factor in 11 reports on diving, caisson, and altitude exposure.[120,121] Three reports found no association with age.[118,122,123] Using the relationship between age and altitude DCS developed from data on 52,000 subjects, Gray[120] estimated a 28-year-old man to be twice as susceptible to DCS as an 18-year-old. Factors associated with age that might affect susceptibility include body fat, degenerative joint disease, changes in pulmonary function, cardiovascular disease, and obesity.[123]

Body fat has been implicated as a DCS risk factor since the earliest observations.[120] The effect of body fat is usually explained by high nitrogen solubility, which increases nitrogen absorption and bubble growth. Three animal studies and 12 human studies report an association of DCS and body fat in diving,

altitude, and caisson work[118,124]; two diving studies found no association.[122,125] Citing the relationship between altitude DCS and weight/height ratios in 49,000 subjects, Gray[120] estimated that a 178 cm (70 inch) tall, 89 kg (196 lb) man was twice as susceptible as a 57.3 kg (126 lb) man of the same height. For altitude exposure, DCS risk increased significantly with the weight/height ratio[89] and with weight.[126]

The reports of Wise[122] and Curley and colleagues[125] stand out in finding no association between DCS and body fat. Wise[122] studied 1131 U.S. Navy divers, 63% of whom experienced DCS; Curley and associates[125] studied 376 U.S. Navy divers, 30% of whom experienced DCS. The reason for the lack of association is uncertain, and several factors are possible:

- Navy divers may be younger and healthier than other subjects.
- Body fat may have a different effect in short dives than in caisson or altitude exposures.
- High body fat may protect against DCS in cold water.
- Modern diving procedures are less severe than earlier procedures.

Carturan and coworkers[87] found that age, weight, and maximum oxygen consumption were significantly associated with Doppler-detected VGE but that the percentage of body fat was not. Dunford and colleagues[57,58] found that age and gender were associated with Doppler-detected VGE (Fig. 7–23). Webb and associates[126] also found a higher incidence of VGE in women exposed to altitude but no difference between men and women in DCS risk. However, women using birth-control medication were more susceptible during the last 2 weeks of the menstrual cycle. Doyle and colleagues[127] had also observed that women using birth-control medication appeared to have a higher DCS risk.

Multiple factors can provide a stronger indication of individual susceptibility. Gray[120] found susceptibility differences of 2:1 and 5:1 with age and body type alone, but differences of 8:1 could be distinguished with age and body type together. Lam and Yau[118] controlled for the effects of multiple variables by logistic regression and found increased individual susceptibility associated with body mass index, previous DCS incidents, and a job as an engineer or miner.

In summary, there is strong evidence that individual factors affect one's susceptibility to VGE and DCS, but many of these effects have been obscured by data and analysis that lack experimental or statistical controls, particularly for differences in exposure.

DECOMPRESSION SAFETY

The problem of decompression safety has been addressed empirically with considerable success for over 100 years, and similar valuable efforts will undoubtedly continue. Although the empirical approach is "good enough" for many practical purposes, it is frustrating and inefficient in the long run. Decompression safety is an unfinished task. More effective procedures are needed to avoid DCS, and improved therapy is needed when it does occur. The following discussion emphasizes the evidence-based approach that has been so successful in such areas of medical research as cancer and heart disease but was only recently applied to environmental physiology and hyperbaric medicine.[128]

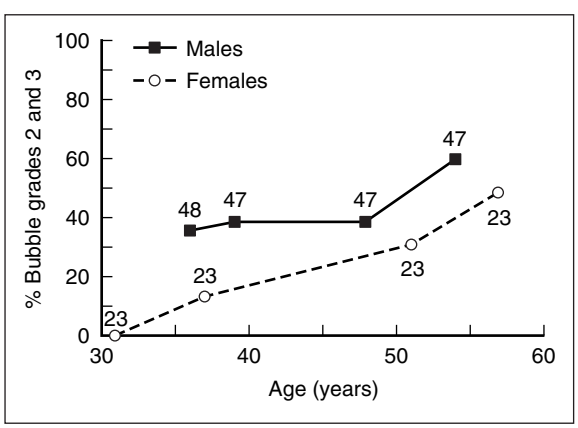

Figure 7–23. The incidence of Doppler bubbles for recreational divers as a function of age and gender.[58] The incidence of Doppler bubble grades 2 and 3 increased with age and was greater for males than for females.

Classification of Decompression Injuries

Medical personnel with adequate training and experience in diving medicine have little difficulty in diagnosing a decompression injury for the purpose of deciding on recompression therapy. Suspicion of decompression injury generally leads to recompression (if there are no contraindications), with the final diagnosis pending therapeutic outcome. A more difficult problem, and the issue addressed in this discussion, is a diagnostic system for guiding epidemiologic research, particularly for differentiating arterial gas embolism (AGE) from DCS. This is necessary to:

- Evaluate therapies that might be specific for each form of injury
- Prevent spurious cases of AGE from confounding the development of decompression procedures
- Support prognostic decisions such as whether and how to evacuate a patient with a decompression injury from a remote location

The classification of decompression disorders has evolved with three entangled purposes: therapeutic, occupational, and investigational. The U.S. Navy classified decompression injury as DCS or AGE since at least 1945 and described DCS as "pain-only" or "serious symptoms."[129] The point was to select appropriate therapy. Pain-only DCS was treated with air at 100 fsw (30 msw; 6 ata) on Treatment Table 1, and serious symptoms were treated with air at 165 fsw (50.5 msw; 6 ata) on Table 3. For AGE that occurred principally during submarine escape training, treatment was mandatory at 165 fsw on Table 3 or 4.

The terms *type 1* and *type 2 DCS* were introduced by the United Kingdom Medical Research Council (MRC) Decompression Sickness Panel to classify injuries sustained by compressed air workers.[130] Men with type 2 injuries were forbidden from further employment in compressed air. A report on the construction of the Dartford tunnel was the first publication to classify DCS as type 1 or type 2.[130] In this paper, type 1 DCS was described in 650 men with pain in and around the joints, and type 2 was described in 35 men who had symptoms other than pain or who had abnormal physical signs. Two men with lung cysts were considered to have type 2 DCS, although the disease described appears to have been AGE. The MRC classification did not distinguish between AGE and DCS because its purpose was occupational health and safety rather than identifying causes. This approach reflected an opinion stated later that the "differential diagnosis between decompression sickness and pulmonary barotrauma is not an urgent problem as the immediate treatment of both conditions is the same."[131]

With the introduction of the U.S. Navy Oxygen Treatment Tables, pain-only symptoms were treated with recompression to 60 fsw (18 msw; 2.82 ata) on the 135 min Table 5; serious symptoms were treated at 60 fsw on the 285 min Table 6.[132] AGE continued to require treatment at 165 fsw with the introduction of Tables 5A and 6A. The U.S. Navy later adopted the MRC terminology and began to refer to type 1 and type 2,[133] but etiology remained the guiding principle for selecting therapy for many years.[134]

Francis and Smith[39] questioned the value of etiology-based therapy because:

- AGE and type 2 DCS can be etiologically indistinguishable.
- AGE can result from arterialized VGE as well as from pulmonary barotrauma.[135,136]
- Animal data and clinical experience suggested that AGE and neurologic DCS responded equally well during therapy at 60 fsw.[137]

If the traditional classifications—type 1 DCS, type 2 DCS, and AGE—have neither etiologic nor clinical utility, Francis and Smith[39] argued they should be combined and proposed to include all three under the name *decompression illness* (DCI). They reasoned that real progress in understanding causes of decompression injury would come from minimizing examination bias by defining a core body of information to describe each case and standardize physical examination. When cases were distilled into the traditional classifications, much of this essential information was lost.

This therapeutic recommendation has been adopted widely. The U.S. Navy, for example, dropped the requirement for treating AGE at 165 fsw.[133] The proposal to replace the terms AGE and DCS by DCI has been more controversial. Distinguishing AGE from DCS may be unnecessary for the clinical management of decompression injury, but such distinction remains a valid goal for understanding cause, pathology, and prognosis and for improving therapy and decompression procedures.

Diagnosing Decompression Sickness and Arterial Gas Embolism

The nonspecific nature of AGE and DCS suggests an exclusionary process for diagnosis (Fig. 7–24). Step 1 is to gather information that describes a case. Step 2 involves differential diagnosis to judge whether the case is DCI or involves another cause. If DCI is not excluded, the patient enters the clinical treatment phase, undergoes recompression, and is treated according to clinical response. Following treatment, the purpose of diagnosis is to complete insurance claims or study DCI. Step 3 is to gather information about therapy and outcome. Step 4 is to judge whether AGE and DCS occurred simultaneously.[138] If not, Step 5 tests whether AGE occurred alone; if AGE is ruled out, Step 6 is to judge whether the case was one of DCS.

Clinical judgment based on adequate training and experience in diving medicine is essential for physicians who execute the process shown in Figure 7–24. The case description of Step 1 must include enough information or the diagnosis will be uncertain, if not impossible. Experience indicates that the important information includes:

- A measure of the diving exposure
- The patient's medical history
- The onset times of signs and symptoms after the exposure
- The time and nature of each therapeutic intervention
- Signs and symptoms determined by physical examination before and after each intervention

Laboratory or imaging investigations also may be helpful.

Step 2 of Figure 7–24, differential diagnosis of DCI, begins with determining whether a decompression exposure actually occurred because DCI can be ruled out in the absence of such an exposure. Next, other causes are ruled out by medical history, manifestations uncharacteristic of DCI, concurrent illness, pharmacologic effects, or underlying medical or psychiatric conditions. Table 7–1 summarizes terms used to describe DCI characteristics by three groups of diving physicians: U.S. Navy Diving Medical Officers reporting on 434 definite cases of DCS and 464 marginal cases during experimental dive trials[139]; a workshop on describing DCI[39]; and civilian physicians reporting on 474 recreational divers treated for suspected DCI.[140] Table 7–1 indicates commonality among the three groups but differences in specific terms.

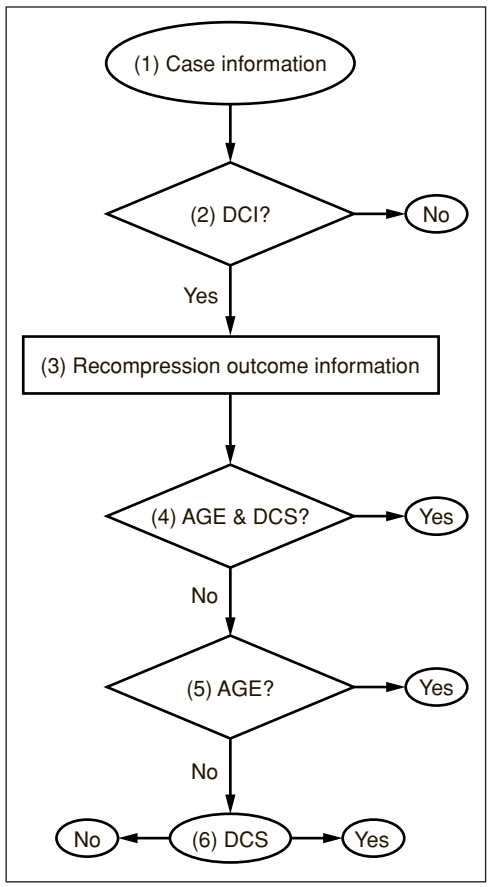

Figure 7–24. Decision tree for the exclusionary diagnosis of arterial gas embolism and decompression sickness.

Differentiating AGE from DCS requires additional information about the diving exposure because AGE can occur after virtually any compressed gas dive, whereas DCS is unlikely above a minimum (although uncertain) exposure. The best exposure information is a recording of the depth-time profile, and this is sometimes available from patients who bring their downloadable dive computers to the recompression chamber; more often, however, a poorly remembered dive profile may be all that is available for differentiation and to decide whether the profile was severe enough to cause DCS.

The end points for investigating DCI are clinical progress and therapeutic outcome. Relevant information about outcome, Step 3, includes the times, durations, and dosages of oxygen and recompression as well as any adjuvant therapy (see Chapter 10). The time course with which signs and symptoms

Table 7–1. Terms used to describe decompression illness by the U.S. Navy (USN),[139] Decompression Illness Workshop (DCI),[39] and Divers Alert Network (DAN)[140,163]

	Manifestation		
Manifestation Category	***USN***	***DCI***	***DAN***
Higher function	Mental sluggishness, poor concentration, memory lapse, "dopey," groggy, convulsions	Aberration of thought, memory loss, personality change, dysphasia, altered consciousness, seizures	Mental status, personality change, dysphasia, calculation, consciousness, mood, orientation, alertness
Coordination	Romberg sign, unsteadiness		Coordination, ataxia, gait, balance, Romberg sign
Vision	Blurred vision, visual haziness, scotoma, diplopia	Visual impairment	Visual fields, diplopia
Hearing and inner ear	Tinnitus, hearing loss, vertigo, nystagmus	Tinnitus, hearing loss, vertigo, nystagmus	Tinnitus, hearing loss, vertigo,
Motor*	Tired feeling or "heaviness" in limb, paresis, decreased strength	Motor weakness, strength, cranial nerves	Bladder or bowel dysfunction, motor weakness, paresis, paralysis, paraplegia, erectile dysfunction, hemiparesis, reflexes
Cardiorespiratory	Dyspnea, postural hypotension, chest tightness, chest pain on inspiration	Cough, shortness of breath, chest pain, hemoptysis, cyanosis, subcutaneous emphysema, pneumothorax, voice change	Shortness of breath, respiratory distress, cough, hemoptysis, voice change, cyanosis, postural hypotension
Sensory*	Paresthesia, numbness, tingling, cold or burning sensation, "pins and needles," hypersensitivity, anesthesia, sensory deficit, decreased sensation, proprioception	Paresthesia, numbness, temperature sensation, vibration, proprioception	Paresthesia, numbness, tingling, abnormal sensation, decreased skin sensitivity
Pain*	Joint, muscle, abdominal	Girdle, limb	Joint, muscle, girdle
Lymphatic*	Swelling	Swelling, enlarged or painful lymph node	Edema, swelling, enlarged or painful lymph node
Constitutional	Headache, nausea, fatigue, general weakness, cold sweat, pale, lightheadedness, malaise	Headache, nausea, excessive fatigue, anorexia, vomiting, malaise	Headache, nausea, fatigue, vomiting, dizziness, diaphoresis, malaise, restlessness, anorexia, lightheadedness, heaviness
Skin*	Itching, rash, pruritis, mottling, marbling, erythema	Itching, redness, marbling	Itching, rash, burning, marbling, urticaria

*Specify location.
Onset time is required for all manifestations.

resolve in relation to therapy is essential, but relief by recompression is not absolute confirmation of DCI because nondiving injuries may benefit from hyperbaric oxygen and DCI often resolves spontaneously. Moreover, incomplete relief upon recompression, especially after a long delay, does not necessarily exclude DCI because bubbles can produce persistent secondary damage (see Chapter 8).

The exclusionary process of Figure 7–24 depends on explicit criteria for each step. Two sets of exclusionary criteria are given in Table 7–2, one developed by the U.S. Navy

Table 7–2. Criteria for excluding decompression illness (DCI), arterial gas embolism (AGE), and decompression sickness (DCS) according to the U.S. Navy[139] and Divers Alert Network[140]

Illness†	Category	Agency	Exclusion Criteria*
(2) DCI	Exposure	USN	None: DCS risk was significant in all cases
		DAN	Single dives to < 30 fsw were excluded unless cerebral symptoms were present
	Patient history	USN	Sharp pain consistent with joint pain or impact injury
			Joint pain or fatigue, mild and consistent with recent exercise
		DAN	History indicates a likely nondiving cause
	Symptom onset time	USN	>24 h
		DAN	>48 h
	Signs and symptoms	USN	Skin itch in dry chamber and He-O_2 dives
			Headache, typical and common for the diver
			Vague abdominal or chest pain not related to trauma or barotrauma
			Dyspnea from barotrauma or anxiety hyperventilation syndrome
			Inner-ear signs and symptoms clearly due to barotrauma
		DAN	Signs and symptoms related to concomitant illness or not characteristic of DCI
			Bilateral tingling or numbness without objective signs
(4) AGE and DCS	Symptom onset time	n/a	Cerebral signs or symptoms at >15 min
			No signs or symptoms relating to sensation, strength, or pain
	Resolution	n/a	<15 min
(5) AGE	Symptom	USN	None: AGE is unlikely during chamber dives with Navy divers
	Onset time	DAN	>15 min post dive
			No cerebral symptoms, signs, or findings
	Resolution	USN	n/a
		DAN	Spontaneous resolution in <15 min
(6) DCS	Resolution	USN	Vague symptoms not responding to recompression within 18 h
			Mild pain persisting for <60 min in one joint or for <30 min in multiple joints
			Moderate pain persisting for <30 min in one joint or for < 15 min in multiple joints
			Severe pain persisting for <15 min in one joint or for <8 min in multiple joints
		DAN	Spontaneous resolution in <60 min
			Spontaneous resolution in <20 min with surface oxygen breathing

*Meeting a single criterion is exclusionary.
†(2), (4), (5) and (6) refer to steps in the decision tree of Figure 7.24

for diagnosing DCS after experimental chamber dives[139] and the other developed by the Divers Alert Network (DAN) to diagnose DCI and to distinguish AGE from DCS in recreational diving.[140] The exclusionary criteria of Table 7–2 apply to Step 2 (Figure 7–24) and depend on information concerning exposure, patient history, symptom onset time, and signs and symptoms. In applying these criteria to dive trials, none of the Navy divers was excluded for insufficient exposure because all had been subjected to significant DCS risk. The DAN divers also had been exposed to compressed gas, although not necessarily on dives sufficient to cause DCS. DAN cases involving single dives to less than 30 fsw (9 msw) were excluded from DCI unless cerebral signs or symptoms indicated that AGE might have occurred.

U.S. Navy exclusions for DCI that were based on patient history included the possible effects of recent exercise; for DAN cases, exclusions were based on possible nondiving causes (see Table 7–2). The Navy considered symptom onset times greater than 24 hours to exclude DCI; DAN excluded cases with onset times greater than 48 hours. These differences reflected Navy exposures that occurred on a

single day and DAN exposures that were multiday and often involved flying after diving. The Navy also excluded cases that involved only vague symptoms or symptoms clearly related to aural barotrauma or hyperventilation. DAN excluded cases with symptoms not characteristic of DCI (as indicated by Table 7–1) or that were possibly related to concomitant illness. None of the Navy cases was excluded from being DCI, whereas 60 DAN cases were excluded because information was insufficient to allow diagnosis.

Neither the Navy nor DAN addressed Step 4 in Figure 7–24, the simultaneous occurrence of AGE and DCS, also known as type 3 DCS.[138] In Table 7–2, this severe form of DCI was excluded for cases that did not involve signs or symptoms and onset times compatible with both AGE and DCS.

The Navy did not discriminate for AGE, Step 5 of Figure 7–24, because this possibility was unlikely during chamber dives with Navy divers. For open-water divers, DAN excluded AGE when the onset of cerebral symptoms occurred at more than 15 min after a dive and for symptoms that resolved spontaneously in less than 15 min. Twenty-nine cases were classified as AGE.

For DCS, Step 6 of Figure 7–24, DAN ruled out cases that resolved spontaneously within 60 min or within 20 min for divers who received 100% oxygen at sea level. The Navy excluded vague symptoms that did not respond to recompression given within 18 hours of a nonsaturation dive. All remaining cases were considered DCS or marginal DCS. Marginal DCS (also known as *niggles*[14]) included moderate or severe fatigue; skin itch in immersed, air, or N_2-O_2 divers; skin rash or mottling unless combined with nonpersistent joint pain; and joint pain that resolved spontaneously within the time limits in Table 7–2.

The diagnosis of DCS is usually obvious, but some cases are ambiguous, and the decision tree of Figure 7–24 and exclusionary criteria of Table 7–2 constitute a coarse filter that does not recognize diagnostic uncertainty. With the DAN criteria of Table 7–2, for example, there would be no difference between DCS involving paraplegia with onset 30 min after a dive and a case of mild joint pain at 36 hours. To account for this uncertainty, Neuman[142] suggested that case characteristics be assigned points in which the point total represents a measure of diagnostic certainty. A large total would be relatively specific and select few false-positive cases. A small total would be relatively sensitive and select few false-negative cases. An investigator could choose the total score appropriate for the study's purpose or could analyze at several levels of certainty to assess the importance of diagnostic certainty.

To maximize utility, exclusionary criteria should be developed and validated by community consensus, as was done for the definitions of psychiatric illnesses[143] and acute mountain sickness.[144] The need for consensus also applies to terms that describe DCI (see Table 7–1). These terms should be suitable for translation into other languages to allow comparisons of data from around the world.

The Morbidity of Decompression

The term *morbidity* is used in this discussion to indicate the overall incidence of DCS for all nonfatal, unrestricted exposures in a diving population. The term distinguishes the population risk from the DCS probability, which measures the risk of an individual dive. Table 7–3 lists DCS morbidity rates for air and nitrogen-oxygen diving as reported by various agencies. The morbidity in terms of the number of DCS incidents per 10,000 dives ($DCS/10^4$) was as follows:

- For scientific diving, 0 to 2.7
- For recreational diving, 1.0 to 8.4
- For commercial diving, 4.7 to 30.7
- For U.S. Navy diving, 2.9 to 127.0
- For military dive trials, 435.8.

The military dive trials were conducted by the U.S. Navy, British Navy, and Canadian forces to develop air and nitrogen-oxygen diving procedures.[139] Morbidity is often high in experimental trials when their purpose is to establish operational exposure limits.

For U.S. Navy diving, morbidity rates were determined from the following operational records: 2.9 $DCS/10^4$ refers only to no-D dives at 21 to 55 fsw (6.4 to 16.8 msw) in 1990 to 1994[155]; 3.0 $DCS/10^4$ refers to all no-D dives in 1972 to 1973[156]; and 127 $DCS/10^4$ refers to decompression dives and dives at the no-D limits in 1971 to 1978.[157]

The dive profiles for most populations in Table 7–3 are unknown, and a wide variety of procedure were used; however, for several populations, separate data were available for no-D dives, in-water decompression dives,

Table 7–3. Published and reported morbidity of various diving populations using air and nitrogen-oxygen breathing gases

No. of DCS Cases	No. of Dives	No. of DCS Cases per 10,000 Dives	Description	Reference
0	39,057	0	Scientific (1990–2000)	H. Lang (personal communication)
25	143,839	1.7	Scientific (1989–2002)	S. Sellers (personal communication)
7	26,274	2.7	Scientific (1985–95, 1998)	Vann et al.[149,150]
14	146,291	1.0	Recreational (2001)	Ladd et al.[145]
25	238,501	1.1	Recreational (1992–96)	Hart et al.[146]
67	198,167	3.4	Recreational (1994–98)	Dear et al.[147]
22	37,000	5.9	Recreational (1995–2001)	Vann et al.[140]
84	~100000	8.4	Recreational (1989–95)	Arness[148]
25	52,692	4.7	Commercial (1993–95)	Luby[151]
20	22,000	9.1	Commercial (no dates)	Beyerstein[152]
31	26,296	11.8	Commercial (1986–90)	Overland[153]
68	32,908	20.7	Commercial (1987–90)	Mills[154]
79	25,740	30.7	Commercial (1982–83)	Shields and Lee[116]
48	163,400	2.9	USN operations, No-D dives to < 56 fsw (1990–94)	Flynn et al.[155]
35	114,926	3.0	USN operations, No-D and decompression (1972–73)	Berghage et al.[156]
205	16,147	127.0	USN operations, No-D limits and decompression (1971–78)	Berghage and Durman[157]
338	7,755	435.8	Experimental chamber trials (1944–94)	Temple et al.[139]

DAN, Diver's Alert Network; DCS, decompression sickness; NOAA, National Atmospheric and Oceanic Administration; No-D, no-decompression.

and surface decompression dives. The morbidity rates of these groups are shown in Table 7–4. For six of seven no-D dive populations, the morbidity rate was 0.0 to 2.9 DCS/10^4 or similar to that for recreational diving (1.0 to 8.4 DCS/10^4; see Table 7–3). The seventh group only included dives made in 1971 to 1978 to the full extent of the U.S. Navy no-D exposure limits[115] and carried a morbidity rate of 134.7 DCS/10^4. This observation suggested the hypothesis that DCS risks at the U.S. Navy no-D limits were many times greater than for unrestricted no-D diving within the bounds of the Navy limits. The hypothesis was supported by a Navy study of all operational no-D dives from 21 to 55 fsw (6.4 to 16.8 msw) in which the DCS risk increased with dive time and depth.[155] The morbidity rate for bottom-time quartiles in this depth range increased geometrically (2.2, 2.4, 5.8, and 12.8 DCS/10^4). The morbidity rate for the deepest dives (51–55 fsw; 15.6–16.8 msw) was 7.3 DCS/10^4 dives and far exceeded the 2.8 DCS/10^4 dives morbidity rate for shallower dives (21–50 fsw; 6.4–15.3 msw).

There appeared to be little difference in the morbidity rates of no-D dives at the U.S. Navy exposure limits (134.7 DCS/10^4) and in U.S. Navy decompression dives 126.5 DCS/10^4),[157] but the morbidity rate of commercial in-water decompression dives (22 to 23.6 DCS/10^4) was lower, perhaps because commercial decompression procedures had been modified to make them more conservative than the corresponding U.S. Navy procedures.[116,153] Commercial in-water decompression dives carried a lower morbidity rate (22 to 23.6 DCS/10^4) than commercial surface decompression dives (30.1 to 49 DCS/10^4), suggesting that surface decompression carried a some-what higher risk although, as Shields and Lee[116] pointed out, surface decompression diving is generally used for more severe exposures.[116]

The National Undersea Research Center also conducted subsaturation and saturation decompression diving (see Table 7–4) (L. Horn, personal communication). The subsaturation decompression dives included air and trimix (helium-nitrogen-oxygen) bottom mixes with decompression on nitrox and 100% oxygen. No DCS occurred in 1425 dives. The saturation dives included 3592 excursion dives and saturation decompressions

Table 7–4. Morbidity rates for specific types of diving

DCS Cases	No. of Dives	No. of DCS Cases per 10,000 Dives	Description	Reference
0	39,057	0	All no-D dives, scientific (1990–2000)	H. Lang (personal communication)
1	15,094	0.7	All no-D dives, commercial (1986–90)	Overland[153]
1	8,705	1.1	All no-D dives, commercial (1982–83)	Shields and Lee[116]
17	108,705	1.6	All no-D dives, USN operations (1972–73)	Berghage et al.[156]
25	143,839	1.7	All no-D dives, scientific (1989–2002)	W. Cobb (personal communication)
0	1,425	0.0	In-water decompression, scientific, NURC (1995–2002)	L Horn (personal communication)
48	163,400	2.9	No-D dives to <56 fsw, USN operations (1990–94)	Flynn et al.[155]
13	965	134.7	Just dives to no-D limits, USN operations (1971–78)	Berghage and Durman[157]
7	38,447	1.8	All no-D dives, scientific, NURC (1995–2002)	L. Horn (personal communication)
7	26,274	2.7	In-water O_2 decompression, scientific (1985–95, 98)	Vann et al.[149,150]
10	4,548	22.0	In-water decompression, commercial (1986–90)	Overland[153]
5	2,116	23.6	In-water decompression, commercial (1982–83)	Shields and Lee[116]
192	15,182	126.5	In-water decompression, USN operations (1971–78)	Berghage and Durman[157]
20	6,654	30.1	Surface decompression, commercial (1986–90)	Overland[153]
73	14,891	49.0	Surface decompression, commercial (1982–83)	Shields and Lee[116]
3	3.592	8.4	Saturation, scientific NURC (1995–2002)	L Horn (personal communication)

DCS, decompression sickness; NOAA, National Atmospheric and Oceanic Administration; no-D, no-decompression; USN, U.S. Navy; NURC, National Undersea Research Center.

with three DCS incidents during or after ascent to sea level.

DCS morbidity is an important measure of DCS risk, but clinical severity must also be considered because serious cases are a greater health hazard than mild cases. Type 1 and 2 DCS are the commonly available measures of clinical severity, and Table 7–5 shows that the proportion of type 2 DCS ranged from 25% to 88% in 11 diving populations. In general, the proportions of type 2 DCS were smallest in military dive trials and commercial dives (25% to 44%), whereas proportions were larger (67% to 88%) in recreational, scientific, and U.S. Navy dives.

Table 7–5 also suggests that for populations with higher proportions of type 2 DCS, the chance of complete resolution after the first recompression was lower than for populations with a lower proportion of type 2 DCS. Only 25% of DCS cases in military dive trials were type 2 (despite the highest morbidity), and 83% resolved completely in a single treatment. For recreational DCS data collected by DAN, on the other hand, 69% of cases were type 2 and only 50% were completely relieved after one treatment. These differences have several possible explanations:

- The dive trial subjects were closely supervised by diving physicians, whereas recreational divers self-reported their symptoms, which may have led to under-reporting.
- Dive trial subjects may have experienced better outcomes than recreational divers because they were often treated sooner.
- Differences in dive profiles between dive trials and recreational dives may have predisposed recreational divers to serious DCS.

The chamber trials were 26% repetitive and exclusively single-day,[139] whereas recreational profiles were 80% to 85% repetitive and 40% to 50% multiday.[140,162,163] Unfortunately, the issue cannot be resolved at present because the recreational data are incomplete.

Table 7–5. Type 2 decompression sickness and recovery with therapy

DCS Cases	% DCS Type	% Complete Relief after First Treatment	Diving Population	Reference
338	24.9	83.1	Dive trials	Temple et al.[139]
31	25.8	NA	Commercial	Overland[153]
20	30	NA	Commercial	Beyerstein[152]
25	40	NA	Commercial	Luby[151]
79	44.3	NA	Commercial	Shields and Lee[116]
100	52	34.9	Recreational and civilian	Erde and Edmonds[158]
50	52	NA	Recreational and other	Kizer[159]
68	54.4	NA	Commercial	Mills[134]
279	67.0	42.3	Mostly recreational	Desola et al.[160]
1732*	69.3	50	Recreational	Vann et al.[140,161–163]
7	71.4	NA	Scientific, NURC	L. Horn (personal communication)
48	87.0	NA	USN operations	Flynn et al.[155]
25	88.0	NA	Scientific	W. Cobb (personal communication)

DAN, Diver's Alert Network; DCS, decompression sickness; USN, U.S. Navy; NURC, National Undersea Research Center.
*121 AGE incidents were also reported.

Determining Decompression Safety

Risk depends on the probability and severity of injury.[164] A high-risk activity could have a low probability of severe injury or a high probability of mild injury. Measuring risk involves estimating probability and characterizing severity. For DCS, these characteristics are inferred from population morbidity and the proportion of type 2 DCS cases.

Safety can be defined as *acceptable risk.*[164] For commercial diving, say Shields and Lee, "in considering 'acceptability' one must take into account not only the overall incidence of DCS, but also its manifestations. Pain-only limb bends, although not desirable, might be acceptable as an occupational hazard of diving; neurologic DCS, with the possibility of cumulative and perhaps permanent damage, is not."[116] An overall DCS incidence of less than 50 DCS/10^4 dives was quite acceptable for Shields and Lee, whereas "the only acceptable incidence for type 2 DCS in an occupational situation (other than the exceedingly rare fortuitous event for which no decompression procedure can cater) is zero." For those who expressed an opinion at the Repetitive Diving Workshop, 2 to 10 DCS/10^4 dives was acceptable for type 1 DCS whereas 0 to 2.5 DCS/10^4 dives was acceptable for type 2 DCS.[165]

Acceptable risk is a personal decision for an individual, but an organization determines acceptable risk for its constituents through deliberation, negotiation, and compromise. The role of science in determining safety is to develop information about risk as it relates to exposure and to make this information available to the arbiters of safety. In the absence of such information, the process can be contentious—take the example of flying after diving. Flying too soon after diving was recognized as a DCS risk factor during the 1960s, and limited data were used as the basis for a number of conflicting guidelines on how long to wait after diving before flying was safe. When the guidelines proved divisive, a workshop was held in 1989 to resolve the dispute.[166] The workshop published consensus guidelines based on expert opinion, but experts within the field of recreational diving continued public disagreement that could not be resolved by existing data. A second workshop formulated evidence-based guidelines for recreational diving when further data became available.[167,168] Guidelines should be based on science if safety is to be negotiated with minimal friction.

Estimating the Probability of Decompression Sickness: Models, Data, and Statistics

Table 7–4 indicates that DCS is not a random event because dives at the U.S. Navy no-D limits carried greater morbidity (134.7 DCS/10^4 dives) than shorter dives within the no-D limits (0.0 to 2.9 DCS/10^4

Table 7–6. Experimental no-decompression dive trial to 60 fsw and probabilities of decompression sickness estimated by USN93 and by logistic regression to raw data

Time (min)	DCS	Trials	Observed Incidence (DCS/10^4 Dives)	Logistic Model of Time (DCS/10^4 Dives)	USN93 (DCS/10^4 Dives)	Table or Computer
42	—	—	—	34	139	Aladdin
45	—	—	—	39	151	Monitor
48	—	—	—	44	162	Mares
49	—	—	—	47	166	Datascan2
50	0	11	0	49	170	DCIEM tables, Suunto Vyper
51	—	—	—	52	174	BSAC Tables, Data Master
52	—	—	—	54	177	EDGE/Skinny Dipper
55	—	—	—	64	188	PADI/DSAT tables
56	—	—	—	68	192	Aeris, Oceanic, Pelagic
60	—	—	—	86	206	U.S. and British Navys
64	—	—	—	110	220	1993 U.S. Navy tables
66	0	29	0	126	227	
80	1	14	714	350	292	
90	2	21	954	796	349	
100	2	13	1539	1862	411	

Data concerning no-decompression dive trials to 60 fsw are from reference 139; USN93 data from reference 177; dive table and computer data from reference 183.
BSAC, British Sub-Aqua Club: DCIEM, Defense and Civil Institute of Environmental Medicine; DCS, decompression sickness; DSAT, Diving Science and Technology; EDGE, Electronic Dive Guide Experience; PADI, Professional Association of Diving Instructors.

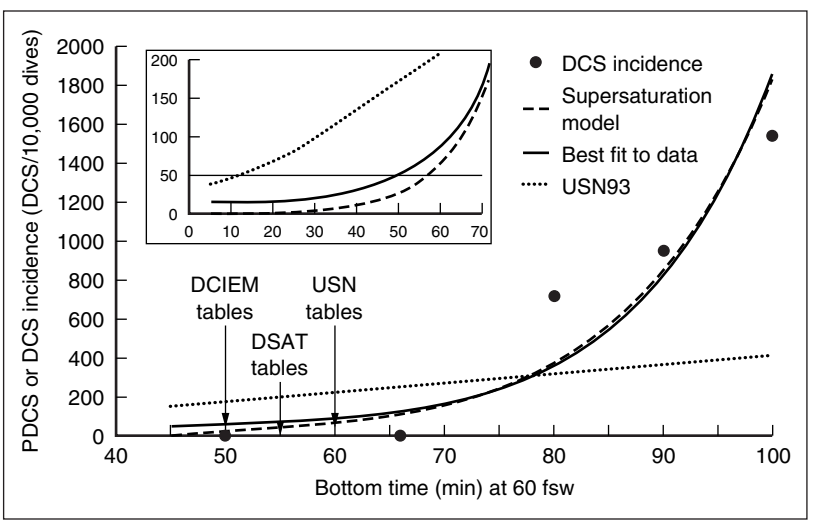

Figure 7–25. The incidence of decompression sickness (DCS) and estimated DCS probability (PDCS) for no-decompression air dives to 60 fsw (18 msw) as a function of bottom time. The *black circles* represent the DCS incidences for experimental dive trials.[139] The *lines* represent estimates of DCS probability as discussed in the text. DCIEM, Defense and Civil Institute of Environmental Medicine; DSAT, Diving Science and Technology; USN, U.S. Navy.

dives). To judge whether a dive's DCS risk is acceptable, we need to know how risk changes with depth and bottom time; this is accomplished by estimating the DCS probability (PDCS) for specific dive profiles.

PDCS is estimated by fitting statistical models to known dive profiles for which the DCS outcomes are also known. Weathersby and associates were the first to apply this approach to DCS.[169] The following discussion adopts U.S. Navy logic in assuming that both type 1 and type 2 DCS can be described by the same model, a logic that was supported by the low incidence of type 2 DCS in dive

trials and the good success of recompression therapy (see Table 7–5).[139]

Table 7–6 and Figure 7–25 provide a simple example of the process. The first three columns of Table 7–6 list the results of 88 no-D dive trials to 60 fsw with bottom times of 50, 66, 80, 90, and 100 min. These data are from a collection of 8578 experimental dives that included 434 DCS incidents and 464 marginal incidents.[139] Table 7–6 also shows the no-D exposure limits at 60 fsw corresponding to 16 dive tables and computers (columns 1 and 8).

The DCS incidence for each experimental bottom time is shown as a solid circle in Figure 7–25. The solid line is the best fit to the experimental data by logistic regression (see the side bar), which estimates PDCS as a function of the bottom time at 60 fsw. Table 7–6 shows the observed DCS incidence (column 4) and the probabilities estimated for the dive trials and for the table and computer exposure limits (column 5). According to these estimates, exposure limits of 45 min (Aladdin/Monitor) and 60 min (U.S. Navy) correspond to PDCSs of 39 and 126 DCS/10^4 dives, respectively. These probabilities are the acceptable risks estimated by logistic regression to the raw data that are implicitly associated with the tables and computers. More than 1000 dives would be required to experimentally distinguish between probabilities of 39 and 126 DCS/10^4. Consequently, uncertainty in comparing the safety of various computers and tables should not be surprising.

Note that the estimated PDCS for the U.S. Navy no-D limit of 60 fsw for 60 min (126 DCS/10^4 dives) is close to the morbidity rate observed for operational dives to the no-D limits (135 DCS/10^4 dives; Table 7–2). To achieve a PDCS that Shields and Lee[116] found acceptable for mild cases (50 DCS/10^4 dives), no-D dives at 60 fsw would have to be limited to 50 min, the exposure limit of the DCIEM tables.[108]

Logistic Regression: A Simple Method for Estimating the Probability of Decompression Sickness[170]

Linear regression finds the best agreement (or fit) of a straight line to continuous experimental data by minimizing the least-squares error between the data and the line. Logistic regression serves a similar purpose for binary experimental data, with values of 0 (no DCS) or 1 (DCS) and a sigmoidal, or S-shaped, curve having values between 0 and 1 that are interpreted as probabilities. The logistic function meets these requirements and is defined as

$$P(DCS) = 1/(1 + e^{(\beta 0 + \beta 1 \bullet x1 + \beta 2 \bullet x2 + \ldots)}) \quad (7–1)$$

where x_1, x_2, … are independent variables, and β_1, β_2, … are parameters whose values are chosen to give the best fit of the binary experimental data to the estimated probabilities. The best fit is found by adjusting the parameters to maximize the *likelihood* (analogous to least squares in linear regression), which is the product of the estimated probabilities for every observation in the data. Thus,

$$\text{Likelihood} = P_1(DCS) \bullet P_2(DCS) \bullet P_3(\text{no-DCS}) \bullet \ldots.$$

where $P_i(DCS)$ is defined by equation 7–1 and $P_i(\text{no-DCS}) = 1 - P_i(DCS)$. Since the product of many probabilities is a small number, the logarithm of the likelihood is often reported.

The simplest approach to the data of Table 7–6 is to set x_1 in equation 7–1 to the bottom time of the dives. The resulting probability estimates appear as a solid line in Figure 7–26. Logistic regression is a useful mathematical function but has no physiologic significance.

The simple relationship of bottom time to PDCS in Figure 7–25 (solid line) is helpful for illustrating the process of fitting probability functions to empirical data, but it cannot estimate PDCS for any but no-D dives at 60 fsw. A more general approach transforms dive-profile data into a computed measure of decompression stress and substitutes this stress for the variable X_1 in the logistic equation (see the side bar). For example, the 60 fsw dive profile data of Table 7–6 can be transformed into the supersaturation in a well-stirred tissue compartment (equation 4–2). The resulting PDCS estimates appear as a dashed line in Figure 7–25 and are quite close to the estimates based on the raw data (solid line). This simple example illustrates the concept of using empirical diving data to derive generalized DCS probabilities.

The simplest approach to estimating DCS probability for diving is to apply a probability model and a deterministic measure of decompression stress to empirical data as

just described. For altitude exposures such as aviation, EVA,[171] or flying after diving,[167] however, this approach is insufficient because PDCS changes with the time at altitude. In these circumstances, survival or failure time analysis can be used to estimate both PDCS and DCS onset time.[172,173] Although onset time is not as essential for modeling PDCS in diving data, onset time provides additional information that can improve model parameter estimation[172] and adds another dimension for comparing the performance of decompression models.[141] Tikuisis and Gerth describe probabilistic modeling in detail, including both incidence-only and onset-time analysis.[173]

The supersaturation model of PDCS (dashed line in Fig. 7–25) could be used to estimate PDCS for any general dive profile, but this would be an inappropriate extrapolation because the model parameters were calibrated from only the 60 fsw data (see Table 7–6). In general, statistical estimation is best when confined to interpolations within the data to which the model parameters were calibrated. The parameters of the U.S. Navy probabilistic decompression models were calibrated against thousands of experimental dives over the range of depths and times that were of operational interest, particularly for long exposures and long decompression dives. The final model (USN93) included a probabilistic version of linear-exponential inert gas exchange[141] (see Chapter 4, Inert Gas Exchange and Bubbles) and was calibrated to the DCS incidence and onset times of 2383 dives[174] and validated in 709 verification trials.[175] There was excellent agreement between predicted and observed DCS for incidences of 200 to 1523 DCS/10^4 dives.[176]

For the experimental trials at 60 fsw, PDCS estimates by the USN93 decompression model appear less satisfactory (dotted line in Fig. 7–25). Although the 88 dives of the 60 fsw data in Table 7–6 were part of the USN93 calibration data, USN93 underestimated the observed DCS incidence at 60 fsw for long bottom times and overestimated the incidence for short bottom times. For example, if the acceptable DCS risk was 50 DCS/10^4 dives, the inset of Figure 7–25 indicates that a no-D dive at 60 fsw could not exceed 17 min and it would be impossible to achieve the morbidity rates of 1.6 and 2.9 DCS/10^4 dives reported for operational U.S. Navy no-D diving (see Table 7–3).

The Navy conducted their dive trials at the limits of anticipated exposures and tolerable risks. This had historical precedent and made practical sense:

- If the most severe dives could be tested safely, less severe dives might be expected to be safe and to need less testing.
- Information relating DCS to diving exposure is obtained only when DCS occurs.
- At a cost of about $500 per dry-chamber trial and at least $1000 for each wet exposure (based on costs at Duke University), tests of low-risk dives would provide little information about DCS at high cost.

This is why the DCS incidence of military dive trials was 436 DCS/10^4 dives instead of less than 50 DCS/10^4 dives, which morbidity estimates suggest is more typical of open-water diving (see Table 7–3).

USN93 was a major advance in predicting DCS risk and provided an excellent fit to its own calibration data,[176] but it overestimated the risks of more operationally common dives such as no-D exposures at 60 fsw for less than 50 min (see Fig. 7–25). Because the calibration data involved few low-risk dives, PDCS estimates for ordinary dives were extrapolated from high-risk data. Future low-risk chamber trials are unlikely, but low-risk data can be collected inexpensively in observational field studies using depth-time recorders and recording dive computers. With a corresponding system to verify the presence or absence of DCS, low-risk observational data and high-risk laboratory data might be combined to provide risk-balanced data for model calibration.

Observational data on multiday repetitive diving might also help resolve the question of the high proportion of serious DCS and less effective therapy for open-water dives as compared with chamber dive trials (see Table 7–5). The Divers Alert Network (DAN) has embarked on a program to collect observational data (Project Dive Exploration); as of 2001, DAN had accumulated 36,711 individual dive profiles from 3787 divers in which 22 DCS incidents occurred (Fig. 7–26).[140]

U.S. Navy Probabilistic Decompression Procedures

In spite of the difficulty in accurately predicting low DCS risk, USN93 has been an important yardstick for grading dive-profile severity and a useful tool for developing decompression procedures.[177]

Upon examining the results of their experimental trials, the U.S. Navy judged that the

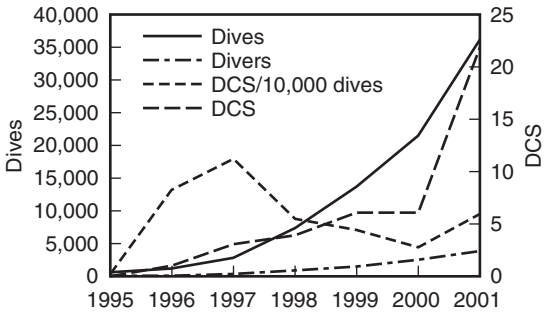

Figure 7–26. Data collection progress for Project Dive Exploration.[140] Data represented include the number of divers in the database, the number of dives collected, the number of divers who underwent recompression for decompression sickness (DCS), and the DCS morbidity per 10,000 dives.

no-D exposure limits that were acceptable corresponded to a PDCS of 230 DCS/10^4 dives. This became the "target" PDCS for dives with decompression times of 0 to 20 min.[175] For decompression times of 20 to 60 min, the target PDCS was allowed to rise from 230 to 500 DCS/10^4 dives. A "sliding" target was used because USN93 estimated much longer decompression times than the corresponding schedules in the approved U.S. Navy Standard Air Decompression Tables.[115] The Navy considered 60 min to be the longest acceptable time for in-water decompression, and dives with longer decompressions were listed as exceptional exposure. The target PDCS for exceptional exposure dives was 500 DCS/10^4 dives until the decompression time reached 180 min, after which the target increased from 500 to 1000 DCS/10^4 dives as the decompression time rose from 180 to 220 min.

The USN93 no-D exposure limits were longer than the Standard Air limits at 90 fsw (27 msw) and deeper and shorter than the Standard Air limits at 30, 35, and 40 fsw (9, 10.7, and 12 msw).[177] USN93 decompression schedules were substantially longer than the Standard Air schedules but with lower estimated PDCS.[175] Because of complexity, the USN93 decompression algorithm did not lend itself to repetitive diving according to the familiar methods of the Standard Air Tables. An alternative method was developed whereby every dive was assigned an A-to-Z "exit state" similar to the Repetitive Group of the Standard Air Tables, and a separate table of schedules was prepared for each exit state. The USN93 tables were as flexible as the Standard Air Tables but not as compact.

Ultimately, the USN93 tables were not accepted by the U.S. Navy as a replacement for the Standard Air Tables (Dr. E.D. Thalmann and Dr. E.T. Flynn, personal communication). The Navy did not perceive a problem with the existing tables that needed to be fixed, and the new tables were thought to reduce capability because:

- Shallow no-D exposure limits were shorter and might restrict a ship's husbandry diving.
- Dives that were formerly available for routine use were now classified as exceptional exposure.
- Repetitive diving procedures were complex.

These views were largely determined by Master Divers—practitioners with strong grounding in tradition. Perhaps this is as it should be. New tactics, equipment, or revolutionary concepts (such as probability in diving) are historically slow to be accepted by the military, with good reason. Change is expensive and time-consuming, and the consequences of being wrong can be catastrophic.

For those less wedded to practice and tradition, probability might be viewed differently given the uncertainty of the present U.S. Navy tables. In 1972 to 1973, only 4% of the 113,007 operational air dives conducted required decompression,[156] and this fraction was less than 4.7% in 1990 to 1994 (Dr. E.T. Flynn, personal communication). U.S. Navy dive trials found specific areas of concern:

- In tests of the 200-min no-D exposure limit at 40 fsw, two DCS incidents occurred (one joint pain, the other with cerebral findings and residual effects) in 91 trials (220 DCS/10^4 dives).[178]
- Trials of Standard Air Decompression schedules resulted in four DCS incidents in 77 trials (519 DCS/10^4 dives)[179] and suggested that some of the Standard Air Schedules would benefit from tripling the decompression time.[174]
- When DCS occurred operationally, the problem was often fixed by ad hoc reductions of bottom time or increases in decompression time.[114]

Table 7–7. Comparison of repetitive dive decompression schedules for 20-min dives to depths of 150–200 fsw according to the U.S. Navy Standard Air Tables (Std), the USN93 Tables ('93), and the INA95 Tables

First Dive						Second Dive						
	USN Stops[*]		*INA Stops*[†]		*PDCS/10*[4]			*USN Stops*[*]		*INA Stops*[†]		*PDCS/10*[4]
D	Std	'93	30′	20′	Dives	D	T	Std	'93	30′	20′	/104
150 (fsw)	9	5	0	15	115	150 (fsw)	20	21	160	0	20	94
160	14	15	0	20	105	160	38	160	0	25	86	
170	19	15	0	20	127	170	43	165	0	25	102	
180	23	15	0	20	148	180	50	NA	0	30	106	
190	28	20	5	25	129	190	60	NA		5	35	92
200	37	20	5	30	153	200	NA	NA		5	40	103

[*] Total time of all stops (min).
[†] 30′ stop on air (min), 20′ stop on O_2 (min).
NA, not allowed; PDCS, probability of decompression sickness.
U.S. Navy Standard Air Table data from reference 115; USN93 data from reference 177; INA95 data from reference 149.

Probabilistic Decompression Procedures for Underwater Archeology

Probabilistic modeling was also used to develop decompression schedules for underwater archeology. In the late 1960s, the Institute of Nautical Archeology (INA) began using in-water oxygen decompression with the U.S. Navy Standard Air Tables during the excavation of ancient shipwrecks in the Mediterranean Sea on the unofficial recommendation of Dr. Robert Workman, then Senior Medical Officer at the U.S. Navy Experimental Diving Unit.[149] Although undocumented, the success of this technique (supported by an on-site recompression chamber with medical personnel for managing diving injuries) led to a formal effort beginning in 1985 with orderly records of diving activity and, in 1988, to a series of probabilistic decompression schedules based on models.[27,180,181] Methods for introducing new diving procedures in the field were adopted as outlined by Schreiner and Hamilton,[182] including:

- Approval of an Institutional Review Board
- Approval of a Decompression Monitoring Board
- Documentation involving written dive logs
- A recompression chamber and diving medical personnel on site
- Incremental introduction of the new procedures

The INA decompression schedules were for dives to a maximum depth of 200 fsw, with bottom times of up to 40 min and oxygen decompression at 20 fsw. There were two dives per day with a 5- to 6-hour surface interval. The diving season was June through September, with 6 dive days per week. The approach to acceptable DCS risk was empirical and similar to that used by the U.S. Navy for USN93. Based on previous INA experience, a target PDCS of 150 DCS/10^4 dives was selected for the first dive and a target of 100 DCS/10^4 dives was selected for the second dive.

Table 7–7 compares the decompression schedules for 20 min dives to 150 to 200 fsw with the Standard Air schedules and the USN93 schedules. The divers breathed air during decompression at 40 and 30 fsw. All other decompression in the INA schedules occurred at 20 fsw while divers breathed 100% oxygen. In 1998, oxygen decompression schedules were introduced for dives to 120 fsw with a bottom gas of 32% oxygen in nitrogen.[150] Seven DCS incidents (3 DCS/10^4 dives) and no oxygen toxicity were reported for 26,274 dives using all INA schedules.[149,150]

CONCLUSIONS

Statistical methods used in probabilistic modeling are not wise in themselves and are simply data-fitting tools that compensate for ignorance regarding underlying mechanisms. Bubble formation, inert gas exchange, and pathophysiology cannot be assumed to be identical in the brain, spinal cord, and limbs. This is why decompression modes should represent premorbid physiology as closely as possible and why understanding this physiology has practical importance. Relating physiology to decompression safety is an epidemiologic problem associated with finding the probability of injury in the context of the individual, the environment, and the exposure. Much will be gained by formalizing operational and clinical methods and by applying analytical techniques used widely in science and medicine.

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CHAPTER 8 Pathophysiology of Decompression Sickness

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This chapter describes the physiologic and pathologic consequences of a gas phase that evolves within tissues or blood as a consequence of reduced ambient pressure. The chapter does *not* detail the effects of changes of ambient pressure on gas phases that normally exist in the body or the consequences of gas phases that evolve in tissues from iatrogenic, traumatic, or infective sources.

MECHANISMS OF DISEASE

The pivotal pathologic event in decompression sickness (DCS) is the formation of bubbles in blood or tissue from dissolved inert gas.[1] This occurs when a state of inert gas supersaturation is achieved during decompression; that is, the tension of dissolved gas exceeds ambient pressure sufficiently for bubbles to form. Multiple organs may be involved. Some, such as the lungs, are injured primarily by intravascular bubbles; others are injured by bubble formation within the tissue. Some organs, such as the spinal cord, may be affected by both intravascular and tissue bubbles.

Inert Gas Bubbles in Blood

Doppler ultrasonic devices have yielded a mass of evidence that intravascular bubbles are associated with DCS in both animals[2–7] and humans.[8–16] However, the exact mechanisms whereby intravascular bubbles are formed from dissolved gas are unknown. Physical theory predicts that the inert gas supersaturation required to overcome surface tension and form bubbles in pure solutions de novo is much greater than could be achieved in a conventional diving exposure[17]; in practice, however, venous bubbles have been detected in humans after air saturation dives at 135 kPa (3.5 msw).[18] Hypotheses to explain this discrepancy focus on bubble formation on hydrophobic surfaces[19] or the existence of preformed gaseous "micronuclei"[20–22] that are stabilized by surfactants[23] and are small enough to remain undetectable.

Although arterial bubbles have been observed in decompressed animals,[7,24–26] bubbles are unlikely to form de novo in large arteries. Inert gas supersaturation sufficient to provoke bubble formation is improbable in arterial blood because the healthy lung essentially equilibrates alveolar and arterial gas tensions in a single pass. Arterial supersaturation may occur in a very rapid ascent of 20 fsw/s^{-1} or greater,[27,28] but arterial bubbles have proven difficult to demonstrate even under these conditions.[29] In contrast, the venous end of capillary beds or venous sinusoids may provide a more suitable environment for bubble formation because the prevailing conditions are of low hydrostatic pressure and high gas tension as nitrogen diffuses out of tissues into the blood. However, this explanation is almost certainly too simplistic; Vann and Thalmann[30] summarize data that demonstrate isolated venous blood to be quite resistant to bubble formation, and complex mechanisms are almost certainly involved.

Whatever the specific location and mechanism of their formation, venous bubbles, unlike arterial bubbles, have been detected frequently in divers.[9,31–36] They range in size from 19 to 700 m,[37] and their number appears to be proportional to the decompression stress.[38] The time course of bubbling may be prolonged. In dogs subjected to a moderately severe decompression stress, central venous bubbles were detected within 5 min of decompression; bubble activity peaked at 25 min, remained stable for 1 to 2 hours, and

decreased thereafter.[39] In humans, bubbles have also been detected within minutes of diving, although latent periods of more than 1 hour have been recorded.[40] In decompression to altitude, there is some evidence that DCS symptoms develop at the peak of venous bubbling,[41] although other investigators have reported that the extent of Doppler-detected venous bubbling correlates poorly with the occurrence of DCS.[40]

Blood vessels are a target for damage by intravascular bubbles. Bubbles may injure both the luminal surfactant layer[42] and endothelial cells,[43–46] which reduces the integrity of the vessels.[42,47] Although the interactions between bubbles and blood vessels are relevant to the development of lung injury in DCS (see later), the greatest impact of these interactions is on the cerebral circulation (see Chapter 9).

Bubbles interact with formed elements of blood and plasma proteins. Bubbles may:

- Stimulate platelet aggregation[48–54] and reduce platelet count[55,56]
- Denature lipoproteins[57]
- Activate and aggregate leukocytes[50,58–60]
- Increase release of cytokines[61]
- Activate the complement,[62–66] kinin,[67] and coagulation systems[50,68,69]
- Cause both capillary leakiness and hemoconcentration by means of these proinflammatory events[70–75]

Individual differences in susceptibility to DCS might be affected by variability in the activation threshold or vigor of the humoral response to bubbles.[61,63,76] This response might also explain the failure of recompression treatment in some cases[75] because, once activated, the humoral response is unlikely to be immediately terminated by the resolution of bubbles. Not surprisingly, the response to intravascular bubbles remains a focus for the development of potential therapeutic interventions (see Chapter 10).[77–80] However, although these bubble–blood interactions are often demonstrated in vitro and in severe in vivo models of DCS, the relevance of such interactions to milder human cases is less certain. For example, it has been shown that the activation of coagulation,[81] complement,[79,82,83] and neutrophils[84] is not significant after bubble-forming decompression with or without mild DCS in humans. Similarly, although significant falls in platelet count have been detected following human dives, these are not reliably associated with symptoms of DCS.[55] Indeed, the presence of venous bubbles without clinical manifestations has long been recognized[40] and is known as "*silent" bubbling.*

In the absence of right-to-left shunting, the important target organs for damage by venous bubbles are the lungs and the spinal cord (both targets are discussed later). Despite the trapping of numerous bubbles in the hepatic sinusoids after decompression in vivo[39] and the observation of portal vein bubbles after human decompression,[85] DCS affecting the liver is not considered an important clinical entity. Portal venous bubbles may nevertheless cause subclinical liver tissue damage. Elevation of liver enzymes has been reported after severe DCS in vivo[86,87] and in human divers with and without clinical DCS.[88,89] It has been suggested that portal venous bubble impaction might impair metabolism of drugs used to treat DCS.[39]

Pulmonary DCS

The first microvessels encountered by venous bubbles are the pulmonary capillaries, and it has been demonstrated in vivo that bubbles generated by decompression or directly infused to the venous circulation become trapped there.[90–97] The time course for subsequent bubble resolution by diffusion into the alveoli is inversely proportional to the volume of embolic gas[96] but in the case of air can be accelerated by oxygen breathing.[95,98] In vivo, it is possible to establish a steady state in which the rate of venous gas infusion is equaled by its clearance by the lungs.[92]

The obstruction of pulmonary vessels by bubbles may be accompanied by damage to endothelium,[46,80,99] accumulation of leukocytes,[99,100] release of thromboxanes and leukotrienes,[101] damage to the blood-lung barrier,[102] and release of vasoactive substances.[103,104] Not surprisingly, the pulmonary artery pressure is elevated,[92,105–107] a state that may be accompanied by a decrease in cardiac output.[94,108,109] There may be hypoxemia[110] due to either a ventilation-perfusion mismatch[110–112] or pulmonary edema generated by elevated transcapillary pressure and leakage of plasma through damaged or inflamed endothelium.[95,109,113–117] Ultimately, there may be cardiac decompensation, respiratory arrest, and death.[107,118]

The extreme decompression stress, or direct venous gas infusions, used to demonstrate such manifestations in vivo are of uncertain clinical relevance, not least because overt pulmonary DCS is very rare. Pulmonary artery pressure was not elevated following human hypobaric decompressions that generated high venous bubble grades.[119] In addition, whether subclinical pulmonary injury occurs in typical human diving exposures is controversial. Neubauer and colleagues have shown that the concentration of leukotrienes in pulmonary condensate does not rise after human wet chamber dives to 50 m, despite an inevitable degree of venous bubble formation from such dives.[120] On the other hand, pulmonary CO transfer has been found to decline significantly in divers with no symptoms of DCS.[121–123]

Right-to-Left Shunting of Venous Bubbles

The lungs can trap and excrete venous bubbles. Without this capability, compressed gas diving would be associated with a much higher arterial bubble load. However, the pulmonary bubble filter may be overwhelmed by excessive venous bubbling,[91,93,94,124,125] although there appears to be both intra- and inter-species variability in the threshold.[125] Factors other than the degree of bubbling have also been identified as promoting, or being associated with, bubble redistribution through the pulmonary capillary bed. These factors include:

- Elevation of pulmonary artery pressure[124]
- Decrease in mean systemic arterial pressure[125]
- Recompression[126]
- Administration of aminophylline[93]
- Pulmonary oxygen toxicity[127]

Venous bubbles may also cross an interatrial shunt such as a patent foramen ovale (PFO).[128] In asymptomatic persons, flow across a PFO, if any, is usually from left to right (see Chapter 25). Such a shunt has to be reversed for venous bubbles to enter the arterial circulation. Butler and coworkers[129] showed that mild decompression may not generate sufficient pulmonary arterial hypertension to cause flow reversal, and Glen and associates, using Doppler, could detect no middle cerebral artery bubbles after relatively conservative dives by four divers with a PFO.[35] However, flow reversal may be achieved following diving: Vik and colleagues found arterial bubbles in all six of a group of pigs with a PFO that were subjected to severe decompression stress.[128] In addition, in contrast with the findings of Glen and coworkers, a small study by Gerriets and associates suggested that arterial emboli were more likely to be detected after decompression in divers with a PFO.[130] Factors thought to increase shunting across a PFO include lifting, straining, and coughing, but not immersion in water or exercise without lifting.[131]

There is evidence that such shunting may be important in human DCS. Using transthoracic echocardiography, Moon and colleagues demonstrated a PFO in 11 of 30 patients (37%) who had suffered DCS and in 11 of 18 patients (61%) who had suffered severe neurologic manifestations.[132] Wilmshurst and coworkers reported a similar series that included a control group of divers with no history of DCS. Fifteen of 63 controls (24%) had a PFO, compared with 41% of 61 patients who had suffered DCS and 66% of 19 patients who had suffered early-onset neurologic manifestations.[133] In a more recent study, Wilmshurst and Bryson found medium to large shunts in 52.0% of 100 divers with neurologic DCS, compared with 12.2% of 123 diver controls without DCS.[134] In a subgroup of 38 divers with spinal DCS, 26 (68.4%) were found to have medium to large PFO shunts. The same authors also found a strong association between cutaneous DCS and PFO.[135] They showed that 47 of 61 divers with cutaneous DCS had a PFO, compared with 34 of 123 divers who had never suffered DCS. Thirty (49.2%) of the 61 cutaneous patients with DCS had large spontaneously shunting PFOs, compared with 6 (4.9%) of the 123 controls. Using magnetic resonance imaging, Knauth and associates detected multiple asymptomatic brain lesions only in those divers with a large PFO.[136] Unfortunately, the lack of a nondiving control group in this study seriously limits its impact.[137] Finally, anecdotal data suggest that the relationship between serious DCS and a large PFO also holds true for hypobaric DCS. Kerut and colleagues[138] report that three of four cases of serious DCS arising from extravehicular activity simulations were found to have a spontaneously shunting PFO.

Notwithstanding these reports, caution should be exercised in interpreting the

relevance of a PFO in DCS. It is pertinent that 20% to 34% of "normal" humans have a PFO,[139] and three studies of divers who had never suffered DCS found a PFO in 27% to 31% of subjects.[135,140,141] Kerut and coworkers[142] used transesophageal echocardiography to survey three diver groups (similar in composition to those defined by Wilmshurst and associates[133]) and found no difference in the prevalence of PFO between any two groups. Even in the studies reported by Moon and Wilmshurst, divers with a PFO were not over-represented among victims of mild DCS.[132,133] This may be explained by the failure of small numbers of shunted bubbles to produce symptoms or by the failure of venous bubbles to cross a PFO in the first place,[129,143] especially if bubble numbers are low and the PFO is small. At the least, it seems clear that large numbers of uneventful dives are performed by divers with a PFO.

The evidence that PFO plays a role in the pathogenesis of DCS is mounting, but the implications for diving medical practice remain controversial. It seems inescapable that a large PFO increases the relative risk of serious neurologic DCS. However, such events are infrequent when considered against the large number of dives performed by the general diving population; also, the increment in absolute risk implied by a PFO may be small.[144–146] Using an estimated incidence of neurologic DCS of 2.28 per 10,000 recreational dives, Bove calculated an odds ratio of 2.5 for a diver with a PFO.[145] The prevalent attitude among diving physicians is that this risk does not justify bubble-contrast echocardiography screening of all divers.[137,145] However, investigation for PFO following "undeserved" DCS is widely advocated.[147,148] In this context, it seems prudent to delay exposure to bubble-contrast agents for a month after suspected vascular gas injury in order to allow inflammatory processes to settle, even though such agents do not appear to damage normal cerebral microvasculature.[149] Moon and Kisslo suggested that a diving candidate with a known PFO be counseled against diving, especially if the candidate is risk-averse[137] or if the lesion was identified after a previous episode of DCS.[150,151] However, this recommendation is not justified by the current incidence of DCS and the known prevalence of PFO.

Inert Gas Bubbles in Tissue

Although pulmonary DCS can be explained entirely by the formation of inert gas bubbles in venous blood, DCS in the other organ systems may be partly, or entirely, due to bubble formation within the tissues themselves. Bubbles in tissue are more difficult to detect than intravascular bubbles, and it therefore has proven difficult to implicate "autochthonous bubbles" as a cause of DCS. Nevertheless, events such as altitude-induced[152] or nitrous oxide–induced exacerbation of previously resolved symptoms have been observed in a fixed anatomic location[153] and suggest an autochthonous bubble cause, although such a conclusion has been challenged.[154] Tissues that are relatively poorly perfused and therefore wash out dissolved gas more slowly during decompression are considered most vulnerable to autochthonous bubble formation. Examples include spinal cord white matter, periarticular tissues, adipose tissue, and the inner ear.

Neurologic Decompression Sickness

SPINAL CORD

Arterial Bubble Embolism Hypothesis

Ever since Hill and Macleod observed the circulation in the vessels of a bat's wing and frog's web during and after decompression,[155] it has been recognized that bubbles of gas can be detected in the arterial circulation. After an extensive series of experiments using a goat model of DCS, Boycott and colleagues[156] concluded that such bubbles would grow if lipid-rich tissues were embolized and that this was probably the pathogenesis of spinal cord lesions. In more recent times, Neuman and Bove[157] have supported this hypothesis for some presentations of DCS. Dunford and coworkers[32,36,158] and Wilmshurst and Bryson[134] have provided further evidence for the presence of arterialized venous bubbles after decompression.

The pathologic findings in the spinal cord of punctate white matter hemorrhages and necrosis with pial sparing have been described as being compatible with ischemic necrosis,[159] and this has been used to

support arterial bubbles as pathogenic mediators.[160,161]

On the other hand, many of the animal studies cited have been performed on small rodent species that were subjected to near-explosive decompression insults in order to generate an injury. As described earlier, the weight of evidence from nonexplosive decompressions is that bubbles first appear on the venous side of the circulation and that arterial bubbles are rare and only associated with severe disease or right-to-left shunts.

In the absence of pulmonary barotrauma, a PFO, or other right-to-left shunt, the only other way for bubbles to appear in arterial blood is if venous bubbles traverse the pulmonary filter. As we have seen, this may occur in the presence of substantial intravascular bubbling, but this process is likely to be time-consuming[94] and accompanied by pulmonary symptoms. Thus, this mechanism is unlikely to be relevant when the onset of DCS occurs either during, or shortly after, decompression.

Even if it is accepted that bubbles formed from dissolved inert gas may appear in the arteries by de novo formation or right-to-left shunting, a further problem with the arterial bubble embolus theory relates to distribution of these bubbles. Hallenbeck and Anderson criticized embolic mechanisms of spinal cord injury in DCS by citing the apparently unique distribution of central nervous system lesions.[162] In other clinical embolic conditions such as subacute bacterial endocarditis, fat embolism, and mural thrombus of the left atrium, the brain is the principle target organ. The authors quoted Blackwood's observation that arterial embolism of the cord is extremely rare. Of the 3737 autopsies Blackwood reviewed on patients that died with neurologic diseases, he found not a single case of spinal cord embolism.[163] If emboli are responsible for the pathologic findings in DCS, it is the *brain,* rather than the spinal cord, that should be preferentially embolized because it constitutes some 98% of the mass of the human central nervous system and receives 75 to 85 times the blood flow of the spinal cord. Moreover, as is discussed in Chapter 9, bubbles of gas may be released into pulmonary capillaries as a result of barotrauma. It is surely pertinent that this almost invariably causes cerebral rather than spinal symptoms. Similar observations have been made regarding the distribution of central nervous system injury in patients undergoing cardiac surgery, who are commonly exposed to substantial numbers of arterial gas emboli arising from air left behind in the heart chambers or other sources.[164,165] Exposure to such emboli has been correlated with frequent perioperative cerebral injury,[166–170] yet such patients almost never suffer spinal injury (unless there is concomitant aortic surgery). Thus, although the brain is clearly a target for arterialized bubbles, their importance in spinal disease is much less clear.

There is also a question as to whether an embolic-ischemic mechanism is compatible with the pathologic appearance of spinal cord DCS. There is evidence that the gray matter, rather than the white matter, is preferentially injured by both ischemia[171] and gas emboli.[172] In a canine model of DCS in which the onset of dysfunction was delayed (30 min), spinal cords showed no histologic evidence of the white matter hemorrhages consistently found in the short-latency disease.[173] This indicates that the mechanism in nonfulminant DCS may be different from that of short-latency disease and possibly compatible with the subtle acute histologic changes that occur with ischemia following bubble embolism.

Marzella and Yin have questioned whether ischemia plays a significant role in the pathogenesis of spinal cord DCS.[174] They used microspheres to show that lumbar spinal blood flow in a small rodent model increased during the onset of disease. Although it is unclear whether the lumbar cord was injured in these animals and the microsphere technique has insufficient resolution to detect the focal ischemia that is likely to occur in DCS, these findings challenge global ischemia as a mechanism for the disease.

Other Embolic Theories

End[175,176] proposed that an initiating event in DCS is the agglutination of formed blood elements by some undisclosed mechanism during decompression. He proposed that these aggregates then act as emboli. Certainly, rheologic changes occur in DCS. As we have seen, increased hematocrit and a loss of plasma volume are common in both animals and humans. This tends to increase

blood viscosity and reduce tissue perfusion. The aggregation of blood components such as platelets[48–54] and leukocytes,[50,59,60] the formation of rouleaux,[73] and the finding of endothelial cells,[50,177] fat, and bone marrow emboli[177–182] have all been described. However, these phenomena may be explained as being secondary to the nucleation of bubbles in blood or bone marrow and need not be primary events in DCS. Furthermore, as Walder observed, the sludging of blood occurs in other conditions without resulting in the manifestations of DCS.[183] An example is disseminated intravascular coagulation (DIC), in which many of these hematologic events occur on a considerable scale. However, the more common consequences of DIC (hemorrhagic necrosis of the gastrointestinal mucosa, congestion of the abdominal viscera and microscopic occlusion of capillaries by thrombi with surrounding secondary, focal necrosis) are not typical of DCS. Furthermore, spinal cord involvement in DIC is most unusual.

A consequence of the development of the cardiopulmonary bypass technique for open-heart surgery was to impose massive rheologic changes on the patient. These include the denaturation of plasma proteins, the clumping of formed blood elements, and the generation of fat emboli.[184] Bubble oxygenators in the cardiopulmonary bypass circuit cause arterial gas embolism,[185,186] and although the technology has improved, patients continue to be exposed to bubbles from other sources.[164,165] In some respects, therefore, this insult produces functional disturbances similar to those occurring in experimental models of DCS. As we have seen, the brain (rather than the spinal cord) is the target of these disturbances. Thus, even if rheologic changes were an initiating event in DCS, it is unlikely that they could account for spinal cord injury.

Finally, an observation that is difficult to explain using a theory based on the impaction of solid emboli as the principal pathologic event is the dramatic improvement in DCS that often occurs with recompression. If embolic phenomena are responsible for the condition, this observation would be more readily explained by compressible, gaseous emboli.

Venous Infarction Hypothesis

Haymaker and Johnston[187] raised the theoretical possibility that under conditions of extreme DCS, bubbles in the epidural vertebral venous plexus (EVVP), combined with back pressure from bubble-laden lungs transmitted through venous anastomoses between the spino-vertebral-azygous and pulmonary vasculature, may cause venous engorgement of the spinal cord. Haymaker developed the hypothesis after noting Batson's observation that the EVVP is a large, valveless, low-pressure system that would make it a favorable site for the formation of bubbles.[188–190]

Hallenbeck and associates went further.[191] They reasoned that gas bubbles are not inert in the blood stream, but—as a result of a 40 to 100 Å layer of electrokinetic forces at the blood-gas interface—they cause structural alterations in plasma proteins. This may result in the activation of the coagulation, complement, and fibrinolytic cascades; the release of kinins; and complex alterations to hemodynamics. The authors demonstrated that one of these systems, the coagulation cascade, was accelerated by the presence of bubbles.[68] By direct visualization of the venous drainage of the spinal cord in an animal model of DCS, they demonstrated many elements of the hypothesis that bubbles accumulate in the venous drainage of the cord; the presence of these bubbles, combined with the activation of clotting, resulted in slowing and eventual cessation of venous outflow. This, the authors observed, causes congestion and ultimately venous infarction of the spinal cord.[74,192–195] In support of this view, they considered that the scattered, punctate, mainly white matter hemorrhages of DCS were compatible with the venous infarction of the spinal cord described by Henson and Parsons.[196]

This theory also has its shortcomings. First, there is some doubt that the characteristic lesions of spinal cord DCS are compatible with a venous infarction mechanism.[197] In rats, for example, obliteration of the EVVP is associated with vasogenic edema of white matter but not frank infarction,[198] although Martinez-Arizala and colleagues described hemorrhagic tissue necrosis as occurring at 24 hours and involving the gray matter more than the white.[199] Again, in monkeys, it is principally the gray matter that is involved.[200] In humans, hemorrhage in the spinal cord that is associated with venous obstruction tends to be massive and centrally located and involves both gray and white matter.[201] Venous infarction of the spinal cord is a very rare condition,[202]

perhaps because the EVVP, being an extensive plexus, is difficult to obstruct. If this plexus were to be completely blocked at any given level, it is probable that the resulting venous congestion and infarction would be more extensive than what occurs in DCS. Even obstruction at the level of the radicular veins might be expected to result in one or more lesions with a segmental distribution. As we have seen, such a distribution is not typical of the lesions of DCS.

Another problem with the venous infarction mechanism relates to the frequent finding of "silent" intravascular bubbles in asymptomatic divers[31–36] and in cases of pulmonary DCS, particularly in aviators, that involve no spinal symptoms.[203] How is it that silent bubbling, which presumably provokes rheologic changes similar to those of symptomatic bubbling, fails to compromise spinal cord drainage? Although it may be argued that such bubbling fails to exceed some arbitrary threshold, it is difficult to understand why aviators with sufficient venous bubbling to cause "chokes" invariably do not also suffer spinal cord injury.

Activation of Complement

As we have discussed, studies in both rabbits and humans have shown that the activation of the complement system may be an important event in the generation of the symptoms of DCS. However, in recent studies, complement activation was not significant in humans despite venous bubble detection[82,83] or symptoms of DCS.[79] Furthermore, treatment of rats with a soluble complement receptor (sCR-1), which has been shown to be beneficial in complement-dependent disease, failed to prevent DCS.[77] Similarly, pre-decompression administration of the anti-C5a antibody failed to protect the pulmonary artery endothelium[80] or the blood-brain or blood-lung barriers in a rabbit model of DCS.[204] Finally, the comparison with cardiopulmonary bypass is again pertinent because cardiopulmonary bypass has been shown to activate complement in a manner similar to that of decompression, yet without generating a syndrome similar to DCS.

It has been claimed that variation in susceptibility to DCS in both rabbits and humans correlates with the sensitivity of the complement system to activation by bubbles.[63,76] However, other workers have questioned the validity of these conclusions on the grounds that the extent of complement activation varies greatly over time and thus predicting susceptibility to DCS on the basis of a single measurement cannot be justified.[205] Furthermore, a recent human study involving repetitive dives showed no association between the activation of complement in vitro and clinical disease.[79] Thus, although complement may be activated in DCS, its role in the development of the manifestations of the condition remains far from clear. With respect to the spinal cord, it has never been shown how the activation of complement could result in the characteristic lesions of DCS.

Autochthonous Bubble Hypothesis

Another possible mechanism whereby the spinal cord may be injured in DCS is through the liberation of a gas phase in situ. This theory is attractive because the spinal white matter in which most of the characteristic punctate hemorrhages, spongiosis, axonal swelling, and myelin degeneration appear[206] contains lipid-rich myelin with a high solubility for inert gas and has relatively low blood flow. Sharp and Broome[207] point out that these tissue characteristics could be expected to favor bubble formation during decompression, although their work using a porcine model of DCS failed to show a correlation between regional spinal cord lipid content and white matter hemorrhages.

The first serious proposal of an autochthonous bubble mechanism was by Keyser,[208] who noted Vernon's observation that fat can absorb five times more nitrogen than water.[209] He went on to propose that bubbles of nitrogen may form in many fatty tissues following decompression and, although such bubbles occurring in adipose tissue or omentum may be asymptomatic, those forming in spinal cord white matter are likely to cause a neurologic deficit.

More recently, Hills and James, following a study of the mechanical properties of the spinal cord, proposed that spinal cord ischemia could result if, during decompression, enough gas bubbles nucleate to increase spinal cord volume by 14% to 31%. They argued that such a volume increase would raise the tissue tension sufficiently to collapse the arterioles and cut off the blood supply.[210]

The major problem with the autochthonous bubble theory has been that until the late 1980s, except for the observations of

Boycott and coworkers[156] in the goat and vague references to "air lacerations" or "stippling" of the white matter in early descriptions of human DCS,[211,212] extravascular bubbles in the spinal cord had rarely been described. In animals, the evidence was limited to the finding of bubbles scattered throughout the spinal cord white matter of 6 of 16 dogs with fatal DCS[213] and in the cords of decompressed fingerling salmon.[214] In humans, nonstaining round spaces were described in the cerebral and spinal cord white matter of a diver who died shortly after taking only 20 minutes to surface from a 4-hour dive to a depth of 40 m.[215] Numerous similar lesions were described in the cerebral white matter of two scuba divers who had apparently died prior to being brought to the surface from 140 ft.[216] Sadly, the spinal cords were not examined.

A possible reason why autochthonous bubbles have so rarely been demonstrated is that their presence in the cord may be transitory. Sykes and Yaffe examined the spinal cords of dogs that had been perfusion-fixed following recompression treatment for DCS (3 or more hours after the diagnosis).[217] Although they described abnormalities of myelin that may have been a consequence of local bubble formation, no overt bubbles could be demonstrated by light or electron microscopy.

In the mid-1980s, Francis and associates adapted a well-established canine model of severe DCS that had been employed for the assessment of therapeutic interventions in acute DCS.[218] Fixation of the tissue within about 20 minutes of the diagnosis of the condition allowed the demonstration of very early changes. The authors found that by embedding the tissue in epoxy resin, nonstaining space-occupying lesions (NSSOLs) (Fig. 8–1) could be found in the spinal cord white matter of animals with DCS. NSSOLs were not found in undived control specimens or dived specimens in which no loss of

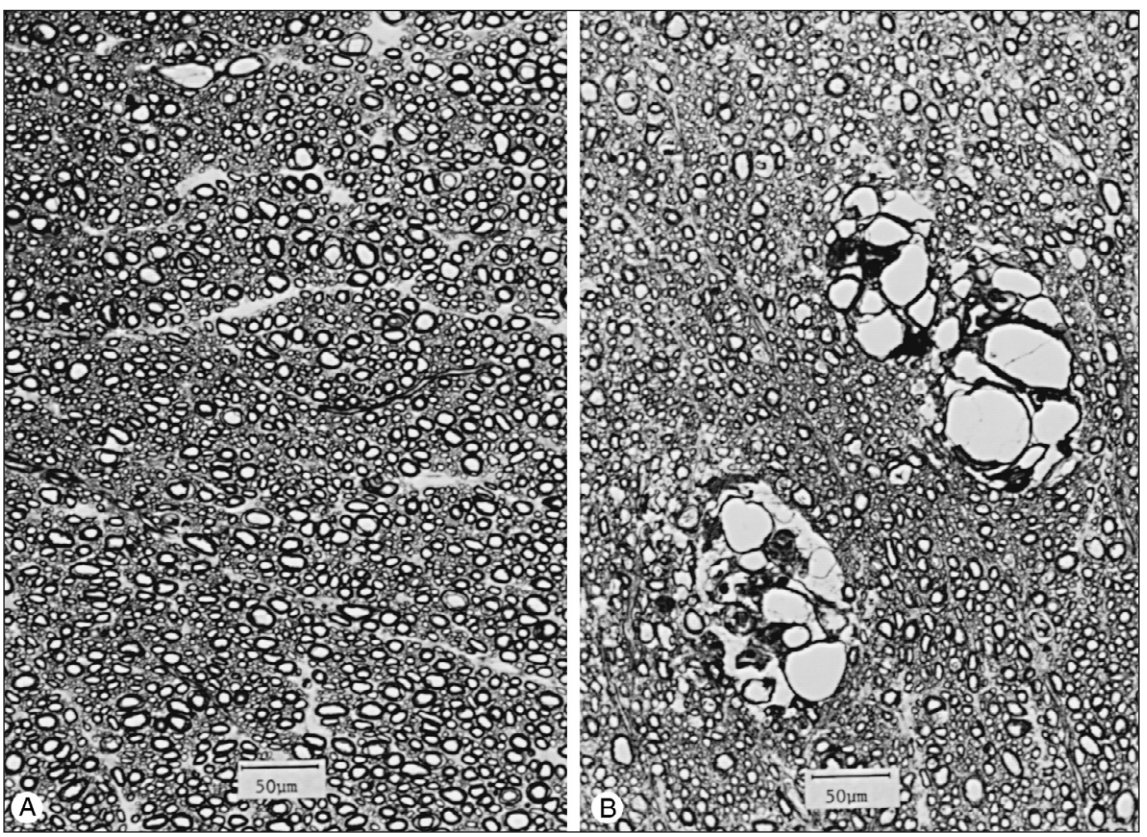

Figure 8–1. Canine spinal cord white matter stained with multiple stain solutions (bar = 50 µm). *A,* Control. *B,* Spinal cord rapidly fixed after the onset of decompression sickness. Large nonstaining spaces contain disrupted myelin figures and some compression of normal surrounding tissue. (Methodology described in Francis TJR, Pezeshkpour AH, Dutka AJ, et al: Is there a role for the autochthonous bubble in the pathogenesis of spinal cord decompression sickness? J Neuropathol Exp Neurol 47:475–487, 1988.)

function occurred. When paraffin wax was used as the embedding material, occasional artifactual NSSOLs were found to be caused by the section tearing as it was cut. The size of the decompression-induced NSSOLs ranged from 20 to 200 μm in diameter. That these lesions were likely to have contained gas in vivo was inferred from the observation that the surrounding tissue appeared to be compressed, as would occur with an expanding bubble of gas. Burns and colleagues reported similar findings from another canine model of DCS, which employed a less stressful dive profile.[219] However, these authors demonstrated most elegantly that these lesions were gas-filled by immersion-fixing the tissue in formalin at different pressures. They showed that the size distribution of NSSOLs varied in accordance with Boyle's law.

The question arises as to how these lesions might provoke tissue dysfunction. Using computerized morphometry, Francis and associates calculated that although the proportion of spinal cord white matter occupied by bubbles was small (always < 0.5%),[220] autochthonous bubbles would account for the loss of cord function if 30% to 100% of the bubble-displaced fibers were rendered nonconducting. The possible means whereby conduction might be nullified are:

- *Destruction of axons at the site of bubble formation.* It was estimated that this effect would account for only 1% of the functional deficit.
- *Stretching and compression of axons around the growing bubble.* This neurapraxia is an attractive mechanism because the onset is rapid (unlike ischemia in the cord) and reversible.[221–224] Such neurapraxia could thereby help account for the most fulminant presentations of the condition, the improvement commonly seen if recompression is undertaken early, and the common and more gradual spontaneous recovery.
- *A biochemical insult akin to the complex interaction between blood and bubbles.* If this effect were limited to those axons adjacent to the bubble surface, it would account for at most 50% of the loss of function. Thus, if there is such an effect, it is likely to be contributory to, rather than the sole cause of, the loss of function.

Broome described another mechanism by which the cord may be injured by autochthonous bubbles.[225] While studying a porcine model of DCS, he correlated functional outcome with the extent of hemorrhage into the tissue.[226] It is likely that expanding bubbles in spinal white matter disrupt not only axons but also the delicate microcirculation. Lacking connective tissue support, these vessels might be uniquely vulnerable to such an insult. The resulting hemorrhage might be expected to be punctate in distribution. Broome proposed that hemorrhage precipitated by bubbles explained why spinal DCS is frequently refractory to recompression treatment.[227] Moreover, he showed that at least some of this hemorrhagic damage appeared to be precipitated by compression of the bubble during early recompression treatment.[225]

There are limitations to the circumstances in which autochthonous bubbles may form. First, a degree of supersaturation is necessary to provide the number of molecules required for bubbles to form and grow. In a study of the spinal cords of 18 animals that were saturated for 4 hours at a fixed pressure and subjected to induced cardiac arrest prior to decompression, few bubbles formed at a saturation pressure of less than 3.6 ata (equivalent to diving to a depth of 26 msw).[173,228] This would indicate that bounce dives to depths much less than this are unlikely to provoke autochthonous bubble formation. Second, the intact cord will off-gas increasingly with time following a dive. Unless bubbles form early, the probability of their formation decreases with time. In the series of canine experiments undertaken by Francis and colleagues, the onset of spinal cord dysfunction occurred more than 30 minutes after surfacing in only two animals; in these specimens, examination of the cord showed no evidence of autochthonous bubbles. The appearance of these two cords closely resembled that of bubble embolism.[173]

Since the description of autochthonous bubbles in the spinal cords of dogs with DCS, other investigators have found such bubbles,[229,230] although in the second of these studies the number found was thought too small to account for the observed loss of function. Despite convincing evidence of extravascular bubbles, their exact location in the spinal cord continues to be debated. In the most recent histologic study, Palmer challenged the view that autochthonous bubbles arise within the tissues themselves, proposing instead that the appearance of spinal NSSOLs arising after provocative decompres-

sion in goats is most compatible with blood vessels overdistended by bubbles.[231]

It is unlikely that a single mechanism can account for spinal cord DCS across its range of latency. It seems likely that cases with a very rapid onset that follow a dive to more than 25 m of seawater are most likely to be associated with autochthonous bubble formation in spinal white matter. In cases that occur with longer latency or from shallower dives, an embolic or possibly a venous infarction mechanism is more likely to be responsible for the loss of function.

Decompression Sickness in the Brain

Autochthonous bubble formation has been observed in the brain following decompression in vivo.[206,232,233] However, in typical human pressure exposures, the relatively luxurious cerebral perfusion is widely considered to limit inert gas supersaturation, thereby preventing clinically significant autochthonous bubbling.[234] This contention is supported by the finding of autochthonous bubbles in both the brains and the spinal cords of dogs in which the circulation was stopped prior to decompression,[232] but mainly in the spinal white matter of dogs or pigs in which the circulation was stopped after decompression.[206,235] Using a cranial window, Pearson and coworkers observed that the initial event causing cerebral dysfunction after provocative decompression in dogs was the arrival of arterial bubbles.[26] The authors noted that these bubbles grew at an accelerated rate after impacting in the cerebral circulation, presumably because dissolved inert gas diffused from tissues into the bubbles. This same group described post-decompression histopathologic changes that were most compatible with an arterial embolic injury.[236] Similarly, a more recent study by Nohara and Yusa, using rats subjected to extreme decompression, showed cerebral changes that, on balance, appeared to favor a vascular mechanism of injury.[237] However, the source of decompression-induced arterial bubbles in these studies is not immediately clear. Other recent studies in decompressed rats showed increased cerebral leukocyte sequestration, but this might be provoked by either vascular or autochthonous bubbles.[238,239]

Thus, although there are reports of cases in which cerebral symptoms are not clearly referable to embolic events,[240] the prevalent theories of dysbaric brain injury focus on embolism by intravascular bubbles.[241]

Musculoskeletal Decompression Sickness

Because some divers have consistent pain localized to a specific joint and others complain of a migratory polyarthralgia and polymyalgia,[242] joint pain in DCS probably cannot be explained by one mechanism. Most hypotheses focus on an autochthonous bubble mechanism, but the actual site is controversial. One or more of the following hypotheses may be valid in certain circumstances.

GAS IN THE JOINT

Although the most obvious hypothesis, the presence of a gas phase within the joint space is an unlikely mechanism for DCS because even large bubbles inside joints do not produce symptoms. Vann and Thalmann[30] summarized the historical data demonstrating that free gas in the knee joints after hypobaric decompression was not reliably associated with pain, whereas gas in the peri-articular tissues (discussed later) was frequently painful. Articular cartilage is also unlikely to be involved in anything other than decompression from saturation because it is avascular and hence will take up inert gas only extremely slowly. Furthermore, articular cartilage is also an aqueous tissue and, consequently, would be expected to absorb only a modest gas burden compared with more fatty tissues around the joint.

GAS IN THE MARROW CAVITY

Bubbles forming de novo in bone marrow are associated with a rise in marrow cavity pressure that correlates with limb pain in sheep and may be a precursor to dysbaric osteonecrosis.[243] It is unclear where, or how, bubbles form in the marrow cavity. One obvious possibility is that, as in the spinal cord, gas is absorbed into the fatty marrow

and this generates autochthonous bubbles on decompression. The resulting pain may result from irritation of local nerve endings or may be an ischemic-type pain resulting from a marrow compartment syndrome caused by the expansion of bubbles inside a rigid cylinder.[243] Increased intramedullary pressure is known to cause pain in other illnesses.[244] Alternatively, bubbles may cause distension of bone marrow sinusoids; because these are innervated with pain fibers, such distention may be the origin of limb pain.[245] In support of this hypothesis, such pain is dull and poorly located, as is common in DCS. An alternative possibility is that embolic bubbles are delivered to the marrow and subsequently grow in situ.

GAS IN PERIARTICULAR SOFT TISSUE

Bubbles that form in poorly compliant tissues, such as tendons, could compress or distort sensory cells.[246] Tendons may be a favorable site for bubble formation because, although they are largely an aqueous tissue and consequently might be expected to have a relatively low gas content, the blood flow through tendon vessel beds is discontinuous.[247] Indeed, bubbles have been seen growing and others simultaneously disappearing in different regions of the same tendon.[247] However, it is strange that only tendons should be involved; if this mechanism operated with any frequency and affected ligaments equally, pain should have been reported in other locations.[245] An alternative pain-sensitive periarticular site in which autochthonous bubbles may form is the joint capsule itself.[248,249] Isolated reports of rhabdomyolysis in divers with DCS[250] raise the possibility that bubble formation in muscle itself might provoke pain via associated sensory fibers.

REFERRED PAIN

Another possibility is that the pain is referred from bubble-induced injury in the central or peripheral nervous systems.[251] This would explain the frequent concurrence of joint pain and neurologic deficits in the same limb. Back and girdle pain are considered to be referable to involvement of the spinal cord. On the other hand, Palmer and associates question the role of neurogenic pain on the basis that no brain or spinal lesions were found in goats affected only by limb pain.[252]

Finally, a generalized release of pain-mediating chemicals such as the kinins can occur in DCS as part of a nonspecific inflammatory response to bubbles.[67] This proposal is well suited to those divers with general malaise, polyarthralgia, and polymyalgia.[242]

Constitutional Decompression Sickness

The precise cause of the constellation of symptoms making up "constitutional DCS" is unknown, but this condition is unlikely to be explained by bubble formation in any discrete location. Indeed, the most plausible explanation for systemic constitutional symptoms arises from the known pro-inflammatory effects of bubbles in whatever location they form. As discussed earlier, activation of the complement or kinin systems secondary to tissue injury, or by direct bubble stimulation of inflammatory cells such as neutrophils with consequent elaboration of cytokines, could result in constitutional symptoms.

Cutaneous Decompression Sickness

Buttolph and colleagues recently published an analysis of the histologic appearance of the cutis marmorata form of cutaneous DCS using a swine model.[253] They described congestion, vasculitis, edema, perivascular neutrophil infiltrates, and occasional frank hemorrhage at the site of the skin discoloration. These findings were progressively less frequent at the margin of the lesion, in grossly unaffected skin from the same biopsy, and in skin biopsied from sites distant to any lesion. Only congestion occurred with any frequency in all samples, and this probably represents an artifact resulting from the biopsy procedure. Ultrastructural changes seen on electron microscopy included neutrophil rolling, adhesion, and transmigration; extravasation of erythrocytes; and reactive changes in endothelial cells. Monocytes and platelets were also involved occasionally. Of interest, the

ultrastructural changes were detected predominantly in capillaries and venules.

Although the clinical and histologic appearance of cutis marmorata is well described, there is a dearth of literature on the mechanisms involved. Ferris and Engel[254] hypothesized that cutis marmorata is caused by the release of extravascular gas bubbles and that these cause vascular spasm that results in the stasis that can be observed clinically. However, these authors provided no histologic or other evidence to support their theory. Another possible cause of cutaneous vascular injury is arterial gas embolism of the microcirculation. This hypothesis has been recently supported by the strong association between large right-to-left shunts and cutaneous DCS reported by Wilmshurst and coworkers.[135] The description provided by Buttolph and associates is inconclusive with respect to mechanism.[253] The ultrastructural vascular changes they reported in the capillaries and venules were consistent with bubble injury by arterial gas embolism or the drainage of bubbles formed locally. It is likely that more than one mechanism is involved, and the possible role of vasoactive mediators and complement cannot be ignored.

A number of early investigators noticed that pruritus with rash is more common when there is little sweating[255,256] and that chilling the skin during decompression increases the frequency of this condition.[257] One interpretation of these observations is that the transcutaneous migration of gas may contribute to the pathogenesis of the pruritus and that the vasoconstriction caused by cooling may reduce the rate at which gas is cleared from the skin. This condition is associated particularly with dry chamber dives and is not usually a harbinger of more serious disease.

Audiovestibular Decompression Sickness

The vestibulocochlear end organ can be damaged by either barotrauma or inert gas bubble formation in DCS. Both processes are detailed in Chapter 22. Vestibular and cochlear DCS have been demonstrated in vivo,[258] and the involvement of either or both systems in divers[259] or aviators[260] with DCS has been reported. The precise mechanism by which inert gas bubbles injure the inner ear is uncertain. The mechanism may involve formation of bubbles in the perilymph, endolymph, or associated blood vessels.[261,262] In addition, Fraser and colleagues reported the intriguing in vivo observation of fractures in the semicircular canal walls following severe decompression stress in animals, suggesting that bubbles forming in restricted spaces may generate immense distractive forces.[263] The clinical significance of this phenomenon has never been established.

It is notable that "pure" inner ear DCS is uncommon following air diving within the recreational diving range but became well recognized with the rise of deep diving using oxygen-helium mixtures.[262] This has been attributed to expansion of silent vestibulocochlear helium bubbles by inward diffusion of nitrogen following gas switching from oxygen-helium mixes to air during decompression.[259]

Lymphatic Decompression Sickness

Rarely, DCS may manifest as localized soft tissue swelling.[264] Other than the assumption that this swelling arises from obstruction of lymphatic vessels by bubbles, little is known about the pathophysiology of this form of DCS.[264–267]

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CHAPTER 9 Pulmonary Barotrauma

Tom S. Neuman

Pulmonary barotrauma (PBT) results from overexpansion of the lungs when the victim, breathing compressed gas, cannot properly ventilate the expanding pulmonary gas volume during a reduction of ambient pressure. A rare form of PBT occurs on extremely deep breath-hold dives wherein lung compression results in vascular engorgement and ultimate bleeding into the alveolar spaces (see Chapter 5). This chapter is devoted to PBT caused by lung overexpansion.

Air embolism secondary to PBT ranks second only to drowning as a cause of death in the recreational diving community.[1–10] However, because pathologists inexperienced in diving medicine frequently classify almost any death that occurs in water as a drowning, PBT with arterial gas embolism (AGE) may actually be the leading cause of death,[11] although other reports suggest that cardiac disease is the leading cause of fatalities associated with scuba diving in men over the age of 45.[12] Also, because water is considered a "hostile environment," cases in which gas embolism leads to unconsciousness in the water with subsequent drowning are then classified as drowning. Regardless, PBT with secondary AGE is certainly one of the more frequent causes of death in the scuba-diving community.

PBT and secondary AGE occur more frequently in novice or inexperienced divers.[13] A combination of factors probably accounts for this; less familiarity with the equipment and monitoring of the air supply, relative unease in the water, and—perhaps because of this unease—a greater likelihood of panicking in the event of an out-of-air situation. In addition to out-of-air and panic ascents, other specific activities that especially carry a risk of PBT and AGE include submarine-escape training, out-of-air emergency ascent training, and buddy-breathing ascent training.[14,15] These activities now have either been discontinued or been significantly modified to reduce the risk of PBT and AGE.

CAUSES OF PULMONARY OVERINFLATION

Pulmonary overinflation is caused by excessive intrapulmonary pressure and over expansion of the lungs, which, in diving activities, results from the failure of expanding gases to exit the lungs. During ascent, expanding gases may not exit the lungs because of breath holding or local pulmonary obstructions (such as bronchospasm, pulmonary secretions, or broncholiths).[16] Other characteristics of the lungs may also predispose to PBT, and it has been suggested that restriction may play a greater role in idiopathic PBT than had been thought (see later). It is especially interesting to note that the classic paper that implicates intrinsic obstruction in the lung from a broncholith as the cause of AGE documents bilateral overinflation of the lungs at autopsy when the presumed cause of the AGE (the broncholith) was unilateral.[16]

A review of sport diving accident statistics suggests that breath holding during ascent, associated most frequently with out-of-air situations or panic induced by unfamiliarity with equipment (e.g., regulator loss or mistakes with the use of buoyancy compensators), is the most common cause of PBT and AGE.[17] However, one report suggests that as many as 50% of cases of AGE secondary to PBT may be idiopathic.[13] In submarine-escape trainees, breath holding did not appear to be a major factor, leading to the conclusion that some intrinsic abnormality of the lungs was the cause of the injury in most cases. Even so, these simplistic explanations do not fully explain the observation that PBT leading to AGE rarely occurs in dry chamber dives. Thus, the pertinent question is whether immersion plays some significant role in the pathogenesis of PBT or divers simply hold their breath more in the water and fail to report it.

MECHANICS OF LUNG RUPTURE

Under experimental conditions, it has been demonstrated that a transpulmonic pressure (the difference between the intratracheal and the intrapleural pressure) of 95 to 110 cm H_2O is sufficient to disrupt the pulmonary parenchyma and allow gas into the interstitium.[18,19] Gas can then migrate through the perivascular sheaths of the pulmonary vasculature to cause mediastinal emphysema and pneumothorax.[20] However, in the case of pneumothorax, it is not clear whether rupture of the visceral pleura or of the parietal pleura is responsible. Gas can also dissect into the retroperitoneum and subcutaneous tissues of the neck. In any event, when intrathoracic pressure drops (e.g., at the time of the first breath after the PBT), extraalveolar gas can pass into torn blood vessels and then travel to the left side of the heart, from where it enters the arterial circulation as gas emboli.[21] There are, however, well-documented cases of fatal AGEs in which the injury is sustained entirely under water without any apparent opportunity for the victims to take a normal breath.

Decreased pulmonary compliance may also be a factor in the development of PBT. Colebatch and colleagues[22] reported that divers with reduced pulmonary compliance are at increased risk for PBT, and there is a case report of an air embolism in a chamber in a patient with asymptomatic sarcoidosis.[12] A person with areas of decreased pulmonary compliance may be more susceptible to PBT because less compliant zones of the lungs may be exposed to excessive pressure. Another factor that may predispose to PBT is the overly forceful attempt to exhale during very rapid ascents (e.g., during submarine-escape training). In humans, simple immersion has been demonstrated to result in airway closure and air trapping at low lung volumes; it has therefore been hypothesized that vigorous attempts to exhale at such low lung volumes could predispose to PBT and AGE.[23] Mild obstruction, however, does not seem to be a risk factor for AGE. In a study using a standard battery of pulmonary function tests on submarine-escape trainees, only a small forced vital capacity was a slight risk factor for PBT.[24] However, case reports suggest that small cysts or perhaps other poorly ventilated areas may predispose to AGE.[25,26] Interestingly, binding the chest wall, which physically limits thoracic overexpansion, appears to be protective.[18,19,27]

PATHOPHYSIOLOGY OF ARTERIAL GAS EMBOLISM

Understanding of the pathophysiology of AGE is based on a few clinical series of human AGE victims during the last 60 years and on a larger number of experimental studies in animals. In addition, studies of thromboembolic stroke, which shares many of the clinical features of gas embolism, have aided the understanding of AGE. Even so, *there are significant differences between the clinical appearance of human victims of AGE and the appearance of animals with experimental AGE.* As a result, caution must be exercised in extrapolating from animal models to human experience.

Animal Studies

Gas entering ruptured pulmonary vessels reaches the left chambers of the heart via the pulmonary veins and enters the arterial system either as foam or as large gas bubbles that distribute according to the relative blood flow.[28] In animal models in which air is injected directly into the cerebral circulation, the air distributes until it blocks blood vessels with a diameter of 30 to 60 μm. Even if air is injected into one internal carotid artery, volumes of air distribute widely to the brain.[29] Cerebrospinal fluid pressure rises secondary to an increase in cerebral blood volume because of reactive hyperemia.[30–32] Systemic arterial hypertension is also associated with this model of experimental air embolism. This hypertension, which can be dramatic, appears to be produced by increases in circulating catecholamines and possibly also by the release of vasopressin.[33] An alternative contributor to systemic hypertension may be the increase in cardiac output associated with this model.[34] In the areas of hyperemia, autoregulation is lost and cerebral blood flow responds passively to changes in systemic blood flow and blood pressure, resulting in further increases in cerebral blood volume.[31,32] During the next 30 to 60 min after the injection of air, cerebrospinal fluid pressure falls.[32] Perfusion

studies indicate that the hyperemic areas are adjacent to areas of low blood flow.[35] Although the basis of these findings is unknown, they are presumed to be due to a failure of reperfusion secondary to interactions between vascular elements and the endothelium.

An electroencephalogram (EEG) or evoked-potential analysis reveals immediate cessation of normal function after embolism,[32] observations typical of ischemic brain injury. Cessation of blood supply to neuronal tissue causes decreases in neuronal adenosine triphosphate and increases in lactic acid production.[36,37] Increased synaptic activity associated with increased serum lactate levels can further increase cellular damage. Investigations of stroke have demonstrated a relationship between glucose levels and the size of infarctions.[38] This infarction is probably mediated through lactate, which is the major metabolic end product of anaerobic glucose metabolism. Glucose effects on central nervous system injury are also discussed in Chapter 10.

Experiments with small animals in a different model of cerebral air embolism suggest that the blood-brain barrier opens immediately after embolization.[39] Permeability to large molecules becomes maximal 30 to 60 min after embolism but falls off rapidly. Permeability to small molecules is also increased after embolism, but it remains increased for up to 24 hours. In this situation, edema appears to be vasogenic; because the period of edema appears to be relatively brief, there is no firm theoretical basis for the use of steroids. Similarly, although cytotoxic edema may also occur (secondary to ischemia), steroids do not appear to have a major effect in this form of edema,[40] and the recommendation for their use is not firmly based on an experimental model. It is noteworthy, however, that bubbles distributed to the cerebral circulation cause sustained occlusion of the cerebral vasculature only if the volume of bubbles is large enough to block several generations of branching vessels—presumably because the surface tension of the air then exceeds the cerebral perfusion pressure.[41] Otherwise, gas bubbles will generally pass through the cerebral circulation after causing transient obstruction.[42,43]

The direct cardiac effects of AGE have also been studied in experimental models.[44] In these studies, air was infused into the left ventricle and cardiac arrest was hypothesized to occur by one of two mechanisms. The first is direct gaseous embolism of the coronary arteries. Experimental animals in this model first become hypotensive, then experience depression of left-ventricular function, then show evidence of myocardial ischemia on an electrocardiogram (ECG), and finally die. The second hypothetical mechanism is based on the observation that embolization of the cerebral circulation results in severe hypertension and marked arrhythmias. However, the process and the time course by which cardiac arrest occurs in these animal studies is markedly different from the time course of cardiac arrest in human victims,[45] suggesting that these mechanisms may not play a major role in the pathophysiology of cardiac arrest in human victims.

In studies of laboratory animals with experimental cerebral embolism, although multiple premature ventricular contractions and periods of nonsustained ventricular tachycardia may occur, cardiac arrest does not occur suddenly during the initial embolization. Cardiac arrest, when it occurs, develops late and only after spontaneous ventilation ceases; it does not generally occur in animals in which ventilation is supported. In animals with coronary artery gas embolism, cardiac arrest occurs only after the animals develop hypotension and depressed left-ventricular function. Once again, sudden cardiac arrest (which occurs in 50% of the AGE victims who die) does not occur in these animal models.

Human Studies

Evidence regarding the effect of air embolism on coronary arteries in humans has been obtained from case reports of iatrogenic injuries occurring during coronary angiography.[46–48] By and large, sudden death is not a feature of such embolism; rather, transient hypotension, chest pain, and ECG abnormalities occur. Lethal arrhythmias and frank infarction have not been reported.

On review of well-documented cases of sudden death from AGE, it appears that some of these cases are associated with a massive gas filling of the central vascular bed.[49,50] In the past, on the basis of an animal model in which a compliant, gas-filled balloon was placed in the left ventricle, air filling of the left ventricle was not considered to be a

cause of sudden death.[51] In this model, neither cardiac arrest nor sudden cessation of cardiac output could be produced. However, in the human cases of sudden death associated with AGE (described earlier), air appears to fill not only the left ventricle but also the aorta, carotid arteries, and subclavian veins; at autopsy, the pulmonary arteries and the right ventricle are found filled with air as well. Thus, it seems more likely that complete filling of the central vascular bed by gas secondary to PBT, not hypothetically induced arrhythmia, is the mechanism of sudden death in AGE.

In addition to its effects on the cerebral circulation and the cardiovascular system, gas embolism also produces a variety of biochemical and hematologic effects, which in all probability are due to direct embolism of other organ systems of the body. The creatine kinase level is routinely elevated in victims of AGE.[52] The predominant isoenzyme is the MM fraction, although the MB fraction is also frequently elevated; the BB fraction is rarely detected. The degree of creatine kinase elevation correlates well with the eventual neurologic outcome of the patient. In addition to this evidence of direct muscle embolization, elevations of lactate dehydrogenase, serum glutamic-oxaloacetic transaminase, and serum glutamic-pyruvic transaminase routinely occur.[53] The increases in these enzymes correlate with the degree of elevation of creatine kinase. Presumably, these abnormalities in lactate dehydrogenase, serum glutamic-oxaloacetic transaminase, and serum glutamic-pyruvic transaminase reflect widespread systemic embolism with secondary damage to the endothelium of embolized organ systems. Intravascular gas damages the vascular endothelium; in fact, one experimental technique to isolate endothelium is to use intravas-cular gas injection to separate the endothelium from blood vessels.[54] This mechanism is used to explain the hemoconcentration in decompression sickness (DCS) (see Chapter 8). Similarly, hemoconcentration also occurs routinely in victims of AGE, presumably by the same mechanisms as in DCS.[55] With the inability of the lung to act as a complete bubble filter in cases of DCS,[56–58] it would not be surprising to see the same, or similar, biochemical abnormalities in cases of DCS associated with arterialization of gas. In addition to overwhelming the filtration capacity of the lung, arterialization of gas through a patent foramen ovale[59] might also produce similar biochemical changes.

CLINICAL MANIFESTATIONS OF ARTERIAL GAS EMBOLISM

Victims of AGE manifest signs or symptoms within minutes of ascent, as would be expected from the pathophysiology of this condition (Table 9–1). Patients with AGE can be divided into two groups based on their initial presentation.[60,61] In the first group, patients experience apnea, unconsciousness, and cardiac arrest immediately after embolism. This catastrophic condition occurs in approximately 5% of patients and, as previously discussed, probably results from complete filling of the central vascular bed with air, or it might occur secondary to coronary artery embolism or cerebral embolism. The observation that many of these patients are unresponsive to cardiopulmonary resuscitation and advanced life support measures tends to support the former mechanism as the cause of sudden cardiac arrest.

In the second group of patients, varying neurologic and systemic signs and symptoms are present but the vital signs are preserved. The most frequently observed signs are loss of consciousness or stupor and confusion, but other less striking findings (see Table 9–1) are also described.[62] If prompt recompression is initiated, most patients in

Table 9–1. Clinical manifestations of arterial gas embolism

Group 1
Apnea
Unconsciousness
Cardiac Arrest
Group 2
Loss of Consciousness
Stupor
Confusion
Seizure
Collapse
Hemiparesis
Cortical Blindness
Vertigo
Headache
Sensory Changes

From[60–62]. See text for explanation of groups.

this group recover fully; however, one fourth to one third of the victims in this group deteriorate secondarily during hyperbaric treatment.[63] This deterioration generally develops approximately 20 min after the initial embolism. The signs and symptoms of this secondary process are similar to the initial presentation but are more gradual in onset and may include headache, progressive stupor, visual disturbances, and convulsions. A variety of different processes may be responsible for this syndrome. Hypothesized mechanisms include edema, increased intracranial pressure, and the effect of vasoactive substances released from lungs damaged by antecedent PBT.[64] All of these processes are thought to contribute to the syndrome by reducing cerebral blood flow. Unlike thrombotic or embolic stroke, many victims of AGE (as noted earlier) lose consciousness as the initial manifestation of the process. Whether this is due to bilateral cerebral embolization and loss of function of both cortices, embolization of the reticular activating system, or profound hypotension and associated generalized cerebral ischemia is not clear; however, infarction of the pattern occurring in classic stroke is distinctly unusual in victims of AGE.

This organizational schema, however, is predominantly derived from observations of submarine-escape trainees. These victims differ in several important aspects from divers. Perhaps the most important difference is that submarine-escape trainees are usually treated within seconds to minutes of the embolism. Thus, in submarine-escape trainees in whom cardiac arrest does not occur, there is often a dramatic and complete resolution of symptoms.[60] In the diver, who often does not have immediate access to a recompression chamber, a complete and immediate response to treatment is less common, although most victims experience complete neurologic recovery. In some victims who are some distance away from a recompression chamber, spontaneous improvement occurs over a period of minutes to hours in varying degrees before definitive treatment is initiated. It is tempting to hypothesize that this improvement is the clinical manifestation of gas clearing spontaneously from the cerebral circulation or from clearing of gas from the central circulation with improved cerebral perfusion pressures. One can also speculate that because rapid treatment, particularly in the sport diver, is infrequent, the clinical manifestations of the secondary deterioration that occurs in submarine-escape trainees are obscured in sport divers by the still-present initial manifestations of the AGE. Alternatively, changes in the sport diver's clinical status that could represent the secondary deterioration might go unrecognized in the extended prehospital setting, in which the observers usually have little medical training. Whatever the cause, secondary deterioration is not reported frequently in sport scuba divers.

Perhaps second in importance is the fact that the sport scuba diver frequently experiences a gas embolism at the end of a dive that has produced some degree of gas loading, leading to a combination of AGE and DCS. This combination does not occur in the milieu of submarine-escape training. The effects on each other of the intravascular gas phase of AGE and the unloading of gas in solution in the tissues remain speculative; however, it does appear that AGE can precipitate DCS in divers who are well within the United States Navy no-stop limits and who otherwise would not be expected to experience DCS.[65,66] Frequently, the DCS occurring in this setting is extremely resistant to the usual forms of therapy.

Finally, whereas victims of AGE in the setting of submarine-escape training are most often removed from the water immediately, victims of AGE in the sport diving community frequently suffer near-drowning episodes when they lose consciousness in the water.[52] As previously discussed, near drowning or even drowning subsequent to loss of consciousness or loss of motor ability may be the ultimate cause of morbidity or mortality in some victims of AGE (see Chapter 14). Once again, the role of hypoxemia in the interactions between intravascular gas from AGE, tissue gas unloading, and the resultant rheologic disturbances awaits further research.

As previously mentioned, the majority of diving accident victims who have sustained an AGE recover fully with appropriate treatment. However, a minority of patients are left with residual neurologic problems.[52] On rare occasions, clear evidence of cerebral infarction is apparent on either computed tomography or magnetic resonance imaging[67]; however, many patients with neurologic injury do not have clearly defined lesions visible by computed tomography or magnetic

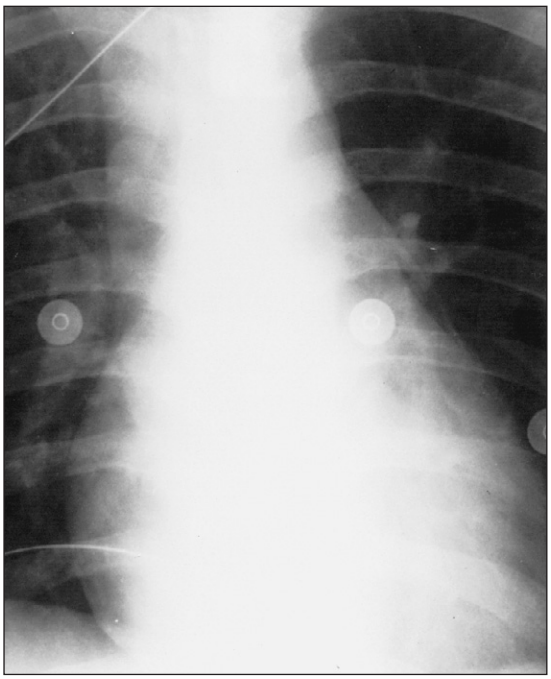


Figure 9–1. Pneumomediastinum. In this radiograph, radiolucencies representing air can be seen only along the border of the left side of the heart and along the descending aorta.

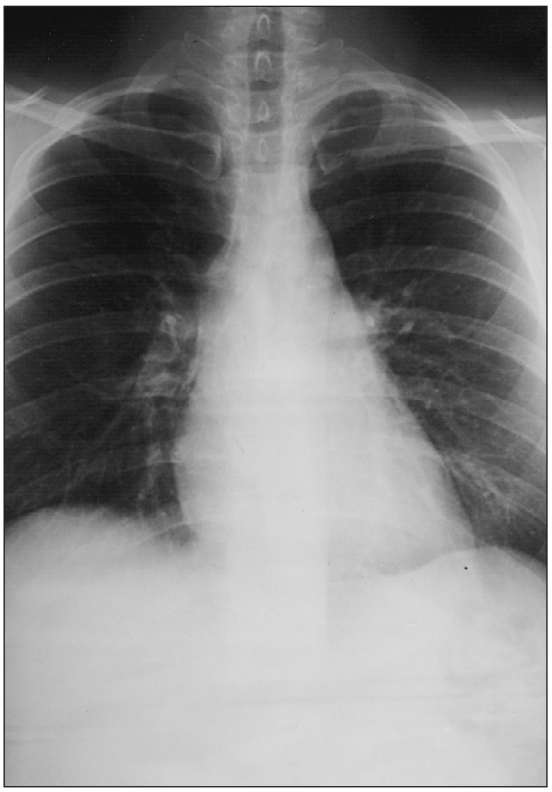


Figure 9–2. Pneumomediastinum. An air lucency is visible on this radiograph as a small semilunar crescent immediately above the left pulmonary artery.

resonance imaging. Other imaging techniques have not proven useful because of a lack of sensitivity and specificity.[68] The lack of correlation between neurologic injury and visualization of lesions by imaging modalities suggests that a portion of the injury may be related to a more generalized insult that could be the result of concomitant near drowning or some other diffuse process. Gas bubble–induced lesions may be smaller than the resolution of current imaging techniques. With better magnetic resonance images and techniques, we soon may become able to routinely visualize the area of injury to the brain in victims of AGE.[69]

Patients with AGE have, by definition, suffered acute antecedent PBT; however, radiographic evidence of PBT is generally present in fewer than half of AGE victims.[62] PBT is most frequently demonstrated as mediastinal emphysema. Mediastinal gas can be difficult to detect and should be carefully looked for along the borders of the pulmonary arteries, the aorta (including the descending aorta), and the heart (Figs. 9–1 and 9–2). Although pneumopericardium has been described as a form of PBT and has occasionally been reported in autopsies, most radiographs that purportedly demonstrate pneumopericardium in reality show pneumomediastinum with the anterior portion of the pleural reflection being displaced off the border of the left side of the heart (Fig. 9–3). Less frequently, subcutaneous emphysema, pneumothorax, and pneumocardium (Fig. 9–4) are radiographically apparent in patients with AGE.

OTHER ASPECTS AND MANIFESTATIONS OF PULMONARY BAROTRAUMA

Isolated PBT without AGE can also occur in the setting of rapid or breath-hold ascents. In one study of 170 consecutive submarine-escape trainees examined radiographically after training, 2 had subclinical evidence of extraalveolar air.[70] Gas expanding within alveoli causes rupture of the pulmonary vasculature, and that is the presumed point of entry of air into the vascular system. As a result, local hemorrhage occurs, which may

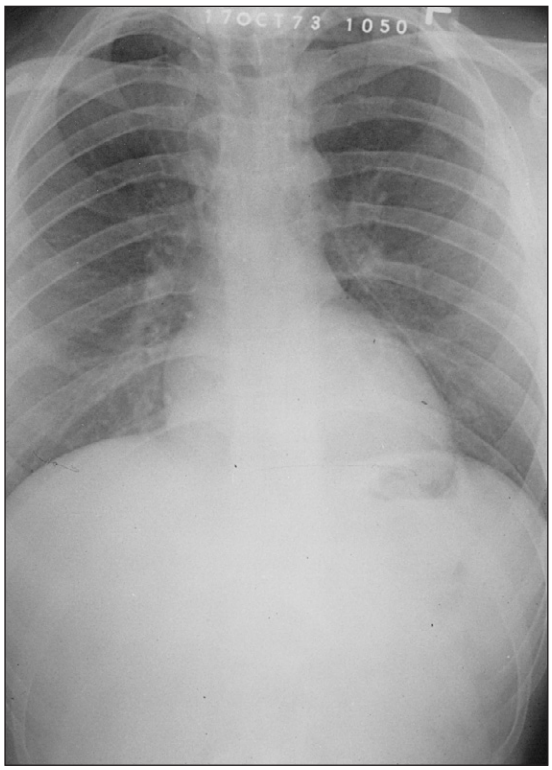


Figure 9–3. Pneumomediastinum. In this radiograph, the anterior reflection of the pleura is pushed away from the border of the left side of the heart. This condition is often misinterpreted as pneumopericardium.

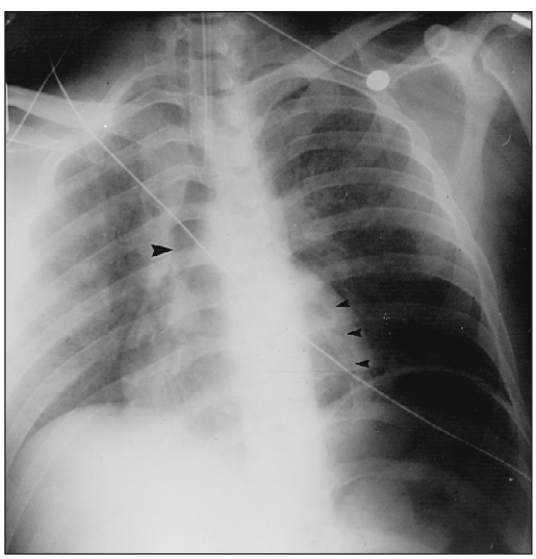


Figure 9–4. Chest radiograph of a patient who died of gas embolism. Gas lucencies are visible within the heart *(small arrows)*, the aorta *(large arrow)*, and the subclavian vessels. In the original radiograph (not shown here), air lucency was also visible in the body of the liver. Gas in these organs was confirmed at autopsy.

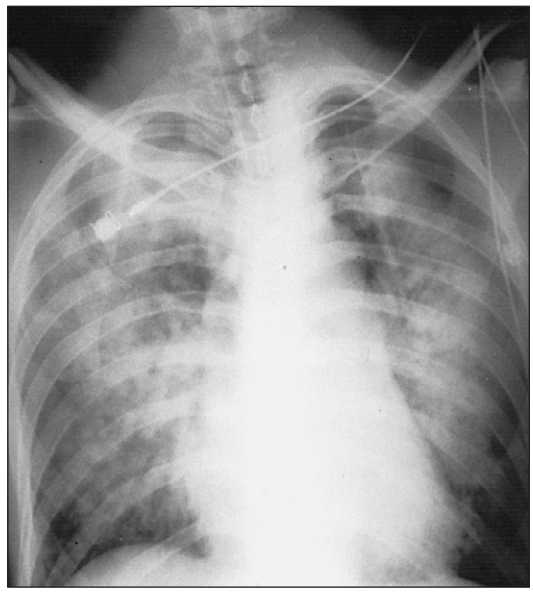
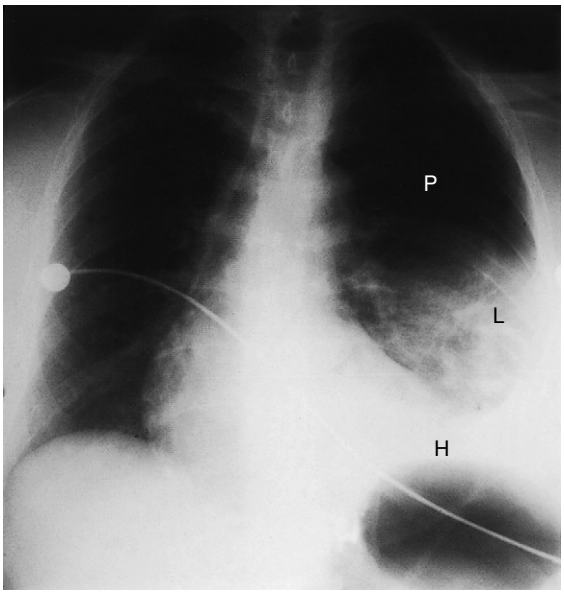


Figure 9–5. Chest radiograph of a victim of a gas embolism who had immediate extensive hemoptysis in conjunction with initial neurologic deficits. The patient continued to have significant hemoptysis (a further 800 mL of blood expectorated) after being taken to a local clinic. He subsequently suffered cardiorespiratory arrest secondary to hypoxemia from continued hemoptysis.

then result in hemoptysis and which, on rare occasion, can be massive and even life-threatening (Fig. 9–5). When gas dissects back along the perivascular sheaths, it can (as mentioned previously) enter the mediastinum. Mediastinal emphysema is generally associated only with mild substernal pain, and even that is present in only a minority of victims. This pain has been described as either a dull ache or a tightness. It may be exacerbated by inspiration, coughing, or swallowing and may radiate to any portion of the upper torso. Unless severe, this condition is not usually associated with respiratory symptoms. On physical examination, a crunching sound synchronous with cardiac action may be auscultated (Hamman's sign), and a chest radiograph confirms the diagnosis. Treatment is usually not necessary.

When air continues to dissect superiorly from the mediastinum, subcutaneous emphysema may become clinically apparent. The signs and symptoms of subcutaneous emphysema include swelling and crepitus in the root of the neck and in the supraclavicular fossa, sore throat, hoarseness, and dysphagia. Radiographs may be helpful in



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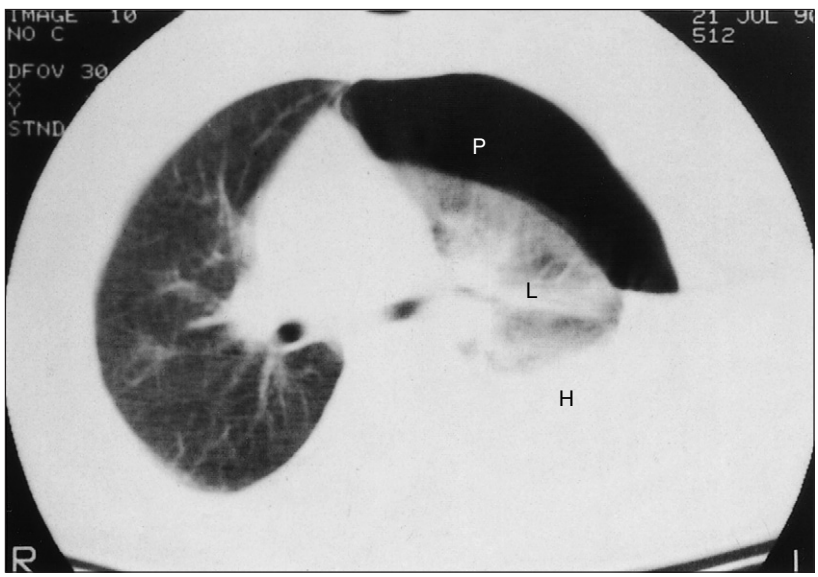

B

Figure 9–6. Chest radiograph *(A)* and computed tomograph *(B)* of a victim of PBT after an out-of-air ascent. The patient experienced a hemopneumothorax. The hemothorax yielded over 1000 mL of blood when it was evacuated and continued to drain blood for several hours after the insertion of a chest tube. P, pneumothorax; L, collapsed lung; H, hemothorax.

detecting subtle cases. Finally, should extraalveolar gas rupture into the pleural space (either from the mediastinum through the parietal pleura or from the perivascular tissues through the visceral pleura), a pneumothorax can occur.

Pneumothorax is uncommon, occurring in fewer than 10% of cases of PBT.[71] Occasionally (as occurs in spontaneous pneumothorax), hemothorax can complicate a pneumothorax (Fig. 9–6). The initial symptom of a pneumothorax is sharp chest pain. Theoretically, recompression and then subsequent decompression can convert a simple pneumothorax to a tension pneumothorax; however, this has not been reported as a frequent problem, and standard therapy is appropriate in such cases.

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CHAPTER 10 Treatment of Decompression Illness

Richard E. Moon

This chapter reviews the therapy of decompression-related illnesses. Collectively, these disorders are called *decompression illness (DCI)* and consist of disorders due to bubbles formed by supersaturation of inert gas in tissues (*decompression sickness,* or *DCS*), and disorders due to lung barotrauma with subsequent arterial gas embolism (AGE). Although the pathophysiologic aspects of the two disorders are different (see Chapters 8 and 9), the signs and symptoms are similar; aside from a few exceptions mentioned in this chapter, therapy is essentially the same for both. When pertinent, the specific disorder is noted, and when discussion is relevant to the two disorders together, the term *DCI* is used.

HISTORY OF TREATMENT OF DECOMPRESSION ILLNESS

During the early experience with compressed-air illness, spontaneous recovery was all one could hope for, perhaps with some optimistically administered medicine. In France, Pol and Wattelle had observed that reentry by compressed-air workers into the high-pressure environment could relieve symptoms and suggested systematic treatment in this manner.[1] Based on his experience with caisson workers during construction of a bridge over the Seine, Foley recommended construction of a portable recompression chamber capable of sustaining a pressure of 2.5 atm.[2] The first major American construction job in which compressed air was used was during construction of the bridge (now named the Eads Bridge) over the Mississippi River at St. Louis. There were 91 reported cases of DCS, of which 30 were classified as serious; 2 persons were crippled for life, and 13 died. No recompression therapy was employed, although some of the severe cases improved spontaneously.[3] In 1872, Dr. Andrew Smith, Surgeon to the New York Bridge Company, observed 110 cases severe enough to require medication in caisson workers. His pharmacologic therapies included atropine, calomel (mercury chloride), ergot, whiskey, and ginger. He too suggested use of a recompression chamber, but it was not implemented.[4,5] The impression of the workers and engineers at the time was that most of the mildly afflicted men recovered, irrespective of Smith's treatments, although it was recognized that neurologic DCS could cause permanent disability or death.

DCS in divers was later described in the medical literature.[2,6–9] The majority of patients were observed to recover spontaneously, with only approximately 10% permanently affected by slight paresis, generally of the anterior muscles of the legs.[9] Complications of spinal-cord DCS were reported as cystitis, decubitus ulcers, and meningitis.

Recompression treatment for DCS was not systematically used until Ernest Moir assumed responsibility for excavating a tunnel under the Hudson River between Manhattan and New Jersey.[10] Injured men underwent recompression using air to two thirds of working pressure followed by slow decompression to atmospheric pressure over 25 to 30 min. This procedure dramatically reduced the mortality rate of DCS, previously at 25% of the workforce per year.

Implementation of recompression therapy for divers took longer. It was not until the 1924 edition of the *U.S. Navy Diving Manual* that a standard recompression therapy was recommended.[11] Over the next few years, in both compressed-air and diving practice, a variety of empirical air recompression

regimens were developed. Treatment pressure was based on either the depth of the dive (or a fraction or multiple thereof) or the depth of relief.[12]

Oxygen administration was not routinely used until much later, although its roots can be traced to Paul Bert's investigations in Paris in the 1870s. He first observed that when 100% oxygen at 1 ata was administered to animals after decompression, some of the signs would resolve.[2] Surprisingly, Bert did not try hyperbaric oxygen, which was first proposed by Zuntz several years later.[13] Initial results were actually somewhat disappointing, probably because the therapy was too brief. In 1909, Keays reported that oxygen had been given to several compressed-air workers during recompression for severe DCS but seemed to afford no appreciable benefit.[14]

In 1939, two U.S. Navy medical officers, Yarbrough and Behnke, first published results of DCS treatment using compressed oxygen,[15] but despite their success, the technique was not initially adopted. Instead, for the next 20 years, the U.S. Navy continued to recommend a variety of air tables despite long treatment duration and a high failure rate.[16–19] In the early 1960s, the U.S. Navy instituted another series of investigations into low-pressure oxygen tables. A high recurrence rate with the 33 ft (2 ata) recompression depth led to the development of treatment tables requiring an initial recompression to 60 ft.[19] The new tables are the ones most commonly used today: U.S. Navy Treatment Tables 5 and 6 (Figs. 10–1 and 10–2). A two-step configuration of the treatment tables, with a period at 30 ft following initial compression to 60 ft, was implemented to allow safe decompression for the tender.

The U.S. Navy developed different tables for the treatment of AGE. Despite evidence in an animal model that a compression depth of 100 ft is adequate,[20] a table that starts at 165 ft was recommended on the basis that diving medical officers would not accept a shallower treatment table.

Developments since the 1960s have included the design of saturation treatment tables[21,22] and treatment tables designed mainly for treating deep uncontrolled ascents ("blowups") when more than 60 min of decompression have been missed (refer to U.S. Navy Treatment Table 8; Fig. 10–3).[22] First espoused in the early part of the twentieth century, the basic principle of breathing oxygen under pressure remains the standard of care.

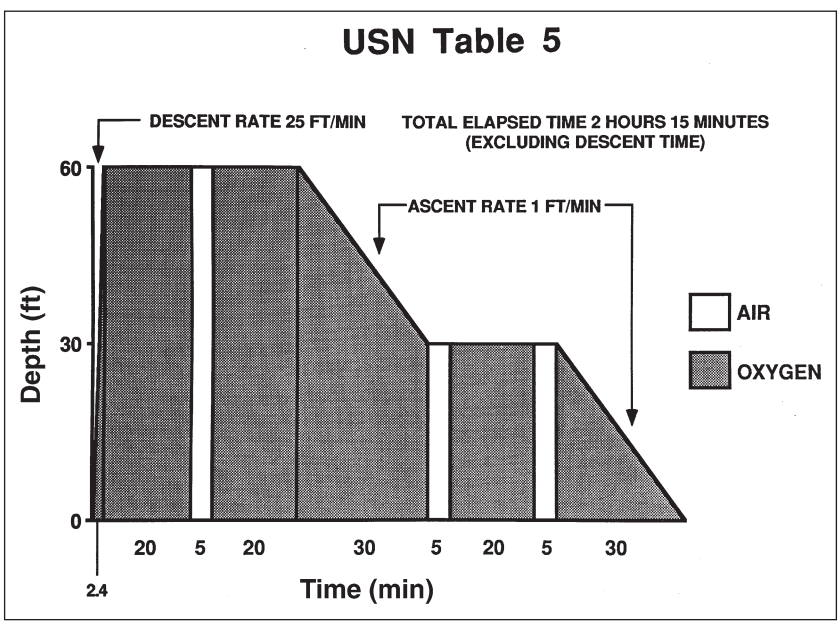

Figure 10–1. U.S. Navy Table 5 (Royal Navy Table 61). The U.S. Navy recommends this table for use in divers with decompression sickness consisting only of pain or cutaneous symptoms. Results of the neurologic examination must be normal, and all symptoms must be relieved within 10 min of reaching maximum pressure (2.8 ata, 18 m, 60 ft). Otherwise, a longer treatment table, such as Table 6 (see Fig. 10–5), should be used. (Redrawn from Moon RE, Gorman DF: Treatment of the decompression disorders. *In* Bennett PB, Elliott DH [eds]: The Physiology and Medicine of Diving. Philadelphia, WB Saunders, 1993, pp 506–541.)

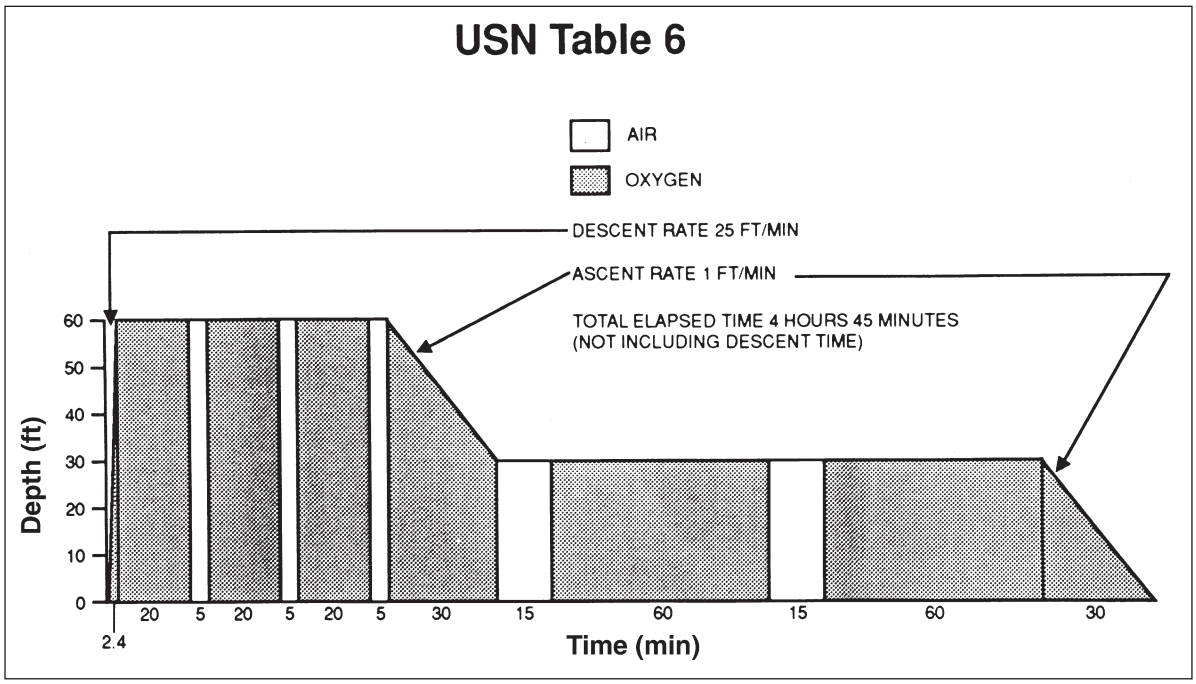

Figure 10–2. U.S. Navy Table 6 (Royal Navy Table 62). This is the most widely used recompression table. Additional oxygen cycles can be administered at both pressures (60 and 30 ft). (Redrawn from Moon RE: Treatment of gas bubble disease. Probl Respir Care 4:232–252, 1991.)

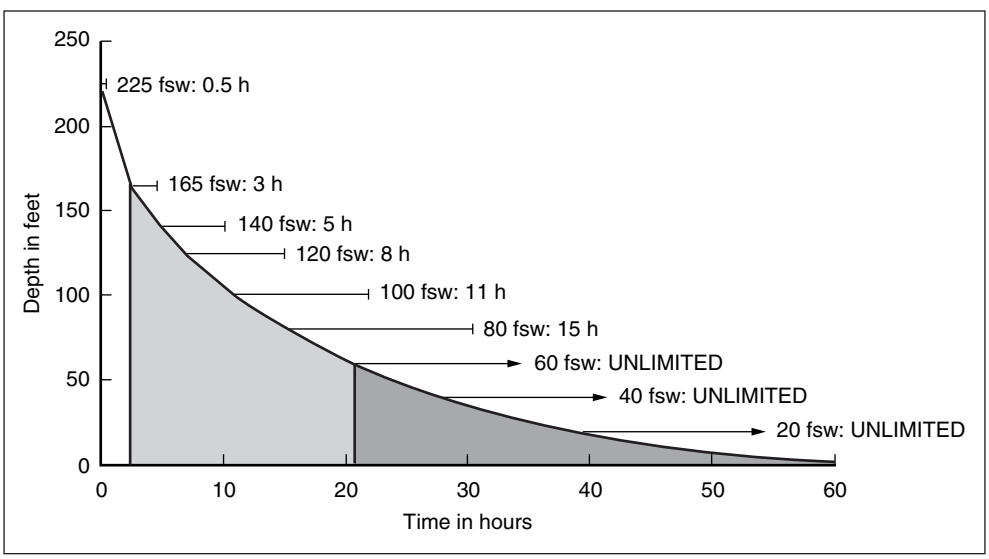

Figure 10–3. U.S. Navy Treatment Table 8. This table is designed for treatment of deep blowups in which missed decompression stop time exceeds 60 min. The table can be used in other situations, such as to compress to a depth greater than 165 ft (50 m) or to stop decompression between 165 ft and 60 ft. Maximum times at each depth are shown; times at 60, 40, and 20 ft are unlimited; decompression occurs in increments of 2 ft. When decompression occurs deeper than 165 ft (50 m), a 16%–21% O_2 in helium mixture can be administered to reduce narcosis. Four treatment cycles, each consisting of 25 min of "treatment gas" followed by 5 min of chamber air, can be administered deeper than 60 ft. Treatment gas used deeper than 60 ft is 40% O_2 in either He or N_2; at 60 ft (18 m) or shallower, treatment gas is 100% O_2. For O_2 administration at 60 ft or shallower, U.S. Navy Treatment Table 7 guidelines are used. Further details can be found in the *U.S. Navy Diving Manual.*[22] (From Moon RE, Dear GDL, Stolp BW: Treatment of decompression illness and iatrogenic gas embolism. Respir Care Clin North Am 5:93–135, 1999.)

ASSESSMENT OF THE PATIENT

Medical History

Breathing compressed gas is usually a precondition of DCS, although DCS has been reported after repetitive breath-hold dives.[23,24] The history should include the dive profile, rate of ascent, symptom onset time, and changes in symptom type or intensity. Whereas pulmonary barotrauma and AGE can occur after a breath-hold ascent of only a meter or two,[25] in situ gas formation requires a significant time at depth to allow sufficient inert gas to dissolve in tissues.

An independent account from a dive buddy or instructor is often useful, particularly if the patient has been unconscious and is unable to relate details because of lack of awareness. However, a secondhand account is often less accurate than a history obtained directly from the diver. Although the depth-time profile cannot be used to establish or exclude the diagnosis of DCS, comparison of recent dive profiles with an established decompression table can provide qualitative assessment of inert gas load. If depth-time details cannot be accurately established by history, it may be possible to obtain them directly from a decompression computer worn by the diver. Slit-lamp microscopy to detect bubbles in the eye-surface tear film and A- and B-mode ophthalmic ultrasonography to detect bubbles at the lens–vitreous interface empirically correlate with inert gas load,[26] but whether these methods are useful in assessing the diver with DCS has yet to be established.

When allowable depth-time limits have been egregiously exceeded, the risk of DCS can be exceptionally high and even approach 100%. However, DCS can also occur even when appropriate decompression procedures have been followed. If there are no risk factors for gas embolism, such as rapid ascent or breath-holding, serious DCS symptoms are rare unless the maximum dive depth is greater than approximately 70 ft (20 m). Inner-ear DCS usually (but not universally) requires a maximum depth of at least 80 ft (24 m).

The time of onset of symptoms varies with the type of diving. In recreational divers reporting symptoms of DCI of all types (i.e., DCS and AGE) to the Divers Alert Network (DAN), the median time of onset (time at which 50% of symptoms have occurred) is 30 min in the absence of altitude exposure. Within 24 hours, 90% of all cases have become symptomatic.[27] The most severe cases present shortly after surfacing, as evidenced by a systematic review of over 1000 cases of neurologic DCI in recreational and professional divers.[28] In that series, 50% of divers became symptomatic within 10 min of surfacing and 85% did so within 1 hour. Of divers with cerebral symptoms usually caused by AGE, 96% became symptomatic within 1 hour. Therefore, the longer the delay between surfacing and the onset of a symptom, the less likely it is due to DCI. However, symptoms can occur after 24 hours in rare cases.

DCI can be a localized or a multisystem disease. In early descriptions of DCS in compressed-air workers[14] and in military divers[29] pain was present in over 90% of cases, with other symptoms, particularly neurologic ones, occurring in the minority. However, neurologic symptoms are considerably more frequent in recreational divers. Among cases of DCI in recreational divers reported to DAN in 2000, nearly 75% had neurologic symptoms or signs (Tables 10–1 and 10–2).

Comparing the two types of divers, one might expect professionals to be less likely to deviate from established procedures than recreational divers. Indeed, more than half of diving accidents in recreational divers reported to DAN in 2000 were associated with procedural problems such as buoyancy, rapid ascent, missed decompression, and being out of gas. Thus, recreational divers are more likely to experience pulmonary barotrauma and intraarterial gas. Moreover, professional divers, who almost invariably dive in close proximity to a chamber, are likely to be treated quickly after the onset of symptoms, whereas treatment delay is likely with recreational divers, thus allowing their symptoms to worsen. Indeed, in 2000, only 24% of recreational divers were treated within 6 hours and 39% within 12 hours.[27] Although the first symptom was neurologic in 39% of these cases, neurologic symptoms eventually developed in nearly 75% (see Tables 10–1 and 10–2).

Altitude exposure, even in the cabin of a commercial aircraft, can precipitate DCS. In recreational divers, the majority of altitude-precipitated DCS cases occur when the flight takes place within 24 hours of surfacing from a dive.[30] The most common symptoms of

Table 10–1. Frequency of symptoms of decompression illness in 605 recreational dive accidents reported to the Divers Alert Network in 2000*

Symptom/Sign	Occurrence as a First Manifestation (%)	Total Occurrence (%)
Pain	39.6	59.8
Paresthesias	26.3	60.3
Headache	7.4	20.2
Fatigue	6.6	18.8
Dizziness or vertigo	5.3	15.5
Skin	3.5	8.2
Strength	3.1	15.9
Nausea	2.8	12.9
Mental status	2.0	9.5
Altered consciousness	1.8	2.3
Pulmonary	1.2	5.2
Coordination	0.2	6.2
Reflexes	0.2	0.7
Cardiovascular	0.2	0.5
Bladder or bowel	0.0	2.2
Hearing	0.0	1.0
Lymphatic	0.0	0.8

*Cases include both decompression sickness and arterial gas embolism.

Table 10–2. Severity of signs and symptoms of decompression illness in 699 recreational dive accidents reported to the Divers Alert Network in 2000*

Severity	% Cases
Mild neurologic	46.5
Severe neurologic	27.2
Pain only	19.5
Cardiopulmonary	3.3
Constitutional	2.9
Lymphatic/skin	0.7

*Cases include both decompression sickness and arterial gas embolism.
Mild neurologic: Numbness, tingling, abnormal sensation, twitching, dizziness, vertigo; *Severe neurologic:* Bladder or bowel dysfunction, impaired consciousness, incoordination, gait abnormalities, ataxia, hearing impairment, tinnitus, vision impairment, abnormal mental status, dysphasia, abnormalities of mood or memory, disorientation, personality change, motor weakness, abnormal reflexes; *Pain only:* Ache, cramps, discomfort, joint pain, pressure, spasm, stiffness. *Cardiopulmonary:* Arrhythmias, palpitations, pulmonary, cough, hemoptysis, shortness of breath, respiratory distress, voice change. *Constitutional:* Fatigue, headache, nausea, vomiting, chills, diaphoresis, heaviness, lightheadedness, malaise, restlessness. *Lymphatic/skin:* Swelling, burning, itching, marbling, rash.

such exposure are pain, paresthesias, and numbness. Altitude exposure after a saturation dive can precipitate symptoms even several days after the end of a dive.[31]

Obtaining a history of previous health problems may provide information about risk factors for DCI and the potential that symptoms are due to a condition unrelated to diving (see later).

Physical Examination

General physical examination should include measurement of vital signs and a search for evidence of pulmonary barotrauma (pneumothorax, pneumomediastinum, subcutaneous emphysema) and otic barotrauma (erythema or rupture of the tympanic membrane, blood or fluid in the middle ear). Rarely, in cases of AGE, bubbles can be observed in the retinal vessels. In DCI, a nonspecific skin rash occurs occasionally. Lymphedema, although rare, may indicate obstruction of the lymphatics by gas bubbles, usually in the trunk. A highly specific sign is a marbled pattern in the skin (discussed later under Cutaneous Decompression Sickness [Skin Bends]).

The examination of a patient with pain-only (musculoskeletal) DCS usually reveals no evidence of joint inflammation; occasionally, there is pain on movement. One physical sign is alleviation of pain when a sphygmomanometer is wrapped around the affected joint and inflated.[32] Similarly, pain in the hips or legs may diminish when the patient stands up, presumably because the resulting

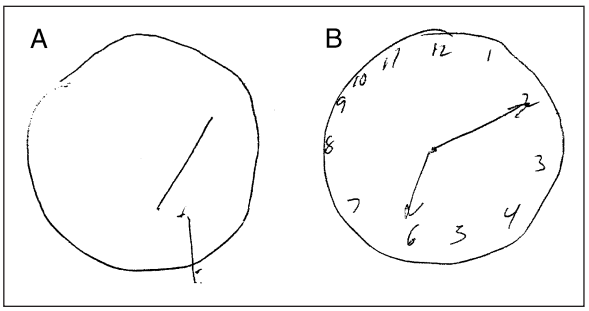

Figure 10–4. Drawings of a clock by a 47-year-old male after two dives to depths of 175 and 106 fsw, respectively. Vertigo, vomiting, ataxia, and confusion developed shortly after he surfaced from the second dive. Abnormal neurologic signs included right–left confusion and dyscalculia. The clock faces indicate parietal lobe dysfunction before and after resolution. *A,* After three U.S. Navy Table 6 treatments. *B,* Improvement after an additional hyperbaric oxygen treatment.

increase in local tissue pressure tends to compress bubbles. However, the exclusion of DCS cannot rely on the absence of this sign.

Physical examination specific to DCI should always include a neurologic evaluation that includes an examination of brain and spinal-cord function. The patterns of abnormality observed in DCI are usually different from of those in stroke. In DCI, patchy areas of hypesthesia, isolated urinary sphincter abnormality (usually urinary retention), and ataxia are common but may not be detected by an abbreviated neurologic examination with the patient lying on a stretcher. In particular, the patient should be asked to walk and perform tandem gait (heel-to-toe walking). A normal person should be able to perform tandem gait forward and backward with the eyes open or closed. An inability to perform these maneuvers supports the diagnosis of DCI. The sensory examination should also be thorough. If urinary retention is suspected, elevated residual bladder volume can be confirmed by inserting a urethral catheter after the patient has attempted to void. Cerebral involvement can produce signs pertinent to the area of injury (Fig. 10–4).

Diagnostic Tests

Abnormalities on chest radiographs, which have been observed in DCI, include pulmonary edema in cardiorespiratory DCS ("chokes"),[33] focal opacities due to aspiration of water or vomitus, or pulmonary overdistention.[34] Demonstration of these abnormalities by imaging rarely affects treatment. However, conditions that can predispose to pulmonary barotrauma, such as cystic lesions or bullae,[35,36] and evidence of barotrauma, such as subcutaneous or mediastinal gas, may support the diagnosis of gas embolism if other evidence is weak. Furthermore, a significant pneumothorax may dictate the insertion of a thoracostomy tube before recompression treatment is undertaken.

Abnormalities have been reported using neuroimaging techniques such as computed tomography (CT),[37–39] magnetic resonance imaging (MRI),[40–44] single photon emission tomography (SPECT),[45–47] and positron emission tomography (PET).[48] MRI can detect abnormalities in the spinal cord.[40,49,50] However, the value of imaging techniques in the management of DCI has not been demonstrated, except to exclude unrelated conditions that require different therapy, such as hemorrhage. For follow-up to investigate the cause of a diving accident, chest CT can detect possible predisposing factors (such as bullae or cysts) that are not easily observed with plain chest radiography.[35,36,51,52]

Tests of abnormal neurophysiology, such as electroencephalography,[38] brain stem auditory evoked responses, and somatosensory evoked responses,[41] have been investigated as end points for DCI. Although most electrophysiologic techniques are neither sensitive nor specific enough to be recommended routinely, they are of considerable value in DCS and barotrauma involving the inner ear. In these conditions, both audiography and electronystagmography are more accurate than clinical examination. Indeed, 4 to 6 weeks after an injury involving the vestibular apparatus, a normal clinical assessment can belie the existence of residual damage, which can only be detected using electronystagmography with caloric stimulation.[53,54]

Some laboratory abnormalities have been described in DCI, but there are no specific blood markers of the disease. Serum creatine phosphokinase can be elevated in AGE (predominantly the MM and MB isoenzymes),[55,56] presumably because of myocardial or skeletal muscle injury. Hemoconcentration may

Box 10–1. Conditions that can mimic decompression illness

Contaminated breathing gas (carbon monoxide)
Near drowning and hypoxic brain injury
Seafood toxin poisoning
  Ingestion
  Ciguatera[221–223]
  Puffer fish[222]
  Paralytic shellfish[222, 224]
  Envenomation
  "Sea stroke"[225]
  Sea snake[222]
  Cone shell[222]
Migraine[226–228]
Guillain-Barré syndrome
Porphyria
Multiple sclerosis
Transverse myelitis
  Spinal cord compression (e.g., due to disk protrusion, hematoma, tumor)
  Middle ear or sinus barotrauma with cranial nerve compression[62-69]
  Inner ear barotrauma[53, 62]
  Post-ictal state after unrelated seizures
  Ischemic[229] or hemorrhagic stroke
  Subarachnoid hemorrhage
  Cold water immersion pulmonary edema[230–233]
  Unrelated seizure (hypoglycemia, epilepsy)
  Functional abnormality[234]
  Acute psychosis[235]

also be a feature of severe DCI,[57–59] probably due to endothelial damage and the consequent loss of plasma into tissues ("third space" loss). When contaminated breathing gas is suspected because of symptoms or signs of carbon monoxide poisoning, a blood carboxyhemoglobin level decides the diagnosis. Other metabolic causes of encephalopathy, such as hypoglycemia and drug or alcohol intoxication, can be confirmed by appropriate blood or urine assays.

In experienced hands, neuropsychological tests can demonstrate abnormalities in DCI that may not otherwise be evident.[60] Short neuropsychological tests can be used to follow the course of treatment, but their administration requires trained personnel. Psychometric testing remains a potentially useful technique in the evaluation and treatment of DCI, and additional study of this modality is clearly warranted.[61]

Differential Diagnosis

Onset of pain, rash, dyspnea, or a neurologic abnormality after a dive is usually (correctly) assumed to be due to DCI. Symptoms that develop during a dive, while the diver is on the bottom, are not usually caused by DCI. Unrelated disease processes may coincidentally become evident during or shortly after a dive and can therefore sometimes go undiagnosed. Barotrauma of ascent can cause compression of cranial nerves V_2 (due to increased pressure in the maxillary sinus) and VII (due to increased middle ear pressure).[62–69] Strains, sprains, and nerve entrapment syndromes could easily be attributable to trauma or swelling. On the other hand, DCS involving lymphatics and muscle can present with swelling and tenderness.[70,71] DCS can also present as a mononeuropathy involving the ulnar, median, and deep peroneal nerves.[72–74] Severe symptoms that begin after more than 6 hours following decompression without altitude exposure, and any symptom occurring more than 24 hours after surfacing, should raise the suspicion of another diagnosis. A diagnosis of DCI should also be reevaluated in a diver who fails to improve despite prompt recompression treatment. Several diagnoses with which DCI may be confused are listed in Box 10–1.

TREATMENT

Natural History of Untreated Decompression Illness

Although anecdotal reports indicate that DCI, particularly isolated musculoskeletal DCS, can resolve spontaneously without recompression treatment,[3,9,14,75] experience with untreated neurologic DCS before recompression therapy was available indicates substantial morbidity and mortality.[2,3,8,9,14,76] In a series of serious DCS cases in the Royal Navy between 1965 and 1984, spontaneous recovery occurred occasionally (8 of 187 divers).[77]

Emergency Treatment

AIRWAY, BREATHING, CIRCULATION

As with any emergency, immediate first aid must include assessment of the airway, establishment of ventilation, and ensuring that blood pressure is adequate. Hypotension should be treated initially by placing the patient in the supine position, or if the blood pressure does not respond, in the head-down position. Dehydration and hemoconcentration are common in DCI[58,59,78] and can be severe in serious cases.[57] Fluid administration therefore should be instituted (see the section on Fluids later in this chapter).

If patients are apneic, foreign material should be removed from the airway and artificial ventilation administered. If divers are unconscious and breathing, they should be placed on the side to minimize the risk of aspiration of vomitus. In unconscious or apneic patients, an endotracheal tube should be inserted as soon as possible for airway protection and, if necessary, mechanical ventilation. If divers are pulseless, external chest compression should be instituted.

BODY POSITION

Traditional teaching included a recommendation that the patient with AGE should be positioned head-down. It was thought that if some residual air remained in the pulmonary veins or left side of the heart, this position would reduce the probability of additional embolization. This idea was supported by the dog studies of Van Allen.[79] However, the volumes of air used in those studies were extremely large and may not be clinically relevant. In embolized cats, Atkinson demonstrated that the head-down position might have another beneficial effect: the tendency to increase intravascular pressure and reduce the size of air bubbles in intracranial arteries, forcing them to move distally into smaller vessels.[80]

Evidence suggests that the head-down position does little to ameliorate the effects of venous gas embolism[81] or prevent arterial gas from entering cerebral arteries,[82,83] and this position can promote cerebral edema.[80,84] However, for treatment of hypotension, a brief period in the head-down position, pending intravenous fluid administration, may augment cardiac filling and increase blood pressure.

Although the head-down position is no longer routinely recommended, placement of the patient in the supine position has advantages over upright posture. In the dehydrated diver, the supine position may prevent postural hypotension. The supine position also significantly enhances inert gas washout.[85,86]

OXYGEN

Supplemental oxygen administration is appropriate to treat hypoxemia, which in the diving accident victim can be due to pulmonary abnormalities such as aspiration of water or vomitus, pneumothorax, venous gas embolism or hypoventilation. Even in the absence of lung disease, hyperoxygenation of the blood augments oxygen delivery to hypoperfused tissue. Finally, when one is breathing 100% oxygen, the absence of inert gas in the inspired mix enhances washout of inert gas from tissues, thus increasing the partial pressure gradient for diffusion of inert gas from bubble into tissue (Fig. 10–5). When 100% oxygen is administered to experimental animals, bubbles shrink more rapidly than if the animals breathe air.[2,87–90] Clinical experience also bears out the effectiveness of oxygen administration to injured divers. In recreational diving accidents reported to DAN, symptoms resolve spontaneously significantly more frequently when first-aid oxygen is administered.

Although any increase in inspired oxygen concentration is probably helpful, it is likely to be most efficacious when administered at a concentration of 100%, that is, via a tightly

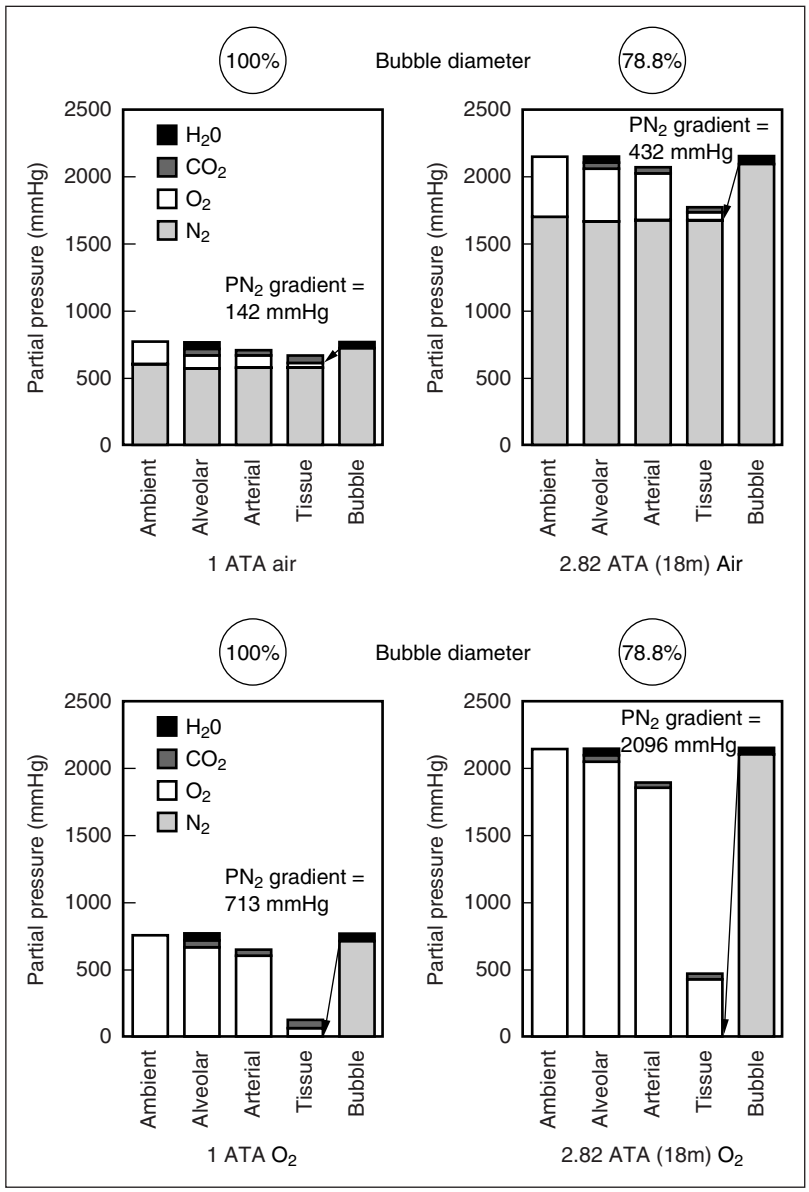

Figure 10–5. Partial pressures of gases in tissues as a function of inspired gas and ambient pressure. One of the factors determining bubble shrinkage is the difference in partial pressure between the inert gas in the bubble and the surrounding tissue. The initial gradient of 142 mm Hg is increased threefold if ambient pressure is increased to 2.8 ata while air is breathed. Breathing 100% oxygen substantially magnifies this gradient, especially at increased pressure. The *circles* represent the relative sizes of spherical bubbles under the different pressure conditions. Note that for a spherical bubble, an increase in pressure from sea level to 2.8 ata (18 m, 60 ft) reduces bubble diameter by only 29.2%. (Redrawn from Moon RE, Gorman DF: Treatment of the decompression disorders. *In* Bennett PB, Elliott DH [eds]: The Physiology and Medicine of Diving. Philadelphia, WB Saunders, 1993, pp 506–541.)

fitting mask, either from a demand valve regulator or closed-circuit apparatus (Fig. 10–6).

IN-WATER TREATMENT

When DCI occurs in remote areas, it may be tempting to reenter the water and accomplish immediate recompression by descent using a scuba breathing apparatus. Hawaiian fishermen have provided anecdotal evidence of this system's efficacy.[91] This technique has several disadvantages, however, including additional uptake of inert gas, the inability of the diver to communicate effectively during treatment, and the potential for a diver who is inattentive or with impaired consciousness to lose the mouthpiece and drown.

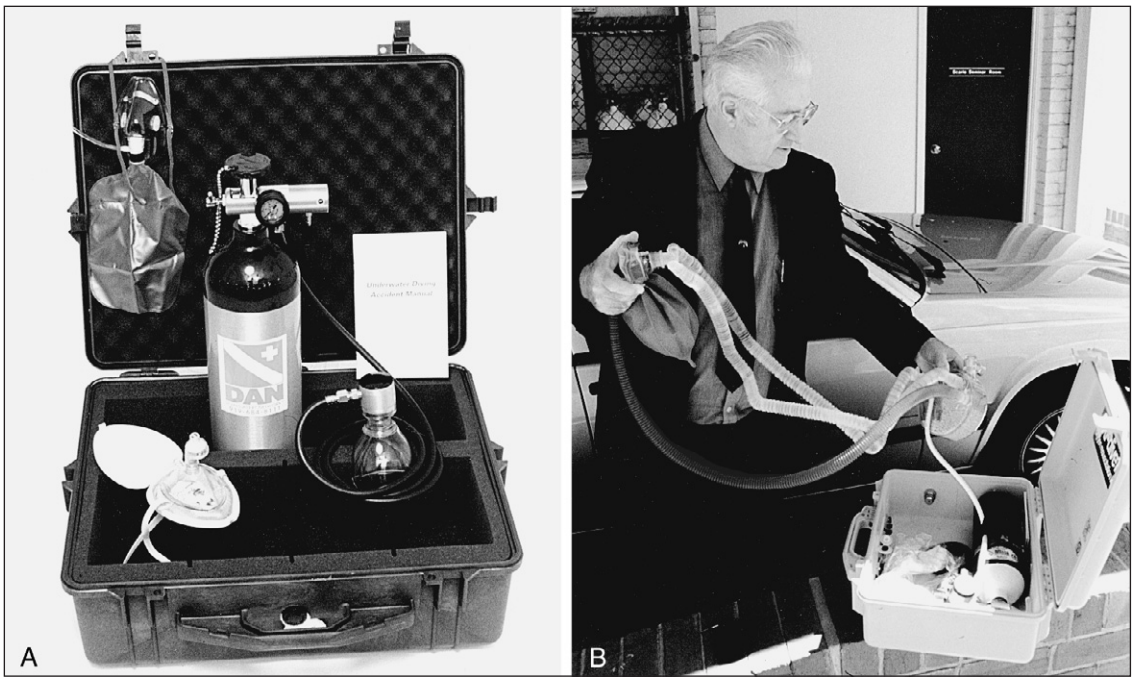

Figure 10–6. Emergency oxygen administration kits. *A,* Emergency nonrebreathing oxygen kit (available from the Divers Alert Network). Included with the cylinder of oxygen containing 646 L (Jumbo "D" cylinder) is a nonrebreathing mask *(upper left),* a tightly fitting mask with demand valve *(below right),* and a pocket mask *(below left)* that allows the rescuer to provide mouth-to-mask ventilation with a high concentration of inspired oxygen. *B,* Emergency rebreathing oxygen kit. Oxygen is delivered to the system from a cylinder containing 320 L of oxygen (a 480 L capacity tank is an option). The mask is connected to a low-pressure circuit, with each limb containing a unidirectional valve. Exhaled gas is passed through a carbon dioxide absorber (held in the left hand). The right hand holds a bag that provides a volume "buffer" and facilitates positive-pressure ventilation of an apneic patient. The nonrebreathing apparatus has the advantage that when used with a tightly fitting mask the oxygen delivery rate can be significantly lower (as low as 0.5 L/min). In order to eliminate nitrogen from the circuit and ensure a high inspired oxygen concentration, the system must be periodically flushed with oxygen. (Photos courtesy of Dr. Harry Oxer.)

These disadvantages have been addressed by the use of a full-face mask to administer 100% oxygen, which allows the diver to communicate while underwater and protects the airway of a semiconscious diver. The Australian technique incorporates oxygen delivered to the diver from a tank on the surface with a nonreturn valve between the supply line and the mask. The suggested initial compression depth is 9 m (30 ft). Time at depth is usually 30 min, although this can be extended for 30 or 60 min in severe cases. Ascent is at a rate of 12 min/m (4 min/ft). After surfacing, the diver is given 100% oxygen for 1-hour periods, interspersed with similar periods of breathing air. A diver attendant should always accompany the patient, and the injured diver should have adequate thermal protection.[92–95]

Underwater oxygen treatment is frequently used by Australian professional abalone divers and pearl divers of northwest Australia, but it has also been successful in colder waters. It has been reported to be efficacious in treating even serious cases of DCI.[95] Underwater oxygen treatment should only be considered if timely evacuation to a conventional hyperbaric facility is not feasible.

Definitive Treatment

Definitive treatment of DCI starts with recompression to reduce bubble size and promote bubble resolution. Immediate recompression treatment can both reverse the acute effects of bubble formation (e.g., tissue distraction and vascular occlusion) and prevent secondary effects. Delay of recompression treatment may result in endothelial leukocyte adherence, platelet deposition, fibrin clot formation, and neuronal damage or death due to ischemia and its consequences. Delayed recompression treatment may there-

fore be less successful initially and require multiple hyperbaric oxygen treatments and adjunctive therapy as described later.

Recompression

RATIONALE

The volume of tissue gas is reduced by an increase in the ambient pressure. Compressed-air workers in the nineteenth century reported that DCS symptoms, which initially occurred after decompression from the high-pressure environment, would often disappear when they reentered the environment on the next shift. The efficacy of recompression treatment was systematically examined by Keays[14] and since then has been supported by clinical experience. Although compression alone cannot reduce bubble volume to zero, the volume reduction is usually sufficient to reduce symptoms. Subsequent decompression must then be controlled at a rate that allows resorption or dissipation of the bubbles.

BREATHING GASES

Although recompression with the gas used during the dive (most commonly air) results in bubble shrinkage, the recompression itself engenders additional uptake of inert gas. During the subsequent decompression, this gas load then may augment bubble growth and precipitate new or recurrent symptoms. Therefore, oxygen is preferable, which is metabolized by tissues and therefore does not accumulate as an inert gas does. This results in a reduction of total gas pressure in the tissues surrounding the bubble, enhancing the rate of diffusion of inert gas from the bubble into the surrounding tissue. This is called the *oxygen window* (see Fig. 10–5).

Hyperbaric oxygen administration has other potential benefits, such as oxygenation of ischemic tissue, reduction of central nervous system edema, and possibly inhibition of endothelial leukocyte accumulation.[96]

Oxygen can be administered safely in a dry hyperbaric chamber at ambient pressures up to around 3 ata. The most commonly used treatment tables were designed to allow 100% oxygen breathing at the highest practical ambient pressure while avoiding oxygen toxicity. Higher partial pressures of oxygen present a significant risk of central nervous system oxygen toxicity (see Chapter 12). Therefore, if a higher ambient pressure is used, oxygen must be diluted with an inert gas, such that its partial pressure does not exceed 3 atm.

It is sometimes preferable to use helium as the inert gas diluent instead of nitrogen. Anecdotal evidence suggests that in divers who have breathed helium-oxygen (heliox) and decompressed all the way to the surface using this gas, nitrogen breathing can exacerbate the symptoms of DCI.[97] It therefore seems logical that if symptoms develop after a diver surfaces from such a dive, then the diver should undergo recompression using either oxygen or heliox. Experience in the U.S. Navy suggests otherwise, that air/O_2 recompression tables can be satisfactorily used to treat divers with DCS experienced while heliox is used.[98]

The work of Hyldegaard and coworkers in animal models indicates that at 1 ata heliox breathing can promote faster air-bubble resolution than air or oxygen breathing.[88,89,99,100] Studies after a compressed-air exposure in which animals were recompressed to 18 m equivalent depth indicate that 100% O_2 and 50-50 He-O_2 administration resolve bubbles faster in tendons, muscles, adipose tissue, and the spinal cord than either air or 80-20 He-O_2.[90] Bubble-resolution rates for 100% O_2 and 50-50 He-O_2 are similar. Whether heliox should be used routinely during recompression for DCI experienced during air dives remains an open question, but heliox appears to have little downside other than cost when treatment takes place at depths exceeding 18 m equivalent depth.

SPECIFIC TREATMENT TABLES

Several recompression schedules (commonly called *treatment tables*) have been empirically developed. The most widely used of these are U.S. Navy Treatment Table 5 and U.S. Navy Treatment Table 6 (see Figs. 10–1 and 10–2) or similar equivalents. The two-step algorithm has periods of oxygen breathing at 60 fsw (18 msw, 2.8 ata) and 30 fsw (9 msw, 1.9 ata). U.S. Navy Table 6 can be "extended" to provide additional breathing cycles at both depths, the extreme example of which is the "Catalina" table (Fig. 10–7).[101]

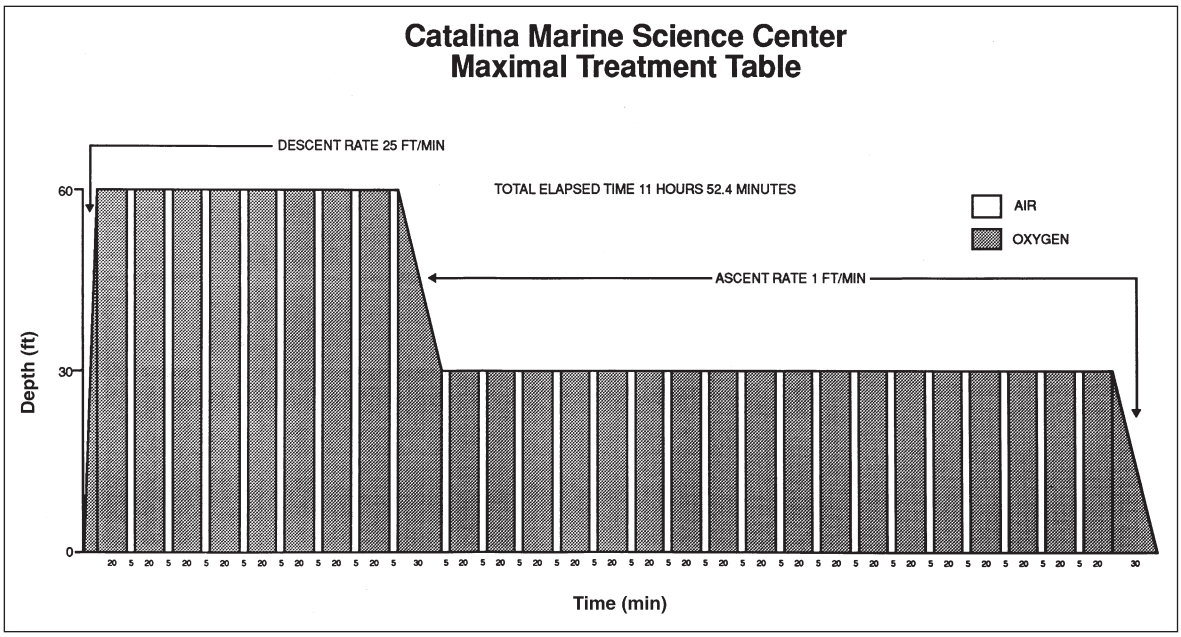

Figure 10–7. "Catalina" table. This table, developed at the recompression facility at Catalina Island, California, allows up to eight oxygen cycles at 60 ft, as shown. Oxygen cycles at both depths last 20 min, followed by 5 min of air breathing. As originally described, the number of required oxygen cycles at 30 ft depends on the number administered at 60 ft, as follows. *Three cycles at 60 ft:* minimum 6 cycles at 30 ft; *4 cycles at 60 ft:* minimum 9 cycles at 30 ft; *5–8 cycles at 60 ft:* minimum of 12 cycles at 30 ft. Up to 18 cycles at 30 ft can be administered. The tender should breathe 100% oxygen for the last 60 min at 30 ft and during ascent to the surface (total, 90 min). If there have been only 3 cycles at 60 ft and 6–8 cycles at 30 ft, then only 30 min of oxygen are required for the tender at 30 ft, although the full 60 min for all treatments probably reduces the risk of DCS for the tender and is preferred by many diving physicians. Pilmanis[101] provides additional details of this table. (Redrawn from Moon RE: Treatment of gas bubble disease. Probl Respir Care 4:232–252, 1991.)

U.S. Navy guidelines for the use of Table 5 require that it be reserved for pain-only or cutaneous DCS and that symptoms must resolve within 10 min of reaching 60 fsw. For all other situations, a different table must be used, usually U.S. Navy Table 6. In fact, many practitioners prefer to use U.S. Navy Table 6 for all instances of DCI (i.e., DCS and AGE). The vast majority of DCI cases in dives originating at the surface (e.g., nonsaturation dives) can be managed using Table 5, Table 6, or Table 6 with extensions. Oxygen cycles are administered at 60 fsw (2.8 ata) until the symptoms are relieved or until the patient is clinically stable, for a maximum time allowed by the guidelines of the particular table.

A deeper option is available, using Stolt Offshore (formerly Comex) Table 30, which incorporates initial pressurization to 30 msw (4 ata) while 50% O_2 is administered in He or N_2 (Fig. 10–8).

Saturation treatment may be an option for divers with major neurologic abnormalities who (1) continue to improve but who have reached the maximum time limit at 60 fsw (2.8 ata) or (2) experience major deterioration during decompression. In this form of treatment, the chamber pressure is maintained until clinical stability has been reached, typically for 12 hours or more. Once saturation treatment has been initiated, decompression must occur at a considerably reduced rate, and oxygen cycles have to be administered using a different schedule in order to reduce pulmonary oxygen toxicity. Saturation treatment at a chamber depth of 60 fsw (2.8 ata) is convenient because air can be used for the chamber atmosphere.[22,102,103] The U.S. Navy implementation of saturation treatment at 60 fsw (2.8 ata) is known as Table 7.[22]

Other saturation depths have been proposed[21] and may be imperative for persons undergoing decompression from either deep bounce dives or saturation dives. In such situations, both the atmosphere and treatment gases must be specially mixed such that the partial pressure of oxygen does not exceed safe limits (typically 0.5 and 3.0 ata, respectively).

Although administration of fluid and evaluation of neurologic status during treatment

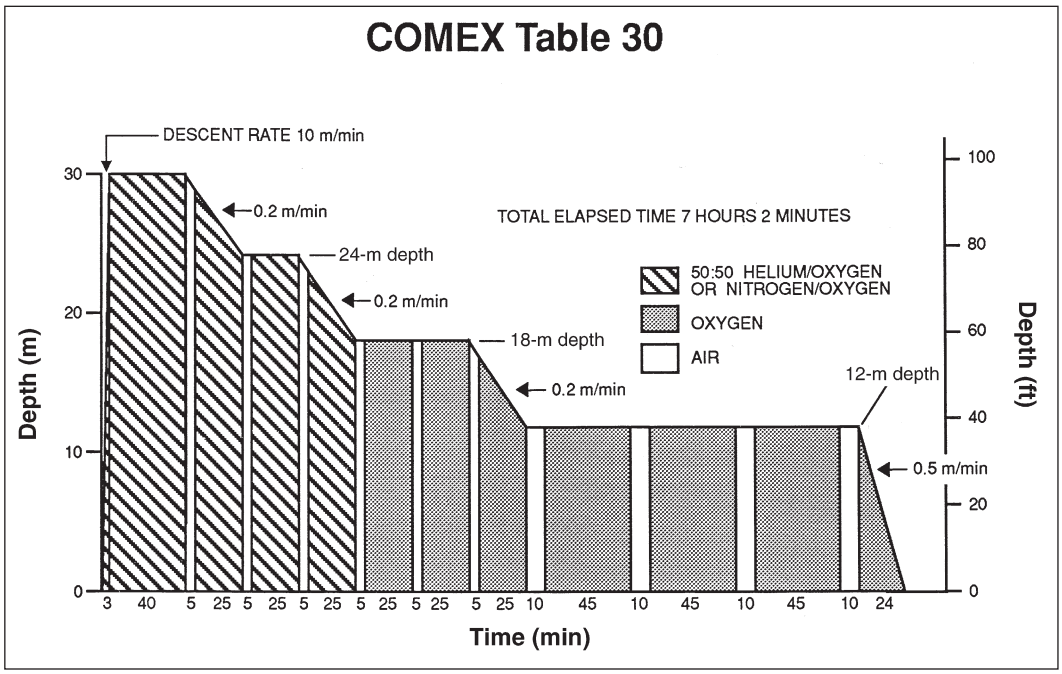

Figure 10–8. Stolt Offshore (Formerly Comex) Table 30. This table is an option for treating serious decompression illness, with a maximum pressure of 4 ata (30 m). Some anecdotal evidence suggests that He-O_2 may be more efficacious than other breathing gases, and this table allows the option of using heliox when the pressure is greater than 2.8 ata. (From Moon RE, Gorman DF: Treatment of the decompression disorders. *In* Bennett PB, Elliott DH [eds]: The Physiology and Medicine of Diving. Philadelphia, WB Saunders, 1993, pp 506–541.)

is easiest in a multiplace chamber, DCI can also be effectively treated in monoplace chambers. Modifications of the U.S. Navy tables have been described for the monoplace chamber not equipped for air breaks. Kindwall's tables for pain-only or cutaneous DCS ("skin bends") are as follows[104,105]:

- 2.8 ata (18 m, 60 ft, 26 psig) for 30 min
- 15 min decompression to 2 ata (10 m, 33 ft, 14.7 psig)
- 2 ata for 60 min
- 15 min decompression to 1 ata

This schedule requires that all symptoms resolve within 10 min of the diver reaching 2.8 ata. If not, the longer table that follows must be used.

For neurologic DCS, AGE, or pain-only or cutaneous DCS that fails to resolve within 10 min on the table just given, the following schedule is used:

- 2.8 ata (18 m, 60 ft, 26 psig) for 30 min
- 30 min decompression to 2 ata
- 2 ata for 60 min
- 30 min decompression to 1 ata

If symptoms have not resolved, the table may be repeated after 30 min of breathing air at 1 ata.

The monoplace table designed by Hart specifies 100% oxygen administration at 3 ata for 30 min followed by 2.5 ata for 60 min.[106,107] These shorter, shallower tables appear to be effective in most cases,[108] although they have not been prospectively compared with the more commonly used schedules such as U.S. Navy Table 6, and their equivalence to the longer oxygen tables in severe DCI has not been confirmed.

Monoplace chambers can be used to administer standard U.S. Navy tables and provide air breaks by installation of a mask delivery system ("built-in breathing system") for breathing air.

Treatment Algorithms

SURFACE-ORIENTED OR SCUBA DIVING

The initial Navy experience with oxygen recompression at an equivalent depth of 18 m (60 ft, 2.8 ata) was so successful that it has become the mainstay of modern recompression therapy.[109] At a pressure of 2.8 ata, 100% oxygen can be breathed with a low probability of oxygen toxicity, and both divers being treated and their tenders inside the chamber can undergo decompression relatively quickly. Most cases of DCI in diving

that commenced at the surface can be satisfactorily managed by compression to 18 m equivalent depth while the diver breathes 100% oxygen; then, one of many available treatment profiles is used. U.S. Navy Table 5 (see Fig. 10–1) can be used if the diver's symptoms are pain-only and if all symptoms resolve completely within 10 min of reaching pressure.[110] Most other cases can be managed using Table 6 (see Fig. 10–2), often with additional oxygen cycles.[110] When only a monoplace chamber is available, standard U.S. Navy tables can be administered if the chamber is equipped with a built-in breathing system that can deliver air to the diver. If there is no mechanism to deliver air breaks, a monoplace table (e.g., the Hart, Kindwall tables discussed earlier) is an option.

A single treatment is often sufficient to resolve symptoms completely. However, with residual neurologic signs or symptoms, additional treatments should be administered daily or twice daily until there is no stepwise clinical improvement. Formal analysis of 3150 cases of DCI in recreational divers from the DAN database suggests that this end point is likely to be reached with no more than seven treatments.[111,112] However, in divers with severe neurologic DCI (e.g., motor weakness that interferes with normal activities such as walking), attainment of a treatment plateau may require a greater number of recompressions. Multiple treatments for isolated musculoskeletal (pain-only) DCS is generally not recommended.

If the chamber complex and staff can support saturation therapy, then this treatment can be considered for divers with severe neurologic DCI and either continued improvement at 2.8 ata (even after reaching a maximum number of oxygen cycles) or significant deterioration during decompression.[102] In a series of DCI cases treated in Scotland, when response to U.S. Navy Table 6 was inadequate with either heliox saturation treatment at greater than 2.8 ata or an air-saturation table at 2.8 ata, 66% of patients showed a major response to treatment or better.[113]

Deeper recompression (e.g., to 6 ata—see U.S. Navy Table 6A; Fig. 10–9) can be considered for severe cases with incomplete response at 2.8 ata. Although animal studies[114,115] and a published case review[116] provide little support for treatment at pressures greater than 2.8 ata, the clinical experience of experienced commercial diving physicians is that a trial of additional recompression often produces additional improvement. This is most likely to occur shortly

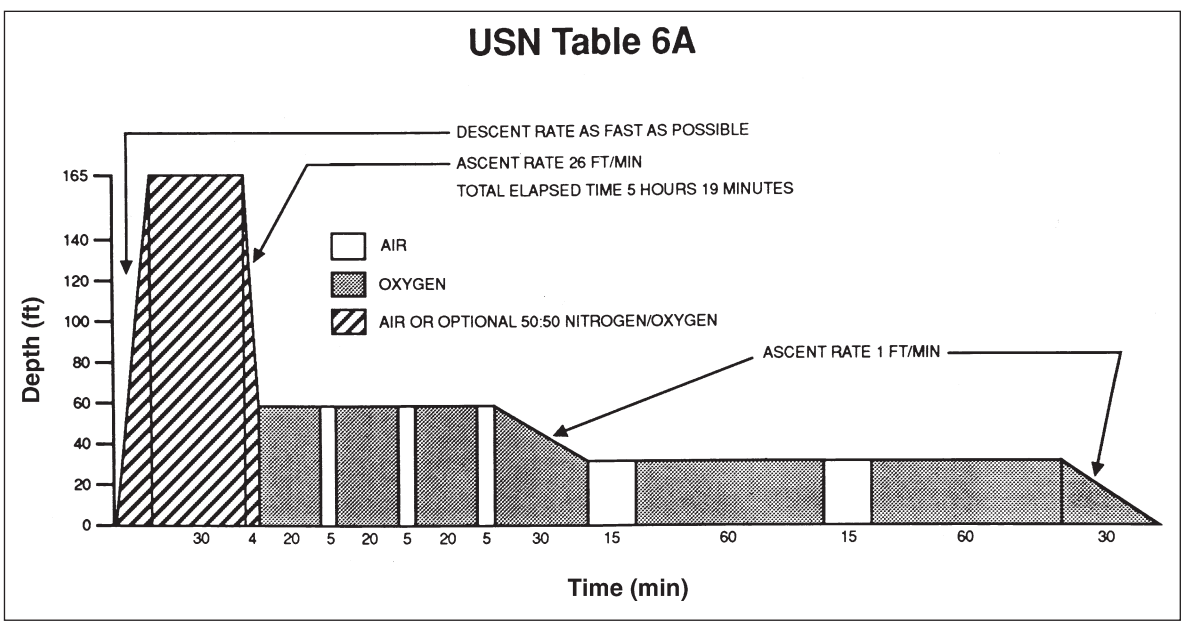

Figure 10–9. U.S. Navy Table 6A. A period of exposure to 6 ata (50 m, 165 ft) precedes the schedule of U.S. Navy Table 6. During the time at 6 ata, the diver can be given air (original implementation) or enriched-oxygen mixtures (40%–50% oxygen). Originally designed by the U.S. Navy to treat arterial gas embolism, this table has been modified by others[117] and used to treat severe cases of DCI in which treatment has been delayed (see text). (From Moon RE: Treatment of gas bubble disease. Probl Respir Care 4:232–252, 1991.)

after the onset of symptoms, at which stage symptom relief is most related to a reduction in bubble volume. After bubbles have initiated secondary pathophysiologic processes, a reduction in bubble size is only one component of multifactorial therapy, which may include rehydration, hyperoxygenation, and administration of adjunctive pharmacotherapy. In addition to reducing the volume of existing bubbles, increasing the pressure to equal or exceeding the tissue inert gas partial pressure may prevent the evolution of new ones. U.S. Navy Table 8 has been designed for treatment of deep blowups, in which missed decompression stop time has exceeded 60 min (see Fig. 10–3).

Lee and colleagues[117] published retrospective data reporting treatment using four 15 min cycles consisting of 40% oxygen and 60% nitrogen breathing (10 min), then air breathing (5 min) at 6 ata (50 m, 165 ft), followed by staged decompression over 40 min to 2.8 ata (18 m, 60 ft), and then following U.S. Navy Table 6. This table was used to cure 70 of 99 divers with neurologic or cardiorespiratory DCS (70.7%) despite delays in treatment of up to 96 hours. The authors report that this table is superior to U.S. Navy Table 6A, although the study was retrospective and not randomized and not detailed enough to allow comparisons of the patients in each group.

Immediate recompression has the greatest success, with delays tending to worsen prognosis.[118] However, the effect of delay on long-term outcome in individual cases is unpredictable: Figure 10–10 depicts a diver who presented with paraplegia but was able to return to almost normal function despite a 24-hour delay in recompression. Nevertheless, if a suitable transport chamber and a person knowledgeable in the assessment and treatment of DCI are both available, administering oxygen at increased ambient pressure during transport to a definitive recompression facility may be beneficial.[119] A collapsible, lightweight, transportable chamber is shown in Figure 10–11.

Notwithstanding the large number of options, the vast majority of cases of DCI in scuba divers or professional divers performing bounce dives can be managed by using U.S. Navy Tables 5 or 6 or, if air breaks cannot be administered, the monoplace tables described earlier. Other types of tables are best left to persons with the necessary training and experience.

CLOSED-BELL AND SATURATION DIVING

Treatment of DCS that occurs during deep closed-bell diving may require a different approach. If symptoms occur after surfacing, the diver should undergo recompression using 100% oxygen as the breathing gas. A patient who responds clinically can be treated according to U.S. Navy Table 6. If the response to the first few oxygen cycles at 2.8 ata is inadequate, saturation treatment or deeper compression using a mixed gas (usually helium-oxygen or nitrogen-oxygen)

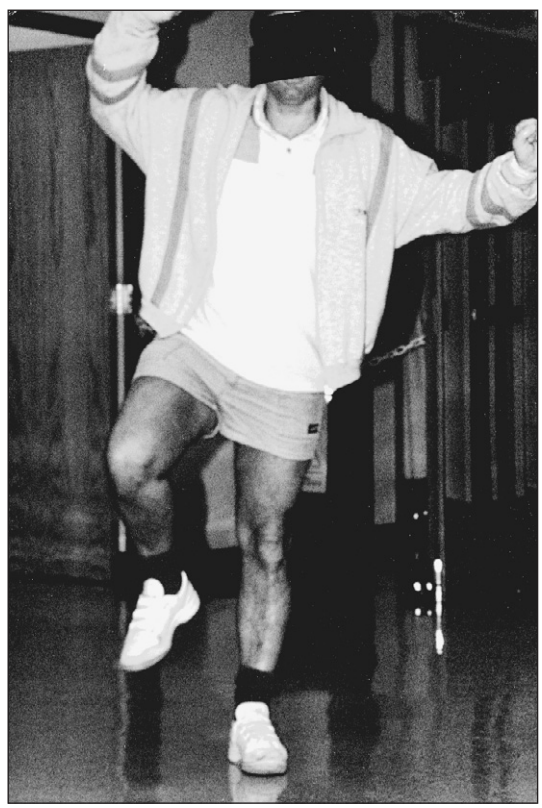

Figure 10–10. Diver after recovery from severe spinal-cord decompression sickness. This 43-year-old man suffered spinal cord decompression illness after a 37 m, 25 min dive. Immediately after surfacing, he experienced back pain and numbness in his legs and feet, which was followed by progressive leg weakness and inability to walk or void. Over 24 hours later, he arrived at a recompression facility with absent leg strength and a sensory level at T_{10}. The photograph was taken 7 months after treatment, which included recompression and intensive rehabilitation. Although he had residual paresthesias and some incoordination, he had regained the ability to walk, run, and ride a bicycle and had control of urinary and anal sphincters. The prognosis with spinal-cord decompression sickness is usually considerably better than after a clinically equivalent traumatic spinal cord injury.

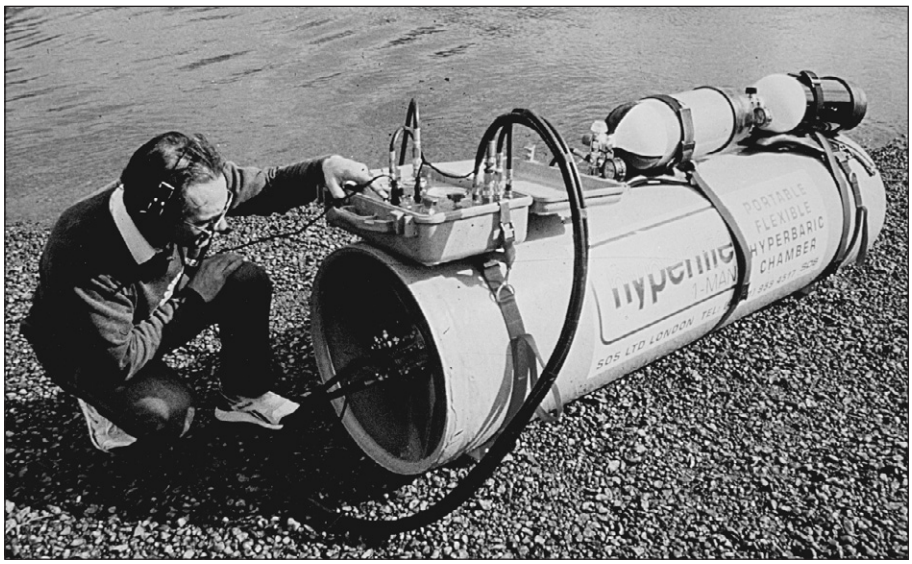

Figure 10–11. Transportable recompression chamber. This chamber, made of Kevlar by SOS Ltd (London), can be carried to remote dive sites in a collapsed state, like a concertina. The chamber can be compressed using an air cylinder to 2.8 ata, and the diver can be given 100% oxygen by mask. The unoccupied weight of the chamber equipped with compressed air and oxygen supplies is approximately 40 kg.

is an option (e.g., to a depth at which symptoms are relieved, or 10 m beyond). After a suitable time at a fixed depth at which the diver is breathing enriched oxygen mixtures, recompression can then be accomplished using special tables, such as Lambertsen-Solus Table 7A, U.S. Navy Table 8,[22] or the guidelines of the United Kingdom Association of Diving Contractors/European Undersea Biomedical Society.[120]

DCS occurring during decompression from a saturation dive can be treated by stopping the decompression, administering oxygen-enriched breathing gas (P_{O_2} = 1.5 to 3.0 ata), recompressing to the depth of relief, or combinations of these options.[121] The review of Van Meter[122] provides additional details on the practical aspects of treating DCI and other injuries in divers on site, under pressure.[122]

Exceptions and Controversies

RESOLUTION OF SYMPTOMS BEFORE RECOMPRESSION

Sometimes symptoms resolve spontaneously or after oxygen administration at 1 ata. Symptoms can return when the oxygen is stopped, and the long-term consequences of untreated DCI are poorly described. Whether nonrecompression treatment is adequate for DCI caused by diving is unknown. Pending more data, recompression therapy for divers is recommended for diving-related DCI whenever it can be administered in a timely fashion, even if the symptoms have resolved in the short term.

CUTANEOUS DECOMPRESSION SICKNESS (SKIN BENDS)

Several types of skin bends occur in divers. Pruritus, without a visible rash, is most common after use of a hyperbaric chamber or when a dry suit is worn. A nonspecific macular rash is the most common form of skin bends in scuba divers. Urticaria can occur when a diver breathes one gas (e.g., air) while surrounded by another, more diffusible gas (e.g., heliox).[123,124] A potentially serious form has a "marbled" appearance (often called *cutis marmorata*).[121,125] This form of skin bends is thought to be initiated by obstruction and congestion in the subdermal capillaries and venules.[125,126]

Urticaria usually resolves with an appropriate change in breathing gas or recompression, or both. Cutis marmorata can herald more serious symptoms and thus requires close monitoring and preferably recompression treatment. However, most diving physi-

cians think that, in the absence of other symptoms or signs, the two milder forms of cutaneous DCS will resolve spontaneously or with surface oxygen breathing.

ALTITUDE DECOMPRESSION SICKNESS

DCS due to acute hypobaric exposure without a preceding dive also responds to recompression treatment as outlined earlier and is recommended for neurologic symptoms. Less severe symptoms, however, often require only 100% oxygen breathing for 2 hours.[110,127]

LONG DELAY TO TREATMENT

There is no well-defined window of time beyond which a response to hyperbaric oxygen is not expected. Several reports have indicated a favorable response to recompression treatment, even after several days.[117,128,129] Massey and associates[130] reported significant clinical improvement in iatrogenic gas embolism even after 42 hours. Minor improvements, often subjective, may occur even after weeks of delay, but major improvement in dense motor weakness is unlikely after more than a few days.

Adjunctive Therapy

BLOOD GLUCOSE CONTROL

There is evidence that hyperglycemia can worsen central nervous system injury in both brain and spinal cord.[131,132] This enhancement of injury is thought to be due to accelerated production of lactate and the ensuing intracellular acidosis. Evidence from series of humans with head injuries[133] and rats undergoing global ischemia[134] suggests that the effect becomes significant above a threshold plasma glucose of around 200 mg/dL (11 mM). There is also evidence that administration of even small amounts of glucose (e.g., 1 L of intravenous 5% dextrose solution) may worsen neurologic outcome, even in the absence of significant hyperglycemia.[135] Except when hypoglycemia must be treated, it is advisable to avoid intravenous solutions that contain glucose and to measure plasma glucose if it might be elevated (e.g., if high-dose corticosteroids are prescribed).

FLUIDS

Fluid administration may help by replenishing intravascular volume and reversing hemoconcentration. Thus, blood pressure may be maintained and microcirculatory flow augmented. Indirect evidence suggests that aggressive hydration can result in more rapid elimination of anesthetic gases,[136] suggesting that a similar approach in divers with DCI may accelerate the washout of excess inert gas. Indeed, interventions that increase central blood volume and cardiac preload—such as supine position,[85] head-down tilt,[137] and head-out immersion[85,137]—significantly increase the rate of inert gas washout. Therefore, fluid administration may offer an advantage even in divers who are not dehydrated.

Rapid intravenous administration of fluids with an osmolality less than that of plasma can cause central nervous system edema.[138] A reduction in oncotic pressure with unchanged osmotic pressure has no effect, however, and colloidal solutions have no advantage over crystalloids.[139,140] Therefore, isotonic fluids without glucose, such as normal saline, lactated Ringer's solution, or Normosol-R (Abbott), are recommended. Hypertonic saline may improve control of intracranial pressure in patients with traumatic brain injury and intracranial hypertension,[141] although it has not been specifically tested in DCI.

Intravenous administration of fluids is preferable to the oral route in critically ill patients, but it is unclear whether parenteral fluid administration helps divers with less severe disease. The fact that oral fluids satisfactorily manage even moderately severe dehydration due to exercise or disease (e.g., cholera) suggests that the enteral route is adequate for hydration, although slower.

The composition of an ideal oral fluid should be such that absorption of both water and electrolytes is optimized. Sodium is not absorbed if its concentration in the fluid is less than 55 mM.[142] Glucose in the solution enhances water absorption, but sodium absorption is reduced at concentrations exceeding 150 mM.[142] Maximum water absorption occurs at a sodium concentration of 60 mM and glucose concentration in the

range of 80 to 120 mM. Although the osmolality of most commercially available soft drinks and juices is higher than that of plasma, water absorption is greater when osmolality is low.[143] Water absorption may also be enhanced by low concentrations of bicarbonate (18 mM)[144,145] and citrate,[146] although these anions probably do not add to the enhanced absorption provided by glucose alone. It has been suggested that an ideal solution for rehydration in diarrhea would contain approximately 30 to 60 mM sodium, contain 70 to 150 mM glucose, and have an osmolality of approximately 240 mOsm/kg.[147,148]

Rehydration after oral fluid administration is related to the rate of transport of water and electrolytes across the intestinal mucosa and the rate at which ingested fluid is delivered to the intestine. Studies using solutions with a sodium concentration of 77 mM revealed that oral fluids could restore plasma volume within an hour.[149,150] Provided the patient is not vomiting, an oral intake of 1000 to 2000 mL of fluid per hour is safe and tolerable. The gastric distention that occurs after oral fluid intake stimulates gastric emptying. However, if the ingested fluid contains protein or high glucose concentrations (over 5% or osmolality > 252 mOsm/kg), then gastric emptying can be slowed.

Ingestion of plain water is preferable to none at all, although the inhibition of vasopressin secretion caused by hypoosmolality can produce a falsely reassuring increase in urine output that can belie residual hypovolemia.[149,151] When a hot, dry environment was used to induce dehydration of 4% of body weight (12% reduction in plasma volume) in normal volunteers, plain water or glucose-electrolyte solution (sodium 22 mM, osmolality 444 mOsm/kg) failed to normalize plasma volume[151] despite an increase in urine output of 180 to 380 mL/h.

Although almost all commercially available beverages are low in sodium and high in carbohydrate, certain beverages, such as Gatorade, contain sodium and glucose concentrations that are close to ideal. World Health Organization oral rehydration salts are widely available; reconstitution of these salts with the appropriate amount of water produces a solution containing 90 mM sodium and 111 mM glucose. A reasonably palatable oral rehydration fluid with appropriate electrolyte and carbohydrate concentration can be improvised by mixing one part orange or apple juice with two parts water and adding a teaspoon of salt to 1 L of the mixture (35 fl oz). Alternatively, in lieu of adding salt, one part sea water diluted with nine parts fresh water can be used to dilute the juice.

End points for fluid therapy should at least include normal hemodynamics and hematocrit. Urine output should exceed 1 mL/kg/h, keeping in mind that if large volumes of hypotonic fluids are used, the urine output may falsely reflect the degree of plasma volume repletion. Fluid should not be withheld just because an ideal liquid is not available.

An alternative temporizing method, in which rehydration is simulated, is immersion to the neck in water. This results in redistribution of blood from the extremities to the central circulation and augmentation of cardiac output. Provided that the diver can be kept warm, head-out immersion, although rarely practical during transport, is likely to result in greater tissue blood flow and inert gas washout.[85]

CORTICOSTEROIDS

Pharmacologic doses of glucocorticoids have been used in DCI to reduce edema. In a review of cases of AGE, secondary deterioration after initial improvement occurred less often in divers who had received glucocorticoids.[152] However, this was a retrospective analysis and selection bias may have influenced the results. Another series of retrospectively analyzed AGE cases found no benefit with glucocorticoid administration.[153] Glucocorticoids have not been shown to be beneficial in the treatment of head injury[154–156] or in animal models of DCI.[84] However, in traumatic spinal-cord injury, early administration (within 8 hours after injury) of very high doses of methylprednisolone (30 mg/kg–1 given intravenously over 1 hour followed by 5.4 mg/kg–1/h–1 for 23 hours) apparently can improve outcome at 6 months after the injury.[157–159] However, primary outcomes in these studies were negative and the post hoc effects were small. Moreover, other studies did not observe a benefit.[160,161] A study using a similar high-dose regimen as a prophylactic in pigs revealed that methylprednisolone treatment did not protect against severe DCS in this model, and mortality was higher among

the treated animals.[162] Therefore, corticosteroids are not recommended for treatment of DCI.

LIDOCAINE

In models of AGE in both cats and dogs,[163,164] lidocaine administration designed to achieve plasma drug levels similar to those used clinically in humans has improved short-term neurologic outcome. A trial of lidocaine in spinal-cord DCS in pigs failed to show a benefit,[165] but the infusion was maintained for only 5 hours. A controlled trial of open-heart surgery demonstrated improved neuropsychological outcome in patients infused with lidocaine for 48 hours after the operation; anecdotal reports in DCS and AGE also support the use of lidocaine.[166–169] However, controlled trials in human DCI have not yet been published.

ANTICOAGULANTS

Because of evidence that bubble–blood interaction may cause platelet deposition and vascular occlusion refractory to recompression, agents that inhibit platelet function and soluble clotting factors might be beneficial in DCI. Administration of aspirin and other antiplatelet drugs reduces the mild drop in platelets that follows dives.[170,171] A single case report of heparin administration to a patient with neurologic DCS indicated neither benefit nor harm.[172] However, animal studies in which single agents were administered have shown no benefit of anticoagulants, except for one canine model of AGE,[173] in which only a triple combination of indomethacin, prostaglandin I_2, and heparin resulted in a short-term benefit. Furthermore, histologic evidence of hemorrhage has been described in AGE,[20] inner-ear DCS,[174] and spinal-cord DCS.[41,165,175,176]

Thus, given the lack of evidence favoring anticoagulation in neurologic DCI and the potential of worsening intraparenchymal hemorrhage, full anticoagulation is not currently recommended as primary treatment for the disease. However, low-dose anticoagulation (typically in the form of subcutaneous low-molecular-weight heparin) is usually recommended for prophylaxis against deep vein thrombosis (DVT) in patients with leg immobility. Indeed, in divers with leg weakness due to DCI, DVT and fatal pulmonary embolism can occur,[177, 178] delineating the need for some prophylaxis. The conundrum of prophylaxis versus bleeding is similar for patients with spinal-cord trauma or hemorrhagic stroke. For patients with motor-incomplete traumatic spinal-cord injury and evidence of perispinal hematoma, it has been recommended that low-molecular-weight heparin therapy be delayed until 24 to 72 hours after injury.[179] For patients with hemorrhagic stroke undergoing active treatment, elastic stockings or intermittent pneumatic compression are recommended over an anticoagulant.[179] The risks and efficacy of prophylactic measures such as low-molecular-weight heparin in patients with acute spinal-cord or brain injury due to DCI are unknown. However, general clinical principles lead to the recommendation that patients with leg immobility be given some form of prophylaxis against DVT, either pharmacologic or mechanical. If the inability to walk lasts longer than 24 hours, a screening test to detect DVT, such as impedance plethysmography, ultrasound imaging, or magnetic resonance venography, may be advisable within the first few days after injury.

Irrespective of their effects on platelet function, the analgesic and anti-inflammatory properties of nonsteroidal anti-inflammatory drugs are commonly used for the discomfort of pain-only DCS. A randomized trial of tenoxicam, a nonspecific cyclooxygenase-1 and -2 inhibitor, suggests that the agent confers a clinical benefit in pain-only and neurologic DCI.[180] In that study, tenoxicam 20 mg or placebo was administered during the first air break in the initial recompression treatment and continued daily for 7 days. There were no differences in outcome at discharge or 6-week follow-up, but the number of recompressions required to achieve a clinical end point was lower in patients treated with tenoxicam.

BODY TEMPERATURE

Numerous animal models of central nervous system injury have shown that hyperthermia significantly worsens outcome.[181] Two studies of patients after out-of-hospital cardiac arrest reported improved outcome with mild hypothermia (32° to 34°C) maintained for

12 to 24 hours.[182,183] Thus, although a recommendation to induce hypothermia for the treatment of DCI awaits more data, it is recommended that fever in a patient with DCI be aggressively treated.

Rehabilitation

Rehabilitation of the injured diver is more successful than that of the patient with a traumatic spinal-cord injury. Even after recompression treatment has reached a plateau, the patient may continue to improve slowly, usually for several months or even years. Miller and Parmentier[184] further discuss rehabilitation after DCI.

Efficacy of Treatment

The results of rapid treatment of DCI following accepted decompression procedures are excellent. Workman reported 96.3% complete relief of symptoms in 114 cases of DCS in military divers.[109] In the same report, civilian divers treated with the same protocols fared less well, with only 70% experiencing complete relief. More recent data on recreational divers indicate a similar response to recompression treatment. DAN data from 2000 show that recompression treatment completely resolved symptoms in 75% of 728 divers.[27] An additional 20% experienced improved symptoms. At follow-up of 9 and 12 months, residual symptoms existed in only 2.5% and 1.3%, respectively.

A group of 69 recreational divers who had suffered paraplegia were interviewed 2 to 10 years after injury: 50% had no residual symptoms, and 70% had no impairment of activities of daily living.[185] Fifty-eight divers (84%) reported no major difficulty in walking (Fig. 10–12). These outcomes, significantly better than outcomes that follow traumatic spinal-cord injury, occurred in spite of a median delay to recompression of 4 hours.

Multifactorial analysis of 268 divers with DCI treated in Scotland[113] revealed several significant predictors of poor outcome:

- Amateur or scallop divers versus professional divers
- Severe manifestations on referral
- Deterioration in condition from time of referral to admission
- Increasing age
- Relapse after treatment

At discharge, the majority of patients had no symptoms (60%) or a minor problem predicted to be short-term (26%); 14% of patients had a problem predicted to be long-term, and 8% were disabled. Of the subset of patients with severe disease, response to

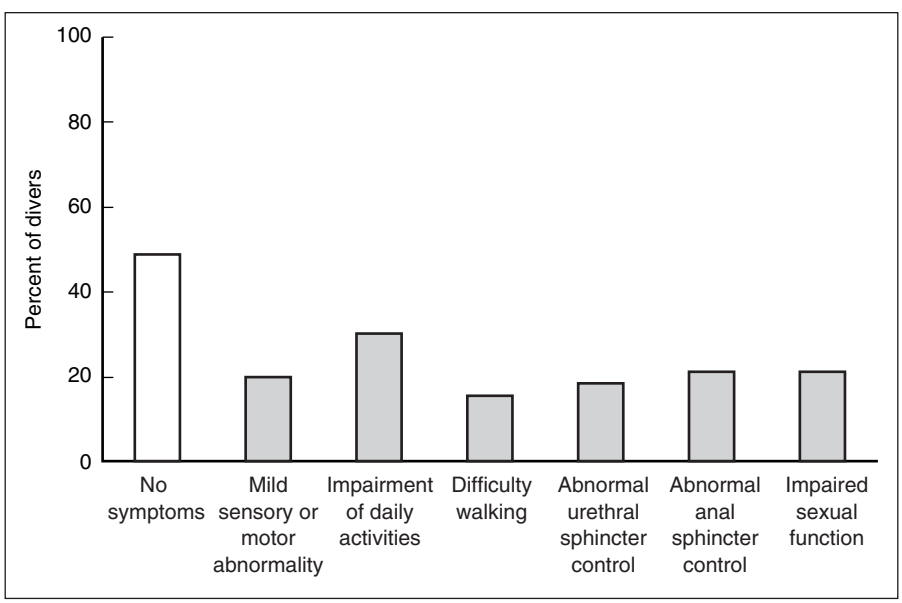

Figure 10–12. Results of long-term follow up in 69 recreational divers with decompression illness presenting as paralysis. (Data from Dovenbarger JA, Uguccioni DM, Sullivan KM, et al: Paralysis in 69 recreational scuba injuries. Undersea Hyperbar Med 27[Suppl]:43, 2000.)

treatment was poor in 25% of cases and a long-term problem remained in about half of these.

Several formal scoring systems have been devised to predict outcome.[118,186–189] Mitchell and colleagues[190] devised a detailed system that weights each symptom according to its specificity for DCI, its natural history if untreated, its potential to incapacitate, and codependence with other signs and symptoms. The final score was derived after adjustment for progression of disease before treatment. The algorithm has been tested in 79 divers and found to be highly predictive of outcome.

Flying After Treatment of Decompression Illness

After treatment of DCI, exposure to altitude can precipitate recurrence of symptoms because of either the pressure reduction (causing residual bubbles to increase in volume) or the lower ambient P_{O_2} (causing marginally perfused tissues to become transiently hypoxic). After patients reach a clinical plateau with recompression treatment, they should spend 3 to 4 additional days at sea-level pressure before flying in commercial aircraft; this period is usually sufficient to prevent symptom exacerbation. Occasionally, altitude exposure precipitates a return of symptoms (usually mild and reversible) after more than a week following recompression treatment. This seems to be most frequent in patients whose symptoms do not completely respond to recompression and is probably due to mild tissue hypoxia induced by the reduction in barometric pressure. If a long flight is required, a short test flight can provide some indication of the potential for a relapse. Commercial airlines can provide in-flight supplemental oxygen, which may enhance safety, although whether altitude-induced symptoms occurring after treatment worsen the long-term outcome is unknown.

Return To Diving

Conditions for a safe return to diving should include (1) no significant increased risk of recurrence and (2) no risk of augmenting tissue damage. All divers who experience DCS or AGE must be evaluated for predisposing factors. If the diver has experienced an incident that is inconsistent with the depth-time profile, it can be argued that there must have been a risk factor, whether or not one is specifically identifiable.

BAROTRAUMA

Risk factors for pulmonary barotrauma include a rapid or breath-hold ascent and lung disease.[35] Although data are insufficient to quantify a possible increased risk, gas trapping in obstructive lung disease such as asthma could theoretically precipitate barotrauma. Chest radiography and spirometry can help identify clinically unapparent disease that might make further diving inadvisable.

Divers with middle-ear barotrauma can safely return to diving once the signs of abnormality have resolved, hearing is normal, and the eustachian tube is functioning. Specialized evaluation and treatment is recommended for inner-ear barotrauma. Because of the potential for recurrence and permanent disability from inner-ear barotrauma, further diving is not recommended.[62]

DECOMPRESSION SICKNESS

Identified or suspected risk factors for DCS include:

- Exceeding recognized depth-time exposure limits
- Rapid ascent
- Dehydration
- Exertion during the dive
- Residual deficits from previous episodes of DCS[191]
- Obesity
- Lung disease
- Intracardiac septal defects, including atrial septal defects[192] and patent foramen ovale[193–195]

For recreational divers, the risk can probably be reduced by shallower diving, reduced bottom times, slower ascent rates (e.g., 5 to 7 m/min), and prevention of dehydration. Routine evaluation for patent foramen ovale is not necessary. However, such evaluation might be useful for a diver whose future dive profiles engender a significant risk of venous gas embolism (e.g., diving deeper than 15 to 20 m, saturation diving).

If all symptoms have resolved and risk factors are absent, U.S. Navy guidelines for returning to diving can be followed. They are as follows.[196] After pain-only or cutaneous DCS managed with the criteria for U.S. Navy Table 5, divers may return to diving after 48 hours; if U.S. Navy Table 6 was required, a symptom-free interval of 7 days is mandated. For DCS consisting of patchy sensory abnormalities, which resolve completely within two oxygen cycles using U.S. Navy Table 6, diving may resume 14 days after treatment. For all more serious cases of neurologic DCS or for divers with AGE, the minimum time before a return to diving is 4 weeks.

It can be argued that all cases observe a minimum of 4 weeks without diving. Intravascular bubbles have been observed after apparently successful treatment of DCI,[197] hemostasis may not normalize until several days after an episode of DCS, and neuropsychological tests and electroencephalography often do not return to normal until 4 weeks after the accident.[38] Especially for divers whose livelihood does not depend on diving, this conservative approach confers few disadvantages.

Future Developments

PERFLUOROCARBONS

Perfluorocarbons, designed as synthetic replacements for hemoglobin, are high soluble in a wide variety of gases, including both oxygen and nitrogen. Pretreatment of experimental animals with perfluorocarbons reduces the mortality from both AGE[198–203] and venous gas embolism.[204,205] In animal studies, intravenous perfluorocarbon administration has enhanced inert gas washout[206] and improved outcome in DCS.[207–210] Perfluorocarbons have been formulated for human use as blood substitutes and may soon be released for general use. However, caution should be exercised before using them for DCI. Increased cerebral oxygen delivery after perfluorocarbon administration could predispose to oxygen toxicity, thus rendering hyperbaric oxygen treatment unsafe.

OTHER ADJUNCTIVE AGENTS

Mechanisms of cell death in central nervous system injury have been described in recent years. Prolonged anoxia can produce cell death within minutes because of depletion of intracellular energy sources. If cell death is not immediate, reperfusion of ischemic brain can result in rapid recovery of cellular respiration and adenosine triphosphate synthesis and return of electrical activity. However, increased production of oxygen free radicals can lead to lipid peroxidation and other mechanisms of free radical injury, as well as delayed neuronal death. Further understanding of the mechanisms of secondary tissue damage may lead to preventive pharmacologic interventions.

In ischemic or traumatic brain injury, excitatory neurotransmitters such as glutamate are released. Increased extracellular glutamate facilitates the entry of calcium into cells, which can be neurotoxic.[211] Calcium can enter the cell via voltage-dependent calcium channels, which open upon neuronal depolarization or with activation of specific glutamate receptors, such as *N*-methyl-D-aspartate (NMDA) α-amino-3-hydroxy-5-methyl-4-isoxazole propionate (AMPA) and 1-aminocyclopentyl-trans-1,3-dicarboxylic acid (t-ACPD).

NMDA receptor blockers protect against focal insults,[212] and AMPA receptor blockers protect against both focal and global injury.[213–218] Excitatory neurotransmitters are released very quickly after the onset of ischemia; thus, these compounds are unlikely to be helpful unless administered immediately after the onset of symptoms. Free-radical scavenging agents have been effective in animal models of ischemic injury[219] and have the potential to reduce neuronal injury after an ischemic event even if administered after a delay.[220]

The next major advance in the treatment of neuronal injury due to DCI is likely to be pharmacologic agents to reduce the effects of acute ischemia, reperfusion injury, and delayed cell death. Unfortunately, despite the efficacy of numerous agents in animal studies, monotherapy thus far has been unsuccessful in human trials of ischemic or traumatic injury of the brain and spinal cord. This may be because of long intervals between symptom onset and seeking medical help, as well as the ineffectiveness of pharmacologic agents when administered after a delay. Such agents might conceivably be more effective in DCI: when serious neurologic symptoms occur the cause is almost always recognized, thus allowing rapid administration of a pharmacologic agent.

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CHAPTER 11 Inert Gas Narcosis and High-Pressure Nervous Syndrome

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This book discusses many of the medical problems associated with diving, as summarized in Figure 11–1. It is apparent that two problems—*nitrogen narcosis* (better known as *inert gas narcosis*) and *high-pressure nervous syndrome (HPNS)*—appear as major limitations. Nitrogen narcosis is likely to occur in scuba divers or in other divers breathing compressed air deeper than 100 ft (4 ata). HPNS occurs only in very deep diving to depths greater than 600 ft (18 ata), when divers are usually breathing oxygen-helium mixtures.

At first glance there seems to be little connection between these two conditions because they have very different signs and symptoms. However, this chapter will show that these conditions are, in fact, very closely related, although in a sense they are opposites. It is not possible to consider all of the research in past and recent years in any detail here. This can be obtained elsewhere.[1–12]

NITROGEN NARCOSIS

The condition known as nitrogen narcosis was first observed as long ago as 1835, when a Frenchman, Junod, noted that when one is breathing compressed air "the functions of the brain are activated, imagination is lively, thoughts have a peculiar charm and in some persons, symptoms of intoxication are present." Green[13] is perhaps the first American to have noted narcosis. At 160 ft (5.8 ata), he reported sleepiness, hallucinations, and impaired judgment, which he thought required an immediate return to atmospheric pressure.

The Royal Navy carried out a thorough investigation[14] when it was found that during 17 of 58 dives between 200 and 350 ft (7 and 11.6 ata), a condition resulting in "semi-loss of consciousness" occurred. The condition was regarded as serious because the diver would continue to give all the normal hand signals at depth but after decompression could not remember any of the events that took place under water.

Much speculation arose about the possible cause, but it was not until 1935 that Behnke and coworkers[15] correctly attributed the narcosis to the raised partial pressure of nitrogen in the compressed air. They characterized the narcosis as "euphoria, retardment of the higher mental processes and impaired neuromuscular coordination."

Signs and symptoms are first noticed at about 100 ft (4 ata) and become increasingly more severe as depth increases. Laughter, loquacity, and a lightheaded sensation may be apparent, with feelings of stimulation and excitement. With increased effort at self-control, it may be possible to overcome such behavior to some extent. There is a slowing of mental activity, with delays in auditory and olfactory stimuli and a tendency to word-idea fixation, as often occurs in hypoxia. The resulting limitation in the powers of association and perception is made especially dangerous because of the presence of overconfidence.

Memory is impaired, especially short-term memory. Errors may be made in recording arithmetic data (Table 11–1). For example, 43 min may be confused with 48 min, and 12:15 may be written as 15:15. Handwriting becomes larger as the narcosis becomes more severe. The sense of time may change. Intellectual capacities are affected more severely than are psychomotor or manual abilities. However, the ability to carry out fine movements is impaired, usually from overexaggeration of movements. If the movements are carried out more slowly than usual, the impairment of efficiency is likely to be less severe.

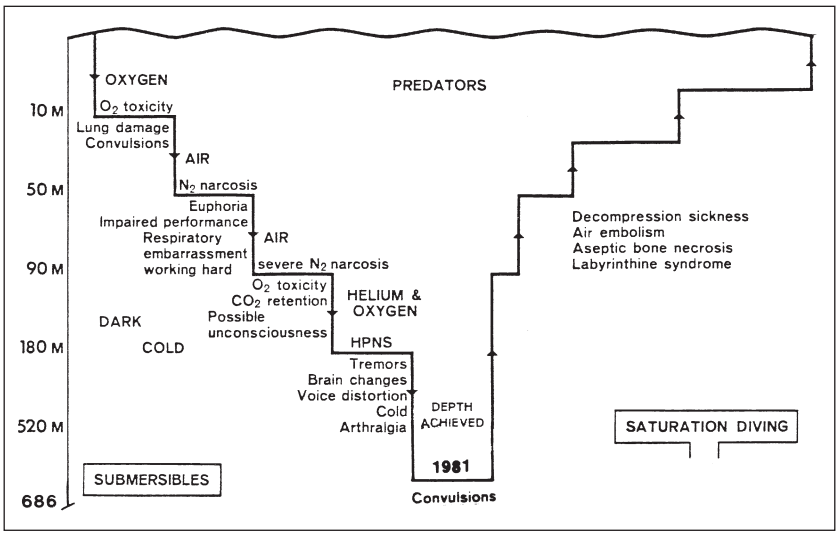

Figure 11–1. Physiologic and medical problems of diving.

Table 11–1. Effect of Pressure on Psychometric Tests

Pressure (ft)	0	90	100	125	150	175	200	225	250	275	300
Mean additional time to solve problems (seconds)	0.35	11.09	6.89	7.65	9.74	11.95	13.98	17.17	26.07	26.53	31.42
Mean additional errors in solving problems	0.18	0.86	0.49	0.42	0.72	0.84	1.22	0.88	2.18	2.66	3.02
Mean decrease in numbers crossed out	—	–0.59	–0.09	–2.26	–2.30	–2.49	–2.55	4.24	–5.85	–6.43	–8.74
Average reaction time (seconds)	0.214	—	—	—	0.237	—	0.242	—	0.248	—	0.257
Mean additional time to solve problems (acclimatized subjects)	1.64	2.55	3.42	3.91	4.66	8.00	11.75	15.73	16.33	17.09	24.36

From Shilling CW, Willgrube WW: Quantitative study of mental and neuromuscular reactions as influenced by increased air pressure. U S Navy Med Bull 35:373–380, 1937.

There may be some numbness and tingling of the lips, legs, and feet as well as a characteristic deadpan look to the face.

At depths greater than 180 ft (6.5 ata), no trust should be placed in human performance or efficiency when breathing compressed air.

At depths greater than 300 ft (10 ata), signs and symptoms are severe, with the possibility of the diver's becoming unconscious. Orders may be ignored. Intensity of vision and hearing, voice reverberation, stupor, and a sense of impending blackout and disorientation occur. Manic or depressive states can also occur, with changes in personality and a sense of levity.[16]

These signs and symptoms are similar to those seen in alcoholic intoxication and the early stages of hypoxia and anesthesia, with an equally wide variation in susceptibility. Nitrogen narcosis is an especially important danger to the compressed-air diver. The narrowing of perception that results may permit divers to carry out a specific task with varying degrees of competency; in the event that something unusual occurs, however, they are unable to function effectively in an emergency. Many divers who chose to ignore the narcosis problem or who thought, as with alcohol, that it was more "manly" to pretend to be unaffected by the condition have perished as a result.

The narcosis is usually more severe immediately on arrival at depth, and there may be some improvement shortly afterward followed by a relatively stable level of narcosis.

Table 11–2. Mean Percentage Impairment in Ability of 14 Subjects to Do an Arithmetic Test During Rest and Work on a Bicycle Ergometer (300 kg/min)

Absolute Air Pressures	4 atm	7 atm	10 atm	13 atm
At rest	–3.2	–6.9	–24.6	–61.6
During exercise	–2.1	–11.6	–39.8	—

From Adolfson J: Deterioration of mental and motor functions in hyperbaric air. Scand J Psychol 6:26–31, 1965.

This is primarily a subjective improvement because objective tests show no change.

Recovery is rapid upon decompression, although there may be some amnesia about events that occurred during the narcotic state. For example, divers may have specific instructions to perform a certain task under water; they will make the dive, become narcotic, and either not perform the task or perform it badly. On return to the surface, they may report that the task was completed satisfactorily!

Many factors, in addition to individual susceptibility, potentiate the severity of the narcosis at a given depth. In particular, any increase in exogenous or endogenous carbon dioxide potentiates the narcosis synergistically. For this reason, the narcosis is likely to be more severe in the swimming or working diver wearing a breathing apparatus than in one in a pressure chamber (Table 11–2). Similarly, hard work facilitates narcosis, as does very rapid compression, alcoholic excess or hangover, and apprehension.

Interestingly, variations in the oxygen percentage of the breathing mixture also affect the degree of narcosis. Thus, at a constant nitrogen pressure, an increase in oxygen partial pressure causes greater narcosis.[17] Although a reduction of the oxygen partial pressure may reduce the narcosis if the nitrogen partial pressure is constant, this is not the case if the reduction means a concomitant increase in the nitrogen partial pressure. For example, Albano and associates[18] noted that at 300 ft (10 ata), seven divers were more narcotic when breathing a mixture of 96% nitrogen and 4% oxygen than when breathing air (Table 11–3), a finding confirmed by Barnard and coworkers.[19]

The novice diver may be seriously affected by nitrogen narcosis, but subjectively, at least, there is some improvement with experience. Frequency of exposure seems to result in some adaptation. However, Hamilton and colleagues[20] studied subjective and behavioral effects associated with repeated exposure to narcosis by exposure to 30% nitrous oxide over 5 successive days. They noted that subjective adaptation can occur without a parallel improvement in performance, which could certainly compromise safety.

Causes and Mechanisms of Inert Gas Narcosis

Although the previous discussion described nitrogen narcosis, the general term *inert gas narcosis* is more correct. *Inert,* in this case, refers to the inability of the respired nitrogen to interact biochemically in the body. Any mechanism of narcosis must therefore be biophysical. Furthermore, nitrogen is not alone in its ability to cause signs and symptoms of narcosis or indeed anesthesia. Behnke and associates[15] related their inference of nitrogen as the causative agent in compressed air to an old, but still very valid, hypothesis that narcotic potency is related to the affinity of an anesthetic for lipid or fat. The Meyer-Overton hypothesis[21] affirms that "all gaseous or volatile substances induce narcosis if they penetrate the cell lipids in a definite molar concentration that is characteristic for each type of animal (or better, type of cell) and is approximately the same for all narcotics." For example, Meyer calculated this concentration as 0.07 moles/L for mice.

In fact, the narcotic potency of inert gases may be related to many physical constants, including molecular weight,[22] absorption coefficients,[23] thermodynamic activity,[24–26] van der Waal's constants,[27] and the formation of clathrates.[28, 29] Of these many constants, lipid solubility gives the best correlation, although polarizability and molar volume are also important in relation to the mechanism of the narcosis (Table 11–4), which involves interaction of the molecule with neuronal membranes. Thus, the size of the molecule and its electric charge are important considerations.

Although nitrogen is widely recognized as the cause of compressed air intoxication, mention must be made of an alternative, but erroneous, theory that has been promoted from time to time.[30–34] This theory implies that the increased density of the breathing gas causes respiratory insufficiency, leading

Table 11–3. Arithmetic Test Results at 10 ata

	Figures Multiplied			Percentage of Errors			Difference	
Subject	(1) Ambient Pressure	(2) 10 atm Air	(3) 10 atm 96% N_2–4% O_2	(4) Ambient Pressure	(5) 10 atm Air	(6) 10 atm 96% N_2–4% O_2	(5) – (4)	(6) – (5)
A.G.	23	18	12	4.35	22.2	41.6	17.85	19.4
P.V.	24	19	15	4.25	79	86.6	74.75	7.6
R.S.	50	43	33	10.00	23	21.8	23.00	–1.2
M.E.	40	20	14	28.00	30	42.8	20.00	12.8
S.V.	36	32	28	7.4	53.6	71.4	25.60	17.8
C.B.	27	24	20		50	60	42.60	10.0
C.U.	45	34	30		26.4	30	26.40	3.6
							M = 32.88	10.0
							t = 4.30	4.2
							p = 0.01	<0.01

From Albano G, Griscuoli PM, Cirulla C: La sindrome neuropsichica de profondita. Lav Um 14:351–358, 1962.

Table 11–4. Correlation of Narcotic Potency of Inert Gases, Hydrogen, Oxygen, and Carbon Dioxide with Lipid Solubility and Other Physical Characteristics

Gas	Molecular Weight	Solubility in Lipid (mol/L)	Molar Volume (cm^3)	Polarizability ($\times 10^{24}$ cc)†	Relative Narcotic Potency*
Helium	4	0.015	32.00	0.20	4.26
Neon	20	0.019	16.72	0.39	3.58
Hydrogen	2	0.036	28.3	—	1.83
Nitrogen	28	0.067	35.4	1.74	1
Argon	40	0.14	28.6	1.63	0.43
Krypton	83.7	0.43	34.7	2.48	0.14
Xenon	131.3	1.7	43.0	4.00	0.039 (surgical anesthesia)
Oxygen	32	0.11	27.9	1.58	—
Carbon dioxide	44	1.34	38.0	2.86	—

*In order from least narcotic to most narcotic.
†The ease with which a dipole is established in an atom or molecule in an electric field of given strength.

to carbon dioxide retention, and this increased carbon dioxide tension is the cause of the narcosis.

In fact, measurements of arterial carbon dioxide (Table 11–5) in humans breathing either compressed air or oxygen-helium mixtures (helium being a very weak narcotic at best) showed that there is no increase in arterial carbon dioxide tension at 286 ft (9.6 ata) and at 190 ft (6.7 ata). However, nitrogen narcosis occurred when nitrogen, but not helium, was present.[35] Similarly, measurements of alveolar carbon dioxide by Rashbass[36] and Cabarrou[37, 38] do not support the carbon dioxide theory. Again, Hesser and colleagues[39] have also shown that the effects of raised pressures of nitrogen and carbon dioxide are merely additive and that carbon dioxide is not the cause of compressed air intoxication.

There can be no doubt that the site of action of the narcosis in the brain is at synapses or nerve junctions, where there is a very small gap of 200 Å between the presynaptic terminal of one nerve and the postsynaptic terminal of another.[34–47] Thus, the mechanism involves interference with the electrochemical mechanisms necessary for the transfer of the electrical potential across the synaptic gap of central synapses. Polysynaptic regions of the brain, such as the ascending reticular activating system and the cortical mantle, are likely to be the regions of the brain most affected.

Just how the synapse is affected remains a controversial area of research. For some years, considerable interest revolved around the so-called *critical volume hypothesis* of Miller and coworkers.[48] This hypothesis suggested that anesthesia (and thus also inert

Table 11–5. Mean Results of Human Mental Performance and Arterial Carbon Dioxide in Air and 20:80 Oxygen-Helium Mixture

	Control	20:80 He-O_2	Air (20:80 N_2-O_2)
	At 286 ft		
Arithmetic correct	16.8 ± 1.78	15.67 ± 2.08	11.0 ± 1.73
Visual analogy test	50.5 ± 5.61	51.50 ± 5.80	44.50 ± 1.21
$Paco_2$	—	35.38 ± 4.36	34.73 ± 3.84
	At 190 ft		
Arithmetic correct	16.8 ± 1.78	18.67 ± 1.53	15.67 ± 2.08
Visual analogy test	50.5 ± 5.61	50.00 ± 5.42	51.70 ± 4.19
$Paco_2$	—	35.05 ± 2.56	32.68 ± 1.60

From Bennett P, Blenkarn GD: Arterial blood gases in man during inert gas narcosis. J Appl Physiol 36:45–48, 1974.

gas narcosis) occurs when the volume of a hydrophobic region is caused to expand beyond a certain critical volume. The theory also allowed an explanation for the "pressure reversal theory," which notes that increased pressure can reverse signs and symptoms of narcosis.[49] Thus, Lever and associates[50] hypothesized that a 0.4% expansion of a neuronal membrane could cause narcosis and, conversely, that a 0.4% contraction of the membrane from pressure alone could result in the condition known as High Pressure Nervous Syndrome (HPNS). However, the pressure reversal of different anesthetics reveals a nonlinearity that led Halsey to infer that narcosis and anesthesia may be produced by interactions at more than one site and that pressure does not necessarily act directly on the same molecular site.[51]

Furthermore, Franks and Lieb[52] have criticized the critical volume theory and other theories in relation to increased fluidity. They have inferred that the membrane protein may be the more likely site by exerting an effect on bilayer permeability or by interfering with normal membrane function in some other way, such as competing for the binding of some endogenous ligand (e.g., a neurotransmitter). Much current research is directed at understanding the presynaptic release and postsynaptic capture of neurotransmitters and the role of calcium ions in this mechanism.

Thus, McLeod and colleagues[53] noted a significant decrease in both dopamine and norepinephrine in the hypothalamus of rats exposed to 20 ata (660 ft) nitrox but moderate increases in the caudate. Balon[54] also noted a 20% decrease in striatal dopamine release in rats exposed to 90 ata (2970 ft), and Risso and associates,[55] using microdialysis, found a 70% decrease at the synaptic cleft at 8.9 ata (294 ft). Vjtosh and coworkers[56] suggested that nitric oxide may play a critical role in the mechanism of nitrogen narcosis.

Table 11–6. Comparative Percentage Impairment in Psychometric Performance of Subjects Compressed to 600 ft and 800 ft While Breathing a 5:95 Oxygen-Helium Mixture[53, 54]

	600 ft (n = 6)	800 ft (n = 4)
Sums correct	–18	–42
Sums attempted	–4	–6
Number of ball bearings	–25	–53

HIGH-PRESSURE NERVOUS SYNDROME

On the basis of the lipid solubilities shown in Table 11–4, it might be expected that helium narcosis would not occur until about 1400 ft (43 ata), compared with narcosis due to compressed air, which would occur at 300 ft (10 ata). As a result, helium was selected[22] as an alternative to compressed air for deep diving.

However, during simulated dives with rapid compressions of 20 to 100 ft/min to 600 ft and 800 ft for 1 to 4 hours in 1965, performance degraded markedly during the first hour of exposure (Tables 11–6 and 11–7), which, unlike nitrogen narcosis, was followed by a

Table 11–7. Mean Percentage Change in Performance in Subjects Breathing a 95:5 Helium-Oxygen Mixture at 600 ft for 4 Hours When Compared with Performance at Atmospheric Pressure[53, 54]

Test	Performance at Surface (Air)	Change in Performance (%) at 600 ft 20 min	600 ft 1½ h	600 ft 2 h	600 ft 2½ h	600 ft 3 h	600 ft 3½ h	300 ft (decompression)
Arithmetic (number correct)	15.67	–18	+1.02	–9.6	+9.6	+10.06	+7.4	+21.25
Arithmetic (number attempted)	19.67	–4.2	–2.61	–7.0	+4.33	+6.95	–0.88	+6.6
Ball-bearing (number of ball bearings)	10.67	–25	+9.37	+17.15	+26.53	+9.37	+15.5	+17.15

slow improvement. Furthermore, in a manner opposite to narcosis, there was a greater decrement in psychomotor tests, such as the ball-bearing test (which required the subject to pick up ball bearings one at a time with forceps and place each in a tube of the same diameter), than in intellectual tasks such as arithmetic efficiency.[57, 58] This was due to the associated presence of a marked tremor (6 to 10 Hz) of the hands, arms, or even whole body, together with dizziness, nausea, and sometimes vomiting. This was the first report at such depths of a condition now recognized as HPNS, which appears to reflect a general excitation of the brain, compared with the decreased excitation seen in inert gas narcosis.

Similar changes were reported by Brauer and associates[59] in mice and monkeys. In these animals, HPNS appears during compression, with tremors and ratchety movements. As the pressure increases, localized myoclonic jerks occur, which progress to clonic seizures. If the animal is maintained at high pressure, intermittent seizure activity occurs for as long as 12 hours. Compression beyond this point results in tonic seizures, coma, and death. Such convulsions have yet to be reported in humans.

Susceptibility to HPNS increases with increasing complexity and development of the nervous system.[60] Noting the fact that during 10 human dives at a compression rate of 24 atm/h, an oxygen partial pressure of 0.5 atm, and a temperature of 30° to 33°C, the mean threshold pressure for the onset of tremors was 26.4 atm (22 to 27 atm), Brauer and coworkers[61] calculated that convulsions should occur in humans under similar conditions at 66.3 ± 7.8 atm, or 2300 ft. The onset of HPNS, however, is markedly affected by the rate of compression; slowing or speeding up the rate of compression results in tremors and convulsions occurring at greater or lower pressures, respectively. Using this and other techniques, humans have, in fact, reached 2250 ft (686 m) without serious HPNS.[62, 63]

In 1968, a dive was performed at Duke University Medical Center to 1000 ft (31 ata) with a compression rate of 40 ft/h without any of the tremors or other signs of HPNS reported during the earlier British dives in 1964 to 1965 down to 600 and 800 ft at 100 ft/min.[64]

However, during a further series of experiments (known as Physalie) by the French,[65] with compression rates averaging about 500 ft/h, four of the dives exceeded 1000 ft (31 ata). Tremors appeared at 21 ata (660 ft), and electroencephalographic changes occurred at about 31 ata (1000 ft), with a marked increase in theta activity (4 to 6 Hz) accompanied by a depression of alpha activity (8 to 13 Hz). As the pressure increased, the EEG changes became worse and were accompanied by intermittent bouts of somnolence with sleep stages 1 and 2 in the EEG. If the subjects had work to do, they were able to function; if they stopped, they lapsed into what has been called *microsleep*. Because of the severity of the microsleep and EEG changes at that time, the deepest dive was aborted after only 4 min at 1190 ft (37 ata).

Subsequently, further depth has been achieved by the use of slower rates of compression, with or without stages. In 1970, two men were compressed for the first time to 1500 ft (46 ata), where they stayed for 10 hours. The compression rate was fast at

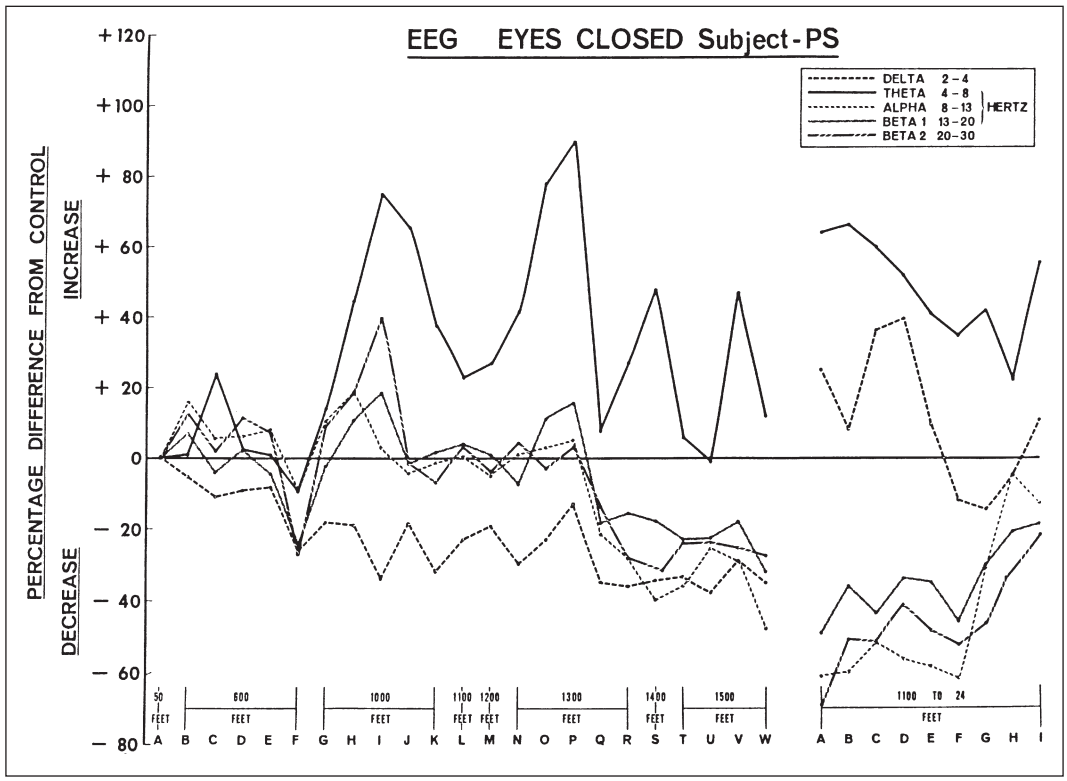

Figure 11–2. Spontaneous cortical electrical activity of the brain (EEG) in a subject compressed in stages to 1500 ft (46 ata) with online frequency analysis shows a rise in theta activity (4 to 8 Hz) with a fall in overall activity from 1300 ft (40 ata). (From Bennett PB, Towse EJ: The high pressure nervous syndrome during a simulated oxygen-helium dive to 1500 ft. Electroencephalogr Clin Neurophysiol 31:383–393, 1971.)

16 to 17 ft/min (7 to 7.5 m/min), but almost 24 hours were spent at 600, 1000, and 1300 ft (19, 31, and 40 ata, respectively). During this dive, the divers were monitored extensively.[66–69] All of the characteristics of HPNS were seen, but the divers were able to function reasonably well. The following points were clarified by this dive.

First, in regard to the EEG, the rise in theta activity was initiated on compression, especially at pressures greater than 31 ata (1000 ft). Theta activity continued to rise for 6 hours, even though compression had ceased, and then fell over 12 hours to lower levels (Fig. 11–2). On recompression, the cycle repeated. The rise in theta activity did not seem to correlate with any of the other signs of HPNS. Individual susceptibility also was apparent in the tremors. One diver showed a considerable increase in tremor, whereas the other had little response (Fig. 11–3). The occurrence of tremor in diving has been reviewed in more detail elsewhere,[4] but it should be pointed out that the tremor is in the frequency range of 8 to 12 Hz, which is a normal resting tremor and not that of Parkinson's disease or cerebellar disease (3 to 8 Hz). Hypothermia, alcoholism, and thyrotoxicosis also cause tremor in the 8 to 12 Hz range.

For the first time, too, it could be clearly seen that helium did not cause signs and symptoms of narcosis. Arithmetic performance was unaffected, but psychomotor tests, such as the ball-bearing and peg board tests, showed a decrement, mostly because of the tremors and muscular jerks.

Prevention of High-Pressure Nervous Syndrome

Because of HPNS, diving to depths beyond 1000 ft (31 ata) imposes considerable limitations on the diver, but their severity can be mitigated by a number of methods. These include choice of a suitable slow exponential rate of compression, use of long stages or holds during the compression to allow adaptation, use of nitrogen (or other narcotic) in

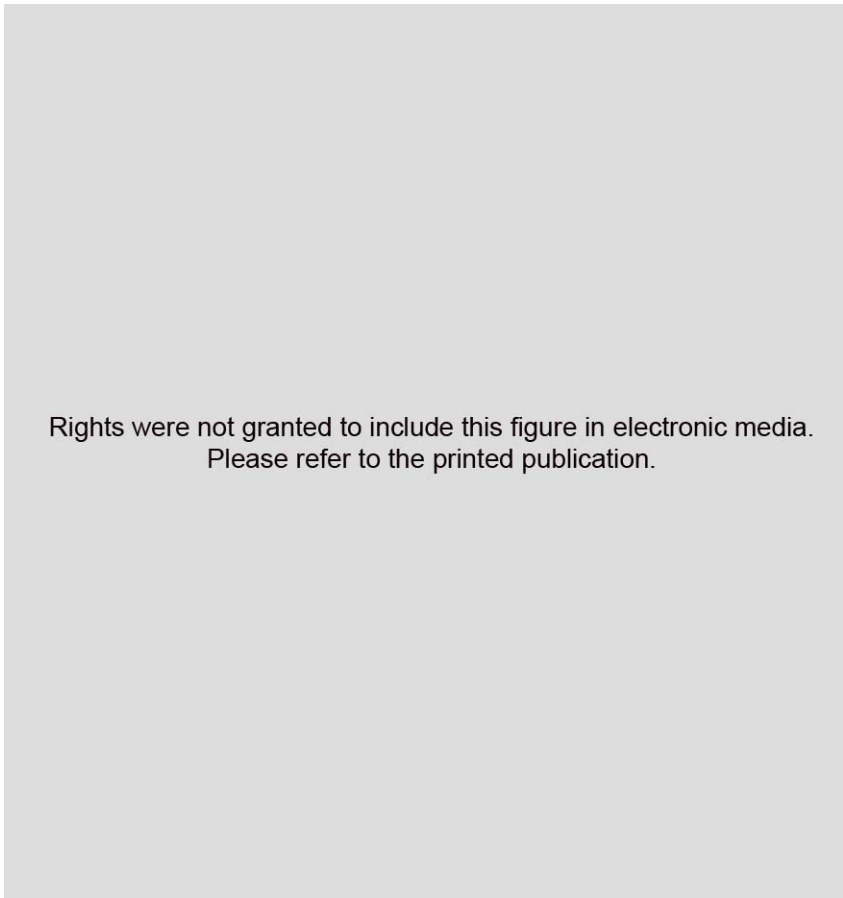

Figure 11–3. Percentage change in tremor of the hand measured by a transducer on the finger of men compressed to 1500 ft (46 ata) with oxygen-helium. Each compression phase causes a marked increase in tremor in one subject but has little effect on the other. The tremor-sensitive subject also shows an increase in base tremor. (From Bennett PB, Towse EJ: Performance efficiency of men breathing oxygen-helium at depths between 100 ft and 1500 ft. Aerosp Med 42:1147–1156, 1971.)

a so-called *trimix,* and selection of the least susceptible divers.[2, 70, 71]

In recent decades, more than 50 deep experimental dives to more than 1000 ft (31 ata) have been made in the United States, the United Kingdom, France, Germany, Norway, and Japan to study HPNS and its prevention.[2, 70, 71] These dives have involved the use of helium-oxygen mixtures, helium-oxygen mixture plus excursions to a greater depth, nitrogen-helium-oxygen mixture (trimix), trimix with excursions, and more recently a hydrogen-helium-oxygen mixture. These dives were made at a time when there seemed little operational need for diving much deeper than 1000 ft (31 ata). Today, operational diving is being done at 410 m (1345 ft), and it seems that current interest is certainly to 450 m (1476 ft). What is the best way to make such dives?

There are only two basic methods: compression with a helium-oxygen mixture or compression with trimix. The former risks incapacitating HPNS; the latter, if not used correctly, puts the diver at risk for nitrogen narcosis and limited HPNS too. Proponents for each method divide along these issues, but in fact neither is necessarily correct.

HELIUM-OXYGEN MIXTURES

During the early 1970s, studies at the British Royal Navy Physiological Laboratory (now AMTE/PL)[66–69] and by the French company COMEX[73–75] showed that depths of 1500 to 2132 ft (500 to 600 m) could be obtained by slow exponential compressions and stages.

Since then, there has been less interest in this type of dive. This is primarily because of the length of time involved with such compressions (e.g., it takes 10 days to reach 2100 ft), which often leaves the diver still affected by varying degrees of HPNS that could be incapacitating in an oceanic situation.

However, in 1976, the AMTE/PL carried out a dive to 300 m (984 ft)[72] (AMTE/PL Dive 5) using a *linear* compression rate of 1 m/min. There was nausea, unspecified epigastric sensation, intention tremor, and impending loss of consciousness. Previously, in 1969, Buhlmann and colleagues[76] made a much faster compression to 300 m (984 ft) at 5 m/min, producing only mild dizziness and an initial decrement in psychomotor tasks that was gone 2 to 3 hours later. The reasons for the differences between these two dives are not clear, but it would seem most likely to be due to personal susceptibility.

One clear characteristic of the subsequent dives with very slow compressions (AMTE/PL Dives 6, 7, and 8) is the absence of nausea and possibly little change in the EEG. However, although very slow compressions do considerably ameliorate or even prevent HPNS in suitable subjects to 300 m (984 ft), at 420 m (1377 ft), even with 6 days of compression, some signs of HPNS are still present, including loss of appetite, periods of unspecified epigastric sensation, and persistent intention tremor with occasional muscle jerks. With a further depth increment of 100 m (328 ft), these become more severe and are compounded by additional signs and symptoms that severely limit functional ability.

Thus, in 1979, the AMTE/PL and the United States Navy Experimental Diving Unit carried out very similar dives. In the British dive (AMTE/PL Dive 9) to 540 m (1771 ft), with compression at 5 m/min and six stages of 4 hours or more for a total of 3 days, 5 hours, there was marked nausea, tremors, dizziness, vomiting, and loss of appetite. Intention tremor and epigastric sensations persisted.

The United States Navy dive was to 549 m (1800 ft), with rates mostly of 30 ft/h (9 m/h) for a compression time of 3¾ days from 650 ft. Fatigue, dizziness, nausea, vomiting, aversion to food with 8% weight loss, stomach cramps, diarrhea, myoclonic jerking, and dyspnea were present. The condition of the divers deteriorated rather than improved with time at depth, but they were able to work at 100 watts in connection with respiratory studies (Spaur, 1979, personal communication).

These experiments showed that even at comparatively slow rates of compression, the increasing hydrostatic pressure produces severely limiting HPNS between 1400 and 1800 ft. This may, in fact, have been the result of still too-rapid rates of compression at the deeper depths; a more exponential rate of compression with slower rates at depth (e.g., 0.1 to 0.2 m/min) would have been more effective.

TRIMIX (NITROGEN-HELIUM-OXYGEN MIXTURE)

In the search for some method to ameliorate or prevent HPNS in deep divers, the use of trimix (a mixture of helium and oxygen with a small percentage of nitrogen) has received special attention. This use was based on the pressure reversal of narcosis theory reported in tadpoles[49] and mice.[50] In studies of the effects of raised pressures of the inert gases nitrogen, argon, and helium on model monolayer membranes, Bennett and colleagues[77] noted that the first two gases caused a fall in surface tension (expansion of the monolayer), whereas the helium caused a rise in surface tension (constriction). Inferring that the fall in surface tension was related to mechanisms of narcosis, and the rise to an HPNS mechanism, and coupling this observation with the pressure reversal theories, Bennett suggested that adding nitrogen to helium-oxygen mixtures might well result in no change in surface tension and thus no narcosis or HPNS.[77–79]

The technique was first used in humans at the F. G. Hall Laboratory at Duke Medical Center in 1974, when divers were compressed with either trimix or heliox to 1000 ft in the remarkably fast time of only 33 min using trimix 18 (that is, trimix with 18% nitrogen). Although trimix did prevent HPNS compared with the heliox (Fig. 11–4), two of four divers experienced euphoria from nitrogen narcosis.[78] Further studies were conducted in 1974, in which five divers were compressed exponentially with three brief holds or stages to 1000 ft, also in 33 min; with the nitrogen concentration reduced to 10%, no performance deterioration, narcosis, nausea, tremors, or EEG changes were detected (Fig. 11–5).[79] Confirmation of the value of trimix was later afforded by French workers in the so-called CORAZ comparative dives, which compared

the value of 9% or 4.5% nitrogen in a 4-hour compression to 1000 ft (305 m). The lower nitrogen concentration appeared best for ameliorating HPNS without narcosis.[80, 81]

In 1980, Norwegian workers also made some comparative dives to 300 m, with divers compressed in 4 hours 4 min using either trimix 10 or heliox.[82] Again, although trimix successfully ameliorated much of the HPNS, there was evidence of nitrogen narcosis.[82] The next year, a similar dive was made to 1640 ft (500 m) with, in one case, a heliox compression of 26 hours 45 min and, in the other, a trimix 10 compression of 41 hours 20 min. Both groups suffered HPNS signs and symptoms, and the trimix group experienced nitrogen narcosis too. The likely reason is that the compression profiles were still too fast in critical places and the nitrogen (at 10%) was too high.

Figure 11–4. Postural tremor of the hand in a subject exposed to 1000 ft (31 ata) either with oxygen-helium alone or with trimix (He-O_2 with 18% nitrogen) with the same compression time of 33 min. Without the nitrogen present, classic tremor may occur. With nitrogen added at 600 ft (19 ata) in the trimix, tremor is suppressed. On changing back to oxygen-helium during the decompression at 850 ft (26.6 ata), tremor returns. (From Bennett PB, Blenkarn GD, Roby J, et al: Suppression of the high pressure nervous syndrome in human deep dives by He-N_2-O_2. Undersea Biomed Res 1:221-237, 1974. ©1974 Undersea Medical Society, Inc.)

The Duke Atlantis dives from 1979 to 1982[58, 59, 83] were designed to determine the relationship between either 5% or 10% nitrogen and either fast or slow rates of compression. Very extensive scientific studies were performed and supported the view that a slow exponential compression rate with stages of about 38 to 40 hours total time to 450 m (1476 ft) with trimix 5 (i.e., 5% nitrogen in heliox) permits divers to arrive at such depths in a fit condition and able to work effectively. It was during these dives that the deepest human exposures to 686 m (2250 ft) were achieved with the divers in a remarkably good condition. These tests also indicated that at depths deeper than 300 or 400 m (984 to 1312 ft), it may not be possible to prevent a small (approximately 15%) decrement in sensitive psychologic test results (Fig. 11–6). However, the men appeared fit and able to work, and this decrement at 686 m was comparable to that at 300 to 400 m.

The French company COMEX initially used baboons[84, 85] to study the effects of trimix compressed beyond 300 m and subsequently reached 3281 ft (1000 m) before the EEG

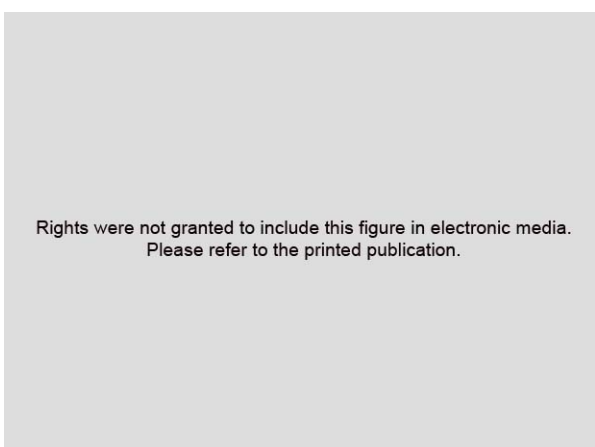

Figure 11–5. Tremor transducer measurements in three subjects compressed in 33 min to 1000 ft (31 ata) in trimix (He-O_2 with 10% nitrogen). No tremors of high-pressure nervous syndrome occur. (From Bennett PB, Roby J, Simon S, et al: Optimal use of nitrogen to suppress the high pressure nervous syndrome. Aviat Space Environ Med 46:37–40, 1975.)

revealed focal seizures. The investigators worked out a technique, tested later in human dives DRET 79/131, ENTEX V, and ENTEX VIII,[86–89] which involved compression at 0.5 m/min to 100 m (328 ft), followed by 0.4, 0.25, and 0.20 m/min for each further 100 m, and 0.14 m/min for the final compression to 450 m. Nitrogen was injected at each 100 m, where the divers were held for 150 min.

This 40-hour compression proved very successful at ameliorating HPNS.[90] Interestingly, using the same procedures in 1983 in ENTEX IX with heliox only, there seemed to be little difference whether the ENTEX dives used trimix or heliox. Indeed, further compression was made to 610 m (2001 ft) for 56 hours for in-water work, although general fitness was not as good as at shallower depths.

Confirmation of the efficacy of trimix in controlling HPNS for operational use was obtained from 18 simulated deep dives from 300 to 600 m (984 to 1968 ft) with 5% nitrogen in heliox and a compression profile based on Atlantis IV (Table 11–8) at the German underwater simulator at GKSS near Hamburg from 1983 to 1990.[91–93] In all, there were 2672 man-days of saturation with 994 man-days of work with few or no signs and symptoms of HPNS, such as nausea, vomiting, tremors, fatigue, sleep problems,

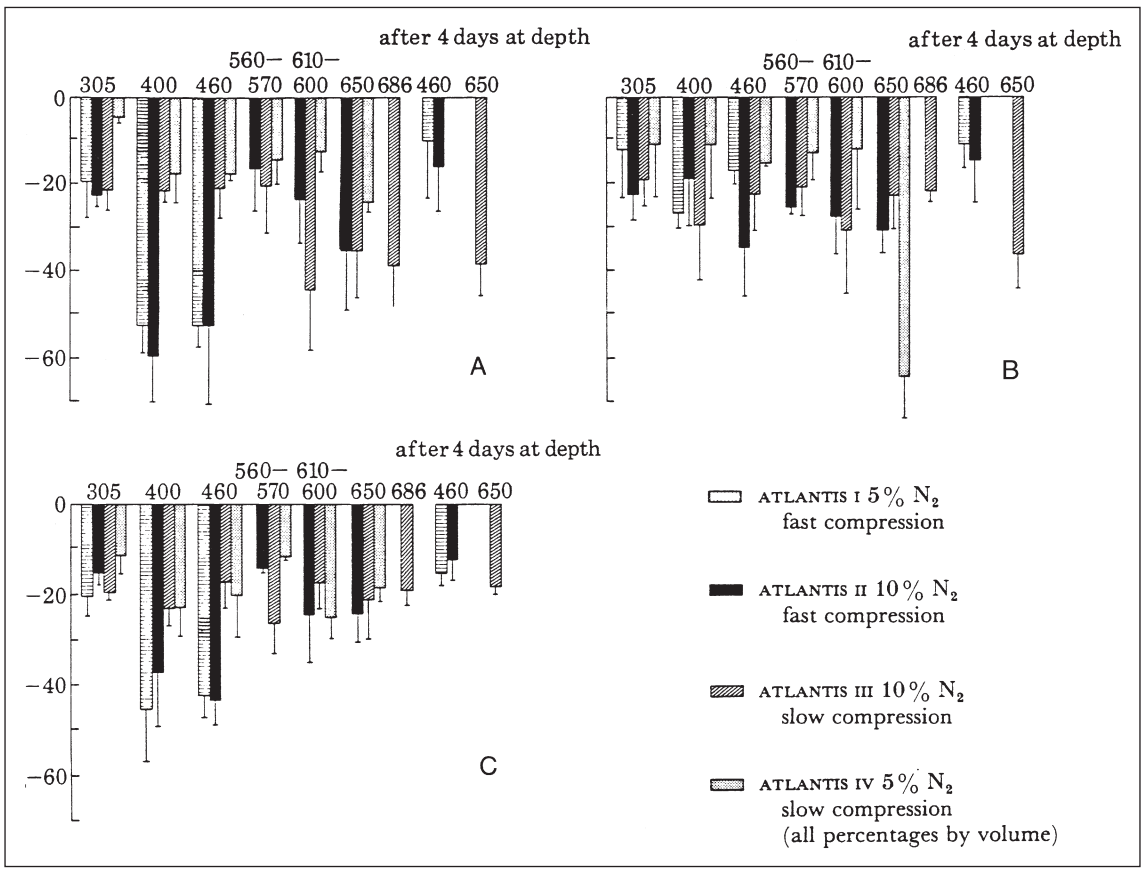

Figure 11–6. *A,* Comparison of the mean percentage decrement of three divers for each of the dives ATLANTIS I, II, III, and IV at the addition test requiring simple arithmetic. The large decrements at 400 and 600 m for ATLANTIS I and ATLANTIS II owing to fast compression are evident, as is the increasing decrement for dives deeper than 500 m during ATLANTIS II and ATLANTIS III and the considerable improvement in ATLANTIS IV. *B,* Comparison of the mean percentage decrement of three divers for each of the dives ATLANTIS, I, II, III, and IV at the ball-bearing test of fine motor dexterity. There is a tendency for the tests performed at lower nitrogen partial pressure to show a smaller decrement, except for ATLANTIS IV, in which, owing to the presence of visible tremors at 650 m, the test indicates decrements of 60% or more. *C,* Comparison of the mean percentage decrement of three divers for each of the dives ATLANTIS I, II, III, and IV at the screw plate or the hand tool test of motor skills. The large decrements at 400 and 460 m resulting from the quick compressions for ATLANTIS I and II do not occur with the slow compressions of ATLANTIS III and IV. Otherwise, the decrements are about 20% regardless of depth, rate of compression, or percentage of nitrogen. (From Bennett PB, McLeod M: Probing the limits of human deep diving. Philos Trans R Soc Lond 304:105–117, 1984.)

Table 11–8. Duke/GUSI Compression to 600 m with Trimix 5 (5% N_2, 0.5 ata O_2, Remainder Helium)

Travel 0–180 m	= 5 m/min (36 min)
Stop at 180 m	= 2 h
Travel 180–240 m	= 3 m/min (20 min)
Stop at 240 m	= 6 h
Travel 240–300 m	= 1.5 m/min (40 min)
Stop at 300 m	= 2 h
Travel 300–350 m	= 0.5 m/min (1 h 40 min)
Stop at 350 m	= 9 h
Travel 350–400 m	= 0.25 m/min (3 h 20 min)
Stop at 400 m	= 2 h
Travel 400–430 m	= 0.125 m/min (4 h)
Stop at 430 m	= 2 h
Travel 430–460 m	= 0.125 m/min (4 h)
Stop at 460 m	= 12 h
Travel 460–490 m	= 0.100 m/min (5 h)
Stop at 490 m	= 2 h
Travel 490–520 m	= 0.100 m/min (6 h 40 min)
Stop at 520 m	= 13 h
Travel 520–550 m	= 0.075 m/min (6 h 40 min)
Stop at 550 m	= 13 h
Travel 550–575 m	= 0.05 m/min (8 h 20 min)
Stop at 575 m	= 16 h
Travel 575–600 m	= 0.05 m/min (8 h 20 min)

From Bennett PB, Schafstall H, Schnegelsberg W, Vann R: An Analysis of fourteen successful trimix 5 deep saturation dives between 150–600 m. *In* Proceedings of the Ninth Symposium on Underwater Physiology and Hyperbaric Medicine. Kobe, Japan, 1986.

increase in brain theta activity, or undue performance decrement at least to 500 m.

Like the French research dives, this work will provide a method for compressing divers to greater depths with relative comfort and safety compared with earlier research dives; the work will also allow the determination of safe decompression schedules, albeit with very slow rates of only 1 m/h (3.3 ft/h) or less.[92]

TRIMIX (HYDROGEN-HELIUM-OXYGEN MIXTURE)

Since 1979, the French have carried out extensive research in the Hydra series of dives on the substitution of hydrogen for the nitrogen of trimix; these studies have been reviewed elsewhere.[2, 94] In 1989, this research culminated in an open-sea experiment called Hydra VIII, with a helium-hydrogen-oxygen mixture (49% hydrogen) at 500 m (1640 ft) with six excursion dives to 520 m (1706 ft) and 26 hours of work. Gardette[94] reports that hydrogen at the correct percentage is an effective substitute for nitrogen, having many of nitrogen's attributes for ameliorating signs and symptoms of HPNS with the additional factor of being a less dense gas, thereby helping breathing and permitting more comfortable and less fatiguing diving.

More recently, in 1992, the COMEX team extended the depth achieved by the 1981 Duke Atlantis team of three divers with a nitrogen-helium-oxygen mixture to 686 m (2250 ft) for 24 hours to one diver breathing trimix at 701 m (2300 ft) for 3 hours.

Whether this technology will ever be used operationally is a question because, today, deep exploration for oil uses more unmanned machines and redesigned well heads that require less use of divers.

Mechanisms of High-Pressure Nervous Syndrome

Bennett and Rostain have extensively reviewed the origins and mechanisms of HPNS.[95] A clear understanding of the mechanism remains elusive in spite of much neurophysiologic and neuropharmacologic research, particularly as regards nitrogen narcosis and neurotransmission.[96] More recent technology involves microdialysis in free-moving animals as reported earlier,[53] increased striatal dopamine,[97] increased serotonin,[98] disrupted γ-aminobutyric acid (GABA) neurotransmission,[99] and decreased 5-HT.[100] More recently, Daniels and colleagues[101] showed that, at the level of glutamate receptors, only *N*-methyl-D-aspartate (NMDA) type receptors are sensitive to pressure. Etzion and Grossman[102] have also reported that helium could act on ion channels. Research has moved a long way from whole minced brain or slices to studies in isolated specific regions of the brain. This has identified complexities wherein a given neurotransmitter might be raised in one brain region but lowered in another, whereas a milieu of whole brain tissue may show no change.

Clearly, much research is needed to elucidate the critical changes in various regions of the brain due to exposure to pressure.

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CHAPTER 12 Toxicity of Oxygen, Carbon Dioxide, and Carbon Monoxide

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OXYGEN

It is paradoxical that the same gas required to sustain life by preventing loss of consciousness and death from hypoxemia is, at sufficiently high pressure and duration of exposure, toxic to all living cells.[1–4] The rate of development of toxic effects is determined by the oxygen partial pressure (P_{O_2}) rather than by the percentage of oxygen in the inspired gas. The specific manifestations of oxygen poisoning that occur in humans or animals are determined by interactions between oxygen dose (with respect to both P_{O_2} and duration of exposure) and the relative susceptibilities of the exposed tissues. Although continued exposure to a toxic P_{O_2} level ultimately causes functional disruption and cellular damage in any organ, effects on the lungs, brain, and eyes are most prominent under practical conditions of exposure. This chapter describes these effects.

Biochemistry of Oxygen Toxicity

Gerschman[5,6] and Gilbert[7,8] were the first to propose that oxygen toxicity is caused by the production of free radical intermediates in excessive concentrations during exposure to increased oxygen pressures. The initial involvement of these agents is now well established, and several excellent reviews have summarized the literature on the biochemistry of oxygen free radicals.[9–13] Although exact mechanisms are not yet known, free radical intermediates—including superoxide anions, hydrogen peroxide, hydroperoxy and hydroxyl radicals, and singlet oxygen—are potentially toxic to cell membranes, enzymes, nucleic acids, and other cellular constituents. Along with a better understanding of oxygen free radicals has come a greater awareness of the dependence of vital biologic processes on cellular antioxidant defenses such as superoxide dismutase, catalase, and the glutathione system. It is now thought that in the absence of these defenses, the same oxygen pressures required to sustain life would cause lethal oxygen poisoning.

Pulmonary Oxygen Toxicity

Studies in monkeys[14–17] have shown that the pathologic response of the lungs to oxygen toxicity can be differentiated into two overlapping phases of progressive deterioration. The first is an acute exudative phase consisting of interstitial and alveolar edema, intraalveolar hemorrhage, fibrinous exudate, hyaline membrane swelling, destruction of capillary endothelial cells, and destruction of type I alveolar epithelial cells. The exudative phase merges into a subacute proliferative phase that is characterized by interstitial fibrosis, fibroblastic proliferation, hyperplasia of type II alveolar epithelial cells, and partial resolution of earlier acute changes. The relative prominence of the individual components in each phase is influenced by interactions of external variables, such as inspired P_{O_2} and the duration of exposure, with internal factors, such as species differences in pulmonary tissue reactivity and susceptibility to hyperoxic exposure.

The lungs of human patients who die after prolonged oxygen therapy demonstrate pathologic changes that are similar or identical to those caused by pulmonary oxygen toxicity in experimental animals.[18–21] Although such alterations are not specific to pulmonary oxygen poisoning, the clinical course of

these patients, in conjunction with the known susceptibility of humans to oxygen toxicity, leaves no doubt that the observed pathologic changes are caused by pulmonary oxygen toxicity. In experimental animals, and presumably also in humans, recovery from pulmonary oxygen intoxication is accompanied by complete resolution of changes typical of the acute exudative phase. However, when exposure to hyperoxia lasts long enough for prominent proliferative changes to occur, recovery from these pathologic effects is greatly delayed, and incomplete resolution may leave permanent residual scarring of the lungs.

Symptoms of pulmonary oxygen poisoning begin insidiously as a mild substernal irritation that becomes progressively more intense and widespread in parallel with increased coughing. In severe poisoning, symptoms appear to originate in the trachea and major bronchi and are characterized by a constant burning sensation, which is exacerbated by inspiration and is associated with uncontrollable coughing. The most severe symptoms are associated with dyspnea on exertion or even at rest. The onset of symptoms varies but usually occurs after about 12 to 16 hours of exposure at 1.0 ata,[22] 8 to 14 hours at 1.5 ata,[23] and 3 to 6 hours at 2.0 ata.[23,24] At oxygen pressures of 2.5 and 3.0 ata, pulmonary symptoms have an earlier onset but are less severe because exposure durations are limited by neurologic manifestations of oxygen poisoning.[23,25]

In humans during and after prolonged exposures to oxygen pressures of 1.0 ata or higher, changes in pulmonary function include decrements in inspiratory and expiratory lung volumes and flow rates, carbon monoxide diffusing capacity, and lung compliance.[2,4,22–28] Arterial oxygenation was maintained at rest during early reversible stages of pulmonary intoxication[23,24,28] but was detectably impaired during exercise after exposure for 48 to 60 hours at 1.0 ata[28] or for 16 to 19 hours at 1.5 ata.[23] Pulmonary mechanical function is impaired earlier and more severely than gas exchange function in normal humans exposed continuously to oxygen pressures ranging from 1.0 to 3.0 ata.[23–25,28]

Although the level of hyperoxia that can be tolerated indefinitely with no pulmonary effects cannot be identified with certainty, normal humans who have been exposed for periods ranging from 7 days at 0.55 ata[29] to 30 days at 0.3 ata[30,31] have shown no detectable manifestations of pulmonary intoxication. However, exposure for 24 hours at 0.75 ata causes pulmonary symptoms in association with a significant decrease in vital capacity,[22] and the rate of pulmonary intoxication increases progressively at higher oxygen pressures.[23,24] Nevertheless, the majority of current applications of hyperoxia in hyperbaric oxygen therapy and diving do not cause pulmonary symptoms or clinically significant functional deficits.[32,33]

Hyperbaric oxygenation may cause pulmonary symptoms in patients when used aggressively for serious conditions, such as severe decompression sickness or arterial gas embolism. Some degree of substernal discomfort is also frequently experienced by commercial divers who use intermittent hyperoxia to hasten inert gas elimination after unusually long or deep dives. When hyperbaric oxygenation is combined with saturation exposure in the treatment of refractory decompression sickness, it is not uncommon for the diving chamber attendants and the patient to experience pulmonary symptoms. In all of these situations, irreversible pulmonary intoxication can be avoided by careful monitoring of symptoms and appropriate alternation of hyperoxic and normoxic exposure periods.

Central Nervous System Oxygen Toxicity

Overt manifestations of central nervous system (CNS) oxygen poisoning include the diverse symptoms and signs listed in Table 12–1. These observations were made in divers who breathed oxygen at pressures of 3.0 ata or higher until they experienced neurologic effects. The studies were designed to develop reliable methods for detecting the onset of CNS oxygen poisoning before convulsions occurred.

Extensive investigation in hundreds of divers failed to identify a consistent preconvulsive index of CNS oxygen poisoning. Minor symptoms did not always precede the onset of convulsions, and even when a preconvulsive aura did occur, it was often followed so quickly by seizures that it was of little practical value. Electroencephalography also proved to be a poor indicator of incipient CNS intoxication because brain electrical activity was not altered consis-

Table 12–1. Signs and symptoms of central nervous system oxygen poisoning in normal men

Facial pallor	Respiratory changes
Sweating	Hiccoughs
Bradycardia	Air hunger
Palpitations	Inspiratory predominance
Depression	Diaphragmatic spasms
Apprehension	Nausea
Visual symptoms	Spasmodic vomiting
Dazzle	Fibrillation of lips
Constriction of visual field	Lip twitching
Tinnitus and auditory hallucinations	Twitching of cheek, nose, eyelids
Vertigo	Syncope
	Convulsions

Adapted from Donald KW: Oxygen poisoning in man, Br Med J 1:667–672, 712–717, 1947.

tently prior to seizure onset. More recent studies have confirmed that electroencephalographic alterations in humans occur only with initiation of the actual seizure.[34,35]

Repeated studies in animals and in humans[1,3,4,34–37] have established that oxygen convulsions are not inherently harmful. However, the conditions under which they occur may make them extremely hazardous. For example, convulsions in an unattended diver can lead to death by drowning. Similarly, convulsions are especially hazardous in patients with fractures, osseous nonunion, head injury, cardiac abnormality, or recent surgery.

CNS oxygen toxicity in association with hyperbaric oxygen therapy is rare. The reported incidence of convulsions is approximately 0.01% when care is taken to adjust for factors that are known to increase the risk of intoxication.[32,38]

EFFECTS OF EXERCISE ON CENTRAL NERVOUS SYSTEM OXYGEN TOLERANCE

Although mechanisms are not known, it is well established that exercise and underwater immersion, independently or together, accelerate the onset of oxygen convulsions and can precipitate their occurrence at oxygen pressures as low as 1.6 ata.[36,37,39,40] Oxygen convulsions are also accelerated by the presence of acute hypercapnia, whether it is induced by an increased inspired partial pressure of carbon dioxide (P_{CO_2}) level, increased breathing resistance, or narcotic depression of ventilation.[3] The adverse effects of acute hypercapnia are mediated by cerebral vasodilatation and delivery of a higher oxygen dose to the brain.[41]

The reduction of CNS oxygen tolerance by exercise may also be explained, at least in part, by the associated hypercapnia.[42] An increase in arterial P_{CO_2} during hyperoxic exercise was shown in men breathing 100% oxygen at 2.0 ata[43] and in divers breathing a mixture of 55% nitrogen/45% oxygen at 4.0 ata.[44] Incremental exercise by men breathing oxygen at 2.0 ata caused a nearly linear increase in arterial P_{CO_2} (Fig. 12–1).[42] An associated increase in cerebral blood flow was confirmed by measuring concurrent increments in middle cerebral arterial blood flow velocity.[45,46] Velocity changes in the middle cerebral artery had been calibrated earlier against concurrent changes in cerebral blood flow measured by ^{133}Xe clearance in normal men exposed to progressive hypercapnia.[47]

Physiologic responses to underwater immersion include improved venous return from the peripheral circulation, increased thoracic blood volume, increased cardiac output, and reduced lung compliance.[48,49] Based on the hypothesis that some combination of these responses might directly or indirectly increase arterial P_{CO_2}, brain blood flow, or both, the ventilatory, arterial P_{CO_2}, and cerebral circulatory responses to incremental exercise were measured in the same subjects under dry conditions and during immersion.[50,46] None of these physiologic responses to exercise was significantly altered by either head-out or total immersion. In a different group of subjects, ventilatory and cerebral circulatory responses to progressive hypercapnia were similarly unaffected by head-out or total immersion.[51,46] These studies do not provide a physiologic basis for the adverse effects of immersion on CNS oxygen tolerance. An alternative explanation is that the increased work of breathing associated with the use of an underwater breathing apparatus reduced the ventilatory response to exercise with concurrent increments in arterial P_{CO_2} and brain blood flow. When combined with the use of oxygen-enriched mixtures in a closed or semiclosed breathing circuit, such physiologic responses

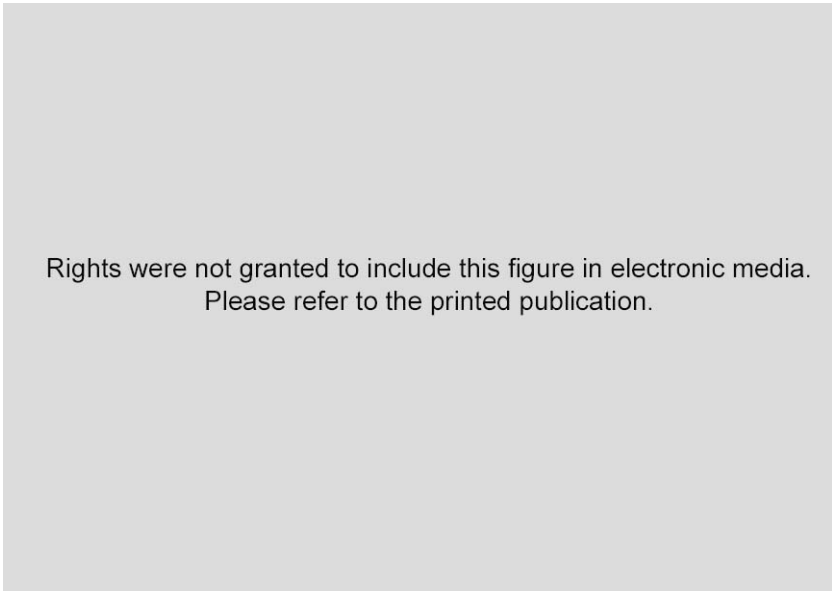

Figure 12–1. Relationship of arterial P_{CO_2} to the rate of CO_2 elimination (at standard temperature and pressure, dry [STPD]) during incremental exercise while breathing O_2 at 2.0 ata. Exercise was performed on a bicycle ergometer at the indicated workloads (in watts [W]) with the subject in a semirecumbent position. Average values for six subjects are shown. (From Clark JM, Gelfand R, Lambertsen CJ et al: Human tolerance and physiological responses to exercise while breathing oxygen at 2.0 ata. Aviat Space Environ Med 66:336–345, 1995.)

could deliver a toxic oxygen dose to the brain and cause convulsions.

Ocular Effects of Oxygen Toxicity

The effect of oxygen on vision is influenced by oxygen dose and other variables such as the age of the exposed person, the method of oxygen administration, and the presence of underlying conditions that may modify susceptibility to oxygen poisoning.[3,52]

RETROLENTAL FIBROPLASIA

Retrolental fibroplasia is a unique condition that may be induced by exposure of the premature infant to any elevation of arterial P_{O_2} above the normal range.[3,52] Risk factors include gestational age less than 30 weeks, birth weight less than 1500 g, and concurrent problems such as sepsis and intraventricular hemorrhage.[53,54] Initial constriction of the developing retinal vessels is followed by endothelial cell destruction and arrest of the retinal circulation at an incomplete stage of development.[3,52,53] The remaining endothelial cells later undergo a disorganized and profuse proliferation to produce a fibrous mass of vascular tissue that ultimately causes irreversible retinal detachment and permanent blindness. Vitamin E therapy is apparently effective in reducing the severity of retrolental fibroplasia.[54]

IRREVERSIBLE EFFECTS ON VISION

Animal studies involving extremely prolonged oxygen exposures have demonstrated severe pathologic effects, such as visual cell death, retinal detachment, and cytoid body formation.[52] In guinea pigs exposed to oxygen at 3.0 ata, histopathologic changes found in the corneal endothelium and lens epithelium, as well as in the retinal plexiform and inner nuclear layers, indicate that pathologic effects may be more severe when the entire eye is exposed to oxygen than when hyperoxygenation occurs only through the arterial circulation.[55]

Histologic studies of oxygen-induced ocular pathology have not been performed in humans. However, one patient who was exposed to 80% oxygen at 1.0 ata for 5 months as therapy for myasthenia gravis developed

nearly total blindness in association with marked constriction and "silver-wire" formation of the retinal arterioles.[56]

REVERSIBLE LOSS OF PERIPHERAL VISION IN HUMANS

Behnke and coworkers[57] first reported the nearly complete bilateral loss of peripheral vision, with only small islands of central vision, in a man who breathed oxygen at 3.0 ata for 3.5 hours. Recovery was essentially complete within 50 min after exposure. Other investigators[34,58] have also observed reversible losses of peripheral vision in similarly exposed subjects.

This phenomenon of reversible peripheral vision loss was recently studied more intensively with repeated measurements of visual fields and acuity in 18 subjects exposed to oxygen at 3.0 ata for up to 3.5 hours.[34] Loss of peripheral vision started at 2.5 to 3.0 hours of exposure and progressed to involve about 50% of the visual field on average, with individual losses as great as 90% at 3.5 hours of exposure. Central visual acuity was not significantly altered. Recovery of peripheral vision was essentially complete within 30 to 45 min after termination of exposure. The mechanisms for this progressive loss of peripheral vision and its rapid recovery are not known.

INDIVIDUAL PREDISPOSITION TO OXYGEN EFFECTS

A person who had recovered many years previously from retrobulbar neuritis in one eye showed an apparently increased susceptibility to visual loss during hyperoxic exposure.[59] While serving as a volunteer for an oxygen exposure at 2.0 ata, this subject experienced a progressive loss of vision in the previously affected eye over the last 2 hours of a 6-hour exposure. The visual field gradually expanded over the first 4 hours of recovery, but two paracentral scotomas remained and gradually cleared over a period of about 3 weeks. The observed visual disturbances appeared to involve two separate processes. One consisted of visual field constriction followed by relatively rapid reversal; the other appeared to represent recurrence of unilateral retrobulbar neuritis, with a much slower recovery.

PROGRESSIVE MYOPIA

Many of the patients who receive daily hyperbaric oxygen treatment for a variety of chronic disease states develop some degree of myopia that usually starts after 2 to 4 weeks of therapy and is progressive thereafter.[60,61] If the patient is initially hyperopic, the refractive error is normalized. At the conclusion of the treatment regimen, the myopia is nearly always completely reversed over a period of 3 to 6 weeks. Occasionally, complete reversal of myopia can require as long as 6 to 12 months.[61] Although the basis for the myopia is not known, elimination of other possible causes implicates a reversible change in lens shape or metabolism.[62] The incidence of myopia has not been determined in a large group of patients. However, there are indications that diabetic and elderly patients are more susceptible to this effect of hyperoxia.[61,62] It is also likely that the incidence of myopia will be higher in patients whose corneal surfaces are directly exposed to oxygen in a hood or monoplace chamber than in those who receive oxygen by face mask; this is because the oxygen tensions of aqueous and lens tissue will be much higher in the former group.[62]

Butler and colleagues[63] documented the occurrence of 1.5 diopters of myopia in a 48-year-old, closed-circuit, mixed-gas scuba diver who accumulated nearly 85 hours of diving at a constant P_{O_2} of 1.3 ata over a period of 21 days. The myopia reversed over a period of about 3 weeks. Subsequently, myopic shifts ranging from 0.5 to 1.5 diopters were found in each of three divers who performed about 45 hours of diving over 15 days at a constant P_{O_2} of 1.3 ata.

In a series of 25 patients, each of whom received a total of 150 to 850 1-hour exposures at 2.0 to 2.5 ata over a period of 2 to 19 months for refractory leg ulcers, cataracts developed in 7 out of 15 patients who had clear lens nuclei at the start of therapy. These cataracts persisted in five persons and were only partially reversible in two others after termination of the therapy series.[64] The lens changes were associated with myopia that was only partially reversible. Fortunately, most clinical conditions that respond

favorably to hyperbaric oxygenation do not require such long cumulative periods of oxygen exposure. However, cataracts have developed in at least one patient who received only 48 exposures over a period of 11 weeks.[65] Each exposure consisted of 90 min of oxygen breathing at 2.5 ata with two 5 min air breaks. The patient was a 49-year-old woman who was not diabetic or taking steroids. Formation of cataracts was associated with progressive myopia, which stabilized at 4 to 6 months after cessation of therapy and remained stable at 8 and 11 months after therapy.

Modification of Oxygen Tolerance

The rate of development of oxygen poisoning in animals and humans can be influenced by a variety of conditions, procedures, and drugs (Table 12–2). Factors that hasten the onset or increase the severity of toxic effects are listed on the left side of Table 12–2. Although none of these factors should be considered to be an absolute contraindication to the application of hyperbaric oxygenation, the presence of one or more of the factors—when part of a disease process or its therapy—should be regarded as an indication for caution.

Table 12–2. Factors that modify rate of development of oxygen poisoning

Hasten Onset or Increase Severity	Delay Onset or Decrease Severity
Adrenocortical hormones	Acclimatization to hypoxia
Carbon dioxide inhalation	Adrenergic blocking drugs
Dextroamphetamine	Antioxidants
Epinephrine	Chlorpromazine
Hyperthermia	γ-Aminobutyric acid
Insulin	Ganglionic blocking drugs
Norepinephrine	Glutathione
Paraquat	Hypothyroidism
Hyperthyroidism	Reserpine
Vitamin E deficiency	Starvation
	Succinate
	Trisaminomethane
	Intermittent exposure*
	Disulfiram*
	Hypothermia*
	Vitamin E*

*Potentially useful as protective agents.
Adapted from Clark JM, Lambertsen CJ: Pulmonary oxygen toxicity: A review. Pharmacol Rev 23:37–133, 1971.

Factors listed on the right side of Table 12–2 have been found to delay the onset or decrease the severity of overt manifestations of oxygen poisoning. Some are potentially useful as protective agents under appropriate conditions of oxygen exposure. Unfortunately, most have side effects or other limitations that preclude their practical use in humans. Furthermore, effective protection against the multiple and diverse effects of oxygen toxicity requires wide distribution of the protective agent throughout all body tissues as well as effective opposition to toxic effects of oxygen on a variety of enzymatic targets. The same agent may delay some toxic effects while hastening the onset of others. For example, disulfiram delays the onset of convulsions in animals exposed to oxygen at 4.0 ata,[66,67] but it enhances the progression of pulmonary intoxication at 1.0[68] or 2.0 ata.[69]

At present, the most effective and practical means for extending tolerance to oxygen in humans is to systematically alternate intervals of oxygen exposure with relatively brief normoxic intervals.[3,4] This phenomenon was initially observed by Soulie,[70] and its practical applications were first elaborated by Lambertsen[71]; its efficacy has been demonstrated in animals[46,72,73] and in humans.[46,74] Intermittent oxygen exposure delays the onset of toxic effects in all organs and tissues and has none of the limitations that are associated with pharmacologic protective agents. The basis for the superiority of this procedure as a means for extending oxygen tolerance resides in the periodic, sequential elevation and reduction of oxygen tension, rather than in the passage of a chemical agent across cellular membrane barriers.

CARBON DIOXIDE

Carbon dioxide is a product of oxidative metabolism and hence is not a toxin in the traditional sense. Intoxication results either from exposure to respiratory gases containing high concentrations of carbon dioxide or from retention of autogenous carbon dioxide because of inadequate ventilatory equipment or pathologic states (e.g., emphysema). In diving medicine, acute carbon dioxide intoxication can be caused by inade-

quate carbon dioxide elimination from closed spaces (e.g., diving bells, submersibles, underwater habitats, recompression chambers) or from closed or semiclosed underwater breathing equipment.[75,76] Carbon dioxide retention from voluntary hypoventilation while diving can result in headaches and may increase susceptibility to oxygen toxicity. This behavior is sometimes found in recreational divers who attempt to reduce gas consumption in open-circuit scuba.

Any physiologic or toxic action of carbon dioxide must be related to an increased partial pressure of molecular carbon dioxide or to an increased hydrogen ion concentration, or both.[76] Because molecular carbon dioxide crosses cell membranes freely to penetrate intracellular as well as extracellular fluid compartments, the potentially toxic actions of carbon dioxide and hydrogen ion are inseparable. In a similar manner, the toxic effects of carbon dioxide are also superimposed on and, to some extent, inseparable from fundamental physiologic influences that include the following:

- Stimulant actions of carbon dioxide on central and peripheral chemoreceptors that provide an important link in the regulation of internal acid-base homeostasis
- Relaxant effects of carbon dioxide on vascular smooth muscle that are involved in the regulation of brain circulation
- Excessive partial pressures (P_{CO_2}) that can depress the activity of the same neural structures that are stimulated by lower levels of P_{CO_2}
- Acidosis-inducing actions of carbon dioxide that influence a wide range of biochemical reactions on both sides of membrane and vascular barriers[75,76]

Acute Hypercapnia

Acute exposure to carbon dioxide at concentrations ranging from zero to greater than 20% at normal atmospheric pressure produces effects that range from barely detectable stimulation of ventilation to loss of consciousness and convulsions, depending on the level inspired (Table 12–3).[75,76] The ventilatory response to carbon dioxide administration is nearly linear over minute volumes of about 12 to 65 L/min for inspired levels of 4% to 10% and gradually levels off to approach 90 L/min for 30% inspired carbon dioxide.[75] The curve for cerebral blood flow resembles the ventilatory curve in monkeys and presumably also in humans, with a nearly linear increase over the arterial P_{CO_2} range of about 30 to 80 mm Hg.[77]

Exposure of humans to inspired carbon dioxide concentrations of 15% to 20% causes an abrupt and violent onset of respiratory distress that is accompanied by rapid loss of consciousness and neuromuscular spasms.[75,76] In therapeutic applications previously employed for neuropsychiatric disorders, exposure to inspired carbon dioxide levels of 20% to 30% in oxygen caused convulsions within 1 to 3 min.[75,76] Any accidental exposure to such a high carbon dioxide concentration would be extremely dangerous because just a single breath can cause mental incapacitation.[75] Electrocardiographic responses to similar levels of hypercapnia include tachycardia, nodal and ventricular premature contractions, inverted P waves, and increased amplitude of T waves.[78,79] In monkeys and dogs exposed to carbon dioxide concentrations of 30% to 40%, cardiac activity was sustained for many hours and remained stable when inspired P_{CO_2} was gradually reduced to zero.[80,81] However, when the dogs were abruptly moved to room air, most of the animals experienced ventricular fibrillation and died.[81] Presumably, the terminal arrhythmias were caused by a failure to allow sufficient time for normal cardiac excitability to be restored by reversal of ionic shifts induced by the prolonged and extreme hypercapnia.[76]

Elevation of inspired P_{CO_2} during exercise interferes with the elimination of metabolically produced carbon dioxide.[76] Under

Table 12–3. Signs and symptoms of acute hypercapnia in normal men

Percent Carbon Dioxide* (Sea-level equivalent)	Effect
0–4	No CNS derangement
4–6	Dyspnea, anxiety
6–10	Impaired mental capabilities
10–15	Severely impaired mental function
15–20	Loss of consciousness
>20	Uncoordinated muscular twitching, convulsions

*Biologic activity of a gas is determined by its partial pressure rather than its concentration. Hence, at depth the effect of an inspired gas becomes greater.

these conditions, a balance between the rates of carbon dioxide elimination and its production is restored by concurrent increments in the arterial P_{CO_2} and the rate of pulmonary ventilation.[82–85] Physically fit young men are able to achieve maximum levels of oxygen uptake ($\dot{V}_{O_2}$) during exposure to inspired P_{CO_2} levels up to 21 mm Hg[82,84] and can tolerate working at 80% of maximum $\dot{V}_{O_2}$ at an inspired P_{CO_2} of 40 mm Hg.[85]

Chronic Exposure to Hypercapnia

Chronic elevation of P_{CO_2} in all body fluids can occur in patients with pulmonary insufficiency,[86] in normal persons who are exposed to increased inspired P_{CO_2} levels for experimental purposes,[83,87] or as a potential consequence of inadequate carbon dioxide removal from a closed-system aerospace or undersea habitat.[76] Compensatory responses to sustained hypercapnia include renal,[88] acid-base,[89–91] respiratory,[86,87] and circulatory[92] adaptations. The kidney responds initially by increasing the tubular reabsorption of bicarbonate and later complements this with increased production of ammonia to enhance excretion of hydrogen ions.[88] Together, these processes augment both extracellular and intracellular concentrations of bicarbonate and other bases to bring hydrogen ion concentrations toward normal levels.[89–91] The acid-base alterations are associated with respiratory adjustments that are manifested in normal humans by a shift of the pulmonary ventilation–arterial P_{CO_2} response curve to higher P_{CO_2} levels with no change in the slope of the curve.[87] Studies in monkeys show an attenuation of cerebral blood flow responses to arterial P_{CO_2} elevation during exposure to chronic hypercapnia as manifested, in this case, by a reduction in the slope of the curve with no apparent change in the initial response threshold.[93]

Normal humans have been exposed to inspired P_{CO_2} levels of 30 mm Hg for up to 11 days and 21 mm Hg for 30 days with no pathologic or residual effects.[83,87] Ventilatory and acid-base adjustments that occurred during the first day of chronic hypercapnia were promptly reversed upon resumption of air breathing. The ability to perform heavy exercise is not impaired by 30 days of chronic exposure to an inspired P_{CO_2} of 21 mm Hg.

CARBON MONOXIDE

Carbon monoxide (CO) is released into the environment by incomplete combustion of carbonaceous materials. The sources of CO are plentiful, and with the exception of carbon dioxide, CO is the most abundant pollutant present in the lower atmosphere.[94] The toxic effects of CO result from its binding to heme-containing cellular proteins. For scuba divers, the typical source of CO poisoning is contaminated air from improperly directed compressor engine exhaust so that the CO is taken up in the air intake system. In addition to environmental sources, CO is also produced endogenously. It is a byproduct of heme catabolism and may account for perhaps 0.5% saturation of hemoglobin in venous blood.[94–96] This discussion focuses on exogenous sources of CO and the clinical impact of such exposure.

Uptake

Inhaled CO rapidly diffuses across the alveoli and binds to the heme porphyrin ring of hemoglobin in erythrocytes. The relative affinity of CO for hemoglobin averages 250 times greater than that of oxygen, with some variation among individuals.[97,98] The uptake is exponential,[99] with the rate depending on the percentage of inspired CO and oxygen, on the ventilatory rate, and on the duration of exposure to CO.[98–100] Binding to many heme-containing proteins can occur, in theory. The affinity of CO varies among heme-containing proteins because amino acid residues on the protein chains modify the binding pocket at the heme porphyrin ring. For example, in the β chain of hemoglobin A, the E7 histidine and E11 valine residues sterically interact with the heme-bound CO and push the ligand off the heme axis. This has a significant effect on the heme-CO bond and on the CO-combination rate. In mutant hemoglobin A chains, or hemoglobin molecules from other animals that do not have these types of steric hindrances, the CO combination rates are much higher.[101] Studies with synthetic iron porphyrin proteins have demonstrated that amino acids can impede ligand binding by presenting steric hindrances to CO.[102] Other more subtle variations in the heme binding pocket, the so-called *docking site,* also influence binding kinetics.

The dissociation rates for CO vary markedly among different heme proteins. These rates cannot be readily explained by steric hindrance but appear more likely to be related to alterations in the polarity within the heme pocket.[103] For example, the amino acid residues surrounding the heme in myoglobin modulate ligand binding affinity. The binding affinity of CO compared with O_2 is reduced approximately 50 times compared with the affinity for the free heme moiety. The difference in myoglobin is thought to be due to the characteristic geometry within the docking site, which impedes CO more so than O_2.[104]

Elimination

CO elimination also displays an exponential relationship.[105–107] The kinetics in any particular instance, however, are complex and appear to depend on the rate of ventilation, inspired P_{O_2}, and possibly the pattern of CO exposure (e.g., whether brief or prolonged, continuous or discontinuous).[99–110] The rate of elimination of CO from the body is most conveniently assessed by monitoring the blood carboxyhemoglobin (COHb) level. Historically, this measurement has also been used to assess the degree of CO exposure and, hence, the severity of poisoning. Clinicians have often attempted to calculate a maximum COHb level for a patient based on an assumed value for the half-life of COHb and an estimate of the time elapsed since a patient was removed from a contaminated environment. Because of physiologic uncertainties, the calculation is not accurate. Furthermore, there is little rationale for performing the task because the mortality and morbidity risks from CO have not been found to correlate with the COHb level.[110–115]

Mechanism of Carbon Monoxide Toxicity

The hypoxic effects of CO were described more than a century ago by Claude Bernard and John Haldane.[116,117] Despite a relatively high P_{O_2} in the vasculature, CO binds to hemoglobin because of its high affinity, which reduces the O_2-carrying capacity of hemoglobin.[118,119] COHb also increases the affinity of unbound hemoglobin for O_2, thus causing a leftward shift and a more hyperbolic shape in the oxyhemoglobin dissociation curve.[120] These later effects cause a lower tissue and intracellular P_{O_2} than would otherwise be expected for a given blood O_2 content. When this happens, the hemoglobin concentration and P_{O_2} of blood may be normal but the O_2 content of the blood is grossly reduced. A clinician must be aware of this fact and that chromatographic measurement of oxyhemoglobin does not adequately monitor oxygenation status. Values reported by pulse oximetry, which is commonly used for clinical monitoring, do not correlate with COHb levels, and oximetry can overestimate arterial oxygenation.[121]

Coburn[122] estimated that at any time approximately 10% to 15% of the total body burden of CO is bound to extravascular hemoproteins. There is little evidence that CO binding to extravascular hemoproteins has adverse effects on organ physiology, except in the case of cytochrome oxidase.[122,123] The affinity of CO for cytochrome *c* oxidase is 10 to 20 times less than that for O_2.[124] Despite this, evidence suggests that mitochondrial electron transport is perturbed by CO and that production of oxidizing species is increased.[125] One possible explanation for this paradox may be linked to cardiac dysfunction, which appears to be mediated by CO-induced hypoxic stress from COHb. Transient cardiac dysfunction impairs tissue perfusion, causing additional hypoxia,[126–129] which increases the probability that CO binds to cytochrome *c* oxidase. A relatively protracted disturbance of oxidative metabolism may occur because the rate of CO dissociation is relatively slow.[124]

CO may also disturb cellular homeostasis because it increases the steady-state concentration of the free radical, nitric oxide (•NO), in and around both platelets and endothelial cells.[130–132] Electron paramagnetic resonance spectroscopy has provided direct evidence that exposure to CO increases the concentration of •NO in lung and brain.[133,134] CO does not increase the activity of nitric oxide synthase in platelets or endothelial cells, nor does CO increase nitric oxide synthase protein concentration in tissues of CO-exposed rats at a time when they exhibit elevated •NO levels.[131–135] In fact, CO partially inhibits nitric oxide synthase activity in rats exposed to 3000 ppm that have COHb levels of approximately 45%.[130] It appears that CO increases the steady-state level of unbound •NO because CO competes for intracellular sites that normally would bind •NO. Toxic

effects on cells are due to the liberated •NO that is available to undergo reactions with superoxide anion to yield the potent oxidizing and nitrating agent peroxynitrite. Peroxynitrite and its protonated form, peroxynitrous acid, can oxidize proteins, membrane phospholipids, and DNA as well as hydroxylate and nitrate aromatic compounds. Peroxynitrite can inactivate mitochondrial enzymes, impair electron transport, and, in some circumstances, accelerate production of reduced oxygen species.[136] A "footprint" of peroxynitrite in vivo is the nitrated form of the amino acid tyrosine. Nitrotyrosine has been found in a perivascular distribution in the brains, lungs, and aorta of experimental animals exposed to CO.[133–135]

Pathophysiology of Carbon Monoxide Toxicity

CO enters the body via the lungs, and the pulmonary parenchyma may be injured by direct interactions without need for delivery of CO by blood-borne hemoglobin. Elsewhere in the body, CO is delivered by hemoglobin. Capillary leakage of macromolecules from the lung and systemic vasculature has been documented in human beings or experimental animals who have been exposed to relatively low CO concentrations for extended periods.[137,138] The capillary leak in skeletal muscle and lungs is mediated by •NO.[134,135]

As COHb levels rise, cerebral vessels dilate[139] and both coronary blood flow and capillary density increase.[140–142] These are acute responses to CO, and as exposure continues, central respiratory depression arises, possibly resulting from cerebral hypoxia.[143] Animal and human reports have described cardiac effects including a myriad of arrhythmias, as well as pathologic changes that include myocardial hemorrhages, degeneration of muscle fibers, leukocyte infiltration, mural thrombi, and multifocal myocardial necrosis.[143–147] Acute mortality from CO is most often due to ventricular arrhythmias caused by hypoxic stress.[144,148–151] There are indications that myocardial impairment may begin at the relatively low COHb level of approximately 20%.

Animals that do not die acutely, instead showing neurologic deterioration over the days subsequent to poisoning, appear to have suffered a combined hypoxic and ischemic insult during the acute exposure. Studies indicate that during the hypoxic stress caused by an acutely elevated COHb level, cerebral hypoperfusion also occurs. Whereas cerebral blood flow normally increases acutely because of CO exposure, with continued exposure this response is thwarted by cardiac dysfunction.[127–129,149] These vascular events, coupled with changes in the endothelium of cerebral vessels, cause leukocyte sequestration, which mediates subsequent injuries by causing oxidative changes.[152,153]

Several investigations have suggested an association between CO-induced neurotoxicity and that caused by excitatory amino acids.[154,155] Although this issue is currently under investigation, in some studies excitotoxicity has been linked to elevations of intracellular calcium, •NO, and superoxide anion.[156,157] Three types of receptors are activated by excitatory amino acids: N-methyl-D aspartic acid (NMDA), D-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid, and kainic acid.[156] Agents that inhibit NMDA receptor activation attenuate CO-mediated delayed neuronal degeneration of pyramidal cells in the hippocampus and cochlear ganglion cells.[158,159] Monoamine neurotransmitters such as norepinephrine and dopamine are elevated after CO exposure, and enzymatic breakdown as well as auto-oxidation will generate reactive O_2 species.[160,161] These agents appear to contribute to oxidative stress after CO poisoning because free radical production in the brain can be diminished by inhibiting monoamine oxidase B, an enzyme located in microglial cells.[162–164] Activated microglia can also mediate neuronal injury by generating •NO-derived oxidants.[165] Microglia can attack oligodendroglia and have been associated with demyelination processes.[166] In experimental CO poisoning, autoimmunity toward myelin proteins appears responsible for delayed neurologic deterioration.[167]

Neuropathology may include neuronal death in the cortex, hippocampus, substantia nigra, and globus pallidus.[168] One of the most common abnormalities is demyelination of cerebral cortex, which occurs in a perivascular distribution along with evidence of a breach in the blood–brain barrier.[168–170] Several neuroimaging techniques have been used to show abnormalities in blood flow and the perivasculature.[171–176] Acute vascular and perivascular changes also have been found in the brains of experimental animals.[111,133,152,177] Moreover, the variability

observed in lesions found in the cerebral white matter and globus pallidus of animals has been correlated with the fall in local blood flow and metabolic acidosis.[128,149,177] Clinical and experimental findings suggest that the effects of CO are global and that variations in the clinical manifestations of poisoning arise because brain regions respond differently to these stresses. Acute neurologic compromise may be due to direct hypoxic stress. The syndrome of delayed neurologic sequelae appears to result from a cascade of events involving oxidative stress and inflammatory responses, as described earlier.

Clinical Findings

Among the earliest complaints associated with a rising COHb level are headache and nausea.[178] With COHb levels less than 10%, there are findings of diminished visual evoked responses, diminished visual brightness discrimination, impaired manual dexterity and precision, and subtle auditory dysfunction; these findings may be more valuable experimentally (to suggest a tissue-level CO insult) than clinically.[179–183]

Symptoms of CO poisoning are generally more severe with higher COHb levels. These include dizziness, vomiting, weakness, confusion, disorientation, visual disturbances, and unconsciousness. Cardiac rhythm disturbances include sinus tachycardia, atrial flutter and fibrillation, premature ventricular contractions, ventricular tachycardia, and ventricular fibrillation. Myocardial infarction can occur, even among patients with normal coronary vessels.[184,185] Pulmonary edema in association with CO is relatively rare; it typically results from congestive heart failure. CO-associated pulmonary edema is more common among persons with concomitant smoke inhalation, in whom the condition may be related to inhalation of a toxic combustion product.[186] Skeletal muscle necrosis can occur and, with it, acute renal failure. Other rare complications include pancreatitis and hepatocellular injuries. *Although COHb levels are loosely associated with symptoms, there is no direct correlation between COHb levels and the severity of symptoms or the risk of mortality or morbidity.* The cherry-red coloration of the skin is often absent; it is an extraordinarily rare occurrence except among the deceased.[187–189]

Table 12–4. Delayed neurologic sequelae of carbon monoxide poisoning

Choreoathetosis	Hemiplegia
Cortical blindness	Hysteria
Dementia	Mutism
Depression	Parkinsonism
Disorientation	Peripheral neuropathy
Epilepsy	Personality changes
Gait disturbances	Speech disturbances
Hearing impairment	Urinary/fecal incontinence

Approximately 30% to 40% of CO victims die before hospitalization.[189] Of those hospitalized, approximately 2% die, 10% recover partially, and 23% to 47% suffer what are described as delayed neurologic sequelae.[147,189,190] The clinical features of delayed sequelae are outlined in Table 12–4.[114,169,191–194] Clinical observations and historical data currently provide the most useful guidelines for stratifying the risk of morbidity and mortality. Risks appear to be greater among patients with previous cardiovascular disease, patients who are older than 60 years, and patients who have suffered an interval of unconsciousness during CO exposure. Although the presence or absence of these characteristics does not always correlate with clinical outcome, the duration of coma or unconsciousness is roughly proportional to morbidity, particularly the development of delayed neurologic deterioration.[111,114,189]

Using traditional neuroimaging techniques, such as computed tomography and magnetic resonance imaging, brain lesions have been sporadically detected in severely poisoned CO patients. Unilateral and bilateral areas of low density in the globus pallidus and white matter are sometimes visible, even in scans performed within the first few hours after poisoning.[171,197–200] The primary shortcoming with these imaging techniques is their limited sensitivity; hence, neuroimaging has not yet provided a reliable method for assessing the severity of CO poisoning. Some recent findings with state-of-the-art neuroimaging techniques have correlated with the clinical improvement in case reports, whereas others may show abnormalities when no clinical changes are noted.[201–203] Recently, more sophisticated neuroimaging techniques have been used to detect abnormalities in some patients who exhibited only subtle neurologic impairments. Abnormalities in resting cerebral

blood flow[174,175] and in cerebral vasoactivity to carbon dioxide[173] have been detected by single-photon emission computed tomography. Other detected changes have suggested that CO causes a disturbance in coupling between neuronal O_2 demand and blood flow. DeReuck and colleagues[172] examined seven patients between 5 and 7 days after CO poisoning using positron emission tomography with $^{15}O_2$. The authors found a global increase in cerebral O_2 extraction along with regional areas of diminished blood flow, especially in the frontal and temporal lobes. Although these observations underscore the vascular nature of CO-mediated neuropathology, they do not assist with clinical assessments of patients. Objective parameters that reliably assess the severity of poisoning are lacking.

Treatment

Initial attention must be focused on restoring or maintaining vital functions. Preservation of a patent airway, ventilation, oxygenation, and adequate perfusion establish the foundation for proper actions in serious CO poisoning. In addition to general supportive care, supplemental O_2 inhalation is a cornerstone in the treatment of CO poisoning. COHb dissociation is hastened by an elevation in the P_{O_2} of inspired gas. Hyperbaric oxygen hastens dissociation beyond a rate achievable by breathing pure O_2 at sea-level pressure[109,204] and therefore has been used to treat severe CO poisoning for several decades. Work with animals indicates that hyperbaric oxygen prevents neurologic injury by inhibiting the adherence of circulating leukocytes to the vascular endothelium.[153,205] Hyperbaric oxygen inhibits the function of adherence molecules called β_2-integrins, and inhibition of neutrophil adherence appears to be the mechanism of action of hyperbaric oxygen in several disorders.[205–210] Hyperbaric oxygen has been shown to also inhibit neutrophil β_2-integrin adhesion in humans.[211]

A number of trials have evaluated the efficacy of hyperbaric oxygen in clinical CO poisoning. In a large retrospective study, Goulon and coworkers demonstrated that hyperbaric oxygen (2 ata), when administered within 6 hours of poisoning, dramatically reduced mortality and morbidity.[212] In a retrospective evaluation of 100 consecutive patients, Gorman and coworkers[213] found that hyperbaric oxygen treatment (2.8 ata) reduced the incidence of neuropsychiatric sequelae; however, they reported that two or more treatments were necessary for an improved outcome.

Six prospective, randomized trials have evaluated hyperbaric oxygen therapy for CO poisoning. Raphael and coworkers[214] randomized patients without loss of consciousness to receive either normobaric O_2 or hyperbaric oxygen (2 ata). In a second arm of the study, patients with loss of consciousness were randomized to receive either one or two sessions of hyperbaric oxygen. Neither arm of the study showed any detectable difference in outcome. Residual neuropsychological effects, determined by a self-assessment questionnaire, were high in all four groups. The study was criticized because of the examination method and because there were long treatment delays.[215,216]

The second prospective trial randomized 26 noncomatose patients with acute CO poisoning to receive normobaric oxygen or hyperbaric oxygen (2.5 ata).[217] Outcome measures included symptoms, electroencephalographic results, and cerebral blood flow responses to acetazolamide administration. The hyperbaric treatment group showed a significant benefit at 3 weeks, but limitations of this trial included small size, inadequate allocation concealment, and the use of surrogate outcome measures.

In the third randomized trial, 60 patients with mild CO poisoning, excluding those with history of unconsciousness or cardiac compromise, received either hyperbaric oxygen (2.8 ata) or normobaric O_2 until symptoms were relieved.[218] Patients were followed with serial neuropsychological testing to detect development of delayed neuropsychologic sequelae. Sequelae developed in 7 of 30 patients (23%) treated with normobaric O_2 and in no patients treated with HBO_2 ($P < .05$). Among those experiencing delayed neuropsychologic sequelae, impairment persisted for an average of 6 weeks and often interfered with normal daily activities. The trial was stopped early because of a treatment advantage in the hyperbaric treatment group.

Scheinkestel and colleagues[219] performed the fourth trial, randomizing 191 CO-poisoned patients of different severity to either continuous high-flow normobaric O_2 for 3 or 6 days or to daily hyperbaric oxygen (3.0 ata

for 60 min) with intervening high-flow O_2 for 3 or 6 days. The CO poisonings were suicide attempts in 69% of cases, and one half of these patients had also ingested alcohol or other drugs. Neuropsychological testing was performed after treatment and 1 month later. Among seven tests performed, only one (the Rey auditory learning verbal test) was significantly different between the groups, in favor of normobaric O_2 treatment. The groups showed no differences 1 month later. Several authors have discussed flaws in the design and execution of this investigation, and they make meaningful conclusions impossible.[220,221] For example:

- The presence of depression and psychoactive substances in many of the patients may have confounded the results of neuropsychological testing.
- Neither treatment protocol followed standard treatment recommendations, and both regimens were potentially toxic.
- Less than half of patients completed the follow-up examination at 1 month.

A recent double-blind randomized trial by Weaver and colleagues was stopped after the third interim analysis, following enrollment of 152 patients, because the hyperbaric oxygen therapy group was found to have a reduced incidence of neurologic sequelae after acute CO poisoning.[223] Among 76 patients treated with hyperbaric oxygen (three treatments at 3.0 ata) 25% had cognitive sequelae compared with 46.1% (35 of 76 patients) in the normobaric O_2-treated group (P = .01). Post hoc subgroup analysis showed that hyperbaric oxygen reduced cognitive sequelae in patients with any of the following: loss of consciousness, COHb greater than 25%, age greater than 50 years, base excess less than –2 mEq/L. In patients with none of these criteria, hyperbaric oxygen therapy did not improve outcome.

An additional randomized trial has been reported, though only in abstract form. Mathieu and associates performed a multicenter study.[222] At an interim analysis, 575 patients had been randomized to one hyperbaric oxygen treatment (90 min at 2.5 ata) versus 12 hours of normobaric O_2. After 3 months, neurologic sequelae were significantly less in the hyperbaric treatment group (8.7%) than in the normobaric group. The difference lessened by 6 months and disappeared by 12 months.

In summary, clinical investigations have provided strong evidence to support the use of hyperbaric oxygen in CO poisoning. Among the investigations that failed to find a benefit to hyperbaric oxygen, poisoned patients were often treated more than 6 hours after their rescue. According to current information, patients with signs of serious intoxication should be referred for hyperbaric treatment.

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CHAPTER 13 Hypothermia

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For tropical, low-altitude, air-breathing homeothermic animals like humans, most of our planet is a hostile place. It is wet, dark, greater than 1 atmosphere of pressure, and cold. The average depth of the ocean floor is 4000 m, the average seabed temperature is 3°C (37.4°F), and, because light penetrates seawater to a depth of only 50 to 80 m, most of the planet remains in constant darkness. The surface temperatures of the majority of the oceans, seas, and inland waters remain below 35°C (95°F), the temperature at which a human can remain immersed at rest indefinitely without becoming hypothermic. So it is, that the combination of cold water and raised atmospheric pressure present a stress that is as great as any the body must endure.

The limited ability of humans to adapt to pressure and cold has meant that they have largely relied on intellect and consequent technology to spread out from their equatorial origins and explore and inhabit the rest of the planet, including the aquatic environment. In so doing, humans have endeavored, through clothing and equipment, to maintain the same conditions around and within the body as applied in their ancestral environment. It is a measure of human ingenuity, curiosity, and adaptability that some of us have swum nearly 60 m under ice with the aid of only a swimming costume and goggles,[1] traveled almost 50 miles underwater in 24 hours using self-contained underwater breathing apparatus (SCUBA) gear,[2] and constructed a normobaric bathysphere that has enabled dives to depths of close to 11,000 m.[3]

Although such achievements can be inspirational, occupational and sports divers are presented each day with a combination of physical and environmental demands that could push them to their physiologic limit. The consequences can then range from impaired physical and mental performance to death. Over the years, cold and hypothermia have been implicated in a number of diving accidents. This chapter attempts to outline some of the risks associated with cold and their amelioration.

HEAT BALANCE

To maintain deep body temperature, the thermoregulatory system of the body must balance heat lost by that gained. In normobaric air, heat may be lost via the physical processes of conduction, convection, radiation, and evaporation from the body surface as well as by convection and evaporation from the largest surface area in contact with the environment, the airways. Alternatively, heat may be gained by the body through conduction, convection, radiation, condensation, and metabolism.

For a naked person in still air, all four routes of heat exchange with the environment are available. In water, radiant and evaporative heat losses from the body surface are minimal. Consequently, for the totally submerged diver, conduction and convection are the major routes of heat exchange from the skin. Despite the reduction in the pathways available for heat loss, a naked human cools 4 to 5 times faster in water than in air at the same temperature.

The difference in the cooling and warming power of air and water lie in their physical characteristics. Although they are both "fluids," air and water have different physical properties: the thermal conductivity of water is about 23 times that of air, and water has a specific heat per unit volume that is approximately 3500 times that of air. The average combined heat transfer coefficient for convection and conduction under several exposure conditions is shown in Figure 13–1.

For divers immersed in cold water at great depths, the calculation of heat loss from the

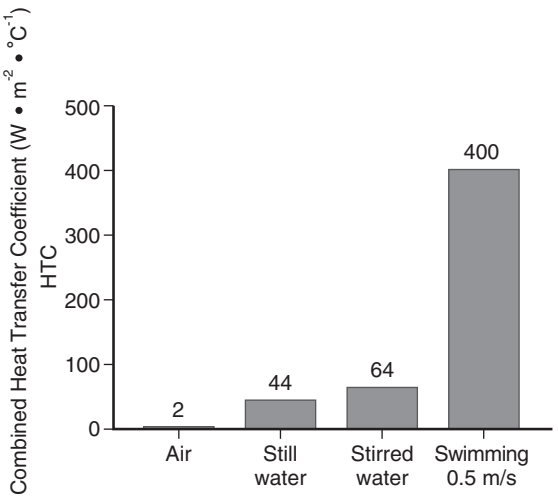

Figure 13–1. Heat transfer coefficient (HTC) for several exposure conditions.

skin is identical to that of an immersed human at sea level. However, heat loss from the respiratory tract increases with pressure, and the temperature regarded as thermoneutral, a thermoneutral temperature is one in which the body can maintain a constant deep body temperature with changes in only peripheral blood flow (i.e., no shivering or sweating), alters and perceived as comfortable. At 1 ata, thermoneutral temperature for a naked person breathing air is about 26°C (79°F) in air, 35°C (95°F) at rest in water, and 26°C (79°F) when exercising at 3 to 3.4 times resting metabolic rate. When the air environment is changed to 80% He and 20% O_2, the thermoneutral zone is elevated and significantly narrowed; thermal comfort requires ambient temperatures of approximately 30°C (86°F). Furthermore, as pressure increases, the ambient temperature required for thermal comfort also increases.

At 1 ata, respiratory heat loss averages 8% to 10% of heat production. This figure is a constant for aerobic exercise because of the linear relationship between minute ventilation and oxygen consumption. In the cold, the proportion of total body heat lost via the respiratory tract can increase because of a reduction in the heat lost by other routes (as a result of vasoconstriction) and increased ventilation to support shivering.

The major cause of respiratory heat loss at 1 ata is evaporation of water from the respiratory tract. Evaporation increases as the inspired air gets drier. At depth, because of the density of the gases breathed, convection becomes the major route of heat loss, and the amount of heat lost depends on the quantity, density, and specific heat of these gases. A diver at 30 ata breathing gas at a temperature of 4°C (39°F) can lose all metabolic heat via the respiratory tract.

TEMPERATURE REGULATION

The body senses temperature via a range of cold- and warm-sensitive receptors. These are located throughout the body but, as much for ease of comprehension as anything else, the receptors have been grouped into those in the central nervous system and those in the skin, with the former containing mostly warm receptors and the latter mostly cold receptors.

The afferent information from peripheral receptors is integrated in the hypothalamus: 60% to 70% of the temperature-sensitive hypothalamic neurons are affected by changes in skin or spinal temperature. The static thermosensitivity of the hypothalamus has been recognized for more than half a century. Thermosensitive neurons have been identified within the anterior, preoptic, posterior, lateral, and dorsal areas of the hypothalamus. Roughly 30% of preoptic neurons are intrinsically responsive to warming and operate in the hyperthermic and hypothermic range; others (~10%) respond to cooling. Hypothalamic sensitivity to cold appears to depend on synaptic input from both the inhibitory postsynaptic potentials of warm-sensitive neurons and the excitatory postsynaptic potentials of temperature-insensitive neurons. Autonomic responses seem to be elicited from the preoptic area, and behavioral responses involve the posterior hypothalamus and cortex. The posterior hypothalamus also plays an important role in the initiation of shivering.

The temperature response of hypothalamic neurons can be altered by a number of nonthermal factors such as glucose concentration and osmolality. Silva and Boulant[4] showed that a large number of preoptic warm-sensitive neurons are also sensitive to osmolality and that many preoptic cold-sensitive neurons are also sensitive to glucose concentration. Although still speculative, these characteristics may be the neu-

rophysiologic correlates of the thermoregulatory impairment that occurs when acute heat stress is accompanied by dehydration or acute cold stress by hypoglycemia.

The mechanism by which body temperature is controlled remains a matter of debate, and several, sometimes contradictory, models have been developed to explain the central processing involved in temperature regulation. Many of these models include a "set point" against which afferent thermal data are compared and appropriate effector responses initiated. Although still popular and a useful conceptual aid, the set point remains hypothetical. Indeed, such a mechanism is not necessary to explain how the thermoregulatory system works.[5]

The thermoregulatory system employs several systems of the body in an attempt to counter and reverse alterations in body temperature. These include the somatic and autonomic nervous systems, the endocrine system, and the skeletal musculature. The primary autonomic effector responses to cold are vasoconstriction and shivering. However, none of these responses reduces respiratory heat loss, a potentially major route of heat loss for divers.

With regard to the maintenance of thermal balance, the effectiveness of the autonomic thermoregulatory responses is dwarfed by that of the behavioral thermoregulatory responses, such as donning clothing and constructing and heating buildings.

RESPONSES TO IMMERSION

Primarily because of the high density of water and the variation in hydrostatic pressure over the immersed body, head-out immersion in thermoneutral water (35°C [95°F]) and, to a lesser extent, upright submersion can produce significant physiologic alterations. During head-out immersion, a negative transthoracic pressure of about 2 kPa is established; as a consequence, the lung is at a relative negative pressure with respect to the rest of the body, and the immersed person is, in effect, negative-pressure breathing.

Although the hydrostatic pressures involved are small in absolute terms, they exceed venous pressure. As a consequence, immediately following head-out upright immersion, there is a cephalad redistribution of blood that can increase central blood volume by up to 700 mL. This is associated with enhanced diastolic filling, a raised right atrial pressure, and a 32% to 66% increase in cardiac output, due entirely to enhanced atrial filling and increased stroke volume.

The shift in blood volume is sensed as hypervolemia by the body, which responds with diuresis, natriuresis, and kaliuresis. Diuresis occurs within 1 to 2 hours, and natriuresis peaks by 4 to 5 hours of immersion. The head-out immersion of fully hydrated, sodium-replete persons can result in a 300% increase in sodium excretion and free water clearance; urine output can reach 350 mL/h.

From a practical viewpoint, there is little evidence to suggest that these changes cause any respiratory or circulatory embarrassment in a fit person, but they may cause problems for someone with a failing heart.

Problems are more likely during rescue. Soon after head-out immersion, the body adapts to its new environment: blood volume, stroke volume, heart rate, and cardiac output have been adjusted to accommodate the higher-density environment. Although blood volume has been reduced, cardiac output is supported by the hydrostatic assistance to venous return. During rescue following protracted immersion in warm water or relatively shorter immersions in cold water (when hypothermia may be present), the sudden loss of the hydrostatic support to the circulation and reintroduction of the full effects of gravity can precipitate a collapse in arterial blood pressure. This can result in unconsciousness or cardiac arrest.

Responses to Cold Immersion

The intense generalized vasoconstriction evoked by immersion in cold water in most body areas, excepting the head, can augment both the redistribution of blood and the responses that occur upon immersion. For example, urine production can reach 0.75 mL/min in the first hour of cold-water immersion. During a dive, urination can significantly reduce the insulation provided by otherwise dry underclothing beneath a diving suit.

Diuresis continues in the cold because of the suppression of antidiuretic hormone, the secretion of atrial natriuretic hormone, and, with more profound cooling, reduced renal

Table 13–1. Stages of hypothermia described by Golden and Hervey (1981)

Stage	Immersion Condition
I	Initial immersion (0–3 min)
II	Short-term immersion (3–30 min)
III	Long-term immersion (30+ min)
IV	Postimmersion (circumrescue collapse)

Data from Golden FS, Hervey GR: The "after-drop" and death after rescue from immersion in cold water. *In* Adam J (ed): Hypothermia Ashore and Afloat. Proceedings of the Third International Action for Disaster Conference. Aberdeen, Aberdeen University Press, 1981.

tubular reabsorption of water and decreased sensitivity of the tubules to antidiuretic hormone. This loss of body water is not prevented by the prior ingestion of glycerol to enhance plasma volume retention, and plasma volume can be reduced by 18% during prolonged dives.

In 1981, Golden and Hervey[6] outlined four stages of immersion associated with particular risk for the naked or conventionally clothed person (Table 13–1). The first three stages are associated with cooling of the skin, superficial tissues, and deep body tissues, respectively. Well-insulated and -designed diving suits ameliorate or delay these responses (see the later discussion on Protection). However, persons wearing poorly insulated, ill-fitting, or damaged garments will be affected.

INITIAL RESPONSES

For those unused to diving in cold water, sudden reductions in skin temperature initiate a set of cardiorespiratory responses collectively known as the *cold shock response.* Even the exposure of naked hands to very cold water (<10°C [<50°F]) may elicit this response in susceptible persons.

During immersion in rough water or submersion, the initial gasp and uncontrollable hyperventilation of the cold shock response can act as precursors to drowning. Novice divers in cold water may panic as their breathing equipment fails to meet the demand placed on it by hyperventilation. This can lead them to think that there is something wrong with their air supply.

Intense vasoconstriction and increased cardiac output combine to place a sudden load on the heart and vasculature. Mean systolic/diastolic blood pressures have been reported to increase from 17.3/10.1 kPa (130/76 mm Hg) at rest to 23.3/12.4 kPa (175/93 mm Hg) after 1 min of ice-water showering. Such responses pose a risk for those with hypertension and cardiovascular disease.

Cardiac arrhythmias, often ventricular in origin, are common on head-out immersion in cold water. Increased excitatory input to the heart in the presence of raised circulating levels of catecholamines is partly responsible for these abnormalities. Electrical disturbances in heart rhythm, particularly supraventricular arrhythmias, are much more common during submersion immediately following breath holding. These disturbances may explain some of the sudden deaths seen on immersion, for which it is difficult to attribute cause at autopsy (see Chapter 25).

As alluded to in the previous paragraph, sudden cooling of the skin on cold immersion stimulates sympatho-adrenal activity, resulting in the secretion of noradrenaline, adrenaline, and cortisol within the first minutes of immersion. Plasma noradrenaline concentration can increase 180% from baseline levels within 2 min. Alterations in the circulating levels of these hormones have implications for substrate utilization (see the discussion of long-term responses).

The cold shock response is now largely regarded as the most dangerous of the responses associated with cold immersion.

SHORT-TERM RESPONSES

The next tissues to cool are the superficial nerves and musculature. Tissues in the extremities are particularly susceptible. The rate of conduction and amplitude of action potentials is slowed with cooling; synaptic transmission is also slowed. For example, the conduction velocity of the ulnar nerve is reduced by 15 m/s per 10°C (18°F) fall in local temperature. Low muscle temperature impairs several chemical and physical processes at the cellular level, and muscle function is impaired when its temperature falls below 27°C (81°F). This can happen in the forearm after just 20 min of immersion in water at 12°C (54°F).

These changes in neuromuscular function can contribute to a reduction in work capacity. In extreme cases, cooling of peripheral

Table 13–2. Functional effects of lowering of deep body temperature

Deep Body Temperature	Effects
35°C [95°F]	Confusion, disorientation, introversion
34°C [93°F]	Amnesia
33°C [91°F]	Cardiac arrhythmias
33–30°C [91–86°F]	Clouding of consciousness
30°C [86°F]	Loss of consciousness
28°C [82°F]	Ventricular fibrillation
25°C [77°F]	Death

motor and sensory nerves leads to severe dysfunction, preventing simple manual tasks that require strength and dexterity such as adjusting gas valves. Such incapacitation can also result in an inadvertent increase in dive duration by increasing the time taken to complete a task.

LONG-TERM RESPONSES: HYPOTHERMIA

All of the changes noted earlier can occur before an immersion victim becomes hypothermic (deep body temperature < 35°C [95°F]). This takes at least 30 min for a conventionally clothed person and possibly up to 2.5 hours for someone wearing a 5 mm wet suit in water at 10°C (50°F). However, with time, a person immersed in water below thermoneutral temperatures becomes hypothermic. The onset of hypothermia can be insidious, and awareness of the early signs and symptoms is essential for its prevention, recognition, and treatment. The effects of progressive hypothermia are listed in Table 13–2. The deep body temperatures are only approximations; individual variation is great. Depending on conditions and whether the airway is not properly protected, death may occur earlier than predicted on the basis of hypothermia alone because of drowning following impairment of consciousness.

There is also great variation in the rate at which different persons cool, even in the same clothing. The underlying causes of this variability include the following:

- Inherent differences in the sensitivity of the metabolic response
- Age
- Gender
- Morphology
- Fitness
- Illness and injury
- Motion illness
- Nitrogen narcosis
- Nutritional state
- Blood alcohol concentration
- Blood sugar concentration
- Ambient carbon dioxide levels
- Ambient oxygen levels
- Previous exposure to cold (cold habituation)
- Environmental pressure

These nonthermal factors can influence the rate of heat exchange with the environment as well as both the sensitivity and the threshold for the initiation of the shivering response.[5]

For example, age, hypoglycemia, and cold habituation all delay the onset of shivering, reduce its sensitivity, and result in faster rates of fall of deep body temperature in a cold environment. In the case of cold habituation and hypoglycemia, these faster rates are coincident with an increase in thermal comfort. This alteration in subjective perception of the thermal state of the body effectively disables the behavioral thermoregulatory system and can be potentially hazardous for divers. It may also contribute to the occurrence of insidious hypothermia.

Drugs are contraindicated for diving, primarily because of their deleterious impact on cognitive function. Many drugs also have a detrimental influence on temperature regulation. Alcohol (75 mL in 200 mL water) does not appear to alter the average fall in rectal temperature over a 30 min immersion in water at 15°C (59°F) compared with controls. However, alcohol does impair thermal perception. Similarly, despite increasing the metabolic rate to a higher level than in control subjects, marijuana (0.739 g) smoked immediately before immersion does not alter the average rectal temperature response over a 60 min immersion in water at 28°C (82.4°F). In contrast, the available literature suggests that the prolonged use of "ecstasy" (MDMA) damages 5-HT nerve terminals in the central nervous system and delays both the onset and the magnitude of the thermoregulatory effector mechanisms. As a consequence, the thermoneutral zone is widened and hypothermia and hyperthermia occur more easily.

Cold exposure in humans is known to increase the oxidation of both carbohydrates and free fatty acids. Cold exposure also enhances the rate of disappearance of plasma glucose, the primary source of

carbohydrate for metabolism in the cold. Compared with normothermic conditions, acute cold stress has been reported to increase the following:

- Free fatty acid mobilization and turnover
- Lipid oxidation
- Glycerol concentrations
- Skeletal muscle glucose uptake

The more rapid uptake of carbohydrate in the cold occurs in the presence of a lowered level of circulating insulin, and the mobilization of free fatty acids may be due to noradrenaline released by the sympathetic nervous system rather than catecholamines from the adrenal medulla.

When body temperature can be maintained by shivering (e.g., relatively warm water, good thermal protection, lost bell), survival time can depend on shivering endurance, which itself is thought to be determined by the time taken for blood glucose to fall to approximately 2.5 mmol/L. Shivering endurance is extremely difficult to predict. The most pessimistic estimations give a time to the cessation of shivering due to hypoglycemia of around 10 hours.

Circumrescue Collapse

Approximately 17% of immersion deaths occur during, or immediately following, rescue. One of the major causes of postimmersion death is drowning, but it is unlikely to be the explanation for the classic anecdotal description of sudden loss of consciousness during, or immediately following, rescue. Originally it was thought that the continued fall in deep body (rectal) temperature that follows immersion, the "afterdrop," was responsible for these sudden collapses and deaths. The afterdrop was thought to be caused by a "convective" mechanism in which cooled blood returning from the extremities lowered the temperature of the deep tissues. This theory provided the rationale for rewarming hypothermic casualties with their limbs out of the hot (40°C [104°F]) water. However, although deep body temperature may continue to fall if those rescued are not adequately insulated or are incapable of generating sufficient metabolic heat, the available evidence suggests that significant blood flow does not return to the extremities of very cold persons until deep body temperature is rising and the central cold vasoconstrictor drive is reduced.

An afterdrop has also been described in moderately cool persons (deep body temperature approximately 34°C [95°F]) who exercise following cold exposure. However, such a fall in deep body temperature is somewhat artificial and not in accord with the classic description of the afterdrop in the context of postrescue death.

Alternative experimental work has suggested that the afterdrop is a conductive rather than convective (mass flow) phenomenon and is most apparent when deep body temperature is measured at sites where local temperature is primarily determined by conductive heat exchange. The rectum, but not the heart, is such a site. Thus, sudden death and unconsciousness during and immediately following rescue are more probably cardiovascular in origin, e.g., the collapse of blood pressure noted earlier. Hypothermia is not an essential ingredient of this phenomenon, although impairment of baroreceptor activity, as a result of cooling, may be a contributory factor, as is hypovolemia. This explanation accounts for the anecdotal descriptions of rescue collapse in persons rescued in tropical waters.

One practical way of reducing the risk of rescue collapse is to remove casualties from the water in a horizontal posture; this helps to maintain venous return and cardiac output. These considerations apply equally to the rescue of survivors who have been adrift in life-saving craft for some time. The rejection of the afterdrop as the cause of death postimmersion means that the limbs can be included in any rewarming procedure, if necessary, but more importantly this rejection focuses management attention on the cardiovascular threat rather than a hypothetical thermal one.

With regard to the postimmersion risk of decompression sickness, it has been concluded that different steady-state levels of shell temperature may not cause alarming elevations in bubble scores. However, a rapid elevation in tissue temperature, such as that caused by hot showering after a cold dive, may increase off-gassing before adequate peripheral blood flow has returned. The resulting increase in bubble formation may make decompression sickness more likely.

Cold Injuries

Cold injuries are another group of pathophysiologic responses to immersion in cold water that are worthy of note. Human tissue freezes at –0.55°C, and exposure to subzero temperatures before, and especially after, a dive can present a significant thermal stress. In addition, because seawater freezes at about –1.9°C, it is theoretically possible for the poorly protected extremities of divers to become frostbitten in the sea. However, just a bit of forethought should prevent this condition.

Nonfreezing cold injury (NFCI) occurs when tissue temperatures remain below approximately 17°C (63°F) for a protracted period, particularly when cooling is coupled with other conditions that can cause circulatory stasis. Classically, the condition occurs in the feet ("immersion foot" or "trench foot"). It is thought that exposure to a tissue temperature less than 5°C (41°F) for more than 30 to 45 min produces the conditions required for injury. At higher ambient temperatures, the exposure time required to produce injury becomes proportionately longer, but little information is available to define the risk of injury more precisely.

On presentation, there is usually a history of a digit, hand, or foot being very cold, ischemic, numb, and pain-free for a long period of exposure. It is usually on rewarming that the initial indications of injury present: the injured part becomes very painful as the circulation returns and a reactive hyperemia is usually present, lasting from days to 4 weeks. At this stage, the peripheral pulses are full and bounding, but capillary refilling is slow (capillary stasis). The affected part is edematous, red, hot, and dry, with some anesthesia and severe paresthesia persisting for some weeks after the part resumes a normal appearance.

Residual sequelae can include cold sensitization (prolonged/sensitive cold vasoconstrictor response), hyperhidrosis (sweating), and, in severe cases, persistent pain. As a consequence, those who have been injured are at greater risk for subsequent injury. The majority of those suffering from NFCI are likely to be symptomatic 6 months after the time of injury, and 10% suffer symptoms 5 years after injury. A smaller percentage continue to be symptomatic for the rest of their lives.

The pathogenesis of NFCI is unclear but appears to involve prolonged cooling, ischemia, hypoxia in nerves, or the liberation of reactive oxygen compounds during reperfusion. The threshold for injury is more easily achieved if persons are dehydrated. All of these factors can coexist during diving. The pathology of NFCI is also obscure. Injury to unmyelinated nerve fibers would be most consistent with the clinical symptoms. However, the most recent experimental evidence suggests that myelinated fibers sustain the greatest damage. The most common observation with regard to the circulation is cold-induced endothelial injury.

NFCI may be prevented by limiting exposure to cold and maintaining adequate peripheral blood flow. This may be difficult in some diving scenarios. During scientific studies at the Institute of Naval Medicine in the United Kingdom, the medical withdrawal criteria used to avoid NFCI during experiments in the cold is a local temperature of 8°C for 15 min or 6°C at any time. During long duration exposure to cold, subjects are withdrawn and slowly rewarmed if any skin temperature falls below 15°C (59°F) for more than 12 hours. Dehydration is avoided whenever possible.

Summary of the Effects of Cold

From the foregoing, it can be concluded that cold has more of an indirect than a direct effect on diver survivability and tends to be a contributory factor rather than the sole cause of a diving fatality. Cold shock can cause panic, which may result in a novice diver removing the mouthpiece and drowning. Cooling of the superficial tissues may impair neuromuscular function and result in difficulties in performing even relatively simple manipulative or life-saving tasks. Delays in completing tasks may extend time at depth, increasing cooling and gas saturation. This in turn can necessitate longer decompression times, which may increase overall body cooling.

Cold-induced vasoconstriction increases the risk of NFCI, which although not life-threatening may have life-long consequences. Vasoconstriction may also limit the removal of inert gas from tissue compartments during decompression, especially during rapid

rewarming following a cold dive. This increases the likelihood of bubble formation and the threat of decompression sickness. It may also increase decompression time and with it the overall level of body cooling.

The impairment in mental performance, which occurs relatively early during hypothermia, makes errors of commission or omission more likely. In addition, the feeling of intense cold can tempt divers to foreshorten their decompression time and take a chance in surfacing prematurely. Thus, apart from the increased solubility of gas with lower tissue temperatures, the likelihood of a diver suffering from decompression sickness increases for other reasons in cold water.

On arrival at the surface, a cold diver can experience considerable difficulty in returning to the diving vessel. Therefore, plans for cold-water diving, in addition to warning divers of the potential problems already noted, should include arrangements for the recovery of those incapacitated by cold.

PROTECTION

Some protection against the deleterious effects of cold can be obtained by physiologic or technologic (behavioral) means. From the physiologic perspective, a good level of fitness, proper nutrition, and hydration enables the thermoregulatory system to perform optimally. Humans habituate to cold. As few as five 2 min immersions in water at 10°C can reduce the cold shock response by 20% to 40% for up to 14 months. Repeated immersions also result in an attenuation of the metabolic response and increased thermal comfort in the cold. Circumstances determine whether or not these alterations are beneficial (e.g., enhance performance on fine motor tasks) or hazardous (e.g., increase the likelihood of insidious hypothermia developing).

For most divers, protective clothing represents the pinnacle of their technologically based protection. This most commonly comes in the form of "wet" or "dry" suits. Wet suits are normally constructed from closed-cell neoprene that is 3 to 6 mm thick. This provides the necessary insulation by trapping air within its cellular structure.

Wet suits do not have waterproof seals but are designed to allow as little water to enter between the suit and the skin as possible. Sizing is therefore important with such suits. A good-fitting wet suit allows only a little water to seep between the skin and the suit, where it will be warmed by the body and become part of the boundary layer. A poorly fitting or damaged wet suit allows water to flush in and out of the skin–suit interface during movement. This disturbs the boundary layer, increases body heat loss (forced convection), and negates the insulation provided by the neoprene. During exercise, such flushing can halve the effective insulation provided by a suit.

Dry suits can be uninsulated or insulated. Uninsulated suits, as their name implies, have little inherent insulation. They are usually constructed from a trilaminate waterproof material composed of a synthetic membrane bonded to two layers of nylon facing fabric. Often, a "breathable," waterproof membrane is bonded to the nylon facing fabrics. These suits usually incorporate waterproof zippers and wrist and neck/face seals. Uninsulated suits are designed to keep the insulation of the clothing worn beneath them dry. This is often normal clothing, which is adversely affected by water leakage into the suit or urination. As little as 500 mL of water can produce a 30% reduction in the insulation provided by such clothing. To reduce the impact of leakage, a "thermal liner" can be worn with the suit. These liners are usually constructed from water-resistant (hydrophobic) materials, which retain more of their insulation when wet than everyday clothing.

Alternatively, an insulated immersion suit may be used. These suits are made from material with inherent insulation (such as closed-cell neoprene) that is unaffected by wetting. Provided, therefore, that water is not flushing in and out of an insulated suit, it is much less affected by leakage than an uninsulated suit worn over everyday clothing. Unlike wet suits, insulated suits are faced with waterproofed nylon fabric and incorporate watertight zippers and seals. Some suits provide inherent insulation by inflation of a chamber in the suit with air or carbon dioxide. Others include "radiant barriers" made of aluminium and plastic. Studies have failed to demonstrate any thermal benefit of this approach.

With regard to the relative performance of these different types of immersion suit, average cooling rates, immersed "Clo", and survival times have been measured and estimated. One Clo is equivalent to the amount of

Table 13–3. Insulation characteristics of different diving dress

Dress	Cooling Rate	Immersed Clo*	Survival Time
Ordinary lightweight clothes	2.3°C/h (4.2°F/h)	0.06	65 min
Uninsulated dry suit over lightweight clothes	1.1°C/h (2°F/h)	0.33	4 h
Full wet suit (4.8 mm closed cell foam covering extremities and trunk)	0.5°C/h (0.9°F/h)	0.5	10 h
Insulated dry suit(4.8 mm closed cell foam covering extremities and trunk)	0.3°C/h (0.5°F/h)	0.7	15 h

*Clo is a unit of clothing insulation. 1 Clo = 1.55 togs = 0.155° C • m^2 • W^{-1} = the insulation provided in air by a standard business suit.

insulation required to keep a seated subject comfortable in air at a temperature of 21°C (70°F), relative humidity of less 50%, with 0.1 m/s air movement. 1 Clo = 0.155°C/m^2/W–1. When measured in water, it is called *immersed Clo*. Approximate values for a thin adult male wearing different clothing assemblies during immersion in calm water at about 12°C (54°F) are listed in Table 13–3.

For the reasons mentioned earlier, these estimations should be regarded only as rough approximations; the estimation of survival time remains more an art than a science.

Although adequate thermal protection may be established for one condition with the careful choice of diving suit, it is unlikely to be appropriate for the entire duration of a dive. For example, a wet-suited diver in cold water may require only 1 immersed Clo of insulation while conducting heavy work but more than 1.5 Clo when resting.

With submersion, hydrostatic pressure compresses clothing, displaces air, and consequently reduces insulation. This, along with the differing physical properties of air and water, explains why clothing assemblies have lower Clo values in water (immersed Clo) than air. For 3 mm and 5 mm neoprene suits, the compression exerted at 100 msw can reduce the effective insulation to one third of its value at 1 ata.

Constant-volume dry suits allow a much wider manipulation of the thermal insulation layer. The choice of undergarments should be dictated by the conditions of the dive (e.g., depth, water temperature, and level of activity). It has long been thought that additional insulation may be obtained from the gas used to inflate the dry-suit compartment, although this requires the diver to wear a separate cylinder containing the suit inflation gas. Inflating a dry suit with a gas such as argon rather than air can theoretically increase the microclimate insulation by as much as 50%. However, this procedure has been reported to have no impact on skin temperature profiles during 60 min inactive prone dives in cold water.

The "passive" systems described are unable to provide the levels of insulation required during deep dives of long duration. In such dives, thermal balance can be achieved only with active heating systems. Wet suits flooded with hot water are one such system. The suits are supplied with hot water (35° to 40°C [95° to 104°F]) via an umbilical cord. The diver can regulate the volume of water flowing through the suit in an effort to maintain a thermally neutral and comfortable microclimate. At depths of 180 msw, the diver requires 1.2 to 1.3 kW of energy in order to achieve thermoneutrality.

The system requires that divers accurately perceive their thermal status and environment. It has been suggested that thermal perception is impaired at depth. However, a recent survey of the thermal status of saturation divers during operational dives in the North Sea found no evidence of such impairment. In this study, the hot-water suits used by the commercial divers enabled them to maintain deep body temperature while working for up to 6 hours at depths to 160 msw in water temperatures ranging from 4° to 6°C (39° to 43°F).

In deep diving, heat loss may be reduced significantly by minimizing heat loss from the respiratory tract. Heating the inspired gas is mandatory at depths greater than 100 msw (330 fsw). This is currently achieved using the same hot water as is used to flood the hot-water suits. Heating the gas improves breathing comfort and minimizes respiratory heat loss by convection. Because the breathing gas normally remains dry, this method does not address the evaporative component of respiratory heat loss.

Head Protection

As a result of the comparative paucity of cold constrictor fibers in the blood vessels of the scalp, heat loss from the head in the cold can account for over half the resting metabolic heat production. In extreme cold, it should be remembered that the face is part of the head.

Hand and Foot Protection

The hands and feet do not produce much heat. Thus, the temperature of these areas depends primarily on the heat delivered by blood flow. Because of this, and their high surface area, the hands and feet cool quickly when blood flow is reduced because of cold vasoconstriction. The best way to protect these regions, in addition to insulating them, is to keep the body warm and thereby help maintain their blood (heat) supply.

Gloves and footwear insulate but do not maintain local blood flow if deep body temperature is falling. Hence, even with gloves and specialist footwear, if deep body temperature falls, hand and foot temperature will also fall, albeit more slowly with extra insulation. The hands are particularly difficult to protect. As the thickness of gloves increases, so does the surface area for heat loss; each individual finger represents a cylinder with a high surface area. This is why mitts are preferred to gloves.

Heated gloves are becoming more widely available. Although these gloves may raise skin temperature locally, thereby slowing the rate of cooling and improving thermal comfort, a danger lies in the fact that they do not increase blood flow in the hands of persons with lowered mean skin and deep body temperatures. Thus, the gloves are heating relatively bloodless tissue; this may be harmful. For the same reason, such heating does not affect whole body thermal balance.

TREATMENT

Much has been written on the clinical signs and symptoms of hypothermia over the last 50 years. These accounts vary between case reports of patients who have been cooled rapidly (e.g., accidental victims of cold-water immersion) or slowly (e.g., as a consequence of thermoregulatory impairment due to intoxication, medication, age, or a variety of pathologic conditions).[7] There is a third group, that is, patients who have been deliberately cooled as a precursor to an elective surgical or therapeutic procedure. Although all three groups of patients may have a lowered body temperature as a common denominator, the similarity generally ends there, at least in regard to the pathophysiologic changes encountered in the mild to moderate levels of hypothermia (35° to 30°C [95° to 86°F]).

The difference in clinical presentation lies largely in the metabolic response to body cooling. In general, in the first group, body temperature is defended vigorously by shivering; in the other two groups, shivering can be attenuated or absent. The consequence of this to the clinical presentation is that vigorous shivering is accompanied by tachycardia, an increase in blood pressure, and a respiratory response necessary to meet the metabolic demands of the shivering muscles. Many reviews of this topic appear to be unaware of this distinction, and one reads of the gradual decline in cardiac and respiratory function in divers as body temperature falls. Additionally, many reviews report (without reference) that oxygen consumption declines as body temperature falls below 35°C (95°F).[8] Closer scrutiny of that paper reveals that it was oxygen extraction that fell rather than consumption, probably as a consequence of an increasing right-to-left pulmonary shunt (the data were obtained from immobilized patients who had been undergoing therapeutic hypothermia for many hours). In fact, in patients suffering acute accidental hypothermia, shivering may still be quite vigorous at a deep body temperature of 30°C (86°F) or below, although at such low temperatures shivering is usually replaced by muscle rigidity. Nevertheless, the demand for oxygen remains high, and with it the associated cardiorespiratory responses, until muscle activity becomes impaired by cold-induced inhibition of enzymatic activity and membrane depolarization.

Thus, references to clinical signs in divers associated with a decline in metabolism are largely irrelevant and may be misleading. In acute hypothermia, such a decline may not occur unless the diver is in the terminal stages of hypothermia, with a body temperature in the region of 24° to 26°C (76° to 79°F) or has some other coexisting condition such

as near drowning. The "cold diver" generally presents with shivering, the intensity of which depends on the influence of nonthermal factors and the level of cooling. Because both protagonist and antagonist muscles are contracting simultaneously during shivering, the limbs are stiff with a marked reduction in manipulative capability. The cold skin usually has a cyanotic hue or, if the water is below 12°C (54°F), may be pink with capillary stasis. If the body temperature is below 34°C (93°F), some degree of mental impairment accompanied by behavioral changes may be evident. Slurred speech, however, may occur at higher temperatures because of shivering in the masseter muscles.

At a body temperature in the region of 30°C (86°F), the pupils become dilated and the diver displays a lack of interest in the surroundings. At this time, atrial fibrillation may be present with some atrioventricular block. Shortly after, if body temperature continues to fall, consciousness is lost. Death from cardiac arrest is likely at approximately 24° to 26°C (76° to 79°F), although ventricular fibrillation is often encountered in the region of 28°C (82°F) if the heart is irritated. At this temperature, the conduction differential between Purkinje tissue and myocardial muscle is lost and thus reentry becomes possible. This is also the likely explanation for the difficulty encountered in conducting successful cardioversion below this temperature.

Much of the foregoing information, however, is really only of academic interest because, if the deep body temperature of a diver has fallen to the region of 30°C (86°F), drowning will likely occur before rescue is achieved. Of course, there are exceptions, such as the diver in a lost bell or a lost surface diver who is eventually rescued after many hours in relatively calm water (in rough water [> sea state 5/6] with moderate to large waves, 6 to 13 feet [183 to 396 cm], drowning from wave splash will very likely occur before hypothermia becomes significant).

In general, therefore, the requirements for treating a "hypothermic" diver are simply those of taking care of one who is very cold but not significantly hypothermic (35° to 31°C [95° to 88°F]). If the level of hypothermia is significant, the chances are that cold water has been aspirated and the priority for treatment will be for near-drowning rather than the more benign condition of hypothermia.

Recovery and Treatment

The principles involved in the treatment of the cold diver are relatively simple. If the diver is alive at the time of rescue, then obviously the prevailing body temperature is not fatal. Thus, it is important to ensure that further cooling does not occur after recovery. Death, if it occurs during or immediately following rescue, is likely to result from some maneuver or intervention that results in cardiovascular instability. At temperatures in the region of or below 33°C (91°F), impairment of physiologic autoregulatory control makes such an event highly possible. Consequently, divers on the threshold of unconsciousness should be removed from the water with care unless their airway is under threat of aspiration, in which case recovery should be through any means feasible and as speedy as possible. Otherwise, divers should be rescued in a manner least likely to impose stress on the cardiovascular system, i.e., they should be recovered in a horizontal attitude to reduce the risk of orthostatic hypotension. This advice also applies to any persons who have been immersed for some time, regardless of water or body temperature, because orthostatic hypotension resulting from hypovolemia is also possible at this time for the reasons given earlier.

After recovery, while the person is maintained in the horizontal attitude, routine ABC assessment should be carried out and appropriate corrective measures (in accordance with standard life support protocols) taken, if necessary. Additional measures, ones not normally detailed in life support protocols, are required to prevent further heat loss. Such loss may occur through:

- Conduction to a cold surface on which the victim is lying
- Convection, especially if the person is exposed to air movement
- Radiation, though only to a small degree
- Most importantly, evaporation

Each liter of water that evaporates extracts about 2428 kJ (580 kcal) of heat. Evaporation can therefore result in significant levels of cooling and contribute to a further decline in body temperature, possibly into a lethal zone. Therefore, once the essential ABC checks have been completed, measures must be taken to insulate the victim against these possible sources of continued heat loss.

Shelter the victim from air movement and adverse ambient conditions before carefully removing all wet clothing. Quickly dry the skin (including the head) by toweling and place the person in a sleeping bag or between blankets. Make sure the head is also covered, leaving the face exposed. If a bunk or similar item is not available, then ensure that the victim is adequately insulated from the ground. If oxygen is available, then provide 100% O_2 through a rebreathing mask. Before finalizing the insulating cocoon, check the lungs for signs of aspiration (rhonchi or rales). Once these measures are complete, check for signs of decompression sickness or any other coexisting problems. Continue to monitor vital signs while the victim slowly and spontaneously rewarms. Provided the victim can swallow easily, warm sweet drinks (preferably noncaffeinated) may be given ad lib.

Fully conscious divers suffering from cold without other complications may be rewarmed rapidly by either showering or immersion in a hot bath. The risks involved (rewarming hypotension) may be reduced if the person is monitored during showering and seated on the floor of the shower if dizziness is perceived. The benefits are immediate and include the following:

- Improved thermal comfort
- Reduced shivering and, with it, decreased oxygen consumption and associated cardiac workload
- Reversal in the decline in deep body temperature
- Peripheral rewarming with a rapid return of peripheral neuromuscular function

However, for the colder diver, whose level of consciousness is impaired, it is prudent to adopt the slower spontaneous method of rewarming described earlier. Regardless of which method of rewarming is chosen, a careful check should be made subsequently for evidence of aspiration and decompression sickness. Should evidence of either condition be suspected, follow the protocols outlined in Chapters 10 and 14.

Should consciousness be lost during recovery or shortly thereafter, adopt procedures to correct possible hypotension pending such information as blood pressure and cardiac function. In the absence of shivering, active measures are required to rewarm the patient to prevent a further decline of body temperature, possibly to a lethal level. Ideally, unconscious cold victims should only be actively rewarmed in a specialized center where facilities are available for invasive monitoring and active intervention should complications arise. However, in remote locations where speedy access to specialist care is not an option, then the emergency procedure of immersion in a bath of hot water at about 40°C (104°F) could be life-saving for a nonshivering, unconscious, hypothermic victim. As well as providing extraneous heat to reverse the continued flow of heat from core to peripheral tissues, immersion has the added benefit of providing some hydrostatic support to a flagging circulation. Once consciousness has returned and the patient is feeling warm again, remove the patient from the water before overheating occurs (with its associated strain on circulating blood volume) and continue rewarming along the passive lines described previously.

Cold Injury Treatment

Because of the paucity of information about the underlying pathophysiology of NFCI, the specific advice in the literature about its treatment is scanty and imprecise. The general consensus of opinion appears to be that affected parts should be elevated, with intermittent passive exercise encouraged, until the edema has resolved. Care should be taken to avoid damaging the affected extremities. In the case of the feet, weight bearing should be avoided. Smoking should be prohibited.

Only slow rewarming by exposure to air should be allowed. The injured area should *not* be immersed in warm water. The early period following rewarming can be extremely painful, even in the absence of obvious tissue damage. Analgesia may be required. Conventional analgesics, systemic and regional, provide only temporary relief or none at all, although *amitriptyline* (10 to 75 mg in a single dose at night) has been shown to have some benefit, particularly if started early. Nonsteroidal anti-inflammatory drugs appear to be of no value.

Once rewarmed, the affected extremities should be treated by exposure to air and early mobilization, ideally under the supervision of a physiotherapist.

Generally recovery is slow, and repeat cold exposure should be avoided. Cases with residual symptoms should be referred to a

specialist neurologic clinic and reviewed 3 to 5 months after injury. If the patient complains of chronic pain, the drug of first choice is, again, amitriptyline. The risk of recurrence following exposure to a less severe stimulus remains high for many years. Sympathectomy should *never* be considered for the treatment of chronic pain or hyperhidrosis resulting from NFCI.

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CHAPTER 14 Near Drowning

Tom S. Neuman

Drowning is reported as the leading cause of death in the approximately 100 to 150 scuba fatalities in the United States each year.[1–10] In reality, arterial gas embolism may be the precipitating factor or even the cause of death in many of these accidents.[11] Indeed, newer reports suggest that the greatest cause of death in male scuba divers over the age of 45 is cardiac related.[12] Nevertheless, near drowning is often a complicating factor in many nonfatal scuba accidents and occurs frequently in the setting of arterial gas embolism.[13] More importantly, drowning is responsible for approximately 7000 deaths in the United States each year, and approximately 90,000 instances of near drowning are reported as well.[14,15] Until recently, drowning was considered to be, after motor vehicle accidents, the chief cause of death among children 4 to 14 years old[16]; however, with the advent of effective automobile child restraint laws, drowning has become the leading cause of death in some states.[17,18] Thus, although scuba diving accidents account for a small percentage of the total number of drowning and near-drowning incidents, the physician who is interested in diving medicine, who is frequently in or around aquatic environments, or who deals with emergencies must be thoroughly familiar with the treatment of these patients.

Drowning and near-drowning incidents can occur in a variety of ways. About 1500 of the total yearly drowning fatalities are related to boating accidents, and approximately 500 of these fatalities occur in victims who die trapped in submerged motor vehicles.[19] Most drownings occur in fresh water (swimming pools, ponds, lakes, and streams), but this may be due to inadequate supervision—approximately 80% of drownings occur at places other than those designated for swimming.[20–24] In contrast, ocean swimming on supervised beaches poses considerably less danger.

The majority of drowning incidents in the toddler group occur in residential pools, generally because of inadequate safety measures in the home.[25–28] In the United States, drowning is approximately five times more common in males than in females, and blacks drown approximately twice as often as do whites.[22] As with fatal automobile accidents, the most important single factor in drowning incidents involving adults is alcohol. Multiple studies in widely different geographic areas, such as the United States,[14,22] New Zealand,[29] Africa,[30] and Australia,[31,32] reveal that alcohol is a significant contributing factor in more than 50% of adult drowning fatalities.

With scuba-related drowning or near-drowning incidents, the most common factor is entanglement or the diver running out of air at depth or in a cave.[1–10] As noted earlier, although events are often reported as "drownings," many of them probably represent cardiac events rather than true drownings or near drownings.

There is strong evidence that intentional hyperventilation before breath-hold diving is associated with both drowning and near-drowning episodes,[33–35] although this is not generally reported in the diving fatality statistics. Hyperventilation reduces the partial pressure of arterial carbon dioxide ($Paco_2$) so that the breath-hold break point is prolonged enough to cause hypoxemia before the person is forced to breathe. In turn, the hypoxemia causes the diver to lose consciousness, and the drowning or near-drowning incident can then occur (see Chapter 5).

Hypothermia has been frequently reported to lead to drowning and near-drowning incidents; however, this is unusual in diving fatalities. It is important to remember that hypothermia per se is rarely the cause of death for people immersed in water. Rather, hypothermia gradually reduces a person's ability to function until the point of unconsciousness or helplessness is reached. At that

point, the victim's head falls into the water, resulting in drowning or near drowning. Perhaps the most famous historical example of this is the sinking of the *Titanic.* When that tragedy occurred, the sea was perfectly calm and help was but a few hours away. There were no fatalities among those in life rafts, and there were no survivors among those in the water.[36] More recently, the *Lakonia* sank, resulting in 200 passengers being immersed in water at 16°C. In less than 3 hours, 113 people were dead.[37] All the victims died from the combined effects of hypothermia and drowning. However, as will be discussed, hypothermia can rarely have a protective effect on the near-drowning victim. There are several documented occurrences of people being submerged for extended periods and recovering completely.[38–41]

Hypothermia is only one of many conditions that can precipitate drowning by causing unconsciousness. In the case of scuba diving incidents, physicians must be aware of the possibility that the diver's air supply may have been contaminated with any of a number of toxic gases. Carbon monoxide is by far the most likely contaminant to produce unconsciousness rapidly underwater, leading to either a fatal or a nonfatal episode.[10] The treatment of carbon monoxide poisoning is beyond the scope of this chapter; however, this condition may have to be treated at the same time as near drowning (see Chapter 12).

Other forms of gas toxicity can produce unconsciousness underwater (e.g., oxygen-induced seizures or adverse effects related to carbon dioxide buildup in closed-circuit or semi–closed-circuit rigs); however, no specific therapy is required other than removing the victim from the exposure to the responsible gas. Recently, oxygen-induced seizures have been implicated as the cause of death in a number of sport divers using oxygen-enriched air as a breathing mixture.[42–44] Indeed, with the increased use of "rebreathers" in the sport-diving population, more and more of these events are likely to occur. Other medical conditions that can produce unconsciousness (e.g., idiopathic seizures)[22,45] in or under the water have been implicated in a number of nondiving fatalities; within the recent past, however, neither idiopathic seizure disorders nor diabetes (in which unconsciousness is induced by hypoglycemia) has been implicated as an underlying cause of a significant number of scuba fatalities in the United States, whether or not they were associated with drowning. The statistics in this regard are compelling enough that several major instructional organizations have developed training protocols for type 1 (insulin-dependent) diabetics (see Chapter 26).

One other important cause of drowning, especially for those involved with aquatic activities, is related to an attempt to rescue other persons. Although the majority of these victims cannot swim themselves (i.e., nonswimming adults trying to rescue children), the risk to a rescuer must always be appreciated.[46] Near drowning can also complicate traumatic injury. Obviously, scuba divers can be struck by boats and suffer closed-head injury or any of a variety of propeller injuries.[10] In addition, the diver can sustain a neck fracture while going through the surf zone. Although uncommon in scuba divers, neck fractures are common in surfers and even more common in persons diving into shallow pools or ponds.[47–49] Occult neck fracture should always be considered in the near-drowning victim. More than half of the 7000 spinal-cord injuries associated with aquatic accidents result in permanent paralysis, and "stick-in-the-mud" accidents (spinal cord injuries from diving into shallow water) are more common in water sports than in all other sporting activities combined.[50]

PATHOPHYSIOLOGY

In recent decades, a considerable amount of experimental evidence largely explains the pathophysiology of drowning and near drowning. Without question, the major pathophysiologic event in near drowning is hypoxemia, with or without aspiration, secondary to immersion in any fluid medium. Hypoxemia explains most of the demonstrable pathology which leads to the long-term morbidity found in near-drowning victims.[51–55]

Approximately 10% to 15% of near-drowning victims appear not to aspirate any fluid during the period of immersion.[56,57] Although it has been hypothesized[51] and generally accepted that reflex laryngospasm occurs and persists until reflex ventilatory activity ceases, no experimental data support this hypothesis. In any event, in these near-drowning victims, the period of hypoxemia is generally only as long as the immersion incident itself; if ventilation can be reestablished prior to the development of any injury sec-

ondary to the hypoxemia, recovery is generally rapid and uneventful. Whether or not these victims actually aspirate no water or trivial amounts of water (such that the effect is clinically insignificant) awaits further study.

When aspiration occurs, the pathophysiologic processes are markedly different than when no aspiration occurs, although hypoxemia remains the underlying primary process in most cases. Unlike the victim without aspiration, the person who aspirates remains hypoxemic even after being removed from the fluid medium and even after ventilation is reestablished. As a result, these patients may experience a longer period of hypoxemia, and secondary damage caused by the hypoxemia is more likely to occur. The continuing hypoxemia is due to direct lung injury from the aspirated fluid, which causes areas of low ventilation/perfusion ratios to develop. It is now clear, however, that the sine qua non of drowning is the aspiration of fluid. The notion that death could take place from drowning without aspiration was based on either misinterpretation or mistranslation of what were considered to be seminal documents.[57] With more recent analysis,[58] the concept of "dry drowning" can appropriately be rejected.

The mechanisms by which hypoxemia develops have not been completely elucidated. With salt-water aspiration, hyperosmotic fluid is thought to cause transudation of fluid into the alveoli, and the aspiration of debris (sand, diatoms, algae) causes a reactive exudate. As a result, the alveoli become filled and are not ventilated, causing hypoxemia. In freshwater aspiration, it is thought surfactant is washed out of the lungs, resulting in regions of focal alveolar collapse, which leads to areas of shunt and a low ventilation/perfusion ratio, again resulting in hypoxemia. These abnormalities then persist until the lung damage resolves or until surfactant can be regenerated.

Because victims often swallow large quantities of fluid during the near-drowning episode, further decreases in ventilatory function can result from elevation of the diaphragm from gastric distention, although an animal model now indicates that gas exchange is actually improved by gastric distention.[59] Vomiting and aspiration of gastric contents can then further complicate the near-drowning episode. Regardless of the cause, hypoxemia and decreased alveolar ventilation have several consequences. Elevations of $Paco_2$ and decreases in pH occur quickly. The former, of course, is due to decreased alveolar ventilation and increased CO_2 production; the latter is due to the combined effects of increased $Paco_2$ and decreased oxygen delivery to the tissues, resulting in increased generation of lactic acid. This metabolic component can be extremely large because the victim often struggles during the near-drowning episode. Finally, cardiovascular collapse can occur, resulting in cardiac arrest. If hypoxemia and decreased cardiac output persist long enough, anoxic cerebral damage can ensue.[38,60–66]

It used to be thought that a significant portion of the physiologic changes associated with near drowning were a result of serum electrolyte changes that occurred with aspiration of salt water or fresh water. These misconceptions were based on a series of carefully controlled experiments in which increasing quantities of salt water or fresh water were instilled into endotracheally intubated dogs. When seawater was instilled beginning at a dose of 1 mL/lb, hypernatremia, hyperchloremia, and hyperkalemia (the average potassium concentration of seawater is approximately 11 mEq/L)[67] quickly occurred, producing what appeared to be lethal electrolyte changes. When fresh water was instilled, hyponatremia, hypochloremia, and hyperkalemia (presumably from hypo-osmolar red-cell lysis) quickly occurred again, resulting in ventricular fibrillation and death.[68–70] Repeated observations in human near-drowning victims, however, conclusively demonstrate that clinically significant abnormalities in serum electrolytes are rare.[53,55,62,65–75]

Although there are minor changes in serum sodium and chloride in the direction expected from the type of aspirated fluid (hypernatremia in salt-water aspiration and hyponatremia in freshwater aspiration), significant changes in serum potassium have not been reported. Indeed, this cannot explain the hyperkalemia that occurred in the original dog studies (allegedly due to red-cell lysis), because the major intracellular cation in dog erythrocytes is sodium rather than potassium.[54] This does not imply that red-cell lysis does not occur with freshwater aspiration but rather shows that the clinical significance of red-cell lysis has perhaps been overemphasized.

In all probability, significant electrolyte changes do not occur in the human near-

drowning victim because the quantity of aspirated water does not appear to be large enough to produce such changes (Table 14–1).[53,55,62,71–76] This appears to be true for the drowning victim as well. In the limited instances in which this issue has been investigated, major electrolyte changes have not been noted in fatal incidents.[77] The exceptions to this situation appear to be submersion victims in the Dead Sea, where electrolyte concentrations are greater than those in usual seawater. Victims of near drowning in that unique environment do have major electrolyte abnormalities, and these disturbances are thought to be responsible for the fatal arrhythmias in that group of victims.[67,78]

The remaining specific consequence of aspiration is pneumonia, which can occur in near-drowning victims and occasionally causes long-term morbidity and mortality.[79,80] A variety of different and unusual organisms have been reported to cause pneumonias in this setting.[81,82] Hemoglobinuria,[83] diffuse intravascular coagulation,[84] and renal failure[85,86] have been reported in near-drowning victims as well as acute tubular necrosis, albuminuria, and rhabdomyolysis.[87,88] The severity of acidosis at presentation also appears to be the best predictor of whether renal insufficiency will develop. Ultimately, all of these are probably nonspecific responses to the hypoxemia, acidosis, and hypotension previously described.

Hypothermia rarely can be protective for the victim who has been immersed in very cold water. Because cardiac arrest occurs secondary to hypoxemia in near drowning, there is presumably a significant delay before the hypoxemia can ultimately cause a cardiac arrest. If the fluid in which the victim is immersed is cold enough, if the surface area/mass ratio of the victim is large enough, and if the victim swallows enough water, the core temperature may decrease sufficiently for oxygen demands to be markedly decreased, protecting the victim from the effects of hypoxemia. However, the exact mechanism of this rapid temperature reduction is unclear, but there is a mathematical model to better understand the contribution of the various factors.[89] It must be stressed, however, that these circumstances are extremely unusual and have been documented most frequently in small children. Current evidence suggests that water temperature generally does not play a role in the neurologic outcome of near-drowning victims.[90]

CLINICAL PRESENTATION

From the previous discussion of the pathophysiology of near drowning, it should be apparent that the clinical presentation of the near-drowning victim can vary considerably. Additionally, the patient's appearance at the scene of the incident (the prehospital setting) may differ from that at the hospital. The patient who is unconscious at the scene and apparently without vital signs may be hemodynamically stable and neurologically intact in the emergency room, whereas the victim initially hemodynamically stable at the scene might deteriorate before arrival at the hospital, independent of the emergency care rendered. Because the clinical presentation can vary so much, it is easiest to describe the clinical status of the cardiovascular, pulmonary, and neurologic systems individually.

Cardiovascular System

Victims of significant near drownings frequently suffer cardiac arrest. The cardiac arrest reportedly responds frequently to resuscitative measures by bystanders (although bystanders may initiate cardiopul-

Table 14–1. Serum electrolytes in human victims of near-drowning

	Na^+ (mEq/L)			Cl^- (mEq/L)			K^+ (mEq/L)		
	No.	*Mean*	*Range*	*No.*	*Mean*	*Range*	*No.*	*Mean*	*Range*
Freshwater victims	22	137	126–146	25	101	88–116	21	4.4	3.0–6.3
Seawater victims	26	147	132–160	28	111	96–127	25	4.2	3.2–5.4

From Modell JH: Blood gas and acid-base changes. *In* Modell JH: The Pathophysiology and Treatment of Drowning and Near Drowning. Springfield, Ill., Charles C Thomas, 1971, pp 44–45.

monary resuscitation [CPR] even in the presence of a pulse), but it is not uncommon for a victim to be brought to the emergency room still requiring CPR. Sinus tachycardia is commonly seen if the victim responds to CPR or if the victim never suffers a cardiac arrest. In the latter case, the sunus tachycardia is usually secondary to hypoxemia and acidosis.[91,92] Usually, the patient is hemodynamically intact (i.e., with adequate blood pressure and pulse, presumably with adequate cardiac output); occasionally, the patient is in shock and requires cardiovascular support. The treatment for the hemodynamically unstable patient is discussed later.

Pulmonary System

Patients with water aspiration may present with few or no respiratory complaints or with severe pulmonary edema.[93] This is due to direct lung injury and is not usually cardiogenic in origin. Patients who have aspirated a significant quantity of water frequently have a widened alveolar-arterial oxygen gradient, and hypoxemia ranging from mild to severe is present. The $Paco_2$ can be low or elevated depending on alveolar ventilation (Table 14–2). Chest radiographs can show patchy infiltrates (Fig. 14–1) (most commonly in the periphery or in the medial basal regions) or frank pulmonary edema (Fig. 14–2).[94–97] The pulmonary edema is noncardiogenic and is a form of acute respiratory distress syndrome.[94] The role that negative pressure may play in the pathogenesis of pulmonary edema in this setting remains speculative.[98,99]

Table 14–2. Arterial blood gas and pH values in patients on admission to the hospital after near-drowning

pH	$Paco_2$ (torr)	Base Excess (mEq/L)	Pao_2 (torr)	Fio_2
In Fresh Water				
6.95	64	–19	245	1.0 R
7.01	38	–22	28	0.2 R
7.05	59	–16	40	1.0 R
7.13	30	–19	67	0.2 R
7.14	45	–14	68	0.2 R
7.18	33	–15	110	1.0 R
7.19	29	–16	108	0.8 R
7.21	37	–13	175	1.0 R
7.22	54	–7	123	1.0 R
7.28	54	–3	35	0.4 R
7.33	41	–4	127	1.0 R
7.40	32	–4	103	0.2 R
7.44	32	–2	76	0.2 R
7.45	35	1	84	0.2 R
In Seawater				
7.03	36	–21		1.0 R
7.08	58	–14		1.0 R
7.20	46	–10	27	0.2 R
7.29	49	–4	364	1.0 R
7.31	35	–8	85	0.8 R
7.35	47	–1	45	0.2 R
7.46	25	–5	71	0.2 R
7.47	26	–3	82	0.4 R

R, mechanical ventilation.
From Modell JH: Blood gas and acid base changes. *In* Modell JH: The Pathophysiology and Treatment of Drowning and Near Drowning. Springfield, Ill., Charles C Thomas, 1971, pp 17–18.

Neurologic Status

The neurologic status of near-drowning patients can also be quite varied. A classification scheme has been suggested to compare results among different groups.[100,101] In this classification system, patients are placed into category A, B, or C based on their initial neurologic status. Category A patients are *a*wake; category B patients are *b*lunted; and category C patients are *c*omatose. Within category C, patients are further classified as C_1, C_2, or C_3, depending on their best motor response. The C_1 comatose patient has a decorticate

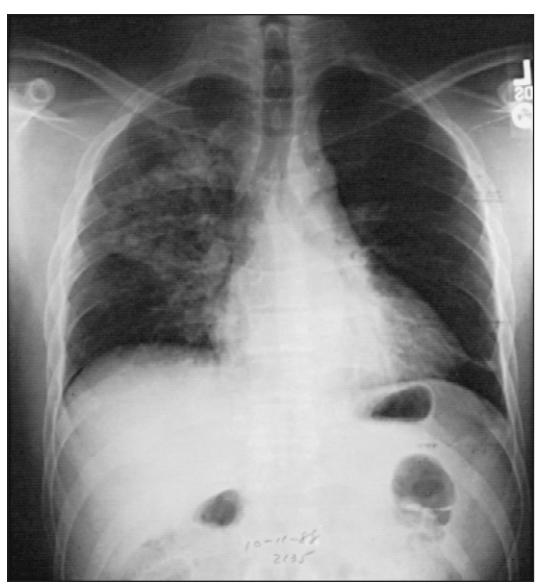

Figure 14–1. Localized, patchy infiltrates in the right lung of a near-drowning victim.

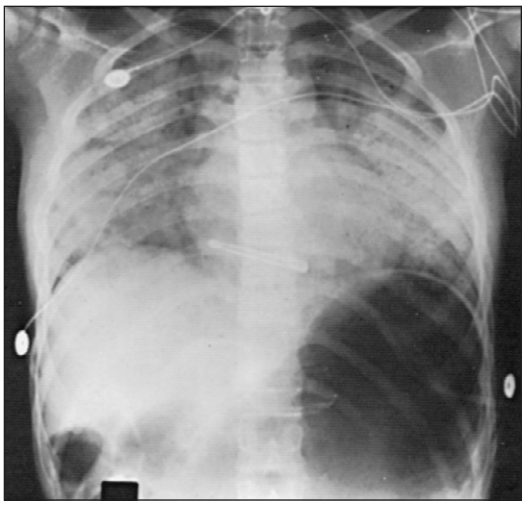

Figure 14–2. Diffuse pulmonary edema in a near-drowning victim. Note gastric distention.

response, the C_2 patient has a decerebrate response, and the C_3 patient has no motor response at all. Treatment for these groups is potentially different and is discussed in the following paragraphs. Unfortunately, there is no uniformly accepted classification scheme for near-drowning victims; as a result, comparison of outcomes among different studies is difficult. Other methods used to classify near-drowning victims are the pediatric risk of mortality score (PRISM),[102] the near-drowning severity index (NDSI),[90] and, of course, the Glasgow coma scale (GCS).

TREATMENT

The near-drowning patient who presents in cardiac arrest should be treated vigorously because recovery with normal neurologic function, although unusual, has been described even after prolonged cardiac arrest.[38–41] Because cardiac arrest in this setting is invariably due to hypoxemia and acidosis, the first goal must be to establish a reliable airway and to supply as high a fractional inspired oxygen concentration (F_IO_2) as possible. Until the results of arterial blood gas (ABG) determinations are available, 100% oxygen should be supplied (preferably through the use of modern high-flow systems that truly deliver a high F_IO_2). Furthermore, because aspiration of stomach contents is a constant threat in the comatose patient, the value of bag-valve-mask oxygenation is at best limited in the advanced care of these patients. The preferred method of establishing an airway is endotracheal intubation, but the possibility of a concomitant unstable neck injury in patients with suspected associated trauma must be considered. In simple near drownings unassociated with trauma, there appears to be little or no risk of associated unstable neck injury[103]; and the risk of aspiration must be kept in mind. Data seem to be insufficient to warrant the routine use of the Heimlich maneuver in near-drowning victims, although this point is controversial.[104] Significant foreign-body aspirations are distinctly unusual, but the possibility of sand or gravel aspiration should be kept in mind. Although not routinely warranted, bronchoscopy is sometimes necessary.[105]

It should also be remembered that profound metabolic acidosis can occur in patients with cardiac arrest secondary to near drowning, and that the doses of bicarbonate necessary to reverse such acidosis may be far larger than the usual doses recommended for patients with cardiac arrest from primary heart disease.[106] Generally, cardiac arrest in immersion incidents is secondary to hypoxemia and acidosis. In cardiac arrest from underlying heart disease, the acidosis and hypoxemia are secondary to the cardiac arrest itself. As a result, ABG measurements are necessary to determine the exact doses of bicarbonate. Concurrent with the resuscitative measures, a nasogastric tube should be inserted to decompress the stomach and body temperature should be measured to rule out hypothermia. In the presence of a significantly lowered body temperature a patient should not be declared dead, and aggressive rewarming measures should be instituted (see Chapter 13).

Once an adequate airway has been obtained and spontaneous cardiac activity achieved, attention must be directed to ensuring adequate oxygen delivery to the tissues by obtaining an adequate Pa_{O_2}. Generally, hemodynamic stability is relatively easy to achieve in the near-drowning victim; rarely, a marked decrease in blood pressure and an associated decrease in cardiac output occur. The initial therapy for hypotension of most etiologies is a trial of fluids, yet this may not be appropriate for a near-drowning victim with pulmonary edema. As a result, this group of patients is best treated with invasive hemodynamic monitoring. Knowledge of pulmonary artery wedge (PAW) pressure and cardiac output

permit more rational decisions concerning the need for fluids or pressors.[93,107] With noncardiac pulmonary edema of any cause, it is important to realize that isolated measurement of the central venous pressure is generally not an accurate method of judging intravascular fluid volume. In addition, changes in central venous pressure (CVP), whether increased or decreased, do not necessarily correspond to changes of the PAW pressure or left-ventricular filling pressure. Thus, central venous pressure measurement has an extremely limited role in the treatment of the near-drowning victim; direct measurement of PAW pressure and cardiac output may be needed in addition to measurement of ABG in patients with hypotension and evidence of pulmonary edema.

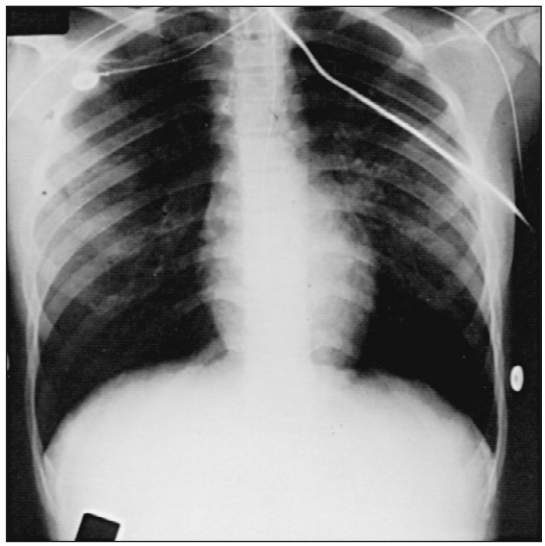

Figure 14–3. Same subject as in Figure 14–2 after positive end-expiratory pressure.

Positive End-Expiratory Pressure

Positive end-expiratory pressure (PEEP) has been shown, both experimentally and clinically, to be extremely effective in reversing the abnormal ventilation/perfusion relationships leading to hypoxemia.[108–112] Only modest amounts of PEEP are usually necessary to achieve adequate oxygenation, and the improvement in pulmonary function can be dramatic (Fig. 14–3; also see Fig. 14–2). PEEP apparently does not alter the course of the underlying pulmonary injury but rather allows for adequate oxygenation while the lung is recovering. PEEP also allows this recovery to occur at a level of inspired oxygen that is not in itself toxic to the lung.[113] The pulmonary injury usually resolves over a period of 48 to 72 hours. Hence, ventilatory support is usually relatively brief unless infections develop.[108] Consequently, in patients who are able to tolerate it, nasal continuous positive airway pressure (BiPAP) may be a reasonable method of short-term ventilatory support; however, the risk of aspiration is not inconsequential with this modality.[114–117] Data appear to be insufficient to warrant the use of inverse-ratio ventilation in cases of near drowning.[118] On the other hand, because evidence is beginning to emerge that PEEP may not be as safe as previously thought,[119] it may be prudent to attempt the use of modern high-flow oxygen delivery systems for relatively brief periods before resorting to more invasive methods of oxygenation.

Surfactants

Given the underlying pathophysiology of near drowning, it makes sense to treat patients with surfactants when necessary. Because the pulmonary insufficiency associated with near drowning is usually relatively easy to manage and usually responds to common measures of ventilatory support, case reports of this modality are few. In the few case reports available, however, surfactants appear to be generally helpful in the treatment of the lung injury; however, it is not clear that these agents confer a significant survival advantage,[120] or that extracorporeal membrane oxygenation does so.[121]

Antibiotics

Near-drowning victims who aspirate ocean water or swimming pool water generally do not need antibiotics except in the settings of fever, new pulmonary infiltrates, or purulent secretions.[122] Prophylactic antibiotics do not seem to improve mortality or decrease morbidity.[52,123] Because most pulmonary infections in the near-drowning victim appear to be hospital-acquired, prophylactic antibiotics seem to select for more resistant organisms.[71] In addition to clinical experience, experimental evidence also suggests that prophylactic antibiotics are not indicated. If the victim aspirates water heavily

contaminated with a known or suspected organism, prophylactic antibiotics may be appropriate.[124] Unfortunately, a percentage of victims do experience infections related to the organisms in the aspirated fluid. A 1997 review details these infections and their associated organisms.[82]

Corticosteroids

Adrenocortical steroids used to treat the lung injury associated with near drowning are also probably unwarranted. Experimental evidence in this and other forms of aspiration strongly suggests that steroids do not improve the long-term outcome or short-term morbidity.[125,126] However, one uncontrolled series of four cases suggests that high-dose steroids may benefit near-drowning victims who present with pulmonary edema.[127]

Cerebral Resuscitation

Cerebral resuscitation is the final aspect of treatment to address at the time of hospital admission. A detailed analysis of the management of the brain-injured individual is beyond the scope of this chapter; however, a brief synopsis may be useful for those who may have to treat a near-drowning victim.

Historically, it has been extremely difficult to estimate the incidence of long-term neurologic dysfunction following near-drowning episodes. Different studies have different selection criteria and different degrees of follow-up. Estimates in large series range from zero to approximately 10%.[28,71,72,91,128,129]

In the late 1970s, after a small experience with near-drowning victims who had a high incidence of long-term neurologic sequelae, it was suggested that aggressive attempts at cerebral salvage could lower the incidence of neurologic dysfunction following near-drowning episodes.[130] This so-called *hyper*-therapy included barbiturate-induced coma, controlled hyperventilation, diuretics, paralysis, intentional hypothermia, and adrenocortical steroids. The rationale for this therapy was to lower intracranial pressure (ICP), reduce cerebral edema, and lower cerebral oxygen demand. All of these measures were to prevent further secondary damage to the neurologic system. This mode of therapy presumes that further damage—after the initial anoxic insult—does occur and that this therapy can prevent such damage.

Unfortunately, after more than a decade of experience with this mode of therapy, it is not clear that morbidity and mortality have improved appreciably. The largest study performed by the group that originally advocated *hyper*therapy reports a neurologic morbidity rate of 7%.[131] This rate is not appreciably different from that reported from multiple studies performed before the advent of *hyper*therapy.[28,71,72,91,128,129] Additionally, other studies have suggested that although high ICPs are associated with a poor outcome,[132] normal ICPs do not ensure neurologic recovery[133] and that *hyper*therapy does not necessarily prevent elevations of ICP. Indeed, it appears that elevations of ICP are the result of brain injury rather than the cause of it.[134] Most authorities certainly agree that, if this therapy is indicated at all,[135] it should be reserved for the most severely affected patients and then carried out only in the setting of an ICU staffed, equipped, and experienced in handling such patients.[101,136] Even in such a setting, the portions of this therapy that are associated with significant morbidity should be reserved for victims whose ICP cannot be controlled by other, more conventional means (e.g., hyperventilation, head elevation, osmotic diuretics). Unfortunately, it appears that no form of specific therapy aimed at cerebral salvage appreciably improves the long-term neurologic outcome associated with near drowning.[137]

The patient's clinical status should be the basis of the decision to admit the near-drowning victim to a hospital. Criteria for admission include significant respiratory symptoms, an abnormal-appearing chest radiograph, a significantly abnormal ABG determination, or the need for supplemental oxygen or ventilatory support. Pulmonary damage is usually evident within a few hours of the event,[52,138] and therefore a patient who has been observed for several hours and remains asymptomatic can safely be sent home.[139] Whether a transient loss of consciousness requires admission is less clear, but the decision should again be based upon the patient's current clinical status.

The previous discussion deals entirely with a victim of near drowning; however, in the setting of a diving accident, an unconscious victim may have sustained an air

Table 14–3. Treatment of the victim of near-drowning

1. Ensure a patent airway. If necessary,
   a. Clear secretions and debris in airway, and suction. (It is not necessary to try to "empty" lungs of water.)
   b. Intubate.
2. Ventilate with as high a percentage of oxygen as can be delivered.
3. If cardiac arrest has occurred, resuscitate according to American Heart Association recommendations.
4. Assess circulation and administer a gentle trial of volume infusion for hypotension.
   If the patient is a diver or has been breathing compressed air, consider the diagnosis of arterial gas embolism and, if needed, arrange for transfer to a hyperbaric chamber.
5. Obtain arterial blood gas levels, a chest radiograph (and C-spine films, if indicated), a complete blood count, a urinalysis, an electrocardiogram, a prothrombin time, and serum electrolyte levels.
6. Insert a nasogastric tube and a Foley catheter, if necessary.
7. Apply positive pressure via PEEP if necessary to maintain adequate Pa_{O_2} and to allow reduction of Fi_{O_2}, or consider nasal CPAP or BiPAP in patients able to tolerate it.
8. For continued hemodynamic instability, consider placement of a Swan-Ganz catheter and measure PAW pressure, PA pressure, and cardiac output.
9. For a deeply comatose victim, consider ICP monitoring. Consider the use of hyperventilation, bed positioning, and diuretics to improve cerebral perfusion pressure. If these measures fail to control the ICP, consider more aggressive measures (e.g., paralysis, barbiturate coma, hypothermia).
10. Treat complications (e.g., pneumonia, seizures, DIC, renal failure) supportively.

BiPAP, biphasic positive airway pressure; CPAP, continuous positive airway pressure; DIC, disseminated intravascular coagulation; Fi_{O_2}, fraction of inspired oxygen; ICP, intracranial pressure; Pa_{O_2}, partial arterial pressure of oxygen; PA, pulmonary artery; PAW, pulmonary artery wedge; PEEP, positive end-expiratory pressure.

embolism as well. Whenever doubt exists, both conditions should be treated simultaneously, if possible (Table 14–3).

PROGNOSIS

The prognosis in the near-drowning victim depends entirely on the duration of immersion, the length of the anoxic period, and the degree of damage secondary to the anoxic episode. The prognosis for patients who are neurologically intact upon arrival at the hospital is excellent; they should survive without any neurologic impairment. Additionally, apparent cardiac arrest does not in itself suggest a poor outcome. If the presumed cardiac arrest responds to first aid at the scene of the accident, the outcome may be excellent. Cardiac arrest that persists through the period of initial first aid and transport to the hospital is a poor prognostic sign,[91] as is an initial rhythm of asystole[140]; however, spontaneous respirations on presentation to the emergency room after cardiac arrest in the field is a good prognostic sign.[141] The duration of immersion correlates with the degree of damage secondary to the anoxic episode and, therefore, with the outcome. For example, estimated immersions that exceed 5 min are often associated with a poor outcome.[128] Finally, it should be emphasized that, with severe hypothermia, predictions of eventual outcome cannot be made.

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CHAPTER 15 Marine Animal Injuries

Carl Edmonds

Serious injury to divers from a marine animal attack is uncommon. Nevertheless, over 1000 marine vertebrates are thought to be either venomous or poisonous. The invertebrates are even more numerous and less well documented.

One comprehensive investigation showed that only 3% to 6% of recreational scuba diver deaths involved marine animals. In most such cases, the provocation lies with the human who threatens the animal or enters its domain. Often the human has intruded with a clear intent to destroy, as with fishermen, divers carrying spear guns, or underwater construction workers. In other cases, a wader, swimmer, or diver intrudes by accident or by choice into the territorial area of the marine animal.

The first group of marine animals discussed in this chapter are those that cause trauma. These animals can use this primitive method of defense or inflict injury as part of their feeding behavior. The forms of physical trauma can vary from biting, to spearing (as from swordfish, sawfish, billfish, and stingrays), to electric shocks from electric rays, eels, and certain fish.[1]

The second group of marine animals inflict pain or cause incapacity by stinging victims in order to obtain food or to protect against predators. These stinging marine animals possess a venom that must be injected by fangs, spines, or stinging tentacles. Some animals hide the venom delivery system until threatened or attacked. Others highlight or display lethal-looking appendages, relying on prevention more than on retaliation. A final *pièce de résistance*, which works well for species survival but which sacrifices the individual animal, is the fish poison. Here a predator succeeds in killing and ingesting the marine animal but then suffers or dies from the effects. Survivors of the predator species thus learn to respect and avoid the poisonous species. There are dozens of fish poisonings, including ciguatera, scombroid, fugu (tetrodotoxin poisoning), paralytic shellfish poisoning, hallucinatory fish poisoning, hypervitaminosis A, and other more common seafood infections, both bacterial and viral.[2]

The toxicology of these poisons is covered in Chapter 16. Marine organism toxicology is as relevant to the general population as it is to divers.

This chapter could have included many other marine injuries, such as marine infections and allergies, but these are too numerous to cover in one chapter. The recommended reading list should help in this regard.

MARINE ANIMALS THAT CAUSE TRAUMA

Although the incidence of serious attacks from marine animals is small, speculation and folklore among enthusiasts have given them a high profile. The types of animal incriminated include killer whales, seals, sea lions, grouper, barracuda, eels, and many types of fish. Two marine animals—sharks and crocodiles—are a cause of genuine concern because they have been responsible for verified fatalities and are the source of much public interest (Table 15–1).

Shark

GENERAL

Our knowledge of sharks has been based more on fiction than fact. Earlier in the last century, sharks were regarded by Europeans as scavengers and cowards, and it was seriously argued whether they ever did, in fact, attack humans. The subsequent controversy resulted in a mass of accumulated data that left no doubt.

Sharks comprise a very efficient and successful evolutionary group of animals

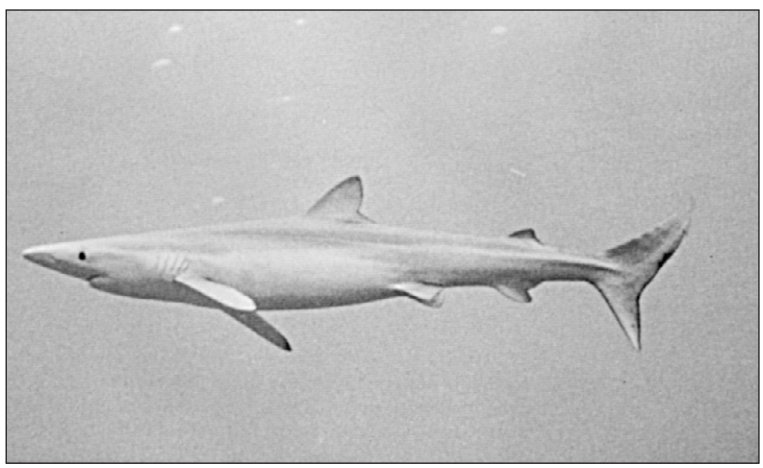
Table 15–1. Examples of marine animals that cause trauma

Shark	Grouper
Barracuda	Killer whale
Crocodile	Miscellaneous biting fish
Eel	Octopus
Electric ray	Coral
Swordfish and sawfish	

(Fig. 15–1). The majority of the 370 species of sharks are marine inhabitants. Many enter estuaries, some travel far up rivers, and a few are freshwater species. Many live in the relatively shallow waters off the major continents or around islands and inhabit the temperate or tropical zones.

The shark is perhaps the most successful of all predators. It has roamed the seas since the very dawn of history, and islanders and seafaring people have incorporated this creature into the center of their folklore. The shark is variously feared, respected, worshipped, idolized, and exploited. Mariners, fishermen, and divers, who are not renowned for their factual reporting, have perpetuated shark obsessions. Rescue and fist-aid groups may have ulterior motives in sometimes exaggerating the risk of shark attack.

Shark attacks on humans are not common. Reporting of these attacks is very country-specific. In 2000, 79 unprovoked attacks were reported, two thirds in the waters of the United States; 70% of these were in Florida. Australia comes second, South Africa third. To put the danger into perspective, many



A

B

Figure 15–1. *A,* Blue shark *(Prionace glauca).* (Photo courtesy Bonnie J. Cardone.) *B,* Great White shark *(Charcharodon charcharias).* (Photo courtesy of Carl Roessler.)

more deaths occur from bee stings and lightning strikes. The estimate has been 1 per 1 million surfer days, whereas 100 million sharks drown in fishing nets each year.

The likelihood of a shark attack depends on two major variables: the number of sharks in the area and the number of people (swimmers, divers, surfers, and board riders). The likelihood of attack is also influenced by the species of shark, the time of day (especially dusk), the water temperature (increases with temperature in most species, but not the *Isuridae*, which have a way of warming their arterial blood supply), and various provocations (see later).

Shark attack remains a genuine but unlikely danger to seafaring people. Although rare, the attack is often terrifying, and the degree of mutilation produced has a strong emotive effect on civilized people. There has been a detectable change in attitude since the advent of scuba diving. Initially, divers engaged in an orgy of destruction against sharks, using spears, powerheads, and carbon dioxide darts. More recently, as divers have observed and then admired the beauty of these animals, attitudes have changed. As in other areas, the camera has replaced the gun. We now look on the sea and its inhabitants as an equally vulnerable and limited resource.

Once a shark attack has occurred, most of those involved would consider the species identification as somewhat academic. This is not necessarily so. Different species have different attack characteristics, and only 30 of the 370 species have been reported as attacking humans. Also, preventive measures must be based on an understanding of shark behavior. Table 15–2 shows typical experience with various shark encounters.

The *Isuridae* are the most notorious of the shark families. They have a fusiform shape, tapered from the pointed snout, with an equally lobed muscular tail. They are capable of fast but stiff-bodied swimming, with short rapid strokes. The large dark eyes testify to the deep-water habits of the porbeagle and the mako, whereas the great white is a more shallow inhabitant (Fig. 15–1*B*).

The great white is the epitome of a man-eating shark. The larger specimens (usually female) weigh between 1 and 2 tons and measure over 6 m long. The teeth are triangular, serrated, and disproportionately large—sometimes over 2 inches long. Great whites are especially found in cold waters with large seal and sea lion populations and therefore are found in shallower areas than the other Isuridae, who feed on deep-sea fishes. The west and northeastern coasts of North America, southern Australia, and South Africa are well-documented areas of habitation and are also favored by surfers and abalone divers.

San Francisco, because of the 120 miles of coast between Tomales Point and Bodega Bay to the north and Santa Cruz and Monterey Bay to the south, is known as the white shark attack capital of the world. The adult females give birth in late summer or early fall in shallow waters south of Point Conception, with the pups remaining inshore and feeding on the prolific fish life. As they grow older, the sharks travel north and offshore to the pinniped (seal, sea lions) breeding areas, around islands. They are not truly territorial and may travel up to 1000 km a month.

Recent decades have seen an increased number of shark attacks along the west coast of the United States on both abalone divers and surfboard riders. Almost four out of every five such attacks are unequivocally due to the great white. It is thought that the attacks are of a feeding type, whereby the surface swimmer or surf craft is mistaken for a seal or sea lion, possibly by immature and inexperienced animals.

Carcharhinids (*requiem* or *gray sharks*) are among the largest and most aggressive, with varied and confusing nomenclature. They range from 1 to 3 m long, usually with the second dorsal fin much smaller than the first and the upper lobe of the tail much longer than the lower.

Table 15–2. Sharks that attack humans

Common Name	Scientific Name	Attacks	Deaths
Great white	*Charcharodon charcharias*	254	67
Tiger	*Galeocerdo cuvier*	83	29
Bull (gray whaler)	*Carcharinus leucas*	69	17

Examples include the tiger shark and the oceanic white tip. The tiger shark is so named because the young are born with stripes. It is also the species most feared by tropical divers. It is pelagic and will eat almost anything. It travels into both the deep and the shallows.

Cousteau described the oceanic whitetip as the most dangerous of all sharks. It is certainly one of the most abundant and congregates rapidly at mid-ocean disasters, such as shipwrecks and airplane accidents.

One of the commonest territorial sharks—and one that can live in the ocean or in fresh (brackish) waterways—is the bull shark. It frequently attacks swimmers, canoeists, and divers.

SHARK ATTACK PATTERNS

There are different types of attack, and these may be identified by the behavior of the animals and the subsequent nature of the injury. Many of these types represent different degrees of a feeding attack, whereas others result from a territorial intrusion into the shark's domain.

The feeding response seems to be related more to the presence of specific stimuli than to the nutritional requirements of the animal. The presence of physical and chemical stimuli, such as those from injured or freshly killed animals, can attract sharks and may result in a feeding response.

Sharks in a feeding pattern tend to circle the victim, gradually increasing their swimming speed. As the circles begin to tighten, the sharks may commence a criss-cross pattern, going across the circle. At this stage, they may produce the first type of injury by contact, when they bump or brush against the prey. The shark's very sharp skin can cause extensive injuries, and it is thought that the information obtained by the animal at this time may influence whether the feeding pattern progresses.

The shark bite is usually performed with the animal in a horizontal or slightly upward direction, with the head swung backward and the upper teeth projecting in a forward direction. This results in a great increase in the mouth size and a display of the razor sharp teeth.

Once the animal has a grip on its prey, if the feeding pattern continues, the mouthful usually is torn out sideways or the area is totally severed. If other sharks are in the vicinity, they may reflexively respond to the stimuli created by the attack and commence another type of feeding pattern behavior: the feeding frenzy.

It has been noted that sharks may swim together in an orderly and smooth manner, but when abnormal vibrations are set up (e.g., by one of the animals being shot or hooked), then the abnormal activity of that animal may trigger feeding responses in the others and this may progressively increase into a feeding frenzy. In this instance, the sharks are likely to attack both the original prey and the predator or any other moving object. During this feeding frenzy, cannibalism has been observed, and the subsequent carnage can be extensive.

This sequence of circling/bumping/biting may not always be followed, especially in the case of the great white, which has the size and strength to attack without warning behavior. Then the first bite on a large animal may be to wound or kill more than to eat. Thus, the prey may be bitten and released ("bite and spit"), to die from blood loss. The shark can then feed without risk of retaliation from its victim.

Once a potential human victim separates from others, the person appears more likely to be attacked. Staying within a group offers some protection. Even going to the aid of a victim rarely results in the rescuer being attacked, although there are at least two cases where this did happen. At least four people have been attacked on more than one occasion, and one had his artificial leg bitten.

A different type of attack is agonistic and involves an animal having its territorial rights infringed on by an intruder, either a swimmer or a diver. It may also happen if the shark is angry, frightened, or engaged in dominance behavior. It is quite unlike the feeding pattern.

The shark tends to swim in a far more awkward manner, shaking its head with a lateral motion, snout upturned with mouth slightly opened, spine arched, and pectoral fins angled downwards. In this position, it appears to be more rigid and awkward in its movements than the feeding animal. This behavior has been described, both in appearance and in motivation, as an animal adopting a defensive and snapping position. If the intruding diver vacates the area, confrontation is avoided and an attack prevented. Most attacks from territorial sharks,

such as the bull shark, are probably of this type. If the intruder does not vacate the area, the shark may snap or rake the victim with the teeth of its upper jaw. This may result in slashing wounds.

Another territorial type of attack may be precipitated by a person entering the water suddenly, onto or near an unsuspected shark. Sharks often follow boats, feeding off the refuse. The "attack" happens with the human falling, jumping, or diving into the water, onto the shark, with the shark responding by snapping at the intruder.

PREVENTION OF SHARK ATTACK

Prevention of shark attack depends very much on the marine locality. The following procedures are relevant in different situations.

Heavily Populated Beaches

The most effective method of reducing the incidence of shark attack is by net enclosures or meshing. Total bay net enclosures are effective in sheltered areas if consistent surveillance is carried out to ensure the integrity of the net. Only small areas are suitable for this technique, and at least one shark attack has occurred within a netted area.

Areas exposed to adverse weather or surf are best protected by meshing. This was introduced to the heavily populated beaches around Sydney, Australia, which had a reputation for shark attack. In 1937, intermittent meshing was introduced along the Sydney metropolitan beaches. Since its introduction, no shark attacks have been recorded at these meshed beaches.

Meshing involves the occasional use of a heavy-gauge net, which is submerged from buoys on the seaward side of the breaking waves. It may be left overnight or for 24 hours and then retrieved. As the animal swims into it, the net wraps around the shark and interferes with its gill function. Because the shark is unable to retreat, it struggles and attempts to push itself forward through the mesh. This results in the shark being further immobilized and thus produces death by suffocation.

The South Africans extended the Australian experience, both by increasing the extent of meshing and by conducting high-quality research into shark morphology and behavior. The Natal Anti-Shark Measures Board is an impressive monument to the importance of this work.

Alternating electric current can also be deployed to protect smaller areas of beach or waterways and is used in South Africa. Protection for individual swimmers and divers has been attempted by carrying small versions of this equipment, producing smaller electric currents (the "shark pod"). Unfortunately, these devices, which can be effective in deterring small sharks, may attract larger ones. This may not be what the diver wants, and at least one diver has been devoured while employing such a device.

Survival Situation

The crash of an airplane into the water and sometimes the noises associated with a ship sinking are effective in attracting sharks to an area. Thus, the survivors of such accidents may become victims of shark attack. The most effective way to prevent this is to use life rafts and have the survivors move into them as quickly as possible. As an alternative, the Johnson shark screen is effective.

The shark screen is a bag of thin, tough plastic with a collar consisting of three inflatable rings. The survivor partially inflates one of the rings by mouth and then climbs into the bag. He fills it with water by dipping the edges, and it appears to any shark as a large, solid-looking black object. The other rings can be inflated at leisure. The bag retains fluid and excreta that may stimulate shark attack.

Swimmers

Swimmers are advised not to urinate in the water or to swim with abrasions or bleeding wounds. Swimmers are also advised to move gently and to not thrash around on the surface. They should stay with a group or at least with a buddy. This is cynically claimed to reduce the chance of shark attack by 50%, but it probably reduces it far more. Swimmers are also advised not to swim in water with low visibility, near drop-offs or deep channels, or toward late afternoon or night, when sharks tend to feed.

It is sometimes claimed that women should not dive or swim while menstruating. There is no evidence to support the belief

that decomposing blood attracts sharks; on the contrary, both experimental and statistical evidence points to the opposite.

Wearing very bright colors is thought to attract some sharks, but these are also the safest colors to wear for other rescue and recovery marine situations.

Divers

The incidence of shark attacks on scuba divers appears to be progressively increasing and now accounts for more than one third of all shark attacks. Wet suits offer no protection and may well increase the likelihood of shark attack, despite popular hopes to the contrary. Divers are advised in the same way as swimmers but with added precautions.

Underwater explosives tend to attract sharks. Shark attacks are more likely at increased depth and can be provoked by playing with or killing fish or other sharks. It is preferable to dive in areas where spear fishing is not performed. Divers are also advised not to tether fish or abalone near their bodies.

Shark feeding, although of commercial value to some dive operators, is a way of conditioning sharks to relate divers and their boats to food. This attracts them to the same area that the diver is occupying. The consequences are obvious.

Powerheads, carbon dioxide darts, and the drogue dart (this has a small parachute attached, which disrupts the shark's orientation and swimming efficiency) are all specialized pieces of equipment which may be appropriate in certain diving situations.

The use of Kevlar incorporated into wet suits has been investigated as a shark bite–resistant material. It is currently being used as bulletproof vest material and is able to stop a .45-caliber bullet. Kevlar does not stop penetration of teeth from a relatively mild dusky shark.

Steel-meshed diving suits definitely discourage an attack but are dangerous unless extra precautions are taken to ensure buoyancy.

If one is diving in shark-infested waters, the use of a *shark billy* can be effective. The shark billy is of greatest value to experienced divers who often encounter sharks. It is a sturdy stick, with a nail stuck into it; the billy is used to push away any curious or interested shark.

More sophisticated techniques involve electrical stimulation (electric prods), chemical or sonic deterrents, and bubble dispensers. Electrical methods (the "shark screen" is a portable variation of the shark pod) are currently in vogue.

Action to Take If Shark Attack Is Threatened

Although the shark may do the unexpected, more often the behavior is predictable. If sharks are encountered, it is best to descend to the seabed or to the protection of rocks, cliff face, or some other obstacle so as to interfere with the normal attack patterns described earlier. If the shark retreats, the diver should slowly move along the shelter or head for the beach or boat when the shark is at its greatest distance. Always stay in a group, both under and on the surface.

If the shark is involved in feeding behavior, separate yourself from the probable source of the stimulus. Abandon any caught fish. If you are producing the stimulus, such as by overhand swimming, kicking, or prying abalone off rocks, then desist in the activity. As calmly as possible, and without heading into open water, leave the area. Continue to face the shark.

If the attack is agonistic, with the typical posturing described previously, remain motionless for a few seconds while you appraise your situation. Face the shark, be prepared to fend it off with anything at your disposal, and calmly vacate the area, swimming backward.

If the shark comes within a meter or two, any action may disrupt the feeding or agonistic pattern, so that yelling, blowing bubbles, or sudden body actions may be of value. Kicking or striking the shark in a sensitive area—eyes, snout, gills—with a knife, snorkel, or other instrument may terminate the attack.

Deterrents of Historical Interest

A vast number of experiments have been carried out to demonstrate ways of preventing shark attack. The less attention given to the repeated failures with electrical, chemical, sound, and bubble deterrents, the better.

Table 15–3. First aid and treatment for shark injuries

Rescue the victim and stop the bleeding.
Lay the patient head-down and elevate the affected limb.
Call in medical assistance.
Intravenous fluid replacement and treatment of shock takes precedence over transfer to the hospital.
Emergency surgical procedures include débridement, cleansing of the wound, removal of foreign bodies (shark teeth), and antisepsis prior to reconstructive surgery.

TREATMENT

Apart from rescuing the victim from the water, and the source of further injury, the basis of treatment is to prevent hemorrhage and treat for shock. Because the wound is exceptional in promoting excessive blood loss, this should be countered by pressure, a tourniquet, or both should be used for arterial bleeding, and elevation of the wounded limb should be used for venous bleeding. The majority of attacks are nonfatal but require extensive surgical repair of lacerations.[3] Fatal attacks usually result from blood loss due to laceration of a major artery.[4] Excessive movement results in more bleeding and clinical deterioration. Table 15–3 summarizes current recommendations for management of a shark injury.

Crocodiles, Caimans, and Alligators

GENERAL

These reptiles, survivors of the dinosaur age, are found in the tropics and subtropics of the Americas, Australia, Indo-Pacific islands, Asia, and Africa. Crocodiles, despite their unlovable appearance, live a highly developed social life. They are very territorial and communicate underwater by a variety of deep-throated sounds and higher-pitched oral noises. They display emotions by specific body postures and become more aggressive during breeding times. The young are hatched from eggs and are protected by both parents.

The species considered as man-eaters are the saltwater crocodiles and the Nile crocodile, which grow to 8 m in length, and the American crocodile and American alligator, which grow to 3.5 m. South American caimans are of the same family as alligators. The Indian mugger crocodile may attack humans if provoked while nesting. All crocodilians are carnivorous. They grow from 1 to 10 m long; the larger specimens are the ones more potentially dangerous to humans. The largest grow up to a ton in weight.

South American caimans can grow up to 5 m long, although most are much shorter. As one moves north to the Gulf of Mexico, the southern United States, and the Caribbean, crocodiles are more numerous. The crocodiles of Florida were nearly wiped out by hunting in the Everglades and during land development of the Keys. Conservation attempts in recent decades have resulted in a rise of some crocodilian populations, but this also has consequences that are difficult to reconcile. As the animals grow older and larger, conflicts with humans emerge. Crocodiles are often thought to be responsible for damage to fishermen's nets, and they may prey on both domestic animals and humans.

Alligators are slower-moving and generally less dangerous to humans. Crocodiles have narrower snouts than alligators, and the fourth tooth on each side of the lower jaw is usually visible when the mouth is closed. The fact that the animal is found in fresh water may signify that it has swum inland from the estuary, not that this is necessarily a freshwater crocodile. The latter are found in lakes and rivers that have no connection with the sea, and in some countries these crocodiles may be large and dangerous. For reptiles, crocodiles have very complex brains and are intelligent enough to stalk a human, strong enough to destroy a water buffalo, and gentle enough to release its own young from the eggs with its teeth. The animal even carries the newly hatched babies in its massive jaws.

Crocodiles tear their food off the carcass by twisting and turning in the water. They then swallow it whole. Stones are also swallowed, and these increase the animal's specific gravity so as to be neutrally buoyant in water. The animal often lies along the banks of rivers, with the nostrils intermittently protruding above water to breathe. Prey, especially land animals, that come to the riverbank may be suddenly grabbed in the crocodile's immensely strong jaws and

twisted off their feet. Once the prey is in the water and does not have the traction usually produced by its feet, it is more vulnerable to panic and drowning. Crocodiles can also move fast on land and can attack there or while free swimming, as has been demonstrated by recent attacks. On land, attacks usually occur at night when the animal commonly stalks for food. Crocodiles move surprisingly fast (faster than most humans), issue a hissing sound, and sometimes attack by sweeping the victim with their powerful tails.

Barracuda

Barracuda are carnivorous, fast-swimming fish, greatly feared in some parts of the world (Fig. 15–2). They often travel in schools, although very large ones are sometimes seen alone. The larger and more aggressive specimens are encountered throughout most tropical and subtropical waters. Many grow to a length of 2 m and weigh up to 40 kg. They are known to herd fish into areas and then decimate their prey with razor sharp teeth, slashing from side to side. After barracudas have killed enough fish, they are devoured at leisure.

Occasionally a barracuda stalks a diver, often at a distance of only a few meters, and sometimes into water no more than knee deep. These fish are territorial, and the stalking behavior is usually related to intrusion into the barracuda's domain. This behavior rarely results in an attack, and the barracuda can be chased away by an aggressive or forward movement of the diver. The fish, however, may only travel a short distance before it turns and continues the stalking behavior. This intimidation is aggravated by the barracuda's dental structure, which has an adverse psychological effect on the diver. Leaving the barracuda's territory usually eliminates the stalking behavior. Most barracuda bites are usually on victims who were themselves the predator, having speared barracuda or hauled them inboard on a fishing line. Although divers have been attacked in the Pacific—one spearfisherman had his kneecap bitten off—such attacks are rare.

The danger of barracuda attack is greatest when one is diving at night with a light. This is thought to blind the fish and cause it to panic, resulting in possible injury to any diver in its path. The barracuda may also act reflexively by slashing with its teeth at any fast-moving or brightly colored object near it. It is also attracted to the abnormal movements of a speared fish. One such fatality off Key West in Florida involved an airline pilot who wore fluorescent swimming trunks. The barracuda slashed his groin and buttocks and then hovered around the area as rescuers tried to protect him and then transport him to the hospital. He bled profusely and died en route.

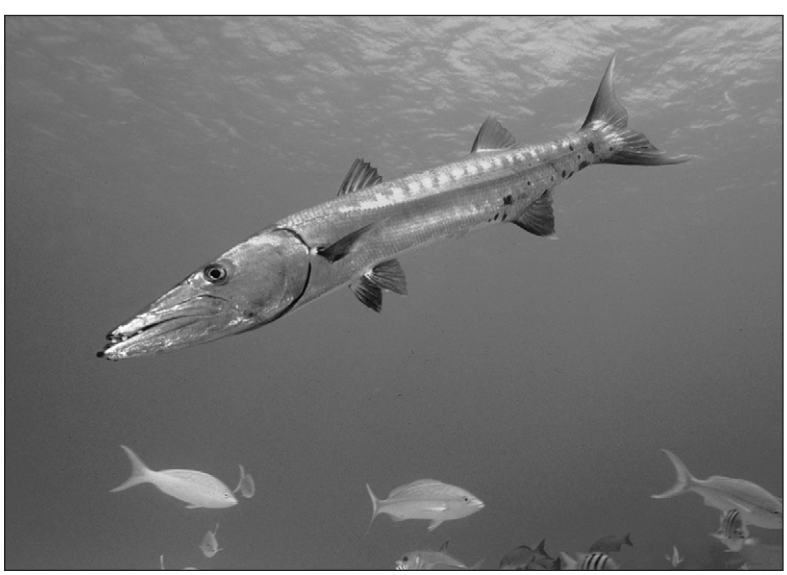


Figure 15–2. Barracuda. (Photo courtesy of Jeri Murphy.)

Eels

The distribution of moray and conger eels is in tropical, subtropical, and temperate waters. There are many instances of morays being tamed and fed by divers, using cut urchins, sausages, or pieces of fish. Some dive resorts have their own pet moray, which can be handled and fed if the diver moves slowly. The eel then learns to equate divers with food and may become aggressive when food is not offered. Attacks sometimes occur when divers wave their hands near the eel, when the eel is speared, or when surfers dangle their feet or hands over their boards. In the last situation, the eel attacks in open water, behavior that I did not see in my earlier diving years but that now happens frequently where eels are fed.

Sharp, slashing lacerations may occur and are probably examples of defensive rather than feeding behavior. The presence of any significant venom apparatus is questionable or unlikely. The wounds are usually of a torn and ragged type (Fig. 15–3). Initially there may be profuse bleeding. Secondary infection in the area is particularly common. A major complication is blood loss with shock. The clinical features include a sweaty (cold and clammy) appearance, rapid pulse, hypotension, and syncope on standing (fainting).

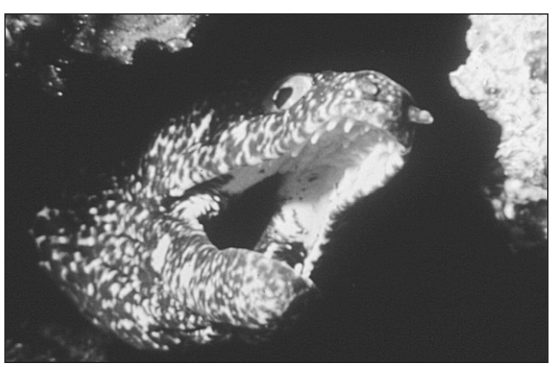
PREVENTION

- Do not spear eels.
- Do not intrude into their domain.
- Do not feed eels.
- Wear heavy protective clothing (boots, gloves) when contact is possible.

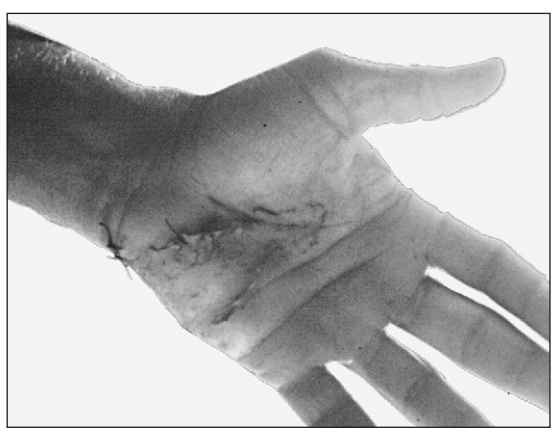
Electric Rays

Electric rays (Fig. 15–4) are found in temperate and tropical oceans, as is the other marine fish that produces an electric discharge, the stargazer. Rays are commonly encountered by divers because these animals are found in relatively shallow depths and can be submerged in mud or sand.

The electrical discharge varies from 8 to 220 volts and is passed between the electrically negative ventral side of the ray to the positive dorsal side. The thick electric organs are usually discernible on each side. Activation of an electric discharge is a reflex action, the result of tactile stimulation. The ray can deliver a successive series of discharges, but these are of lessening intensity. There is a latent period in which the fish recuperates its electric potential. It is not necessary to actually touch the ray to get a shock. The electric shock may have a serious effect in disabling a human temporarily and presumably could be more hazardous to a child. Subsequent danger may come from drowning or aspiration. The affected skin usually shows no local manifestations. Recovery is uneventful, and treatment is not usually required.



A



B

Figure 15–3. *A,* Moray eel showing the open mouth and arrangement of teeth. (Photo courtesy of Alfred A. Bove, MD, PhD.) *B,* Moray bite that demonstrates the outline of the V-shaped mouth. This injury required surgical débridement and suture closure of the wound. (Photo courtesy of Peter R. Lynch, PhD.)

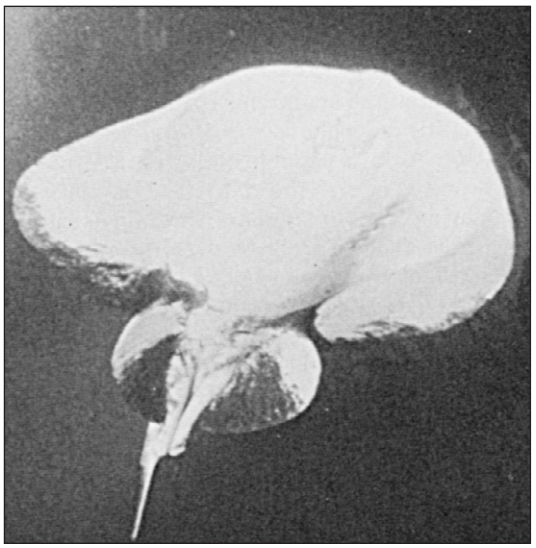


Figure 15–4. Electric ray (torpedo ray). Electric discharge up to 200 V can briefly paralyze a diver. (Photo by Bonnie J. Cardone, with permission from Peterson Publishing, Los Angeles.)

Grouper

Groupers are usually found in tropical, subtropical, and temperate waters. They are the heavyweights of the sea. They may grow to 100 to 200 kg. Groupers are the most friendly of all fish to divers. They live in wrecks, caves, and coral caverns and are protected by overhangs. They are often photographed, being very curious, and appear to make good friends. There is a slight danger when hands and feet are waved around, simulating perhaps the activity of fish, which may certainly attract the attention of the myopic grouper. This is responsible for most of the minor injuries that have occurred, although the appendage usually is spat out while still attached to an arm or a leg, when it is unable to be swallowed.

Most of their food is swallowed whole despite the jaws, which are capable of ripping most prey apart. Unprovoked attacks on swimmers and divers have been reported, and in some areas such as the Torres Straits, groupers are feared more than sharks. They have been known to grab the hands, feet, and even the whole bodies of divers and surfers, verifying the potential of groupers to cause injury. Occasional fatalities have been described, although not well documented. The only case known to me involved a small female child.

PREVENTION

- Do not dispute the territorial rights of a grouper.
- Ensure that there is no contact with injured fish. Do not handle or carry speared fish.
- Some claim that traces of grease or perspiration attract this animal.
- Most scare techniques used by divers against sharks (e.g., blowing bubbles, making a noise, moving toward the fish) are of no avail with groupers.

Killer Whale

The killer whale, or Orca, is distributed throughout all oceans of the world but more frequently in the polar regions. Said by some to be one of the largest and most dangerous creatures (a mammal) to be found in the sea, it grows up to 9 m in length and has a blunt, rounded snout. It is shiny black with white over the eyes and on the belly, giving it a spotted appearance.

The killer whale is strictly carnivorous and is exceptional in being ferocious without provocation. Attacks are not related to environmental temperature, and the whale, which is really a dolphin, has been known to leap out of the water to attack and destroy boats and to up-end ice floes to reach its victim (usually a seal). The bite is achieved with 10 to 14 interlocking conical teeth on each jaw. Killer whales hunt in packs of up to 40, preying on other marine animals, including whales, seals, and penguins. The name is actually derived from the fact that it is a killer of whales.

Attempts by divers to approach these animals in the wild have been unsuccessful, with the whales retreating rapidly. This behavior is not consistent with the animals' horrendous reputation. In 1991, in Canada, a female university student slipped into a holding area for killer whales. She was towed into the center of the area by the animals and pulled underwater on a number of occasions until she finally drowned. This is predictable killer whale behavior toward other prey. For over an hour, attempts to throw ropes to her or rescue her were unsuccessful. At autopsy, there was no significant physical trauma.

Miscellaneous Biting Fish

PIRANHA

The piranha (*Serrasalmus* spp) probably have the worst reputation of all small fish, and although they are carnivorous and can be ferocious and voracious, they do not deserve their terrifying reputation. Abundant in the rivers of South America, most of the 20 or more species of piranha are harmless. The largest grow up to 45 cm long; others grow only a few centimeters. Only the black piranha and its relatives need cause concern. When there are enough of them, they are thought to be able to remove all the flesh from a large animal within a few minutes.

Piranha have a very deep body, a blunt head, and strong jaws with triangular razor-sharp teeth that interdigitate during the severing bite. They are thought to be attracted by blood, even though the usual source of food is other fish. When they do attack larger prey, each bite cleanly cuts a chunk of flesh.

OTHERS

Taylor, or blue, fish (e.g., *Pomatomous saltatrix*) do not have the same reputation as the piranha, although they certainly work in large schools and have caused occasional injury to bathers. When caught on fishing lines, these fish can react violently, and there have been reports of fingers being amputated. Taylor fish commonly travel in large numbers along the east coast of the United States, and on August 12, 1974, the beaches near Bakers Haulover in Miami had to be closed when swimmers were driven from the water by large numbers of bluefish which, although probably attracted by schools of mullet hugging the coastline at that stage, attacked many swimmers. A number of fingers and toes were badly injured, and many people needed sutures.

Spanish mackerel (e.g., *Scomberomorus maculatus*) have also occasionally attacked and injured swimmers en masse.

Some fish not usually known to bite humans may do so under certain circumstances. The very beautiful and famous bat fish (*Platax* spp) around Heron Island in the Great Barrier Reef, an area that has for long been a marine reserve, are used to being fed by divers and are likely to move in large numbers and nip at divers' exposed skin.

Most of the puffer fish (*Tetrodon* spp) have four strong biting teeth and are slow-swimming. They are encountered frequently by divers and fishermen. Because the jaws are designed to crunch through crustaceans, the tip of a human finger is not beyond a nibble.

Octopus

ANATOMY AND PHYSIOLOGY

The octopus is an eight-armed cephalopod of the order Octopoda. They inhabit all oceans from the Arctic to the Antarctic, from the surface waters to depths of 5000 m. The largest is the Pacific *Octopus hongkongensis,* which has an arm span of up to 9.5 m. The common octopus, *O. vulgaris,* lives in tropical and temperate waters and can have an arm span of 3 m and weigh 22.5 kg.

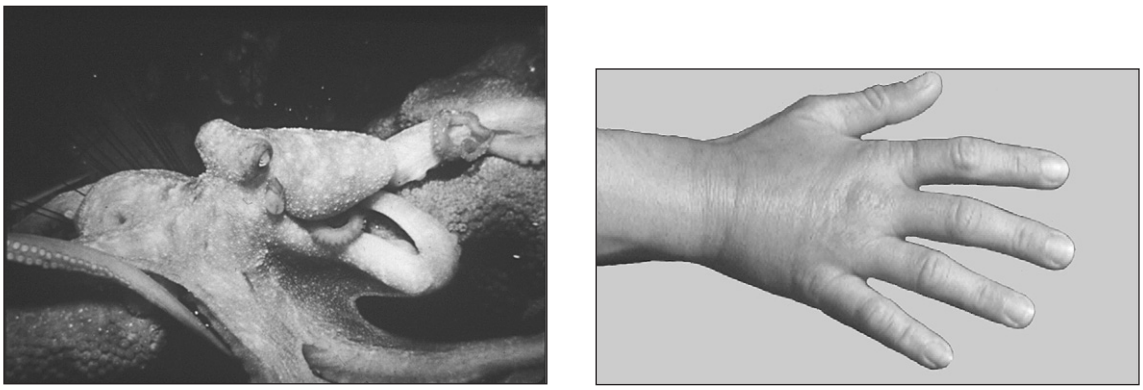
During the octopus massacres by scuba divers of previous decades, octopuses with a span of 5 m and weighing over 50 kg were commonly caught in the Puget Sound of Washington State.

The octopus has a beak capable of piercing shellfish; suction pads that are strong enough to pry open bivalves and oysters; and defensive sepia or "ink" that can be used as a "smoke screen," as a falsely aggressive threat, as a decoy to distract large predators, and to confuse predators with a scent similar to that of the octopus.

HUMAN INJURY

The bite from an octopus can cause various symptoms in humans, sometimes causing pain very quickly (Fig. 15–5). At other times, a severe tissue reaction or inflammation occurs, with gross swelling and numbness.[5] It may take many days to resolve and often produces itching. The medical literature yields only a dozen or so reports of octopus bites to humans. The sepia can produce corrosive dermatitis.

By far, the commonest conjecture regarding damage from octopus to divers is the possibility of the octopus fixing itself to an undersea structure while holding onto a



A **B**

Figure 15–5. *A,* Common octopus indigenous to the tropical and temperate oceans. *B,* Hand of a diver who allowed an octopus to reside on the dorsum of the hand. The bite is noted as a raised area at the base of the middle finger, the hand is edematous, and the patient complained of joint stiffness. (Photos courtesy of Peter R. Lynch, PhD.)

skindiver with the suckers on its other arms. It would be easy to write this off as poetic license had it not been for the experiences of some divers who would otherwise be classified as reasonably credible.

Cases have been reported in which an octopus has actually attacked divers. Occasionally they have been unprovoked, but more often the octopus is retaliating against a spear or knife.

Squid, the relative of the octopus, are potentially more dangerous than octopus.

Swordfish (Billfish) and Sawfish

Swordfish and sawfish are distributed in tropical, subtropical, and temperate waters. Although uncommon, there are enough documented cases of death and injury from these fish to warrant mention. Injury can be caused by the large spearlike or sawlike extension of their jaw.

Billfish are renowned for their fighting abilities, and marlin are the epitome of game fish. Other billfish include the swordfish and spearfish, which can be of a similar size and nature, and the usually smaller sailfish.

These fish have been known to attack whales, other fish, and humans. Injury to humans has usually occurred after the capture of a fish, usually by a game fisherman, and during the attempt to bring the fish on board.

Garfish have also been known to spear humans. These fish are smaller and include many species in temperate and tropical waters. They are known as needlefish, long tom, flying fish, and skipper (alluding to the habit of jumping out of the water and skimming along the surface for short distances). The slender spearlike beak is really an elongated jaw.

Garfish can often be seen in schools and leap sometimes up to 2 m above the surface.

These fish can impale themselves into humans as they skim over the surface. In one series of garfish injuries in Papua New Guinea, of the 10 accidents to humans, 3 died, 3 recovered after exploratory surgery, 2 were blinded, and 2 experienced no sequelae.

Sawfish can grow up to 6 m in length and weigh up to 450 kg. They are sometimes captured by fishermen using nets, although sawfish can usually chop through most fishing nets as they can through schools of fish (destroying many and eating few). If, by accident, the saw penetrates a large fish, then the latter is rubbed off on the seabed, the sawfish scraping the victim off the saw before consuming some of its remnants.

Occasional unprovoked attacks have been reported. One was an attack on a 15-year-old boy who had been collecting shiny cans from the water. The cause of death was thought to be a combination of severe blood loss from the abdominal wound and subsequent drowning.

Coral Cuts

Because of the coral's sharp edges and the awkwardness of humans in the sea, corals often cause lacerations. The sequelae of

such cuts may well equal the intensity of the more impressive marine animal injuries. Not only is the coral covered by bacteria-laden slime, but pieces of coral or other foreign bodies often remain in the laceration. Some of the manifestations, especially initially, could be due to the presence of discharging nematocysts. Patients occasionally have been affected by the marine organism *Erysipelothrix.* Certain *Vibrio* organisms may be present in the marine environment and can cause serious infections. These may be cultured on conventional blood agar plates at 37°C. However, other marine organisms must be cultured in special saline media and at different temperatures to permit identification.[6] This is also relevant to other forms of marine trauma and skin abrasions.

A small, often clean-looking laceration is usual on the hand or foot. It causes little inconvenience at the time of injury and may well go unnoticed. A few hours later, there may be a burning sensation, especially during washing. At that stage, there is a mild inflammatory reaction around the cut. Within the next day or two, the inflammation becomes more widespread, with local swelling, discoloration, and tenderness. Severe cases may lead to abscess formation with chronic ulceration and even osteomyelitis. After healing, there may be a small numb area of skin with a fibrous nodule beneath it, a keloid-type reaction to the coral foreign body.

Treatment involves thorough cleansing of the area, removal of the foreign material, and application of an antibiotic powder or ointment such as neomycin. Systemic antibiotics may be needed; if this is so, third-generation cephalosporins may be used until the organism is identified or culture and sensitivity results are obtained. One sequela of coral cuts is sometimes a very unpleasant pruritus that can be troublesome for many weeks. It responds to the use of a local steroid preparation.

VENOMOUS MARINE ANIMALS

Venomous animals selected for discussion in this chapter include sea snakes; fish (stonefish, stingrays, and others); box jellyfish, *Physalia,* and other coelenterates; and miscellaneous invertebrates (cone shells, blue-ringed octopus, sea urchins, and sponges) (Table 15–4). Many such creatures are not mentioned specifically, but their effects and treatments can be extrapolated from similar animals (such as with fish stings). Some, such as sea lice, are a combination of a variety of others (coelenterates, small biting crustaceans, and allergy-producing organisms). Many others are not mentioned at all.

Table 15–4. Examples of venomous marine animals

Seasnakes	
Fish	
Stonefish	Old wife
Stingrays	Rabbitfish
Miscellaneous scorpionfish	Stargazer
Weeverfish	Surgeonfish
Catfish	
Coelenterates (Jellyfish)	
Portuguese man-o-war, *Physalia*	Jimble
	Mauve stingers
Chironex, sea wasp, box jellyfish	Fire coral
	Sea anemone
Cyanea	Stinging hydroid
Irukandji	
Miscellaneous Invertebrates	
Cone Shell	Sea urchin
Blue-ringed octopus	Sponge

Sea Snakes

GENERAL

Sea snakes are air-breathing reptiles that number some 50 species (Fig. 15–6). They are usually found in tropical or temperate zones and are most frequent in the Indo-Pacific area. Sea snakes can be subdivided into two major types according to their feeding habits.

The *bottom feeders* can dive to considerable depths (over 100 m) to locate and devour their prey (eels, fish). The *Laticauda*, or banded sea snakes, are characteristic of this type. They are necessarily restricted to coastal and relatively shallow waters, often breed and lay their eggs onshore in crevices or caves, and can exist for long periods out of water.

The second group is the pelagic *blue water* type, exemplified by the yellow-bellied sea snake, *Pelamis platurus.* These snakes are surface feeders that drift with the warm

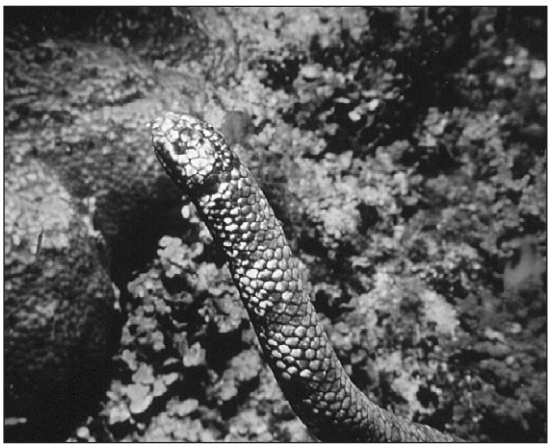


Figure 15–6. Sea snake. Several species are present throughout the Indo-Pacific. Usually docile except during mating season. The toxin is more potent than that of the King Cobra, but fortunately the fangs are quite short and located posteriorly. Human envenomation is rare. (Photo courtesy of Paul Cianci, MD.)

tides. Mating takes place at sea, and the snake is viviparous. It may be found in packs far out in the ocean, but it dies if washed up on beaches or land. This snake does not tolerate extremes of temperature and is rarely found when the average sea temperature drops below 20°C. The lethal limit for the snake's body temperature is 33° to 36°C, and the snake avoids high temperatures in tropical regions by diving into the deep cool waters. This is why these snakes are more frequently found on the surface during rain or on cloudy days.

The sea snake has adapted to the hypertonic saline environment by developing salt-excreting glands under the tongue. It has also developed a flattened paddle-shaped tail and a laterally compressed body that make it an efficient swimmer. It is capable of remaining submerged for 2 hours, perhaps by decreasing its metabolic rate and developing an increased tolerance for hypoxia. The lung may function as a hydrostatic organ, regulating the snake's buoyancy. Sea snakes are inquisitive and are sometimes aggressive, especially if handled or trodden on. They appear to be attracted by fast-moving objects (e.g., divers being towed by a boat), and under these circumstances they can congregate and become troublesome. They are also caught in trawling nets and pose a frequent hazard to fishermen.

Land snakes may also take to the water, sometimes making identification difficult. A sea snake is characterized by a flattened, paddle-shaped tail. No land snake has this tail. Sea snake venom is much more toxic than cobra venom, but less of it is delivered, and only about one quarter of those bitten by sea snakes ever show signs of envenomation.

These snakes show some reluctance to inject venom even when they do bite. Nevertheless, the fresh venom of one adult sea snake of some species is enough to kill three men. In most species, the apparatus for delivering the venom is poorly developed even though the mouth can open widely; in other species, the mouth is small and the snake has difficulty in biting wide enough to pierce the diver's clothing or any other protective material. Unfortunately, in some species, such as the olive-brown and the Stoke's sea snakes, the venom is very toxic and their fangs are well developed and could easily pierce wet suits.

Sea snake venom is a heat-stable, nonenzymatic protein that appears to block neuromuscular transmission by acting on the postsynaptic membrane and affecting the motor nerve terminals. The venom has a specific action in blocking the effects of acetylcholine. Autopsy findings include patchy and selective necrosis of skeletal muscles and tubular damage in the kidneys if the illness lasts longer than 48 hours.

CLINICAL FEATURES

An initial puncture when bitten is usually noted. The number of fang and teeth marks varies from 1 to 20, but usually there are 4, and the brittle teeth may remain in the wound. Usually, there is little or no local pain or swelling, although occasionally the wound can be more extensive. After a latent period without symptoms, which may vary from 10 minutes to several hours, generalized features are noted in approximately one quarter of cases.

Mild symptoms include a psychological or toxiconfusional reaction, such as euphoria, anxiety, or restlessness. The tongue may feel thick. Thirst, dry throat, nausea, and vomiting occasionally develop. Generalized stiffness and aching may then supervene, and muscle weakness may be noted. If weakness does progress into paralysis, then it is usually of the ascending Guillain-Barré type, with the legs involved an hour or so before the trunk, then the arms and neck. Another manifestation of paralysis extends centrally

Table 15–5. Management of parenteral envenomation by marine animals

Pressure Immobilization

Pressure immobilization is used as a first-aid treatment for potentially lethal toxins that are fast-acting and induce little pain and inflammation. Sea snake, blue-ringed octopus, and cone shell injuries are typical. This technique is not recommended for fish stings, jellyfish stings, and urchin spine injuries, because the pathology of these injuries could be aggravated by the constriction.

Pressure bandages, using a wide strap and about the same tension as for a sprained ankle (50 to 70 mm Hg), are wrapped around the area of the bite and proximal to it. If combined with immobilization of the area, this wrapping inhibits both venous and lymphatic drainage of the area. Give reassurance if needed, and otherwise keep patient exertion to a minimum. The affected limb or body area must be immobilized. Removal of the pressure bandage should await the availability of resuscitation facilities, skilled personnel, and the acquisition of appropriate antivenom, if available.

Thus, pressure immobilization may be employed for a few hours while awaiting appropriate resuscitation facilities. Once this is obtained, the pressure should be removed. If there is sudden deterioration, as the venom is circulated, the pressure immobilization can be temporarily reemployed.

from the area of the bite, e.g., from the bite on the hand to the forearm, arm, other arm, body, and legs. Usually the proximal muscle groups are the most affected, and trismus and ptosis are characteristic. Muscular twitching, writhing, and spasms may occur, and the patient may experience difficulty with speech and swallowing as the paralysis extends to the bulbar areas. Facial and ocular palsies then develop. Respiratory distress due to involvement of the diaphragm may result in dyspnea, cyanosis, and finally death. Cardiac failure, convulsions, and coma may develop terminally.

Myoglobinuria may develop some 3 to 6 hours after the bite in serious cases. When this occurs, one must consider the other possible effects of myonecrosis, namely, elevated plasma creatine kinase, acute renal failure with electrolyte and potassium changes, uremia, aggravation of the muscular paralysis, and weakness. The myonecrotic syndrome with renal failure usually supervenes on the other muscular paralysis and may thus prolong and aggravate this state. When recovery occurs, it is usually rapid and complete. Coagulation and hemolysis, as occur with terrestrial snake bites, are uncommon.

TREATMENT

First Aid

It was originally thought that a venous ligature above the site, together with removal of the surface venom, was indicated. The current treatment is to apply pressure immobilization (Table 15–5).

If possible, the snake should be retained for identification because, although it may be harmless, the treatment certainly is not. Serum can be assayed for sea snake venom, verifying the diagnosis. In the event of severe manifestations, mouth-to-mouth respiration may be required.

Medical Treatment

Apart from the above first-aid procedures, full cardiopulmonary resuscitation may be required. Treatment may be necessary for the cardiovascular shock and convulsions. Sea snake antivenom should be used cautiously in serious cases (those with evidence of envenomation). Each ampule contains 200 μg. Care must be taken to administer it strictly in accordance with the directions in the brochure. The antivenom can be dangerous to allergy-prone patients. Emergency precautions for anaphylactic shock (including subcutaneous adrenaline/epinephrine) are required, and most practitioners employ prophylactic treatment against an allergic reaction (e.g., epinephrine, antihistamines) prior to the antivenom administration. The sea snake antivenom is made of separate antivenoms, each with a specific action. Unfortunately, although it does counter the 12 most common venoms, some may not be affected. The antivenom usually retains its potency for at least 8 years, but this varies with storage procedures. Tiger Snake or Polyvalent Land Snake antivenom can be used if the sea snake antivenom is unavailable, although their value has yet to be established.

Fluid and electrolyte balance must be corrected, and acute renal failure is usually

obvious from the oliguria, elevated blood urea, and electrolyte changes. Hemodialysis may result in a dramatic improvement in the muscular paralysis and in the general clinical condition. The acute renal tubular necrosis and the myonecrosis are considered temporary if life can be maintained.

Patients bitten by sea snakes should be hospitalized for 24 hours because of the delay in symptom development. Sedatives may be required, and it is reasonable to administer anxiolytics as required, sedating the patient without interfering significantly with respiration.

Fish Stings

GENERAL

Many fish have spines and a venom apparatus, usually for protection and occasionally for incapacitating prey. Spines may be concealed, only becoming obvious when in use (e.g., stonefish), or may be highlighted as an apparent warning to predators (e.g., butterfly cod or firefish).

Some fish envenomations have resulted in death, especially by the stonefish and stingray. These animals are described separately. Others, such as the scorpionfish and firefish (family Scorpaenidae), catfish (family Plotosidae and Ariidae), and stargazers (family Uranoscopidae), have also been responsible for occasional deaths in humans. Weeverfish (family Trachinidae), toadfish (family Batrachoididae), rabbitfish (family Siganidae), and some species of leatherbacks (family Carangindae) are also thought to have a potentially serious venom. Even some sharks (family Heterodontidae) have spines and venom apparatus.

As a general rule, fish that have been damaged—such as those in fishing nets—cause fewer problems clinically, probably because some of the envenomation system may have been triggered. Wounds that bleed profusely are less likely to be associated with intense symptoms. Some spines are inexplicably not associated with venom sacs and therefore produce few symptoms. Other fish may produce injury by knifelike spines that may or may not result in envenomation, e.g., old wife fish (family Enoplosidae), surgeonfish and unicornfish (family Acanthuridae), and ratfish (family Chimaeridae). In many cases, the slime that exists on the spines may contribute to symptoms and to subsequent infections as much as the possible venom. Identification of the species of fish responsible for a wound is not always possible. Fortunately, the symptoms do not vary a great deal.

CLINICAL FEATURES

The first symptom is usually an immediate local pain that increases in intensity over the following few minutes. It may become excruciating, but pain from an average sting usually lessens after a few hours (more rapidly with a minor sting and longer with a major sting). Maritime folklore attempted to reassure victims with the adage that the pain will lessen "with the turn of the tide."

The puncture wound is anesthetized, but the surrounding area is hypersensitive. Pain and tenderness in the regional lymph glands may extend even more centrally. Locally, the appearance is of one or more puncture wounds, with an inflamed and sometimes cyanotic zone. Surrounding the cyanotic zone is an area that is pale and swollen, with pitting edema. Generalized symptoms are sometimes severe. The patient is often very distressed by the degree of pain, which is disproportionate to the clinical signs. This distress can develop into a delirious state. Malaise, nausea, vomiting, and sweating may be associated with temperature elevation and leukocytosis. Occasionally, a cardiovascular shock state may supervene and lead to death. Respiratory distress may develop in severe cases. Chronic localized inflammation, edema, necrosis, and severe disability may continue for many months. This is usually due to marine infections, a foreign body reaction, or venom effects. This is especially likely if first aid and medical treatment have been inadequate or delayed.

TREATMENT

First Aid

The patient should be laid down and reassured. The affected area should be rested in an elevated position. Because these fish toxins are usually heat-labile, arrangements can then be made to immerse the wound in hot (up to 45°C) water for 30 to 90 minutes or until the pain no longer recurs. Unaffected skin as well as the wound should be immersed to avoid scalding. If the area

cannot be immersed, as on the head or body, hot packs may be applied. The total duration of the hot water immersion depends on the symptoms. If the injury site is removed and the pain recurs, it should be reimmersed. The wound should be thoroughly irrigated (preferably with isotonic saline) and cleaned after the immediate treatment is no longer required. As an alternative, if other methods are not available and if the therapist is prepared to risk any legal repercussions, a small incision can be made across the wound and parallel to the long axis of the limb to encourage mild bleeding and to relieve pain. A ligature or tourniquet is contraindicated. Local vasoconstriction is already a hazard to tissue vascularity without aggravating it with further circulatory restriction.

Figure 15–7. Stonefish *(Scorpaenea plumieri).* This species is prevalent along the Atlantic coast from Massachusetts to Brazil. They are very difficult to locate because of their perfect camouflage. (Photo courtesy of Paul Cianci, MD.)

Medical Treatment

Medical treatment includes first aid as described. If injected through the puncture wound, a local anesthetic, e.g., 5 to 10 mL lidocaine 1% without adrenaline (epinephrine), affords considerable relief. It may need to be repeated frequently, possibly within the hour. Local or regional anesthetic blocks may also be of value. Treatment may be needed for generalized symptoms of cardiogenic shock or respiratory depression. Systemic analgesics or narcotics are rarely needed, although they may be of value in severe cases.

Local cleansing and debridement of the wound, with removal of any broken spines or their integuments, is best followed by the application of a local antibiotic such as neomycin or bacitracin. Tetanus prophylaxis may be indicated if there is necrotic tissue or if the wound has been contaminated. If the stings are severe, they can mimic the lesions described under the headings of stonefish or stingray. The treatment sections of these injuries should be referred to because the principles (other than the use of antivenom) apply generally to all fish stings. Cellulitis, abscesses, and osteomyelitis were not rare in the pre-antibiotic era.

Stonefish

General

Perhaps the most venomous fish known, stonefish (Fig. 15–7) inhabit the whole tropical Indo-Pacific region. Many species similar to *Synanceja verrucosa* and *S. trachynis* are found in other tropical areas. Some of the Scorpaenidae, such as the spotted scorpionfish of the Caribbean, probably have comparable toxicity.[7]

This fish grows to about 30 cm in length. It lies dormant in shallow waters, buried in mud, coral, or rocks, and is practically indistinguishable from the surroundings. It can catch a small passing fish by sucking it into its gaping mouth. The 13 dorsal spines, capable of piercing wet suit booties, sneakers and skin, become erect when the fish is disturbed. Apart from the tip of the spine, the fish is covered by loose skin or integument. When pressure is applied, two venom glands discharge along ducts on each spine into the penetrated wound. Each spine has 5 to 10 mg of venom that can be neutralized by 1 mL of antivenom produced by the Australian Commonwealth Serum Laboratories. Occasionally, a stonefish spine is associated with no venom. It is thought that the venom is regenerated very slowly, if at all. The fish may live for many hours out of the water.

The venom is an unstable protein, with a pH of 6.0 and a molecular weight of 150,000. It produces an intense vasoconstriction and therefore tends to localize itself. It is destroyed by heat (2 min at 50°C), alkalis and acids (pH > 9 or < 4), potassium permanganate, and Congo red. The toxin is a myotoxin that acts on skeletal, involuntary, and cardiac muscles, blocking conduction in these tissues. It releases acetylcholine,

substance P, and cyclooxygenase. This results in a muscular paralysis, respiratory depression, peripheral vasodilation, shock, and cardiac arrest. The toxin also can produce cardiac arrhythmias.

CLINICAL FEATURES

Whether local or general symptoms predominate seems to depend on many factors, such as the geographic locality, number of spines involved, depth of spine penetration, protective covering, previous sting, and first aid treatment.

Local

Immediate pain is noted. This increases in severity over the ensuing 10 min or more. The pain, which is excruciating, may be sufficient in some cases to cause unconsciousness and thus drowning. Sometimes the pain comes in waves, a few minutes apart. Ischemia of the area is followed by cyanosis, which is probably due to local circulatory stasis. The area becomes swollen and edematous, often hot, with numbness in the center and extreme tenderness around the periphery. The edema and swelling may be gross, extending up the limb. Paralysis of the adjacent muscles is said to immobilize the limb, as may pain. The pain is likely to spread proximally to the regional lymph glands, e.g., in the axilla or groin. Both the pain and the other signs of inflammation may last for many days; delayed healing, necrosis, and ulceration may persist for many months. Swelling can likewise continue, although to a gradually lessening degree. These long-term sequelae are not as common in patients treated correctly in the first few days with antitoxin, debridement, cleansing, and local antisepsis.

General

Signs of mild cardiovascular collapse are not uncommon. Pallor, gross sweating, hypotension, and syncope on standing may be present. Respiratory failure may be due to pulmonary edema, depression of the respiratory center, cardiac failure, paralysis of the respiratory musculature, or a combination thereof. Bradycardia, cardiac arrhythmias, and arrest are also possible. Malaise, exhaustion, fever, and shivering may progress to delirium, incoordination, generalized paralysis, convulsions, and death. Convalescence may take many months and may be characterized by periods of malaise and nausea.

TREATMENT

First Aid

See the earlier discussion of fish stings.

Medical Treatment

Medical treatment depends on the site and severity of the symptoms. A local anesthetic agent without adrenaline, infiltrated into and around the wound, is the treatment of choice, especially if administered early. It may also remove the pain in the regional lymphatic area. A repeat injection will probably be needed and often reduces central pains (probably lymphatic in origin). Systemic analgesics and narcotics are seldom indicated or useful, although intravenous narcotics are sometimes used.

Elevate the affected limb to reduce pain and swelling and apply local antibiotics to prevent secondary infection. After the initial resuscitation and analgesia have been effected, débridement of necrotic tissue must be considered if there is any significant tissue damage or embedding of integument or spine. Otherwise, both local and generalized symptoms can continue for many months. Even when treatment has been inadequate or delayed, surgical excision of the damaged area may be necessary to reduce symptoms and hasten recovery. Ultrasound or another imaging technique is used to localize foreign bodies, although they are not excluded by a negative result.

Stonefish antivenom may be administered. One mL neutralizes 10 mg of venom (i.e., the venom from one spine). Initially, 2 mL of antivenom is given intramuscularly, although in severe cases the intravenous route can be used. Further doses can be given if required, but antivenom should never be given to patients with horse serum allergy. Antivenom should be stored between 0° and 5°C but not frozen, protected from light, and used immediately on opening. Tetanus prophylaxis is sometimes needed, and systemic antibiotics may be used because secondary infection is likely.

Appropriate resuscitation techniques may have to be applied. These include external cardiac massage and defibrillation and endotracheal intubation with controlled respiration. Monitoring procedures should include records of clinical state (pulse, respiration), blood pressure, central venous or pulmonary pressure, electrocardiogram, arterial P_{O_2}, P_{CO_2}, and pH. Clinical complications of bulbar paralysis should be treated as they arise.

PREVENTION

Wear thick-soled shoes when in danger areas. Be particularly careful on coral reefs and while entering or leaving boats. A stonefish sting is said to confer some degree of immunity for future episodes.

Stingray

GENERAL

Stingrays (Fig. 15–8) are found extensively from the tropics to the temperate regions. They are bottom dwellers; their flat bodies are often submerged in sand and only detectable by a protruding eye or two, a piece of tail, or the spiracles showing above an elevated disc of sand or mud.

Damage from the spine may cause death either from physical trauma, such as the penetration of the body cavities (pleural, pericardial, or peritoneal), or from the venom of the spine.[8, 9] In Australia, deaths tend to be from the former; in the United States, venom-related death is more likely. In the United States, stingrays are said to cause some 1500 injuries per year.

The stingray is not aggressive but can protect itself against intruders. It buries itself in sea or riverbeds; an unwary wading victim may step on its dorsal surface or a diver may descend over it. The stingray swings its tail upward and forward, either producing swordlike lacerations or driving the spine into the limb (especially the ankle) or body of the victim. An integument over the serrated spine is ruptured. Venom escapes and passes along the ventrolateral grooves into the perforated wound. Extraction of the saw-shaped spine results in further tissue damage due to the serrations and retropointed barbs and may leave spine or sheath within the wound.

The venom is a protein (molecular weight > 100,000) that is heat-labile and water-soluble, and it has an intravenous median lethal dose of 28 mg/kg body weight. Low concentrations may cause electrocardiographic changes (an increased P-R interval) associated with bradycardia. A first-degree atrioventricular block may occur with mild hypotension. Larger doses produce vasoconstriction, second- and third-degree atrioventricular blocks, and signs of cardiac ischemia. Most cardiac changes are reversible within 24 hours. Some degree of respiratory depression is noted with greater amounts of venom. This is probably secondary to the neurotoxic effect of the venom on the medullary centers. Convulsions may also occur.

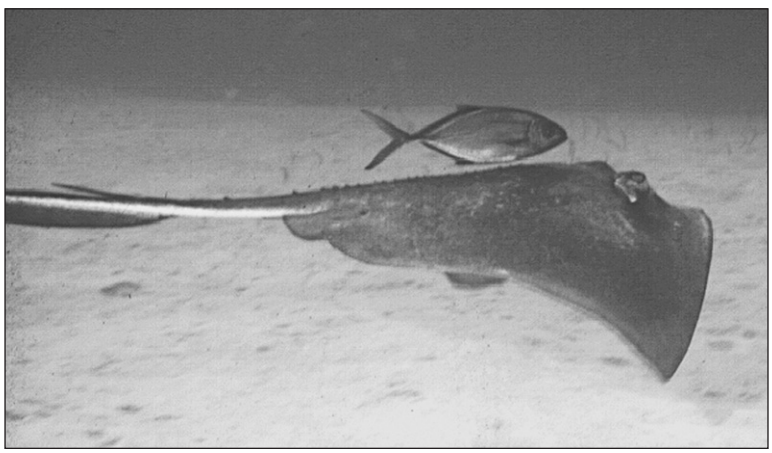


Figure 15–8. Southern stingray with hovering bar jack. Note the visible barb protruding above the tail. The barb can be forcefully driven into a diver's extremity by a powerful forward whip of the tail, but this is rare unless there is contact with the ray's dorsal surface. (Photo courtesy of Alfred A. Bove, MD, PhD.)

CLINICAL FEATURES

LOCAL

Pain is usually immediate and is the predominant symptom, increasing over 1 to 2 hours and easing after 6 to 10 hours. However, it may persist for some days. The area is swollen and pale, with a bluish rim. It is several centimeters in width and spreads around the wound after an hour or two. The pain may be constant, pulsating, or lancinating. Bleeding may be profuse, and deaths have resulted from blood loss. In lesser amounts, bleeding may relieve the pain. A mucoid secretion may follow. Integument from the spine may be visible in the wound, which may gape and extend for a few centimeters in length.

Aggravation of pain within days or weeks may be due to secondary infection. Local necrosis, ulceration, and secondary infection are common and, if unchecked, may incapacitate the patient for many months. In earlier years, amputation was performed. Osteomyelitis in the underlying bone has been reported. More frequently, recurrence of local symptoms within a week or two implies retention of a foreign body (integument or spine). Ultrasound and other imaging techniques may identify the cause, but a negative finding does not exclude a foreign body.

GENERAL

The following manifestations have been noted: anorexia, nausea, vomiting, diarrhea, frequent micturition, and salivation. Pain extends centrally, to the area of lymphatic drainage. Muscular cramps, tremors, and tonic paralysis may occur in the affected limb or may be more generalized. Fainting, palpitation, hypotension, cardiac irregularities (conduction abnormalities, blocks), and ischemia are possible. Respiratory depression may occur, with difficulty in breathing, cough, and pain on inspiration. Other features include nocturnal fever with copious sweating, nervousness, confusion, or delirium. Fatalities are possible, especially if the stingray's spine perforates the pericardial, peritoneal, or pleural cavities.

The initial symptoms last from hours to days but may recur or persist for weeks or months after the injury, even though the wound may have closed over. These include a dull ache over the area and a swelling that may develop under the influence of gravity. Thus, an ankle may become painful and swollen after standing or walking. Resting with the foot elevated may alleviate pain. A radiograph or ultrasound image should be used to demonstrate a foreign body (stingray spine) in the soft tissues.

TREATMENT

FIRST AID

See the earlier discussion of fish stings. Hemostasis may be required if the spine has severed an arterial supply. If the spine or integument is still present, it should be gently extracted. Following pain relief, the limb should be immobilized in an elevated position and covered with a clean dressing, such as an unused newspaper. The patient's state may become far more serious than it first appears.

MEDICAL TREATMENT

Pain is relieved via infiltration of local anesthetics without adrenaline (epinephrine) into and around the wound or by regional block. Systemic analgesia may be required. X-ray or ultrasound imaging may demonstrate foreign bodies and bone injury. The basic physiologic signs (e.g., temperature, pulse, respiration, blood pressure, central venous pressure, urine output), serum electrolytes, electroencephalogram, and electrocardiogram are monitored as indicated. Broad-spectrum antibiotics (e.g., doxycycline and local application of neomycin) are used at an early stage. Symptomatic treatment is given for the clinical features. Tetanus prophylaxis is indicated if wounds are necrotic or contaminated.

Wound cleansing, débridement, and reconstruction, if required, are performed as early as permitted by the patient's general state. Serious cases, especially those involving the major body cavities, require the removal of all necrotic tissue and foreign bodies; otherwise, the wounds will break down during the second week. Bacterial infection may complicate wound healing.[10]

In chronic cases, wherein foreign body reaction predominates, damaged tissue and fibrotic nodules can be removed if the area is surgically explored; rapid recovery

follows this minor surgery. The nodule may be due to a tissue reaction from a piece of spine or sheath. Antibiotics do not help at this stage.

PREVENTION

Divers, swimmers, and waders are advised to shuffle their feet when walking in the water. This gives the ray time to remove itself, which it cannot do if a foot is on its dorsum. Although wearing rubber boots decreases the severity of the sting, the spine penetrates most protective material. Care is needed when handling fishing nets. Divers should not swim within a meter of the seabed because this stirs up both silt and stingrays.

Coelenterates

GENERAL

Coelenterata is a phylum of 9000 species containing jellyfish, sea anemones, fire coral, stinging hydroids, and so on. It constitutes one of the lowest orders of the animal kingdom and has members that are often dissimilar in appearance and mobility. Although many of these creatures appear flowerlike, all are carnivorous animals. The common factor among the coelenterates is the development of nematocysts, or stinging capsules. They are like coiled springs held within an envelope, the shape of which varies with the species. The tentacles that carry the nematocysts adhere to the victim by either sticky mucus or specialized nematocysts with penetrating spines. The triggering mechanism responsible for the firing of the nematocyst is thought to be initiated by many factors (e.g., trauma or the absorption of water into the nematocyst capsule, causing it to swell).

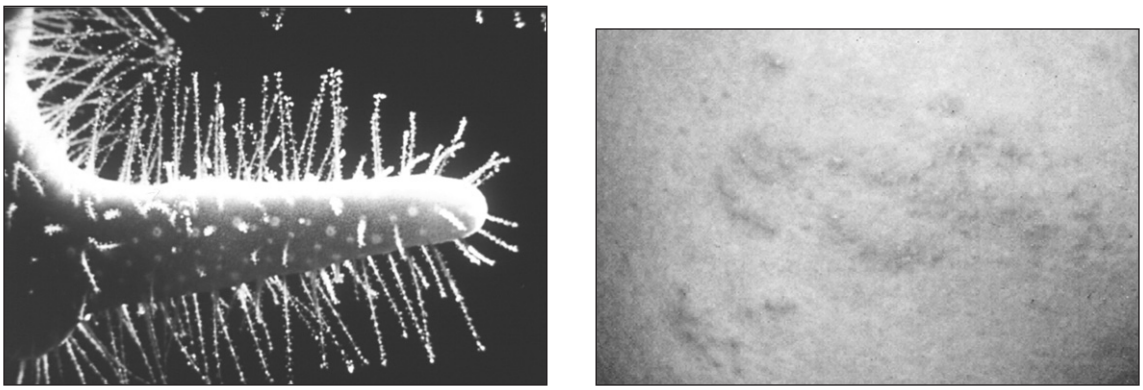
The function of the nematocysts is to rapidly incapacitate and then retain prey, which is eaten. The pattern of nematocyst stings may be characteristic, depending on their aggregation on the tentacle of the coelenterate and the morphology of the tentacles. Thus, the Portuguese man-o-war usually produces a single long strap with small blisters along it, whereas the box jellyfish has multiple long red lines, often with the tentacle adherent. The nematocysts of different types of coelenterates may be identifiable; therefore, a skin scraping is of value in the differential diagnosis of marine stings. Stinging hydroids (Fig. 15–9) and fire coral (Fig. 15–10*A*), being nonmobile, sting only when touched by the diver.

Figure 15–9. Stinging hydroids from the Sea of Cortes. Fine fronds contain hundreds of nematocysts. (Photo by Bonnie J. Cardone, with permission from Peterson Publishing, Los Angeles.)

CLINICAL FEATURES

Clinical factors may vary from a mild itch locally to severe systemic and lethal reactions.[11–13] The local symptoms vary from a prickly or stinging sensation, developing immediately on contact, to a burning or throbbing pain. The intensity increases over 10 minutes or so and the red inflamed area (Fig. 15–10*B*) may develop blisters or even necrotic ulcers in severe cases. The pain may spread centrally, with lymphadenopathy, and may be associated with abdominal pain and chest pain.

Generalized symptoms include fever, increased secretions, gastrointestinal disorders, cardiovascular failure, respiratory distress, and a toxic-confusional state.



A **B**

Figure 15–10. *A,* Close-up photograph of a branch of fire coral. The fine filaments contain hundreds of nematocysts that attach to the skin when contact is made. *B,* Typical erythematous rash that results from contact. (Photos courtesy of Alfred A. Bove, MD, PhD.)

The intensity of both local and generalized manifestations of coelenterate stinging may vary according to the following:

- The species involved (the box jellyfish is often lethal, whereas many other jellyfish can be handled with impunity)
- The extent of the area involved
- The maturity of the animal
- The body weight of the subject, with the generalized symptoms being more severe in children than in adults
- The thickness of the skin in contact
- Individual idiosyncrasies such as allergic reactions and preexisting cardiorespiratory or other disease
- The use of protective materials, such as Lycra suits and water-resistant sunscreens

Many complications may develop after coelenterate stings. These include anaphylaxis and allergies, vasculopathy, neuropathy, myopathy, lymphadenopathy and lymphedema, hematologic complications, and the Irukandji syndrome.

Because coelenterates of the Cubomedusae family (box jellyfish) are the most dangerous, they are dealt with in detail. *Physalia*, or Portuguese man-of-war, is so widespread that it is also dealt with specifically.

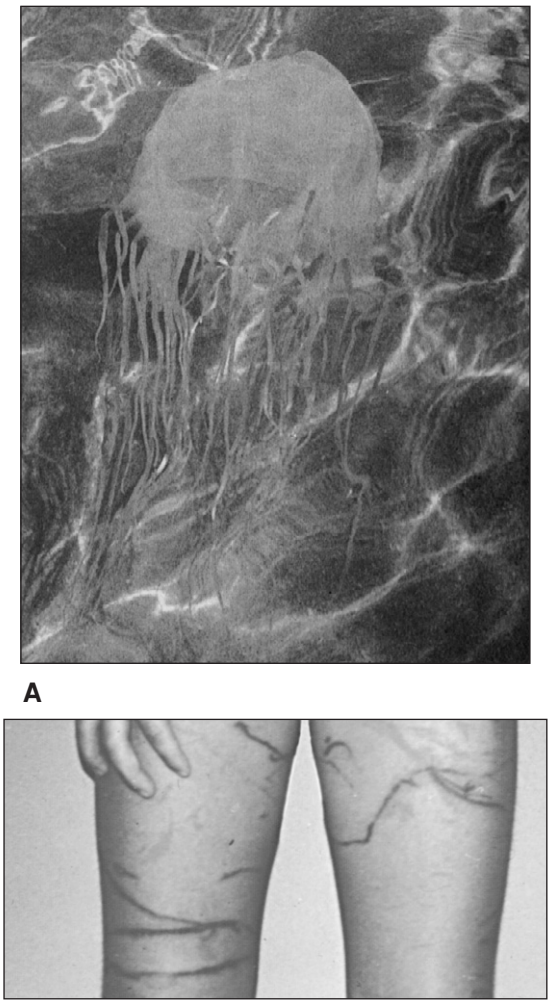
Cubomedusae (Box Jellyfish, Sea Wasp)

GENERAL

These animals (*Chironex*, *Chiropsalmus,* and similar species) are most plentiful in the warm waters of the Indo-Pacific region, with 80-plus documented fatalities in the waters off northern Australia (from November to April) and about 50 deaths a year in the Philippines. Cases are frequent but not well documented in Southeast Asia and Melanesia. *Chiropsalmus* has also caused deaths in the Gulf of Mexico. These animals have caused deaths and associated disruption of Japanese tourism in Okinawa. Other species, such as Morbakka, Carybdea, and Tamoya from the Gulf of Oman, are found in the equatorial waters of the Indo-Pacific.

The *Chironex* box jellyfish (Fig. 15–11*A*) is said to be the most venomous.[11] It is especially dangerous to children and patients with cardiorespiratory disorders (e.g., asthmatics). Its box-shaped body can measure 20 cm along each side and has up to 15 tentacles measuring up to 3 m in length on each side of its four pedalia (arms). *Chiropsalmus* is smaller, about 7 cm across, with fewer and shorter tentacles, and is less potent (but still potentially lethal).

The severity of the sting increases with the size of the animal, the extent of contact with the victim, and the delicacy of the victim's skin. Deaths have occurred from contact with as little as 6 to 7 m of tentacle. Adjacent swimmers may also be affected to a variable degree. The tentacles tend to adhere with a sticky jellylike substance, but bystanders can usually remove the tentacles because the thick skin on the palms of the hands affords ample protection. This protection is not always complete, and stinging can occur even through surgical gloves. The venom is made up of at least three different



A

B

Figure 15–11. *A,* Box jellyfish or sea wasp *(Chironex fleckeri),* perhaps the deadliest animal in the sea. An inhabitant of the estuarine areas on the northern coast of Australia, where the turbid water makes detection difficult. Death has been reported within minutes of envenomation. (Photo courtesy of Paul Cianci, MD.) *B,* Typical scarring from contact with the tentacles of *Chironex.* (Photo courtesy of Keven Reed, MD.)

fractions, one with a molecular weight of approximately 75,000 and one with a molecular weight of 150,000. The lethal, dermatonecrotic, and hemolytic fractions are specific antigens for each species, but some cross-immunity seems to exist, at least for *Chironex* and *Chiropsalmus.*

The effects on the cardiovascular system include an initial rise in arterial pressure, which is followed by hypotensive/hypertensive oscillations. This condition is probably due to interference with vasomotor reflex feedback systems. The hypotensive states are related to bradycardia; cardiac irregularities (especially delay in atrioventricular conduction) are due to cardiotoxicity, baroreceptor stimulation, brain-state depression, or a combination thereof. Ventricular fibrillation or asystole precedes cerebral death.

CLINICAL FEATURES

The patient usually screams as a result of the excruciating pain that occurs immediately on contact and increases in intensity, often coming in waves. The patient then claws at the adherent tentacles (whitish strings surrounded by transparent jelly). The patient may become confused, act irrationally, or lose consciousness and hence may drown.

Local

Multiple interlacing whiplash lines—red, purple, or brown, 0.5 cm wide—develop within seconds. The markings are in a beaded or ladder pattern (transverse wheals) and are quite characteristic. These acute changes last for hours. If death occurs, the skin markings fade. If the patient survives, the red, swollen skin may develop large wheals, and, after 7 to 10 days, necrosis and ulceration develop over the area of contact. The skin lesions may take many months to heal if deep ulceration occurs. Itching may also be troublesome and recurrent. Pigmentation and scarring at the site of these lesions may be permanent (Fig. 15–11*B*).

General

Excruciating pain dominates the clinical picture; impairment of the conscious state may proceed to coma and death. The pain diminishes in 4 to 12 hours. Amnesia occurs for most of the incident following the sting. If death occurs, it usually does so within the first 10 minutes; survival is likely after the first hour.

Cardiovascular effects dominate the generalized manifestations. The patient may develop cardiac shock, with a disturbance of consciousness. Hypotension, tachycardia, and a raised venous pressure may also occur. It is also possible that the clinical state will oscillate within minutes from episodes of hypertension, tachycardia, rapid respirations, and normal venous pressure to those

of hypotension, bradycardia, apnea, and elevated venous pressure. The oscillation may give a false impression of improvement just prior to the patient's death.

Respiratory distress, pulmonary congestion, edema, and cyanosis may be due to the cardiac effects or to a direct midbrain depression. Paralysis and abdominal pains may occur. Malaise and restlessness may persist, with physical convalescence requiring up to a week. Irritability and difficulty with psychological adjustment may take weeks or months to disappear. Immunity to the sting is thought to occur following repeated and recent contacts.

TREATMENT

First Aid

Prevent drowning. Apply copious quantities of vinegar or mild denaturing agents (e.g., stale wine or carbonated beverages if vinegar is not available) to reduce the likelihood of discharge of the nematocysts. The tentacles should be removed as rapidly and gently as possible. Cardiopulmonary resuscitation may be needed and may need to be repeated on a number of occasions.

Medical Treatment

Repeat the traditional vinegar irrigation, as it is thought to have some prophylactic value. Local applications also may include lidocaine or other local anesthetic ointment and local steroid, later. Analgesics include morphine (15 mg) or meperidine (Demerol—100 mg) administered intravenously; these may also protect against shock. Some authors think that pressure bandages delay systemic spread of the toxin,[14] but this therapy is not universally recommended (see Table 15–5).

Intravenous steroids may be administered every 2 hours if needed. Local steroid preparations are valuable for treating local manifestations such as swelling, pain, itching, and urticaria. Intermittent positive-pressure respiration, possibly with oxygen, replaces mouth-to-mouth artificial respiration, when needed. This requires constant attention because of the varying degree of respiratory depression. General anesthesia with endotracheal intubation and controlled respiration are of value if analgesia cannot otherwise be obtained.

Anxiolytics, tranquilizers, or other sedatives may be of value after the immediate resuscitation because they assist in calming the patient without causing significant respiratory depression. Other drugs may be used but are unproved in this clinical disorder. They include noradrenaline (Levophed) or isoproterenol (Isuprel) drips. Electrocardiogram monitoring is indicated, as are measurements of pulse rate, blood pressure, central venous or pulmonary pressure, respiratory rate, arterial gases, and pH levels. External cardiac massage and defibrillation are given if required.

Box jellyfish antivenom has been developed by the Australian Commonwealth Serum Laboratories and is derived from the serum of hyperimmunized sheep. Twenty thousand units may be sufficient to control the effects of a moderate sting on adults. This may need to be increased to 100,000 units for a child with massive injury.

PREVENTION

Prevention includes the wearing of adequate protective clothing (e.g., overalls, Lycra suits, wet suits, body stockings). Swimming or wading should be restricted to the safe months of the year. Care is especially needed on cloudy days toward the end of the hot season. Dragging a section of a beach with a 2.5 cm mesh has been used, but not always successfully, to clear the area for bathing.

***Physalia* (Portuguese Man-o-War)**

GENERAL

There was one reported death in Florida from *Physalia*, and others have been claimed. Many cases have required resuscitation. The animal has a pneumatophore (a gas-filled transparent sac, usually blue), which can reach 15 cm in length and which allows it to drift at a 45-degree angle to the wind (Fig. 15–12). It floats on the surface of the water and trails many short frilled tentacles and one or more long "fishing" tentacles. The latter may extend for many meters and is responsible for most stings.

One toxin of *Physalia* is called *hypnotoxin* and is a peptide or protein material. It causes neurologic depression, affecting motor and

Figure 15–12. Pacific Portuguese man-o-war *(Physalia Utriculus).* This species is smaller than the Atlantic variety but equally potent. The float can attain a length of 13 cm, and tentacles may hang to a depth of 12 m. Care should be taken when walking the beach after on-shore storms because portions of the tentacles may have washed ashore. (Photo courtesy of Paul Cianci, MD.)

sensory areas, and has edema-producing properties. Respiratory depression occurs in envenomated animals and has been observed in human victims.

CLINICAL FEATURES

LOCAL

Initially, there is a sharp sting. This may be aggravated by pulling on the tentacle, rubbing the area, or applying fresh water. The sting rapidly increases to an intense ache that spreads to involve surrounding joints and then moves centrally. The axilla or groin may be affected, and the associated draining lymph glands become tender. The duration of the severe pain may range from a few minutes to hours. It is superseded by a dull ache that lasts a similar period.

The affected area develops a red line with small white punctate (pinpoint) lesions, and in severe cases a central weal or blister develops after the erythema. The wheals only last a few hours and look like a string of beads; the erythema clears within 24 hours. Rarely, ulceration, discoloration, and scarring occur.

GENERAL

General signs are not uncommon but rarely last longer than a day. The patient may be slightly shocked and may experience syncope, with a rapid pulse and hypotension. Generalized chills and muscle cramps may develop. Abdominal symptoms include nausea, pain, and vomiting.

Neurologic signs have been noted, with the patient showing irritability and confusion. Death is possible if the patient experiences respiratory depression.

TREATMENT

FIRST AID

First aid includes rescuing the victim from the water, laying the victim down, and giving reassurance. Any technique used for burn treatment may give some relief (cold packs, ice, and local antiburn sprays). Many household substances may be of value, but alcohol can aggravate the condition. Cold tea, carbonated beverages, and stale wine have all been suggested. The best application for relief of pain probably is a local anesthetic agent (e.g., lidocaine 5%). The tentacle should be removed as gently as possible to reduce the likelihood of further nematocyst discharge.

MEDICAL TREATMENT

Local anesthetic (topical) is often effective as a pain reliever and is usually superior to other applications. In a series of stingings performed on volunteers, both immediately and after a 10-minute delay, the following results were obtained. Lidocaine 5% ointment was superior to both Ultralan 0.5% (a steroid) and lidocaine gel, and both of these were better than Benadryl cream (an antihistamine). Methylated spirits was the least effective. Commercial preparations were effective for only a short time.

If the eye is affected, it would be logical to apply a nonaqueous local anesthetic solution (e.g., cocaine eye drops or ointment) followed by a steroid ointment. Aqueous drops should be avoided. Homatropine and cocaine drops may be instilled later, although some tend to use a steroid antibiotic eye ointment combination. Antibiotic is used to ensure that the corneal ulcers do not become infected. Ophthalmologic advice is required for treatment of the specific complications, such as conjunctivitis, keratitis, iritis, and glaucoma.

Despite the paucity of documented deaths, it would be wise to monitor severely

affected cases, as one does with *Chironex* stings, and to give cardiovascular and respiratory assistance if needed. Tranquilizers or anxiolytics may be indicated in distressed patients.

It is possible that allergy-prone patients are more susceptible to the *Physalia* and other coelenterates. The use of intravenous steroids may be indicated in these persons. Severe itching may develop within a few days, but this responds to steroid ointments.

Irukandji Syndrome

An array of systemic symptoms, known as the *Irukandji syndrome,* may follow an almost negligible sting from a variety of jellyfish.[15]

The name Irukandji was given after a local aboriginal tribe living near Cairns, Australia, where the injury was first described. The cause was a small box jellyfish, now known as *Carukia barnesi* (Fig. 15–13).

This animal is rarely observed by the victim, though the stinging may occur near the surface and in shallow water. It is classically a small box jellyfish with a transparent body about 1 to 2 cm long and with four tentacles varying from 5 cm to 1 m in length, depending on the degree of contraction. Nematocysts, appearing as clumps of minute red dots, are distributed over the body and tentacles. The delayed injury is proportional to the duration, extent, and location of the sting. Similar clinical symptoms may accompany stings from other coelenterates. Symptoms have been confused with decompression sickness.

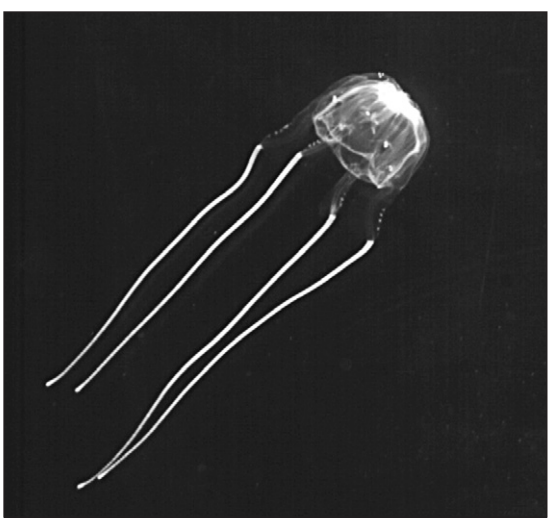


Figure 15–13. Irukandji box jellyfish *(Carukia barnesi).* This coelenterate is usually about 2 to 3 cm in diameter, with tentacles that can reach up to 1 m in length. It is transparent and often invisible in the sea. (Photo courtesy of Robert Hartwick, MD.)

CLINICAL FEATURES

Local

A few seconds after contact, a stinging sensation is felt; this increases in intensity for a few minutes and diminishes during the next half-hour. It is usually sufficient to cause children to cry and adults to leave the water. It may recur at the commencement of the generalized symptoms but is overshadowed by them.

A red reaction, 5 to 7 cm, surrounds the area of contact within 5 minutes. Small papules (pimples) appear and reach their maximum in 20 minutes before subsiding. "Kissing" lesions occur where the original skin lesion comes into contact with other skin (e.g., near joints). The red coloration can occasionally last up to 3 hours, and there is a dyshidrotic reaction (skin dry at first, with excessive sweating later) over the area. Occasionally, in severe cases, the area may remain swollen for many hours. There is usually a latent period of 5 to 120 minutes between contact and the development of generalized symptoms. The patient may not relate these symptoms to the local reaction unless specifically questioned about this.

General

Pain usually dominates the clinical presentation. Abdominal pains, often severe and associated with spasm and boardlike rigidity of the abdominal wall, often come in waves. Muscular aches such as cramps and dull boring pains occur, with increased tone and muscle tenderness on examination. This especially involves the spine but also involves hips, shoulders, limbs, and chest. Headache may also be severe. Profuse sweating, anxiety, and restlessness may develop, as may nausea and vomiting. Respiratory distress with coughing may occur, and grunts may precede exhalations. Pulmonary edema has been described, usually many hours after the stinging. There is often a marked increase in blood pressure and pulse rate, with possible arrhythmias and hemor-

rhages. Deaths have resulted, often from cerebral hemorrhage.

Later symptoms include numbness and tingling, itching, smarting eyes, sneezing, joint and nerve pains, weakness, rigors, dry mouth, and headache. Temperature usually remains normal, although there may be an increased pulse rate.

Symptoms diminish or cease within 4 to 12 hours. Occasionally, malaise and distress may persist and convalescence may take up to a week.

PREVENTION

Wear protective clothing (e.g., wet suits, Lycra). Once stingings have been reported, the water should be avoided.

TREATMENT

First Aid

It is currently thought that the copious use of locally applied vinegar for at least a minute may reduce subsequent discharge of nematocysts. The use of compression bandages has been recommended but is controversial. The worry is that these bandages may themselves traumatize the nematocysts and increase their discharge rate.

Medical Treatment

During the severe phase with abdominal pains, spasms, and coughing, intravenous narcotics may be needed and repeated as necessary. Promethazine, with an intravenous dose of 0.5 mg/kg to a maximum of 25 mg, not only reduces the symptoms of nausea and vomiting but also reduces the subsequent amount of narcotics required.[15] α-Adrenergic blockers have been recommended for the control of hypertension due to catecholamine release. Hydralazine has also been used. Other medications that have been used include anxiolytics, antihistamines, and anesthesia. General anesthesia with assisted respiration can be used if the conventional techniques prove insufficient.

Monitoring of fluid and electrolyte state, together with cardiorespiratory parameters, would seem indicated, especially if there are any respiratory symptoms. Pulmonary edema has been treated with intubation and controlled ventilation, high inspiratory oxygen, and positive end-expiratory pressure. If signs or symptoms of pulmonary edema develop (which may occur after 10 to 12 hours), then repeat the creatine kinase and admit the patient to a coronary intensive care unit. During the latter part of the illness, when only fleeting neuralgic and arthralgic symptoms predominate, simple analgesics may be effective.

Sea Bather's Eruption

Sea bather's eruption describes the erythematous rash found in bathers and divers who are exposed to the larvae of the thimble jellyfish.[16–18] Divers refer to the disorder as "sea lice." The larvae, which contain active nematocysts, become trapped under the bathing suit, diving skin, or wet suit and discharge into the skin along the edge of the garment (Fig. 15–14).

CLINICAL FEATURES

Bathers and divers demonstrate a painful, itchy rash on the skin under the edges of the bathing suit or diving dress. Dive masters and guides working in tropical waters, subjected to repeated exposures to the larvae, acquire allergies to the toxins and develop a complex skin eruption that is a combination of the direct effects of the sea nettle toxin on the skin and an allergic eczema.[18] The rash is erythematous with vesicles; pustules may indicate a secondary bacterial infection.

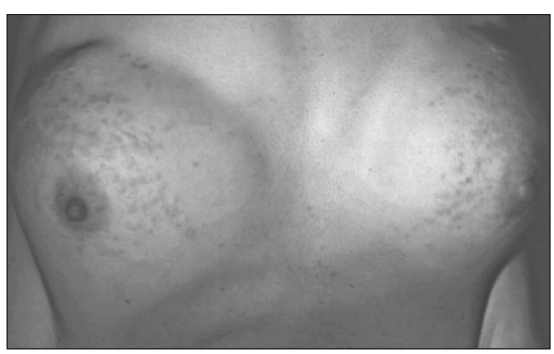


Figure 15–14. Sea bather's eruption. The macular erythematous rash is noted along the edges of the bathing suit, where the coelenterate larva become trapped and discharge nematocysts into the skin. (Photo courtesy of Bruce Miller, MD.)

TREATMENT

Topical steroids usually reverse the allergic reaction and lead to a cure. In some cases, the subject may be required to refrain from swimming or diving in affected waters for several weeks until the skin is clear. Topical antibacterial ointments should be used if secondary bacterial infection is suspected.

Cone Shells

GENERAL

Highly favored by shell collectors of the tropics and subtropics, these attractive snails, univalve molluscs (Fig. 15–15), have a proboscis that extends from the narrow end but that can reach most of the shell. Holding the shell even by the "big end" may not be safe and may court a sting with a resultant 25% mortality rate. The cone shell inhabits shallow waters, reefs, ponds, and rubble. It is usually up to 10 cm in size but may be larger. It has a siphon, sometimes ringed with orange, that detects its prey and may be the only part visible if the cone burrows under the sand. The proboscis, which delivers the coup de grace, carries up to 20 radular teeth (harpoons) that penetrate and inject venom into its prey, thus immobilizing the victim.

Probably only the fish-eating cones are dangerous to humans, but because these are difficult to distinguish at first sight, discretion on the part of shell collectors is recommended. The venom is composed of a variety of small peptides. One interferes with neuromuscular activity and elicits a sustained muscular contraction; others abolish nerve fiber excitability and summate with tubocurarine but are uninfluenced by eserine. The major effect appears to be directly on skeletal muscular activity. Children are especially vulnerable.

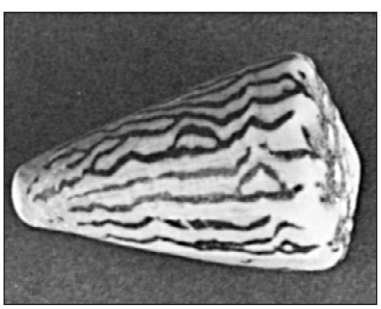


Figure 15–15. Cone shell *(Conus princeps).* The proboscis can reach almost to the large end of the shell. (Photo by Bonnie J. Cardone, with permission from Peterson Publishing, Los Angeles.)

CLINICAL FEATURES

Local

The initial puncture effects may vary from no pain to extreme agony and may be aggravated by salt water. The wound may become inflamed and swollen, sometimes white and ischemic, with a cyanotic area surrounding it, and it may be numb to touch.

General

Numbness and tingling may ascend from the sting to involve the whole body, especially the mouth and lips. This may take about 10 minutes to develop. Skeletal muscular paralysis may spread from the site of injury and may result in anything from mild weariness to complete flaccid paralysis. Difficulty with swallowing and speech may occur prior to total paralysis. Visual disturbances may include double and blurred vision (paralysis of voluntary muscles and pupillary reactions). These changes may take place within 10 to 30 minutes of the sting. Respiratory paralysis may dominate the clinical picture. This results in shallow rapid breathing and a cyanotic appearance, proceeding to apnea, unconsciousness, and death. Other cases are said to result in cardiac failure, although this is probably secondary to the respiratory paralysis. The extent of neurotoxic damage varies. Patients who survive are active and mobile within 24 hours. The sequelae and especially the local reaction may last a few weeks.

TREATMENT

First Aid without Paralysis

The patient should be rested and constantly reassured. The limb must be immobilized, and a pressure bandage may delay venom absorption.

First Aid with Paralysis

Mouth-to-mouth respiration may be needed. This may have to be continued for hours or

until medical facilities are accessed. This artificial respiration is the major contribution to saving the patient's life. External cardiac massage as well as mouth-to-mouth resuscitation are needed if patients have neither pulse nor respiration. Patients may be able to hear but not communicate and thus require reassurance. If patients are in shock, place them in a prone position with the feet elevated.

Medical Treatment

With respiratory paralysis, administer artificial respiration with intermittent positive pressure adequate to maintain normal Po_2, Pco_2, and pH levels of arterial blood. Endotracheal intubation prevents aspiration of vomitus and facilitates tracheobronchial toilet, when indicated. With total paralysis, the following regimen is needed: intravenous nutrition (nothing by mouth), eye toilet and protection, and attention to pressure areas. External cardiac massage, defibrillation, vasopressors, and so forth may be indicated by the clinical state and electrocardiogram monitor. Local anesthetic can be injected into the wound. Respiratory depressants, respiratory stimulants, and drugs used against neuromuscular blockade are not usually of value. Anti-cholinesterases may be of some benefit.

Blue-Ringed Octopus

GENERAL

This animal (*Octopus maculosa* or *Hapalochaena maculosa*) usually weighs 10 to 100 g and is currently found only in the Australasian and Central Indo-Pacific region. Its span, with tentacles extended, is 2 to 20 cm but is usually less than 10 cm. The octopus is found in rock pools at low tide. The color is yellowish brown with ringed markings on the tentacles and striations on the body. These markings change to a vivid iridescent blue when the animal is feeding or becomes angry, excited, disturbed, or hypoxic. The heavier specimens are more dangerous, and handling these attractive creatures has resulted in death within a few minutes. Many such incidents have probably escaped detection by the coroner. Autopsy features are nonspecific, and the bite fades after death.

The toxin (maculotoxin) is more potent than that of any land animal. Analysis of posterior-salivary extracts demonstrate a hyaluronidase and cephalotoxins of low molecular weight (<500). This has similar effects to tetrodotoxin (from puffer fish poisoning) and produces a temporary blockage of sodium channels in nerve tissue (see Chapter 16).

CLINICAL FEATURES

Local

Initially, the bite is usually painless and may thus go unnoticed.

General

A few minutes after the bite, a rapid, painless paralysis dominates the clinical picture, which progresses in the following order: abnormal sensations around mouth, neck, and head; nausea, vomiting, or both; dyspnea with rapid, shallow, and stertorous respirations leading to apnea, asphyxia, and cyanosis; visual disturbance (involvement of the extraocular eye muscles results in double vision, blurred vision, and ptosis, whereas intraocular paralysis results in a fixed dilated pupil); difficulty in speech and swallowing; and generalized weakness and incoordination progressing to complete paralysis.

The duration of paralysis is 4 to 12 hours, but the weakness and incoordination may persist for another day. The patient's conscious stage is initially normal, even though the patient may not be able to open the eyes or respond to the environment. The respiratory paralysis (causing hypoxia and hypercapnia) finally results in unconsciousness and then death, often within minutes of the commencement of symptoms, unless resuscitation is continued. Cardiovascular effects of hypotension and bradycardia occur in severe cases.

This clinical sequence may cease at any stage, that is, the effects may end with the local reaction or with a partial paralysis or may proceed to a complete paralysis and death. Less severe bites may result in generalized and local muscular contractions, which may continue intermittently for some hours. This occurs with a subparalytic dose. Other symptoms noted in mild cases include a lightheaded feeling, depersonalization, paresthesia, weakness, and exhaustion.

TREATMENT

First Aid before Paralysis

Immobilize the limb and apply a pressure bandage to reduce the absorption of venom. Rest patients, preferably on their side in case of vomiting, and do not leave them unattended. Obtain medical assistance and give reassurance. Do not remove the pressure bandage until full resuscitation facilities and personnel are present.

First Aid with Paralysis

Apply mouth-to-mouth respiration to ensure that the patient does not become cyanotic. Attention must be paid to cleaning the airway of vomitus, tongue obstruction, dentures, and so forth. If an airway is available, this should be inserted, but this is not essential. Artificial respiration may have to be continued for hours, until the patient reaches a hospital. If delay has occurred, then external cardiac massage may also be required. Reassure these patients, who can hear but not communicate, that they will be all right and that you understand their condition. Enlist medical aid but never leave patients unattended.

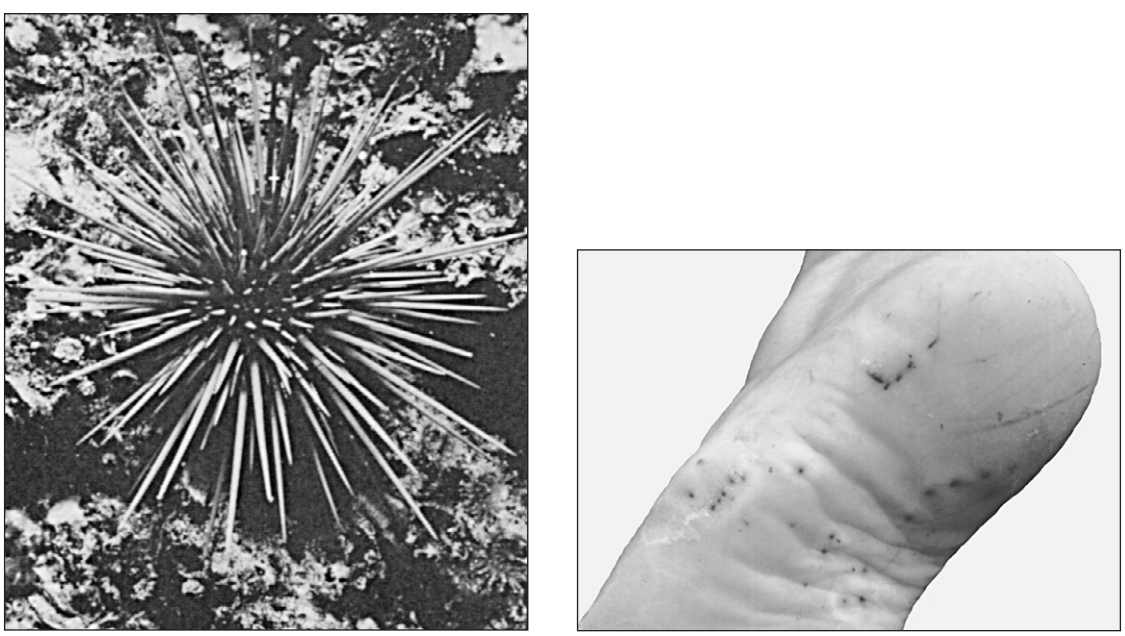
Medical Treatment

For respiratory paralysis, artificial respiration with intermittent positive pressure respiration is necessary to maintain normal P_{O_2}, P_{CO_2}, and pH levels of arterial blood. Endotracheal intubation also prevents aspiration of vomitus and facilitates tracheobronchial toilet, when indicated. Oral foods or fluids are contraindicated. Eye toilets and protection are needed. Respiratory stimulant may be of some value in the recovery phase.

Sea Urchins

GENERAL

Of the 600 species of sea urchins (Fig. 15–16*A*), approximately 80 are thought to be venomous or poisonous to humans. They belong to the phylum Echinodermata, named after the Porcupine (Echinos) because of its many-spined appearance. In some, such as *Diadema setosum*, the long-spined or Black Sea urchin, the damage is mainly done by the breaking off of the sharp brittle spines after they have penetrated the diver's skin (Fig. 15–16*B*). Sometimes the spines disappear within a few days, but in other cases



A **B**

Figure 15–16. *A,* Red Sea urchin *(Strongylocentrotus franciscus).* The long spines break off readily in the skin when contacted. (Photo by Bonnie J. Cardone, with permission from Peterson Publishing, Los Angeles.) *B,* Plantar surface of a foot with numerous sea urchin spines embedded. An area of pallor surrounds some spines. (Photo courtesy of Peter R. Lynch, PhD.)

they become encrusted and may remain for many months to emerge at sites distant from the original wound. The spines are covered by a black pigment, which can then be mistaken for the actual spine during its removal.

The most potent sea urchins are the Toxopneustidae, which have short thick spines poking through an array of flowerlike pedicellariae. Deaths have been reported from this family, and the venom is thought to be a dialyzable acetylcholine-like substance.

The starfish *Acanthaster planci* (crown-of-thorns) can also cause damage by the spines piercing the skin, but these seem to have a far more inflammatory action suggestive of a venom. Injuries from the crown-of-thorns have been more commonly reported since divers attempted to eradicate them from reefs. A characteristic general symptom is nausea or vomiting (sometimes also experienced with other urchin injuries). It is proclaimed that the crown-of-thorns destroyed reefs at a rate of 5 km per month.

TREATMENT

The long spines tend to break easily and therefore need to be removed vertically, without any horizontal movement. A local anesthetic may be required if surgical extraction is contemplated. Drawing pastes such as magnesium sulfate have been used. Some find relief with the use of heat, and others have removed the spines by the use of a snakebite suction cup.

One technique that would be described as barbaric, were it not for the fact that it seems to work, is to apply extra trauma and movement to the area in order to break up the spines within the tissue. Of course, this should be limited to those in nondangerous areas and if surgery is not contemplated. It does seem as if, in these cases, activity is more beneficial than rest and immobilization. With the latter, the limb tends to swell and become more painful. Because the spines are proteinaceous, they are usually absorbed.

Attempts at surgical removal are sometimes necessary, especially if the spine is in a potentially dangerous area, such as penetrating a joint, or if symptoms persist. Under these conditions, an operating microscope is of value.

The use of hot water baths and local anesthetic as treatment of the crown-of-thorns sting seems to be of some value in early stages. Treatment of Toxopneustidae stings must be based on general medical principles.

Sponges

GENERAL

Sponges are sedentary animals that require some defense from mobile predators, and they have developed a skeleton of calcareous and siliceous spicules. They also have a toxin that is not well defined. About a dozen sponges from the 5000 or so known species have been incriminated as toxic, and these are found mainly in the temperate or tropic zones. Skin lesions have developed from sponges that have been deep-frozen or dried for many years.

CLINICAL FEATURES

One group of symptoms relates to the contact dermatitis associated with the areas of sponge contact. After a variable time, between 5 minutes and 2 hours, the dermal irritation is felt. It may be precipitated by wetting or rubbing the area. It may progress over the next day or so and feel as if ground glass has been abraded into the skin. Hyperesthesia and paraesthesia may be noted. The symptoms can persist for a week or more, with inflammatory and painful reactions around the area. The degree of severity is not related to the clinical signs, and some patients may be incapacitated by the symptoms without any objective manifestations.

The dermal reaction may appear as an erythema, with or without papule and vesicle development. Desquamation of the skin sometimes occurs in the second or third week, but in other cases the skin lesions have recurred over many months.

TREATMENT

The only adequate treatment is prevention, using gloves when handling sponges and not touching anything that has been in contact with the sponge. The use of alcohol, lotions, or hot water usually aggravates the condition. Local application of a cooling lotion, such as calamine, may be of some value, but

in general the skin lesion is treated with the conventional dermatologic preparations, though with limited success.

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CHAPTER 16 Marine Poisoning and Intoxication

Peter R. Lynch
Alfred A. Bove

Seafood is a favorite source of nutrition and enjoyment. However, various forms of seafood are known to carry toxins. This chapter reviews the toxicity of ingested marine animals *(ichthyotoxism).*

Some 1200 species of marine fauna are known to be poisonous. They vary from the simplest unicellular protistans to large chordate mammals. Outbreaks of seafood-related disease occur in all the seas of the world. In a few oceanic regions, they give rise to serious public health and economic problems. Seafood oral toxins are relatively stable and are not influenced by cooking, freezing, or drying. The symptoms of ichthyotoxism can present a confusing differential diagnosis, but a careful history with special attention to ingested seafood and a physical examination can clear the picture.[1–4]

SHELLFISH TOXINS

Trace amounts of the known shellfish toxins are often found in commercial shellfish from various areas of the world,[5] but most toxins are present in concentrations that do not affect health. These toxins can be identified in shellfish using high-pressure liquid chromatography (HPLC), and safe concentrations of the toxins have been established.[5] Many of the marine toxins are produced by Protistans, the protozoan, unicelled algae, diatoms, and bacteria widely distributed throughout all the marine waters from the polar regions to the tropics. Most of the toxic species are in the order Dinoflagellata, which are Pyrrophyta ("fire algae") associated with "red tides." Some believe that the Biblical mention of Aaron striking the waters of Egypt with his staff was the first written account of a red tide: "…and the fish that were in the river died; and the river stank; and the Egyptians could not drink of the waters of the river; and there was blood throughout all the land of Egypt" (Exodus 7:21). What people call red tides are often dinoflagellate blooms turning the water brick-red, brownish, green, or even yellow in the inshore bays, rivers, and sea. The high concentration of Protistans may deplete the sea of oxygen and cause death of fishes, but mollusks can filter these organisms and concentrate the toxin in their muscles.

Paralytic Shellfish Poisoning

Paralytic shellfish poisoning is usually found in temperate to tropical oceans worldwide. This poisoning occurs in humans after ingestion of shellfish that are contaminated by the toxins. Contamination may be found in mussels, beach and surf clams, razor clams, and butter clams, among others.[6] The dinoflagellates *Alexandrium tamarense* and *Alexandrium catenella* have both been shown to produce the toxin. The poison is a potent neurotoxin called *saxitoxin* or *neurosaxitoxin*[7,8] that acts by blocking voltage-dependent channels in muscle and nerve cells. This effect is similar to the effect of tetrodotoxin (see later), but opposite to the effect of ciguatoxin, which opens sodium channels.[7] The resulting disease is what we call paralytic shellfish poisoning.[9,10]

The illness may take on different manifestations that may be related to more than one form of saxitoxin. A gastrointestinal form is related with nausea, vomiting, diarrhea, and abdominal pain. Symptoms usually appear about 12 hours after eating the shellfish. In some subjects, the symptoms are typical of an acute allergic reaction with bronchospasm and oral mucosal edema. The paralytic type begins with sensations of tingling in the mouth and lips 5 to 30 min after eating a contaminated shellfish. The sensation

spreads to the rest of the body, and numbness may follow the tingling. Ultimately, motor paralysis develops and respiratory failure may occur.

Symptoms include circumoral paresthesias, headache, ataxia, slurred speech, vertigo, muscle weakness, peripheral paralysis, cranial nerve dysfunction, nausea, vomiting, dizziness, increased salivation, thirst, dysphagia abdominal pain, respiratory failure, and diarrhea (less common). An unusual symptom in some victims is a floating sensation when walking. Symptoms can last days to months for some of the myopathies. There is no specific treatment for this disease, but reversal of toxic effects has been shown in guinea pigs using a specific antitoxin.[11] If the oral sensations are perceived, the food should be immediately removed from the mouth. When systemic symptoms are found, the food should be removed from the stomach by gastric lavage. Vomiting should be induced if a gastric tube is not available. When muscle paralysis is present, one should begin careful monitoring for respiratory insufficiency and respiratory support, if necessary (usually over the first 24 hours). Diagnosis may be aided by a history of ingested shellfish and physical findings related to the symptoms. Mouse bioassay of the food eaten or HPLC spectrophotometry[3] can identify the specific toxin.

Amnesic Shellfish Poisoning

Amnesic shellfish poisoning has only recently been discovered in California, New England, and Prince Edward Island, Canada.[9,10,12] Planktonic algae or one-celled plants called *diatoms* (*Pseudonitzschia australis* and *Pseudonitzschia pungens*) produce domoic acid, a potent neurotoxin that is ingested by filter feeders such as bivalve shellfish (clams, oysters, scallops, and mussels). Unsafe levels of domoic acid have been found in anchovies, razor clams, butter clams, and the viscera of crabs (not the meat) in California.

Symptoms appear within 30 min to 24 hours after ingesting toxic shellfish. Initial symptoms include nausea, vomiting, abdominal pain, diarrhea, headache, malaise, tremor, and mental confusion. In 3 to 4 hours, dyspnea, bradycardia, seizures, coma, and death can occur. Hemiparesis and ophthalmoplegia also can occur. Survivors of severe cases often demonstrate permanent loss of short-term memory, hence the term *amnesic shellfish poisoning.* Although most symptoms last for hours, the memory loss may be permanent. Respiratory support may be needed in the early phase of exposure.

A human illness designated as *possible estuarine-associated syndrome* by the Centers for Disease Control and Prevention has been associated with estuaries inhabited by toxin-forming dinoflagellates, including *Pfiesteria piscicida* and *Pfiesteria shumayae.*[12–15] Divers and fishermen may be exposed through direct contact with the estuarine water or by inhalation of aerosolized toxins in the local air. Symptoms most commonly reported are cough, diarrhea, headache, fatigue, memory impairment, skin rash, difficulty concentrating, light sensitivity, burning skin upon contact with water, muscle ache, and abdominal pain. Less frequent symptoms are upper airway obstruction, shortness of breath, and red or tearing eyes. Resolution with cholestyramine treatment suggests a neurotoxin-mediated illness.

Neurotoxic Shellfish Poisoning

Neurotoxin initially causes gastrointestinal symptoms such as nausea, diarrhea, and abdominal pain. These are followed by neurologic symptoms that can include circumoral paresthesia, vertigo, ataxia, convulsions, and respiratory paralysis. Other symptoms may include bradycardia, headache, cramping pain in the lower extremities, and dilated pupils. Symptoms usually appear within minutes of ingestion but may be delayed by as much as 3 hours after ingesting the toxic shellfish. Patients requiring respiratory support recover within a few days. The offending Dinoflagellate appears to be *Ptychodiscus brevis*, formerly known as *Gymnodinium brevis.* The toxin is called *brevetoxin.* Geographic distribution includes the Gulf of Mexico, Japan, France, Portugal, and New Zealand.

Diarrhea Shellfish Poison

Diarrhea shellfish poison produces predominately gastrointestinal symptoms. They include incapacitating diarrhea, nausea, vomiting, abdominal pain, and chills. Neurologic symptoms are absent. Recovery

usually occurs within 2 to 3 days, with or without medical assistance. To our knowledge, no fatalities have been associated with this disease. The toxic substance is *okadaic acid,* found in clams, oysters, and mussels. Okadaic acid is a potent inhibitor of protein phosphatases 1 and 2A,[16] alters chloride transport across cell membranes,[17] and, in animal studies, has neurotoxic properties that cause loss of short-term memory.[18] In one report, Phytoplankton from contaminated mussels were dominated by the Dinoflagellate species *Dinophysis norvegica.*[19] Diagnosis of okadaic acid poisoning is made with HPLC mass spectrometry of polyether toxins, clinical signs, and mouse bioassay. Treatment is supportive and includes replacement fluids and antidiarrheal medications.

FISH POISONS

Scombroid Fish Poisoning

Scombroid fish poisoning occurs when fish that are usually safe to eat are left for several hours at room temperature or outside in a warm climate. When at room temperature for several hours, histidine in the muscle tissues is converted to saurine and histamine by bacteria (e.g., *Clostridia, Salmonella*).[20,21] Toxic levels of histamine produce anaphylactoid symptoms. This is one instance wherein bacteria have a role in producing the toxin. The toxin, however, is not bacterial.

This form of poisoning was originally associated with the Scombridae family of fish, such as tuna, albacore, mackerel, and bonita.[6] Other fish, such as mahimahi (dolphin), amberjack, yellowtail, snapper, and bluefish, can produce this toxin if they are poorly handled and not refrigerated immediately after landing.[21] Epidemics have been related to canned tuna. Because scombroid fishes are shipped widely, this illness can be found in sites remote from the source of the fish. Scombroid outbreaks have been identified in fish caught by amateur fishermen.[22]

Symptoms include diffuse erythema and flushing,[21,23] palpitations, headache, nausea, vomiting, diarrhea, abdominal pain, anxiety, hypotension, bronchospasm, urticaria, and hypotension within 30 to 60 min of ingesting toxic fish. Thirst and dysphagia follow the acute phase of the disease. In general, the more fish consumed, the more severe the symptoms. It should be noted that cooking the fish does not inactivate the toxin. Diagnosis is made by clinical signs; laboratory tests are usually not necessary. Histamine levels can be measured if a sample of fish is available. An electrocardiogram is useful in identifying arrhythmias. The patient may describe the fish as having a unique pepperybitter taste.[24]

Treatment includes gastric lavage, support of circulatory collapse, and support for respiratory insufficiency. Antihistamines are considered to be effective and should be administered parenterally if the poisoning is severe.[24] Both H_1 and H_2 antagonists have been used as effective treatment for abolishing the symptoms, but an H_2 antagonist alone may be sufficient[25] and is less likely to cause sedation. The disease is usually self-limiting but can cause significant discomfort. Antihistamines such as diphenhydramine, cimetidine, and ranitidine[21] have been used successfully. Following treatment, the prognosis is usually excellent. Symptoms usually abate in 8 to 12 hours. Scombroid poisoning can be mistaken for a true seafood allergy. An epidemic outbreak identifies the nonallergic nature of the intoxication.

Ciguatera Fish Poison

Ciguatera poisoning is the most common fishborne nonbacterial poisoning. This poisoning is caused by many of the common fish found in tropical waters and is considered to be the most common foodborne illness related to consumption of fin fish.[26,27] This type of poisoning is caused by eating reef fish that have been feeding on the dinoflagellate algae *Gambierdiscus toxicus.* The toxin *(ciguatoxin)* accumulates in large fish and causes severe symptoms. Ciguatoxin has been a known tropical hazard for centuries in areas with extensive coral reefs.[28] Because of the rapid transport of fish by commercial fisheries, ciguatera disease can appear in any inland location. A case of coelenterate-borne disease has been reported.[29] In the tropical areas of Florida and Hawaii, home products are sold to detect ciguatera in locally caught fish (e.g., Hawaii Chemtect, 626-568-8606). Species of fish that most often carry ciguatoxin include grouper, red snapper, barracuda, and large grunts. Cases have occurred from moray eels. Fish larger than 2 kg can contain large amounts of ciguatoxin. The toxin is thought to be more common following

upwelling from deep ocean waters.[30] Ciguatoxin is heat-stable (unaffected by cooking), colorless, and odorless, and it does not change the taste of the fish. The toxic effects are caused by activation of voltage-dependent sodium channels resulting in hyperexcitability, decreased conduction of action potentials, and a prolonged refractoriness of nerve and muscle cells.[7]

Symptoms usually occur within 12 hours of ingesting the affected fish but can appear within minutes of ingesting the ciguatoxin and increase in severity over the subsequent 4 to 6 hours. Presentation may vary from minor symptoms to coma and death. Gastrointestinal symptoms are often the first to appear and may last for several days. They consist of abdominal pain, nausea, vomiting, and diarrhea. Neurologic symptoms usually occur within a few hours to 3 days after the meal and can last for months. The first symptoms are often lingual and circumoral paresthesias, followed by painful paresthesias of the extremities. Muscle weakness, headache, myalgias, ataxia, vertigo, and respiratory paralysis follow the initial symptoms. Paradoxical sensory effects (e.g., cold objects feel hot, hot objects feel cold), occurring in one third of patients, are likely due to exaggerated nerve depolarization in small sensory fibers affected by the toxin.[31] The often-experienced weakness and dizziness probably reflect the bradycardia and hypotension commonly found in these patients. The hypotension results from the fluid loss, bradycardia, and peripheral vasodilation and perhaps from the direct toxic effects on the myocardium. These symptoms usually resolve in a few days. Other symptoms include dyspnea, sweating, chills, neck stiffness, and pruritus. Dehydration following the vomiting and diarrhea is common. Palafox and colleagues[32] listed the clinical features of 24 patients with ciguatera poisoning (Table 16–1). Neurologic symptoms dominate the clinical presentation. Although mortality is usually low, an outbreak in Madagascar following ingestion of a shark affected 500 people and resulted in a mortality rate of 20%.[33]

The active disease is most severe for 6 to 10 (average, 8.5) days,[34] but many victims complain of symptoms for prolonged periods that may extend to a year or more. Prolonged orthostatic hypotension may result from autonomic dysfunction.[35] Some victims experience recurrence of symptoms when any fish or fish product is ingested; severity may be related to repeated subclinical exposures[36] or to a history of previous intoxication. There are no diagnostic laboratory tests, although there are immunoassays,[37] and bioassays[38,39] for the toxin in suspected fish products.

Table 16–1. Clinical features of ciguatera poisoning in 24 patients

Sign or Symptom	%
Circumoral or facial paresthesia	96
Paresthesias in extremities	96
Cold-to-hot reversal	79
Paresis	17
Coma	8
Abdominal pain	42
Diarrhea	38
Nausea	33
Vomiting	33
Muscle pain in extremities	75
Arthralgia	75
Hypotension	12

Treatment of ciguatera poisoning is nonspecific and primarily supportive.[26] A variety of treatments have been suggested. These include calcium gluconate, corticosteroids, atropine, vitamin B complex, pyridoxine, and amitriptyline.[40] Following the initial case report by Palafox and coworkers,[32] which suggested that mannitol may be helpful in comatose patients to manage cerebral edema, several other studies confirmed the value of mannitol in treating the acute phase of ciguatera poisoning even without cerebral edema.[26,41,42] Amitriptyline may be useful in treating chronic manifestations.[40] Serotonin and norepinephrine reuptake inhibitors are reported to relieve symptoms of pruritus and dysesthesias by blocking fast sodium channels that have been activated by ciguatoxin.[40] Acetaminophen is useful in treating headache. Nonsteroidal anti-inflammatory drugs also can be used to relieve myalgias and arthralgias. Diphenhydramine can be used to relieve pruritus.

During the recovery period, the patient should avoid ingesting all fish products, shellfish, alcohol, and nuts. Ciguatera outbreaks occur in Hawaii and Florida each year. The poisoning is seldom lethal, but children are involved more often in life-threatening cases. The disease becomes more common each year and occasionally appears in inland locations because fish are shipped by air all over the world.

Tetrodotoxin or Pufferfish Toxins

Poisoning from pufferfish, globefish, or blowfish toxins results in more human deaths than any other fish toxin.[43,44] This form of poisoning is typified by Fugu poisoning in Japan, which occurs from ingestion of the Asian pufferfish.[45] In this fish, the gonads and the liver contain lethal amounts of the poison. Microorganisms produce the toxin, which is then ingested by the pufferfish and accumulated in the tissues. The toxicity of these fish has been known for centuries and may account for the admonition found in the biblical passage from Leviticus (11:9–12), which states, "these shall ye eat of all that are in waters, in the seas and in rivers, them shall ye eat. And all that have not fins and scales in the seas and in the rivers, of all that move in the waters, and of any living thing which is in the waters, they shall be an abomination unto you. They shall be ever an abomination unto you; ye shall not eat of their flesh, but ye shall have their carcasses in abomination. Whatever hath no fins nor scales in the waters, that shall be an abomination unto you." Pufferfish and globefish have no scales.

The Japanese consider *fugu,* or pufferfish, a delicacy. There, some 50 people a year die from feasting on this expensive gourmet dish. Other unreported cases probably account for many more deaths throughout the tropics. Toxic outbreaks have also been described after ingesting shellfish.[46] Tetrodotoxin is a neurotoxin that blocks the transmission of sodium ions through cellular membranes preventing depolarization and propagation of the action potential[45] and affects neuronal transmission in motor and sensory peripheral nerves, the central nervous system, and muscle cells. Cardiac conduction and contractility may be affected. Tetrodotoxin derives its name from the pufferfish family Tetraodontidae. This class of fish includes pufferfish, globefish, and blowfish. Interestingly, this toxin has been found on the skin of salamanders,[47] the skin of atelopoid frogs ("poison dart frogs"), and the saliva of the blue ringed octopus *(Hapalochlaena maculosus)* (see Chapter 15). In the pufferfish, the toxin is most concentrated in the gonads, liver, and skin. The concentrations of these toxins vary throughout the year in the offending fish species.[43] It has been demonstrated that the toxin is derived from an algae covered with the bacterial species *Alteromonas.* In Japan, specially trained fugu chefs are considered more expert if they leave small amounts of the toxin in the fish to provide a mild tingling sensation in the mouth. The mortality rate from a large dose of toxin can reach 60%.

Symptoms occur in the first 15 minutes after the ingestion of a pufferfish. Rare incidences occur as late as several hours after the meal. The first symptoms are circumoral or tongue paresthesias, followed by facial and extremity paresthesias. Nausea, vomiting, abdominal pain, and diarrhea follow the initial sensory symptoms. Neurologic involvement often begins with muscular fasciculations and twitching; it may progress to total flaccid paralysis. Muscle weakness, neuralgias, ataxia, dysarthria, and rapid ascending paralysis occur within 4 to 24 hours. Speech and respiration may be compromised, and ocular paralysis may occur. Hypotension and cardiac arrhythmias occur concomitantly with the neurologic involvement. A curare-like state ensues wherein the patient is totally immobile but conscious. Seizures may be followed by apnea and coma. Death can occur within 4 to 6 hours from respiratory paralysis.

A history of ingesting fugu is the key to proper diagnosis. Cardiopulmonary resuscitation must be initiated early and should include endotracheal intubation for oxygenation and airway protection in case vomiting occurs. If vomiting occurs, gastric lavage may not be necessary, but every effort should be made to remove gastric contents. Intravenous therapy may be needed to support blood pressure or to treat cardiac arrhythmias. Although no drug has been discovered that can reverse the effects of tetrodotoxin, symptomatic treatment is often successful. Activated charcoal and neostigmine have had some success. When paralysis occurs, respiratory support is required for 24 hours or more. The toxic effects usually remit spontaneously within 48 hours if the patient is otherwise supported.[48] Sedation should be given to the totally paralyzed patient. No specific treatment has proven to be effective.

Patients with documented pufferfish poisoning should be placed in an intensive care unit for at least 24 hours, on a respirator if necessary. The mortality rate is approximately 50% even with the best of care. Prognosis is good if the patient survives the

first 24 hours after ingestion. This type of poisoning requires the reporting physician to contact the local authorities.

CONCLUSIONS

Besides the well-described infections from shellfish exposed to water contaminated with hepatitis A or Norwalk virus,[49] paralytic shellfish poison, diarrhea shellfish poison, amnesic shellfish poison, and neurologic shellfish poison all are related to ingestion of edible, commonly ingested shellfish. Fish transmit ciguatoxin, the most common poison, scombroid poison, and the deadly but fortunately rare tetrodotoxic poison.[50] To avoid these problems, avoid eating unknown species of fish or shellfish, check with local authorities for the safety of their seafood, do not eat shellfish collected from an area that has suffered a red tide, and keep seafood refrigerated.

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Related Web Sites

http://umed.utah.edu/net
http://www.chbr.noaa.gov
http://www.emedicine.edu

CHAPTER 17 Human Performance Underwater

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Human performance underwater takes many forms, such as when a commercial diver inspects a pipeline, a sports diver engages in underwater photography, or a Navy diver repairs a propeller. For many years, diving medicine has emphasized physiologic problems such as decompression, which is largely a matter of getting the diver safely to the worksite and back without untoward physical problems. However, as Bennett observed in 1965,[1] while this emphasis is "of vital necessity, it should not be forgotten that this constitutes wasted effort if man, working under pressure, is not in perfect physical and mental condition." Perfection may not be achievable, but it is certainly a worthwhile goal.

In recent years, the emphasis has shifted to an understanding of those factors that affect the diver's ability to perform effectively and safely underwater. This has resulted in a change in the physician's focus from an almost exclusive concentration on physiologic factors to an awareness of the elements of human performance that are crucial to safety and efficacy. Because emotional elements (such as anxiety and panic) and factors such as environmental limitations, work objectives, equipment limitations, decompression obligations, and gas mixtures affect diver performance, the physician's normal physiologic orientation must expand to include a psychological component.

The problem has been compounded somewhat by a blurring of the distinctions between commercial and military diving and sport diving (Fig. 17–1). Increasing numbers of sport (or recreational) divers are participating in diving activities that involve decompression. A recent highly publicized trend involves technical diving, an activity in which a relatively small number of divers are extending the limits of traditional diving for exploration and personal achievement (Fig. 17–2). These divers consider the traditional limits of air diving (namely, to depths not to exceed 130 fsw with no decompression) to be restrictive and unnecessary and think that they should not be limited in the pursuit of their goals while accepting the responsibility for their own safety. Unfortunately, the educational training programs, technical support, and short-range perspective necessary for implementing such programs are currently lagging behind the marketing of these programs. What was originally an activity involving breath-hold diving or relatively shallow, no-decompression dive profiles now has requirements when pursuits, such as wreck diving or cave diving, necessitate divers' staying longer at greater depths. In addition, some cave divers have developed dive profiles for cave dives to as deep as 700 ft, which require the use of helium-oxygen breathing mixtures.

Developments increasing the availability of enriched air (nitrox) and other breathing mixes have also been of interest to divers because they allow an extended bottom time within the limits of oxygen tolerance. Wreck diving and cave diving are diving activities that require profiles and, perhaps, breathing mixes that differ from those used in standard scuba and skin-diving procedures, but many divers still refer to such activities as "sport" or "recreational" diving. However, once a diver enters a decompression-schedule dive or uses a mix that is not a standard compressed-air mixture (and the two are often related), the dive should no longer be considered recreational. The planning and operation of a decompression dive, the use of breathing mixes such as nitrox, heliox, trimix, or some other exotic gas mix, and the need for different support equipment make such dives classic *working* dives. Even if the purpose of the dive is adventure or recreation, the character of the dive is different,

Figure 17–1. Recreational scuba. (Photograph courtesy of Mark Lonsdale.)

Figure 17–2. Deep air system with nitrox and oxygen. (Photograph courtesy of Mark Lonsdale.)

and the diver should be considered a working diver, who needs to be more knowledgeable about operations and equipment than the shallow, no-decompression, warm-water diver.

Figure 17–3. Dry suit diver in technical gear.

The technical diver who uses exotic gases such as helium or hydrogen in addition to standard mixes needs a number of different tanks for the various stages of the dive (Fig. 17–3). Each tank requires a specifically different identification for its intended use. The skill in differentiating tank and its contents from another at depth, as the diver shifts from one gas mix to another as demanded, is one that requires exceptional ability and overlearned skills. There is always the risk that degrading factors, such as nitrogen narcosis or cold, dark water, will impair these skills and affect performance.

This position does not suggest that divers should reject activities such as wreck diving or cave diving as recreational pursuits; however, divers must recognize that the demands are more severe and that the requirements for sound physical and emotional conditions, as well as training and diving skills, are dramatically increased. Likewise, the demands on the diving physician who evaluates candidates for diving programs are increased. It is clear that the technical diver today is oriented to using the tools that have traditionally been associ-

ated with commercial, military, and scientific endeavors. The latter programs have always been founded on a strong base of training and technical and logistical support; however, such training and support are not available to most divers who wish to perform extended-range dives. It is refreshing to note that some groups of technical and public safety divers are developing protocols that require all of the team members to wear standardized equipment configurations in order to minimize risks. Because of the risk with extended-range dives, the preparation and execution for each and every dive require strict attention.

Another development in technical diving has recently reached the recreational diving community: the rebreather. The concept of the rebreather is not new, having a long history of military use since World War II, but the introduction of the technique to the civilian diving community is new. Sawatsky, in a series of three articles on the physiology of rebreathers,[2] quotes sources in the diving industry who predict that within a few years, 30% of recreational divers will be using rebreathers. This estimate may be overly optimistic, but several manufacturers of rebreathers have put several thousand devices into the field. Each device has its own unique set of requirements for optimal safety. Rebreathers vary in cost as their complexity increases; a state of the art model will likely cost well over $10,000. Cost alone is unlikely to be a significant deterrent to growth, but the complex training and labor-intensive preparations may well slow the widespread use of rebreathers in the recreational market.

The concept of the rebreather is simple. Instead of exhaling into the water, the diver exhales through a carbon dioxide absorbent filter that removes CO_2 before the exhaled gas is balanced with oxygen and, if needed, diluent gas to reestablish the tidal volume of gas at an appropriate mixture needed to breathe comfortably and safely at the diving depth. As the diver breathes the reconstituted gas from the breathing bag, it is taken into the lungs and needed oxygen is extracted before the exhalation cycle begins again. This balancing cycle is repeated with each breath. Because pure oxygen and, if need be, diluent gas is introduced into the breathing bag in small amounts, the size of the supply tanks can be dramatically reduced.

Rebreathers that can provide the proper gas mix for the depth of the dive permit deeper excursions for longer periods. Although this appears to be a distinct advantage, the diver's task load increases exponentially, as does the risk of injury or death. The high level of training and the dedication to detail necessary for the operation and maintenance of today's equipment limits recreational closed-circuit to a relatively small number of divers. Variables such as proper control of the mouthpiece and predive checks—including assembly, pressurized testing of the components, and quality control of gases—must all become a matter of strict routine.

Because the use of rebreather technology for deeper, longer dives poses major task-loading issues, constant monitoring of redundant readout displays and awareness of time and depth parameters are critical. The O_2 partial pressure must be maintained at less than 1.4 ata and more than 0.16 ata to avoid serious problems. Such constant monitoring is needed to avoid hypoxia, hyperoxia, or hypercapnia, all possible causes of deaths on rebreathers.[3] In a recent analysis of 13 deaths in the United Kingdom involving divers using a modern rebreather, Bevan[3] stresses the element of high task-loading as a problem. He observes, "The very requirement to check the handset every single minute or even oftener during a dive suggests that the set's operation needs extremely vigilant monitoring." Bevan also notes the relatively large number of instruments to be monitored, more than 15 in all. "Seven of these controls," he observes, "are out of the view of the diver." This task loading requires the diver to be aware of the location of the controls and to be able to perform needed functions rapidly and under conditions of poor visibility. Although the absolute number of deaths on these rebreathers is small, the ratio compared with other diving techniques such as scuba is high. The fatality rate for members of the British Sub-Aqua Diving Club in the year 2000 was 1 death per 6666 members. As Bevan observes,[3] assuming there were 2000 modern rebreather divers over the period of 3.5 years of reporting, with a total of 13 fatalities, the ratio would be an average of 1 fatality per 570 divers, or 12 times higher. Sawatsky cites 9 deaths out of 1000 modern rebreather dives.[2] Although the advantages of rebreather technology are significant, so are the risks.

What does this mean for human performance? It simply means that a diver using a modern rebreather may be able to dive deeper and stay longer, but only at the cost of constantly monitoring the life support system. This divided-attention task loading may seriously reduce one's ability to perform complex tasks or even enjoy an adventurous dive. This loading certainly increases risk. Future developments may reduce the expense and labor-intensiveness of rebreathers, but, in the near term, widespread use of the rebreather technology seems limited by the task-loading issues.

ELEMENTS OF PERFORMANCE

All too often performance is described in general and abstract terms, as when one says that a diver "inspects" or "repairs" an object underwater. Planning a dive operation requires more than a general description of an overall task. It is much more valuable to specify particular behaviors that make up the entire performance in a complex task. Such specificity is also valuable to the scientist or physician who wishes to evaluate the ability of the diver to perform a task. For example, it was questioned whether a diver working in dark waters needed to have perfect color vision. The dive master planning an operation might not be concerned if a diver working under turbid conditions lacked optimal color vision but might be concerned if this same diver was the operator of a gas manifold or hyperbaric chamber, with which recognition of color-coding would be important. Even here, the experienced operator can learn the placement of certain valves and perhaps overcome the lack of color discrimination. Divers can be assigned more effectively to jobs, and efficiency and safety can be increased, if the requirements of the task and the characteristics of the diver are known and objectively evaluated. A change in any aspect of a diving operation imposes new demands, however subtle, on divers, who must modify their capabilities in order to remain in control of the situation.

Psychologists in performance research agree on three elements that constitute an overall performance sequence:

- *Perception*: sensing the stimulus event (which may be visual, tactile, or auditory)
- *Information processing*: cognitive or mediational processing through which the information perceived is evaluated and a course of action (a motor response) is selected
- *Motor response*: performing the motor activity that completes the task

In various analyses of these factors, behavioral components have been further delineated. One such classification that attempts to delineate performance characteristics with greater specificity was reported by Christensen and Mills[4] and by Berliner and colleagues[5] and was later modified by Bachrach.[6] In this system, the performance elements include the following:

Perceptual Processes

- *Searching for and receiving information:* The performer (operator) detects, inspects, observes, reads, and surveys.
- *Identifying objects, actions, and events:* The operator discriminates, identifies, and locates.

Mediational (Cognitive) Processes

- *Information processing:* The operator categorizes, calculates, codes, computes, and itemizes.
- *Problem solving and decision making:* The operator analyzes, calculates, computes, compares, estimates, plans, and selects a course of action.

Motor Processes

- *Simple or discrete processing:* The operator activates, closes, connects, disconnects, and sets.
- *Complex or continuous processing:* The operator adjusts, aligns, tracks, regulates, and synchronizes.

This model permits assessment of elements that are most affected by external events. For example, in a review of the effects of alcohol on driving behavior, Moskowitz and Austin[7] concluded that the primary effect of alcohol is on mediational processes, which include information processing and response selection, rather than on sensory or perceptual inputs or on the motor response. In other words, the driver under the influence of alcohol may have the capacity to detect the red light (sensations/perception) and to activate the brake pedal (motor response), but the information processing needed to interpret the significance of the red light and to make a decision based on this input is adversely affected. Similarly, Bradley[8] suggested that cold is a major factor in commercial diving fatalities in the

North Sea, largely because of impaired judgment and decision-making components of information processing imposed by an external event—cold.

In the original classification of performance elements, Christensen and Mills[4] added the element of *communication processes*, which is very important in task completion. In communication processes, the operator advises, answers, reports, transmits, requests, answers, and instructs. Communication is a part of the operator's performance that may be adversely affected if, for example, cold has distracted the diver's attention so that inputs from topside are not perceived, or, as seen in Bradley's study,[8] a loss of judgment resulting from cold exposure interferes with information processing. Problems in communication were also significant disruptors of perception and information processing in the Moskowitz and Austin report on the effects of alcohol.[7]

Task Loading

In earlier discussions of diving methods such as technical diving and the use of rebreathers, task loading is mentioned as a major problem in such diving. *Task loading* refers to the increase in decision-making and tasks with such dives. Adding gauges to monitor, jobs to perform, and time limits to observe (owing to decompression or gas supply) are among the factors that stress a diver's ability to function effectively. As the earlier discussion of performance elements showed, it is useful to break down performance into specific factors that can be analyzed. This is also true of task loading.

In 1952, a psychologist named Hicks came up with what has since been called *Hicks' law*. Hicks stated that a reaction time increases approximately 150 msec when the response option increases from one to two. For every choice added, the response time essentially doubles. He also held that constancy in processing information is needed to evoke a response, even when complex decision-making is required, a premise that has been experimentally confirmed.[9] The time to respond with a decision to act is proportional to the log of the number of alternative courses of action. Faced with a number of choices in a dive situation the diver must respond as effectively as is possible. Because a number of choices involves time of response, the necessary performance becomes one of *queuing*, in which the most critically important response is selected as the first action. When a mask leaks, a sea-state changes for the worse, and a shark appears, it is not time to check the contents of the collection bag. Thus, task loading and its inevitable primacy must be emphasized as an element of performance. The skill pattern of queuing, to order responses in terms of their immediate urgency, should be a critical part of emergency dive training.

With this classification of the key elements in human performance, we may proceed to evaluate the factors that make human performance underwater a special field of research and practice. The primary factors to be considered in this chapter as they relate to performance are the environment, diver training, diver condition (this includes emotional factors, principally panic), and the type of work performed. Other chapters in this book deal with the all-important performance factor of equipment (Chapter 3) and the effects of hypothermia (Chapter 13). The effect of drugs is discussed later in this chapter.

Environment

Among the environmental factors of primary interest in underwater human performance are the water medium, visibility, and temperature.

WATER MEDIUM

The water significantly affects diver performance. Being a different medium from air, it necessarily results in alterations in performance. Movements in a viscous medium are patently different from those performed in air. In a study conducted at the University of California, Los Angeles (UCLA), Weltman and colleagues compared the learning of an underwater assembly task (the UCLA pipe puzzle) in two groups of divers; the first group was trained to perform the task on dry land, whereas the second group learned to perform the task underwater.[10] The dry land group had the advantage of direct contact with the training staff as well as immediate corrective feedback on their performance. Nevertheless, the water-trained

group achieved a 25% faster mean completion time on the task, suggesting not only that the movements are different in water but also that performance differences (and accordingly, differences in performance assessment) must exist between water and dry land. Many performance assessments use dry-land baselines. The studies by Weltman and his colleagues suggest that a more fitting baseline might be performance under *optimal* diving conditions (clear, comfortable, low-current water, with a minimum of protective gear), so that the assessment can compare performance under given diving conditions with the optimal conditions, not against dry-land or hyperbaric chamber baselines, which do not include underwater characteristics.[10]

The water environment reduces performance in many ways. The relative weightlessness in a tractionless milieu is a major problem that markedly affects the use of tools because of recoil and the difficulty of maintaining a stable posture. Conditions of high current greatly compound this problem when swiftly flowing or surging water displaces the diver, making it difficult to maintain stability and control over tools and equipment.

Low visibility is another environmental factor that decreases performance. Turbidity and other obscuring elements in the water make observing and performing a task more difficult. The diver often has to resort exclusively to tactile sensory inputs when faced with a loss of visibility, but this is a problem when the turbid water is also cold, necessitating the use of protective gloves, which markedly diminish sensation. Side-scan sonar imaging is new technology that offers promise as an aid for "seeing" in dark waters.

VISION AND PERFORMANCE

Various aspects of vision are altered underwater (see Chapter 2). Although some visual adaptation occurs, correct visual perception is required as the complexity of underwater tasks increases. Most alterations in underwater vision are largely based on the fact that radiant energy changes as it travels through water. Water has much lower transmission efficiency than air, largely because of suspended particles of matter that cause blurring and loss of contrast. Absorption of energy in the denser medium varies with the clarity of the water. In Antarctica, where the water is extremely clear and the sun is extremely bright, it is possible to see high-contrast items at a range of 800 ft or more. This range may be reduced to inches in turbid water. Distortion also varies considerably. The distortion in the optical image is caused by the refraction of the light rays at the interface of the mask with the faceplate as the rays pass from the water into the gas environment within the mask, where the speed of the light is greater. These limiting parameters lead to several consequences.

The underestimation of distances underwater is due to the fact that, when the light rays are refracted at the perpendicular to the interface with the mask lens, a virtual image of the object is formed at a distance that is three quarters of the actual distance of the object from the interface. Changing the angle of refraction results in a change of the usual 3:4 ratio. Objects viewed at a decreasing angle from the perpendicular through a flat lens are distorted further; thus, looking to the side of a mask results in larger images than looking straight ahead. The extent of refraction depends on the angle of incidence of the light rays, altering the shape and position of the optical image and resulting in changes in perceived size and location.

A further distortion occurs during diving for size-limited items such as lobster. The lobster appears larger underwater because the underwater retinal image of the lobster, formed from the virtual image, is larger than what would be perceived in air. This occasionally embarrassing problem can be somewhat mitigated by adaptation through experience.

Finally, because objects underwater are not physically located where they appear to be, hand-eye coordination and visual motor skills may be disrupted. Experience and adaptation can be valuable in reducing this problem.

Visual fields of divers are often limited by the skirt portion of the mask that creates the seal to the face and causes the refraction of light at the air–water interface. The various faceplate configurations generally result in a form of tunnel vision. The eye can see the inside perimeters of the mask, which act as the sides of the tunnel. Most divers become adept at accommodating this physical limitation, and many divers prefer low-volume, close-fitting masks. The nose, however, creates a problem because the covering over

the nose pocket significantly restricts the lower visual field. This becomes annoying when the diver is unable to view the equipment located on the front of the body.

Color perception is an important aspect of underwater diving because color is important in the enhancement or camouflaging of many underwater objects. The visibility of colors is largely a function of the clarity of the water and the available light. The use of fluorescence has become increasingly popular for underwater markings. It appears that fluorescent colors convert short-wave energy, to which the eye is relatively insensitive, into energy of longer wavelength, to which the eye is more sensitive; the result is a brighter image. In general, colors close to each other on the visual spectrum are hard to distinguish, whereas colors from opposite ends of the spectrum are more readily discriminated.

COLD STRESS AND EFFECTS ON PERFORMANCE

The most disruptive of all environmental conditions is temperature; the most severe stress in diving is cold exposure. Not only does cold itself adversely affect performance, but protective gear such as gloves and other clothing can diminish mobility and manual performance. Although great strides have been made in the field of thermal protection, particularly in the area of dry protection, cold remains a major factor limiting prolonged exposures (see Chapter 13).

Webb[11] observed that "cold is a seriously limiting factor in many diving operations" and that it is likely to be a causative or contributing factor in commercial diving accidents; however, "because cold exposure is routine in diving, it is accepted and its potential for harm is discounted."

In a 1987 report, Padbury and colleagues[12] offered a similar comment: "The diving industry is skeptical whether undetected hypothermia is a real enough danger to warrant monitoring, let alone development of equipment or procedures to prevent it." Recognition of the dangers and risks associated with hypothermia has greatly improved. Divers should pay the same degree of attention to depleting body heat as they give to depleting their breathing gas. The term *undetected hypothermia* has been used by research workers and field personnel (along with the synonyms *insidious, silent,* and *progressive* hypothermia) to describe the slow effects of cold exposure on the diver. The process is a slow cooling that often occurs without the diver's realizing that significant decrements in performance have occurred. This change occurs anytime a temperature gradient is affecting the diver.

Divers in well-insulated dry suits, working in Antarctic waters where the ambient temperature under thick ice cover is 28.6°F (–1.9°C), can work effectively for periods of 15 to 20 min before hand function is affected severely. Greatly improved thermal protection, involving dry suits and heavy underwear, keeps the divers much warmer in colder water with a significant increase in the effective work time. Hand function and strength during extreme cold exposure remain common limiting factors in underwater work owing to the effects of cold on muscles and peripheral nerves. The central nervous system and associated functions continue to operate reasonably well under extreme conditions.

The progression of hypothermia suggests that the critical factor is the *rate of cooling.* Kuehn[13] commented on this variable, noting that rapid heat loss results in a greater drop in rectal temperature accompanied by more shivering, whereas an identical loss of heat over a longer period results in a small temperature drop (compared with rapid cooling), less shivering, and fewer complaints about cold discomfort. The concern over slow cooling is that the diver may not be aware of progressive changes, which are not as profound as those associated with rapid cooling, creating a potential for hazard. Bradley's study[8] implicates such cold exposure as a causative or contributing factor in 11% of North Sea commercial diving fatalities; in another 15% of fatalities, loss of judgment (a cognitive decrement resulting from cold stress) appeared to be a crucial element in the accident and its unfortunate consequences.

Among the principal physical symptoms associated with progressive hypothermia are muscle stiffness, numbness, weakness, and loss of muscle strength, all of which reduce the diver's ability to respond effectively and greatly affect critical motor functions such as manual dexterity. This degradation of physical capacity, coupled with loss of judgment and decision-making competence, places the diver in a vulnerable position as far as safety and performance are concerned.

Some problems encountered by the diver working in a cold environment are caused by distraction. The diver's concentration on the task requirements may mask the awareness of physiologic changes that are occurring with slow cooling. Another form of distraction is being uncomfortable from cold exposure, which can interfere with optimal performance of the task and lead to serious consequences. As Childs[14] observed, "Distraction due to discomfort may cause the diver to ignore threats to his safety underwater and, finally, realizing he is in danger, he may be in further difficulty because of a loss of power and dexterity in his hands." Distraction was considered to be a major factor in problems in performance, as reported by Vaughan,[15] who found that Navy divers showed impairment in reaction time and target detection early after cold-water exposure. Similar findings were reported by Davis and colleagues,[16] who recorded significant decrements in performing tasks of simple arithmetic, logical reasoning, word recall, and recognition (all cognitive tasks), as well as in manual dexterity, in divers exposed to water at a temperature of 5°C.

Another study suggesting that distraction resulting from cold exposure may be a factor in reduced performance is one by Padbury and colleagues,[12] in which divers were locked out of a 450 m chamber dive in cold water. The divers were wearing open-circuit hot-water suits for warming but showed uneven temperatures. One diver was shown to have undetected hypothermia when his rectal temperature fell from 37.8°C to 36.3°C in a 66 min lockout, although he did not complain of cold or discomfort. It should be remembered that a core-temperature drop of 1°C to 1.5°C moves the diver from the "comfort" range of 37° to 36°C into the "tolerable" range of 36° to 35.5°C. The drop in temperature recorded in this diver over a 66 min lockout was not severe but was approaching the discomfort level. Among the results of this study were the findings that no decrements occurred in manual performance as measured by finger dexterity or arm or wrist speed. However, vigilance (a cognitive task), as measured by speed at arithmetic and visual reaction time, was reduced on the first cold exposures but improved with later trials. The assumption that distraction from cold exposure adversely affects early performance, whereas later trials show improvement, appears valid if adaptation to the novel stimulus of cold exposure over time is considered. Over a number of exposures, humans can adapt and tolerance levels can change by ±0.5°C. However, other factors need to be accounted for. Perhaps of greater importance is the difference between a chamber dive in a wet pot and an open-sea dive. Even when the temperature and pressure are controlled, an open-sea dive poses conditions of danger not present in a controlled environment.

Reeves and associates[17] suggested a neurophysiologic basis for changes in performance in preliminary studies involving subjects in swim trunks in a cold immersion tank, which effectively produced an average reduction in colonic temperature to 35.5°C. The cold exposures either lasted 60 to 90 min or until a core temperature reduction of 1.5°C below baseline was reached, whichever occurred earlier. The techniques used included electrophysiologic evaluation of central nervous system activity and employed visual and auditory event-related potentials to measure the effects of cold immersion on nerve conduction. Related performance measures such as tapping and grip strength were employed to determine behavioral factors in nerve conduction velocity. The authors concluded that the multineuronal conduction velocity in both the central and peripheral nervous systems can be reliably slowed as a result of reducing core temperatures by an average of 1.5°C. Increased latency in nerve conduction was associated with lowered cognitive performance. A reduction in nerve conduction velocity was significantly correlated with the tapping rate (the number of times a subject could finger tap in a set time); no correlation was found between nerve conduction velocity and grip strength.

These data suggest that tapping as a performance task involves intermittent neuromuscular activity, whereas grip strength is a static and steady effort less susceptible to interference from changes in nerve conduction. The study also lends support to the need for specifying performance tasks with precision so that responses can be better evaluated. The authors suggest that further work with sham immersion in cold water, in which subjects would be placed in cold water sufficient to be uncomfortable but not to induce reductions in core temperature, could contribute to a further understanding of the effects found in their study—for

example, whether the increased latency is associated with cold, with discomfort-induced distraction, or, as seems probable, with a combination of both elements.

Another critical performance element in divers affected by cold exposure is memory. Coleshaw and associates[18] reported that memory was impaired as core temperatures fell below 36.7°C in subject divers during a cold-chamber dive. Results revealed a 70% loss of memory for data at core temperatures of 34° to 35°C. A significant feature of this study was the assessment of recall of facts memorized during hypothermic exposures under warm-water conditions following the cold immersion. The subjects were comfortable, although still slightly hypothermic, but showed marked impairment in reasoning ability as well as in memory for facts learned under cold exposure. The 70% loss of memory in this study agrees with the measured loss of 75% of material learned in an underwater task when recalled on the surface post dive, as reported by Stewart of the Scripps Institution of Oceanography.[19]

From this review of selected studies on the effects of cold exposure on diver performance, it is clear that the major effect of cold appears to be on the cognitive, information-processing element so crucial in task completion underwater.

Diver Training

A specific and precise analysis of performance elements helps in the development of more effective diver training programs. The emphasis, particularly in industry and in the military, has recently shifted from selection of a candidate for a particular job, followed by training and field performance, to the reverse. It is now understood that the first step in performance enhancement is to do a task analysis that defines the requirements of the job and the specific behaviors needed to accomplish it. Thus, the tasks and the behaviors associated with the job are first specified, the most effective training procedures to perfect the needed skills are then developed, and, finally, those persons whose aptitudes and backgrounds seem most appropriate for such training are screened and selected.

An important part of training also involves learning responses that are designed not to accomplish a task but to avoid or eliminate events that might interfere with performance. For example, a survey by Egstrom[20] indicated that an important skill required in the field was the ability to escape from entanglement. The survey group consisted of commercial divers who had gone through formal training but had not been trained to escape from entanglement in lines or debris underwater. Such feedback is important, not only in assessing elements of performance, but also in determining procedures to add to standard teaching programs. Similarly, knowledge of emergency procedures is crucial for all divers—recreational as well as military and commercial—and instruction in handling emergencies should be a part of all training programs. Unfortunately, little formal emphasis has been placed on emergency procedures, and such procedures show virtually no standardization.

One of the most controversial emergency procedures is the emergency ascent. When a diver runs out of air or some untoward event occurs, it is desirable to return to the surface rapidly, but carefully, so that the risk of an air embolism is averted. Emergency ascent training has been controversial because many diving trainers and physicians think that the risks involved in teaching the emergency ascent combined with the low frequency of actual emergencies outweigh the possible benefits of such training. In 1977, the Undersea Medical Society convened a group of 35 diving authorities from all areas of diving to resolve the issue of the value of emergency-ascent training. The consensus report said that, if feasible, a controlled emergency ascent was desirable as an independent action on the part of the diver in difficulty.[21] If such independent action were not possible, a dependent action such as buddy breathing was recommended. The experts agreed that training was essential to the success of these emergency procedures and that there was a need for improved standardization of emergency ascent training and the use of equipment.

Over two decades have passed since that important workshop, and standardization of emergency procedures shows no significant progress. Indeed, the proliferation of equipment with differing control mechanisms, and therefore specifically different operational characteristics requiring different skills, has compounded the problem. This is largely a reflection of the failure of training agencies and equipment designers to agree on

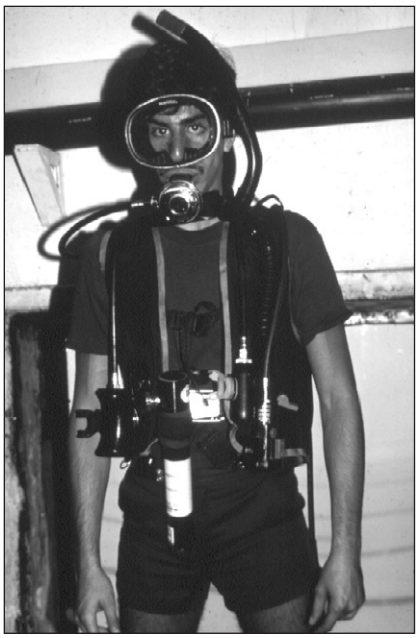

Figure 17–4. A diver wearing several alternative air sources. From right: a secondary regulator attached to the first stage (Octopus), a small cylinder of compressed air (SpareAir), a regulator attached to a buoyancy compensator, and a spare regulator attached to a second compressed air tank (pony bottle). (Photograph courtesy of Glen H. Egstrom.)

emergency procedures. The increase in the number of techniques available to a diver to recover from an out-of-air emergency has led to new levels of complexity as a result of the dozen or more relatively different responses that would be required to share air efficiently and effectively in an emergency. For example, an additional second-stage regulator is used frequently without any standardization in terms of placement of the alternate second stage, creating an uncertainty that could be hazardous during an emergency.[22] Other products include small, independent, compressed air supplies (pony bottles), a variety of autoinflator combinations with second-stage breathing devices built into the buoyancy compensator, a completely redundant regulator system on a single tank, or twin tanks with two regulators. Dozens of alternatives exist for an out-of-air problem that should not occur with well-trained divers. Figure 17–4 shows a few of the commonly used emergency air devices.

There is also an unfortunate lack of standardization of training programs for both commercial and recreational divers. Divers have no means of knowing precisely what skills and experience a fellow diver brings to the dive. The variety of courses offered to potential recreational divers ranges from a few hours' training to a thorough grounding in diving practice. Cooperation among the training agencies to develop optimal levels of training would solve many of these problems.

It is our opinion that one of the major problems in diver training today is the increased reliance on equipment. In the 1950s, when diver training formally began in the United States, there was a requirement for individual skills in water and for being in good physical condition. The diver learned proper weighting techniques so that the weight belt became a means of controlling buoyancy; the ability to use the weight belt (and jettisoning it when necessary) was a personal skill that, for example, also educated the diver about personal physical buoyancy characteristics. This personal skill has now been replaced by training in the use of a buoyancy compensator, which is a useful tool but not a substitute for self-reliance in the event of an emergency. The use of a variety of integrated weighting systems has also added to the number of variables the diver must be familiar with to enable effective emergency response. The increasing reliance on equipment rather than on personal skills also seems to include the belief, in some quarters, that the ability to swim is not necessary for diving; consequently, effective water skills, which were a requirement in the early days, have become a secondary consideration for many entry-level courses.

We have referred to emergencies frequently in the above discussion. The reasons are simple. If no mishaps occur, a dive can proceed safely despite a lack of skill and the level of training and the experience of the diver may not be crucial. However, during evaluation of a diving candidate, the physician or diving instructor should consider the possibility of an emergency and the need for a coping response. The candidate can then be evaluated not only in terms of the training in needed skills but, equally importantly, in terms of physical and emotional condition.

Diver Condition

Perhaps the most important single factor in underwater human performance is the condition of the diver. Given optimal diving conditions, properly planned diving operations, and well-engineered equipment, the diver's physical and psychological condition is criti-

cal to performance. The diver in good physical condition is more likely to perform successfully and to cope readily with problems encountered in the underwater environment. In other words, the diver should have sufficient endurance and strength to withstand the stresses of the underwater environment and the ability to handle the equipment, perform the task, and cope with emergencies that may arise. Behaviorally, it means that the diver in good condition must be confident in personal skills (which is, in very large measure, a function of adequate training) and thus be competent enough to cope with impending problems. The combination of effective training and good physical condition is critical for divers.

With regard to strength and endurance, the level of work in sport diving should be kept well within aerobic limits. Divers should be trained to recognize the signs of overexertion and to react appropriately to reduce the workload until respiration rate and pulse rate are back to a comfortable level. Exertion was a contributing factor in about 60% of the cases of decompression sickness between the years 1987 and 1993.[23]

One further comment about the diver's physical condition: In 1979, Eldridge[24] reported on a number of deaths in scuba divers, all in cold water and all involving males 35 to 55 years old. These deaths were called *sudden unexplained death syndrome,* and cardiovascular mechanisms in older males were thought to be involved. In a discussion of this report, Bachrach[25] suggested that vagotonic changes in older males should be a factor in diver evaluation. There have been recent reports of "sudden death" in divers, reviving an interest in this occurrence. When evaluating a diver's physical condition, especially that of older males, the diving physician should carefully consider factors such as the vasovagal state, factors that also involve behavioral elements. As Angel noted,[26] "Vasodepressor syncope occurs in our machismo culture more commonly among men than women, especially in settings in which the man feels the ambiance to be one of strong social disapproval of any display of weakness," a description of what has come to be known as *type A behavior.*

PROBLEM OF PANIC

Continued interest in the problem of panic is clear from discussions of this phenomenon. In his writing on technical diving, Nadeau[27] states that "we know the number one cause of dive accidents is panic, brought on by stress." Vikengo[28] recognized that "panic can be a life-threatening experience" in his work on panic control.

Of all the factors that interfere with effective performance underwater and, in many cases, lead to serious consequences such as death, panic is clearly foremost. Lack of confidence and competence and a less-than-adequate physical condition can lead to a loss of control, which appears to be a factor in most diving fatalities. In a study of sport diving fatalities by Sand,[29] panic was the major factor leading to fatality in 80% of the cases. Most diving researchers would agree that panic, with the diver losing control, is indeed the leading cause of fatalities; several authors have addressed the subject in recent years.[30–33] As Bachrach and Egstrom observed,[34] a certain level of apprehension may be expected in a diver experiencing a novel situation, such as entering a kelp bed for the first time. It is not possible for a training program to cover every conceivable hazard a diver may encounter, although, as noted, a good training program teaches emergency coping procedures and self-confidence in handling novel situations. Gathering advance information about the particular dive, such as the conditions, the dive plan, the necessary equipment, and the potential problems, can reduce apprehension and help performance. Emphasizing the positive aspects of the dive—the beautiful sights to be encountered, the opportunity to perform exciting tasks such as underwater photography, and the pleasure of being underwater—can create anticipation and reduce apprehension.

Although apprehension may be controlled by proper planning, adequate knowledge, good role modeling, and reassurance by the dive master, panic is a loss-of-control state in which divers perceive that they are losing control but are unable to extricate themselves from danger.

In most fatalities reported, equipment failure does not appear to be a major causative factor. Rather, the condition of the victims leads to the inference that human error is at fault. The victim usually has air left in the tank, the weight belt is still in place, and the buoyancy compensator is uninflated, all indications of a human error resulting from a lack of problem-solving skills, leading to panic.

An early sign, frequent in most panic situations, is a change in breathing rate and pattern that is often the first response to any type of stress (heat, cold, altitude, or diving) and is readily observable. In the apprehensive diver, the breathing pattern changes from smooth and regular to rapid, irregular, and shallow. The latter type of respiration produces an inefficient exchange of oxygen and carbon dioxide and leads to a sensation of needing more air, which may further exacerbate feelings of panic. Divers in a panic condition on the surface are frequently reported to struggle with their arms and legs to keep their heads above water; the struggle results in the head being higher, but it increases the workload on the body and increases the pulse and respiration rates. The struggling diver, supporting the weight of the head (approximately 17 lb), can sustain this workload for a matter of seconds; if the shoulders are out of the water, the weight to be supported could easily increase to 30 to 36 lb. The problem is compounded by exhaustion from the struggling. Divers have been reported to sink in a few seconds or, at the most, in about a minute. It has been assumed that the panic and struggle lead to cardiovascular consequences, including the potential for the sudden death syndrome mentioned earlier.

Another early sign of panic is *agitation,* which is associated with the change in breathing rate and pattern discussed earlier or with erratic movement. A diver in control moves along smoothly, with controlled and regular respiration and swimming movements, whereas jerky and irregular movements are associated with apprehension. Bevan,[32] from an observation of a diver on the verge of panic, noted that such a diver is apt to bring the knees forward and swim with short, jerky strokes rather than with smooth movements of the thighs and legs for propulsion.

Another sign of agitation concerns *orientation.* A controlled diver is oriented toward the water ahead, toward the bottom to observe sights and events, or toward the diving partner to maintain contact. The apprehensive diver, who may be approaching panic, is oriented toward the surface, often checking orientation toward the presumed safety of the surface and the dive boat. A diver in control makes frequent equipment checks to ensure enough air in the tank for a safe ascent and monitors bottom time carefully; the apprehensive diver checks equipment too often, manifests discomfort, and may be preoccupied with the gauges.

Preventing panic is largely a function of being in good physical condition and having adequate training, both of which lead to competence and confidence.[32] Acquisition of skills is highly important to appropriate coping responses in a problem situation. For example, it is not rare to witness a diver venturing into an unfamiliar surf condition and failing not only to engage in the appropriate behavior but also to respond to pertinent directions from the dive partner. Such behavior often results in failure to take simple precautions such as holding on to the facemask when the surf is about to break. The subsequent loss of the facemask caused by the surge of the water creates additional stress and leads to panic in what should be just a minor emergency.

An effective program sequence of diver training is crucial so that actual use of the required skills is built in to the diving program. A necessary skill in the water for the apprehensive diver is to be able to properly gain positive buoyancy, a priority for a fatigued or anxious diver. The diver who still has a snorkel or regulator in the mouth should be trained to backfloat with the mouthpiece removed for increased air passage.[34] Floating on the back, with the buoyancy compensator sufficiently inflated, allows the diver to rest and decrease struggling, which lessens exhaustion and the chance of submergence.

Predicting behavior is not a science, but some identifiable behavioral patterns may help diving physicians and diving instructors recognize persons who may have problems with panic.[22] The diving candidate most vulnerable to panic appears to have three characteristics:

- The person has a high level of generalized anxiety and is likely to respond with apprehension to a wide range of situations viewed as stressful. Psychologists call this general behavioral type *trait anxiety*,[35] as opposed to *state anxiety,* which is more situational.
- The vulnerable person has low self-confidence and low sense of competence and is unsure of being able to cope (has a sense of helplessness)[22] in situations of potential stress.
- The person has a low level of social support, an inability to work well with

other people, and an inability to provide or receive support from others. In some cases, this represents a type of machismo, a need to excel and succeed on one's own.

A proper screening of divers should identify characteristics that need attention from diving instructors and diving partners. Good training programs can develop skills and self-confidence, along with the recognition of the importance of the social support that can help ameliorate a tendency for anxiety.

DRUGS AND DIVING

The drugs considered here are not the medications divers may take, such as antihistamines, but controlled substances, such as marijuana and cocaine. The behavioral effects of such compounds are similar in many ways to those of alcohol, with profound effects on information processing, decision-making, and judgment. Marijuana, for example, is known to affect the sense of timing.[22] The awareness of the passage of time is a critical variable in dive planning—information necessary to monitor air consumption and decompression limits. Tzimoulis[36] comments on the problem of sleepiness as a result of marijuana intoxication. The impairment of information processing resulting from marijuana use may not be a problem in an uneventful dive, but the diver's ability to adapt to an emergency is severely diminished. Marijuana is also known to cause hypothermia. Studies by Pertwee[37] have shown that tetrahydrocannabinol, the active ingredient in marijuana, lowers body temperature by acting centrally (primarily in the anterior hypothalamus) to reduce heat production in response to cold.

Cocaine is reported to be "the most commonly abused central nervous system stimulant."[38] Cocaine affects information-processing abilities drastically, placing the diver at risk. Physiologically, the risks are major ones. As a stimulant, cocaine produces a hypermetabolic state that "may place the diver at risk of subsequent fatigue, mental depression, acidosis, and an inability to respond to life-threatening emergencies."[38]

A life-threatening physiologic effect of cocaine is alteration of cardiac function. In a report of the cardiac consequences of cocaine use, Isner and colleagues[39] showed that ventricular arrhythmias and sudden cardiac death were serious and often fatal consequences that, in recent years, have been sadly demonstrated in young, otherwise fit athletes.

Alcohol, the most widely studied drug, is known to reduce performance in accordance with the dose-response progression of the drug.[40] In addition to its well-documented impact on judgment, alcohol has a major effect on slowing the neural transmission capability of the central nervous system. Contrary to the popular view that a few drinks do not significantly affect performance is the finding that even small amounts of alcohol reduce the performance level of any task that requires information processing from more than one source. For example, a fundamental diving task is the monitoring of different aspects of a dive, which requires the diver's attention to be divided among several tasks; a performance function markedly affected by alcohol intake.

Type of Work: Changing Technologies

When evaluating human performance underwater, one must recognize that in recent years the role of the diver has changed significantly. In place of the "mud diver," who performed alone in a hard hat with simple tools, advances in diving technology have required more sophisticated working divers trained in the use of new diving techniques and procedures. From the working diver operating in a surface-supplied hardhat diving mode, to the saturation diver performing at greater depths for longer durations free of stringent decompression obligations, the changes in technology and the resulting physiologic stresses have been profound. With saturation diving, the necessity for mixed-gas breathing, such as use of helium-oxygen mixtures, to replace air at depth in itself created the problems of heat loss, speech intelligibility, and neurologic effects such as the high-pressure nervous syndrome (see Chapter 11). Different parameters of performance are affected by shallow and deep diving, as discussed by Biersner,[41] who noted that neuromuscular performance is most affected by deep dives, whereas the cognitive, information processing capabilities are less affected. The opposite appears to be true for shallow dives that use

compressed air rather than heliox as a breathing mix: Intellectual functions are affected more than motor responses.

The concerns about neurologic effects of deep diving, particularly the possible long-term effects on divers, were in part responsible for an increased emphasis on diving technology and the development of diving techniques such as 1 ata diving systems (e.g., JIM and WASP) and remotely operated vehicles (ROVs), which are now a standard part of diving procedures in deep-sea exploration and work. The utility of ROVs is demonstrated in such outstanding deep dives as the discovery of the *Titanic* and, in tandem with 1 ata systems such as the WASP, in the work on the deep archeological dive on the wreck of H.M.S. *Breadalbane*.

Busby[42] observes that "There seems to be no stopping the ROV now. The concept of sending a human being into the cold, dark high-pressure environment of the deep sea has had its day." He supports this view by referring to performance tasks accomplished using the ROV SCARAB in recovering the voice and data recorder from the downed Air India flight number 182 in 1985 at a depth of 6601 ft off the coast of Ireland. He notes that this was a task that could not possibly have been performed by a human diver, just as the work on the *Titanic* at a depth of 2 miles could not have been accomplished with human divers. Busby sums up his position by noting that the human diver has obviously not been replaced by the ROV in all circumstances but that "the ROV has demonstrated that the human diver need not be subjected to potentially hazardous situations and that it is frequently not economical to do so." In a real sense, this parallels developments in the space program in which, as Busby observes, "the romantic aspects of human involvement have given way to the practical and economic aspects of getting the job done."[42]

The human diver is not going to be replaced by a machine in all circumstances, and the need for human performance will continue. If the mechanical systems are a more practical alternative to humans for work at depth, for both physiologic and economic reasons, what does this mean for the study of human performance underwater? The emphasis has necessarily shifted from concerns about the physiology of deep diving to the development of improved training techniques; more effective decompression schedules for working dives; better equipment such as improved tools, diving suits, and other protective gear; and technologic aids for the diver. The last item includes the earlier-mentioned acoustic imaging techniques that help the diver "see" in turbid waters. The use of the operator in the 1 ata diving system WASP in tandem with an ROV on the wreck of H.M.S. *Breadalbane* was a breakthrough in diving technology because the human operator in the 1 ata system could communicate with topside personnel to direct the ROV with a camera to any position on the wreck.

Thus, the future of human performance underwater largely rests on the effective use of technology and human skill.

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CHAPTER 18 Medical Supervision of Diving Operations

Edward T. Flynn, Jr

Physicians and diving medical technicians are an important part of most military and civilian diving organizations. Long before diving starts, they review diving techniques and procedures for safety and determine who is medically qualified to dive. Once diving commences, these health professionals turn their attention to the differential diagnosis and treatment of diving accidents. When diving is over, they conduct debriefings and institute monitoring and surveillance programs to ensure the health of divers. These results provide feedback for new strategies to prevent diving accidents and new criteria for selecting divers for future operations.

This chapter focuses on the actual diving operation itself—specifically, what on-site medical personnel need to know to be effective. Two areas are highlighted: predive planning and differential diagnosis of diving accidents. Treatment of diving accidents is not discussed because this topic is addressed in the preceding chapters.

PREDIVE PLANNING

Preventing a diving accident is the number one goal of medical personnel involved in a diving operation. This goal is achieved primarily through meticulous predive planning. Medical personnel review dive plans to ensure that equipment, tools, gas mixtures, and decompression procedures are appropriate for the dive and that the divers are fully trained. Lessons learned from previous operations are carefully considered. Each phase of the dive is then reviewed to identify potential problems and to confirm proper contingency plans. Careful attention is paid to mitigating environmental and occupational hazards as well as to scheduling dive time to maximize diver efficiency and safety. Immediately before the dive, the divers are examined to ensure their fitness to dive. The following discussions elaborate on each of these key aspects of dive planning.

Selection of Underwater Breathing Apparatus

Although a few dives are performed by breath holding, the vast majority employ some type of underwater breathing apparatus (UBA). The type of UBA selected and the gas mixtures used depend on many factors, including the depth and duration of the dive, the type of work to be performed, and the amount of decompression required.

UBAs are generally classified in three ways:

- Whether the unit is self-contained (i.e., scuba) or has an umbilical gas supply
- Whether it employs air, oxygen, or mixed gas (e.g., nitrogen-oxygen or helium-oxygen mixtures)
- Whether it has an open-circuit, a semiclosed-circuit, or a closed-circuit breathing loop

Semiclosed- and closed-circuit systems are commonly called *rebreathers.* Chapters 3 and 6 and other standard references describe these systems.[1–3] Only those features important to dive planning, accident prevention, and medical diagnosis are reiterated here.

AIR DIVING

Air is the most economical gas for diving because it can be compressed on-site and made available in unlimited quantities.

The opinions in this chapter are those of the author and do not necessarily reflect the views of the U.S. Navy or the naval service at large.

Consequently, most of the world's dives are air dives. Most air is compressed with oil-lubricated compressors that introduce the possibility of diver's air contamination with carbon dioxide, carbon monoxide, and various gaseous and particulate hydrocarbons. Even with a perfectly functioning compressor, improper placement of the compressor inlet near exhaust fumes (including those of the compressor itself) can introduce contaminants into the diver's air supply.

Nitrogen narcosis limits the depths that can be reached on air. Nitrogen narcosis begins to impair an air diver's performance at approximately 132 fsw (40 msw). Deeper than 200 fsw (60 msw), impairment becomes marked. From a practical standpoint, 200 fsw can be considered the depth limit for air diving (see Chapter 11 for further discussion).

The depth and duration of air diving is also limited by the absorption of nitrogen by body tissues, which creates a requirement for decompression. Decompression times become increasingly long as dive depth and duration increase. Oxygen toxicity, on the other hand, is almost never a limiting factor. Increasing levels of nitrogen narcosis and lengthy decompression times generally prevent the diver from diving deep enough and long enough to exceed oxygen-exposure limits.

Three types of UBAs are commonly used for air diving: open-circuit demand scuba, surface-supplied demand helmets, and surface-supplied free-flow helmets.

Open-circuit demand scuba is the most widely used system. Compressed air from a high-pressure cylinder is inhaled through a demand regulator (i.e., one that supplies air on demand) and is subsequently exhaled into the water. The breathing circuit is called "open" because no gas is rebreathed. Dive time is limited by the volume of air carried by the diver and is inversely proportional to the diver's depth and respiratory minute volume. Because of the limited air supply, careful planning is necessary for dives requiring decompression, and no-decompression dives are strongly encouraged. Dives with open-circuit scuba are generally restricted to 130 fsw (40 msw) or less because of the limited air supply.

The *surface-supplied demand helmet* is the most common helmet in use today. Unlike scuba, the surface-supplied demand helmet provides the diver with head protection, communications with the surface, and an umbilical air supply that allows long-duration dives. The umbilical air supply feeds a demand regulator that works just as it does in scuba. The diver inhales from the regulator and exhales into the water. An oronasal mask keeps respiratory dead space to a minimum. The flow resistance of modern demand regulators is low enough that alveolar hypoventilation and carbon dioxide retention are rare, even with heavy work. If the demand regulator fails, the diver can open a valve that allows air to flow freely through the helmet. If the umbilical air supply fails, the diver can open another valve that connects the regulator to a reserve cylinder of air carried on the back. Air diving with the surface-supplied demand helmet is generally restricted to 200 fsw (60 msw) or less because of the incapacitating effects of nitrogen narcosis.

The *surface-supplied free-flow helmet* is the classic diving system of yesteryear. It is now little used, except for certain specialized operations such as diving in contaminated water. It is an open-circuit system. Compressed air flows through the helmet continuously at rates of 30 to 180 L/min. The diver manually adjusts airflow to match workload and prevent accumulation of carbon dioxide in the helmet. Proportionally higher flows are needed for work than for rest. The principal problem with this system is accumulation of carbon dioxide in the helmet during heavy exercise. High noise levels associated with high airflow may also be a problem.

OXYGEN DIVING

Pure oxygen may be used as a diving gas instead of air, but only for very shallow dives. Pure-oxygen diving is primarily a military specialty, but its use is increasing in the recreational and scientific diving communities. One hundred percent oxygen is rebreathed in a closed-circuit breathing system. No gas escapes from the apparatus, and hence there are no bubbles to attract unwanted attention. Fresh oxygen sufficient to meet metabolic requirements is supplied to the rebreathing circuit from a high-pressure cylinder. Exhaled carbon dioxide is absorbed in a chemical bed. Dive time is limited by the size of the cylinder oxygen supply and the size of the absorbent bed. Dive time varies inversely with the diver's workload, as it does in open-circuit scuba, but is independent of the diver's depth (see Chapter 29 for further discussion).

Table 18–1. Representative depth-time limits for 100% oxygen diving

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

Adapted from *U.S. Navy Diving Manual*, Revision 4, Change A. Publication No. SS521-AG-PRO-010, Naval Sea Systems Command. Washington, D. C., Department of the Navy, March 2001.

Central nervous system (CNS) oxygen toxicity is the biggest problem with pure-oxygen diving. To avoid trouble, divers must stay within prescribed depth-time limits, such as those shown in Table 18–1. These limits vary from apparatus to apparatus depending on the design of the oxygen makeup system and the purging procedures employed at the beginning of the dive to eliminate nitrogen from the breathing loop. The apparatus must be purged to avoid hypoxia during the dive. If a significant amount of nitrogen remains in the breathing loop after purging, all of the oxygen in the breathing loop may be consumed before fresh oxygen is added. Purging procedures for each UBA are designed specifically to avoid hypoxia while providing a level of residual nitrogen that reduces the likelihood of CNS oxygen toxicity. Thus, the allowable depth-time limits may vary from apparatus to apparatus.

Carbon dioxide accumulation in the rebreathing circuit resulting from a failed absorbent bed is another major concern with pure-oxygen diving. As with all closed- and semiclosed-circuit UBAs, the average life of the CO_2 canister is established through empirical testing, then a healthy safety factor is applied. Dive planners must be aware of these canister limits when planning dives with oxygen rebreathers. Unfortunately, faulty packing of the canister or leakage of water into the canister may cause early canister "breakthrough." Decompression sickness (DCS) is never a problem during pure-oxygen diving because there is not enough nitrogen in the breathing loop to create a decompression requirement. Pure-oxygen diving is generally limited to a depth of 50 fsw (15 msw) or less because of the risk of CNS oxygen toxicity.

NITROGEN-OXYGEN (NITROX) DIVING

Dives may be performed with nitrogen-oxygen mixtures having higher oxygen content than air. The principal advantage these mixtures offer over air is a reduced decompression requirement. Nitrox diving may be performed using open-, semiclosed-, or closed-circuit systems.

Open-Circuit Systems

Scuba and surface-supplied demand helmets are by far the most common systems employed for nitrox diving. With open-circuit systems, the gas mixture may be premixed in high-pressure cylinders or blended on-site by mixing pure oxygen with air. Two gas mixtures are commonly used in open-circuit operations: *32% oxygen, balance nitrogen* and *40% oxygen, balance nitrogen.* However, any gas mixture with an oxygen fraction of 22% to 50% may be used. To ensure safety, all gases must be analyzed for oxygen content before use.

The main concern with open-circuit nitrox diving is avoidance of CNS and pulmonary oxygen toxicity. To use oxygen exposure tables, it is first necessary to calculate the partial pressure of oxygen (P_{O_2}) in the breathing mixture. P_{O_2} is the product of the mixture's oxygen fraction and the absolute pressure at the dive site:

$$P_{O_2} = F_{O_2} \times PB$$

where P_{O_2} = partial pressure of oxygen, F_{O_2} = fraction of oxygen in the mixture (percentage/100), and PB = absolute pressure at the dive site.

Example: For a dive to 132 fsw (5 ata) on 32% oxygen:

$$P_{O_2} = 0.32 \times 5 = 1.60 \text{ atm}$$

Table 18–2 gives the P_{O_2}-exposure time limits established for nitrox diving by the National Oceanic and Atmospheric Administration (NOAA).[2] For each P_{O_2}, two limits are shown: a single dive exposure limit and a 24-hour cumulative exposure limit. For P_{O_2} above 1 atm, the two limits are based on avoidance of both CNS and pulmonary toxicity; for P_{O_2} below 1 atm, the limits are based on avoidance of pulmonary toxicity. The U.S. Navy imposes similar limits but does not allow open-circuit nitrox diving at a P_{O_2} higher than 1.4 atm. Setting oxygen exposure limits involves both a scientific assessment

Table 18–2. NOAA oxygen exposure limits for nitrogen-oxygen, helium-oxygen, and helium-nitrogen-oxygen diving

Oxygen Partial Pressure (atm)	Maximum Single Exposure Time (min)	Maximum Exposure per 24 hours (min)
1.60	45	150
1.55	83	165
1.50	120	180
1.45	135	180
1.40	150	180
1.35	165	195
1.30	180	210
1.25	195	225
1.20	210	240
1.10	240	270
1.00	300	300
0.90	360	360
0.80	450	450
0.70	570	570
0.60	720	720

Adapted from Joiner JT (ed): NOAA Diving Manual: Diving for Science and Technology, 4th ed. Flagstaff, AZ, Best, 2001.

of the risk and an organizational judgment of how much risk is acceptable in a given situation. The acceptable risk may vary from job to job.

Semiclosed-Circuit Systems

Semiclosed-circuit scuba was invented shortly after World War II to support clandestine military operations at depths beyond the range of pure-oxygen diving. Semiclosed-circuit scuba diving is now widely practiced in the military and to a lesser extent in the recreational diving community. A nitrogen-oxygen mixture with an oxygen fraction ranging from 32% to 70% is metered into the breathing loop at a flow rate that meets the diver's metabolic demand for oxygen. Fresh gas may be added to the breathing loop either at a constant mass flow rate (most systems) or in direct proportion to respiratory minute volume. Exhaled carbon dioxide is absorbed in a chemical bed. Although most of the gas in the loop is rebreathed, a small amount continually escapes to the water, hence the term *semiclosed.* In constant mass flow systems, the oxygen fraction in the breathing loop varies inversely with the diver's workload, being highest at rest and lowest during hard work. Dive time depends on the volume of supply gas carried by the diver and the size of the absorbent bed. For most semiclosed systems, dive time is independent of dive depth, a key advantage of semiclosed-circuit scuba over open-circuit scuba.

Several medical problems can be anticipated with semiclosed-circuit scuba. These include CNS oxygen toxicity (if P_{O_2}-exposure time limits are exceeded), hypoxia (if the mixed-gas injector system fails), and hypercapnia (if the CO_2-absorbent bed fails or if the diver hypoventilates to reduce respiratory workload). Alveolar hypoventilation is a particular problem for rebreathers that impose not only flow-resistive but also elastic and hydrostatic loads on the respiratory system. The hyperoxic gas mixture in the rebreathing circuit contributes to the problem by decreasing the CO_2 sensitivity of respiratory control centers.

CNS oxygen toxicity is the primary factor limiting the depth to which semiclosed-circuit scuba may be used. Dives must be kept within the P_{O_2}-exposure time limits deemed acceptable by the organization. For maximum depth calculations, it is customary to use the oxygen fraction in the supply gas rather than in the breathing loop to compute the P_{O_2} exposure. Because the oxygen fraction in the breathing loop is always lower than in the supply gas, this custom introduces a degree of conservatism that helps compensate for the likelihood of some increase in arterial CO_2 tension during the dive—a potent risk factor for CNS oxygen toxicity. An alternative approach is to use actual loop P_{O_2} values and adopt lower P_{O_2}

limits for semiclosed-circuit operations than for open-circuit operations. A major limitation of this approach is that breathing-loop P_{O_2} is continuously variable and hard to characterize. To stay within oxygen limits, the oxygen fraction in the supply gas of semiclosed-circuit units must be reduced as the diver's depth increases.

Closed-Circuit Systems

Closed-circuit nitrogen-oxygen scuba offers all the advantages of semiclosed-circuit scuba plus longer dive times and tighter P_{O_2} control. These units are used both by the military and by recreational divers engaged in specialized forms of diving, such as deep diving and cave diving. Unlike semiclosed-circuit scuba, in which the P_{O_2} varies with both depth and workload, these highly sophisticated UBAs maintain the P_{O_2} in the breathing mixture at a constant value independent of depth and workload. The UBA has two independent gas supplies: a cylinder of pure oxygen and a cylinder of inert gas (either nitrogen or helium). As oxygen is consumed from the breathing loop, it is replaced molecule for molecule from the cylinder containing pure oxygen. Inert gas is added to the breathing loop only when needed to maintain constant loop volume. The major addition of inert gas occurs during descent to offset Boyle's law compression of the breathing loop. Periodic minor additions compensate for gas leaks from the breathing loop. Exhaled carbon dioxide is absorbed in a chemical bed.

An oxygen sensor in the loop controls addition of oxygen to the breathing loop. The P_{O_2} may be controlled at any value, but the common choices range from 0.7 to 1.6 atm. CNS oxygen toxicity is of concern with these units. Because P_{O_2} remains constant both on the bottom and during decompression, divers are exposed to elevated P_{O_2} longer than in dives with open-circuit or semiclosed-circuit UBAs. Therefore, these units require more conservative oxygen limits. The U.S. Navy has adopted a limit of 1.3 atm for both nitrogen-oxygen and helium-oxygen closed-circuit diving.[1] Other organizations have endorsed somewhat higher limits (1.4 to 1.6 atm). The choice depends on the risk the organization is willing to take.

Dive time with closed-circuit mixed-gas scuba is limited by the size of the oxygen cylinder and the absorbent bed. Dive time is independent of dive depth. Problems that can be anticipated with this apparatus are similar to those expected with semiclosed-circuit scuba. They include hypoxia (if the oxygen addition system fails shut), CNS oxygen toxicity (if the oxygen addition system fails open), and hypercapnia (if the CO_2-absorbent bed fails or if the diver hypoventilates to reduce respiratory workload). When nitrogen is used as the diluent inert gas, nitrogen narcosis limits dive depth to approximately 150 fsw (45 msw). This limit varies somewhat with the P_{O_2} set point chosen.

HELIUM-OXYGEN (HELIOX) AND TRIMIX DIVING

To avoid nitrogen narcosis, dives deeper than 200 fsw (60 msw) require a switch from nitrogen-oxygen mixtures to helium-oxygen mixtures. The reduced density of helium compared with nitrogen also helps alleviate problems with breathing resistance and prevents alveolar hypoventilation and carbon dioxide retention until great depths are attained. A switch from nitrogen-based to helium-based mixtures should be considered whenever inspired gas density exceeds 6 g/L, even if narcosis is not yet a problem.

Gas contamination problems are unusual in helium-oxygen diving because the component gases are meticulously monitored for purity. CNS oxygen toxicity is a major concern. Oxygen exposure limits, such as those shown in Table 18–2, should not be exceeded. The U.S. Navy does not allow heliox diving at a P_{O_2} higher than 1.3 atm.

Helium-oxygen dives can be divided into those of 300 fsw (90 msw), shallower dives, and deeper dives. For dives to 300 fsw, the surface-supplied demand helmet is the most common UBA employed. For added diver safety, an open diving bell is frequently used in conjunction with the helmet. Semiclosed-circuit scuba and closed-circuit constant P_{O_2} scuba may also be used in specialized applications. These UBAs are often equipped with open-circuit "bail-out" systems for these deeper dives.

Several different gas mixtures may be employed during the course of a dive to 300 fsw with an open-circuit helmet system. The descent is typically begun with a mixture having an oxygen fraction near 21%. During descent or on the bottom, the diver *downshifts*

to a mixture with a lower fraction of oxygen (typically 10% to 16%) to remain within the oxygen exposure limits. Decompression from these dives may involve successive switches to air, 50% nitrogen/50% oxygen, and 100% oxygen during ascent. This is called *upshifting* because the oxygen fraction is successively increased during the decompression to reduce decompression time. The on-site use of multiple gas mixtures presents the constant danger of acute hypoxia or oxygen toxicity due to inadvertent shifts to a mixture with too low or too high an oxygen fraction for the depth.

Some organizations advocate helium-nitrogen-oxygen mixtures *(trimix)* rather than helium-oxygen mixtures in the depth range of 150 to 300 fsw. Advantages attributed to trimix include improved diver thermal comfort, improved communications, and reduced decompression time, especially for short dives. As with helium-oxygen mixtures, the oxygen fraction in trimix is selected to keep the diver within the oxygen exposure limits. The nitrogen fraction is selected so that the sum of the oxygen and nitrogen partial pressures at depth does not exceed a specified *equivalent narcotic depth* on air, usually 70 to 120 fsw. The balance of the mixture is helium. A typical mixture for a dive to 297 fsw (10 ata) might be 16% oxygen, 24% nitrogen, 60% helium. The equivalent narcotic depth on air for this dive is 99 fsw (4 ata). The oxygen and nitrogen partial pressures are added when computing equivalent narcotic depth under the assumption that oxygen and nitrogen have equal narcotic potencies. This is a conservative assumption.

SATURATION DIVING

Although the open-circuit demand helmet is fully capable of supporting dives deeper than 300 fsw, the in-water decompression requirement for such dives becomes prohibitive. Also, the risk of serious DCS while a diver is still in the water becomes significant. Dives deeper than 300 fsw are generally performed with saturated divers in a deep-diving system. The deep-diving system consists of a *deck decompression chamber,* in which the divers live under pressure for up to 1 month at a time, and a *personnel transfer capsule (PTC)* or diving bell, in which the divers are transported under pressure to and from the worksite. The divers exit the PTC on umbilically supplied UBAs. These UBAs are usually open-circuit demand helmets fitted with gas-reclaiming devices to return expired gas to the PTC or to the surface for carbon dioxide removal, oxygen addition, and ultimate return to the diver. The reclaiming devices are fitted with safeguards that prevent sudden depressurization of the helmet and the consequent squeeze. While saturated in the deck decompression chamber, the divers breathe a helium-nitrogen-oxygen mixture with an oxygen partial pressure of 0.4 to 0.6 atm. Gas mixtures for excursion dives outside the PTC are generally helium-oxygen mixtures and are chosen to avoid both hypoxia (during excursions above the depth of the PTC) and oxygen toxicity (during excursions below the depth of the PTC). The U.S. Navy restricts the P_{O_2} on downward excursions to a maximum of 1.25 atm. Other organizations have set different limits. Saturation diving techniques may also be used for long jobs in shallow water. These dives use air and other nitrogen-oxygen mixtures. Chapter 6 discusses saturation diving, including the techniques used for shallow operations.

Table 18–3 summarizes the major types of available UBAs, their principal uses, their depth limits, and the associated medical problems.

Selection of Protective Garments

After the UBA itself, protective garments are the most important piece of the diver's equipment. These garments should be selected to protect against thermal insult, dangerous marine life, cuts and abrasions, and chemical and microbiologic pollution.

The requirements for thermal protection in cold water are fairly well defined and are available in several reference sources.[1,2,4] Generally, thermal protection is afforded by wet suits, dry suits, or hot-water suits. *Wet suits* are sufficient for short, shallow operations, even in icy waters. They can be donned speedily, interfere minimally with the diver's mobility, and require little weighting. Because of compression of the closed-cell neoprene foam, the insulating value is reduced at depth: Consequently, wet suits offer the greatest thermal protection near the surface. *Dry suits* are required for deeper,

Table 18–3. Commonly available underwater breathing apparatus and associated medical problems

Type	Depth Limit (fsw)	Hypoxia	Hypercapnia	Oxygen Toxicity	Nitrogen Narcosis	Contaminated Gas	Decompression Sickness	Principal Uses
Demand scuba	130 (N_2-O_2*)	0	+	++	+	+	+	Recreation, search and recovery, scientific diving
	130 (air)	0	+	0	+	+	+	
Surface-supplied demand helmet	130 (N_2-O_2*)	0	+	++	+	+	+	Salvage, ship husbandry, underwater construction
	200 (air)	0	+	0	++	++	++	
	300 (He-O_2*)	0	+	++	0	0	++	
Free-flow air helmet	200	0	++	0	++	++	++	Salvage, underwater construction
100% oxygen scuba	50	+	++	+++	0	0	0	Combat swimming, recreation
Semi-closed mixed-gas scuba	130 (N_2-O_2*)	++	++	++	+	0	+	Combat swimming, mine clearance
	180 (He-O_2*)	++	++	++	0	0	++	
Constant Po_2 mixed-gas scuba	150 (N_2-O_2†)	+++	++	+	++	0	++	Combat swimming, mine clearance, recreation
	300 (He-O_2†)	+++	++	+	0	0	++	
Deep-dive system with demand helmet	1500 (He–O_2‡)	0	++	+	0	0	+	Deep-water salvage, deep-water search and recovery, deep-water construction

*32% oxygen.
†Po_2 = 0.7—1.4 ata.
‡Po_2 = 0.4–-1.6 ata.
0, improbable; +, possible; ++, probable; +++, very probable.

longer missions. These suits are more cumbersome; they reduce the diver's mobility and require more weighting. In addition, they require a source of inflation gas to avoid suit squeeze on descent and to maintain the insulating value of the undergarments. They are prone to leaks. Despite these limitations, dry suits offer superior insulation compared with wet suits and the insulation is not reduced at depth. *Hot-water suits* offer the best thermal protection but require support equipment not available at many dive sites. Although hot-water suits prevent the intense shivering and painfully cold hands and occasionally experienced by divers wearing wet or dry suits, hot-water suits can be associated with an insidious, asymptomatic hypothermia that leads to undetected mental incapacitation. Burns from improperly regulated hot-water sources may also occur. With helium-oxygen dives deeper than 400 fsw, heating of respiratory gas is required in addition to thermal protection of the body.[5] (See Chapter 13 for a discussion of thermal stress and diving.)

The requirement to dive in hot water is rapidly increasing in the commercial diving sector. Typical locations are cooling water outfalls and nuclear reactors. Water temperatures may exceed 50°C. For exposures of less than 1.5 hours at water temperatures below 40°C, open-circuit free-flow air helmet systems are effective.[6] The large flow of incoming cool air provides sufficient convective cooling. Above a water temperature of 40°C, water-cooled suits are required.

For diving in waters polluted with chemicals or microorganisms, a dry suit and helmet-breathing system are preferred. The suit should have attached hard-sole boots and attachable dry gloves; the helmet should mate to the suit at the neck without leaks and must cover the entire head. Ordinary demand scuba regulators are considered unacceptable for this type of diving because they allow seepage of water around the mouth and through the exhaust valve. Full-face masks are also considered unacceptable; they leave part of the head exposed and may become dislodged. The helmet should be equipped with a gas reclaimer on the exhaust side or with a double exhaust valve to prevent backflow of water. For demand helmets, the inhalation diaphragm should be isolated from the water to prevent contaminated water from seeping through pinhole leaks. Vulcanized rubber suits have proved to be easy to disinfect after exposure to bacterially polluted water. Wet suits and nylon-coated dry suits, on the other hand, have proved to be very difficult to decontaminate. Because of the extreme danger, divers should never be allowed to dive in water contaminated with acetic anhydride, acrylonitrile, bromine, carbon tetrachloride, chlordane, cresol, dichloropropane, epichlorohydrin, ethyl benzene, methyl chloride, methyl parathion, perchloroethylene, styrene, trichloroethylene, or xylene.[2,6] Recent publications have summarized techniques for diving in contaminated water.[2,6–8]

Regardless of the need for thermal or pollution protection, divers should always wear leather or rubber gloves and hard-soled boots or booties to protect the hands and feet from trauma.

Environmental and Occupational Factors

Predive planning must always consider environmental and occupational factors. *Environmental factors* to be considered include surface weather, sea state, water temperature, currents, surge, underwater visibility, and dangerous marine life. Immediate access to the surface may be restricted; typical examples include diving in tanks, pipes, caves, and under ice. These dives require special preparation. *Occupational factors* include the presence of chemical and biologic pollutants in the water; the noise produced by tools, sonars, and other sound sources; and the potential for injury related to electric shock, radiation exposure, and mechanical trauma.

Noise exposure is of particular concern to divers because it may lead to long-term debilitating hearing loss. High levels of low-frequency noise may also cause diver disequilibrium and pulmonary damage. Noise may be continuous or impulsive, and different exposure standards apply to each type. The type of equipment used by the diver is an important determinant of allowable exposure levels. A helmeted diver whose head is surrounded by gas will tolerate less sound than a diver whose head is surrounded by water. The helmeted diver hears by air conduction; the wet diver hears by bone conduction, which is less sensitive. Wet-suit hoods offer significant protection against underwater sound and should be used whenever noise levels are high.

When interpreting noise exposure guidance, it is important to understand that the decibel scales used to quantify sound pressure level (SPL) may differ from one application to the next. SPLs referenced to 20 micropascals (μPa) are commonly used for SPL measurements in air; SPLs referenced to 1 μPa are commonly used for measurements in water. SPLs with a 20 μPa reference will be 26 dB lower than SPLs with a 1 μPa reference.

A "doubling rule" is commonly used to guide noise exposure. The maximum allowable SPL is determined experimentally for one exposure time. SPLs for other exposure times are then calculated using the doubling rule selected. The U.S. Navy uses a 4 dB doubling rule for diving applications.[1] Exposure time can be doubled for each 4 dB reduction in SPL from the reference condition. For example, if 105 dB is determined to be the maximum safe SPL for a 15 min exposure, the corresponding safe SPLs for 30 min and 1-, 2-, 4-, and 8-hour exposures are 101, 97, 93, 89, and 85 dB, respectively. The doubling rule can be used if detailed guidance for a particular application is lacking.

Electricity is used underwater for welding; for powering tools, lights, and cameras; and for cathodic protection of ships and underwater structures. The hazards of underwater electrical fields have been reviewed by Bove and others.[9,10] Much remains to be learned about tolerable exposure levels and mechanisms of injury. The U.K. Association of Offshore Diving Contractors has established standards for the safe use of electricity underwater based on current knowledge.[11] These standards have been adopted worldwide. A recent U.S. Navy publication provides additional information specific to welding.[12] Low-voltage devices, properly functioning ground fault interrupters, and avoidance of 50 to 60 Hz alternating current are key components of underwater electrical safety. Impressed current cathodic protection systems are a special situation. These systems should be secured during diving operations whenever possible. If they cannot be secured, divers should be advised of safe standoff distances and wear a full wet or dry suit with hood and gloves when working in the vicinity of the anode, dielectric shield, or reference cell.

Mechanical trauma is a major concern whenever divers are working around wrecks and underwater structures and when using power tools such as water jets, jackhammers, bolt cutters, spreaders, grinders, and underwater chainsaws. Divers must be particularly aware of various sources of suction that may trap and kill them. Nichols and colleagues describe a fatality related to suction injury in a water treatment facility.[13] All sources of suction must be secured before diving begins. Underwater blast is also of concern.[14] Blast may be a planned part of the diving operation or may result unexpectedly from the accumulation of explosive gases in enclosed spaces. *Liveboating,* diving from a vessel while it is underway, can be particularly hazardous if safety precautions are not followed.[15]

Divers sometimes work in enclosed, gas-filled spaces, such as submarine ballast tanks and underwater welding habitats. The purity of the gas in these spaces is of concern because divers may doff their UBA and breathe the ambient atmosphere while working. Gas purity must be measured before divers enter the space, and in some cases during the operation itself, if the work adds contaminants to the space. Special care must be taken not to contaminate the atmosphere of saturation diving bells and deck chambers by bringing back contaminants from the worksite. In some instances, divers have to wear special protective garments that can be shed before they enter the diving bell.

Proper scheduling of dives is essential to avoid diver fatigue, to remain within oxygen limits, and to avoid excessive decompression obligations. The most difficult decision is how to divide the total bottom time required for completion of the mission into discrete individual dive packets to maximize efficiency and safety. Despite advances in decompression technology, the risk of DCS with most decompression tables increases as the depth and bottom time of the dive increase. A greater number of shorter dives, therefore, may prove safer in accomplishing the mission than a lesser number of longer dives. When decompression times in the water become long, it may be prudent to switch to surface decompression techniques or use hyperoxic decompression mixtures in the water.

Medical Equipment and Supplies

Predive planning also includes preparation to diagnose and treat diving accidents if they arise. Proper diagnostic and therapeutic

equipment must be available on-scene and must be checked for operability. It is also important to establish communications with backup medical and recompression facilities and to plan for possible patient transport to these facilities. A frequently overlooked piece of emergency equipment is a source of 100% oxygen that the diver can breathe while being transported.

Predive Physical Examination

Other chapters of this book extensively address the standards for physical selection of divers. The general requirement is for a vigorous, emotionally stable person who is free of cardiovascular, pulmonary, neurologic, and otolaryngologic disease. The on-scene physician or diving medical technician should perform a predive physical examination to rule out temporary conditions that may disqualify the diver. Such conditions include acute upper or lower respiratory tract infections, sinusitis, otitis media, alcohol or drug intoxication, hangover, excessive fatigue, gastrointestinal upset, recent orthopedic trauma, and seasickness. The diver should also be questioned about the use of prescription drugs and over-the-counter medications and dietary supplements.

The U.S. Navy and NOAA diving manuals discuss planning of military and civilian scientific dives in great detail.[1,2] Much of this information is also applicable to recreational diving. The Association of Diving Contractors *Consensus Standards for Commercial Diving and Underwater Operations* gives specific requirements for diving in the commercial sector.[15]

COMMON MEDICAL PROBLEMS IN DIVING OPERATIONS

Other chapters detail the medical problems unique to diving. Some of these conditions are common, others are rare, and a number are related to specific types of diving equipment. One of the most useful ways to organize this information is to review what can go wrong during each phase of the dive. The value of this approach is twofold. First, the phase of the dive in which a problem occurred is one of the most important clues to diagnosis. Second, knowledge of what can go wrong during each phase of the dive is essential for anticipating problems and developing contingency plans.

Medical Problems During Descent

The following medical problems should be considered during descent.

BAROTRAUMA

By far, barotrauma is the most likely injury during descent. *Middle-ear squeeze* is extremely common and occurs more frequently than do other forms of barotrauma. Up to 10% of divers may be affected at any given time. The presenting symptom of middle-ear squeeze is pain. The diagnosis rests on the characteristic symptoms and the presence of a hemorrhagic tympanic membrane upon surfacing. *Barotrauma to the paranasal sinuses* is the second most common form of barotrauma during descent, but it is considerably less frequent than middle-ear squeeze and affects less than 1% of divers at any given time. Pain in the involved sinus on descent and a bloody nasal discharge on ascent are diagnostic. In the case of *maxillary sinus barotrauma,* pain may have been referred to the upper molars. *Face-mask squeeze, dry-suit squeeze, tooth squeeze,* and *external-ear-canal squeeze (reversed ear)* are all relatively uncommon and are easily diagnosed. *Lung squeeze* is exceedingly rare because of better buoyancy and descent control in modern equipment and the all-important presence of nonreturn valves in umbilically supplied equipment. Few physicians ever see this injury.

Frequent and forceful attempts to equalize middle-ear pressure during descent may produce transient vertigo—generally, just as the diver reaches the bottom and performs the last equalization maneuver. This condition is called *alternobaric vertigo (ABV) of descent.* Difficulty in clearing the ears may also produce actual inner-ear injury, a condition known as *inner-ear barotrauma (IEBT).*[16,17] A perilymph fistula through the oval or round window may or may not be a part of this injury. The symptoms of IEBT are

sustained neurosensory hearing loss and vertigo (see Chapter 22).

CALORIC VERTIGO

Transient caloric vertigo may occur when a diver enters the water or during the early phases of descent if cold water enters one external ear canal faster than the other. Obstruction of one ear canal by cerumen, otitis externa, or a tight-fitting wet-suit hood is generally the cause. Sudden transient caloric vertigo may also occur if the tympanic membrane ruptures from barotraumatic injury and cold water enters the middle ear.

HYPERCAPNIA

Transient arterial hypercapnia with attendant cerebral symptoms may occur during very fast descents because of rapid compression of alveolar gas. This condition may also occur in the older-style helmet systems without neck dams. In the latter instance, carbon dioxide accumulates in the diving suit while the diver is on deck before the dive; the gas is then forced into the helmet when the diver enters the water, and the gas is compressed by the descent. This problem is quickly remedied by ventilating the helmet. Hypercapnia is a much more common problem on the bottom. The factors that can lead to hypercapnia in diving are discussed at length under Medical Problems on the Bottom.

HYPOXIA

In air diving, hypoxia cannot occur during descent because the oxygen fraction is fixed at 21% and air-diving systems are all open-circuit. In open-circuit mixed-gas diving, however, hypoxia may occur at the surface or in the early phases of descent if the gas mixture has too low an oxygen fraction for the depth. Divers often have become unconscious in the first few moments of a dive when the gas supply was inadvertently switched to pure helium or nitrogen. Hypoxia near the surface is a particular problem for most semiclosed systems. Heavy exertion near the surface rapidly lowers the P_{O_2} in the breathing loop, often into the hypoxic range. The risk of hypoxia from this mechanism lessens as the depth increases.

OXYGEN TOXICITY

Oxygen toxicity is not a problem during descent in routine air diving. In mixed-gas diving, oxygen toxicity is also quite unlikely because the descent time is short and because the P_{O_2} remains low for most of the compression. However, oxygen toxicity can occur during descent on a mixed-gas dive if the diver is inadvertently breathing a gas mixture with a high oxygen fraction.

CONTAMINATED GAS SUPPLY

Gas supplies contaminated with aliphatic, aromatic, and halogenated hydrocarbons or with carbon monoxide may lead to problems on descent if the concentrations of these contaminants are high enough. The gas supply can easily become contaminated if cleaning solvents are not completely removed from cylinders, valves, and piping systems before they are placed back in service after cleaning. Faulty operation of compressors can also lead to contamination of the gas supply. Gas supplies should be routinely checked for the presence of contaminants. The U.S. Navy has established a 25 ppm limit for total gaseous hydrocarbons (in methane equivalents) and a 20 ppm limit for carbon monoxide.[1] Other organizations have adopted even more stringent limits. Mixed-gas diving mixtures are prepared from pure gases; carbon monoxide poisoning is therefore very rare on mixed-gas dives. The one exception is when air is used to form the mixture. Any gas with a pronounced or abnormal odor should not be used until a definitive analysis can be made.

Medical Problems on the Bottom

Once a stable depth has been reached, barotrauma ceases to be a problem. The one exception is delayed onset of vertigo from a rupture of the labyrinthine window sustained during descent. The following possible problems should be considered.

HYPERCAPNIA

Hypercapnia is most likely during the bottom phase of the dive. Hypercapnia may occur in two ways. First, carbon dioxide may be present in the inspired gas because of contamination of the supply gas, inadequate gas flow in helmets, or weak or exhausted CO_2-absorbent beds. For each 1 kPa rise in inspired carbon dioxide partial pressure, arterial CO_2 partial pressure rises 1 kPa if pulmonary ventilation does not increase to compensate. Such compensation may be difficult in a hard-working diver breathing a dense gas. Second, although the inspired gas may be free of carbon dioxide, pulmonary ventilation may be too low relative to carbon dioxide production. This condition is called *alveolar hypoventilation* and is common in divers. Hypoventilation at depth results from increased resistance to breathing caused by dense gas flowing through regulators, valves, hoses, and the diver's airway; from dead space in the breathing apparatus; and from an elevated inspired oxygen partial pressure that suppresses respiratory drive. Both alveolar hypoventilation and carbon dioxide accumulation in the inspired gas may be present simultaneously. Hypercapnia is most likely on the bottom because the breathing resistance is greater, the inspired oxygen partial pressure is generally higher, and the carbon dioxide production is elevated because the diver is working.

Hypercapnia is most likely with closed- and semiclosed-circuit systems but may occur with open-circuit systems as well, particularly when gas density is high. In closed- and semiclosed-circuit diving systems, carbon dioxide can accumulate in the breathing loop because of wet, depleted, or defective carbon dioxide absorbent or because of improper packing of absorbent canisters. Accumulation is greatest during exercise, when the load on the canister is greatest. Breathing bags may foster alveolar hypoventilation by imposing elastic and hydrostatic loads on the respiratory system that are minimal or absent in open-circuit systems. With closed- and semiclosed-circuit systems, the diagnosis of hypercapnia is usually a retrospective one made by coupling the characteristic symptoms of carbon dioxide excess with a visual inspection of the faulty absorber.

In open-circuit free-flow helmets, the diver must set the flow of fresh air through the helmet at a rate high enough to wash out exhaled carbon dioxide; otherwise, carbon dioxide will accumulate in the helmet. Diver workload is the most important determinant of helmet ventilation, but depth also has some effect. At the same workload, greater helmet ventilation is required at depth to compensate for the small quantity of carbon dioxide always present in the compressed air supply. Most divers using free-flow air helmets experience some elevation in the inspired carbon dioxide level. This elevation may be the primary cause of hypercapnia during exercise or may be a contributing factor. One can usually diagnose hypercapnia in a diver with a free-flow helmet when symptoms of carbon dioxide excess disappear when the diver stops work and ventilates the helmet with a large volume of fresh air.

Excess carbon dioxide in the inspired gas is rarely a problem with open-circuit demand scuba or demand helmets. Air supplies are monitored for carbon dioxide level, and gas mixtures are usually blended using certified pure components. Hypercapnia may still occur with these systems, however, if the diver hypoventilates to compensate for excessive breathing resistance or if the diver voluntarily skip-breathes to conserve gas supplies.

HYPOXIA

The P_{O_2} in air increases linearly with the diver's depth. As a result, hypoxia will not occur on the bottom on air dives unless the demand regulator fails completely and the diver becomes asphyxiated. Even with a free-flow helmet, the air flow would have to be reduced to extremely low levels for hypoxia to occur. Barring complete failure of the helmet air supply, severe carbon dioxide intoxication would occur before hypoxia. Hypoxia is also very unlikely in open-circuit mixed-gas demand systems. A diver at 132 fsw (5 ata) would have to be switched to a gas mixture containing less than 3% oxygen (15% oxygen, surface equivalent) before hypoxia would occur.

Hypoxia is much more likely in closed- and semiclosed-circuit rigs. In closed-circuit diving gear controlled by oxygen sensors, hypoxia may occur at the bottom if the oxygen sensing system fails and new oxygen is not added to the rig. The onset of symp-

toms will be gradual as the diver slowly consumes all the available oxygen within the breathing circuit. In semiclosed-circuit systems, complete failure of the injector system will also lead to hypoxia, with a gradual onset similar to that in closed-circuit systems. Partial failure of the injector system, however, may or may not lead to hypoxia depending on the degree to which the supply gas flow is reduced, the oxygen content of the supply gas, and the diver's depth and oxygen consumption. When the diver is deep and at rest, the Po_2 may not fall low enough to produce hypoxia. However, the Po_2 in this situation will be considerably lower than it should be for the depth. Hypoxia can be expected during ascent.

Hypoxia may occur on the bottom with certain 100% oxygen rebreathers if proper purging procedures have not been followed. In this instance, the breathing bag contains a significant amount of residual nitrogen derived both from air that was incompletely washed out of the bag and lungs at the beginning of the dive and from nitrogen washout of body tissues during the dive. Oxygen in the bag can be completely consumed before the bag volume becomes small enough to interfere with tidal respiration or to prompt new oxygen addition. The onset of hypoxia is gradual as the diver slowly consumes all of the available oxygen in the breathing circuit.

In breath-hold diving, excessive hyperventilation may lead to body stores of carbon dioxide low enough to produce hypoxia during the breath hold before there is an adequate stimulus for terminating the breath hold and breathing.[18] This accident occurs most frequently during competitive breath-holding in swimming pools.

OXYGEN TOXICITY

CNS oxygen toxicity is not likely on the bottom during routine air diving; the depth and bottom time restrictions imposed by nitrogen narcosis and decompression do not allow enough oxygen to be breathed. The probability of CNS oxygen toxicity in mixed-gas diving is higher and depends on the Po_2 and the time spent at depth. CNS oxygen toxicity is more likely when Po_2-exposure time limit curves are approached or exceeded. The sudden onset of involuntary muscle twitching, visual disturbances, nausea, vertigo, or a feeling of impending doom should prompt this diagnosis. Hiccups may also be indicative of oxygen toxicity.

CONTAMINATED GAS SUPPLY

Gas supplies contaminated with carbon monoxide or hydrocarbons may lead to serious problems at depth. In the case of carbon monoxide contamination of a gas mixture with a fixed fraction of oxygen (such as air), the partial pressures of both carbon monoxide and oxygen rise linearly with depth, leading to complex effects. The rise in oxygen partial pressure offsets the rise in carbon monoxide partial pressure and results in an equilibrium carboxyhemoglobin level at depth that is not much different from that on the surface. The rate at which equilibrium is approached, however, is accelerated at depth in direct proportion to the elevated carbon monoxide partial pressure.[19] This is because more molecules of carbon monoxide are available for uptake from each breath. Thus, the diver would be expected to get sicker faster on the bottom than on the surface but to have comparable illness at equilibrium were it not for still a third effect: the ability of the elevated oxygen partial pressure on the bottom to maintain oxygen transport in the presence of inactivated hemoglobin. This third factor may completely mask symptoms of ongoing poisoning. With heavy carbon monoxide contamination of the air supply, symptoms of carbon monoxide intoxication will almost certainly appear on the bottom. With lesser levels of contamination, however, intoxication may be delayed until ascent, when the protective effect of oxygen is lost.

In contrast with carbon monoxide poisoning, symptoms arising from other gas contaminants are most likely to appear on the bottom. The partial pressures of these contaminants are at their highest, time is available for absorption, and the toxicity of the contaminants is not offset by the elevated oxygen pressure.

Special care must be taken not to introduce contaminants into the sealed environment of a saturation diving system. This can be a problem with underwater welding habitats. It can also happen when prohibited items are locked into the deck decompression chamber or when a diver working in chemically polluted water reenters the PTC at the end of a dive.

NITROGEN NARCOSIS

In both air and nitrogen-oxygen diving, nitrogen narcosis begins to produce significant cerebral symptoms when the nitrogen partial pressure exceeds 4.0 atm. Progressive intoxication and impairment of performance occurs as nitrogen partial pressures increase beyond that point. At a nitrogen partial pressure of 8.0 atm, the diver becomes severely affected. Narcosis is not a problem on helium-oxygen dives because helium has no narcotic properties. However, if a diver breathing a helium-oxygen mixture is suddenly switched to air because of a failure of the helium-oxygen supply, the degree of narcosis will be greater than if a dive to the same depth was made on air. This is a transient phenomenon, but it can be momentarily incapacitating.

COMPRESSION ARTHRALGIA

Compression arthralgia may appear on deep dives. The syndrome consists of pain, popping, or cracking in one or more joints on movement. The shoulders, knees, wrists, and hips are the joints most commonly affected. Occasionally, incapacitating low back pain or xiphisternal pain may occur. Symptoms of compression arthralgia generally begin at 200 fsw (60 msw), increase in severity as the depth increases, and are greatly aggravated when the affected joint is moved under load, as during exercise. Rapid compression is also an aggravating factor, but even extremely slow compression does not eliminate the problem entirely.[20] On saturation dives, compression arthralgias are observed to abate gradually over a period of days at depth, although this is not always the case. On nonsaturation dives, symptoms abate during decompression in reverse order of their appearance during compression. Upon surfacing, the diver will be free of symptoms unless the joint was further injured by exercise on the bottom. In the latter case, it may be difficult to distinguish residual compression arthralgia injury from DCS. Compression arthralgia is most common on helium-oxygen dives but also may be encountered on air dives deeper than 200 fsw.

HIGH-PRESSURE NERVOUS SYNDROME

On dives deeper than 600 fsw, high-pressure nervous syndrome may occur during descent and on the bottom. Divers complain of tremors, dizziness, imbalance, nausea, dysmetria, and intermittent somnolence. The severity of high-pressure nervous syndrome is proportional to the speed of compression and the ultimate depth achieved (see Chapter 11).

HYPOTHERMIA/HYPERTHERMIA

During long exposures in cold water, hypothermia may become a problem, particularly if thermal protection is inadequate. Confusion, lethargy, and inability to perform even simple self-help tasks are the worrisome signs and symptoms. Severe shivering may or may not be present, depending on the thermal garment selected. After a very long exposure to cold, nonfreezing cold injury to the hands and feet is possible. Hypothermia can develop in hot-water suits without the diver's experiencing any sensation of cold. In the increasingly frequent instances of diving in very warm water, heat exhaustion or heat stroke may become a problem.

OCCUPATIONAL INJURY

On the bottom, trauma may occur from contact with hazardous marine life, an accident with tools, contact with debris from wreckage, or an underwater blast. Aural injury may result from excessive noise levels, arising either internally within the helmet from gas flow or externally from sources such as tools and sonars. Electric shock may occur. In semiclosed-circuit and closed-circuit units, divers may suffer a caustic slurry of carbon dioxide absorbent on the face, in the mouth, in the nose, or in the hypopharynx. There may be exposure to radiation as well as to various chemical and microbiologic pollutants in the water. The effects of these latter agents are generally delayed and occur after surfacing.

INERT-GAS COUNTERDIFFUSION

On the bottom, switching from one gas mixture to another may cause problems. A diver who has breathed a nitrogen-oxygen mixture at depth for a significant period and who is then suddenly shifted to a helium-oxygen mixture is at risk for symptoms of

DCS even though ambient pressure remained the same. This condition has been called *deep-tissue isobaric counterdiffusion* and is associated with skin rash, joint pains, and circulating venous gas bubbles. A second type of dangerous gas switch at depth involves a dry deck decompression chamber filled with helium-oxygen deeper than 200 fsw when the diver begins to breathe a nitrogen-oxygen mixture. Differential diffusion of nitrogen and helium across the skin leads to bubble formation within the skin and eventually to venous gas embolism. This condition is called *superficial isobaric counterdiffusion.*

DROWNING

Drowning is always possible, particularly in diving systems that do not employ a helmet. The precipitating event is generally one that impairs consciousness, such as hypoxia, hypercapnia, hypothermia, oxygen toxicity, contaminated gas supply, or electric shock.

Medical Problems During Ascent

The following medical problems should be considered during ascent.

BAROTRAUMA

Ascent may be slowed by a ball-valve–like obstruction in the outlet of one of the paranasal sinuses, a relatively infrequent occurrence. Gas trapped in the sinus is prevented from escaping by the ball-valve action of a redundant mucosa, polyp, or cyst. Pain is the predominant symptom. The diver can usually be returned to surface pressure by alternately descending and ascending. When the maxillary sinus is involved, an ischemic neuropraxia of the infraorbital nerve may occur, with attendant neurologic findings.[21,22] When one or both ears fail to vent properly during ascent, ABV may result. The onset of ABV is sudden and is often preceded by a feeling of fullness in one or both ears. A few feet of descent can provide relief, and this maneuver is diagnostic. In a number of persons, the facial nerve is exposed to middle ear pressure as it traverses the temporal bone. If the middle ear fails to vent during ascent, an ischemic neuropraxia of the facial nerve may occur, resulting in unilateral facial palsy of the peripheral type (including paralysis of the forehead musculature). Generally, 10 to 30 min of overpressure is necessary for symptoms to occur; ABV often coexists. Full facial function returns 5 to 10 min after relief of the overpressure. Molvaer and Eidsvik[23] provide an excellent review of this condition.

Vertigo secondary to a perilymph fistula incurred during descent may not actually appear until the diver ascends. Such a delayed presentation is fairly common.

Facial emphysema has been reported during ascent in persons with healing facial fractures or with a recent history of dental extractions.[24] Pain in a tooth, or *aerodontalgia,* may be experienced during ascent.

HYPERCAPNIA

Hypercapnia is considerably less likely during ascent than during the bottom phase, where the diver is essentially at rest. The one exception occurs when a CO_2-absorbent bed is completely exhausted. An exhausted bed is most likely to cause problems in the late phases of decompression.

HYPOXIA

Hypoxia may occur during ascent if the mixed gas or oxygen injectors of semiclosed-circuit or closed-circuit equipment have partially failed, if a diver fails to purge a 100% oxygen rebreather thoroughly before ascending, or if the oxygen content of the supply gas mixture in open-circuit systems is too low for the intended depth.

CONTAMINATED GAS SUPPLY

Ascent is the most likely phase for carbon monoxide poisoning during air diving because the oxygen partial pressure is decreasing and sufficient time has elapsed for carbon monoxide uptake. New contaminants may cause problems during ascent if the diver is shifted to a new gas mixture for decompression. The diagnosis can usually be confirmed by shifting the diver to an alternative source of that particular gas mixture.

CEREBRAL ARTERIAL GAS EMBOLISM

Cerebral arterial gas embolism (CAGE) is most likely in an inexperienced diver making an emergency ascent. However, some cases occur in apparently healthy, experienced divers ascending at normal rates. Symptoms are expected in the final stages of the ascent, when the expansion of trapped intrapulmonary gas is greatest. Gas embolism should be high on the list of diagnostic possibilities if neurologic symptoms, especially loss of consciousness, occur while the diver is ascending to a shallow decompression stop or to the surface. Other stigmata of pulmonary barotrauma, such as pneumomediastinum or pneumothorax, are not usually apparent until the diver surfaces (see Chapter 9).

DECOMPRESSION SICKNESS

DCS under pressure, either in the water or in a chamber, is possible at any point during the ascent but is most likely at the terminal decompression stops. For most ordinary dives, DCS under pressure is not expected unless the decompression is very long or inadequate. DCS under pressure, however, is quite common with saturation dives and nonsaturation dives deeper than 300 fsw. It may be difficult to differentiate DCS from other conditions while the diver is in the water. The diagnosis becomes easier when classic symptoms such as joint pain occur and when the decompression requirements have been substantial. Vertigo or tinnitus and hearing loss that occur within a few minutes after a decompressing diver breathing helium-oxygen is switched to air at depths greater than 100 fsw should be regarded as manifestations of inner-ear DCS. One case of inner-ear DCS has been reported with an air change as shallow as 60 fsw.[25]

OXYGEN TOXICITY

The ascent should be divided into two phases when evaluating the risk of oxygen toxicity: *phase 1,* the initial ascent and any decompression stops during which the diver breathes the bottom mixture, and *phase 2,* decompression stops during which the diver breathes 100% oxygen or oxygen-enriched gas mixtures to accelerate decompression. Oxygen toxicity is unlikely during phase 1 because the P_{O_2} is steadily decreasing. The probability of oxygen poisoning is also lower during this phase because the diver's exercise level and carbon dioxide production are less than on the bottom. The one exception to the rule is the diver who is on the verge of a convulsion on the bottom: A convulsion may be triggered by the rapidly falling P_{O_2} during the early ascent. This effect has been called the *off phenomenon*.

Once oxygen or oxygen-rich mixtures are breathed in the water to accelerate decompression (phase 2), the chance of oxygen toxicity greatly increases. Indeed, most decompression procedures have an entire set of rules to deal with this contingency. The problem for on-site medical personnel is to distinguish oxygen toxicity from other conditions that may occur at the oxygen stops but that require different interventions. The differential diagnosis includes ABV, CAGE, neurologic DCS, contaminated gas supply, hypercapnia from an exhausted carbon dioxide absorber, and hypoxia from having shifted to the wrong gas mixture. The most important clue to diagnosis is the time of onset of symptoms. Oxygen toxicity requires time to develop, and it becomes the most likely cause for symptoms as the time on oxygen lengthens. By contrast, ABV presents immediately upon ascent or within 1 min of the diver's arrival at a stop. Hypoxia should present within 1 min of completion of the gas shift. CAGE should also present very shortly after arrival at a stop and should be excluded as a diagnosis after 10 min at the stop. DCS may present at any time during a stop but is most likely during or immediately after ascent to the stop. Hypercapnia from an exhausted carbon dioxide absorber or intoxication from a contaminated source of hyperoxic gas may lead to a symptom complex similar in presentation and time course to oxygen toxicity. These conditions can be distinguished from oxygen toxicity by shifting the diver to the secondary gas supply and by switching to an open-circuit mode of operation that bypasses the carbon dioxide absorber.

RAPID UNCONTROLLED ASCENT ("BLOWUP")

Blowup is a special problem that occasionally confronts medical personnel. The diver

loses buoyancy control because of overinflation of a buoyancy compensator or dry suit and makes a rapid uncontrolled ascent to the surface. If no decompression stops have been missed, such persons should be watched closely for emerging signs of pulmonary barotrauma. If decompression stops have been missed but the diver remains asymptomatic, then the diver should be immediately returned to pressure for further decompression. If the diver is symptomatic, then the diver should be recompressed and treated according to the appropriate therapeutic table. Various diving manuals provide rules for dealing with blowup.[1,2]

Medical Problems After Surfacing

Medical problems that may arise after the diver has surfaced include hypoxia, hypercapnia, oxygen toxicity, ABV, IEBT, CAGE, pneumothorax, pneumomediastinum, pneumoperitoneum, DCS, hypothermia, hyperthermia, and various forms of dermatitis. The key to separating these various conditions is the presenting symptom and the time of its onset after the diver has surfaced (Table 18–4).

HYPOXIA

Hypoxia, with loss of consciousness, may occur immediately upon surfacing or during any period following surfacing in which the diver continues to breathe from the UBA. The risk is greatest whenever divers must perform heavy exercise (e.g., swimming against a strong current) in semiclosed- or closed-circuit gear. Hypoxia may also occur in open-circuit systems if gas switches are performed when the diver surfaces or if the oxygen fraction in the original supply gas is too low to allow surfacing. Divers recover rapidly from hypoxia as soon as they breathe ambient air.

HYPERCAPNIA

With some older styles of free-flow air helmets and associated dress, it is not possible to climb a ladder out of the water without first securing the air supply. If the air supply is not secured, the arms will balloon out as soon as the spring-loaded exhaust valve clears the water. In these systems, carbon dioxide excess may occur while the diver climbs the ladder and walks to the dressing stool. Cerebral and respiratory symptoms of hypercapnia clear rapidly once the diver breathes ambient air.

OXYGEN TOXICITY

An oxygen-induced seizure may occur within the first 1 to 2 min following surfacing from an oxygen-breathing decompression stop (off phenomenon). A seizure developing more than 2 min after surfacing should not be ascribed to this cause.

Table 18–4. Differential diagnosis of conditions presenting after surfacing by time of onset

Time of Onset After Surfacing		
<2 min	***2–10 min***	***>10 min***
DCS	DCS	DCS
Hypothermia	Hypothermia	Hypothermia
IEBT	IEBT	IEBT
CAGE	CAGE	
Pneumothorax	Pneumothorax	
Pneumomediastinum	Pneumomediastinum	
Alternobaric vertigo		
Hypercapnia		
Hypoxia		
Oxygen toxicity		
Carbon monoxide poisoning		

CAGE, cerebral arterial gas embolism; DCS, decompression sickness; IEBT, inner-ear barotrauma.

ALTERNOBARIC VERTIGO

ABV occurs at the moment of surfacing or very shortly thereafter. In general, vertigo that develops more than 2 min after surfacing should not be considered ABV. However, occasional cases may persist after surfacing (see later discussion of ABV under Differential Diagnosis of Vertigo).

PULMONARY BAROTRAUMA

Conditions associated with pulmonary barotrauma (pneumomediastinum, pneumothorax, pneumoperitoneum,* and CAGE) should become apparent on or shortly after surfacing. The rapid onset of neurologic signs followed by unconsciousness upon surfacing is highly suggestive of CAGE. Generally speaking, pulmonary barotrauma should not form part of the differential diagnosis of symptoms that begin more than 10 min after the diver has surfaced.

DECOMPRESSION SICKNESS

DCS may occur upon surfacing, but its onset is usually delayed. When neurologic symptoms or signs arise more than 10 min after surfacing, DCS is the most likely diagnosis.

INNER-EAR BAROTRAUMA

When auditory-vestibular symptoms are the sole neurologic symptoms arising 10 min after surfacing, IEBT must be considered in addition to DCS. Most forms of IEBT should manifest on surfacing, although minor impairments may go unnoticed until more than 10 min after the diver has surfaced. In addition, in IEBT with perilymph fistula, the diver may first become symptomatic after heavy lifting or straining. Thus, symptoms can occur well after surfacing. The differential diagnosis of DCS versus IEBT is discussed later under Differential Diagnosis of Vertigo.

HYPOTHERMIA

Hypothermia with mental clouding, nonresponsiveness, and semiautomatic behavior may occur after surfacing. Symptoms may worsen when the afterdrop in core body temperature occurs. For divers requiring decompression, the condition must be differentiated from DCS, which may also be associated with similar mental changes. Measurement of core body temperature is diagnostic.

DERMATITIS

Various forms of dermatitis, all unusual, may occur after the dive. They include such conditions as sea bather's eruption, sea louse dermatitis, soapfish dermatitis, sponge dermatitis, seaweed dermatitis, and dermatitis from chemical contaminants in the water. Recently, cases of *Pseudomonas* folliculitis have been related to the use of wet suits.[26,27] Fisher[28] and Sims[29] provide excellent discussions of dermatitis.

Other Medical Problems

OTITIS EXTERNA

Otitis externa is a very common and important condition that can adversely affect diving operations. It is particularly troublesome for saturation divers, who are continuously exposed to a hot, humid environment for many weeks. Up to 50% of worker-hours available for diving may be lost because of this condition. Severe cases are associated with pain, fever, lymphadenopathy, and inability to bite or chew. In the saturation environment, systemic antibiotics and local measures may barely be able to control the infection once it has begun. Prevention is paramount. One highly successful regimen is twice-daily irrigation of the external ear canal with a 1% acetic acid/aluminum acetate solution.[30]

SEASICKNESS

Seasickness is another very common and debilitating problem on diving operations. Vomiting into masks and regulators can be

*Pneumoperitoneum is a very rare and generally asymptomatic condition usually associated with pulmonary barotrauma. Because it may arise from a ruptured viscus, however, the first manifestation may be abdominal pain and signs of peritonitis that appear some time after the dive is completed.

extremely dangerous because of the possibility of pulmonary aspiration or laryngospasm. Seasickness in small boats can be minimized by the diver's refraining from going below deck, finding a spot on the deck that has the least motion, fixing on the horizon, and entering the water quickly once the boat is at anchor. Inhalation of diesel fumes should be avoided. To be effective, antimotion drugs must generally be taken before exposure to the stimulus. A recurring concern with drug therapy is its potential to reduce diver alertness, impair performance, and produce side effects that may be confused with DCS or CAGE. Currently recommended medications include dimenhydrinate, meclizine, cyclizine, transdermal scopolamine, and oral scopolamine combined with dextroamphetamine.

Two studies examined the psychomotor and side effects of a preparation of transdermal scopolamine (Transderm Scop) during diving in a dry hyperbaric chamber.[31,32] No alterations in psychomotor performance were noted, but side effects included fatigue, difficulty concentrating on tasks, and blurred vision. Further evaluation of these drugs in open water is required. Some physicians advocate the use of a "sea band" to avoid seasickness before diving. This is a wrist strap that puts mechanical pressure on acupuncture sites on the wrist or stimulates these sites electrically. The efficacy of these devices in combating seasickness remains to be demonstrated.

SUNBURN

Sunburn is also a very common and painful sequela of open-water diving operations. It is exacerbated by windy conditions. Sunburn can be prevented by the proper use of clothing and the active use of sunscreens containing para-aminobenzoic acid derivatives or other absorbents of ultraviolet radiation in the 290 to 320 nm range. The United States Weather Service publishes a daily ultraviolet index for various locales in the United States. This index describes the expected intensity of ultraviolet exposure when the sun is at its highest position in the sky; the index is based on a variety of measurements, including atmospheric pressure, temperature, ozone level, and expected degree of cloudiness. Index values of 0 to 2 indicate minimal exposure levels; values of 10 to 15 indicate very high exposure levels. Protective strategies should be based on the index (or an estimate of it) and an individual's susceptibility to burning.

At low exposure levels, simple application of a sunscreen with a low skin protection factor (SPF) may be sufficient. At high exposure levels, a sunscreen with an SPF of 30 to 45 and wearing hats, sunglasses, and special sun protective clothing are indicated. The risk of burning is highest at midday. On high-index days, midday exposure should be limited. For diving, a waterproof sunscreen is best. To be effective, sunscreen should be applied 30 min prior to exposure. Water does not absorb ultraviolet irradiation completely. At a depth of 3 fsw, the intensity of ultraviolet radiation is about 70% of its intensity on the surface. Swimmers and snorkelers therefore may be at significant risk for sunburn even though they are in the water.

Summary

Table 18–5 summarizes the medical problems that may be expected during the various phases of an air scuba dive to 130 fsw. This is the most common form of diving encountered by the average physician or diving medical technician. Air dives to greater depths increase the risk of narcosis, DCS, and hypercapnia. Hypercapnia is more likely in open-circuit free flow helmets than with demand equipment. If open-circuit mixed gas equipment or rebreathers are chosen, hypercapnia (from alveolar hypoventilation or absorbent failure, or both), hypoxia (from gas injector failure or wrong gas selection), or oxygen toxicity (from injector failure, wrong gas selection, or violation of oxygen limits) may be added to the list. On very deep dives, additional problems related to compression arthralgia, high-pressure nervous syndrome, and thermal stress may be encountered.

DIFFERENTIAL DIAGNOSIS

The previous discussion reviewed medical disorders that can be anticipated during various phases of the dive. One can considerably simplify diagnosis by knowing the point in the dive at which the problem occurred. This discussion elaborates on the

Table 18–5. Potential medical problems associated with each phase of a 130-fsw scuba dive on air

	Descent	On Bottom	Ascent	After Surfacing
Aural and sinus barotrauma	Most likely injury	Impossible except for delayed perilymph fistula	Most likely injury	Impossible, except for delayed perilymph fistula
Hypercapnia	Unlikely	Unlikely unless: • CO_2 is present in air • Regulator resistance is extensive • Skip breathing is used	Unlikely	Not possible
Carbon monoxide poisoning	Unlikely • Inadequate time for uptake • Increased P_{O_2} protects	Unlikely (increased P_{O_2} protects)	Most likely time • Adequate time for uptake • Loss of P_{O_2} protection	Unlikely
Alternobaric vertigo	Possible	Possible immediately after arrival on bottom	Most likely time	Within first 2 min only
Nitrogen narcosis	Slight; aggravated if descent rapid	Slight	None	None
DCS	Not possible	Not possible	Rare	Most likely time
Occupational injury	Possible	Most likely time	Possible	Possible
CAGE	Not possible	Not possible	Possible in late stages	Possible; onset of symptoms within 10 min of surfacing
Pneumothorax, pneumomediastinum	Not possible	Rare; usually associated with ditch-and-don exercises	Possible in late stages	Possible; onset of symptoms within 10 min of surfacing

CAGE, cerebral arterial gas embolism; DCS, decompression sickness; P_{O_2}, partial pressure of oxygen.

differential diagnosis of several common presenting complaints. Headache is not included because it has been the subject of a recent review.[33]

Musculoskeletal Pain

The differential diagnosis of musculoskeletal pain following a dive generally centers around two possible causes, traumatic injury and DCS.* The pain of DCS is generally described as a deep, dull ache or a deep, boring sensation, although it may be throbbing. DCS most commonly involves the shoulders or elbows on short-duration dives and the knees on long-duration dives. However, any synovial joint may be affected. Lam and Yau[34] reported the involvement of more than one anatomic site in 54% of 793 cases of musculoskeletal pain in tunnel workers. The sensation of discomfort may extend well beyond the joint into the surrounding muscle; indeed, the joint may not be the primary focus of the complaint. The pain tends to be relatively insensitive to movement and usually lacks a trigger point. The area is not usually tender to palpation.

Factors that favor a diagnosis of DCS include:

- Clear onset of pain after a dive at or near the no-decompression limit or after a dive having a decompression requirement

*The severe generalized musculoskeletal pain produced by envenomenation by the sea-wasp jellyfish *(Chironex fleckeri)* in the Indo-Pacific region may be confused with severe DCS. See Chapter 15.

- Lack of known injury to that region either before or during the dive
- Involvement of more than one site
- Other signs of DCS
- Gradual increase in severity of pain

Even in the presence of previous trauma, however, DCS often cannot be excluded with certainty because DCS tends to develop in previously injured areas. Pain that is relieved by the application of a blood pressure cuff to the affected joint may suggest DCS.

It is often impossible to distinguish between traumatic injury and DCS. In such cases, a diagnostic test of pressure is indicated. The victim undergoes recompression to 60 fsw (18 msw), breathing oxygen for a period of 20 to 30 min. If no relief is obtained, the condition is regarded as a traumatic injury and the patient is returned to the surface and treated accordingly. If relief is significant, the condition is regarded as DCS and the hyperbaric treatment is continued. Some practitioners suggest completing a treatment table once started even if pain does not respond to recompression.

Focal Neurologic Dysfunction

With the exception of vertigo and hearing loss, the onset of focal neurologic signs after decompression almost always indicates either DCS or CAGE. Before either of these diagnoses is entertained, however, several forms of traumatic nerve injury should be ruled out. These conditions include an isolated facial nerve injury secondary to middle-ear overpressure, an isolated infraorbital nerve injury secondary to maxillary sinus overpressure, brachial plexus injury secondary to shoulder harnesses or shoulder trauma, and lateral femoral cutaneous nerve injury (meralgia paresthetica) secondary to heavy weight belts. All of these conditions are relatively uncommon.

DECOMPRESSION SICKNESS VERSUS CEREBRAL ARTERIAL GAS EMBOLISM

In the past, it was generally considered important to distinguish CAGE from DCS. This was because conventional treatment of CAGE called for recompression to 165 fsw (50 msw), whereas treatment of DCS called for recompression to only 60 fsw (18 msw). Experience has shown that it is often difficult, if not impossible, to distinguish these two conditions.[35] This has led to the adoption by some physicians of the term *decompression illness,* which includes both DCS and CAGE, and to the selection of recompression protocols on the basis of patient symptoms rather than presumptive diagnosis.[1] Nevertheless, it is still important to distinguish the two conditions for the purposes of accident analysis, therapeutic trials, development of new decompression tables, and prognosis.

Time of Onset

The manifestations of CAGE usually begin during ascent or immediately after surfacing. In a review of 188 cases derived from submarine escape training and diving activities, Pearson[36] noted that when coma was the dominant manifestation, symptoms developed within 30 sec to 1 min of surfacing. In patients showing a variety of lesser focal signs, manifestations developed within 5 min, with the singular exception of one patient in whom CAGE manifested after 8 min. A time lapse of more than 10 min between surfacing and the onset of symptoms is generally not consistent with the diagnosis of CAGE, although there are exceptions.

DCS may also begin during ascent or immediately after surfacing. Such a rapid onset is not unusual, particularly in fulminant forms of the disease involving major disruptions of spinal cord or cerebral function. In an analysis of 1070 major cases of type 2 DCS, Francis and colleagues[37] noted that in 50% of the patients, manifestations occurred within the first 8 min of surfacing. In an analysis of 100 cases of DCS, Erde and Edmonds[38] noted 22 cases of cerebral involvement that presented within the first 3 min of surfacing. An additional four cases presented between 3 and 10 min. Three patients experienced spinal-cord involvement in the first 3 min on the surface; signs developed in an additional five patients between 3 and 10 min after surfacing. Minor focal neurologic signs, on the other hand, tend to be delayed and have a slower time course for evolution, similar to the time course for type 1 DCS. Focal neurologic signs presenting more than 10 min after surfacing are likely to be DCS; those presenting within 10 min could represent either CAGE or DCS.

Clinical Presentation

CAGE usually presents with an onset of neurologic manifestations that suggest brain involvement, such as aphasia, dysarthria, vertigo, visual disturbances, unilateral sensory and motor changes, convulsions, and loss of consciousness. Bilateral sensory or motor changes occur in only 10% of cases.[36] Relatively rapid spontaneous recovery may occur. A lucid interval occurs occasionally. When present, coexisting signs of pulmonary barotrauma, such as pneumomediastinum or pneumothorax, support the diagnosis.[39] Hemoptysis, if present, also supports the diagnosis.

Neurologic DCS usually presents with focal manifestations that suggest involvement of the spinal cord or a peripheral nerve, such as sensory losses in nerves or nerve-root distribution, paraparesis or paraplegia, loss of bladder and bowel function, and the Brown-Séquard syndrome. Loss of sensory and motor function is often patchy, and in contrast with CAGE, bilateral involvement is common. When spinal-cord tracts are disrupted, a distinct motor and sensory level related to the spinal segments becomes apparent. Unfortunately, a wide variety of cerebral symptoms may also be the primary manifestation of DCS. These symptoms frequently have a rapid onset and are difficult to distinguish from CAGE. Recent surveys have suggested that up to 30% of cases of type 2 neurologic DCS may involve the brain.[37,38] Symptoms of cerebral DCS include confusion, personality changes, amnesia, aphasias, scotomata, visual field defects, headache, dizziness, motor or sensory disturbances involving only one limb, and a variety of cerebellar signs including ataxia. Focal or generalized seizures and loss of consciousness may occur. The acute onset of stocking or glove anesthesia in DCS may indicate cortical involvement, although a spinal lesion is also possible.

Other Diagnostic Considerations

Neurologic signs of DCS regress without treatment, but the rate of regression is significantly slower than for CAGE. A weak differential diagnostic point is that headache is a more prominent feature of DCS than of CAGE. Coexisting signs and symptoms, such as musculoskeletal pain and skin rashes, can help support the diagnosis of DCS. The absence of these correlates does not argue against DCS, however. In a recent survey of U.S. Navy divers, Torrey and colleagues[40] observed coexisting arthralgia in only 21% of cases of type 2 neurologic DCS. Erde and Edmonds[38] reported musculoskeletal pain in 38% of cases with cerebral involvement, in 27% of cases with spinal-cord involvement, and in 64% of cases with peripheral-nerve involvement.

The depth and time of the dive often help to distinguish CAGE from DCS. Dives well within the no-decompression limits, especially those associated with emergency (e.g., out-of-air) ascents or emergency ascent training, are most likely to result in CAGE. Dives in which ascent is normal but the diver requires considerable decompression or omits a large portion of the decompression are most likely to result in DCS. This is not an iron-clad rule, however: Some fulminant cases of spinal-cord DCS have occurred within the no-decompression limits.

It is worth noting that CAGE and DCS may coexist. When decompression has been omitted through emergency ascent, DCS can occur right after CAGE. Also, both animal experiments and human cases suggest that CAGE may predispose to spinal-cord DCS in situations in which spinal-cord involvement would not ordinarily be expected.[41]

As mentioned earlier, the overlap in manifestations can make it difficult to clearly distinguish between CAGE and DCS. Failure to arrive at a firm diagnosis is not likely to have serious consequences for the individual patient for three reasons:

- Treatment by recompression benefits patients with both diseases.
- Most current recompression protocols recommend starting at the same depth for both conditions.
- The physician always can recommend alteration of the treatment tables if the patient's clinical response is unsatisfactory.

Peripheral Oxygen Toxicity

A focal neurologic symptom that is a special diagnostic pitfall for the uninitiated is intense numbness in the fingertips in a diver breathing oxygen during decompression or during recompression treatment. This condition does not represent DCS or CAGE but rather peripheral oxygen toxicity. The numbness subsides several hours after oxygen breathing is discontinued. This condition does not herald the onset of CNS oxygen toxicity, and oxygen breathing need not be discontinued.

Chest Pain and Dyspnea

Chest pain and dyspnea are fairly uncommon in diving, but they can pose diagnostic dilemmas. The nature and location of symptoms, the time of onset during the dive, and the physical examination are key to the differential diagnosis. The following conditions should be considered.

PULMONARY DECOMPRESSION SICKNESS (CHOKES)

Chokes may begin at any point after the diver leaves the bottom, but this condition is most likely after surfacing. The onset of symptoms is sometimes delayed for several hours after surfacing. Symptoms consist of progressively worsening substernal burning pain or feeling of distress, paroxysmal cough, and shortness of breath. These symptoms are greatly aggravated by deep inspiration and by smoking. Tachypnea is invariably present. An electrocardiogram (ECG) may show a peaked P wave, right-axis deviation, and evidence of right-ventricular strain. Chokes usually require a severe decompression stress (i.e., either a large portion of the required decompression was omitted or decompression was very long and arduous). Often, other stigmata of DCS develop and aid diagnosis. Erde and Edmonds[38] reported that 52% of patients with respiratory symptoms had coexisting musculoskeletal symptoms, whereas 91% of patients had one or more findings relating to the CNS or the inner ear.

PULMONARY EDEMA

Symptomatic pulmonary edema has occurred in both divers and surface swimmers.[42–46] This is a relatively rare condition whose cause is uncertain. Divers may dive for many years before first experiencing symptoms; thereafter, they may experience recurring episodes interspersed with periods of normal diving. Symptoms may begin on the bottom, during ascent, or shortly after ascent and consist of cough, shortness of breath, and hemoptysis. Chest pain is notably absent, which helps to eliminate chokes as a diagnostic possibility. Chest examination reveals rales, and chest radiographs show the classic pattern of pulmonary edema. Arterial unsaturation may be significant. Symptoms and signs usually resolve spontaneously over 24 hours. Episodes appear to be precipitated by factors that increase cardiac preload and afterload, including immersion in water (particularly cold water), heavy exercise, negative-pressure breathing, and predive fluid overload (see Chapter 25 for further discussion).

SPINAL-CORD DECOMPRESSION SICKNESS

Back pain followed by lancinating radicular or dull girdle-like chest pain occurring shortly after surfacing often heralds the onset of paralytic spinal-cord DCS. Such a condition often occurs after dives requiring only minimal decompression. Pneumothorax should quickly be ruled out. Other stigmata of evolving spinal cord DCS, such as numbness and weakness, usually appear rapidly and aid diagnosis.

PULMONARY OXYGEN TOXICITY

The symptoms of pulmonary oxygen toxicity are quite similar to those of chokes but develop much more slowly. During most conventional dives, the dose of oxygen delivered to the lungs is inadequate to produce pulmonary oxygen toxicity, thus excluding this diagnosis. If the exposure to oxygen has been long enough to cause pulmonary oxygen toxicity, pulmonary oxygen toxicity can be distinguished from chokes by its very gradual onset and the absence of other stigmata of DCS. Sometimes, DCS may not be possible. Pulmonary oxygen toxicity is most likely when the diver is breathing oxygen in the chamber. Chokes, on the other hand, are most likely after the diver is at the surface, breathing air. The unit pulmonary toxicity dose or some other quantitative index of oxygen toxicity risk[47–49] can be used to estimate the likelihood of pulmonary oxygen toxicity in a given situation.

PNEUMOMEDIASTINUM

Symptoms associated with pneumomediastinum first appear during or after ascent, although the problem may begin at the bottom during ditch and don exercises. The principal symptom is a substernal ache or

tightness. Occasionally, the pain may be sharp and may radiate to both shoulders, the back, or the neck.[50] The discomfort is often aggravated by deep breathing, coughing, swallowing, moving the neck and trunk, or lying flat. The quality of the voice may change. Dyspnea is present in more severe cases, and subcutaneous crepitation may be felt over the clavicles. In contrast with chokes, progressively worsening cough and shortness of breath do not accompany the pain. Examination of the chest may reveal subcutaneous crepitus above and below the clavicles, decreased heart sounds, decreased area of cardiac dullness, and a crunching or crackling sound on auscultation *(Hamman's sign)*. There may be some precordial tenderness. A chest radiograph is diagnostic.

PNEUMOTHORAX

Simple pneumothorax may result from lung overinflation but is considerably less common than pneumomediastinum. Pneumothorax should be suspected when there is pleuritic pain, especially pain located over the more lateral aspects of the chest wall. Decreased or absent breath sounds and tactile fremitus, increased resonance to percussion, tracheal deviation to the affected side, and decreased chest wall motion on the affected side aid diagnosis. These signs may be difficult to elicit in a small pneumothorax. An erect chest radiograph in full expiration or a lateral decubitus radiograph is required.

Tension pneumothorax may result from lung overinflation during ascent, or it may result from recompression therapy for simple pneumothorax. The dominant manifestations of tension pneumothorax are those of circulatory collapse (arterial hypotension, cyanosis, dyspnea, and tachypnea) rather than chest pain. Tracheal deviation away from the affected side, absent breath sounds, bulging interspaces, distended neck veins, and hyperresonance of the affected side may be present.

MYOCARDIAL ISCHEMIA

Severe or prolonged substernal or precordial chest pain usually indicates myocardial ischemia. Two possibilities exist: (1) The pain is the manifestation of underlying coronary artery disease, that is, an anginal attack unrelated to the diving environment per se, or (2) the pain is the result of CAGE associated with pulmonary barotrauma. Although involvement of the coronary arteries is not common in pulmonary barotrauma, it has been documented in humans,[51] and one study suggests that coronary artery involvement may be more prevalent than previously thought.[52] Other stigmata of pulmonary barotrauma with neurologic involvement should be present. An ECG and cardiac enzymes are essential for ruling out this possibility.

ANXIETY-HYPERVENTILATION SYNDROME

Anxiety-hyperventilation syndrome is not uncommon in novice divers and may produce shortness of breath and substernal tightness. The other characteristic findings, such as lightheadedness; dizziness; numbness of the hands, feet, and perioral area; carpal pedal spasms; and a positive Chvostek sign aid diagnosis. Anxiety-hyperventilation syndrome should not be diagnosed until pneumomediastinum and chokes have been ruled out.

TRAUMA

Injury to the chest wall is always possible on any working dive. A history of injury and local tenderness or evidence of trauma establish the diagnosis. Trauma to the respiratory muscles may occur during long dives when the breathing resistance is high. After the dive, the diver complains of generalized chest-wall soreness.

Loss of Consciousness

The differential diagnosis of unconsciousness rests heavily on the phase of the dive during which the problem occurred, the hazards in the surrounding environment, the type of equipment employed, the nature of the prodromal symptoms, and the abruptness with which consciousness is lost. It is often impossible to establish the diagnosis with certainty. The following conditions should be considered.

HYPOXIA

Hypoxia is perhaps the most common cause of unconsciousness in diving and is an especially strong possibility when semiclosed- and closed-circuit breathing systems are being used. Any phase of the dive may be involved. Gradual impairment of consciousness with attendant euphoria is the usual presentation with semiclosed- and closed-circuit breathing sets and results from gradual depletion of oxygen in the breathing loop. In open-circuit demand systems, the loss of consciousness is abrupt if a gas shift is involved and is only slightly more gradual if the diver is ascending to a shallower depth than that allowed for the gas mixture.

HYPERCAPNIA

Hypercapnia is unlikely to cause abrupt loss of consciousness during any phase of the dive. Lesser symptoms of restlessness, lightheadedness, dizziness, weakness, confusion, throbbing frontal or bitemporal headache, nausea, and, occasionally, breathlessness or suffocation usually appear before levels of carbon dioxide become narcotic. The brightness, colors, or shapes of objects may appear distorted. If warning symptoms go unnoticed or unheeded, frank loss of consciousness supervenes. When the buildup of carbon dioxide is rapid, the warning period may be brief. The bottom and ascent phases of the dive are most commonly involved.

A special syndrome called *shallow water blackout* has been recognized with the use of 100% oxygen rebreathers. For reasons that are not entirely clear, accumulation of carbon dioxide in the rebreathing bag because of a faulty absorber does not trigger the usual compensatory hyperpnea in some persons. The arterial partial pressure of carbon dioxide rises rapidly in such persons, causing them to lose consciousness.[53] The typical occurrence involves a young and inexperienced but highly motivated diver undergoing initial training with the apparatus. The loss of consciousness typically appears early in the dive, often during or after a period of hard work. The depth of the exposure ranges between 10 and 25 fsw, too shallow to incriminate oxygen toxicity. Approximately 50% of affected divers do not experience (or remember) warning signs prior to the loss of consciousness.

OXYGEN CONVULSIONS OR SYNCOPE

Syncope or convulsion with abrupt loss of consciousness is often the first sign of CNS oxygen toxicity. The depth of the dive, the P_{O_2}, the length of exposure, the exercise level, and the degree of carbon dioxide retention all influence the probability of oxygen poisoning. When the P_{O_2} has been less than 1.3 atm, convulsions or syncope are very unlikely, which is useful in differential diagnosis. Prodromal symptoms such as irritability, involuntary muscle twitching, narrowing of the visual fields, nausea, auditory hallucinations, or vertigo, followed by abrupt loss of consciousness in a hyperoxic environment, strongly suggest oxygen poisoning. The sudden loss of consciousness upon moving a diver from an environment with a high P_{O_2} to one with a low P_{O_2} could also represent oxygen toxicity (off phenomenon), although the presence of a hypoxic gas mixture must be ruled out. During an oxygen seizure, a single expiratory sigh or cry is commonly heard as the paroxysm begins.

TRAUMA

Trauma to the head or other serious injuries may cause abrupt loss of consciousness on the bottom. Trauma is especially likely when the diver is working around a wreck or platform or is operating from a diving bell. The cause is usually self-evident.

CONTAMINATED GAS SUPPLY

Gas contaminated with carbon monoxide or hydrocarbons may produce unconsciousness if concentrations are high enough. Unconsciousness is generally preceded by a period of increasing cerebral dysfunction resulting from pulmonary uptake and circulatory distribution of the offending agent to the brain and other body tissues. This rate is accelerated by exercise. The likelihood of unconsciousness from carbon monoxide poisoning is maximal during ascent because enough time has passed to allow carbon monoxide uptake during the bottom phase and because the oxygen partial pressure, which helps in maintaining oxygen transport and in preventing dissolved carbon monoxide from attaching to hemoglobin and cytochromes, is rapidly

decreasing. With carbon monoxide intoxication, the diver may first experience tingling in the fingers and toes and a feeling of tightness across the forehead. This is followed by increasing confusion, euphoria, throbbing at the temples, headache, nausea, weakness, dizziness, and tinnitus. Loss of muscle control and dimming or blurring of vision may be experienced before consciousness is lost. Intermittent convulsions and Cheyne-Stokes respiration may then occur. Carbon monoxide poisoning was misinterpreted as CAGE in one case.[54] With other contaminants, the problem is most likely to occur on the bottom, with the specific symptom pattern depending on the specific contaminant. Sometimes it is possible to shift gas sources. Disappearance of symptoms after the shift strongly suggests the presence of contaminated gas. This can be confirmed by analysis of the questionable gas.

CEREBRAL ARTERIAL GAS EMBOLISM

CAGE is an unlikely cause of loss of consciousness until the final stages of ascent and surfacing are reached. Then, CAGE becomes one of the major diagnostic possibilities. Loss of consciousness is abrupt and is often not preceded by a prodrome other than perhaps some vague feeling of chest discomfort or pain. Consciousness is lost during the actual ascent or within the first few minutes of reaching the decompression stop or the surface. Unconsciousness late in a decompression stop or more than 10 min after surfacing is unlikely to result from gas embolism. Hemoptysis may help suggest this diagnosis.

ELECTRIC SHOCK

Electric shock may be severe enough to cause unconsciousness. The onset is abrupt, with the victim crying out one or more times before losing consciousness. The setting is usually a military or commercial dive, rather than a recreational dive, and the problem generally occurs during the bottom phase.

POSTURAL HYPOTENSION

Divers who have intravascular volume depletion from extended dives in cold water or who are peripherally vasodilated from hot-water suits may experience dizziness and lightheadedness when climbing back into and standing inside a deep-diving bell or PTC. The sudden loss of the hydrostatic support provided by the water leads to postural hypotension. Momentary unconsciousness may occur.

ANXIETY-HYPERVENTILATION SYNDROME

Excessive hyperventilation, particularly in inexperienced, anxious divers, can produce lightheadedness and dizziness. By itself, hyperventilation rarely leads to unconsciousness. Numbness of the hands, feet, and perioral area; obvious hyperpnea; carpal pedal spasms; and a positive Chvostek sign aid the diagnosis. Whether a vigorous Valsalva maneuver performed by a hyperventilating diver (e.g., to equalize middle-ear pressure) can convert a simple disturbance of consciousness to a loss of consciousness in the water is controversial. The phenomenon is well known on land, but in the water circulatory factors protect venous return to the heart. Still, this possibility requires further investigation.

VASODEPRESSOR SYNCOPE

Fainting (vasodepressor syncope or vasovagal reaction) is a debatable cause of loss of consciousness in divers.[53] The increased central blood volume during immersion is thought to protect against the hypotension and subsequent reduction in cerebral blood flow. In very warm water, however, some of this protection may be lost because vasodilatation reduces the extent to which central blood volume is increased. I know of two cases of syncope in subjects immersed in water at 35°C. In both instances, syncope was related to flushing of an indwelling arterial catheter. Diagnosis of vasodepressor syncope should require a clear antecedent cause (e.g., pain). If the diver is recovered from the water quickly, significant bradycardia should be present. Hunger, fatigue, and hangover predispose to vasodepressor syncope.

CARDIAC ARRHYTHMIA

Serious cardiac arrhythmia leading to confusion and helplessness, frank loss of con-

sciousness, or even death is always possible in the water, as it is on land. In water, several factors may increase the chances of arrhythmia: a dilated right side of the heart from increased central blood volume, increased circulating catecholamine levels, activated autonomic nervous system reflexes by cold exposure, and, occasionally, respiratory acidosis from carbon dioxide retention. One study showed that arrhythmia was 22 times more likely in water than on dry land.[55]

Arrhythmias have been implicated in several situations.[53] One example is the diver who, while swimming back to base after completion of a shallow no-decompression air dive, fails to keep up with companions, complains of fatigue and perhaps breathlessness, calmly requests help, and then passes out and sinks. The time on the surface is too long to permit diagnosis of CAGE. In another situation, an open-circuit air scuba diver on a shallow dive signals to buddy-breathe but rejects the regulator when it is offered and then passes out. The diver's cylinder contains ample air, and no contaminants are found. Unless the diver is being monitored during the event (a rare situation even in commercial and military diving), the diagnosis of arrhythmia has to be one of exclusion. Only a few suspected cases show evidence of coronary occlusion at autopsy. One unusual case revealed endomyocardial fibrosis.[56] An arrhythmia should be suspected when loss of consciousness occurs without obvious explanation in a middle-aged diver on a working dive in cold water, especially if the diver has a history of cardiac disease and is taking cardiac or antihypertensive drugs. Further discussion of cardiac arrhythmias can be found in Chapter 25.

NITROGEN NARCOSIS

Nitrogen narcosis can produce severe disturbances of consciousness and even loss of consciousness on air dives deeper than 300 fsw (90 msw). At lesser depths, however, euphoria, poor judgment, and impaired performance, but not loss of consciousness, can be expected.

HYPOTHERMIA

Moderate hypothermia occasionally occurs in divers. The diver may appear confused, may perform assigned tasks robotically, or may fail to respond to verbal commands. Apart from an accidental or uncontrolled exposure of a diver to cold water, however, hypothermia sufficient to induce unconsciousness would not be expected. The diagnosis of hypothermia can be based on the environmental exposure, the gradual onset of symptoms, the absence of other known causes for the disturbance in consciousness, the cold, blue skin, and a rectal temperature lower than 35°C.

HYPERTHERMIA

Heat exhaustion, or even frank heat stroke, can develop after dives in hot water. The diver is also at risk for these conditions during recompression therapy in hot climates, especially when the chamber is exposed to direct sunlight. Heat exhaustion leads to mild confusion but not to loss of consciousness. Heat stroke, on the other hand, is characterized by confusion, delirium, disorientation, seizures, and eventual coma; focal neurologic signs may be present. Hyperthermia should be suspected as a reason for loss of consciousness when the water temperature is greater than 35°C. Heat stroke can be diagnosed from knowledge of the environmental conditions; hot, dry skin; hypotension; and a rectal temperature higher than 40°C.

DECOMPRESSION SICKNESS

Complete loss of consciousness from DCS is unusual. More often, the practitioner encounters cases of DCS with collapse and semiconsciousness. The diagnosis of DCS should be considered when the decompression obligation has been extensive and the condition presents late in the decompression or after surfacing. Collapse and complete or partial loss of consciousness occurring more than 10 min after a diver leaves the water permit a presumptive diagnosis of DCS. Almost all other causes can be eliminated by this time.

Vertigo

Vertigo is common in divers, and the average physician or diving medical technician can

spend many anxious moments trying to sort out the probable cause. Many medical conditions are associated with vertigo, and the differential diagnostic list is very long. Divers with a history of recurrent benign vertigo may aggravate the disorder by diving. This discussion covers conditions unique to diving.

When confronted with the general complaint of dizziness, medical personnel must first establish whether true vertigo is present. This is done by eliciting a history of a sensation of motion—patients feel themselves moving or feel that the environment is moving around them. Vertiginous symptoms are generally described as whirling, spinning, rotating, tilting, rocking, or undulating. Oscillopsia may be present. True vertigo in the diving setting is most often accompanied by pallor, sweating, nausea, and occasionally vomiting.

Vertigo may be caused by lesions of the membranous labyrinth, of the eighth cranial nerve, or of the vestibular nuclei and their central connections. In most diving instances, these lesions are destructive, leading to loss of function. Vertigo thus results from the unopposed signals emanating from the normal side. Many sophisticated tests distinguish among peripheral, eighth-nerve, and central lesions, including audiometry; auditory brain stem evoked response; stapedius reflex measurement; electronystagmography with caloric, rotational, positional, and optokinetic stimulation; smooth harmonic acceleration testing; and saccadic eye and smooth eye pursuit. Several authors[57–59] have described the usefulness of these tests in the diagnosis of diving injuries. Unfortunately, few if any of these sophisticated tests are immediately available to the diving physician. In the field, the diagnosis rests almost exclusively on the history (i.e., at what point in the dive the problem occurred) and the physical examination.

PHYSICAL EXAMINATION IN DIVERS WITH VERTIGO

The physical examination should be used to establish the presence of a vestibular disorder and to distinguish between a central and a peripheral vestibular lesion. This distinction is important because central lesions always require recompression therapy, whereas some peripheral lesions (e.g., IEBT) may not. Unfortunately, the various tests to distinguish central from peripheral findings are not 100% accurate. Some central lesions may behave like peripheral ones, and vice versa. Nevertheless, these tests should always be performed and interpreted in the context of the patient's overall presentation. The following examinations are recommended.

Examination of General Balance

Positive results with general tests of balance, such as the Romberg test and the past-pointing, tandem-walking, and clock-walking tests, suggest a vestibular disorder. In a peripheral vestibular lesion, the Romberg test yields positive results, with the patient falling to the side of the lesion. Past pointing to the affected side also occurs. These general tests of balance are nonspecific with regard to localization.

Examination for Spontaneous Nystagmus

Spontaneous nystagmus indicates a vestibular disorder. In acute peripheral labyrinthine lesions, spontaneous horizontal nystagmus is generally present, with the fast component directed to the side opposite the lesion.§ The amplitude and frequency of the nystagmus *increase* when the eyes are directed 30 degrees from the midline in the direction of the fast component and *decrease* when the eyes are directed 30 degrees from the midline in the opposite direction. In mild cases, nystagmus may be present only when gaze is directed toward the fast component. If visual fixation is broken (e.g., with Frenzel lenses), the amplitude of nystagmus is enhanced. In mild peripheral involvement, spontaneous nystagmus may appear only when visual fixation is abolished.

Enhancement of nystagmus by loss of visual fixation is characteristic of a peripheral lesion. In a central lesion, spontaneous nystagmus is unchanged or inhibited when visual fixation is abolished. Peripheral labyrinthine nystagmus involves both eyes (i.e., it is always conjugate), and both eyes beat in the same direction. Spontaneous vertical nystagmus is always a sign of a central lesion.

Examination for Positional Nystagmus

Peripheral labyrinthine vertigo is generally exacerbated by head movement. Indeed,

after the initial insult has subsided, vertigo and nystagmus may occur only after changes in head position. Thus, tests for positional nystagmus are always indicated. In the Nylen-Bárány test for positional nystagmus (also called the Hallpike maneuver), the patient's head is first rotated 45 degrees to the right or left; the patient is then rapidly moved from the seated to the supine position, and the head is allowed to hang over the edge of the examining table by 45 degrees. The eyes are kept open and in midposition. In a peripheral labyrinthine lesion, nystagmus appears after a latency period of 2 to 10 sec, reaches a peak in 2 to 10 sec, and then rapidly subsides. The nystagmus lasts approximately 30 sec. The nystagmus is horizontal and rotary and beats toward the affected ear when it is placed lowermost. Intense vertigo usually accompanies the nystagmus. When the affected ear is uppermost, no nystagmus or vertigo results. If the test is repeated immediately, the resultant nystagmus is generally diminished or absent, indicating fatigability of the response.

When the Hallpike maneuver is performed in cases of vertigo secondary to central causes, a different pattern emerges. The nystagmus starts immediately (i.e., it has no latency) and generally persists as long as the head remains in the dependent position (or at least for 1 min); it is associated with little or no vertigo. Nystagmus occurs in both right and left head positions, and the rapid component is generally directed upward in both cases. The direction may be variable, however, and may be upward, downward, or changing. This central type of response does not fatigue.

In addition to the Hallpike test, the patient should be examined for sustained spontaneous nystagmus that appears only in certain head positions. The direction of this nystagmus is variable. Failure to suppress spontaneous positional nystagmus with visual fixation suggests a central disorder.

Examination of Eye Pursuit

Saccadic eye pursuit and smooth eye pursuit should be normal in peripheral labyrinthine lesions. Overshooting or undershooting of the target during saccadic-pursuit testing or frequent corrective saccades during smooth-pursuit testing suggests a central lesion. Spontaneous nystagmus may present the uninitiated examiner with difficulties in separating the two types of eye movement.

Examination for Associated Hearing Loss

Associated tinnitus or neurosensory hearing loss suggests a peripheral lesion. Vertigo and hearing loss generally do not coexist in a central disorder.

Examination for Associated Neurologic Findings

Convulsions, unconsciousness, and cranial nerve findings (except for findings related to the eighth nerve) are not found in peripheral labyrinthine lesions.

DIFFERENTIAL DIAGNOSIS OF VERTIGO

Table 18–6 summarizes the differential diagnosis of central and peripheral vertigo in diving.

Determination of the cause of the diver's vertigo generally rests on the phase of the dive in which the vertigo first became manifest, the duration of the attack, and the associated symptoms. The following conditions should be considered in the differential diagnosis of vertigo.

Caloric Vertigo

Rupture of the tympanic membrane causes violent vertigo lasting up to 1 min when cold water enters the middle-ear space. This condition generally occurs during descent but may occur at any depth because of an underwater blast, sound bursts from a sonar device, or other shock waves. Generally, the diver knows exactly what happened. Examination reveals a ruptured tympanic membrane. Examination should disclose no vertiginous symptoms.

Unilateral obstruction of the external ear canal generally produces a mild and relatively short-lived episode of caloric vertigo shortly after the diver enters cold water. Examination shows an obstructed external canal secondary to cerumen or otitis externa. Vertigo is caused by cold water gaining access to one ear and not the other. Examination should disclose no vertiginous symptoms.

Transient caloric vertigo may occur during dives with hot-water suits if the hot

Table 18–6. Differential diagnosis of central and peripheral vertigo in diving

	Peripheral Vertigo	Central Vertigo
Symptoms	Generally intense with nausea and vomiting	May be intense but are often milder
	Generally affected by head movement; one head position may be critical	Only slightly responsive to head movement
Spontaneous nystagmus	Horizontal or rotatory, never vertical	All forms possible
	Suppresses with visual fixation	Unchanged or enhanced by visual fixation
	Gaze direction dependent	May depend on gaze direction
	Always conjugate	May be disconjugate
Positional nystagmus	2–10 sec latency period	No latency
	Short-lived	Generally persists
	Positive when affected ear is positioned downward	Positive when either ear is positioned downward
	Direction fixed	Direction changing
	Response fatigues on repeat testing	Response does not fatigue
Saccadic and smooth eye pursuit	Normal	Abnormal
Associated auditory findings	Frequent	Very infrequent
Neurologic examination results	Normal*	Abnormal

*Peripheral labyrinthine lesions in decompression sickness and cerebral arterial gas embolism may be associated with an abnormal result on neurologic examination.

water gains access to one ear and not the other. Examination should reveal no vertiginous symptoms.

Alternobaric Vertigo

ABV developing during descent generally lasts less than 1 min and often follows a forceful Valsalva maneuver by a subject with difficulty clearing the ears. ABV developing during ascent generally lasts for only seconds but may persist for up to 10 min. In approximately 3% of cases, the ABV lasts 10 to 60 min; in 1% of cases, the ABV lasts 60 min to 10 hours.[60] ABV of ascent always starts while the diver is moving upward in the water column and is usually associated with fullness or pain in the affected ear. Immediate relief of symptoms by descent is diagnostic. Molvaer and Albrektsen[61] have reviewed the risk factors for ABV.

Inner-Ear Barotrauma with Perilymph Fistula

IEBT with perilymph fistula is characterized by the sudden onset of sustained vertigo in a subject who has experienced difficulty clearing the ears during descent. IEBT usually presents during descent or shortly after reaching the bottom. However, the onset of vertigo may be delayed until after surfacing, when a small fistula is suddenly enlarged or the perilymph loss is suddenly increased by straining during a bowel movement or while lifting heavy weights. A perilymph fistula may also develop during ascent secondary to a large overpressure in the middle ear.

A small perilymph fistula may lead to only a complaint of unsteadiness or ataxia during walking or to a complaint of episodic vertigo related to changes in head position, sneezing, or coughing. A large fistula produces steady vertigo, at least initially. Perilymph fistula is usually associated with a sensation of fullness, deafness, and tinnitus or a sensation of bubbling in the affected ear. The tinnitus may be described as roaring. Although coexisting auditory involvement has been absent in some cases,[62–64] involvement of the auditory mechanism is common. The hearing loss is neurosensory. In one series, only five cases of normal hearing were detected in 40 proven cases of fistula.[62] The hearing loss fluctuates in intensity. Sounds may be distorted, and the patient may be intolerant of loudness.[62] Speech discrimination scores were less than 80% in 75% of cases in one series.[62] Hearing may improve slightly when the affected ear is positioned upward. Vertigo and nystagmus associated

Table 18–7. Differential diagnosis of vertigo after surfacing

	ABV	IEBT	DCS	CAGE
Onset	Within 2 min	Anytime	Anytime	Within 10 min
Duration	Usually short, <10 min	Persistent	Persistent	Persistent
Associated neurologic findings	Absent*	Absent	Possible	Common
Decompression stress required†	No	No	Yes	No
Difficulty clearing/evidence of MEBT	Not required	Generally present	Not required	Not required
Coexisting auditory signs	Unusual	Very common (88%)	Common (38%)	Less common
Nystagmus	Peripheral	Peripheral	Central or peripheral	Central or peripheral
Fistula test	Unknown	May be positive	Unknown	Unknown

*Except for alternobaric facial palsy.
†Sufficient time has elapsed on the bottom to allow for inert gas absorption. For sensitive individuals, this may be inside the no-decompression limits.
ABV, alternobaric vertigo; CAGE, cerebral arterial gas embolism; DCS, decompression sickness; IEBT, inner-ear barotrauma; MEBT, middle ear barotrauma.

with positional testing are of the peripheral type described earlier.[62]

A fistula test may help to establish the diagnosis. A good seal of the external ear canal is obtained with a pneumatic otoscope, and several puffs of air are delivered. A positive response consists of a forced deviation of the eyes away from the side of the stimulus. This may or may not be followed by a few beats of nystagmus. Fresnel lenses help to reduce the suppressive effect of ocular fixation and aid in observing the response. This test yields positive results in 25% to 40% of known fistula cases.[62,63] However, a 10% to 20% rate of false-positive results has also been reported.[63,65] Using impedance audiometry to generate precise pressure fluctuations and electronystagmography to record the results, Daspit and colleagues[65] reported a diagnostic accuracy of 90.8%. The false-positive and false-negative rates were 4.5% each. Recently, transtympanic electrocochleography has been proposed as a test for perilymph fistula.[66] Unfortunately, impedance audiometry, electronystagmography, and electrocochleography are not available in the field.

When symptoms of IEBT first appear during ascent or after surfacing, CAGE or DCS must be ruled out. This is important because inappropriate recompression might cause further damage in cases of IEBT.[67] In practice, the distinction may be very difficult. The differential diagnostic points are summarized in Table 18–7. The diagnosis of perilymph fistula should be considered when:

- There is a clear history of difficulty in equalizing middle-ear pressure and otoscopic evidence of middle ear barotrauma.
- The dive did not require decompression.
- No emergency ascent was involved.
- Coexisting auditory signs are present.
- No other focal neurologic signs are present.
- Other stigmata of DCS such as musculoskeletal pain are absent.

If the onset is delayed 10 min after surfacing, CAGE can be ruled out. Both a perilymph fistula and inner-ear DCS were present in at least one case.[68]

Inner-Ear Barotrauma Without Perilymph Fistula

IEBT without perilymph fistula occurs in conditions essentially identical to those for perilymph fistula with comparable symptoms except surgical exploration of the middle ear reveals no fistula. When this problem first appears during or after ascent, CAGE and DCS must be ruled out, as described earlier.

Decompression Sickness

Vertigo is a common manifestation of DCS. Dizziness, vertigo, or symptoms relating to the vestibular mechanism are reported in 4.4% to 18% of the cases reported in various series.[37,69,70] In deep, nonsaturation helium-oxygen decompression diving, vertigo may be the dominant symptom of clinical DCS.[71] The

shallowest reported case occurred on a 25 msw air dive with a 40 min bottom time.[72] Vertigo may occur from lesions affecting the labyrinth, the eighth cranial nerve, or the central vestibular nuclei and their connections. More than one site may be involved.[57] Vertigo may occur after the long pull to the first stop on deep dives, with deep gas switches from helium to air, or during the later stages of a particularly arduous decompression,[73] but vertigo is most common some time after surfacing. In one case reported by Farmer and coworkers,[73] vertigo occurred 206 min after the diver surfaced. Vertigo is often, but not invariably, associated with tinnitus and neurosensory deafness in the affected ear. In 16 cases of inner-ear DCS and vertigo, Farmer and coworkers[73] noted coexisting auditory symptoms in six patients. Other stigmata of DCS (rashes, limb pain, hypesthesia, paresis, or chokes) are variously present. Of 23 patients with inner-ear DCS reported by Farmer and coworkers,[73] 5 (22%) had other symptoms of DCS, generally pain. In 18 patients with inner-ear symptoms, Erde and Edmonds[38] noted coexisting musculoskeletal symptoms in 33%. Before inner-ear DCS is diagnosed, IEBT and gas embolism must be ruled out (see Table 18–7).

Cerebral Arterial Gas Embolism

The sudden onset of vertigo during ascent or within 10 min of surfacing may be a sign of CAGE. Central vestibular mechanisms are most commonly involved, although isolated embolization of the internal auditory artery or its vestibular branches may occur. Pearson reported that vertigo was a presenting symptom in 22 of 100 cases of gas embolism.[36] Vertigo secondary to gas embolism is infrequently associated with tinnitus and neurosensory hearing loss. It is most often accompanied by other rapidly evolving focal neurologic signs. The patient may rapidly become unconscious. Subcutaneous supraclavicular crepitus may be present, suggesting pulmonary barotrauma as the cause. Hemoptysis is not a constant sign but, when present, helps support the diagnosis. The differential diagnosis includes DCS and IEBT. Severe DCS may be ruled out if the dive was well within the no-decompression limits. IEBT should be considered in the absence of focal neurologic signs (see Table 18–7). Parell and Becker[74] report one case in which CAGE and a documented perilymph fistula occurred simultaneously.

Central Nervous System Oxygen Toxicity

CNS oxygen toxicity may lead to the sudden onset of vertigo while the diver is breathing oxygen at a high partial pressure on the bottom or during decompression stops. Vertigo may or may not be associated with other symptoms of oxygen toxicity such as nausea, tunnel vision, or muscular twitching. The vertigo rapidly abates when the oxygen partial pressure is lowered, which confirms the diagnosis.

Inert-Gas Isobaric Counterdiffusion

This type of sustained vertigo occurs under two conditions: (1) at stable depths of approximately 600 fsw or greater when the diver breathes nitrogen or neon while the body is surrounded by helium[75] and (2) during decompression from deep nonsaturation helium-oxygen dives when a transition from a helium-oxygen mixture to air occurs deeper than 100 fsw.[73] The first condition exists only in the experimental laboratory and is unlikely in a diving operation unless a mistake in gas switching is made. This type of vertigo has not been associated with auditory findings. The second situation is more likely on a dive because deep air shifts are common. Farmer and colleagues[73] noted coexisting auditory findings in one of four cases related to an air shift. Care must be taken to distinguish true vertigo from the intense nitrogen narcosis that can be experienced with a sudden shift from a helium-oxygen mixture to air. If true vertigo is present, the diver should be restored to the complete helium environment and undergo recompression. The condition should be treated as DCS.

Nitrogen Narcosis and High-Pressure Nervous Syndrome

Dizziness and unsteadiness may be associated with nitrogen narcosis and high-pressure nervous syndrome. In the latter condition, the cerebellum appears to be the target organ. Neither condition is associated with true vertigo.[25]

Hearing Loss

Complaints of tinnitus and hearing loss in diving are common. The first step is to determine whether a true hearing loss exists when

a patient complains of hearing loss, stuffiness of the middle ear, or tinnitus. In the clinic, this is best done by pure-tone audiometry; in the field, tuning forks, watches, and the whispered and spoken voice must be used. If a hearing loss is discovered, it is imperative to determine whether this loss is conductive or neurosensory because neurosensory losses may require more urgent therapeutic intervention. In the clinic, audiometry is used to make this determination. Tuning forks must be used in the field.

Three tuning fork tests are useful in distinguishing conductive from neurosensory losses: the Rinne test, the Weber test, and the Schwabach test. A 512 Hz fork should be used at the start. In the Rinne test, the tuning fork is struck and placed firmly on the mastoid process of the ear to be tested. When the patient no longer hears the vibrating fork, it is placed 2 cm opposite to the auditory meatus until the sound disappears. The test may also be performed by alternately placing the fork on the mastoid bone and opposite the meatus until the sound disappears in one of the two locations. Normally, the fork is heard longer and more intensely by air conduction than by bone conduction. A conductive loss is present if the air conduction time is less than the bone conduction time, or if the sound is heard more intensely by bone conduction than by air conduction. If a conductive loss is found, the result should be confirmed with a 1024 Hz fork. The 512 Hz fork indicates that bone conduction is greater than air conduction when the conductive loss is 20 dB or greater; the 1024 Hz fork so indicates when the conductive loss is 25 dB or greater.

In the Weber test, the tuning fork is struck and placed against the center of the patient's forehead, and the patient indicates the ear in which sound is heard best. With a conductive loss, hearing is better in the affected ear; with a neurosensory loss, hearing is better in the normal ear. In the Schwabach test, examiners compare their own hearing by bone conduction with that of the patient's affected ear. In a conductive loss, the patient hears longer; in a neurosensory loss, the examiner hears longer. Table 18–8 summarizes the tuning fork tests.

Tuning fork tests may be very difficult or impossible to conduct adequately in a noisy shipboard environment. When such tests are not possible, the presence of middle-ear barotrauma, as determined by otoscopic examination, may suggest a conductive loss, but it does not absolutely rule out neurosensory loss.

In the clinic, pure-tone audiometry with air and bone conduction should be used to assess whether the hearing loss is conductive or neurosensory. In addition, a wide variety of specialized tests are available to pinpoint the location of a lesion in neurosensory losses as either cochlear or retrocochlear. These tests include speech reception threshold and speech discrimination, Békésy audiometry, the alternate binaural balance test for recruitment, the short-increment sensitivity index, tone decay, acoustic reflex tests, auditory evoked response tests, and various tests for central auditory function. These tests have been described in detail by Katz,[76] and their use in diving cases has been illustrated by Caruso and colleagues[57] and Shupak and associates.[58]

Conductive losses are the most common hearing losses in divers, and the cause can usually be confirmed by otoscopic examination. The major conditions that lead to conductive hearing loss in diving are severe middle-ear squeeze with or without tympanic membrane rupture, obstruction of the external ear canal by cerumen or severe otitis externa, disarticulation of the auditory ossicles or dislocation of the stapes footplate secondary to middle-ear barotrauma, and serous otitis media.

The major conditions that lead to neurosensory hearing loss in diving are noise trauma, IEBT, DCS, and gas embolism. Neurosensory hearing loss has been reported as a consequence of carbon monoxide poisoning.[77]

Table 18–8. Comparison of various tuning fork tests to determine type of hearing loss

Type of Loss	Rinne Test	Weber Test	Schwabach Test
None	AC > BC	Midline	Equal
Conductive loss	BC > AC	Lateralizes to affected ear	Patient hears longer
Neurosensory loss	AC > BC	Lateralizes to unaffected ear	Examiner hears longer

AC, air conduction; BC, bone conduction.

Noise trauma is, by far, the most common cause of neurosensory hearing loss in divers. Transient auditory threshold shifts of 20 to 30 dB lasting up to 24 hours are not uncommon after noisy dives.[78] Tinnitus is usually present. Comparison of pre- and postdive audiograms reveals the extent of the loss. The diagnosis is usually straightforward and can be based on the presence of high noise levels during the dive and the absence of other probable causes. Repetitive noise trauma leads to permanent neurosensory losses, and most divers show such changes. The hearing loss most commonly affects auditory frequencies above 4000 Hz.

IEBT (with or without perilymph fistula) presents as tinnitus, a feeling of ear blockage, and neurosensory hearing loss, often but not invariably accompanied by vertigo. The tinnitus may be described as roaring. Three patterns of hearing loss have been observed:

- A flat line, that is, a major loss across all frequencies from 250 to 8000 Hz
- A linear decrease in auditory acuity as frequency increases
- A relative preservation of auditory acuity at the lower frequencies with a precipitous falloff at higher frequencies[64,79–82]

One case of a midfrequency loss concentrated at 1000 Hz has been reported.[83] Speech discrimination may be very poor.[62,64] A fistula test may yield positive results. Molvaer and associates reported that 30 of 83 inner ears (36%) damaged by barotrauma displayed only cochlear symptoms.[84] For purposes of differential diagnosis, therefore, it can be stated that isolated hearing loss is not uncommon, and the absence of vertiginous symptoms should not rule out this diagnosis. Auditory symptoms related to IEBT may begin during descent, during ascent, or after surfacing. DCS and CAGE must be ruled out in cases that occur during ascent or after surfacing. As in the differential diagnosis of vertigo, this can be done when the history reveals difficulty in equalizing middle-ear pressure, when other stigmata of DCS and CAGE (e.g., joint pain or other neurologic signs) are absent, when the depth and time of the dive are within no-decompression limits, and—in the case of CAGE—when the symptoms begin more than 10 min after surfacing (see Table 18–7). The absence of middle-ear barotrauma on otoscopic examination does not rule out the possibility of IEBT.[85]

DCS is a common cause of neurosensory hearing loss in divers. Tinnitus and hearing loss occur during or following ascent from a dive, most commonly a deep dive requiring a fair amount of decompression. The hearing loss may be partial or complete. There is no characteristic audiometric pattern. Vertigo is often, but not invariably, present. The other stigmata of DCS are frequently present and aid diagnosis. DCS-induced hearing loss has been reviewed.[86]

Gas embolism of the internal auditory artery may present as the sudden onset of tinnitus and deafness during ascent or within 10 min of surfacing from any dive. It is usually associated with vertigo. Other focal neurologic deficits are also generally present, representing the effects of emboli at other locations. This condition may be very difficult to distinguish from DCS. A short or shallow dive, the onset of symptoms within 10 min of surfacing, and the absence of joint pain or skin rash favor this diagnosis.

Diving may also be associated with disruptions in central auditory processing, although pure-tone audiometry results are normal.[57] Such lesions are almost always the sequelae of DCS or CAGE.

CONCLUSIONS

Most diving operations will be free of major medical problems if medical personnel select the divers and equipment carefully and engage in meticulous predive planning. Each phase of the dive should be carefully evaluated, and comprehensive contingency plans should be established. Such an exercise may result in different equipment being chosen or in different approaches being taken. Once an operation is underway, medical personnel should be vigilant in detecting incipient problems. Thorough familiarity with the equipment and knowledge of what may go wrong during each phase of the dive are essential for effective monitoring of diving operations.

Once a problem has occurred, the nature of the presenting complaint and the phase of the dive in which the symptoms first became apparent are the most important clues for diagnosis and treatment. Barotrauma of the ears and paranasal sinuses is the most common complaint, followed by seasickness, sunburn, and various traumatic and envenomation injuries. Depending on the tables

used, DCS occurs in approximately 1% of divers requiring decompression; the deeper and longer the dive, the more likely the occurrence of DCS. Oxygen toxicity may occur when oxygen limits are approached or exceeded. Carbon dioxide intoxication, hypoxia, and contaminated gas episodes are infrequent. Pulmonary barotrauma also is infrequent, but evidence for this condition should always be sought whenever respiratory or neurologic symptoms are present.

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[†]Pneumoperitoneum is a very rare and generally asymptomatic condition usually associated with pulmonary barotrauma. Because it may arise from a ruptured viscus, however, the first manifestation may be abdominal pain and signs of peritonitis that appear some length of time after completion of the dive.

[§]A useful mnemonic device is **COWS,** which stands for **C**old **O**pposite, **W**arm **S**ame. Although it applies to the results of caloric testing, it can also be used to describe the behavior of a paretic or dead (cold) labyrinth or an irritated (warm) labyrinth.

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CHAPTER 19 Women in Diving

Maida Beth Taylor

Since the 1970s, the sport diving world has seen a sweeping set of changes. Once diving was a sport for men. Now women account for 30% to 35% of the community of active recreational divers and have established their place in commercial, scientific, and military diving. Manufacturers now design equipment to suit the physical differences between men and women. The publication of *Scuba Diving: A Woman's Guide* is an indicator of how much things have changed.[1]

Men and women divers have more in common than not. But there are anatomic and physiologic differences that can influence diving hazard and risk. In writing about women and diving, the major differences are generally addressed and, after that, largely ignored. The most obvious and serious differences emanate from female reproductive functions, namely pregnancy and menstruation. To a lesser extent, female gender limited diseases, usually urogenital disorders, may influence fitness to dive. This chapter examines female sports performance, the physiology of pregnancy and fetal gas exchange in utero, and specific questions on common health concerns of women. The chapter also defines particular problems encountered by women divers and seeks to answer the question: Are women different from men at depth?

The first part of this chapter briefly reviews the cardiopulmonary and musculoskeletal differences in women that alter sports performance. The impact of heavy athletic activity on adolescent development, endocrine function, and reproductive function is discussed. The literature on diving disorders, particularly decompression syndrome (DCS), in women is reviewed at length, and risk factors specific to women, such as increased body fat, menstruation, and contraception, are discussed. A detailed review of the effects of diving and hyperbaric conditions on the fetus and pregnancy follows.

As the population of divers continues to grow and as the relative and absolute numbers of women divers grow, the issues covered in this chapter will become increasingly important to medical personnel practicing and consulting in the field of diving medicine.

ANATOMIC AND PHYSIOLOGIC SEX DIFFERENCES AFFECTING SPORTS PERFORMANCE

Although the differences may not be universal, owing to great genetic and individual variation and overlap, women typically have a lower peak performance capacity than do men by virtue of smaller cardiovascular, pulmonary, and skeletal systems. Smaller bones with smaller articular surfaces carry less weight. Leg length is shorter and accounts for 51% of total height in women compared with 56% in men. The shoulder width is narrower, while the hips are wider, with an attendant increased valgus angulation of the knee inward and a greater varus angulation at the hip outward. These skeletal modifications, in conjunction with a lower center of gravity, provide women with a narrower stance for balance.

Physiologically, women possess less potential for power, speed, work capacity, and stamina than do men. At a specified height, a woman has a smaller heart than does a man. This, together with smaller lungs, a smaller thorax, and smaller stroke volume, means that women cannot functionally achieve the maximal oxygen consumption capacity that men can. Despite training, women have a higher percentage of body fat. For example, sedentary college-age women have approximately 25% body fat, whereas trained athletic women reach 10% to 15% body fat. Trained

males, however, average 7% to 10% body fat. In trained males, muscle makes up 40% of total body mass, whereas comparably fit women have only 23% muscle.[2]

Women possess other physical differences with seemingly more positive aspects. Using a number of mechanisms, women's bodies conserve energy better than do men's. Increased body fat provides better insulation from heat loss and allows increased buoyancy. The sudorific response (sweating) occurs at a higher core temperature, again conserving energy but making women perhaps more susceptible to heat stress. The basal metabolic rate is lower in women, which lowers basic caloric needs and resting oxygen demand. Women also demonstrate more tendon laxity and flexibility, allowing for greater range of motion but also predisposing to torsion and dislocation injuries.

The functional anatomy of women predisposes them to certain injuries in sports. The increased angulation of the knee and hip joints causes lateral malposition of the patella. This angulation, coupled with overuse, makes women prone to a set of abnormalities known as patellofemoral stress syndrome, a nonspecific term that includes patella subluxation, chondromalacia patellae, lateral patella compression syndrome, and patella tendonitis. All variations of the disorder are usually treated with a combination of rest for acute symptoms and exercise aimed at strengthening the vastus medialis to help stabilize lateral slip of the patella. Severe cases in competitive athletes may require surgical stabilization of the patella.

Women are also at increased risk for shoulder injuries, including anterior subluxation, shoulder impingement, and thoracic outlet syndrome. This group of injuries often occurs in swimmers because tendon and muscle hypertrophy puts stress on points of insertion and articulation. Neck injuries, particularly ballistic forms of trauma such as whiplash, are also common. Cranial size, volume, and weight are only slightly smaller in women, whereas the bones and muscles of the neck are significantly smaller. When the head accelerates or decelerates suddenly, the force generated may be similar to that in a man, but the supporting structures are much smaller and more vulnerable to injury. This type of injury is not common in the surface swimmer, but platform and springboard divers are at risk.

EXERCISE, DEVELOPMENT, AND REPRODUCTIVE ENDOCRINOLOGY

Pubertal Development

Since the beginning of the twentieth century, the age of menarche in the industrialized world has fallen from 17 to 12. Generally, one can say that girls are reaching their terminal height at an earlier age, and soon thereafter they reach the critical body mass necessary for initiation of cyclic ovarian hormone production and, eventually, ovulation. *Puberty* is a vague term assigned to the triad of developmental landmarks—thelarche, pubarche, and menarche. These events are linked, temporally and physiologically, with breast and pubic hair development starting between ages 12 and 13 years. The onset of these developmental landmarks varies greatly, as does the rapidity of their progression and their interrelationships; in general, however, in 66% of girls, breast development starts first, followed by growth of pubic hair, and then an accelerated rate of linear growth before menstrual function starts. The most constant predictor of menses is the decrease in the rate of growth—the deceleration phase of the peak height velocity. The decline of the rate of growth heralds the onset of menarche, which usually occurs within 6 months.

Pubertal development is mediated and altered by many factors including weight, height, heredity, nutrition, environment, and climate. Because of its effects on weight, body fat, and caloric requirements, exercise can profoundly alter developmental landmarks. Most of the research in this area has been done by Warren and Frisch and others,[3–7] studying developmental delay in young ballet dancers, but their findings also apply to other young, competitive athletes who run, swim, or bicycle. First, menarche is delayed often by 2 to 4 years. Bone age is also consistently 2 to 4 years behind chronologic age in premenarcheal athletes. Menstrual cycles, once initiated, are often infrequent or irregular. The high incidence of observed oligomenorrhea and irregular cycles continues into young adult life if high levels of activity persist.

Athletes reach their terminal height at a later age than do controls, owing to later closure of the epiphyses, but the final height

does not appear to be altered. Similarly, breast development is delayed. Young female athletes at puberty gain weight more slowly, gain less, and stop gaining sooner than do sedentary controls. During periods of inactivity, such as during summers when intramural competitions cease or during times of injury, young athletes may experience rapid developmental progression, implying that rest allows energy to be directed to development rather than to sports activity.

Interestingly, both Marcus and colleagues[8] and Warren and associates[9] have reported increased orthopedic problems in young female athletes who are amenorrheic. Warren and coworkers found an increased incidence of scoliosis in amenorrheic dancers and found that the incidence correlated with duration of amenorrhea. Similarly, Frisch and colleagues[5] noted an increased incidence of stress fractures in teenaged and young adult runners, with the injury rate again increasing with the duration of amenorrhea.

Girls who continue their athletic endeavors into young adult life and women who undertake intensive athletic training during their reproductive years are likely to have continued impaired reproductive function. The incidence of amenorrhea in top female athletes is reported to range from 3.4% to 66%, compared with 2% to 10% in the general population. Vigorous exercise induces a progressive impairment of gynecologic endocrine function.

Initially, the luteal phase of the menstrual cycle shortens from the normative 14 days to 8 to 10 days. This aberration can be detected by both a lower peak and a shortened duration of the rise in basal body temperature. Serum progesterone levels parallel the temperature changes, with lower progesterone levels in the luteal phase in athletes. Peak levels of progesterone are lower and persist for a shortened duration, reflecting the impaired function of the corpus luteum. As the endocrine abnormality progresses, anovulatory cycles ensue, with loss of the luteinizing hormone (LH) surge at midcycle, no ovulation, no corpus luteum formation, and cessation of progesterone secretion.[10–14] Uterine bleeding may continue to occur as scant, irregular bleeding or as light flow at irregular, long intervals. The disorder then progresses to a state of euestrogenemic amenorrhea, with serum estrogen levels high enough to maintain secondary sex characteristics and bone density but not high enough to cause endometrial proliferation.

Further progression of endocrine dysfunction leads to hypoestrogenemic amenorrhea. At this stage, the regulatory mechanisms in reproduction are profoundly disrupted. Endocrine tests reveal prepubertal hypothalamic function with low tonic level of LH and follicle-stimulating hormone (FSH), decreased pulse frequency of LH secretion, and an LH/FSH ratio of less than 1.0.[15,16] Prolonged, continued impairment of the hypothalamus offers little ovarian stimulation, and serum estrogen levels—most notably the level of estradiol, the potent estrogen of the reproductive system—fall. If low levels of estradiol persist, regression of secondary sex characteristics, particularly breast development, can occur.

Bone Development

More important than its effect on sexual development, from the vantage point of the coach, trainer, or physician, is the role estradiol plays in maintenance of bone. The skeleton is composed of two types of bone, which are structurally and metabolically different. Cortical bone comprises the shafts of the long bones and is traditionally measured at the junction of the lower and middle thirds of the radius. Trabecular bone comprises the vertebral bodies, pelvis, flat bones, and ends of the long bones. Measures of trabecular bone are done on the vertebral column or on the wrist or distal radius. Vertebral measures, though more important clinically in determining bone density, are less sensitive and less reproducible than measures of the wrist or radius. Cortical bone is less metabolically active than trabecular bone. Cortical bone remodels at a rate of 10% per year, whereas trabecular bone turns over at 40% per year. Thus, trabecular bone is more vulnerable to agents or events impairing bone metabolism.

Bone mineralization reaches a maximum rate in early adult life and continues through age 35. One might think of the early adult years as a time for making deposits in a "bone bank" account. This prepares a woman for the withering of bone later in life when estrogen levels fall. Before menopause, cortical bone is lost at a rate of 0.3% to 0.5% per year. In early menopause, this rate of loss increases to 2% to 3%. Being more metabolically active, trabecular bone is lost at a rate of 1.2% to 2.4% per year prior to menopause.

The rate accelerates to 6% per year for the first 2 years after the climacteric. Thus, 25% of cortical and 32% of trabecular bone is lost between ages 50 and 80.[17]

Amenorrheic athletes experience osteoporosis; their rates of bone loss approximate the accelerated rates of bone loss in menopause.[18–21] The losses can be stopped and reversed by exogenous hormone administration or by the resumption of normal cycles.[22] New data, however, suggest that the losses may not be completely reversible and that amenorrheic athletes never recover normal bone density. This makes eminent sense when one remembers that these women are losing bone at a profound rate at a time when they should in fact be building bone, a form of double jeopardy. Osteoporosis is also exacerbated by low dietary levels of calcium. In weight loss or in calorie-restricted diets, the kind of diet often undertaken by young women athletes, dairy products with their generally high fat levels are often left out. Because the substrate needed for bone mineralization is limited, another level of risk is imposed on these women. Therefore, in dieting or training when dairy intake is limited, calcium supplementation is imperative. Poor protein intake also reduces bone mineralization. Smoking, alcohol consumption, and thyroid disease add to the risk of osteoporosis.

Gravity and weight-bearing exercise affect bone density positively. Working against gravity is the best means of promoting bone mineralization. Standing, walking, and running in the presence of normal estrogen levels all lead to heavier, denser bones. However, weight-bearing exercise cannot correct for a lack of estrogen. Women runners who are cyclic have denser bones than do sedentary women, and postmenopausal runners have 40% more bone than do controls.[23] Amenorrheic runners have less bone mass than do cyclic runners, and though bone mass is greater in long-distance runners than in sedentary women, it is still well below the levels of cyclic runners. Swimming and other non–weight-bearing exercises do not increase bone density except in highly trained persons and do nothing to counter hypoestrogenemic osteoporosis. Swimming can be regarded as a safe alternative form of aerobic activity for the osteoporotic or injured athlete.

A long career in commercial diving may damage the microcirculation of bone, with osteonecrosis now a well-recognized outcome. No evidence exists that osteonecrosis occurs in sport diving, but female commercial divers are likely to incur the same risk for dysbaric osteonecrosis as their male counterparts. When this osteonecrosis is superimposed on the risk for osteoporosis, women divers may be at risk for more profound disability than their male counterparts.

Exercise-Induced Amenorrhea

Several predisposing factors have been proposed and confirmed for exercise-induced amenorrhea. The most important measurable cause of amenorrhea in athletes is weight loss coupled with loss of body fat. As more weight loss occurs in training, the incidence of amenorrhea increases. Amenorrhea correlates with low body weight at the start of training, actual weight loss, and percentage of body fat lost. The age of onset of training also correlates with incidence of athletic amenorrhea. Women who start training prior to the initiation of regular menstrual cycles are more likely to be amenorrheic than women who start training after their cycles are established. In a similar mode, women who have had a previous pregnancy, evidencing maturity and stability of the reproductive axis, are less likely to become amenorrheic when they undertake strenuous training.

If the hypothalamic axis is inherently unstable, a rather modest level of exercise can cause disruption of cycles. Some women with highly erratic cycles may become amenorrheic by running only 9 miles per week. This translates into a 3-mile run three times per week, a level of aerobic activity equal to the lower end of the recommended scale for cardiovascular conditioning. Stated another way, some women possess an inherently fragile reproductive balance and may suffer significant reproductive impairment at very low levels of training.

Both Prior and associates[10] and Shangold and Levine[24] report that even women with stable reproductive function experience menstrual irregularity if training is intense. The degree of irregularity and the incidence of dysfunction increase directly with intensity of training. Shangold and Levine found that as college runners increase their mileage, menstrual irregularity increases. Prior and coworkers[10] demonstrated similar

events in runners, swimmers, and cyclists, although the latter two groups showed less severe aberrations at equivalent levels of energy expenditure. Interval, sprint, and other forms of speed work tend to disturb cycles more than steady aerobic demand does. Periods of intensive training, during which the person is trying to increase levels of performance and fitness, lead to more abnormalities than does maintenance of a stable level of activity.

Several other poorly defined factors in athletic amenorrhea have been proposed. The stress of training is not easily quantified or studied. Amenorrheic runners often feel that training is more stressful than do eumenorrheic runners. This may reflect an inherent difference in the personality of these women. Athletes who perceive training as pleasurable or satisfying may feel less stressed by the high demand of their endeavor. Perhaps more competitive persons find training more stressful. The differences in perceived level of stress may reflect differences in inherent athletic ability. Persons with natural talent and ability may not have to work as hard to reach a level of performance, and therefore they experience training as less physiologically demanding and less psychologically stressful.

Although not clearly elucidated, menstrual dysfunction in female athletes results from the complex interaction of neurotransmitters, hormone-releasing factors, and peripheral sex steroids. Several acute hormonal changes occur in response to exercise, but they are generally mild and short-lived. These include a decline in LH and increases in prolactin, estradiol, and progesterone. The level of FSH is unaltered. Intensive exercise ultimately results in diminished LH pulse amplitude and pulse frequency, chronically lowered estradiol levels, and failure of ovulation with an absence of progesterone secretion. Researchers in this area surmise that chronic, daily changes in hormones may lead to cumulative effects on the endocrine system, especially if intense workouts are long and closely spaced. The overall effect of training may be greater than is implied by the small acute changes measured after a single training session. Amenorrheic runners also have been found to have increased levels of endorphins and enkephalins during exercise.[25] Research has implied that increased levels of these compounds lower FSH and LH levels, and the lowering effect can be blocked with the narcotic antagonist naloxone.

Fertility and Exercise

Athletes with exercise-induced amenorrhea are anovulatory and therefore infertile. Because estradiol levels are low, this type of infertility does not respond to clomiphene citrate, the agent most commonly used to induce ovulation. Clomiphene acts as an antiestrogen, inducing the hypothalamus to secrete increased levels of LH to drive the ovary to produce more peripheral sex steroids. The desired response normally induces follicle growth and development. Therefore, in the hypoestrogenemic female with impaired LH metabolism, clomiphene offers no therapeutic effect. The use of more dramatic, complex, and expensive ovulation induction agents, such as gonadotropin-releasing hormone, FSH and LH extracts, and human chorionic gonadotropin, poses a difficult medical and ethical dilemma. If an individual is so active and hypoestrogenemic that her body cannot support ovulation, how well will she support a pregnancy? The safest therapy for exercise-induced infertility is a decrease in level of activity, increased calorie consumption, and weight gain. Ovulation may take some time to recur because a higher percentage of body fat is needed to reestablish cycles than to initiate them.

Pregnancy and Exercise

Gestation imposes increased physiologic and metabolic requirements, and the pregnant athlete superimposes the demands of training on the demands of pregnancy. Both pregnancy and exercise elicit hyperdynamic physiologic responses; therefore, the pregnant athlete has to be a superwoman to meet all these demands. When one is assessing the benefits and risks of exercise in pregnancy, the accomplishments and rewards of the gravida as athlete must be balanced with the needs of the developing fetus, who is a noncompetitor but not a nonparticipant. Sound training principles protect the athlete from injury, and even more stringent guidelines need to be observed during pregnancy to protect the fetus from inadvertent harm.[25,26]

Aside from the obvious gross changes in the uterus as it enlarges, major physiologic adaptations occur in the respiratory and cardiovascular systems to support the needs of the fetus. Although the diaphragm elevates owing to compression by the expanding uterus, total lung capacity remains unaltered because the chest wall splays laterally and the anteroposterior diameter of the chest increases. Inspiratory capacity and tidal volume increase, while there is an attendant decrease in functional residual capacity, residual volume, and end-expiratory reserve volume. Although respiratory rate remains unchanged, tidal volume increases and thus minute volume increases, resulting in an increase in oxygen uptake and maximum ventilatory capacity.[27]

Cardiovascular alterations, mediated by hormonal changes, occur during the first trimester. These changes are dramatic even though the uterus remains rather small and the actual hemodynamic demands are low. Heart rate increases by 10% to 15% coincident with a 20% to 40% rise in stroke volume, resulting in a 30% to 50% increase in cardiac output. Blood volume expands by 20% to 100% (mean 50%), causing a 33% increase in red cell mass and a 50% increase in plasma volume. Therefore, hematocrit falls to 33% to 36% (the so-called *dilutional anemia of pregnancy*), but overall oxygen-carrying capacity improves greatly. Coordinated with increased levels of erythrocyte 2,3,-diphosphoglycerate causing an increase in oxygen affinity in red cells and shifting the oxyhemoglobin dissociation curve to the left, oxygen delivery at the tissue level also improves. Mediated by progesterone and its relaxing effects on smooth muscle, peripheral vascular resistance falls, lowering systolic and, even more so, diastolic blood pressure. Thus, pronounced venous pooling occurs in the lower extremities and uterus.[28]

Also of importance in discussing exercise in pregnancy are musculoskeletal alterations, most notably the increased joint laxity and ligament loosening, again mediated by progesterone. Although promoting relaxation of the pelvic floor joints and ligaments to facilitate delivery of the fetal head, these changes destabilize joints in the extremities and predispose the pregnant woman to joint injury, torsion accidents, and dislocations. The growing uterus imposes a passive increase in weight bearing, increasing the lordotic curve of the spine, increasing stress and compression on lower back vertebrae and nerve roots, and predisposing to sacroiliac syndrome, back strain, and sciatica. The hips, knees, and ankles are also at increased risk for stress injury. As pregnancy progresses and the uterus comes out of the pelvis, becoming a true abdominal organ, the center of gravity of the gravida shifts forward. During the late second and entire third trimesters, this center changes almost constantly, producing gait and balance instability and increasing the woman's vulnerability to injury.

During pregnancy, the acute and chronic responses to the demands of exercise are altered. Exercising while pregnant is like exercising with weights on.[29–32] Aerobic capacity can be maintained through all trimesters if exercise training and intensity are maintained, though a slight degradation in V_{O_2}max occurs, seemingly attributable to the increase in body mass. If a pregnant woman maintains a fixed level of activity, her fitness actually increases as the pregnancy progresses because she is performing more work as the passive loading increases with fetal and uterine growth. If the activity declines slightly, the fitness level is actually maintained. Untrained women may undertake a slow-paced progressive training program and develop cardiovascular fitness while pregnant. In a group of untrained women, training during pregnancy increased fitness by 15% and increased maximal oxygen consumption capacity by 33%, whereas sedentary controls lost 10% of maximal aerobic capacity.[33] Maternal oxygen consumption also increases throughout gestation. Seventy-five percent of increased oxygen consumption results from increased weight bearing, whereas 25% is attributed to increased metabolic load.[34,35] In spite of all the positive study findings, many female athletes stop exercising during pregnancy, overtaxed by morning sickness, fatigue, musculoskeletal stress, and other discomforts.

Animal studies demonstrate that uterine blood flow decreases with exercise and that the decrease correlates with intensity and duration of activity. In sheep exercised at 70% of maximal output for 40 minutes, uterine blood flow decreased 40%. Placental blood flow also decreased but less dramatically, and actual fetal oxygen delivery fell only 11%.[36] Because the fetal oxygen delivery system holds a 50% reserve, this decline is

not significant in normal pregnancy. Exercise produces a number of other physiologic effects that, in theory (though not clearly in practice), may lower oxygen delivery to fetal tissues. Increased maternal oxygen consumption and muscle blood flow play only a small role. Increased catecholamines, particularly epinephrine, are known to decrease uterine blood flow, but sympathomimetics increase uterine blood flow. Overall, exercise increases sympathetic tone. Increased maternal temperature directly increases fetal temperature and metabolic rate. After exercise, fetal cooling lags behind maternal cooling, but this has not been demonstrated to produce adverse effects.

Animal studies of exercise and pregnancy have not demonstrated adverse fetal effects. In 1980, Clapp[37] exercised ewes to exhaustion, and although uterine and umbilical blood flow decreased, uterine and umbilical oxygen uptake was unaltered. Maternal lactic acidemia developed with no demonstrable uteroplacental or fetal lactic acid excess. Bagnall and colleagues[38] exercised rats throughout gestation and found decreased maternal weight gain but complete fetal sparing. Bell and colleagues[39] also studied ewes and measured seven metabolic parameters, including glucose, lactate, insulin, and glucagon, noting rapid changes of maternal and fetal levels of all metabolites measured but also rapid return to baseline level after activity ceased. They found that lambs of ewes that exercised regularly were larger, were fatter, and had more muscle glucagon than did lambs of controls. This single paper has been responsible for the notion that exercise promotes fetal well-being and enhances intrauterine growth. No other work has confirmed improved fetal growth with maternal exercise in any species. Mottola and colleagues[40] found that sedentary rats had, on the average, three more pups per litter than did exercised rats, and that pups in both groups had comparable birth weights. This suggests that some fetuses were resorbed to compensate for the increased demands of exercise, but those surviving were spared any ill effects.

Studies of fetal responses to maternal exercise in human subjects fail to demonstrate any short- or long-term damage to the fetus, nor do these studies show any benefit to infants of active mothers. Most human studies have been conducted at low, submaximal levels of activity, and the results cannot be extrapolated to committed or elite athletes. Most studies have been done at 65% to 80% of maximal oxygen consumption, and at worst a short-lived fetal bradycardia has been observed in a few subjects with no adverse fetal outcomes at term.[41–47] Carpenter and colleagues[48] are the only researchers to report on maximal levels of output. In third-trimester women, these authors recorded fetal hearts in 85 submaximal and 79 maximal exercise sessions. One instance of fetal bradycardia occurred in the submaximal group in a woman who experienced a vasovagal episode. In the maximal output group, 16 cases of bradycardia were observed, all occurring within 3 min of cessation of exertion, and all women recovered without incident. The flaw in the study is that the women went from maximal activity to rest, probably causing a profound drop in cardiac output, an increase in peripheral venous pooling, and, very likely, a rapid fall in uterine blood flow. Good training principles mandate a cool-down to avoid hypotension and decreased cerebral perfusion; the uterus probably deserves the same.

Exercise has no clearly documented effects on pregnancy outcomes, fetal or maternal.[49] No differences in birth weight, length, or Apgar scores can be confirmed. One study[50] demonstrates a higher 1-min Apgar score in offspring of fit mothers but no significant difference in 5-min Apgar scores. No study has been done on cord blood gases of "fit" infants.

No consistent trend can be identified on how exercise affects intrapartum events for the mother. Intermittently, studies report a shorter second stage of labor and decreased incidence of cesarean section in athletes, but maternal outcomes usually do not seem to differ in regard to induction of labor, augmentation of labor, anesthesia, midpelvic operative delivery, cesarean delivery, episiotomy, or duration of stages of labor.[45] In addition, maternal exercise does not affect lactation. The volume of milk, energy output of the mother, and weight gain of the infant are comparable in exercising mothers and in nonexercising controls.[45,50–54]

Studies on elite and endurance athletes, published by Clapp and others[55–57] in a collection of excellent papers, reveal interesting data on the fetal effects of intense maternal activity. The results, in general, are reassuring. In 1984, Clapp and Dickstein[55] reported that infants of ultramarathon runners had

birth weights that were reduced approximately 400 g, with no other significant problems identified. Between 1991 and 1992, Clapp reported continuing observations on a group of recreational runners and aerobic dancers. A subset of these women stopped exercising voluntarily after becoming pregnant.

Elite and dedicated amateur athletes often stop exercising because of morning sickness, fatigue, pain during exercise, and a host of other factors including increased perception of stress. Even a world-class athlete may have to stop exercising, as did Mary Decker Slaney through the greater part of her first pregnancy. Clapp's study, although not randomized, did have a sedentary control group well matched for age, height, weight, percentage of body fat, body mass index, preconception resting pulse as an index of fitness, and other measures. Birth weights in the exercise group averaged 407 g less than did weights in the now-sedentary group.[58] It should be noted that although the sedentary controls had curtailed their endurance training, they were probably still more active than the average American parturient. Labor outcomes of the two groups were also markedly different, with the "sedentary" group having statistically older gestational age at onset of labor, more frequent arrest of the second stage, higher incidence of artificial rupture of membranes, a longer active phase of labor, higher rates of forceps and cesarean delivery, more oxytocin use, and more fetal distress and meconium staining. Most of the differences in these outcome measures can be linked to the differences in gestational age and birth weights between the two groups, with longer gestational age and higher weights leading to longer, more difficult and complicated labor.

A second similarly constructed study published at the same time offered more detailed information on infant outcomes, particularly on neonatal morphometrics.[59] Although birth weight in the "exercise" group was 310 g lower, the length and the head circumference were unaffected. Thus, the decrease in weight was asymmetric, not affecting head size and, implicitly, not affecting brain development. Seventy percent of the difference in weight was attributable to a lower fat mass in the infants of the exercising mothers. Thinness in infants is not associated with increased perinatal mortality, and lower body fat does not lead to increased morbidity in the neonate. Lower fat mass, however, may contribute to the need for more frequent feedings, to the risk of cold stress, to lability of blood glucose, and to other subtle problems not easily detected by morbidity and mortality measures. Simply stated, exercise does not harm the fetus in utero but exercise confers no fetal benefits either.

Subjective observations of fit women have given the impression that they tolerate the physical demands of pregnancy well, have a better attitude in labor, experience less fatigue post partum, and recover sooner than their unfit counterparts. These triumphs may not be the result of exercise and conditioning but rather may reflect the intrinsic personality, positive outlook, and behavior of health-conscious women. In the past, our society has promoted the perception that the puerperium is a time when women are weak and ailing. Less than 30 years ago, women were kept at bed rest for 2 to 4 weeks post partum. Women, families, and whole cultures treat pregnancy as an illness. Active, athletic women clearly reject these antiquated views.

When a woman's pregnancy is a high-risk one, exercise is generally contraindicated, especially in disorders predisposing to impaired uteroplacental exchange or prematurity. Doppler flow studies in high-risk diseases such as pregnancy-induced hypertension and diabetes suggest that blood flow to the fetus does diminish significantly during exercise.[60] Weight-bearing exercises also appear to stimulate more uterine activity than non–weight-bearing exercises and should be limited or proscribed if a patient is at risk for preterm labor.[61]

Fetal Physiology and Gas Exchange

Uterine blood flow approximates 0.5 L/min, or 10%, of cardiac output of the mother, which is equal to cerebral blood flow levels. Eighty percent of flow distributes to the placental bed and 20% perfuses the uterine musculature. Within the closed fetal circulation, umbilical blood flow reaches 270 mL/min, 50% of the total fetal cardiac output. Twenty percent of the umbilical flow is shunted and does not participate in fetal gas exchange. Gas exchange occurs in the intravillous space and is characterized by a multivillous streaming system, maximizing

the surface area for gas exchange. Gas exchange between the two circulations is passive and is limited by diffusion. The rate of exchange is affected by a long list of hematologic and hemodynamic factors including intervillous blood flow, placental blood flow, oxygen tension in the maternal arterial blood, oxygen tension in fetal blood, oxygen affinity of maternal and fetal bloods, hemoglobin concentration in each system, oxygen-carrying capacity of each circulation, placental diffusion capacity, placental vascular geometry, the ratio of maternal/fetal blood flow in the exchange areas, shunting, and placental and uterine oxygen consumption.

The fetal environment is severely hypoxic, and Bancroft[62] has called the fetus "a maskless mountaineer atop of Everest in utero." The tissue defenses of the fetus mimic adult hypoxic protections, although the fetal responses are quantitatively different. Fetal erythropoiesis is higher, maintained by a higher, chronic level of erythropoietin. High hematocrit, however, is limited by the flow dynamics of Poiseuille's law, with a dramatic increase in viscosity over 80%. Fetal hematocrit averages 60%. Maximum blood flow of the most oxygenated blood is directed to critical tissues—the brain, coronary arteries, and adrenals. The oxygen dissociation curve is shifted markedly to the left. This is achieved by an altered molecular configuration of the fetal hemoglobin molecule. The beta chains are replaced by gamma chains, and the attendant binding of oxygen is tighter, with higher oxygen saturations at lower oxygen tensions. This altered binding does not depend on 2,3,-diphosphoglycerate as does in the adult. But just as occurs in adult hemoglobin, increased temperature, increased carbon dioxide, and increased hydrogen ion concentration all cause a shift of the curve to the right and decreased oxygen affinity.

Gas transfer takes place in the intervillous space. The gradient of oxygen tensions is exactly the same as the gradient in the alveolar capillary bed—10 mm Hg. The transit time for the red cell in the capillary also equals the transit time in the lungs, which is about 1 sec. Fifteen percent to 20% of uterine blood flow is shunted through the myometrium, and 15% to 20% of umbilical flow is shunted through fetal and placental channels, with a resultant 30% of flow uninvolved in gas exchange. Little reduction in the shunt is physiologically possible, and there are limits on increased rates of exchange. Another limiting factor in exchange comes from the fact that placental oxygen consumption is higher than that of the lung, with lung metabolic need being almost negligible.

In the placenta, the maternal/fetal blood flow ratio ranges from 0.4 to 2 with a mean of 0.8, close to the predicted optimum of 1. Areas of high maternal/fetal blood flow ratio are equivalent to areas of high ventilation-perfusion in the lungs. This wasted maternal circulation or over-arterialization of flow offers the fetal-maternal system a considerable reserve. Increases in the maternal or fetal flow rates do little to increase oxygen transfer. The system is functioning at an ideal, maximal level at most times. If it is stressed or compromised, gas exchange does not improve but rather is maintained because the resting state holds a 50% physiologic reserve. Increases in inspired maternal oxygen do little to alter fetal gas delivery, even at maternal arterial oxygen tensions of 500 to 600 mm Hg. However, increases can be achieved using hyperbaric states.

The medical community in the former Soviet Union claims extensive experience treating both maternal and fetal hypoxic conditions with hyperbaric therapy. They also use hyperbaric oxygen (HBO) to manage diseases presumed to be due to poor maternal-fetal exchange. The literature cites HBO treatment of cyanotic heart disease, pulmonary hypertension, anemic disorders, intrauterine growth retardation, preeclampsia, diabetes, and even habitual abortion. The statistics are poor, with almost no analysis beyond reporting of percentages of improved outcome. Treatment schedules do not approximate any of our common treatment tables. HBO is administered at 1.5 to 2 ata, often for extended periods of time, with patients laboring in chambers and with delivery and even cesarean section being performed in a pressured environment.

In acute hypoxic situations, the fetus responds by differential redistribution of flow to three classes of circulatory beds: nonnegotiable, negotiable, and expendable. Blood flow is centralized to critical tissues, paralleling the diving reflex of marine mammals. Liggins and colleagues,[62] working with the Weddell seal, found that the fetus demonstrates a diving reflex during maternal dives, with centralization of flow and bradycardia in both the carrier and the carried.

The human fetus "dives" during hypoxia, with a reflex mediated afferently by the trigeminal-vagal nerve plexus. Hypoxia induces bradycardia and peripheral vasoconstriction. Note also that fetal adrenal secretion is dominated by norepinephrine, the dominant adrenergic mediator in diving mammals. Smith and Nelson[63] summarize the physiology in this paraphrase of the classic tenet of genetics: "The physiologic ontogeny of the human fetus may very usefully recapitulate the phylogeny of the diving reflex."

In considering the possible effects of diving, pressure, and hyperbaric states on the fetus, one needs to know the effects that changes in concentration of inspired gases have on the fetal-maternal unit. Moderate levels of hypoxia, hypercapnia, and hypocapnia do little to alter placental gas exchange. Marked changes in inspired gases, however, cause decreases in fetal perfusion. Research from the anesthesia literature reveals that when inspired oxygen drops to 6%, maternal cardiac output increases, systemic vascular resistance decreases, and uteroplacental vascular resistance increases, with concomitant decreases in uterine blood flow. The same changes occur at 12% oxygen concentration, but less dramatically. When P_{CO_2} increases to 60 mm Hg in maternal circulation, uterine blood flow increases. With P_{CO_2} over 60 mm Hg, vascular resistance increases and uterine blood flow declines. Diminished levels of carbon dioxide probably cause no great changes, but the anesthesia literature cites uteroplacental vasoconstriction with an attendant decline in uterine blood flow, fetal hypoxia, acidosis, and neonatal depression when P_{CO_2} falls to 17 mm Hg. Fall of P_{CO_2} in the study reviewed was achieved by mechanical hyperventilation and is thought to be due to an artifact of mechanical positive pressure ventilation.[64]

WOMEN AND DIVING

The diving literature has paid little attention to the special anatomic, physiologic, and psychologic differences between men and women. To extract relevant information is difficult because reports are scant in number and substance. Data can be derived from sources in diving and hyperbaric medicine, anesthesiology, and aerospace science.[65–67]

Because of their smaller stature and smaller muscle mass, women possess different strength and energy potential during exercise. Thermal stress is one of the major energy burdens in diving. Anatomically and physiologically, women respond to cold in subtly different ways. The surface area/volume ratio is slightly higher in women, increasing the area of conductive heat loss. It is agreed, however, that the differential is small in practical terms. More important, women possess much less muscle mass, with less metabolically active tissue to generate heat during activity. Although women carry more subcutaneous fat than do men, the relative insulation value is poor. Women demonstrate a greater ability to vasoconstrict limb blood flow, thereby conserving heat, but again the advantage is unclear. Of interest is work by Hong, who studied Korean Ama divers (see Chapter 5). He quantitatively demonstrated that men and women divers lost the same amount of heat when working in cold water for 60 min. Men worked in 27° C water and cooled to a core temperature of 36.4°C. Women worked in 22.5°C water and cooled to 35°C. The physiologic mechanisms in response to cold stress appeared to differ between the two groups. Men seemed to produce and lose much more metabolic heat, whereas women did not appear to "compensate" for rapid thermal losses. The women, in fact, voluntarily tolerated a state of prolonged hypothermia. Skin-fold thickness is presented as an explanation of the differences. Although most measures and parameters put women at a disadvantage in tolerance to cold exposure, practice offers contradictory evidence. Long-distance open-water swim records are often held by women such as Diana Nyad. Even if women are more vulnerable to hypothermia, thermal stress should not pose a sex-specific hazard for women divers when properly equipped.

Women possess a lower aerobic capacity than do men, with significantly less upper body strength. During sport diving (generally not a severe aerobic endeavor), these differences should hold little influence. Most experienced divers insist that less work and exertion helps to conserve air and extend bottom time. In commercial diving and when swimming in strong currents, women performing the same workload as men are likely to become exhausted sooner, especially when cold stress and hyperventilation are superimposed. Male divers and instructors need to be aware that they must avoid

pushing female divers into situations demanding overexertion.

Women and Decompression Sickness

Early reports from hypo- and hyperbaric medical literature suggested that women are at increased risk of DCS. In 1973, Bassett[68] cited seven cases of aerospace DCS in 3190 exposures in female nurse flight trainees versus two cases in 9056 exposures in male pilots. A second study in 1980[69] reaffirmed an apparent 3.6-fold increased risk of aerospace DCS in women. These data have been repeatedly criticized because the level of technical training and physical fitness in the male pilots far exceeded the preparation of the female nurses in the program.

The impression of exaggerated risk was reinforced when Bangasser,[70] in 1978, reported on data derived from a retrospective questionnaire sent to divers. Six hundred forty-nine women divers with varied levels of training and experience logged 88,028 dives, with 29 reported cases of DCS, a rate equal to 0.033% per dive. When different levels of experience were used in calculating rates, the incidence in basic divers rose to 0.043% and in instructors fell to 0.023%. When the rate of DCS in women instructors is compared with the rate in male instructors (0.007%), a three- to fourfold increased risk appears to exist for women, the same order of magnitude as reported by Bassett. The similarity of the risk ratio in these three studies gave increased weight and credence to the idea of women being at increased risk for DCS.

Weien and Baumgartner (1990) compiled data from 528 altitude chamber and aircraft DCS cases. After excluding wet diving and air diving cases, 429 cases remained. The incidence of DCS for women was 206 per 100,000; the rate for men was 48 per 100,000. Thus, women incurred a relative risk of 4.3 ($P < .001$).[71] No adequate explanation of the differences has been forthcoming. Sex differences in body fat, lean body mass, fluid retention, peripheral vasoconstriction, limb perfusion, hormonal influences, platelet aggregation, and complement levels have not consistently correlated with rates of DCS in men or women. Some workers in the field have postulated that women may tend to report symptoms more often than do men. The prospective study by Webb and associates[72] demonstrated no difference between men and women exposed to altitude, although male subjects had more venous gas emboli. These authors discuss possible causes for the findings of earlier altitude studies[68,69] that described a higher female incidence of DCS.

Most of the field studies on divers under observation and with protocols for testing and reporting do not confirm an increased incidence of DCS in women. During the Tektite saturation diving project, Beckman and colleagues[73] collected data on DCS from a dive team consisting of 5 women and 50 men. One woman and five men experienced symptomatic DCS, rates of 20% and 10%, respectively. The numbers of cases of DCS are too small to infer statistical significance. During Project Hydrolab, a similar saturation project, Miller and Koblick[74] reported no cases of DCS in the 58 women and 285 men studied. An aerospace study of DCS during simulated space missions, authored by Waligora and colleagues,[75] found that the rates of reported pain and detectable Doppler bubbles in male and female subjects were similar. Of the 14 women, 9% reported pain and 18% were Doppler-positive for venous bubbles. Of the 15 men, 6% had pain and 23% had detectable bubbles. Zwingleberg and colleagues[76] published data on Navy divers performing air dives to 120 to 285 fsw and heliox dives to 120 to 300 fsw. For the 988 dives by women, the incidence of DCS was 0%; the rate for men was 1.3%. During 60 dives in which men and women were paired, two men and no women suffered from the bends. Fife and colleagues[77] reported on archeologic research divers performing approximately 10,000 dives from 140 to 190 feet. Women accounted for 33% of the divers, and even though they performed 50% of the deepest dives, the rates of DCS for men and women were comparable, 0.04% and 0.03%, respectively. Countering the common perception and wisdom, a 1995 report from the United Kingdom, surveying 2250 divers, 46% of whom were women, estimated the rate of DCS in men to be 2.6 times higher than that for women with the same level of diving experience.[78]

Robertson,[79] in a letter to the Undersea and Hyperbaric Medicine Society, reported on 111 cases of DCS from the Australian Navy. Though the cases showed no correlation

with age, dive experience, dive profile, and other measures, the rate of type II DCS for women was 4.3 times higher than the rate for men (confidence interval, 1.2 to 15.8). This report admonishes us that the data on DCS in women are far from conclusive.

Most of the data obtained from dry-dive studies also indicate that women are not at increased risk for DCS. Eckenhoff and Olstad[80] studied a diverse group of people during chamber dives with Doppler bubble detection at precordial and subclavian sites. The authors found no differences in the rates of bubble formation across weight, height, and age variations. A trend toward greater bubble formation was seen, with longer duration of exposure and with increasing age. Dunford and Hampson[81] published a report in 1992 that found no increased risk for women during chamber dives. Between 1976 and 1990, approximately 7910 hyperbaric treatments took place in the chamber at Virginia Mason Medical Center, with 8424 inside attendant exposures during these treatments. Twenty-six instances of DCS occurred that required treatment, a rate of 0.31%. The men/women exposure ratio was 0.38:0.62, whereas the men/women DCS ratio was 0.31:0.69, a difference that is not statistically significant. A similar study by Dietz and Myers from the University of Maryland[82] records 25,164 exposures in 439 tenders, with a DCS rate equaling 0.78% in men and 0.76% in women.

A troubling finding from the analysis of Dunford and Hampson was that when the data were analyzed in relation to the timing of menstruation, a cluster emerged such that the relative risk for chamber dive–related DCS appeared to be increased 7.6-fold during the menstrual phase of the cycle. Dunford also reported on DCS in a survey of open-water divers[83] and did not find a clustering of DCS related to the menstrual cycle. But two other researchers from the aerospace field have reported increased rates of DCS during the menstrual phase. In 81 cases of DCS in women trainees, Rudge[84] reported that 62 had pain only and 18 had neurologic symptoms and that the later the women were in their menstrual cycle, the fewer the instances of DCS. In a study by Dixon and colleagues, assessing the hypobaric exposure profiles of 30 female astronaut candidates,[85] all cases of aerospace DCS occurred in women who were menstruating. None of the researchers in the field has proposed a hypothesis that might satisfactorily explain the menstrual clustering in aerospace and chamber-related DCS, nor has anyone offered an explanation as to why or how menstruation might enhance this risk.

The conventionally recognized risk factors for DCS, such as age, increased body fat, and poor conditioning, do not provide any insights into the clustering of events in the menstrual phase of the female reproductive cycle. Each phase of the normal menstrual cycle is characterized by pronounced differences in sex steroid levels and ratios and other physiologic functions. High levels of estrogen are prothrombotic. High levels of progestins may exert some anti-inflammatory effects. However, the menstrual phase of the cycle is characterized by low levels of both of these hormones. In animal models and in human testing, estrogen potentiates nitric oxide–mediated vasodilation. Thus, the propensity for DCS during menses might be due to impaired vasoregulatory mechanisms. Peripheral blood flow is altered during menses. Terregino and Seibold[86] used plethysmography to study digital arterial flow in men, women, and women with Raynaud phenomenon. They found that digital flow ceases at 13.7°C in men, at 18.1°C in women, and at 26°C in women with Raynaud phenomenon. At the time of menstruation, however, all the normal women experienced responses to cold exposure that were similar to the responses of women with Raynaud disease and were least "cold tolerant" at this phase of the cycle, although none evidenced the blanching changes that occur in true Raynaud phenomenon.

Scientific study contradicts the notion that increased body fat predisposes women to high rates of DCS.[87] When active and sedentary persons are compared, differences in the rate of DCS seemingly relate to levels of fitness, not fatness. In addition, fewer Doppler-detectable bubbles were present in fit persons, suggesting that increased vascularity may aid the elimination of dissolved gas. In 1995, Broome and colleagues[88] demonstrated that pigs who were preconditioned with exercise training prior to hyperbaric exposure suffered fewer and less severe instances of DCS, thereby providing additional support for the idea that fitness exerts a protective effect in DCS. If fitness explains the apparent differences in rates of DCS, all divers—men and women—should strive to maintain their aerobic conditioning

and level of fitness to lessen the potential risk of DCS. A recent report suggests that "moderate, intermittent arm or leg exercise during decompression may accelerate inert gas evacuation and reduces the incidence of intravascular bubbles after diving."[89] Post-dive exercise, however, potentiates bubble formation and risk.

Most recently, St. Leger and coworkers[90] published results of a retrospective survey of recreational divers in the United Kingdom. The questionnaire included information on general health, smoking, alcohol, drug use, diving history, and profiles from all divers; physician- and self-diagnosed episodes of DCS were collected from female subjects only. Of the 2250 respondents, 47% were female. A total of 458,827 dives were logged, 31% of which were performed by women. The decompression illness part of the study included confirmed and unconfirmed cases. Six percent of subjects reported 159 events: 86 men and 49 women. Thirty-seven physician-confirmed cases of DCS occurred in women and 50 cases in men. Some interesting gender differences were found in the basic diver demographics and in diving injury patterns. Overall, looking at both confirmed and self-diagnosed DCS, highly experienced divers, those with more than 26 years of experience had low DCS rates. This was true and apparent for both males and females, with an event rate of less than 0.01 per 1000 dives.

In assessing the whole data set, women appeared to participate in less aggressive diving activities according to the safety stops reported in their dive profiles. When the physician-confirmed cases of DCS were assessed, the rate for women was 0.262 per 1000 dives and the rate for men was 0.157 per 1000 dives. This suggests a 1.67 relative risk for women. Further refinement of the data, limiting the analysis to a subset of subjects with confirmed dive profiles, found a relative risk in women of 2.46. The authors then corrected the data for the type of diving activity, the depth, the duration, and other "risk" markers. When a correction factor for the dive characteristics was applied, the data produced an increased risk for men. Overall, men appeared to have a relative risk of DCS 2.57 greater than women, assuming the "correction" formula used for this calculation is statistically valid.

Other interesting findings included no correlation of accident rate with fatness as assessed by the body mass index of respondents. Interestingly, the women had a lower incidence of obesity than the men in the study population, perhaps suggesting a higher degree of fitness as well, which might offer some explanation of the lower DCS risk. Twenty-two percent of DCS accidents in women occurred while they were menstruating. Seventy-eight percent of events occurred during other phases of the cycle, or the menstrual status at the time of injury was unknown. Again, estimating 7 to 8 days at most for the menstrual phase, one would expect 25% of cases to occur if the distribution was uniform throughout the monthly cycle. The outcomes here do not support the hypothesis that DCS clusters in the menstrual phase of the cycle. The authors admit that there may be some inaccuracy in these data.

There appeared to be no increased rate of DCS in women using oral contraceptives. Women appear to have a higher rate of multisystem DCS: 72.4% for women versus 54.8% for men. This is consistent with other reports of type II DCS being more common in women. Women were more likely to be treated with oxygen at the scene, suggesting that women are more likely to report symptoms or that symptoms are more severe. The authors acknowledge that the data are not robust because of the retrospective nature of the study and the potential for biases to be affected by respondent biases. The authors also state that the DCS numbers are estimates, not true rates. Nonetheless, these data represent one of the most sophisticated and objective studies delineating differences between male and female divers.

In summary:

- Women are probably not at any substantial increased risk for DCS when diving within recreational limits.
- Although women may be at increased risk for altitude DCS, newer studies show no sex difference in DCS.
- At altitude, women tend to exhibit a lower incidence of venous gas emboli, with a similar rate of DCS.
- Altitude DCS events appear to cluster in the menstrual phase, the first week of the cycle.
- Coagulation and hormonal factors may be implicated in the enhanced risk during the menstrual phase of the cycle but have not been elucidated.
- The data on DCS in "wet" diving do not suggest an increased risk during the menstrual phase of the cycle.

- Both no-stop diving DCS and altitude DCS are rare (0.1% to 0.5%)
- When afflicted with a DCS injury, women may incur a higher rate of type II DCS.
- All women presenting for hyperbaric recompression therapy should have detailed menstrual and contraceptive histories, including last menstrual period.
- Last menstrual period and contraceptive use should be routine parts of diving log data for all professional women divers.
- All female chamber attendants should accurately chart their menstrual cycle. If women sense that they are less than par during their menstrual cycles, they would do well to limit their excursions in terms of time, depth, or both to provide safer margins for their dives.

Other Diving Accidents and Injuries

Between 1987 and 2002, the Divers Alert Network Diving Accident Reports[91] have tabulated diving accidents in the sport-diving community. The accident rate for women ranged from a low of 21.6% in 1986 to a high of 30% in 2000. Women are presently estimated to compose 33% to 35% of the diving population. Women generally are represented proportionally to their participation in the sport in the accident numbers but typically show up as a much smaller percentage of diving deaths. For example, in the most recently reported year, 2000, 12 female deaths and 76 male deaths were listed, women accruing 13.6% of the deaths. Generally, women appear to be underrepresented and under-enumerated in the mortality body count. The disparity is probably due to difference in diving activities. Women hold fewer advanced diving certifications and seem less likely to engage in "high-risk" diving activities such as caving and technical diving.

Reports do not suggest an increased incidence of air embolism in women. A patent foramen ovale is thought to contribute to the risk of embolism, but there is no sex differential in the incidence of this cardiac anomaly.[92] All divers should remember that the risk of air embolism is greatest during compressed-air diving at shallow depths. Other risks of compressed-air diving include oxygen toxicity and nitrogen narcosis. Oxygen-related seizures are reported in the literature to occur in as few as 1 in 10,000 treatments (0.0001%) or in as many as 5%. Hampson and colleagues[93] recorded the incidence of oxygen-related seizures during protocols for carbon monoxide poisoning. The incidence of seizures in patients treated at 3.0 ata, 2.8 ata, and 2.5 ata was 2.5%, 1.9%, and 0.6%, respectively. The rates did not vary with age, sex, level of carboxyhemoglobin, or level of consciousness.

Frequently Asked Questions About Women and Diving

Since the 1980s, the Divers Alert Network[94] has become a prime source for data collection in the sport-diving community. In addition to triage and management of diving accident victims, the Divers Alert Network provides medical information and advice regarding the most commonly encountered dilemmas in diving medicine. In 1981, call volume was a trifling 180, but by 1995, the advice line logged 14,642 calls.[91] Volume has declined somewhat since then, with 10,046 calls in 2001. Queries are tabulated and tallied, and a set of online and fax-accessible answers are generated. The most commonly asked questions about women and diving are listed in Table 19–1.

Diving and Menstruation

There is no evidence of increased shark attacks on menstruating female divers. Hygiene in remote locales and on dive boats may be troublesome and inconvenient if

Table 19–1. Topics of commonly asked questions regarding women and diving

Breast Cancer, cancer, and surgery
Breast implants
Breastfeeding
Endometriosis
Hysterectomy
Menstruation during diving activities
Oral birth control
Osteoporosis
Ovarian cancer
Pregnancy
Premenstrual syndrome
Return to diving after giving birth

Data from http://www.diversalertnetwork.org/

there are no sanitary facilities or privacy. For women with severe menorrhagia, episodic heavy blood loss may limit aerobic exercise capacity. Losses as high as 200 to 300 mL per cycle are not uncommon. St. Leger and colleagues[95] reported on the diving behavior and performance of women during menstruation, collecting data both retrospectively and prospectively. In the retrospective cohort of 1050 respondents, 93% of women continued to dive while menstruating, engaging in an average of 37 dives per year. Seventy-one percent admitted to having some "premenstrual tension," and 34% admitted to subjective decrement in performance due to menstruation. Eighty-one percent of those in the prospective cohort of 420 women reported premenstrual tension, and 40% felt that their ability to perform was impaired. Feelings of panic, anxiety, loss of control, dizziness, and cold were exaggerated while menstruating.

Some authors worry about the effect of menstruation on diving, but others have asked whether hyperbaric exposure alters menstrual cycles. No reports in the aerospace or hyperbaric chamber literature have suggested that exposure alters the cycle length or duration and amount of flow. One published work[96] involved only three subjects who were pressurized to 5 ata seven or eight times during each menstrual cycle. The authors detected no changes in cycle length, LH, FSH, estradiol, progesterone, testosterone, or ovulation.

PREMENSTRUAL SYNDROME AND PREMENSTRUAL DYSPHORIC DISORDER

Premenstrual syndrome (PMS) is an aggregate of physical, behavioral, and psychological symptoms that occur during the luteal phase of the menstrual cycle. Premenstrual symptoms of some kind occur in 80% of women, but the diagnosis of PMS requires one or more of the symptoms listed in Table 19–2 during the 5 days prior to menses in each of three prior menstrual cycles.

The diagnosis requires the exclusion of other underlying or intercurrent physical or psychiatric disorders and requires that symptoms be severe enough to disrupt social or work performance.[97]

Five percent of women suffer from a more severe form of the ailment, premenstrual dysphoric disorder (PMDD). Premenstrual dysphoric disorder is more pervasive and disruptive than PMS. The diagnosis requires at least five of the symptoms listed in Table 19–2 but also includes decreased interest in activities, difficulty concentrating, lack of energy, changes in appetite, change in sleep pattern, and feelings of being overwhelmed or out of control. PMDD produces more symptoms of greater intensity, leading to great social disruption.[98]

Women with severe PMS and PMDD need to be evaluated to rule out other underlying psychiatric disorders. Menstrual mood disorders can be confused with more pervasive depressive disorders such as dysthymia, atypical depression, and hypomania, a milder version of bipolar disease. By definition, premenstrual disorders must fluctuate with the menstrual cycle. Menstrual mood disorders can be distinguished from other psychiatric disorders by the following:

- Women must be symptom-free for some period of time each cycle.
- Symptoms always resolve shortly after the start of menstrual flow.
- The psychological manifestations are usually accompanied by physical manifestations.
- The symptoms resolve with pregnancy.
- Selective serotonin reuptake inhibitors (SSRIs) are highly effective.

The underlying pathology in both disorders is not hormonal; rather, normal fluctuations in the changing hormonal milieu throughout the female reproductive cycle interact with an underlying neurotransmitter defect.[99] SSRIs work remarkably well and are the current standard treatment. Most often,

Table 19–2. Symptoms required during the five days prior to menses to diagnose premenstrual syndrome*

Depression
Angry outbursts
Irritability
Anxiety
Confusion
Social withdrawal
Breast tenderness
Abdominal bloating
Headache
Swelling of extremities

*Symptoms should occur in each of three prior menstrual cycles.

they are given continuously, despite the seeming cyclicity of the symptoms. Studies have shown similar responses using "intermittent" dosing wherein SSRI use is confined to the luteal phase of the menstrual cycle.[100] No adverse effects of SSRI drugs have been reported in sport divers.

The diagnostic challenge in a fitness-to-dive evaluation of a female presenting with PMS or PMDD consists of separating women with mild to moderate menstrual-related problems from those with another underlying severe depression or sociopathy. Careful assessment of mood should include any of the common depression scales, such as Hamilton's or Beck's; symptoms should be charted through at least three menstrual cycles to exclude a more pervasive mood disorder. Any individual—male or female—who displays evidence of antisocial tendencies, suicidal ideation, destructive behavior, or other severe psychiatric disturbances should be disqualified from diving.

Diving and Endometriosis

A theoretical concern has been heard from an unknown quarter; the concern is that the high partial pressures like those encountered in HBO treatment or during diving might exacerbate the underlying process of endometriosis. No scientific literature supports this concept. On the contrary, scientists from the former Soviet Union have used HBO, with no supporting scientific rationale, to treat endometriosis. Baskakov and associates[101] reported on the treatment of 31 women with genital endometriosis and "nephroptosis." Surgical nephropexy with a muscular flap was carried out in all cases, followed by adjuvant treatment with hormones and HBO. All patients reportedly did well over 3 years of observation. The Russians used HBO for such conditions as intrauterine growth retardation, recurrent abortion, infertility, maternal cyanotic heart disease, maternal anemia, fetal cardiac disease, maternal valvular disease, and multiple gestations. Women with severe cyanosis or deoxygenation were treated for prolonged periods in HBO environments, living at pressures of 1.5 to 2.0 ata, often for the entire late third trimester; they remained at pressure while laboring or during caesarean delivery in the operating room in a multiplace, multichamber medical complex. The data of Baskakov and colleagues counter speculation about adverse consequences of HBO exposure in women with endometriosis, but the report is of limited scientific utility.[101]

Inasmuch as endometriosis increases bleeding, cramping, and the amount and duration of menstrual flow, this disorder may present a disadvantage for the female diver. A woman with severe, symptomatic, disabling endometriosis is at increased risk, not only from the underlying illness but also from various drugs used to treat the condition, especially sedatives or narcotics used for pain.

Diving and Contraception

ORAL CONTRACEPTIVE AGENTS

Twelve percent of women aged 14 to 44 years currently use oral contraceptive pills. Concerns regarding the safety of birth control pills first surfaced in 1969 when several British retrospective epidemiologic surveys[102–104] reported increased rates of deep venous thrombosis, pulmonary embolism, cerebrovascular accidents, and myocardial infarctions in pill users. A better understanding of the attributing risk and of the interactions of sex hormones with host cofactors has since emerged. During this interval, the estrogen dose in oral contraceptives declined from 85 to 150 μg to 20 to 50 μg and the progestin dose dropped from 5 mg to an average of 1 mg for the norethindrone-containing and norethindrone acetate–containing brands. While dosage was declining, prospective data were collected on pill performance and safety. These new data, coupled with improved multivariate analysis, now demonstrate that almost all of the increased arterial vascular risk attributed to the pill is linked to the synergistic interaction of the pill with tobacco smoking. Women who smoke fewer than 25 cigarettes a day experience a threefold increased risk of myocardial infarction, whereas heavy smokers (those using more than 25 cigarettes per day) have a 23-fold increased risk.[105] Oral contraceptives do not increase the risk of myocardial infarction in women under age 35 who do not smoke, and the pill is also not atherogenic.

It has been suggested that at least 50% of thromboembolic events that occur in women taking birth control pills may be due to

Table 19–3. Thrombotic events related to factor V Leiden and oral contraceptives

Group	Relative Risk	Annual Incidence (%)
Control	1	0.008
Oral contraceptives only	3.8	0.030
Factor V Leiden only	7.9	0.057
Factor V plus oral contraceptives	30	0.285

Adapted from Vandenbroucke JP, Koster T, Briet E, et al: Increased risk of venous thrombosis in oral-contraceptive users who are carriers of factor V Leiden mutation. Lancet 344:1453–1457, 1994.

interactions of the medication with heritable and acquired clotting disorders. The most common of the thrombophilias evolves from the substitution of a glutamine for an arginine in the structure of coagulation factor V, rendering it resistant to cleavage by activated protein C. The prothrombotic activity of factor V is thus upregulated. The resultant disorder, activated protein C resistance, is an autosomal dominant disorder, and it is estimated to occur in 5% to 10% of Northern European whites. Whereas the relative risk was increased sevenfold for heterozygous persons, it was increased 80-fold for homozygous subjects.[106]

There appears to be synergy between the thrombophilias and oral contraceptives. Thrombotic events are greatly increased by having the Leiden mutation and taking oral contraceptives,[107] as indicated in Table 19–3.

There is also evidence that two of the newer progestins in oral contraceptives, desogestrel and gestodene, may modify the coagulation cascade, increasing activated protein C resistance.[108] Similar findings have also been reported with postmenopausal hormone therapy.[109, 110] Clinical tests for activated protein C resistance are sensitive enough to identify the changes in coagulation factors in response to exogenous hormones. Other thrombophilias also interact with the prothrombotic effects of oral contraceptives. Other common entities include protein S deficiency, protein C deficiency, lupus anticoagulant, and anti–thrombin III deficiency.

The implications of these findings are unexpectedly important for diving. Underlying coagulation defects have been implicated as potential risk factors for DCS, osteonecrosis, and idiopathic aseptic necrosis of the femoral head and a host of other vascular complications. Thrombophilic disorders should be considered when encountering unexpected, undeserved DCS or DCS that seems more severe than predicted given the dive exposure history.[111] A natural and logical hypothesis is that oral contraceptives might increase the frequency and severity of DCS injuries by increasing thrombotic activity at the site of a gas induced occlusive lesion. Fife and Fife,[112] who subjected pigs to a bounce dive profile, showed no difference in the rates of DCS in controls versus animals remedicated with oral contraceptives. The study found that extent of injuries was identical in the treated and control pigs. However, pigs are not afflicted by thrombophilias as discussed earlier.

Other studies examining the relationship of oral contraceptive use to DCS have yielded mixed results. Schirmer and Workman[113] found that altitude-chamber trainees appeared to have no increased susceptibility to DCS in relation to phases of menstrual cycle or oral contraceptive use. In a survey of 1000 women divers, Fife and Fife[114] reported no increased incidence of DCS in the users of oral contraceptives. The Divers Alert Network published data on the relationship of menstruation to decompression illness. The database of DCS cases from 1989 to 1995 was culled for female diving accidents. Nine hundred fifty-six complete records were found and assessed for depth and time of dive, ascent rate, age, and diving experience: 38.2% of victims were menstruating at the time of DCS accident. The expected percentile assuming menstrual flow duration of up to 8 days would be 25%. For the 654 women of reproductive age (13 to 51 years), assuming cessation of menses at age 51, 21.6% were menstruating at the time of the accident, a percentage probably not significantly less than the expected rate based on assumed menstrual norms. For the

261 women in the cohort who were taking oral contraceptives, 85.5% were menstruating. This may suggest an increased rate of DCS associated with oral contraceptive use. The rate is even more alarming because for women on oral contraceptives, the duration of flow is attenuated, shortened to 3 to 4 days from the norm of 5 to 7.[115]

OTHER CONTRACEPTIVE METHODS

In recent years, three contraceptives have been introduced in the United States that offer unique new options for women. Ortho Evra is a combination transdermal contraceptive patch that contains 6.00 mg norelgestromin (17-deacetyl-norgestimate, the active metabolite of norgestimate, the progestin in the Ortho-cyclin and Ortho-TriCyclen birth control pills) and 0.75 mg ethinyl estradiol. The patch releases 150 μg of norelgestromin and 20 μg of ethinyl estradiol to the blood stream per 24 hours. At the time of this writing, acceptance of this new method has been dramatic. It is now the second most commonly prescribed contraceptive after birth control pills. It has been on the market for too short a time for any meaningful clinical experience to accumulate. From clinical trials, there appear to be no adhesion problems in sports activities, including swimming and hot tub use. "Experience in more than 70,000 Ortho Evra patches worn for contraception for 6 to 13 cycles showed that 4.7% of patches were replaced because they either fell off (1.8%) or were partly detached (2.9%). Similarly, in a small study of patch wear under conditions of physical exertion and variable temperature and humidity, less than 2% of patches were replaced for complete or partial detachment."[116] Diving effects on patch adherence have not been documented. Note that because this method provides the same amount of bioavailable ethinyl estradiol as 20 μg oral contraceptives, any cautions that apply to pills apply to the patch regarding coagulation and thrombotic risk.

Another nonoral combination method was approved by the Food and Drug Administration recently; the NuvaRing (etonogestrel/ethinyl estradiol) consists of a flexible, transparent, colorless vaginal ring about 2.1 inches in diameter containing the hormones etonogestrel and ethinyl estradiol. Like oral contraceptives and the contraceptive patch, NuvaRing provides a high degree of efficacy and carries the same risks as other contraceptives containing estrogen-progestin. The ring is left in the vagina for 21 days and then removed for 7 days. Women are advised not to remove the ring for more than 3 hours during the active "treatment" part of the cycle. Other side effects of NuvaRing may include vaginal discharge, vaginitis, and irritation. NuvaRing releases only 15 μg of ethinyl estradiol, half of the amount found in most currently marketed oral contraceptives. Again, the reported clinical experience of this method is insufficient in terms of divers or on the effect of prolonged immersion on the performance of the device.

Long-acting, injectable progestins have been available for more than 20 years but have only recently come into use in the United States. Injectable and implanted progestins are highly effective, uninfluenced by what contraceptive researchers call "user-related" failures. The long-acting progestational agent Norplant (subcutaneous norgestrel-containing Silastic implant) has fallen into disuse after a wave of litigation alleging that the device causes major health problems, such as autoimmune disease and systemic allergies, claims that are not supported by epidemiologic data. Injectable medroxyprogesterone acetate (Depo-Provera) and the newer depo-medroxyprogesterone acetate plus estradiol (Lunelle) are becoming increasingly popular, especially with younger women who desire a highly effective but non–coital-related method of contraception. None of the physiologic changes induced by depo-progestin–only methods suggests that they would interact adversely with hyperbaric physiologic changes. Depo contraceptives with estrogen, as in Lunelle, confer thromboembolic risks similar to those conferred by oral contraceptives. The most common side effect of depo-progestins is irregular and unpredictable bleeding, an inconvenience for the woman diver but 1clearly not a serious health risk. Ultimately, a high percentage of women using depo-progestins become amenorrheic, a side effect that many women might regard as an advantage, particularly for sports participation.

It can be speculated that progestational agents may offer the female diver protection from tissue damage if she does experience DCS. Studies published in the 1970s found

that the incidence and severity of sickle cell crisis were reduced in women using high-dose progestins. The mechanism of sickle cell crisis—trapping of erythrocytes, release of thromboplastins, and thrombus formation—is similar to the mechanism of thrombus formation in response to bubbles in DCS. Isaacs and colleagues[117] reported that 80% of women receiving progesterone in oil (10 mg weekly, taken intramuscularly) experienced a 75% reduction in the severity-duration scores for their sickle cell crises. Medroxyprogesterone acetate and megestrol acetate have also been shown to irreversibly decrease sickle cell counts.[118, 119] Progesterone in oil, 10 mg weekly, has a potency equivalent to that of medroxyprogesterone acetate, 150 mg intramuscularly every 3 months, the dose currently used for long-term contraception, and is roughly the equivalent of the progestin dose in low-dose oral contraceptives. Sex steroids reduce the risk of thrombus formation by stabilizing cell membranes and decreasing cell fragility.

INTRAUTERINE DEVICES

Three intrauterine contraceptive devices are available in the United States. In the past, intrauterine devices (IUDs) increased menstrual flow. Generally, Lippes loops and copper 7 and T devices increased flow from 35 to 40 mL to 50 to 60 mL per cycle and increased dysmenorrhea, undesirable events for female athletes. IUDs also carried an increased relative risk of ectopic pregnancy. Although IUDs decrease intrauterine and extrauterine gestation, they are much more effective at preventing intrauterine pregnancies. Therefore, pregnancies that occur with IUDs are associated with a high proportion of ectopic pregnancy. As many as 20% of pregnancies with the Progestasert progesterone-impregnated IUD were in the fallopian tube. Ectopic pregnancy clearly is a major problem in a remote diving locale.

The Mirena intrauterine system is a steroid-embedded contraceptive device that delivers very high local levels of the progestin levonorgestrel directly to the endometrium. The device releases 20 μg of the hormone daily and downregulates estrogen receptors in the endometrium. The uterus becomes unresponsive to the stimulation by endogenous (and exogenous) estrogens. Although the device is associated with an increase in the number of days of spotting, menstrual losses are dramatically reduced. Measurements of hematocrit, transferrin, and iron stores all confirm the profound reductions in bleeding. At the end of 13 cycles, women generally evidence a 95% reduction in flow. Moreover, there is a resultant reduction in dysmenorrhea. The device has even been shown to induce regression of endometriosis. This new method might prove to be a great benefit for a female diver plagued by heavy, prolonged bleeding.[120]

BARRIER METHODS

Barrier methods such as diaphragms, caps, foams, creams, jellies, and films present no risk for diving, but diving may decrease the efficacy of the methods by diluting the concentration of nonoxynol, the active contraceptive chemical agent in the vagina, something that might also occur during swimming. No increase in failure rates of barrier methods in women who participate in water sports has been reported.

In summary, as is often the case when reviewing issues about women and diving, the lack of clinical information allows only theorization, speculation, and inference. An extrapolation from the scant data suggests that steroidal contraception does not appear to constitute an increased risk for women divers. Oral contraceptives pose significant risks only to smokers, on land or in the water. Though it is highly speculative to say so, depo-progestins may actually offer protection from tissue injury in the event of a diving accident. IUDs and barrier methods may pose nuisances but not hazards for women divers.

Diving and Pregnancy

Diving during pregnancy might seem to be a short, pleasurable activity for the mother. Like swimming, the perceived weightlessness of diving may give the woman temporary respite from the burden she carries on land. However, the short-term pleasure of diving must be balanced against the potential long-term effects on the fetus as a passive passenger at depth.

Most workers investigating DCS and fetal risk agree that the fetus is at no increased

risk for bubble formation during decompression. In fact, three researchers[121–124] demonstrated that the fetus is more resistant to bubble formation than the mother. Only Fife and colleagues[125] found an increased risk in fetal lambs, but later Stock and colleagues[126] repeated the experiment and asserted that the increased risk was an artifact of instrumentation. Studies of DCS have also measured rates of birth defects after induced DCS in animals. Most of the experiments have been done at pressures in excess of those encountered in sport diving (6.4 to 7.1 ata). Despite the high pressures and high rates of DCS imposed on the study animals, only one of three studies in the literature demonstrated an increased rate of malformation after DCS. In the study of Gilman and colleagues,[127] hamsters with untreated DCS experienced increased rates of defects whereas treated animals did not.

Of more importance, perhaps, than birth defects is the very high rate of fetal death in utero found by animal researchers. Studies of dogs and rats show no increased rates of fetal death, but virtually all sheep studies show high rates of fetal loss. The impression is that the closer to term the fetus is, the greater the risk. The fetal circulation depends on the large patent foramen ovale and ductus arteriosus for the delivery of well-oxygenated blood from the umbilical vein directly to critical tissues, bypassing the systemic and pulmonary circulations. The fetal cardiovascular system lacks an effective filter; thus, any bubbles formed are likely to be directed to the brain and coronary arteries. This selective perfusion scheme probably accounts for the lethality of DCS in animals. Thus, researchers in the field concur that any bubble in the fetus is more ominous than several bubbles in the mother.

Concern about potential fetal oxygen toxicity comes from two quarters. First, diving at depth in compressed air increases the partial pressure of oxygen in fetal circulation. Second, diving accidents often necessitate hyperbaric therapy, exposing the fetus, sometimes repeatedly, to high levels of oxygen in utero. Miller and colleagues[128] exposed rats to 100% oxygen at 2 to 3 ata for 6 hours and found not only an increase in cardiovascular malformations but also an increased rate of fetal resorption. One hundred percent oxygen at 1 ata and air at 3 ata caused no increase in adverse outcomes. Fukikara[129] treated rabbits with 100% oxygen at 3.6 and 4 ata for 2 to 3 hours and found high rates of retrolental fibroplasia, similar to that seen in infants treated for prematurity with high oxygen. Gilman and colleagues[130] used standard United States Navy Treatment Table 6 (see Appendix 4) on hamsters with no increased rate of defects in the offspring.

Human data on HBO and the fetus are very limited. The former Soviets, as mentioned earlier, have used HBO repeatedly and for long durations in mothers, though at relatively low pressures. They have not reported any fetal problems. Van Hoesen and colleagues[131] reported a case of maternal carbon monoxide poisoning treated with 100% oxygen at 2.4 ata for 90 min with delivery of a normal infant 5 weeks later. Hollander and colleagues[132] also published a similar successful case. A few cases of maternal air embolism have been treated with HBO (see later). All fetuses died, probably because of the magnitude of the insult, not as a consequence of therapy.

Two surveys have questioned the outcome of women who dived while pregnant. In her survey, Bangasser[70] included questions on birth defects and losses in women who dived; the author found no increased rates. Bolton[133] also took a retrospective questionnaire. Limiting the study to the most recent pregnancy, 109 women dived before and during gestation. Sixty-nine dived before pregnancy but stopped when pregnancy was diagnosed. Although no statistical analysis was done, the survey suggests higher rates of low birth weight, birth defects, neonatal respiratory difficulties, and other problems in the dive group that continued diving perinatally. Of particular interest is the list of defects reported: multiple hemivertebrae, absence of a hand, ventricular septal defect, possible coarctation of the aorta, hypertrophic pyloric stenosis, and a birthmark. No major defects were reported in the nondive group. The cardiac anomalies are worrisome, but the first two defects listed are rare and dramatic. They were also associated with deep diving—120 and 160 feet, respectively. Much attention should be paid to these two items, and a good measure of caution is indicated. The need for caution is reinforced by a distressing case reported by Turner and Unsworth,[134] excerpted here:

We have seen a baby born with arthrogryposis and some dysgenic features whose

mother had been scuba diving in early pregnancy. The mother was a 22-year-old primigravida. She and her husband went on holiday from the 40th to 50th days [after the] last menstrual period. The mother dived at least once daily to a total of 20 dives in these 15 days. Most dives were to a depth of 60 feet or less, but three were to 100 feet and one to 110 feet. The ascent rate used by the mother and her husband was 60 ft/min, though this was usually estimated rather than timed. When decompression was required, a modified version of the U. S. Navy tables was used. All the dives except one were without complications. The exception involved an "equipment failure" of the husband, whom she was accompanying, at the end of a strenuous 15 min bottom time dive at 60 feet. The rate of ascent of both was described as "very rapid." She felt well but tired after this dive. No medications were used apart from oral Sudafed (pseudoephedrine), 60 mg, on two or three occasions early in the holiday to aid ear cleaning.

The rest of the pregnancy was uneventful. The abnormalities noted in the baby were unilateral ptosis, small tongue, micrognathia, and short neck. The penis was adherent to the scrotum. The upper limb joint movements were all normal except the hands. The fingers were fixed in flexion with some webbing between the 3rd, 4th, and 5th fingers, the thumb was digit-like but had two phalanges. The hip joints were dysplastic with reduced range of movement, and one hip was dislocated. There was fixed flexed deformity of the knees and bilateral equinovarus deformity of the feet. The head circumference was normal and motor development was appropriate for the baby's age at 3 months. Karyotype, electromyogram, and muscle biopsy were all normal.

No data, reports, or discussions of air embolism and its effects on the mother and the fetus in pregnancy appear in the diving literature. Fifteen cases of embolism from orogenital sex have appeared in the obstetric journals, all in young women in the second or third trimester. The embolism occurred from air being forcibly blown into the vagina. The first 12 cases reported maternal and fetal death in all instances. The next patient reported was treated with HBO 39 hours after the event and lived; however, she retained moderate neurologic deficits. Her infant was stillborn.[135] Another woman lived with no therapy and delivered a healthy but premature infant. The good outcome in this case has been ascribed to entrance of only a small volume of air into the arterial circulation.[136] The most recently reported case occurred at 22 weeks of pregnancy, and the patient was treated 9 hours after the insult. The mother survived without sequelae, but the infant was stillborn 3 weeks later.[137] These reports demonstrate the lethality of embolism in the fetus if air evolves in the uteroplacental bed. Overpressure diving accidents with pulmonary air embolism would surely present less gas directly to the uterine bed, and the mother's circulation would act as a filter. Because the volume of gas lethal to the fetus has not been measured and is probably very small, and because bubbles would be preferentially delivered to the heart and brain, even shallow diving presents grave fetal risks.

The hazard of diving during pregnancy extends beyond DCS and air embolism. Because both of these injuries usually require HBO therapy, the safety of treatment merits examination. The medical literature offers no evidence of adverse fetal outcomes from controlled hyperbarism. The classic paper on maternal and fetal effects of hyperbaric states, by Assali and colleagues[138] in 1968, detailed changes in uterine and fetal blood flow during administration of HBO. On 100% at 1 ata, maternal arterial P_{O_2} reached 500 mm Hg, but fetal umbilical vein P_{O_2} increased by only 10 to 15 mm Hg. When pressure was increased to 3 ata, maternal P_{O_2} rose to 1300 mm Hg; umbilical vein P_{O_2} rose to 300 mm Hg, but umbilical artery P_{O_2} levels reached only 50 mm Hg. Maternal and fetal arterial pressures did not change significantly. Placental and umbilical flow rates decreased slightly during HBO administration. The major finding was alterations in fetal blood flow pattern. Ductus arteriosus flow decreased dramatically when the oxygen tension in the pulmonary blood rose. At the same time, flow increased in the ascending aorta but effective fetal cardiac output decreased. Apparently, the fetal pulmonary bed is exquisitely sensitive to oxygen tension and responds with vasodilatation as oxygen tension rises. Thus, HBO causes a shift from a fetal blood flow pattern to a neonatal pattern. The shift reverses when oxygen tension returns to normal. One can only speculate on the long-term effects of prolonged in utero exposure of the fetus to high oxygen because of neonatalization of the fetal circulation.

A more immediate problem, which is still speculative, is concern that basic physiologic changes in pregnancy may compound diving risks. Many divers experience some anxiety at the outset of a dive. Combining the increased exercise demand, cold stress, pregnancy load, and sympathomimetic reflex of anxiety, potent vasoconstriction is possible; therefore, the potential for decreased uterine blood flow may be significantly increased for the pregnant diver. Although diving is submaximal in its cardiovascular demands, short, hard bursts of activity are needed occasionally. Such episodic demands are more likely to compromise uterine blood flow than gradual increases or sustained requirements of aerobic activity. Abrupt shifts in flow dynamics may produce unrecognized, short episodes of decreased fetal perfusion. For this reason, racket sports and power weightlifting are generally less than ideal forms of exercise during pregnancy. If diving elicits erratic cardiopulmonary responses, it should be avoided.

During pregnancy, maternal body fluid distribution is altered, with increased interstitial fluid and edema. These third-space fluids have a diminished exchange of dissolved gases in the central circulation. Though not addressed in the literature, maternal third-space fluid might offer a reservoir for nitrogen retention. The potential sites for nitrogen sequestration include the increased deposits of body fat found during pregnancy. On the average, women increase body fat from 28% to 33% (normal) to 33% to 36% during pregnancy. Combining third-space and fat stores as harbors for nitrogen, off-gassing time for pregnant women may not correspond to the limits established in the standard repetitive dive tables. Some of this loss of effective circulation may be counterbalanced during diving, by centralization of maternal circulation during immersion, which has recently been observed.[139]

Fluid retention during pregnancy also causes nasopharyngeal swelling. Women with no prior allergic symptoms often complain of nose and ear stuffiness in pregnancy. Obviously, the risk of ear and sinus squeeze is increased. Many pregnant women become dependent on decongestants and incur the risk of rebound congestion when the medication wears off. If rebound occurs during diving, ascent may be slow and arduous.

During the early months of gestation, approximately two thirds of pregnant women experience some degree of gastrointestinal dysfunction, including nausea, vomiting, increased gastric acidity, and gastric reflux. Later in pregnancy, as the uterus enlarges, reflux increases. Gastric emptying time is delayed; obstetricians and anesthesiologists have come to regard all pregnant women as having full stomachs regardless of the timing of the last meal. If motion sickness on the dive boat adds to morning sickness, the pregnant diver experiences a difficult dive. Consequently, she is at high risk for vomiting into her regulator, an accident few sport divers are prepared to handle safely.

Pregnancy induces a state of relative vasodilatation accompanied by an increased basal metabolic rate. Although vasodilatation may increase the risk of hypothermia, a higher metabolic rate may increase the risk of hyperthermia. During diving, the thermal risk is defined by the water temperature, the length of the dive, and the quality of the diving garment. Heavy activity in a heavy wet suit in warm water leads to hyperthermia. Light activity with no protective clothing in cool water may lead to hypothermia. A paper from Finland[140] cited increased neural tube defects in fetuses born to women who used saunas during pregnancy. Though no other confirmatory reports demonstrate adverse outcomes of hyperthermia, obstetricians generally advise women to avoid hyperthermia when pregnant. Ama divers, who endure repeated cold stress, were reported to have a higher incidence of low-birth-weight infants. Because these women incur exceptionally high levels of exertional demand as professional divers and often consume fewer calories than are required to compensate for thermal debt, the smaller sizes of their infants may not be due solely to thermal stress.

Comfort during diving is not a medical issue, but pregnancy introduces problems in fit and function of dive gear. The changing size of the abdomen throughout gestation soon stresses even the most flexible wet suit. The safe location and placement of weight belts under, over, or around the gravid belly may create an unforeseen inability to ditch them during an emergency. Over the uterus, the belt does not fall free; under the uterus, the belt may be poorly positioned for quick release. Balance with the fetus in front and tanks in back may pose an interesting challenge. Balance instability, although obviously not a problem in water, makes leaving or entering a boat more difficult.

Any diving disaster threatening the mother poses similar threats to the fetus. In

a litigious society such as ours, diving and pregnancy seem incompatible. The same woman who eschews coffee, shuns hair coloring, and avoids vitamins with yellow dye, none of which is a fetal hazard, will ask if she can continue to dive. Laypersons fail to see that oxygen at pressure is a potent drug. No clear risk can be defined and confirmed from the very limited data available on pregnancy and diving, but neither are we reassured by the few available reports. In view of the elective nature of sport diving, when one weighs a relatively short hiatus from diving imposed by pregnancy against the possibility of a lifelong disability created in utero by pressurized gas, the rational answer seems clear: Pregnant women should not dive. And because many inadvertent teratogenic exposures occur early in the first trimester before a pregnancy is recognized or confirmed, women actively trying to become pregnant should be advised to put off diving until after delivery.

RETURN TO DIVING AFTER PREGNANCY

Diving, like any other sport, requires a certain modicum of conditioning and fitness. The postpartum return to diving should follow guidelines suggested for other sports and activities. Generally, after vaginal delivery, women can resume light to moderate activity within 1 to 3 weeks, depending on their prior level of conditioning, exercise and conditioning during pregnancy, pregnancy-related complications, and postpartum fatigue and anemia. Women usually resume exercise programs and sports participation in earnest at 3 to 4 weeks post partum.

Obstetricians generally recommend avoiding sex and immersion for 21 days post partum to allow the cervix to close, thereby decreasing the risk of introducing ascending infection into the genital tract. Thus, I think a good rule of thumb is to recommend that women wait 4 weeks post partum before returning to diving.

After caesarian delivery, one must also consider wound healing. Most obstetricians advise waiting at least 4 to 6 weeks after operative delivery before resuming full activity. Recovery of aerobic conditioning, coupled with wound healing and the significant strength needed to carry dive gear, suggests a longer recovery time than prescribed for resumption of the daily routine. A delay of 8 to 12 weeks post caesarian section is recommended before returning to diving. Any severe medical complications of pregnancy, such as twins, preterm labor, hypertension, and diabetes—which may have been treated with prolonged bed rest and which may have led to profound deconditioning with loss of aerobic capacity and muscle mass—will further delay return to diving. For medically complicated parturients, medical screening and clearance should be done to ensure, as in the case of the gestational diabetic, that the patient does not have continued clinically significant glucose intolerance.

Postpartum anemia, with hemoglobin under 10 g/dL, may be slow to resolve. The additional nutritional demands imposed by lactation may slow recovery from anemia. Women should undergo postpartum hemoglobin measurement before returning to diving. Caring for a newborn, a rigorous and demanding time in life characterized by poor sleep and fatigue, may interfere with a woman's attempts to recover her strength and stamina. A new mother with a colicky or demanding infant needs a clear and honest reality check on the capacity to handle the demands of diving. The infant should be her priority; the oceans can wait.

DIVING AND BREASTFEEDING

The duration of breastfeeding in Western cultures is usually short, and complete breastfeeding, with breast milk as the only source of infant nutrition, rarely exceeds 6 months. There is no risk of nitrogen accumulating in breast milk. There is no risk of an infant swallowing dissolved nitrogen. Diving causes centralization of circulation, and dehydration from immersion may interfere with milk production. Enteric bacteria in water might grow on the skin under a wet suit and theoretically might increase the risk of mastitis or infant diarrheal illness. Careful cleaning of the breast after diving and before feeding is advised.

Diving and Breast Implants

Silicone, saline, and silicone-saline implants were exposed to a variety of depth/time profiles simulating recreational diving. Minor changes in bubble size during the course of dives ranged from 1% to 4%. The implants were exposed to ambient HBO in a chamber,

not in situ. Thus, gas could directly diffuse into the implant from the ambient gas in the chamber, a very different dynamic than gas diffusion from the circulation into the breast tissue and then into an implant in vivo. Saline implants absorbed less nitrogen because N2 is less soluble in aqueous medium than in silicone. The greatest volume change occurred in the silicone-saline implant, with silicone acting as a reservoir for dissolved nitrogen. The amount of volume change in the implant was not enough to predispose to rupture. Gas bubbles resolved over time. Sudden decompression to hypobaric pressures simulating flight caused some increase in the volume of the implants. However, in real life, abrupt, dramatic shifts in pressures do not happen.[141, 142]

From a practical patient management vantage, diving poses a hazard only in the immediate postoperative period. All suture lines should be well healed to help avoid infection. Pressure on the suture lines should be avoided to minimize the risk of wound dehiscence. Women with implants should be advised to avoid putting buoyancy compensator straps over implants to prevent any undue pressure on the implant bag.

Breast implants filled with saline are neutrally buoyant. But silicon implants are heavier than water and may alter buoyancy and attitude in the water, particularly if the implants are large. Appropriate training and weighting obviate such difficulties. Any woman who has recently undergone surgery and who is returning to diving would do well to perform a safety checkout dive to be sure she is properly weighted and that she has good buoyancy control.

Breast Cancer and Breast Surgery

As the diving population ages, questions regarding fitness to dive after treatment for serious illness will continue to escalate for both men and women. The following remarks can be applied to men and women treated for malignancies.

First, adequate postoperative recovery, scar stability after biopsy or mastectomy, and adequate mobility (enough to move comfortably in the water and to use gear properly) are necessary before diving. Buoyancy and fit of gear should be adjusted before open-water diving is resumed if extensive tissue has been removed or if reconstructive procedures used saline or silicone implants. Patients undergoing radiation therapy and chemotherapy should not dive. These treatments carry a number of risks, including immunosuppression, diarrhea, chemotherapy-induced nausea and vomiting, dehydration, anemia, and fatigue.

Long-term complications of treatment include a significant risk of pulmonary fibrosis with decreased diffusing capacity after use of chemotherapeutic agents and from radiation damage. Agents such as bleomycin, methotrexate, mitomycin, busulfan, and the nitrosoureas may cause pulmonary damage. These agents may cause interstitial pneumonitis, alveolitis, and pulmonary fibrosis. Methotrexate and procarbazine may cause an acute hypersensitivity reaction. Cytosine arabinoside has been found to cause noncardiogenic pulmonary edema. Multiple drugs, coupled with radiation therapy or preexisting lung disease, may potentiate pulmonary damage. Radiation pneumonitis, usually occurring 2 to 6 months after completion of radiation therapy, are common when the dosage exceeds 40 Gy. Symptoms may be delayed for years after treatment. Patients treated with doxorubicin and cyclophosphamide are at risk for cardiotoxicity.[143] These same cautionary notes should be sounded after treatment of ovarian cancer and other neoplasms.

Any question of limited pulmonary reserve should trigger a complete pulmonary evaluation and consultation. If a patient has been exposed to agents likely to cause pulmonary damage, pulmonary function tests and diffusion capacity are indicated.

Hysterectomy and Other Pelvic Surgeries

All that has been said about diving after a caesarean applies to diving after major surgery. It takes 6 weeks for the vaginal cuff to close completely after surgical extirpation. If surgery is complicated in any way (e.g., infection, anemia), diving should be delayed even further. These recommendations apply to total abdominal hysterectomy, vaginal hysterectomy, hysterectomy plus salpingo-oophorectomy, subtotal hysterectomy, and laparoscopically assisted vaginal hysterectomy.

Aging and Diving: The Menopausal Diver

To date, there is not a large population of postmenopausal women in the diving community who are at risk for osteoporosis: The average age at menopause is 50, with the onset of osteopenia at age 60 to 65, osteoporotic fractures becoming highly prevalent at age 70 to 75. Osteonecrosis and osteoporosis are unrelated conditions, save for both affecting bones. Osteoporosis results from decreases in bone formation by osteoblasts and relative increase in bone resorption by osteoclasts, resulting in loss of bone mineral content and ultimately in bone matrix. Osteonecrosis results from infarction of the microcirculation of bone. Clearly, having both disorders would be an unfortunate coincidence. All that can be said is that older women might do well to dive conservatively to minimize the risks of osteonecrosis, thus avoiding imposing one bone-damaging disease on top of another.

DIVING AND HORMONE REPLACEMENT THERAPY

The world of menopause medicine experienced an abrupt and startling upheaval in 2002. The Women's Health Initiative (WHI),[144] the first large, prospective, randomized clinical trial designed to assess the effects of several health practices on the rates of diseases in an aging female population, reported its first results dramatically, with early termination of one of the largest segments of the study. The WHI hormone replacement study is actually two trials. The hormone replacement study included 16,609 women taking most commonly prescribed hormone treatment used in the United States—conjugated equine estrogens, 0.625 mg, plus medroxyprogesterone acetate, 2.5 mg (marketed as Prempro). Half of the subjects received hormones; the other half received a placebo. A separate study includes women who have undergone hysterectomy and who have been randomized to conjugated equine estrogens or placebo, a study that continues at the time of this writing.

The study of combined hormone therapy was stopped in July 2002. The main reason for stopping was an increase in invasive breast cancer from 0.30% to 0.38% per year of use. In the hormone group, nonfatal myocardial infarctions increased from 0.23% to 0.30% per year (Table 19–4). Annual nonfatal stroke incidence increased from 0.03% to 0.04%. These adverse events were offset somewhat by a reduction in the risk of colorectal cancer and a reduction in hip fractures. Annual mortality, however, was not changed.

The WHI has been criticized for having a mean age at entry of 63 years. The study was specifically constructed and weighted with one third of the population under 60 and two thirds over age 60. Although no significant interactions with age, race, body mass index, smoking status, blood pressure, diabetes, aspirin use, or statin use were found for the effect of hormone replacement therapy on coronary heart disease, stroke, or venous thromboembolism in the WHI study,[144] a trend can be noted toward a greater cardiovascular risk in the youngest women. The hazard ratio for women taking Prempro

Table 19–4. Women's health initiative study outcomes

Outcomes	Placebo (8102)		Treatment (8506)	
	Number	*Percent**	*Number*	*Percent**
Nonfatal myocardial infarction	96	.23	133†	.30
Nonfatal stroke	59	.14	94†	.21
Venous thromboembolism	67	.16	151†	.34
Breast cancer	124	.30	166†	.38
Colorectal cancer	67	.16	45†	.10
Hip fracture	62	.15	44†	.10
Total mortality	218	.53	231	.52

Adapted from Women's Health Initiative Writing Group: Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women's Health Initiative randomized controlled trial. JAMA 17:321–333, 2002.
*Annual percent incidence.
†Significant at $P < .05$.

compared with controls in the age group 50 to 59 years is 1.67, in the 60 to 69 group 1.26, and in the 70 to 79 group 1.18.[145]

The rate of cardiovascular disease events in women over 60 was 0.7%, only one tenth the rate for women in the general population in this age group. The expected rate is 7% to 8%. Thus, the WHI represents a "best worse case" scenario.

The American College of Obstetrics and Gynecology has advised practitioners and women to limit the use of hormone therapy to the lowest dose, for the shortest duration of time, for the management of symptoms only.

Given this new reality, diving physicians would do well to add hormone replacement therapy to the list of risk factors predisposing to nonfatal myocardial infarction, stroke, and venous thromboembolism. Female divers who present with neurologic abnormalities after diving need careful evaluation to differentiate DCS or arterial gas embolism from a primary cerebrovascular event, especially if they are on hormone replacement therapy.

CONCLUSIONS

It is obvious that women are different from men and that female divers are different from male divers; however, when all the data are carefully considered, the rate of diving-related injury and illness is no greater for female divers than for male divers. Female divers differ in that reproductive health issues, such as contraception and pregnancy, may interfere with diving activities at specific, limited times during a woman's life. Issues of health and safety apply equally across gender lines. Guidelines for fitness to dive and suggestions to enhance diving safety are, in the main, gender-neutral. Diving experience, physical fitness, and dive planning are far more important to risk management than the difference between an X and a Y chromosome. As the diving population ages, all divers, male and female, need periodic reassessment of fitness to dive.

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CHAPTER 20 Diving in the Elderly and the Young

Alfred A. Bove

Age limitations are commonly imposed on commercial and military divers, but there are no formal limitations on the sport diver. Divers under the age of 10 years and older than 75 years are known to participate in sport scuba diving. A few shallow-air commercial divers and scientific divers continue to work into their sixth and seventh decades of life. Military divers are usually limited to age 45 and younger (see Chapter 29). Limitations with the young and the elderly follow different patterns, but youth and age alone should not be considered as contraindications.

DIVING IN THE ELDERLY

Although there has been a trend toward increased physical activity in older persons, most elderly people do not exercise and indeed are often discouraged from participating in regular exercise. Good physical condition is essential for diving. Physical capacity is known to decline with age[1–6] because of age-related changes in cardiovascular function, skeletal muscle, and other organ systems as well as the reduction in physical activity that is common in older persons. However, maintaining good health remains an important goal in preserving physical function. In an analysis of over 100,000 medical beneficiaries, Cooper and Kohlmann[7] found that heart and lung disease and back pain were important causes of reduced physical capacity. Careful attention to these disorders is important when assessing an elderly individual for recreational diving.

Causes of reduced muscle mass in the elderly (sarcopenia) are multiple and include effects of detraining, chronic illness, and changes in energy metabolism[8,9] Reduced physical activity in the elderly is to some extent caused by social factors that relegate physical activity and exercise to the younger population. Recent data suggest that endurance exercise in the elderly may result in a compensatory decline in energy expenditure during nonexercising periods and may be counterproductive to fat loss and overall conditioning.[9] Isometric exercises have been shown to improve muscle mass, exercise endurance, and aerobic capacity in the elderly[10,11] and are an important part of an overall conditioning program for all elderly persons, including divers. The combination of factors causing loss of muscle mass in the elderly, when associated with reduced physical activity, results in considerable reduction of muscle strength, which may interfere with safe diving. Therefore, most elderly divers cannot sustain the exercise workload or the strength capacity of younger persons.

In a study by Vincent and colleagues,[11] maximum oxygen consumption was in the range of 20 to 25 ml/kg/min (6 to 8 METS), depending on level of training. This level of physical capacity does not allow for responses to unsafe conditions that require extreme physical exertion for safety. Most elderly divers therefore require a diving partner who can provide assistance when physical exertion is needed for safety. Many elderly persons have osteoporosis and are prone to bony fractures with minimal trauma. Several studies have shown an improvement in muscle energy metabolism with a combination of endurance and resistance exercise in the elderly.[9,12] Combined endurance and resistance training may reduce the severity of osteoporosis.[13]

The reduction in physical capacity, increased susceptibility to fractures, and higher incidence of pulmonary, vascular, and metabolic disorders must be accounted for when advising older divers. Several studies have examined older athletes and found marked physiologic differences in these

persons when compared with age-matched, nontrained younger persons.[14–16] Many of the cardiovascular and skeletal muscle changes are first noted in the fifth decade of life and decline by 8% to 10% per decade.[17] However, the decline in physical capacity with age can be minimized by continued physical training.[7,17] In addition, some evidence indicates that cognitive function and decision-making responses are improved by physical conditioning in the elderly.[18] Lord and Menz[19] found a correlation between a number of physiologic and psychological health scores and physical conditioning. Thus, conditioning programs are essential for safe diving with elderly divers. Holm and colleagues[20] found that elderly male Ama divers had adequate conditioning to continue their work, but reflex bradycardia was diminished compared with younger divers. Recommendations for diving in the elderly can be provided when chronic or acute illness does not preclude such activity and when physical condition allows the diver to perform safely. This discussion reviews some of the changes in physiologic responses known to occur with aging and, based on these concepts, provides some recommendations for diving in the elderly.

Cardiovascular System

It is generally accepted that systolic and diastolic blood pressures rise with age.[21] However, elevated diastolic pressure (>90 mm Hg) is still considered abnormal and subject to medical therapy. The accepted range of normal systolic blood pressures is known to increase slowly with age, so that at age 70, for example, an acceptable high limit for normal systolic blood pressure would be higher (i.e., 140 mm Hg) than the acceptable high level of normal in a 20- to 30-year-old. Persons with hypertension should be treated before being cleared for diving.

Systolic blood pressure elevation in the elderly is due in part to alterations in the stiffness of the vascular system.[22] Clinical observations[23] and experimental studies[24] suggest that aortic smooth muscle tone is reduced following prolonged exercise training. Studies in animals[24] suggest that exercise might reduce peripheral vascular resistance and increase aortic compliance, thus lowering systolic and diastolic blood pressure. Training can provide some blood-pressure control and should be recommended as part of a conditioning program for diving.

Peripheral vascular resistance is also known to increase with age. This increase may be caused by a reduction in skeletal muscle mass[25,26]; however, the change is probably caused by multiple factors and is not necessarily related to long-standing hypertension. Because most of the systemic vascular resistance resides in the vasculature of skeletal muscle, the increase in peripheral vascular resistance in elderly persons might result from a combination of increased hormonal sensitivity[27] and reduced muscle mass with partial loss of microvascular channels in the peripheral vascular bed. Studies in hypertensive populations suggest that exercise lowers peripheral vascular resistance.[28,29] A program of endurance exercise training in elderly hypertensives reduces blood pressure because of changes in peripheral vascular resistance and possibly because of changes in the vascular tone of the large distributing arteries. Blood-pressure control in trained elderly men may also be improved because of preserved autonomic nervous system function and preserved baroreceptor sensitivity.[30] Blood-pressure response to acute exercise in older persons is known to be altered by training[14,21,31]; however, elderly persons with apparently mild hypertension may experience marked elevation of blood pressure during exercise, including diving. If exercise produces a significant elevation of blood pressure (diastolic > 110 mm Hg, systolic > 210 mm Hg), antihypertensive treatment is needed before a diving program is begun.

Cardiac Performance

Studies in experimental animals and in humans[32–35] indicate age causes a reduction in the contractile performance of the myocardium. This reduction is small and generally of minimal consequence; however, it can be detected in studies designed specifically to examine the contractile characteristics of the myocardium.[36] Catecholamine responses in the elderly are enhanced[28,37]; however, it is unclear whether catecholamine receptors in the elderly have the same sensitivity as receptors in a younger population.[38] Some studies[27] have demonstrated increased blood catecholamine levels in response to

exercise in the elderly, suggesting that the neuroregulatory control of the heart during exercise requires release of greater amounts of catecholamines to obtain a cardiac response appropriate for the exercise level.

Diastolic ventricular relaxation in elderly subjects is impaired because of increased myocardial stiffness.[39,40] Thus, high heart rates are less well tolerated in older, compared with younger, persons. Although the increased stiffness of the myocardium in the elderly is thought to be a normal aging change, there is some concern that the increased stiffness may ultimately lead to diastolic heart failure. If increased diastolic stiffness raises pulmonary venous pressure to the point of lung congestion, the elderly subject experiences dyspnea on minimal exertion. When combined with central blood shifts due to water immersion (see Chapter 5), the stiffer myocardium in an elderly diver may result in pulmonary congestion. The myocardium in the elderly is also known to be less responsive to catecholamines; thus, exercise requires release of greater amounts of catecholamines to obtain a cardiac response appropriate for the exercise level.

A well-known alteration in cardiac performance associated with age is the decline of maximum heart rate.[3,41,42] The cause of this alteration in heart-rate response in the elderly is not clear; however, it is possible that changes in autonomic tone or in the state of innervation of the heart by the autonomic system are responsible.[38]

Maximum oxygen uptake also declines with age beyond the late twenties.[33,43,44] The rate of decline in maximum oxygen uptake with age may be rapid or slow, depending on the state of physical condition and the continuity of endurance training in older life. Thus, the decline of maximal oxygen uptake with age described from early studies was found to be associated with a state of poor physical conditioning. A program of physical activity continuing over several decades has been found to slow the decline of oxygen uptake originally thought to be exclusively age-related.[45,46] Anaerobic threshold is lower in older, untrained subjects,[47] and lactate production with exercise may be exaggerated. These changes are related to the decline in maximum oxygen consumption. Increased lactate during exercise lowers arterial pH and induces excess hyperventilation to counter the metabolic acidosis. Thus, severe dyspnea may occur during diving-related exercise and induce a panic reaction in an inexperienced elderly diver. Loss of physical strength with age may also result from detraining.[48] Because of the decline in maximal oxygen uptake with age, maximal work capacity is reduced in older persons compared with persons in the third and fourth decades of life; similar changes occur in both male and female populations.[49] Training programs for recreational diving should account for reduced capability in elderly divers. Matching an elderly trainee with peers prevents excess physical stress resulting from pressure to perform with younger, better-conditioned trainees.

Coronary Disease

When assessing risk for coronary disease, age continues to be one of the most important factors.[50] The increased risk of coronary events in elderly divers warrants a careful assessment of coronary status and risk for ischemia (see Chapter 25). Improved physical conditioning lowers the risk of coronary[51] and cerebral[52] vascular events. Thus, assessment of risk for vascular events during diving should include an assessment of physical conditioning and exercise activity.

Ventilatory Performance

Data from Christensson and colleagues[53] show an age-related increase in unventilated lung compartments. These changes are likely due to decreased lung compliance in elderly subjects. Older divers may experience more breathing difficulty than younger divers because of increased work of breathing. Brischetto and coworkers[54] found that eldery subjucts had reduced ventilatory sensitivity to CO_2 production with exercise; thus, these divers may be more susceptible to CO_2 toxicity from hypoventilation.

Superimposition of chronic illness adds to the decline of work performance in the elderly. It is not reasonable to expect elderly divers to perform as well as younger divers. Because of the reduced maximum oxygen consumption, the elderly person experiences greater physical stress when exercising at a given work level compared with younger persons, whose maximum oxygen

consumption is higher than that of the elderly, even when the relative state of physical training is approximately equivalent. Because perception of the intensity of exercise depends on the percentage of maximum uptake at a given workload, moderate levels of physical activity feel more stressful to an elderly person, whose maximum oxygen consumption is low compared with a younger person.

Metabolic Changes

Other contributing factors to consider with elderly divers include age-related alterations in the metabolic state.[55] Primary insulin deficiency in older persons may cause them to have more glucose intolerance than younger persons.[56,57] Because of reduced hypoglycemia awareness, the elderly are more prone to asymptomatic hypoglycemia. Although many elderly subjects with mild hyperglycemia are not affected by the metabolic changes of diabetes, symptomatic hypoglycemia should disqualify a person from diving. Glucose intolerance may be present in half of patients over age 65.[58] Chapter 26 discusses diabetes and diving.

Thermal Stress

Although most recreational diving is done in warm or temperate waters, even in tropical diving locations the ocean temperature may be in the range of 78° to 82°F (26° to 28°C) at usual sport diving depths. This temperature causes a heat loss in diving that can be limited by appropriate diving dress, but in most cases sport divers lose heat even in tropical waters (see Chapter 13). The elderly are particularly susceptible to hypothermia from even tropical ocean exposures. Smolander[59] reviewed the effects of cold exposure in the population older than 60 years. The elderly are less able to maintain core temperature during a cold challenge and have reduced thermal perception during cooling. Normal diurnal temperature modulation is also diminished.[60] The activity of the thyroid gland may be diminished. The elderly person, therefore, is less tolerant of alterations in temperature. Elderly persons are noted to have reduced basal metabolic rates compared with younger persons,[26] and these differences in metabolism also should be considered when evaluating older persons for diving. Diminished thermoregulatory capacity, when added to the attenuated metabolic response to cold stress, places elderly divers at greater risk for hypothermia than younger divers.

Clinical hypothermia is recognized as a serious clinical problem in the elderly,[61,62] although most clinically relevant occurrences are not related to diving.[61] Factors that contribute to the increased risk of hypothermia include reduced sensation of cold, medications that inhibit thermal counter-regulatory responses, chronic illness, and reduced subcutaneous fat. In severe hypothermia, elevated creatine kinase may occur from rhabdomyolysis.[63] This population may also have a reduced corticosteroid response to stress that reduces the response to both heat and cold stress. Both heat and cold tolerance may be reduced in elderly divers.[64] During training, elderly persons should be given careful instructions concerning reduced work capacity and altered cardiovascular responses in extremes of temperature. Considering of these factors provides elderly divers with safe diving programs.

Other Age-Related Alterations

In addition to known cardiovascular, endocrine, metabolic, respiratory, and nervous system changes with age, connective tissue structure also changes. As age progresses, collagen polymerizes from a relatively soluble form to a relatively insoluble form that is stiffer than the younger, nonpolymerized collagen.[65] The changes in collagen structure result in increased stiffness of tendons, ligaments, and joints. In addition, the incidence of osteoporosis increases with age, and minor disabilities can occur from involvement of the knee and hip joints, the spine, and the hands. Alterations in collagen structure cannot be avoided in the elderly, and increased stiffness of joints and tendons with associated reduction in range of motion will reduce the capacity of elderly divers to perform arduous physical tasks related to diving. Most elderly divers or diving candidates will relate joint stiffness or pain, particularly related to the hip, knee and spine.[7] These findings should not prohibit diving unless a significant physical disability results

from limitation of motion of a joint. Among the sport diving population, there is little or no risk for development of dysbaric osteonecrosis, and no joint injury would be aggravated by this disorder in a recreational diver. Medical causes of osteonecrosis should be considered in elderly divers with clinical evidence of this disorder. These include: alcoholism, collagen diseases, hemogolobinopathies, and steroid therapy.[66] In commercial divers, osteonecrosis is a definite hazard after long-term exposure (see Chapter 21). Van Blarcom and associates[67] found that osteonecrosis can progress after diving work has ended; thus, in the elderly or retired diver, osteonecrosis should continue to be considered when complaints of joint dysfunction are found.

In a post mortem study of tympanic membrane function, Jensen and Bonding[68] found an age related weakening of the tympanic membrane that they suggested would make older divers more prone to tympanic membrane rupture.

It remains unclear whether older divers are more susceptible to decompression sickness. Hoiberg showed no age effects on decompression sickness.[69] However, this report involved only Navy divers under 50 years of age. Carturan and colleagues[70] found age to be a contributing factor to bubble formation in a study examining the effects of several factors on bubble formation, however none of their subjects developed overt decompression sickness. In a study of altitude exposure, Sulaiman and associates[71] found an increased incidence of decompression sickness in subjects over age 42, compared to younger subjects; however, they did not report the distribution of subjects over age 42. More recent anecdotal data suggest that elderly divers using established decompression tables are not more prone to DCS then younger persons.

Another important consideration in dealing with diving in the elderly is alteration in neurological function.[37] Normally a slowing of certain central nervous system functions can be noted with age. The elderly may have a lengthening of reflex time and somewhat less precise motor control compared with younger persons. Elderly divers should choose diving that is within the capability of their neuromuscular system. It is important to document a baseline neurological examination prior to diving to avoid misdiagnosis of neurologic decompression sickness when subtle, chronic neurologic changes may be mistaken for a diving related illness.

Evaluating the Elderly Diver

For initial assessment, physical capacity must be assessed in both normal elderly subjects and elderly patients with chronic illness who wish to dive. This evaluation may reveal that physical capacity is significantly reduced compared with younger persons. Some elderly persons who have continuously exercised to remain fit may have surprisingly good physical capacity. Another component of the initial assessment is to identify chronic diseases that would interfere with safe diving. The elderly individual is more likely to have coronary or other vascular disease, which may be undiagnosed. Pulmonary function may be reduced, there may be endocrine metabolic disorders, particularly glucose intolerance, either manifest or undiagnosed, and renal function may be impaired because of arteriosclerosis. Chronic hypertension may result in diastolic cardiac dysfunction that is often first detected by severe dyspnea with mild exercise. Alterations that occur in the elderly as part of the aging process and as a result of chronic illness must be considered in evaluation for diving.

The reduced work capacity of older persons requires low exertion diving programs. Older subjects can be tested for exercise capacity using a standard stress test. In exercise testing, elderly persons in general do not achieve the same levels of exercise capacity as younger persons, and when chronic illness is present, exercise tolerance may be further limited. Severe limitations to physical capacity due to chronic illness or detraining should prohibit diving.

Chronic diseases, known to be of higher incidence in the elderly, present special problems in diving. A significant and important problem in the elderly is the high incidence of cardiovascular disease. Atherosclerosis can affect blood flow to the brain, heart, kidneys, or skeletal muscle, such as the legs. Many times these arterial obstructions are undetected, and high flow demands induced by swimming with diving gear may result in inadequate oxygen supply and abnormal function of a tissue or organ. Because these disorders are higher in frequency in

the elderly, it is important to search carefully by physical examination, history, and appropriate laboratory studies to rule out the possibility of significant atherosclerosis. Of most importance is the presence of coronary atherosclerosis with coronary artery obstruction, which limits flow to the myocardium. Flow demands in the myocardium can increase substantially with diving, and in the presence of severe atherosclerosis with impaired blood flow myocardial infarction, serious arrhythmias, or sudden death may follow. In the elderly avoidance of serious cardiac problems while diving can be achieved through appropriate screening evaluation[72] including an exercise stress test with electrocardiographic and blood pressure monitoring, which documents the physical capacity of the patient and detects coronary artery disease (see Chapter 25). The value of an exercise stress test in this population cannot be overemphasized. This test provides both diagnostic screening for coronary disease and the information needed to judge capacity for diving. A study of this type is essential in elderly persons prior to instituting a diving program.

Consideration of diving in the elderly must also take into account alterations in bone and joint structure and strength to avoid musculoskeletal injury from diving. The osteoporosis of the elderly can be a significant problem if an elderly diver is subjected to trauma that might lead to a fracture. Poorly conditioned persons beginning a diving program should be instructed to avoid heavy lifting or traumatic situations that might result in injury to bones, joints, or tendons, since these structures are weak compared with those of younger divers, and heal more slowly. For poorly conditioned persons who have not exercised for long periods of time, the initial training may result in a musculoskeletal injury that precludes further conditioning. Table 20–1 summarizes the important issues related to diving in the elderly.

In conclusion, it is reasonable to provide clearance for some elderly subjects to undertake sport diving. Programs for the elderly require special considerations because of the reduced physical capacity and alterations in the neurological, cardiovascular, pulmonary, and endocrine systems. Diving candidates should undergo exercise testing in a controlled environment with electrocardiographic and blood pressure monitoring. With a careful evaluation, an elderly individual in good health can be given clearance for diving. Taking into account all of these variables, it is possible and desirable to provide diving programs to healthy, moderately conditioned elderly subjects. These divers, however, should not be considered to be as capable as younger divers.

Table 20–1. Factors to consider when evaluating an elderly subject for diving

Cardiovascular system
Hypertension
Coronary disease
Heart failure
Musculoskeletal system
Back and spine disease
Knee and hip arthritis
Osteoporosis
Reduced muscle strength
Pulmonary system
Reduced lung capacity
Dyspnea
Reduced strength and endurance
Thermal stress intolerance

DIVING IN THE YOUNG

Medical considerations for young divers are directed toward emotional maturity, ability to learn and understand the requisite physiological, physical, and environmental data needed for safe diving, and physical strength necessary for handling diving equipment. In commercial and military diving, the lower age limit is 19 years, but most commercial divers are two to three years older. Sport diving imposes no legal limit, but most training organizations require candidates to be 15 years old for full certification. Training is provided to younger candidates who receive conditional certification until age 15.[73] Pouliquen reported on a diving program for children from 4 to 12 years old.[74] This program provides training and supervised scuba diving and had no claimed problems in 7000 dives undertaken by children. Sport diving training has been organized for children as young as 8 years old. The programs, however, limit diving to shallow depths, and require a trained instructor to accompany all children on their dives.

Questions have been raised concerning injury to developing bones. To date no evidence has been provided to support this concern. Nevertheless, young divers should

use diving profiles that minimize risk for decompression sickness. Shallower, shorter dives for children will remove any concern for bone injury.

Ear and Sinus Considerations

Problems related to eustachian tube dysfunction are well known in children,[75] and the consequent increase in otitis media is well documented.[75] Children have a high incidence of ear barotrauma after flying due to inability to equalize the middle ear.[76] They appear to have difficulty performing a Valsalva maneuver for clearing.[76] Similar difficulties with ear equalization have been described in children divers.[77] Recommendations from a recent symposium[77] suggest that children who are training for diving have a periodic otoscopic examination that includes evaluation of autoinflation. Care should be taken in examination to evaluate hearing, as a perilymph fistula from diving may result in hearing loss that goes undetected in a child.[78] There are varied opinions regarding exposure to water sports in children with tympanostomy tubes. Current practice is to advise against swimming,[79] however, Cohen et al.,[80] found no increase in otitis media in children swimming with tympanostomy tubes but indicated that that diving should be prohibited. In the study by Salata and Derkay,[81] there was no increase in otitis media in children swimming with tympanostomy tubes, but they advised that diving should not exceed 180 cm below the surface. Thus, children with tympanic membrane perforations or with tympanostomy tubes should not dive due to risk of acute otitis media, however, swimming and other surface exposure to water is not likely to increase the risk for ear infections.

Cardiovascular System

Children with congenital heart disease should be evaluated based on information provided in Chapter 25. Cyanotic heart disease is a contraindication to diving, but some children with surgically corrected defects who have normal cardiac function and normal arterial oxygen saturation could be considered for diving. Some children may exhibit cardiac arrhythmias when diving, particularly in cold water. Both tachyarrhythmias and bradyarrhythmias have been identified in children exposed to water immersion.[82] Profound bradycardias induced by exposure to cold water can produce syncope. Children with a history of neurocardiogenic syncope or with unexplained fainting episodes should be evaluated prior to diving to be certain that this disorder is not aggravated by diving. Children with a history of palpitations should be evaluated by electrocardiography. Those with evidence of preexcitation (Chapter 25), who have a history of arrhythmias, should not dive unless the rhythm is treated. Children may manifest the congenitally prolonged Q-T syndrome with recurrent episodes of syncope.[83] Water exposure appears to aggravate arrhythmias related to the Long Q-T sydrome (see Chapter 25). Batra and Silka[84] described a cardiac arrest in a 12 year old with a known long Q-T syndrome. The child developed ventricular fibrillation after diving into cold water and was returned to sinus rhythm by an implanted defibrillator. Children with a documented long Q-T syndrome should be prohibited from diving. Presence of an implanted defibrillator is a contraindication to diving. When performing a physical examination for diving in children, a family history of sudden death should be sought, as this information suggests that a further evaluation should be done to assess risk for the diver.

Thermal Exposure

Children also can exhibit a rapid loss of body heat due to the higher body surface area relative to metabolic rate. Coupled with a reduced sensitivity to reduced body temperature, children may be more prone to develop clinically significant hypothermia while diving. Proper diving dress should be fitted to the child to avoid heat loss in cold or temperate water, and children should be educated about the symptoms of hypothermia and strategies for minimizing heat loss while diving.

Asthma

Asthma is a common disorder of childhood, and is often related to identified allergens. Children with active asthma should not be approved for diving. Protocols for diving in

children with a past history of asthma can follow recommendation for diving in adults with asthma (see Chapter 24).

Equipment must be properly fitted to the young diver. Wet suits, buoyancy compensators, and compressed air tanks designed for adults will be difficult to manage and may be unsafe for a child of small body habitus. As the age for initial diving training is lowered, small size equipment has become available for use by children. It is not appropriate to outfit a child with diving equipment sized for an adult.

Evaluating the Young Diver

A review of factors to be considered in young divers was presented by Dembert and Keith.[85] Medical guidelines presented throughout this text should be applied to all divers, including young divers. Table 20–2 provides a list of important issues to consider in a child candidate. Physical conditioning is usually of less concern than in the elderly diver but must still be considered. Most children active in recreational or organized sports are in good physical condition. Although poor physical condition is less common in the young, those young divers who are poorly conditioned have increased risk of accident or injury. Dembert and Keith[85] suggest that the young diver weigh at least 45 kg and be 150 cm tall or greater. Medical guidelines are similar to adult guidelines. As in the elderly, the young diver is best trained with a group of peers to avoid stress produced by keeping up with older and more physically capable divers.

In summary, there appear to be no physiological constraints to diving in children below age 15. Physical constraints should be considered in small children (<45 kg or <150 cm tall) because of minimum strength requirements for safe diving. Of greatest concern in the healthy child are the capability to learn and understand the physics and physiology needed for safe diving and the presence of a mature attitude toward safe diving. Training programs for children will accept children as young as 8 years old, but training is limited, and diving is usually limited to a pool or highly controlled open environment. Children above age 10 seem to be better equipped for training and for diving in less constrained environments. Walker[86] provides a thoughtful overview for assessing children who are candidates for diving.

Table 20–2. Factors to considered when evaluating a young subject for diving

Emotional maturity
Ability to understand diving physics and physiology
Ears and sinuses
Normal hearing
Absence of chronic otitis media
Absence of tympanic membrane perforation
Ability to equalize ears and sinuses
Cardiovascular (see Chapter 25)
Absence of congenital heart disease
Normal exercise tolerance
Absence of arrhythmias
No family history of sudden death
No long Q-T syndrome (see Chapter 25)
Respiratory (see Chapter 24)
Evaluation for asthma

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CHAPTER 21 Aseptic Necrosis of Bone

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David H. Elliott

Aseptic necrosis of bone is one of the many terms that has been used to describe the changes seen in the bones of some humans following exposure to increased ambient pressure. Now commonly called *dysbaric osteonecrosis,* this is a potentially crippling condition leading to collapse of a major joint that occurs in compressed-air workers and divers. Aseptic necrosis of bone does not affect every bone in the body or indeed all of any one bone but rather seems to be limited to a few specific and circumscribed sites (Fig. 21–1).

The important sites are those at the proximal ends of the humerus and femur adjacent to the joint surfaces, where lesions are called *juxtaarticular.* The normal load carried at such sites may result in collapse of the dead bone and disruption of the normally smooth bearing surfaces (Fig. 21–2). This in turn can lead to nonspecific compensatory changes in and around the joint surfaces to result in a secondary arthritis. Such a sequence of events results in painful and limited movement of the affected joint. In the early stages of necrosis, prior to collapse of the articular surface, the condition is typically symptom-free and may be first detected in working divers by routine radiographic screening.

Surveillance for the early detection of necrosis in a population of compressed-air workers and divers depends on radiology. Magnetic resonance imaging (MRI) has advantages over x-ray examination in the clinical assessment of suspected or established necrosis, but because of its cost MRI is not suitable for the regular screening of large numbers of asymptomatic persons at risk. The epidemiologic studies conducted on divers and compressed-air workers since the mid-twentieth century or so depended on radiologic diagnosis and have not yet been repeated using MRI. Thus, the radiographs taken in accordance with the Medical Research Council of the United Kingdom (MRC) recommendations, and the diagnoses made using the MRC classification of lesions (discussed later), retain an important medicolegal value alongside the magnetic resonance images in any detailed assessment of this condition.

CAUSES OF OSTEONECROSIS

Osteonecrosis (avascular or aseptic necrosis of bone) is the single common end point of a number of different medical conditions.[1,2] A useful classification uses four groups:

- *Idiopathic necrosis.* Typically, idiopathic necrosis of the femoral head occurs suddenly, with no obvious cause in persons aged 30 to 60. The presenting symptom is pain in the groin, and in 30% the opposite hip may be affected a few months later. However, osteonecrosis in the second hip may begin several years after the first. Because the joint space is not involved at the beginning, the condition is presumed to be a vascular disorder of the head of the femur. This condition is not uncommon, even after excluding those with a history of steroid therapy.
- *Arthropathies* in which necrosis may occur. These include such conditions as rheumatoid arthritis, psoriatic arthritis, and the Charcot joints of late syphilis; these conditions need not be considered further here.
- *Following fracture of the neck of the femur.* This is a condition that should be identified easily.
- *Secondary necrosis,* which is the type found in some persons who have been exposed to raised environmental pressure. Table 21–1 presents some conditions associated with secondary necrosis.

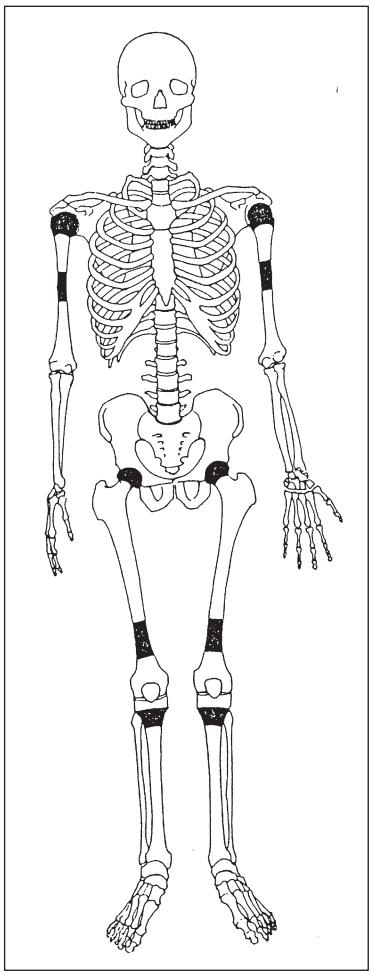

Figure 21–1. Common sites of lesions in divers and compressed-air workers.

CAUSES OF DYSBARIC OSTEONECROSIS

The *bubble hypothesis* of decompression sickness has led to the belief that bone necrosis in divers results from the blockage of some critical nutrient supplying blood vessels to bone by gas bubbles liberated during decompression. However, because there is no absolute correlation between reported attacks of decompression sickness and the subsequent development of bone lesions,[3] several other mechanisms have been suggested to account for bone necrosis in divers. Jones and Sakovich[4] think that fat embolism may be an important factor, and Jones[5] considers that lipids are a factor common to the mechanism of all secondary osteonecrosis. Philp and coworkers[6] think that the blood vessels to bone may be obstructed by platelet thrombi. Hills[7] put forward the unorthodox idea that gas-induced osmosis may be the etiologic agent and that aseptic bone necrosis occurs during compression, as opposed to the generally accepted idea that the decompression is responsible.

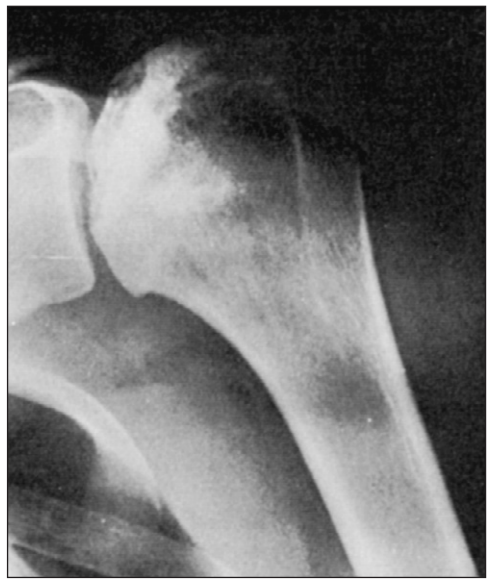

Figure 21–2. Radiographic appearance of a juxtaarticular lesion affecting the head of the left humerus. The articular surface has collapsed.

UNDERLYING PATHOLOGY

Some aspects about the pathology of bone necrosis are illustrated by a comparison of a lesion with the x-ray image. *The only changes in bone that can be seen by radiographic examination are changes in the amount of calcium salts present.* Radiographs taken many years after death may be practically indistinguishable from those obtained on the day of death. Because the amount and distribution of calcium salts in the bone of a living person are seen to change after a bone is damaged, it is clear that some form of circulation must be surrounding the damaged area and perhaps extending into it in places.

When it has been possible to compare the histologic state of a bone with previous radiographs,[8] it has been found, as might be expected from the previous discussion, that the radiograph does not always reveal the full extent of the lesion (Fig. 21–3). When, for example, necrotic lesions affecting the head

Table 21–1. Conditions associated with secondary osteonecrosis

Alcaptonuria	Intravenous drug addiction
Alcoholism	Ionizing radiation
Arteriosclerosis	Liver disease
Decompression illness	Local minor trauma
Diabetes	Pancreatitis
Diffuse lupus erythematosus	Phenylbutazone treatment
Gaucher disease	Pregnancy
Giant cell arteritis	Raynaud disease
Hypercholesterolemia	Rheumatoid arthritis
Hypercorticism	Sickle-cell and other abnormal hemoglobins
Hyperlipidemia (hypertriglyceridemia)	Steroid therapy
Hyperuricemia (and/or gout)	Syphilis

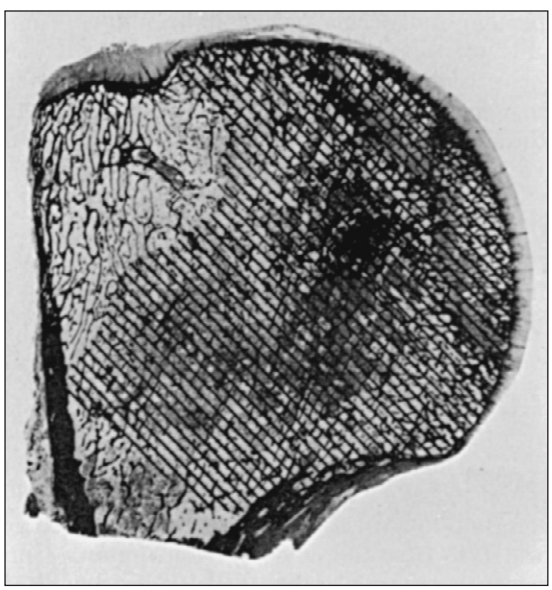

Figure 21–3. Photomicrograph (×2) of the head of the humerus showing aseptic necrosis. Beneath the articular cartilage is an area of necrotic bone bounded by a line of fibrous tissue. The *hatched area* beyond this line shows evidence of dead trabeculae, which have been covered by living bone. This represents the full extent of the original damage.

of the humerus or femur are examined under the microscope, it is found that the original trabeculae no longer contain living osteocytes in their lacunae but that these dead trabeculae have been covered by a layer of new bone containing living osteocytes (Fig. 21–4). This represents the body's attempt to repair the damaged bone, which can occur only in the presence of a satisfactory blood supply.

These changes come about slowly; available evidence suggests that a lesion cannot be detected radiologically until 3 to 4 months after the causal incident.

Histologic examination of the available human material indicates that in addition to the necrosis of bone, fatty marrow necrosis also occurs. Dead fat cells release breakdown products (which stimulate new bone formation[9]) and fatty acids, which combine with calcium to give rise to the diffuse calcified markings typically seen on the radiographs of shaft lesions (Fig. 21–5).

Animal Studies

There can be little doubt that much more would be known about the cause of dysbaric osteonecrosis if it were not so difficult to induce the condition in laboratory animals by simulating diving conditions. This is probably related to the short circulation time, among other factors, in animals smaller than humans, because this would mean that tissue gas tensions would not persist long enough to maintain bubble emboli beyond the time (6 to 12 hours) required for osteocytes to be irretrievably damaged.[10] In addition, the regenerative capacity of bone in small animals is probably better than that in humans.

An interesting approach to the problem has been the intra-arterial injection of spherical glass particles into rabbits to simulate bubble emboli.[11] Lesions of the femoral heads similar to those seen in humans have been produced with this technique. This appears to confirm that the difficulty in producing bone lesions in animals may arise from the short period of persistence of bubble emboli in these species. However,

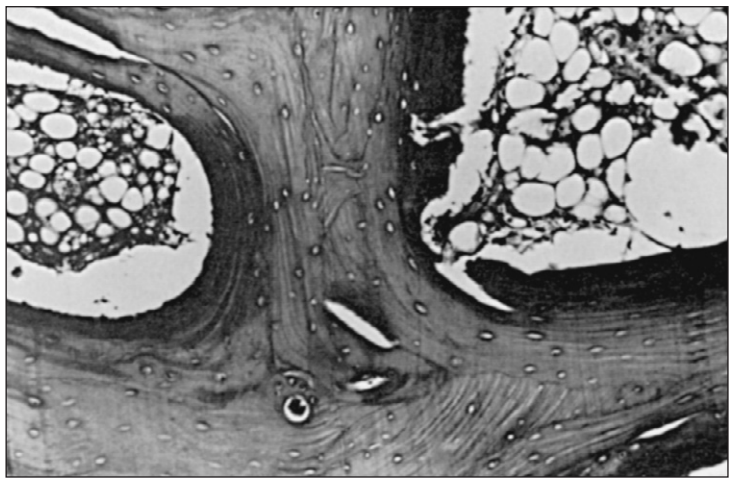

Figure 21–4. Photomicrograph (×125) showing dead trabeculum with empty lacunae onto which a layer of appositional new bone has formed with vital osteocytes in its lacunae.

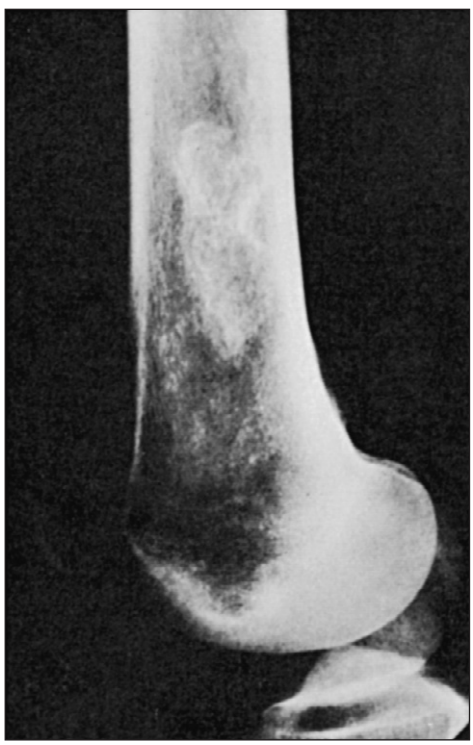

Figure 21–5. Radiographic appearance of a shaft lesion in the lower end of the femur.

there is no evidence that arterial bubble emboli in humans cause dysbaric osteonecrosis.

There have been reports of bone changes in mice[12] and rats[13] after hyperbaric exposure, but the lesions described do not appear to be radiographically identical to those in humans. Commenting on these studies, Behnke[14] wrote, "it is in animals with white fatty marrow in the long bones such as the pig and the cow (certainly not the red marrowed rodent) which are likely to yield the answer." Lanphier and colleagues[15] reported on experimentally produced hyperbaric osteonecrosis in the sheep (an animal that has white fatty marrow in its long bones). However, the initiating insult (rapid decompression) after prolonged exposure to pressure was so severe that the mechanism involved may not be the same as that in humans.

Walder[16] pointed out that the blood flow in fatty marrow is drained by way of venous sinusoids and that these, if distended, give rise to a dull, aching pain. (Such distention is the explanation for the pain of osteoarthritis, which is relieved when orthopedic surgeons perform osteotomy.) It is suggested that distention of venous sinusoids by bubbles is the explanation for the pain of decompression sickness and that the growth of such bubbles impedes the circulation through fat cells, giving rise to necrosis and the release of breakdown products. These products, in turn, stimulate new bone formation and damage the surrounding trabeculae, producing the typical appearance of dysbaric osteonecrosis seen on a radiograph.[17] One further possible factor that until recently has been largely overlooked is the raised partial pressure of oxygen to which divers are exposed. It has been demonstrated that fat cells increase in volume when subjected to a raised partial pressure of oxygen.[18] This would embarrass the circulation of such cells because they are confined to bony trabecular compartments within the marrow space. These findings support the hypothesis that although the decisive factor that results in bone death may be a bubble or some associated disturbance of lipid metab-

olism, some fat cells are already compromised by a diminished circulation brought about by a diver's exposure to a raised partial pressure of oxygen for some hours.

DIAGNOSIS

Clinical

The earliest cases in the literature are those of patients who presented with pain in a hip or shoulder joint, often of sudden onset and probably related to the collapse of subchondral bone. Since the introduction of health surveillance for compressed-air workers and divers in whom the diagnosis is still radiologic, the diagnosis is early and no longer clinical. However, with the introduction of deeper and more prolonged recreational diving and with the diminution of routine bone radiographs in commercial divers because of cost, a return to clinical diagnosis can be anticipated.

The latency of radiologic and clinical lesions is a severe disadvantage when trying to attribute the subsequent damage or disability to a particular dive or other exposure to raised environmental pressure. MRI might be the ideal answer but is not yet sufficiently available to have been evaluated as a screening tool in healthy workers.

Radiological Classification

To standardize the systems used by different centers to describe the radiographic appearances of bone lesions seen in compressed-air workers and divers, the MRC Decompression Sickness Panel in the United Kingdom drew up a classification for juxtaarticular and shaft lesions (Table 21–2) that has proved to be extremely useful.[19] To assist radiologists in recognizing the various lesions mentioned in the classification, a limited number of radiologic atlases were distributed from the MRC Decompression Sickness Team in Newcastle upon Tyne. Many of the radiographs from these atlases have been reproduced by Ilford[20] and can also be found in a book by Davidson.[21]

As mentioned earlier, with the passage of time, symptomless juxtaarticular lesions may progress to structural failure, with associated pain and limitation of movement, and then may develop osteoarthritic changes over the years. Furthermore, it is not yet possible to use the radiologic appearance to predict whether a specific early juxtaarticular lesion will progress to collapse of the articular surface in a few months, as illustrated in Figure 21–2, or whether it will, like the majority of lesions, remain stable for many years and cause no further involvement of the bone or any signs or symptoms. Some predictions as to the probability of the progression to collapse have been made using the different radiologic categories,[22] but in general it is considered that some 10% of persons with an early juxtaarticular lesion experience progression to collapse and that some 10% of this group will need surgery for a joint replacement. It is therefore necessary to keep a lifetime record of bone lesions and occupational exposures of an individual. Care must be taken to prevent the premature destruction of these radiographs for silver reclamation. Conversion to high-resolution digital images would be adequate for long-term image archiving.

In addition to juxtaarticular lesions, some lesions occur away from the articular surface, either deep in the head or neck of a bone or in its shaft. These are called *head, neck,* and *shaft lesions.* These are most frequently found at the lower end of the femur and at the upper end of the tibia.

For accurate diagnosis, one must exclude bone islands. These appear radiographically as an isolated area of increased bone density. Bone islands are usually round or oval, the longer diameter varying between 2 and 15 mm, and the margins may be well defined or irregular and indistinct. In 1973, Conti and Sciarli[23] suggested that bone islands might

Table 21–2. Classification of bone necrosis in compressed-air workers and divers

Juxtaarticular Lesions
A1. Dense areas with intact articular cortex
A2. Spherical segmental opacities
A3. Linear opacity
A4. Structural failure
a. Translucent subcortical band
b. Collapse of articular cortex
c. Sequestration of cortex
A5. Osteoarthritis
Head, Neck, and Shaft Lesions
B1. Dense areas
B2. Irregular calcified areas
B3. Translucent areas

be more common in divers than in nondivers, and indeed it is possible that some authors have classified them as bone lesions. Later, Davidson and colleagues,[24] reviewing the radiographs of 100 Royal Navy divers and the radiographs of 100 nondiving Navy personnel matched for age and rank, were unable to substantiate this finding and said that bone islands are no more common in divers than in nondivers. The areas compared were those normally radiographed according to the MRC Panel's recommendations for diver bone surveys. In the past, radiologists have correctly dismissed the presence of bone islands as insignificant, and hence bone islands have usually not been addressed in routine radiologic reports.

Other Imaging Techniques

Occasionally, the radiographic appearance of a diver's bones gives rise to the suspicion of a lesion, but the evidence is not conclusive. In such cases, MRI may resolve the doubt. If a suspected lesion is accompanied by pain on movement, there may well be a breakdown of the articular surface continuity, which may be revealed only by MRI or tomography. Trochanteric bone biopsy and bone phlebography may be considered.[25]

Other diagnostic techniques that have been employed in asymptomatic persons at risk are those using bone-seeking radioactive isotopes. These have been used successfully in the detection of other bone abnormalities,[26] and there is some evidence of their potential value in the early diagnosis of aseptic necrosis of bone.[27] However, one difficulty is that these methods appear to be almost too sensitive and may indicate lesions that spontaneously heal and never progress to detectable radiographic change or indeed to cause any disability.[28]

More experience is required before techniques other than simple radiography can be adopted universally as alternatives for the routine surveillance of divers.[29]

DIFFERENTIAL DIAGNOSIS

In addition to hyperbaric exposure, many conditions can lead to aseptic necrosis of bone in humans, but most of these conditions can easily be diagnosed and in any case are incompatible with fitness to dive. The presence of chronic alcoholism in healthy divers with bone lesions is rather more difficult to establish, but the evidence is that alcoholism is a significant factor only if severe and prolonged. Matsuo and colleagues[30] showed that the relative risk increases in proportion to consumption in excess of the equivalent of 400 mL of alcohol per week, particularly over some years.[31]

Hyperlipidemia is a complex condition and one of the few alternative causes of necrosis that can be found in apparently healthy divers.[5] Hypertriglyceridemia is the variety most commonly associated with necrosis and may be familial, but the underlying concern is that the pathogenesis of osteonecrosis at the cellular level appears to be the same for virtually all the different causes of secondary necrosis, including raised environmental pressure. Thus, there is a possibility of synergism between hyperlipidemia, alcohol, and diving that may enhance what would otherwise be a low risk.

PREVALENCE OF BONE LESIONS

The problem of aseptic necrosis of bone as it affects divers must be put into perspective. There is no doubt that persons exposed to pressurized environments, such as civil engineers and compressed-air workers and divers, run a risk of bone damage. In one study,[32] a bone radiographic survey was carried out in two groups of manual laborers employed in tunneling work. One group worked in compressed air many times over a period of years, whereas the other group did similar jobs at atmospheric pressure. The prevalence of bone lesions in the 171 compressed-air workers eventually reached 26%, but none of the 120 members of the other group experienced a lesion.

Reports of the prevalence of bone necrosis in divers have differed widely. Ohta and Matsunaga[33] reported a prevalence as high as 50% in Japanese diving fishermen. Surveys by Elliott and Harrison of Royal Navy clearance divers[34] showed an overall figure of 5%, although most of the lesions were found in divers who had engaged in experimental diving (dives in which the decompression schedules used were not of proven adequacy). In 1981, the MRC Decompression Sickness Registry in Newcastle upon Tyne

published the statistics for the 4980 North Sea commercial divers who underwent radiography in the United Kingdom up to that time.[35] Bone necrosis was present in 4.2% of the divers, and the figure remained at that level until the end of 1984, when funding was discontinued by the Health and Safety Executive and the Registry closed.

The lesions of aseptic necrosis of bone are found at the same sites in both compressed-air workers and compressed-air divers (see Fig. 21–1), but the frequency with which the sites are affected seems to differ from one report to another. In the British experience, the most common site for lesions is the lower end of the femur, followed by the shoulder joint. The hip joint is rarely affected in divers, although it is commonly involved in compressed-air workers. Such observations have led to speculation about the reasons for this distribution and whether it can provide some additional clue about the cause of aseptic necrosis of bone.[3] Unfortunately, the Japanese experience with divers[36] is more like that of the British compressed-air workers, with lesions in the head of the femur being common.

SURVEILLANCE FOR OSTEONECROSIS IN DIVERS

The radiation exposure associated with every radiographic examination must be kept to an acceptable limit. Attitudes toward the dangers of ionizing radiation have changed considerably in recent years. It must be remembered that the mean effective dose equivalent associated with a diver's long-bone survey is between 1 and 2 milli-Sieverts, depending on the technique and equipment used.[37] The lifetime fatality probability coefficient for all malignancies arising from exposure to ionizing radiation is estimated to be 0.04 according to Sievert,[38] so that the probability of an individual diver's developing a fatal malignancy after a single radiologic long-bone survey is 0.00008. Although the risk of fatal cancer is miniscule, it is a risk that should be minimized when considering radiologic studies.

In an effort to keep the radiation hazard of the long-bone surveys to a minimum, it has been suggested in the United Kingdom that knee radiographs be eliminated because lesions in this region rarely, if ever, cause symptoms. Such radiography is also a cost burden for the self-employed diver. The procedure for full radiographic examination for those at risk is shown in Table 21–3.

The Registry has no record of aseptic necrosis of bone occurring in working divers who never dived deeper than 30 m. It therefore does not seem justifiable to carry out routine radiographic studies on amateur and sport divers if they do not dive below 30 m, even though a few cases have been reported.[39,40] The prevalence of bone necrosis in nonsaturation air diving to depths of up to 50 m is very low (0.8%). The current recommendation in the United Kingdom is that long-bone radiographs be required before undertaking saturation diving and thereafter on clinical indication or at specific

Table 21–3. Radiography of the bones of compressed-air workers and divers

1. Good definition of the trabecular structure of the bone is essential.
2. The gonads must be protected from ionizing radiation by the use of a lead shield.
3. Projections required:
   a. *Anteroposterior projection of each shoulder joint.*
      Place the patient in a supine position with the trunk rotated at an angle of approximately 45° to bring the shoulder to be radiographed in contact with the table. Partially abduct the arm and flex the elbow. Center 1 inch below the coracoid process of the scapular and cone to show as much humerus as possible. Bring the lateral diaphragm to show only the head and shaft of the humerus.
   b. *Anteroposterior projection of each hip joint.*
      Place the patient in a supine position with the feet at an angle of 90º to the tabletop. The edge of the gonad protector should be as near the femoral head as possible but not in any way obscuring it. Center the cone over the head of the femur, that is, 1 inch below the midpoint of a line joining the anterior superior iliac spine and the upper border of the pubic symphysis.
   c. *Anteroposterior and lateral projections of each knee.*
      Center the cone at the level of the upper border of the patella. The field should include the lower one third of the femur and the upper one third of the tibia and fibula.

request. This does not seem adequate because bone necrosis is not confined to saturation divers. Although a baseline radiographic survey may not be needed for divers in low-risk categories, such as police divers and most recreational instructors, such a survey is indicated in (and should be placed on file for) all working divers whose duties take them regularly below 30 m. The radiographs should be repeated at 1- to 3-year intervals for those who continue to engage in saturation diving; more frequent monitoring is justified in some other circumstances. For those who may be considered to be at special risk after an unusual episode of decompression sickness or after a difficult and extended recompression, there is merit in undergoing *negative* technetium scanning or MRI not sooner than 6 weeks after recompression. Any positive results would need to be followed up for the detection of early radiographic change; should such a change develop, orthopedic management would be made available at the earliest opportunity.

ADVICE

What advice should the doctor give to a diver found to have a bone lesion? This is difficult to answer because factual evidence is still limited. Certainly, juxtaarticular and shaft lesions should be considered separately.

At the moment, it seems reasonable to take the view that, although a shaft lesion represents a failure to protect the diver from the consequences of diving, shaft lesions almost never result in disability, so that the patient may continue to dive. Some caution is necessary: Mirra and coworkers[41] and Kitano[42] have suggested that neoplastic changes have occurred in some shaft lesions of compressed-air workers, although the risk, as currently known, is very low. Divers with shaft lesions should therefore be informed that the lesions exist and should be advised to report to their physicians should any symptoms ever arise in the affected limb. This possibility will have to be monitored very carefully, but there is still no reported case of malignancy in a diver.

If the lesion is juxtaarticular, the situation is quite different. Every juxtaarticular lesion is potentially disabling because there is a 10% to 40% chance that the articular surface will progress to collapse.[22] Even if the potential disability of one major joint were acceptable because it would not compromise in-water safety, the risk that a continuation of diving might result in a lesion in a second major joint cannot be excluded. For these reasons, it seems sensible to consider a juxtaarticular lesion to be a contraindication to future diving. Although the possibility of a further lesion might be reduced by limiting the diving to depths of less than 30 m, the risk of aggravation remains. In any case, it appears that, at present, such a limitation is impractical for working divers because of the need to dive without restriction when required.

TREATMENT

No treatment is indicated for shaft lesions because they are not expected to produce symptoms or to result in disability. The treatment of aseptic necrosis of bone at juxtaarticular sites is not yet satisfactory.

Orthopedic surgeons apply a general principle of treatment to damaged joints before the articular surface has collapsed: Relieve the affected joint of weight bearing in order to give the underlying bone an opportunity to heal. In the case of aseptic necrosis of bone in divers, this means a period of rest lasting for several months. Unfortunately, at present there is no way of determining which lesions will break down and which will never cause pain or limitation of movement. In general, therefore, conservative treatment is neither practicable nor satisfactory.[43]

Attempts to treat advanced lesions in which the articular surface has already become disrupted have included the following:

- Inserting a bone graft via a drill hole through the underlying living bone into the area of dead bone in order to provide a pathway for revascularization[44]
- Gouging out the necrotic bone from beneath the cartilage and packing the cavity with fresh cancellous bone chips[45]
- Realigning the shaft of the bone to change the line of weight bearing, as in a McMurray osteotomy

None of these methods has met with great success.

The most satisfactory method of treating the seriously affected joint may be to subject the joint to arthrodesis or to replace the damaged head of the bone with a prosthesis. Although the use of prostheses is well estab-

lished for middle-aged and older patients, questions about the durability of the prosthesis become of great importance when it is to be used in the treatment of active young persons such as divers.

Persistent or recurring bone or joint pain should not be ignored in a commercial diver or in a sport diver exposed to depths greater than 50 m (i.e., a technical diver). Radiographic evaluation may be diagnostic, but in the absence of radiographic findings, if aseptic necrosis is suspected, MRI may be needed to confirm the diagnosis.

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CHAPTER 22 Ear and Sinus Problems in Diving

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Injuries to the ears and paranasal sinuses are the most common problems produced by exposure to altered barometric pressures. The air-filled middle ear spaces and sinus cavities can become a liability for divers who cannot attain proper pressure equilibration. The air-containing organs of hearing and balance and the paranasal sinuses are sensitive to minute variations in ambient pressure and gas mixtures. The rapid and often uncompensated pressure changes encountered in diving and exposure to varied gas mixtures can present significant otolaryngologic problems. The incidence of exposure to hyperbaric conditions and ear and sinus injury is increasing with greater participation in sport scuba diving and increased use of hyperbaric chambers for medical treatment. Most ear and sinus injury is readily apparent, and early, proper assessment and treatment are often needed before specialist assistance is possible. This chapter provides diving medical personnel with the tools to correctly triage and appropriately treat the injured diver.

HISTORICAL CONSIDERATIONS OF DIVING OTOLARYNGOLOGY/ OTOLOGY

Severe deafness and vestibular problems were reported among other ailments by A. H. Smith in 1873[1] as part of a description of "caisson disease" experienced by compressed-air workers. Injury to the middle and inner ears during compression and decompression in caisson workers at Nussdorf was reported by Alt and Heller in 1897.[2–4] Citing their subsequent investigations in animals, they were the first to suggest that inner ear injuries in diving could occur during compression, in association with inadequate middle ear pressure equilibration, and during decompression, during which injuries were thought to be secondary to interference with the inner ear blood supply by nitrogen bubbles developing in the labyrinthine vasculature. Caisson worker deafness was later reported by Lester and Gomez[5] and Boot.[6] In 1929, Vail[7] expanded our knowledge of the mechanism of ear injury with animal studies that indicated that the inner ear damage occurring during compression was related to inadequate middle ear pressure equilibration, with resulting stasis and hemorrhage in the inner ear. Vail also shared Alt's hypothesis that injuries during decompression were related to nitrogen bubbles causing emboli and necrosis in the inner ear.

By the 1940s, diving-related inner ear injuries were reported infrequently, with the decrease in incidence attributed to improved safety and decompression schedules. During World War II, the increase of diving operations was reflected in literature devoted to the prevention and treatment of barotitis media, which was generally thought to be a reversible process with no serious consequence or disability. When symptoms suggestive of inner ear injuries were described, they were frequently attributed to central nervous system decompression sickness. Shilling and colleagues[8] concluded that the high-frequency sensorineural hearing loss in divers was related both to repeated episodes of barotitis media and to noise exposure; however, many authors from 1945 to 1961 noted sensorineural hearing losses and attributed the deficits to nondiving diseases or excessive noise trauma.[9–12]

As activity in commercial, military, and sport diving to deeper depths increased, reports of diving-related ear problems became

more frequent. Middle ear barotrauma during descent continued to be the most common diving medical problem encountered, but reports of inner ear disturbances with some permanent sequelae during all types and phases of diving appeared with increasing frequency. The book *Otological Aspects of Diving*[13] appeared in 1973 and contained a detailed review of the various causes of otologic problems in diving. In 1974, Kennedy[14] published a review of the literature that summarized the vertigo and disequilibrium that occurred during diving and suggested that vestibular injury incurred while diving was more common than previously suspected. Lundgren[15] in 1965 and Ingelstedt and colleagues[16] in 1974 described and demonstrated inadequate middle ear pressure equilibration and subsequent vertigo during ascent.

In the 1970s, the first writings since the early works by Alt and Vail began to emphasize inner ear injuries in diving. Freeman and Edmonds[17,18] described labyrinthine window ruptures and inner ear injury associated with inadequate middle ear pressure equilibration during the compression phase of shallow diving. Stucker and Echols echoed the earlier works of Alt and Vail and suggested that inner ear injuries during diving could occur from nitrogen bubble emboli forming in the internal auditory artery system during decompression.[19] Ten cases of isolated vestibular or cochlear injuries occurring during or shortly after decompression were described in the same year in a paper by Rubenstein and Summitt.[20] In 1976, Farmer and coworkers[21] enlarged on the 10 cases of Rubenstein and Summitt and presented an additional 13 cases of isolated vestibular or cochlear injuries occurring during or shortly after decompression. Buhlmann and Gehring[22] described additional instances of otologic injury in humans related to decompression from deep helium/oxygen diving. This increase in inner ear disease associated with diving sparked review of specific recommendations for the management of inner ear decompression sickness.

Animal studies by McCormick and colleagues[23] in 1973 demonstrated intralabyrinthine bubble formations and hemorrhages along with decreases in cochlear function in guinea pigs subjected to rapid decompression. In 1977, Landolt and associates[24] described vestibular dysfunction and inner ear pathology in monkeys after rapid decompression. Further studies by Venter and coworkers in 1983,[25] enlarging on the work of Landolt, demonstrated actual fractures of the bony endosteal layers of the semicircular canals in the inner ears of monkeys with inner ear decompression sickness. In 1975, Lambertsen and Idicula[26] described inner ear vestibular dysfunction and injury occurring in divers while at stable deep depths soon after beginning the breathing of different inert gases. This was described as a manifestation of the counterdiffusion phenomenon.

In 1977, Farmer[27] reviewed diving inner ear injuries and pointed out that the pathophysiology and treatment of these problems differed with the phase of diving in which the injuries occurred. Persistent inner ear injuries were classified into (1) injuries occurring during compression (inner ear barotrauma), (2) injuries occurring at stable deep depths, (3) inner ear injuries related to decompression sickness, and (4) sensorineural hearing loss secondary to excessive noise exposure in diving.

Over the last 100 years, consideration of otologic problems in diving has increased as the incidence of life-threatening injury in diving has decreased. Since the 1950s, the tools available to differentiate true vertigo and hearing loss have improved markedly. Diagnostic studies, although helpful, do not supersede a thorough history and physical examination, which can provide important information regarding eventual treatment of the injured diver.

RELATED ANATOMY AND PHYSIOLOGY

A complete review of the anatomy and physiology of the ear, nose, and sinus is beyond the scope of this chapter; however, certain points should be made to better understand otologic and paranasal sinus problems in diving.

The external ear and ear canal serve to capture and direct sound waves toward the middle ear. The external auditory canal (Fig. 22–1) is a self-cleaning blind tube lined by squamous epithelium that is continuous with the squamous epithelium on the outer layer of the tympanic membrane. The keratinized epithelial cells are constantly migrating from the eardrum into bony ear canal and then outward to the cartilaginous canal, or outer one third, where they are mixed with *cerumen,* a colorless, odorless material containing long- and short-chain fatty acids. The water- and fat-soluble fatty acids in

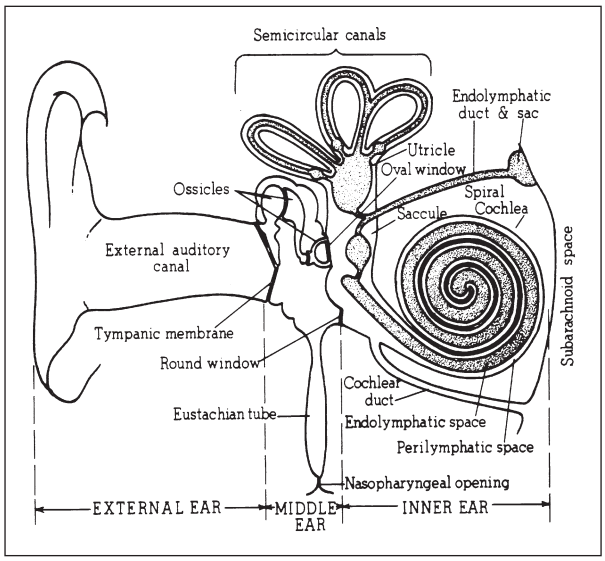

Figure 22–1. Simplified diagram of the external, middle, and inner ear showing air within the external auditory canal, middle ear, and eustachian tube. The fluid-filled inner ear is subdivided into the perilymphatic and endolymphatic spaces, which connect indirectly with the subarachnoid space via the cochlear duct and endolymphatic duct and sac, respectively.

cerumen have a protective function, that of primarily maintaining a slightly acid pH that is bacteriostatic and prevents the epithelium from becoming waterlogged and prone to infection. When cerumen is exposed to air, oxidation and the typical brown color occur.

The middle ear cleft is an irregularly shaped space that communicates with air cells in the mastoid, petrous, and zygomatic portions of the temporal bone. The total gas volume of this complex varies with the pneumatization of these areas. The evolution of an air-containing external and middle ear has presented humans with a device that efficiently transforms airborne sound into the fluid-filled inner ear, where it is transduced into electrical signals. Proper function of this mechanism requires that the external ear canal be patent, that both the external ear canal and the middle ear contain air, and that pressure differentials between these structures and the ambient atmosphere, as well as the inner ear, be avoided.

The pressure-sensitive middle ear can become a liability with the pressure changes encountered in diving. With an intact tympanic membrane, the only communication for pressure equilibration between the middle ear cleft and the ambient atmosphere is through the eustachian tube. This tube is approximately 35 to 38 mm in length in the adult and is directed downward, forward, and medially from the middle ear to the nasopharynx. The nasopharyngeal ostium is normally closed except when opened by a positive middle ear pressure or when opened by the muscular action of the pharyngeal and palatine muscles upon the surrounding tubal cartilage during swallowing. During descent, active attempts must be made to open this ostium by contracting these muscles; the ostium and tube usually open passively during ascent.

The inner ear (see Fig. 22–1) consists of a system of perilymph-filled bony channels within the temporal bone. Membranous structures containing endolymph are located in these channels. Perilymph is biochemically similar to extracellular fluid, whereas endolymph is biochemically similar to intracellular fluid. A resting electrical charge exists between perilymph and endolymph. When acoustical energy enters the cochlea, the basilar membrane is displaced and the electrical charge depolarizes with activation of the neural auditory pathways.

The membranous inner ear structures are divided into two parts: the *vestibular system*, containing the semicircular canal, utricle, and saccule; and the *auditory system*, containing the spiral cochlea. These two systems are interconnected and are separated by the thinnest membranes (two cell layers) in the body. The blood supply to both systems is through the internal auditory artery, which originates from the basilar or the inferior cerebellar artery. This is an end artery that supplies only the membranous inner ear. Alterations in cerebrospinal fluid (CSF) pressure are transmitted to both the endolymph and perilymph fluid compartments, and significant pressure differences between these spaces are usually avoided.

Any maneuver that increases CSF pressure, such as a Valsalva maneuver, can cause increased pressure in the inner ear fluid compartments, with bulging of the round window membrane into the middle ear. With marked pressure changes, possible round window rupture or rupture of inner ear membranes, or both, can occur during shallow or deep exposures (see later).

Respiratory epithelium has a rich vascular supply and lines the eustachian tube, the nasopharynx, the nose, and the paranasal sinuses. This epithelium is constantly secreting a mucous blanket, which is moved into the nasal cavity through the sinus ostia by the beating of the microscopic cilia on the mucosal cell surface. Once in the nasal cavity, this mucus is combined with the mucus secreted in the nose and is swept by ciliary action to the nasopharynx. A healthy adult secretes about 1 L of mucus per day; the inspired and expired air stream evaporates about half of this. The mucous blanket has cleansing, filtrating, bacteriostatic, and protective functions.

Alterations in these functions and obstruction of the airways and sinus ostia can result from chronic inflammatory disease, which is commonly due to one or more of the following underlying factors: allergy, chronic irritation from smoking, prolonged use of nose drops, and chronic obstruction from internal or external nasal deformities or from mass lesions. Frequently, acute or chronic nasal and sinus infections are due to the congestion and airway obstruction from one or more of these underlying factors or the physiologic nasal congestion, increased mucous discharge, and drying effects by breathing cold, dry air. Inflammatory nasal and sinus diseases can result in inadequate eustachian tubal function and otitis media in the absence of atmospheric pressure changes; with diving, such disease can result in an increased likelihood of middle ear or even inner ear barotrauma as well as barotrauma to the paranasal sinuses.

SYMPTOMS OF OTOLOGIC DYSFUNCTION

The common symptoms of otologic dysfunction are ear fullness or pain, hearing loss, tinnitus, and vertigo. A complete discussion of each of these symptoms is beyond the scope of this chapter; however, a brief review is in order.

Ear Fullness and Pain

Ear fullness, or the sensation of a blocked ear, commonly occurs from occlusion of the external auditory canal or from high or low middle ear pressure relative to ambient pressure. The resultant tensing of the eardrum and increased ossicular chain impedance cause a decrease in sound transmission to the inner ear. The patient feels that the ear has become occluded. Ear fullness can also occur with the collection of fluid or blood in the middle ear space, which can result in a decrease in the ability of sound to conduct through the middle ear transformer, thus resulting in a conductive hearing loss.

Pain occurs from sensory pain receptors in the eardrum and middle ear mucosa with marked pressure differentials across the tympanic membrane. Increased pain is felt with eardrum rupture. The inflammation and swelling of the external ear canal in otitis externa also presents as pain. Referred pain to the ear is also common with inflammation or lesions in the nose, hypopharynx, nasopharynx, teeth, maxillary sinuses, and temporomandibular joint.

Hearing Loss

Hearing loss is classified into three types: conductive, sensorineural, and mixed.

CONDUCTIVE HEARING LOSS

Conductive hearing loss results from dysfunction of any component of the sound conduction system, i.e., the external auditory canal or the middle ear transformer (the eardrum or ossicular chain), or both. Complete airtight occlusion of the external auditory canal, such as from a cerumen plug, causes a conductive hearing loss. Partial occlusion or non-airtight seals of the canal usually do not result in hearing loss unless the occluding material lies against the eardrum and impedes vibration. Conductive hearing loss can also occur from any process that interferes with the transmission of sound energy into the inner ear or impedes the movement of the eardrum and ossicles.

Such processes can include inflammation and swelling of the eardrum or middle ear mucosa; middle ear effusion or exudates; changes in middle ear gas density, such as occurs in nontraumatic hyperbaric exposures; pressure differentials across the eardrum; fixation (otosclerosis) or dislocation of the ossicles; loss of elasticity of the eardrum and ossicular fixation from scarring or repeated infections; and large eardrum perforations. Conductive hearing loss is commonly experienced with middle ear barotrauma.

SENSORINEURAL OR NERVE DEAFNESS

Sensorineural or nerve deafness results from dysfunction in the inner ear, auditory nerves, or brainstem cochlear nuclei. Such dysfunction can result from:

- Occlusion of the cochlear blood supply with ischemia
- Mechanical disruption of inner ear or brain stem structures from trauma or bubbles
- Leakage of perilymph from round window rupture with inner ear membrane breaks
- Idiopathic hydrops, or excess fluid pressure in the endolymphatic space (Meniere disease)
- Inflammatory disease in the inner ear (labyrinthitis)
- Autoimmune inner ear diseases
- Idiopathic degenerative processes such as presbycusis
- Trauma-induced degeneration of cochlear structures from excessive noise exposure

MIXED OR COMBINED CONDUCTIVE SENSORINEURAL HEARING LOSSES

A combination loss results from simultaneous dysfunction in the middle and inner ear. This can occur in coexisting middle and inner ear barotrauma, middle and inner ear otosclerosis, or the development of acute middle or inner ear dysfunction with preexisting disease in the other area.

EVALUATION OF HEARING LOSS

Determination of the type of hearing loss is essential in the evaluation and management of any patient with suspected otologic dysfunction. This is best accomplished with soundproof-booth audiometry by a certified audiologist. However, in some instances, such testing is not available or is impractical, and some preliminary information can be gained by testing with a 512 Hz or 1024 Hz tuning fork in quiet surroundings. A 256 Hz fork can be used, but the examiner has to be careful that the patient does not respond to vibratory sensations, which are more easily perceived at lower frequencies.

Weber Test

The struck tuning fork is placed on the forehead or on the upper incisor teeth, and the patient is asked if the sound is louder in either ear or if it is of the same intensity in both ears. With a conductive hearing loss, a sound source placed on either of these midline skull locations will be heard louder in the affected ear; with a sensorineural hearing loss, such sounds will be heard louder in the unaffected ear. With equal hearing in both ears, i.e., normal hearing or bilaterally equal hearing losses, the sound will not lateralize.

Rinne Test

A vibrating tuning fork is alternately placed against the patient's mastoid tip and then held about 2 inches from the ear canal. The patient is asked to ascertain the position in which the sound is louder or heard longer. In a normal-hearing ear, or in an ear with a pure sensorineural hearing loss, bone-conducted sound will be heard less loudly and for a shorter time than air-conducted sound. This phenomenon is due to the enhancement of airborne sound by the middle ear transformer, i.e., the eardrum and ossicular chain. With a moderate or severe conductive hearing loss, bone-conducted sound will become equal to or louder than air-conducted sound, depending on the degree of loss. With mild conductive hearing losses, normal results can be obtained.

Schwabach Test

Examiners should know their own hearing threshold; the examiner first places a vibrating tuning fork on the patient's mastoid tip. At the precise moment the patient no longer hears the sound, the fork is placed on the examiner's mastoid tip. If the examiner then hears the sound, decreased bone conduction

or a nerve hearing loss in the patient's tested ear is suggested.

Watch Test

The frequency of a wristwatch alarm or ticking ranges from 4000 to 6000 Hz. The watch can be used in quiet surroundings to compare the perception of sound between the examiner's and the diver's ears. This is a rough estimation of hearing function, and examiners must know their own hearing threshold.

In general, tuning fork tests are difficult to perform adequately, particularly by untrained or inexperienced examiners. Patient suggestibility, decreased alertness, discomfort, and excessive background noise can adversely affect the results. Also, the presence of mixed hearing losses, unilateral or bilateral, can hamper interpretation of the results. Adequate audiometry by certified audiologists should be obtained as soon as possible to supplement and confirm the results of tuning fork testing. Persons who dive regularly should undergo routine periodic audiometry to detect unnoticed hearing losses and to provide baseline data for future reference.

Tinnitus

Tinnitus, or spontaneous noise in the ear, is difficult to quantitate because different patients experience different types of noise. Also, the perception of severity and the effect on daily life vary. Tinnitus can occur with middle ear disease that results in a conductive hearing loss but usually occurs with inner ear or central auditory pathway disease. With the former, tinnitus is thought to represent the sounds of cochlear and intracranial blood flow that are perceived because the conductive hearing loss results in a loss or a decrease of the masking effect of the usual background noise. Patients may describe a rushing, pulsatile sound that can often be timed with the pulse.

With inner ear disease, tinnitus is thought to be due to the spontaneous firing of injured but viable auditory neurons or cochlear hair cells. However, this is not well understood: Destructive labyrinthectomies or 8th cranial nerve sections in patients with recurrent and disabling vertigo, tinnitus, and nonfunctional hearing due to Meniere disease—which usually relieve the vertigo—have frequently not relieved the tinnitus.

Vertigo

The central nervous system is programmed during the first year of life to associate unequal firing rates between the right and left vestibular end-organs, primary vestibular neurons, and brain stem vestibular nuclei with linear and angular acceleration. These acceleration inputs are integrated with visual and proprioceptive information, which is then translated into signals to limb musculature to maintain balance. When sudden unilateral disease affects the vestibular structures without corresponding alterations in visual and proprioceptive inputs, the cerebral cortex interprets this neural input mismatch as vertigo with rotation, pitching, yawing, or rolling.[28] This type of dizziness must be differentiated from other, less specific symptoms of balance disturbances, such as lightheadedness, unsteadiness, and presyncopal sensations. If a dizzy patient does not have vertigo, then the dizziness is unlikely to be related to primary or secondary vestibular disease. Exceptions to this include persons with slowly expanding intracranial lesions such as acoustic neuromas, who usually describe continuous, progressive unsteadiness with no or brief vertiginous sensations. Another exception is the patient with bilateral vestibular end-organ disease such as that caused by ototoxic drug exposures, who usually have nonvertiginous unsteadiness that can become severe with the loss of a second balance system input, such as decreased vision in dark surroundings or decreased proprioception with peripheral neuropathy.

When vertigo is present, adequate evaluations must be performed to differentiate end-organ from central vestibular dysfunction and to properly determine whether such affected persons are suited for further diving after apparent recovery. This involves a systematic approach to the evaluation and management of dizzy patients. To develop this type of approach, several general points should be emphasized.

SYMPTOM CHARACTERISTICS

The first distinction is whether a dizzy individual is experiencing nonvestibular dizziness or true vestibular vertigo, defined as a specific alteration of spatial orientation

involving the sensation of motion, usually rotary, of either the subject or the environment. If the dizzy person does not have vertigo, the dizziness is unlikely to be related to primary or secondary vestibular system dysfunction, either in the peripheral end-organ or in the central vestibular pathways. However, some persons find it difficult to describe their dizziness. Nausea, vomiting, visual disturbances, presyncope, or other symptoms frequently accompany inner ear dysfunction. Thus, the presence of these symptoms does not necessarily mean a more extensive central nervous system injury.

PAST MEDICAL HISTORY/MEDICATIONS

Most human maladies and many medications are associated with dizziness. Consideration of cardiac, vascular, endocrine, psychogenic, and neurologic medications and other medical conditions is paramount for the differential diagnosis of dizziness.

PHYSICAL FINDINGS

Vestibular dysfunction usually is accompanied by classic nystagmus, with a defined quick and slow component. If by visual observation and by electronystagmography a dizzy patient does not have such accompanying nystagmus, the dizziness is unlikely to be due to vestibular system dysfunction.

Nystagmus resulting from nonacute end-organ vestibular dysfunction is frequently suppressed by visual fixation and, therefore, is not observable. Thus, electrical recordings of ocular motion in the dark or with the eyes closed, i.e., electronystagmography, is important in the evaluation of a dizzy patient.

Once it has been determined that dizziness is likely due to vestibular system dysfunction, the next distinction is whether the disease is located in the end-organ or in the central vestibular system. In some cases, this determination is not difficult because other accompanying neurologic symptoms or signs point to a centrally located lesion. However, in many cases, such accompanying symptoms or signs are lacking, and this determination becomes more difficult.

The presence of accompanying auditory symptoms or the finding of injury to the tympanic membrane or middle ear on otoscopic examination is more frequently, but not always, associated with end-organ injury.

The presence of vertical nystagmus usually means central disease.

DIAGNOSTIC TESTING

Further evaluations, such as electronystagmography, pure-tone and speech audiometry, temporal bone and skull computed tomography and magnetic resonance imaging, and complete otologic and neurologic examinations should be performed as soon as is feasible.

After an acute unilateral vestibular end-organ injury, vertigo characteristically subsides over a varying period of several days to 4 to 6 weeks. This improvement in symptoms usually results from central nervous system compensation and, less frequently, from functional recovery of the injured inner ear. Thus, a disappearance of symptoms does not necessarily mean that the injured part of the vestibular system has been restored to its previous healthy state. Persons who have compensated for permanent end-organ vestibular injury or destruction frequently have no dizziness. Some may experience transient, brief vertigo or loss of spatial orientation, or both, with certain positions or motions. These symptoms can be intensified with loss of some proprioception and vision during underwater conditions. Therefore, specialists should evaluate all divers who experience vestibular injuries after their symptoms have disappeared. Only in this way can rational judgments be made regarding an individual's suitability for exposure to future situations in which further inner ear injury and disability may occur or vertigo and spatial disorientation during diving might endanger the life of the diver or the lives of others.

VERTIGO IN DIVING

Vertigo is one of the most hazardous symptoms to occur during diving. It is frequently accompanied by hearing loss and tinnitus. Vertigo is described in multiple phases of diving.[13] However, many of these reports are not well documented, do not differentiate vertigo from nonvertiginous disequilibrium, or discuss vertigo only as an incidental observation.

Table 22–1. Causes of vertigo in divers

Decompression sickness
Hypoxia
Hypercarbia
Nitrogen narcosis
Seasickness
Alcoholic hangovers
Sensory deprivation
Hyperventilation
Impure breathing gas
Unequal caloric stimulation
Difficulties with middle ear pressure equilibration

Possible causes of vertigo in divers are provided in Table 22–1. One can readily appreciate that these causes can encompass a wide variety of pathologic mechanisms, the management of which is vastly different depending on the mechanism involved. Also, as noted earlier, the dizziness experienced in some of these entities is usually not true vertigo.

Edmonds and coworkers[13,29] undertook a complete review of the various causes of dizziness in diving. Their classification is basically broken down into those causes of vertigo due to *unequal vestibular stimulation,* including caloric stimulations, barotrauma, and decompression sickness; and those due to *unequal vestibular responses* to equal stimuli, such as the result of one vestibular apparatus being more sensitive than the other. Affected persons might have vertigo with caloric stimulation resulting from equal amounts of cold water entering the external ear canals. Also included in this group are divers who experience vertigo resulting from a unilateral hypofunctioning vestibular end-organ in situations in which equal and symmetrical pressure changes occur in the middle ear cavities during ascent and descent.

In addition, this classification includes dizziness noted with nitrogen narcosis; the dizziness, nausea, and tremor described in the high-pressure nervous syndrome; and the dizziness noted during oxygen toxicity and sensory deprivation. Here, we offer a modification of this classification, which separates diving otologic injuries into those with usually transient otologic dysfunction and those with permanent otologic inner ear injury.

Motion Sickness

Motion sickness is commonly experienced during sea, air, and car travel and is characterized by nausea, vomiting, cold sweats, yawning and hyperventilation, and pallor. The current accepted theory of neural input mismatch involves differences between real sensory input from the proprioceptive, vestibular, and visual systems compared with expected sensory input patterns to the central nervous system, which were programmed while the patient learned to crawl and ambulate during infancy.[30]

Divers commonly experience motion sickness on the boat or in the wave motion zone during decompression. Most symptoms resolve upon entering the water and with descent. Habituation from continuous exposure can develop in 2 to 3 days. Nervousness, female gender, young age, and dehydration from overindulgence in alcohol have all been cited as contributors to motion sickness.[29] Positioning oneself amidships while concentrating on the horizon has been suggested to help prevent or reduce the severity of symptoms.

TRANSIENT OTOLOGIC DYSFUNCTION IN DIVING

External Ear Canal Barotrauma (External Ear Squeeze, Reverse Ear Squeeze)

External ear canal obstruction can result in barotrauma to the canal and eardrum with ascent or descent during diving. With such blockage, ear canal pressure becomes negative during descent or positive during ascent relative to ambient and middle ear pressures. The resulting tissue damage includes congestion, hemorrhage, outward or inward bulging, and possible rupture of the tympanic membrane. The common causes of external ear canal obstruction are cerumen, foreign bodies, the use of earplugs, the use of tight-fitting diving hoods, and swelling and congestion of canal skin.

The treatment of external ear canal barotrauma is similar to that for middle ear barotrauma. If significant swelling of the external ear canal has occurred, then the treatment described for otitis externa in the following discussion is useful. Again, if tympanic membrane rupture has occurred, ototoxic eardrops should be avoided.

The best treatment of external ear squeeze is prevention. External ear canal patency during pressure changes must be

ensured. Accumulated masses of cerumen, which can essentially obstruct the ear canal, should be removed by washing the ear in a lukewarm water solution using a rubber bulb syringe. Care should be taken before such washing to ensure that a tympanic membrane perforation does not exist. The use of tight-fitting hoods, solid earplugs, or headphones, which can completely seal the ear canal, should be avoided.

The use of vented earplugs is controversial. They may be helpful in persons who have collapsing ear canals; however, these earplugs may become occluded with debris or cerumen and thus become harmful. Removal of an occluded earplug at depth would allow influx of colder water into the ear canal and potentially cause caloric stimulation vertigo, which can be disorienting and potentially fatal while a diver is submerged.

Otitis Externa

Otitis externa is the second most common diving medical problem, second only to middle ear barotrauma. It is a painful, sometimes debilitating malady encountered in all types of diving. The pathophysiology is related to the effects of moisture or humid atmospheres on cerumen and the canal skin. The external ear canal is lined with squamous epithelium and has a slightly acid pH. Cerumen is produced in the outer cartilaginous ear canal and contains water-soluble bacteriostatic fatty acids in addition to oil-soluble fatty acids. These factors, along with the constant outward migration of the squamous epithelium, provide a natural cleansing mechanism and usually protection from infection in this skin-lined cul de sac. Excessive exposure to water or humid atmospheres produces maceration of the squamous epithelium and can dilute the water-soluble fatty acids of cerumen, resulting in a shift of pH toward alkaline and providing a good medium for bacterial growth. Other contributing factors include collections of ceruminous debris, local trauma from cotton swabs (which should always be avoided) or other instrumentation, the presence of seborrheic dermatitis or eczema, poorly fitting or improperly cleaned earplugs, and swimming in polluted water.

In divers, the prolonged exposure to water or humid conditions frequently results in an alteration of the normal skin flora to a greater number of gram-negative bacteria. Thus, *Pseudomonas* and *Proteus* species, in addition to *Staphylococcus* organisms, are frequently found in otitis externa. Other organisms include diphtheroids, *Escherichia coli, Aerobacter,* and *Streptococcus faecalis.*[31,32] Fungi such as *Aspergillus* and *Candida* can also be seen but are usually noted after prolonged treatment with topical antibacterial agents. Otitis externa is particularly common in divers exposed for prolonged periods to saturation hyperbaria in chambers with high ambient temperature and humidity.

The symptoms of otitis externa in divers are similar to those in the nondiving population and include initial irritation with itching or burning. Later, there is a thin white discharge and pain that is frequently severe. Examination reveals an inflamed, swollen, and extremely tender external auditory canal. With progression, erythema of the surrounding pinna and skin, cervical lymphadenitis, and complete obstruction of the ear canal with subsequent abscess formation and possible involvement of bone and cartilage can occur. Fortunately, the latter complication is rare and practically never occurs in the absence of other debilitating illnesses such as poorly controlled diabetes or other conditions resulting in immune deficiency.

PREVENTION

The best treatment of otitis externa is prevention. Ear canals should be cleaned of ceruminous debris, and local trauma should be avoided. Any seborrheic condition should be adequately controlled before a dive. If ventilated earplugs are to be used, they should be properly fitted and cleaned.

A useful prophylactic topical ear solution during exposure to humid and aqueous environments contains a buffered weak acid such as 2% acetic acid and aluminum acetate. This is commercially available as otic Domeboro solution and should be used after showers, swimming, or diving. Alcohol and alcohol-boric acid preparations have been used as prophylactic measures with variable success. However, alcohol dissolves the cerumen fat-soluble fatty acids considered to be protective; also, solutions that contain alcohol can be irritating to the ear canal, particularly inflamed skin. The prophylactic use of antibiotic eardrops is not recommended because they are not usually effective and may enhance the chances of infection with a resistant organism.

TREATMENT

Once otitis externa is present, treatment principles include cleansing of the external auditory canal from infected squamous debris, topical antibiotic therapy, and normalization of canal pH. Systemic narcotics are often required for adequate pain relief. Cleansing is best accomplished by ear irrigation using lukewarm tap water or 1.5% hydrogen peroxide as described earlier, with care being taken to dry the ear canal afterward. A stream of warm air from a hair dryer, blown into the canal, is useful for this purpose. If an eardrum perforation exists, irrigation of the ear with water should be avoided and suction or gentle cotton wipes used instead. More specific therapy includes the use of an eardrop preparation that has a slightly neutral or acid pH and contains topical antibiotics in suspension. Several eardrop preparations also contain steroids, which are not contraindicated and are thought by some to aid pain relief because of the anti-inflammatory action. Adequate amounts of these agents should be used three to four times daily.

Frequently, swelling of the ear canal prevents these medications from being easily instilled into all of the involved areas of the ear canal skin. In these cases, a cotton wick or commercially available methylcellulose sponge wick should be inserted and the medications instilled onto the wick several times daily. These wicks can usually be removed in 2 to 4 days once the canal swelling has subsided. Systemic antibiotics can be used in the management of severe cases. However, the causative bacterial organisms are frequently *Proteus* or *Pseudomonas,* which are resistant to many antibiotics. Therefore, for the management of severe cases, appropriate cultures are important in the choice of systemic antibiotics.

All swimming and diving should cease until the otitis externa has cleared. This usually requires at least 5 to 7 days.

EXOSTOSES

Bony external auditory canal exostoses can also contribute to otitis externa. These hard lumps in the bony ear canal are not uncommon in swimmers and divers who are subject to repeated cold-water exposures. Recent studies in U. S. Navy and Japanese military divers show a higher rate of exostoses in divers compared with the overall population[33] and increased severity of exostoses in divers subjected to colder diving conditions.[34] The mechanism is unknown; supposedly, the very thin tissue of the external canal overlying the periosteum may render the underlying bone vulnerable to ischemic cold injury, with subsequent reactive hyperemia and repair by exostosis formation.

Middle Ear Barotrauma (Barotitis Media)

The most common diving medical problem is middle ear barotrauma resulting from inadequate pressure equilibration between the middle ear and the external environment. During compression, the nasopharyngeal ostium of the eustachian tube, which is normally closed, can fail to open if the diver does not make active attempts to clear the ears by swallowing or if local inflammation and swelling prevent opening. Thus, middle ear pressure becomes negative relative to the increasing ambient pressure (Fig. 22–2). If a diver descends 2.6 feet and middle ear pressure fails to equilibrate, there is a theoretical pressure differential of 60 mm Hg (Fig. 22–2*B*). Significant mucosal congestion and edema occurs. This further narrows the eustachian tubal lumen, and subsequent ear clearing or pressure equilibration becomes more difficult. Also, with increasingly negative middle ear pressure, opening of the eustachian tube becomes more difficult because of the nasopharyngeal valve effect. At a pressure differential of approximately 90 mm Hg, equivalent to a descent of 3.9 feet, it is usually impossible to open the tube voluntarily (Fig. 22–2*C*). Fullness and pain usually occur at the pressure differential of 60 mm Hg, and the tympanic membrane has been found to rupture at pressure differentials ranging from 100 to 500 mm Hg[35] (Fig. 22–2*E*). With a forceful modified Valsalva maneuver (nose occluded) under these conditions, the existing pressure differential between the inner ear and middle ear becomes greater, possibly leading to round window rupture with leakage of perilymph and inner ear injury or breaks in the inner ear membrane (inner ear barotrauma—see the later discussion) (Fig. 22–2*D*). Animal studies have demonstrated round window

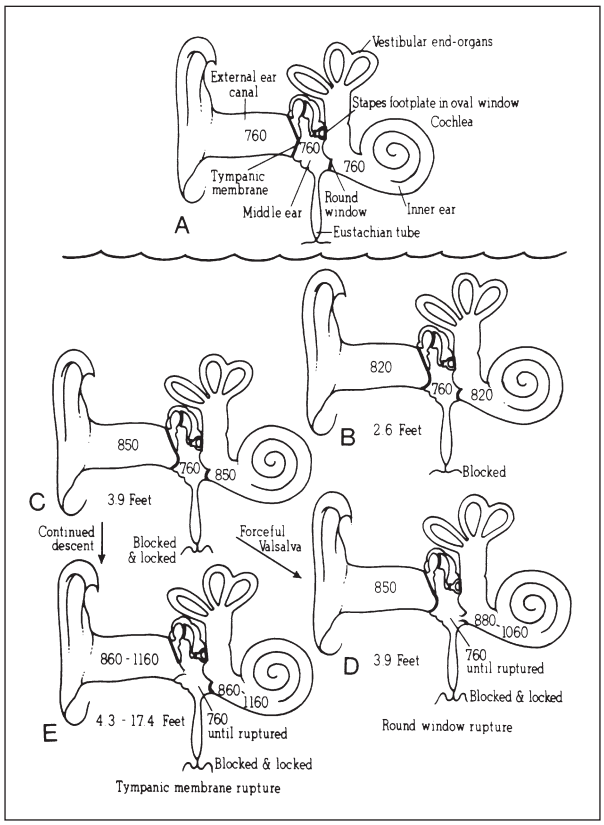

Figure 22-2. Otological barotrauma of descent. Theoretical sequence of changes in the right ear of a diver who does not equilibrate middle ear pressure during descent. Pressures are shown in millimeters of mercury (mm Hg). *A,* Surface condition with equal pressures (760 mm Hg) throughout and a patent eustachian tube with a normally closed nasopharyngeal ostium. *B,* Depth of approximately 2.6 feet after diver failed to open the eustachian tube upon entering the water. Pressure differential of 60 mm Hg exists. Tympanic membrane and round window are bulging into the middle ear. Diver notices pain and pressure in the ear with a conductive hearing loss and possible vertigo. *C,* Depth of approximately 3.9 feet with 90 mm Hg pressure differential and blocked and locked eustachian tube. *D,* A forceful Valsalva maneuver can lead to rupture of the round window with resulting leak of perilymph into the middle ear. The exact pressure differentials at which rupture occurs in humans are unknown. Studies in cats[36] have indicated that round window ruptures occur when a pressure of 120 to 300 mm Hg is added to the cerebrospinal fluid space at 1 ata. *E,* Continued descent can lead to tympanic membrane rupture at pressure differentials of 4.3 to 17.4 feet.[35] The actual rupture point varies highly.

ruptures when CSF pressure has been increased 120 to 300 mm Hg.[36]

CLINICAL PRESENTATION

Symptoms of middle ear barotrauma consist initially of a sensation of ear blockage. With further descent and greater pressure differentials,[9] frank ear pain occurs. A conductive hearing loss is always present but may not be a primary complaint because of ear pain. Mild tinnitus and vertigo can occur. With eardrum rupture, pain usually is severe and vertigo can occur from a caloric effect if water enters the middle ear. If hearing loss, tinnitus, and vertigo are severe in association with a no-decompression dive, possible inner ear barotrauma with round window rupture or other inner ear injury should be suspected.

The presence of pre-dive nasal dysfunction, such as congestion and discharge, increases the chances of inadequate eustachian tubal function and subsequent middle ear barotrauma. Likewise, a history of otitis media, mastoiditis, previous mastoid or middle ear surgery, or middle ear barotrauma with flying is suggestive of inadequate eustachian tubal function in the absence of the atmospheric pressure changes associated with diving. Such persons are certainly more likely to have inadequate eustachian tubal function with exposure to the greater atmospheric pressure changes encountered in diving. A recent study by Miyazawa and collaegues[37] showed that dysfunction of the eustachian tube, measured by sonotubometry and tympanometry before hyperbaric and hypobaric exposures in a normal adult population, was a statistically significant predictor of middle ear barotrauma in a normal patient population. Divers who undertake rapid descent or who do not attempt to equilibrate middle ear pressures every 1 to 2 feet of descent are more likely to experience eustachian tube mucosal congestion and middle ear barotrauma during subsequent dives within the next several days. Multiday, repetitive diving, even in two experienced divers, has been documented to result in eustachian tube dysfunction, increasing negative middle ear pressures and barotitis media in proportion to diving frequency.[38]

PHYSICAL SIGNS

The physical signs of middle ear barotrauma are noted on otoscopic examination. The pathologic changes include edema and hemorrhage in the middle ear mucosa as well as inflammation and collections of serous fluid or blood in the middle ear cleft. Six grades of middle ear barotrauma have been described (Table 22–2).[13]

This classification well describes the pathologic changes; however, the otoscopic findings in middle ear barotrauma frequently include combinations of changes in different grades. Eardrum scarring from previous perforations plus non–diving-related middle ear disease or surgery can obscure the middle ear findings. Occasionally, signs are minimal or absent, but then obvious signs of negative middle ear pressure with eardrum injection and retraction and middle ear inflammation or effusion develop over the next 24 hours. Also, treatment is not completely dependent on which of these grades is present.

TREATMENT

Recommended treatment varies with the type of middle ear barotrauma. Three types of barotrauma are described.

Type 1 Middle Ear Barotrauma

Type 1 describes cases with post-dive symptoms but without otoscopic signs either immediately or within 24 hours after a dive.

Table 22–2. Grading system for middle ear barotraumas*

Grade 0 Symptoms without otoscopic signs
Grade 1 Diffuse redness and retraction of the tympanic membrane
Grade 2 Grade 1 changes plus slight hemorrhage within the tympanic membrane
Grade 3 Grade 1 changes plus gross hemorrhage within the tympanic membrane
Grade 4 Dark and slightly bulging tympanic membrane due to free blood in the middle ear; a fluid level may also be present
Grade 5 Free hemorrhage into the middle ear with tympanic membrane perforation; blood can be seen outside or within the ear canal.

From Edmonds C, Freeman P, Thomas R, et al: Otological Aspects of Diving. Sydney, Australian Medical, 1973.

The recommended treatment for type 1 cases is as follows:

1. Avoid any further diving until (a) any preexisting nasal symptoms have cleared, (b) the patient can easily autoinflate both ears at the surface, and (c) ear symptoms have cleared.
2. Long-acting topical nasal decongestants such as oxymetazoline hydrochloride (Afrin) nose drops, two to three drops into each nostril twice daily with the head extended and supine, can be used. The nose drops should not be used for more than 7 days. Nasal adrenergic sprays are not as effective as drops. Systemic decongestants such as pseudoephedrine (Sudafed), 30 to 60 mg orally three times daily, should be used with caution because the drug may have undesired adrenergic side effects such as temporarily elevated blood pressure, hyperexcitability, and insomnia. Persons with a history of palpitations, coronary artery disease, hyperthyroidism, or diabetes should avoid pseudoephedrine-containing products. Systemic antihistamines and steroid nasal sprays are usually not beneficial unless there is preexisting nasal allergy.

Type 2 Middle Ear Barotrauma

Type 2 describes cases with symptoms plus otoscopic findings and no eardrum perforation. The recommended treatment is as follows:

1. Rest and avoid further diving until otoscopic examination shows complete resolution and the diver can easily autoinflate both ears at the surface. This usually requires 3 to 14 days, depending on the severity of the injury.
2. Use systemic and topical nasal decongestants as described for type 1. Short courses of systemic steroids have been used with reported benefit; however, the well-described contraindications and complications of systemic steroids should be noted.
3. Prophylactic systemic antibiotics are controversial but have been used. Eustachian tubal function is usually poor because of middle ear and tubal mucosal inflammation and swelling. Under these conditions, secondary middle ear bacterial infection becomes more likely. In the presence of purulent nasal discharge or cough with

purulent sputum production, systemic antibiotics should be given.
4. With an intact eardrum, topical eardrops containing antibiotics, steroids, or anesthetic agents are of no benefit because these substances do not readily cross the outer, squamous epithelial layer of the tympanic membrane. An inert oily preparation such as Auralgan, warmed to body temperature and instilled into the ear, may provide partial pain relief.
5. Pain relief is best achieved with systemic analgesics; however, the use of narcotics is generally not needed.

Type 3 Middle Ear Barotrauma

Type 3 describes cases with symptoms and otoscopic findings that include eardrum perforation. The suggested treatment is as follows:

1. Avoid further diving until a complete otologic evaluation has been performed and the middle ear process has resolved with healing or surgical repair of the eardrum. Most of these perforations heal spontaneously, and surgical repair is not necessary. Persistence of poor eustachian tubal function or middle ear inflammation from secondary infection delays healing.
2. If the amount of blood or other debris in the ear canal is significant, the patient requires referral to an otolaryngologist for ear cleansing. Various cleansing solutions may be used, but this is controversial because of the possibility of washing such debris into the middle ear and damage to the middle ear mucosa. Solutions containing alcohol or acids should not be used when an eardrum perforation exists because of a significant irritating effect on the middle ear mucosa.
3. Most commercial antibiotic eardrop preparations contain drugs that are toxic to the inner ear. These should not be used in the presence of an eardrum perforation. The only commercial otic antibiotic eardrop approved for use in the middle ear is a ciprofloxacin HCl and hydrocortisone otic suspension, Cipro HC Otic, which is recommended if purulent discharge occurs. Otic solutions containing topical anesthetics are usually inadequate for analgesia.
4. Systemic and topical nasal decongestants should be employed as described earlier.
5. If purulent discharge is found in the nose, in the tracheobronchial tree, or draining from the ear, these drainages should be cultured and systemic antibiotics should be administered. Prophylactic antibiotics may be given systemically in the absence of purulent discharge because of the increased possibility of secondary middle ear or mastoid infection from contaminated water.
6. If the eardrum does not heal after 1 to 2 weeks of appropriate therapy, the patient should be referred to an otolaryngologist. In addition, further diving should be avoided until the eardrum has healed and middle ear ventilation is adequate. The diver should be cautioned that an eardrum perforation occurring underwater is a potentially serious problem. Fortunately, most eardrum perforations resulting from middle ear barotrauma heal spontaneously, and surgical repair is not required. Poor eustachian tubal function resulting from nasal or sinus disease can impair healing.

PREVENTION

The best treatment of middle ear barotrauma is caution and prevention. An adequate prediving otolaryngologic history and examination, emphasizing nasal and eustachian tubal function, should be performed. Information that suggests poor eustachian tubal function is listed in Table 22–3. Such persons are unlikely to be able to tolerate the pressure changes encountered in diving.

Other important factors in the prevention of middle ear and possible inner ear barotrauma are provided in Table 22–4.

Divers should be aware of techniques of equilibrating middle ear pressure, which are safer than a modified Valsalva maneuver:

Table 22–3. Factors that indicate poor eustachian tube function

Preexisting symptoms or signs of middle ear or nasal disease, or both
Frequent bouts of ear infection or drainage, or both
History of middle ear or mastoid surgery
History of a healed or persistent eardrum perforation
Existence of a cholesteatoma
History of difficulty with ear clearing during descent while flying

Table 22–4. Methods for prevention of middle and inner ear barotraumas

Avoiding diving in the presence of significant nasal congestion or discharge
Not continuing descent without adequate ear clearing every one to two feet
Slowing descent rates
Descending feet first
Avoiding a forceful modified Valsalva maneuver at depth

- *Modified yawn and swallow*. This maneuver is accomplished by thrusting the lower jaw anteriorly and slightly opening the jaw while maintaining the lips pursed around the regulator. This may be followed by a swallow if ear clearing has not occurred.
- *Frenzel maneuver.* With the nose, mouth, and glottis voluntarily closed, the tongue is used as a piston to compress air in the nasopharynx and thus into the eustachian tube. The mass of the tongue is strongly driven backward and acts to compress the nasopharyngeal air space through the soft palate.
- *Soft palate contraction.* The palatal muscles are activated when the patient raises the soft palate. With experience, this maneuver can be mastered without the need to swallow or move the jaw.
- *Toynbee maneuver.* Pressure equilibration is attained by swallowing with the mouth and nose closed, which produces an initial slight increase followed by a decrease in nasopharyngeal pressure. This is less likely to clear a middle ear with existing negative pressure.

The techniques described for gentle eustachian tubal opening and middle ear pressure equilibration during diving are safe in that they are unlikely to induce significant changes in pulmonary, alveolar, arterial, central venous, CSF, or labyrinthine fluid pressures, which do occur with the modified Valsalva maneuver. Thus, dangerous overpressures in these spaces and subsequent pulmonary or inner ear barotrauma are less likely. Also, these maneuvers, in contrast with the modified Valsalva maneuver alone, are more effective because they involve contraction of the tensor palatini muscle, which acts to open the nasopharyngeal orifice of the eustachian tube. Thus, less nasopharyngeal pressure is required for tubal opening.

Alternobaric Vertigo: Transient Vertigo Resulting from Unequal Middle Ear Pressure Equilibration

Lundgren[15] described transient vestibular dysfunction secondary to asymmetrical middle ear pressure equilibration in 1965. Later reports came from Vorosmarti and Bradley[39] and from Terry and Dennison.[40] Lundgren attributed such vertigo to a unilateral increase in middle ear pressure during ascent with resulting unequal vestibular end-organ stimulation. Indeed, some persons who have experienced alternobaric vertigo at depth can produce vertigo and vestibular nystagmus by performing a modified Valsalva maneuver and unequally inflating the middle ears on the surface. Many of these persons have encountered unilateral difficulty with middle ear pressure equilibration during diving. Disappearance of the vertigo has been noted with stopping the ascent or with descending again, shortly after a sudden hissing from the blocked ear, or both. Further work by Tjernstrom[41] and Ingelstedt and colleagues,[16] using a technique for indirectly measuring middle ear pressure changes with simultaneous electronystagmographic recordings, has shown true vestibular nystagmus with an overpressure in one middle ear during decompression in a pressure chamber.

The exact frequency of alternobaric vertigo is not known. A later publication by Lundgren and coworkers[42] involved a questionnaire answered by 2053 Swedish divers; it indicated that of 453 divers who had experienced vertigo during diving, 343 were likely to have had alternobaric vertigo. The vertigo was noted to last from a few seconds up to 10 min. Divers who had experienced vertigo had logged more dives than those without vertigo and had more frequently reported middle ear pressure equilibration difficulties. These divers also noted that the pressure equilibration difficulties were more dominant in one ear.

The occurrence of vertigo during underwater exposures, even those to shallow depths, can be hazardous. The resulting spatial disorientation, with possible nausea and vomiting, may explain some of the previously unexplained deaths of experienced scuba divers.

Prevention is the best treatment. First, persons should not dive if they have

difficulties with ear clearing or if a Valsalva maneuver at 1 ata produces vertigo. Second, divers should take precautions to adequately equilibrate middle ear pressure every 1 to 2 feet of descent. If a diver notices any ear fullness, blockage, or vertigo during compression, further descent should be stopped and the diver should ascend until the ears can be cleared. If vertigo is noted during ascent, and if gas supplies and other conditions permit, the ascent should be stopped and the diver should descend until the symptoms disappear. Diving with a trained companion and avoiding delaying ascent until gas supplies are almost depleted are excellent rules that should be followed.

Alternobaric Facial Paralysis

Transient unilateral facial paralysis in association with ipsilateral middle ear overpressure during ascent has been described in divers by Molvaer[43] and Becker[44] and in flyers during ascent by Bennett and Liske.[45] The divers experienced alternobaric vertigo with an overpressure in the ear on the same side as the facial paralysis. In each instance, the paralysis subsided within 1 hour after onset. Becker[44] postulated that the middle ear overpressure during ascent compressed the horizontal portion of the facial nerve through a dehiscent bony fallopian canal. Another mechanism postulated is excessive middle ear pressure during ascent resulting in gas bubbles entering a nondehiscent fallopian canal through the fenestram of the chorda tympani nerve.

Because alternobaric facial paralysis is an infrequent problem and appears to be transient, treatment should focus on prevention by appropriate equilibration of middle ear pressure and measures to prevent inadequate ear clearing during exposures to altered atmospheric pressures as described earlier.

Intracranial Consequences of Middle Ear Barotrauma

In 1986, Goldmann[46] described an interesting case of pneumocephalus secondary to otologic barotrauma. A 26-year-old healthy male scuba diving instructor noted left ear pain while ascending to the surface after a 60-foot freshwater dive. Upon reaching the surface, his left ear pain suddenly disappeared and he experienced a severe left vertex headache. After several hours of surface interval, he subsequently dove again to 60 feet and noted that the headache improved at depth; however, upon ascent, the headache returned with increasing severity. Upon hospital presentation, the physical examination was unremarkable with the exception of left middle ear barotrauma. Skull radiographs revealed a subdural pneumocephalus localized to the left side of the cranium; computed tomography of the brain revealed air in the subdural space, and a nuclear magnetic resonance scan of the brain 16 days after the incident revealed a small amount of blood in the left epidural space near the base of the skull with blood in both mastoids, more on the left side. He was treated conservatively with prophylactic antibiotics, with gradual resolution of his headache and normal-appearing follow-up computed tomography 1 month later.

During ascent, expanding gas in the middle ear is usually cleared passively through the eustachian tube. However, if the pressure differential between the middle ear space and the ambient pressure is greater than 2 to 3 feet during descent, swelling and congestion of the eustachian tube and middle ear mucosa impede passive clearing of the middle ear during ascent, with subsequent increased middle ear pressure similar to that noted with alternobaric vertigo (described earlier). This increased pressure is distributed throughout the middle ear cleft and mastoid air cell system. Frequently, the bony roof of the middle ear attic area, which also forms the floor of the middle cranial fossa, is thin with dehiscence in approximately 22% of normal human temporal bones, as described by Ferguson and colleagues.[47] Thus, this area may have provided a route for the escape of expanding gas into the intracranial space.

Transient Vertigo from Caloric Stimulation

During most diving conditions, the vestibular end-organs are stimulated equally and vertigo does not occur. Edmonds and coworkers[13,29] described caloric vertigo in divers resulting from unequal vestibular stimulation and unequal vestibular responses to equal stimuli. Vestibular nystagmus was

demonstrated when cold water entered one ear, particularly when the divers were in a position in which the horizontal (lateral) semicircular canal was in a vertical orientation, that is, supine with the head elevated 30 degrees or prone with the head depressed 30 degrees. Water immersion usually results in an equal flow of cold water into both external auditory canals, with subsequent symmetrical vestibular end-organ stimulation and an absence of vertigo. Obstruction of one ear canal with cerumen, foreign bodies, exostoses, otitis externa, earplugs, or diving hoods can increase the chances of unequal entry of cold water into the external auditory canals and caloric stimulation. A tympanic membrane perforation may also result in unequal entry of cold water into the middle ear and caloric stimulation vertigo.

Transient Dizziness Resulting from High-Pressure Nervous Syndrome

Transient symptoms suggestive of vestibular system dysfunction (vertigo, dizziness, and nausea) have been reported in association with high-pressure nervous syndrome.[48–50] Later studies[51–54] have indicated that these symptoms are not accompanied by demonstrable electronystagmographic nystagmus but are associated with decrements in postural equilibrium and bilaterally equal increases in the vestibulo-ocular reflex. Thus, such symptoms during high-pressure nervous syndrome are thought to be related to dysfunction in more centrally located structures and not to unilateral end-organ or primary vestibular neuron dysfunction.

PERMANENT INNER EAR OTOLOGIC INJURY IN DIVING

As discussed earlier under historical considerations, inner ear injury in diving was recognized in the latter part of the nineteenth century and in the early part of the twentieth century. In the 1930s and 1940s, safety procedures for air diving improved and the frequency of recognized inner ear injuries in diving decreased. Indeed, most of the diving literature concerning decompression sickness noted symptoms suggestive of inner ear injury in association with what was thought to be central nervous system decompression sickness, whereby the inner ear symptoms were understandably considered to be of secondary importance. Vertigo was often thought to be related to lesions in the central nervous system.

Hearing loss, tinnitus, or vertigo occurring during or shortly after decompression, in the absence of other symptoms that suggest decompression sickness, were often not treated because isolated inner ear decompression sickness was not generally recognized. The conclusions reached during that time are understandable because isolated inner ear injuries are not usually life-threatening unless a diver notes severe vertigo, nausea, and vomiting while underwater. Also, the diving community did not generally recognize that the vestibular symptoms resulting from permanent destruction of one vestibular end-organ in otherwise healthy persons would usually subside in 4 to 6 weeks because of central nervous system compensation, even though inner ear function had not been recovered.

Beginning in the 1960s and 1970s, with more frequent diving including mixed-gas diving to deeper depths, reports of permanent inner ear injury in multiple phases of diving appeared. The pathogenesis and management of diving inner ear injuries differed with the phase and type of diving in which the injury occurred. These injuries have been classified as follows[21,27,55–57]:

- *Inner ear barotrauma.* Injuries related to middle ear barotrauma, usually beginning during descent or compression
- *Isobaric otologic barotrauma.* Inner ear injuries at stable deep depths
- *Inner ear decompression sickness.* Injuries occurring during ascent, during or shortly after decompression
- *Noise-induced hearing loss.* Sensorineural deafness related to high background noise during diving

Inner Ear Barotrauma

Inner ear injuries associated with relatively shallow diving and those in which the otologic symptoms begin with inadequate middle ear pressure equilibration during the compression phase of deeper diving have been called *inner ear barotrauma.* These injuries were first documented and named by Freeman and Edmonds in 1972[18] and were found to be related to labyrinthine window ruptures by

Edmonds and colleagues[17] in 1974. These cases involved difficulty with ear clearing during descent or evidence of middle ear barotrauma on otoscopic examination, or both. The depth and duration of the dives made decompression sickness unlikely. The sensorineural deafness was noted to be total or partial and occurred with or without varying degrees of vestibular dysfunction.

PATHOPHYSIOLOGY

Goodhill and coworkers[58] proposed that these injuries were secondary to oval or round window ruptures, or both, and postulated implosive and explosive mechanisms for these ruptures related to diving and nondiving stresses. The explosive mechanism depicted in Figure 22–2*D* suggests that, with inadequate middle ear pressure equilibration during descent, middle ear pressure becomes negative relative to intralabyrinthine fluid pressure as well as ambient pressure. Straining or a modified Valsalva maneuver in an attempt to clear the ear results in a further increase in the pressure differential between the labyrinth and the middle ear as a result of increases in CSF pressure transmitted to the inner ear. There is rupture of the round or oval window membrane into the middle ear and a subsequent perilymph fistula. Studies by Harker and coworkers[36] have shown that increased CSF pressure results in bulging of the round window membrane in cats, with ruptures occurring when CSF pressure is increased to levels ranging from 120 to 300 mm Hg.

The implosive mechanisms suggest that with a sudden Valsalva maneuver resulting in middle ear ventilation, the rapid increase in middle ear pressure can rupture the round or oval window into the intralabyrinthine space. Another possible implosive mechanism suggests that, in the presence of a negative middle ear pressure, there is inward displacement of the eardrum, ossicular chain, and stapes footplate. With a significant negative middle ear pressure, the footplate can sublux into the vestibule and an oval window fistula can occur. A third possible and perhaps more likely implosive explanation involves the formation of middle ear overpressure during ascent after inadequate middle ear pressure equilibration and negative middle ear pressure during descent. This causes mucosal congestion and swelling, which narrows the eustachian tube lumen and impedes the clearing of expanding gas during the subsequent ascent; thus, a middle ear overpressure occurs, which may disrupt the labyrinthine windows and cause inner ear barotrauma. Indeed, as noted earlier, middle ear overpressure during ascent has been well described and demonstrated by Tjernstrom,[41] Ingelstedt and colleagues,[16] and Lundgren and assoicates[42] as alternobaric vertigo. In these cases, the degree of middle ear overpressure was probably not sufficient to cause labyrinthine window rupture and permanent inner ear injury.

Inner ear barotrauma in association with middle ear barotrauma with subsequent inner ear auditory or vestibular dysfunction may occur in diving without labyrinthine window rupture. In 1929, the animal studies of Vail[7] showed hemorrhage into the basal turn of the cochlea. Kelemen[59] noted hemorrhage in the middle and inner ears of the temporal bones of two drowning victims. No evidence of inner ear membrane tears or round or oval window fistulae were apparent. Simmons[60,61] postulated that the pressure changes encountered with inadequate middle ear pressure equilibration during the descent phase of diving may result in intralabyrinthine membrane breaks with or without labyrinthine window rupture. Also, in 1981, Gussen[62] noted a break in Reissner's membrane without labyrinthine window rupture in the temporal bone of a woman who suffered severe ear pain with subsequent hearing loss, tinnitus, and vertigo after an airplane trip.

On the basis of these studies, Parell and Becker[63] speculated that the pathology of 14 cases of inner ear injury related to scuba diving could be divided into hemorrhage within the inner ear, labyrinthine membrane tears, and perilymph fistula.[9] It was proposed that inner ear hemorrhage cases presented with absent or transient vestibular symptoms and moderate sensorineural hearing loss. These cases showed excellent hearing recovery without surgery. Cases with labyrinthine membrane tears were proposed to present with similar symptoms and findings, except that recovery audiograms showed a localized, persistent hearing loss in one or two frequencies (a notch hearing loss). The four patients of labyrinthine window fistula were thought to be divers who had vestibular symptoms in addition to sensorineural hearing loss. In one case, the fistula occurred in a round window that was more vertically placed so that most of the membrane could

be directly seen on exploratory tympanotomy, a finding previously noted by Pullen and colleagues[64] and by Singleton.[65] Most labyrinthine window ruptures associated with diving have involved the round window. A few cases have involved the oval window.[66]

DIAGNOSIS AND MANAGEMENT

Any diver who presents with signs of inner ear injury—vertigo, sensorineural hearing loss, loud tinnitus—after dives in which decompression sickness is unlikely should be suspected of having inner ear barotrauma and possible labyrinthine window fistula. Unless other signs indicate pulmonary overpressure accidents and air embolization, divers suspected of suffering inner ear barotrauma *should not be subjected to recompression therapy.* Such therapy exposes the patient to the same pressure changes that contributed to the otologic injury. A complete evaluation by an otolaryngologist, including otoscopic examination, proper audiometric testing, a complete neurologic examination, and tests of vestibular function and the possible presence of an otologic fistula, should be accomplished as soon as feasible.

Treatment recommendations and guidelines include:

- Bed rest with head elevation is recommended.
- Care should be taken that CSF and inner ear pressures are not increased (e.g., Valsalva maneuvers, coughing, nose blowing, and straining at defecation).[17,58]
- Medications that supposedly increase intracranial and inner ear blood flow are generally not effective in this regard and may result in a decrease of intracranial blood flow via shunting of the axial circulation to the periphery and skin.
- Anticoagulants are potentially harmful because of possible hemorrhage from traumatized otologic tissues.
- Eardrops containing ototoxic antibiotics should be avoided.

SURGERY

The treatment of perilymphatic fistula remains a diagnostic challenge with controversial management. Inner ear barotrauma related to diving may not be frequently associated with an active fistula. In a series of 91 patients with documented perilymphatic fistula, Seltzer and McCabe[67] noted a wide variety of signs and symptoms ranging from unilateral tinnitus and aural fullness to sudden and profound hearing loss, roaring tinnitus, and whirling vertigo. Twenty-three percent of the fistulae were associated with head trauma, barotrauma, direct ear trauma, or acoustic trauma. Kohut and associates[68] point out that any one of a combination of the following criteria in a patient with an otherwise healthy ear is highly suggestive of a perilymphatic fistula:

- Sensorineural hearing loss of sudden onset, fluctuating nature, or rapid progression
- Positive Hennebert's sign or symptom (deviation of the eyes or dysequilibrium with changes in external ear canal pressure)
- Dysequilibrium with loud noise exposure
- Positional nystagmus
- Constant dysequilibrium of varying severity between attacks of spontaneous vertigo

In a nationwide survey of otolaryngologists, House[69] found that a patient history suggestive of trauma with a delayed onset of audiovestibular symptoms that progress or fluctuate provides more information for the diagnosis of perilymphatic fistula than a battery of otologic testing.

Some authors have advocated immediate exploratory surgery in all suspected cases of labyrinthine window fistula.[70] Others have suggested reserving surgery for those who do not experience improvement after 48 to 72 hours of bed rest with head elevation.[58] Caruso and colleagues[66] reasoned that, although the majority of labyrinthine window ruptures may heal spontaneously with conservative treatment, such treatment may be associated with progressive hearing loss. They recommend that when the diagnosis is fairly certain, surgery should be performed without delay to prevent further inner ear deterioration. Most authors[71–73] advise that an initial trial of medical management as described earlier should be undertaken and exploratory surgery reserved for those who demonstrate no improvement after 5 to 10 days or if inner ear function worsens in the interim.

Isobaric Otologic Barotrauma: Otologic Problems at Stable, Deep Depths

Sundmaker[74] described inner ear problems occurring at stable, deep depths as observed at the University of Pennsylvania late in the

summer of 1971. Three divers who had been breathing an oxyhelium atmosphere at a simulated depth of 600 feet in a pressure chamber noted the sudden onset of vertigo, nausea, and nystagmus shortly after starting to breathe by mask a gas mixture containing a second inert gas (neon or nitrogen). Follow-up evaluations after the dive revealed permanent end-organ vestibular dysfunction in two of the three affected subjects with no changes in auditory function.

The likely mechanism of the vestibular injury appears to be related to the counterdiffusion of different inert gases with different solubilities between inner ear fluid compartments, with gas bubbles forming at tissue interfaces, such as the partitions in the inner ear between the perilymphatic and the endolymphatic spaces.[26] This bubble formation produces displacement or disruption of the inner ear structures, or both, and occurs without changes in total ambient pressure. A similar reaction has been documented in the skin by Blenkarn and colleagues[75] and Graves and coworkers,[76] who noted gas-filled blister formations following the sequential exposure to various inert gases at constant ambient pressures. These eruptions are thought to represent gas bubble formations in the deeper layers of the skin resulting from the counterdiffusion of different inert gases across tissue interfaces. Farmer[27] suggested that these inner ear injuries occurring shortly after inert gas changes at stable, deep depths could be related to increased volume in and distention of the endolymphatic space resulting from an osmotic flux of fluid due to more rapid accumulation of the dissolved inert gas in endolymph.

Until the exact mechanism of these injuries at stable, deep depths has been established, changes between inert gases at deep depths should be avoided.

Inner Ear Decompression Sickness

HUMAN REPORTS

Inner ear decompression sickness became an accepted entity in the late 1960s, with subsequent documentation in the literature. In 1967, Buhlmann and Waldvogel[77] described 82 decompression accidents from a series of chamber dives ranging in bottom depths from 11 ata to 23 ata and noted that the only neurologic symptoms of the entire series consisted of vertigo, nausea, vomiting, and tinnitus in 11 divers, with hearing loss present in 2 of these divers. In 1971, Rubenstein and Summitt[20] described 10 cases of isolated inner ear decompression sickness after diving. Farmer and colleagues[21] included these 10 cases and added 13 more cases of isolated inner ear decompression sickness occurring during or shortly after decompressions from 4 air and 19 helium-oxygen dives.

None of these cases was associated with symptoms of middle ear barotrauma during compression, otologic symptoms at the maximum depth, uncontrolled or rapid ascents, or other symptoms or signs suggestive of central nervous system decompression sickness. Ten of the divers had vertigo only, seven had hearing loss and tinnitus, and six exhibited hearing loss, tinnitus, and vertigo. Prompt recompression treatment correlated significantly with recovery. The 11 divers who underwent recompression within 42 minutes after the onset of the otologic symptoms experienced relief during recompression; subsequent studies revealed no residual inner ear dysfunction. Three divers underwent recompression within 60 to 68 minutes after symptom onset; one of these persons experienced relief of symptoms, with the remaining two having only partial or no relief and demonstrating significant residual inner ear dysfunction. In the remaining cases, recompression treatment either was delayed longer than 68 minutes after symptom onset or was not given; the divers experienced residual inner ear dysfunction.

Thirteen of the 19 helium dives in this series involved a switch to an air atmosphere at depths ranging from 60 to 150 feet during the latter stages of decompression. Farmer and coworkers[21] postulated that the sudden decrease in helium partial pressure during such an air switch possibly contributed to the formation of helium gas bubbles in inner ear tissues during decompression. Another speculated pathophysiologic mechanism involved the formation of bubbles at inner ear tissue boundaries resulting from the counterdiffusion of two different inert gases between inner ear fluid compartments, similar to the counterdiffusion mechanism suggested by Graves and associates[76] and by Lambertson[26] to explain inner ear injuries noted at stable, deep depths after inert gas changes.

More recently, Nachum and colleagues[78] reported a retrospective 12-year experience of 29 cases of inner ear decompression sickness in compressed air recreational diving. It is important to consider that in this study and others, some of the divers do not appear to violate a decompression schedule; thus, the differential diagnosis between inner ear barotrauma and decompression sickness is uncertain. The authors found that prompt diagnosis and recompression resulted in complete resolution of symptoms in 9 of the 17 patients treated within 6 hours of symptom appearance.

ANIMAL STUDIES

Animal studies have shown interesting findings regarding the pathophysiology of inner ear decompression sickness. In 1975, McCormick and colleagues[23,79] showed that guinea pigs subjected to rapid decompression experienced bubble formation and hemorrhages in labyrinthine fluid spaces and decreases in cochlear potentials. The authors also observed that these deficits in inner ear electrical function could be lessened by treating the animals with heparin prior to the dives, indicating that a key mechanism of inner ear decompression sickness may be hypercoagulation in the inner ear microvasculature as described by Philp.[80]

The most extensive animal studies of inner ear decompression sickness have been performed in Toronto and are reviewed by Landolt and colleagues.[81] These investigations revealed that the inner ear in squirrel monkeys, apparently similar to the inner ear in humans, is susceptible to decompression sickness. Clinical observations plus electronystagmographic recordings and post-dive histologic studies revealed that the injuries occurred during the latter stages of decompression and were related to specific histologic findings in the inner ear. Shortly after the injuries, varying degrees of hemorrhage and blood-protein exudate in the inner ear fluid spaces and tissues were noted. The inner ears of monkeys killed 38 to 383 days following decompression showed the appearance of connective tissue and new bone growth that tended to obliterate the damaged regions of the semicircular canals.

An interesting biophysical mechanism to explain these changes involves the production of significant pressures by bubble enucleation and growth within osteoplastic cell cavities of the endosteal bone immediately surrounding the semicircular canals. During the latter stages of decompression, significant pressure differentials occur between these bony cellular spaces and the adjacent perilymphatic spaces with a sudden implosive fracture of the endosteal bone into the canal space. The implosive nature of such fractures was postulated to cause a pressure wave bolus that moved rapidly along the canal, causing tearing of the endosteum and loosening of the attachments of the membranous semicircular ducts to the canal wall with initial bleeding and a later stimulus for subsequent new bone growth. Indeed, in 1985, Money and coworkers[82] reported similar changes in the inner ear of a professional diver who died of unrelated causes 56 days after suffering left inner ear decompression sickness that did not respond to prompt recompression. The diver experienced a persistent total loss of left vestibular function and partial left sensorineural deafness. Histologic examination of the temporal bone revealed ectopic bone growth and fibrosis in the left ear semicircular canal similar to that seen in the squirrel monkeys sacrificed 38 days or longer after inner ear decompression sickness.

MANAGEMENT

Citing the human investigations, Farmer and colleagues[21] proposed the following measures in 1976; thus far, they appear to be appropriate.

Inner ear symptoms that begin during or shortly after the decompression phase of a dive profile in which scheduled decompression is indicated by dive tables should be considered decompression sickness. *Use of dive tables is suggested in addition to the use of dive computer to determine adequacy of decompression.*

Divers who experience symptoms during or shortly after a switch to an air environment during decompression from a deep helium-oxygen exposure should be switched back to the presymptom helium-oxygen atmosphere and subjected to prompt recompression.

The optimum recompression depth has not been established. Obviously, the depth of relief would be a predictable desired end point. However, in some cases, bubble formation in the inner ear will have caused

structural deformities such as those described in animal studies[81] and in one human report[82] by Landolt and coworkers; thus, inner ear symptoms will not be relieved even though the depth of recompression has been enough to drive the bubbles back into solution. Also, returning to the bottom depth (or deeper) may be hazardous or impractical in some diving situations. Therefore, as Farmer and colleagues[21] arbitrarily suggested in 1976, the optimal treatment depth in these situations is 3 atm deeper than the depth at which symptoms occurred. When symptoms begin after surfacing, prompt recompression using tables suitable for the treatment of central nervous system decompression sickness should be instituted. These recommendations thus far seem to be adequate; however, future studies and observations are needed to more precisely define the optimal recompression profile.

Other measures in the treatment of otologic decompression sickness, such as anticoagulants and low-molecular-weight dextran, have not been adequately evaluated. Anticoagulation could cause additional harm, particularly if inner ear hemorrhage has occurred, as indicated in the animal studies. Therefore, anticoagulation is not recommended. Drugs that supposedly increase intracranial and inner ear blood flow are generally not effective in this regard and result in shunting of blood to the periphery; therefore, these agents are not recommended. Conversely, fluid replacement and other measures such as the administration of oxygen-enriched treatment gases, as advocated in the treatment of decompression sickness, are indicated. Whether steroids and salicylates aid the management of inner ear decompression sickness is not known. If hemorrhage is significant, salicylates are possibly undesirable because of an additional anticoagulation effect. Also, salicylates may be ototoxic in high doses.

Diazepam (Valium), 5 to 15 mg intramuscularly, has been noted to significantly relieve vertigo, nausea, and vomiting, which can be severe during otologic decompression sickness. This drug can suppress the accompanying nystagmus and may thus mask a sign of optimal treatment. In many cases, however, the symptoms are so severe that relief is preferred. Monitoring of the respiratory rate and blood pressure after parenteral admission of diazepam is recommended.

A complete otoneurologic examination, with audiometry and electronystagmography, must be conducted as soon as possible after adequate recompression therapy. Patients with a permanent loss of inner ear vestibular function on one side usually become asymptomatic, provided the inner ear on the opposite side is normal and central nervous system vestibular function is normal. Thus, a disappearance of vestibular symptoms within 4 to 6 weeks after the injury does not necessarily indicate recovery of inner ear function because the central nervous system will compensate.

Divers who suffer permanent inner ear dysfunction as a result of inner ear decompression sickness should not be returned to diving. Some authors think that further inner ear injury to the same ear is more likely and could result in extreme danger at depth from the associated vertigo and possible nausea and vomiting. Also, injury to the opposite inner ear during future diving could result in significant disability for nondiving activities, particularly occupational and life skills involving communication and balance.

A diver should be disqualified from future diving if any of the following is present:

- Persistence in either ear of a pure-tone audiometric threshold greater than 25 dB in the frequency ranges of 500 to 2000 Hz
- Speech discrimination score of less than 90%
- Electronystagmographic abnormalities indicating persistent inner ear vestibular dysfunction

DIFFERENTIAL DIAGNOSIS OF INNER EAR BAROTRAUMA AND INNER EAR DECOMPRESSION SICKNESS[56,57]

In some instances, inner ear barotrauma is difficult to distinguish from inner ear decompression sickness. An accurate, prompt diagnosis is important because the proper treatment of these two entities is significantly different. Prompt recompression therapy is essential in the appropriate management of inner ear decompression sickness; recompression therapy should be avoided in cases with inner ear barotrauma unless there is central nervous system decompression sickness or an air embolism. Decompression sickness can occur even when the decompression tables and dive-computer schedules are accurately followed;

Table 22–5. Characteristics of inner ear barotrauma and inner ear decompression sickness

	Inner Ear Barotrauma	Inner Ear Decompression Sickness
Time of symptom onset	During compression (associated with middle ear barotrauma)	During or shortly after decompression
Dive characteristics	Dives not requiring staged decompression Can occur during compression phase of deeper dives Dives with rapid descents Reported cases associated with air diving—can probably occur with helium diving	Dives requiring staged decompression Dives without proper, staged ascents More common during decompression from helium dives—can occur with air diving
Possible associated symptoms	Difficulty with ear clearing and/or ear pain or drainage—frequent May have history of preexisting nasal, sinus, or middle ear disease	None or other symptoms of decompression sickness
Possible associated physical findings	Signs of middle ear barotrauma—frequent	None or other symptoms of decompression sickness

From Farmer, JC: Otologic and paranasal sinus problems in diving. *In* Bennett, PB, Elliott DH (eds.) The Physiology and Medicine of Diving, 4th ed. London, WB Saunders, 1998, p 294.

thus, the differential diagnosis may be difficult if the related dive involves an exposure close to the no-decompression limits. Also, divers occasionally do not know when the symptoms began during the dive, and signs of middle ear barotrauma suggesting inner ear barotrauma or other signs of decompression sickness may not be present. Usually, an accurate history and physical examination allows the physician to differentiate these two entities; however, when the differential diagnosis is difficult, the major factors to be considered include the following (Table 22–5):

- *Time of symptom onset.* Otologic symptoms occurring during compression indicate possible inner ear barotrauma; otologic symptoms starting during or shortly after decompression are more likely related to inner ear decompression sickness.
- *Knowledge of the dive type and profile.* Inner ear dysfunction is more likely to be related to inner ear barotrauma when associated with (1) shallow dives wherein decompression sickness is unlikely, (2) rapid descent, or (3) the lack of conscious efforts to adequately equilibrate middle ear pressure during descent. Also, inner ear barotrauma seems to be more common with air diving, whereas inner ear decompression sickness appears to be more common with deeper, mixed-gas diving.
- *Presence or absence of associated symptoms*. Ear pain, blockage, or fullness during compression is more likely to be related to inner ear barotrauma. Other symptoms of decompression sickness (e.g., central nervous system symptoms, joint symptoms, skin itching) are more likely to be associated with inner ear decompression sickness.
- *Presence or absence of associated physical findings*. Findings indicating middle ear barotrauma are more likely to be associated with inner ear barotrauma. Divers who exhibit inner ear symptoms and who have other signs of decompression sickness, such as other neurologic deficits or skin rashes, should be suspected of having inner ear decompression sickness.

Noise-Induced Hearing Loss: Otologic Injuries Related to High Background Noises

Several studies[12,13,83] have concluded that the high-frequency sensorineural deafness that occurs in the usual diving population could be explained by previous nondiving noise exposures. Divers with no history of previous excessive noise exposure had high-frequency hearing losses similar to the overall nondiving population when allowances were made for such factors as age and cardiovascular disease. Recent studies in Japanese fishery divers and Norwegian professional divers have documented deteriora-

tion in divers over time after elimination of age and comorbid conditions.[84,85] Other authors[86–88] have concluded that professional divers have a higher incidence of sensorineural hearing loss than the nondiving population and that such losses could not be attributed to excessive noise exposure but may be related to otologic barotrauma or decompression sickness.

Investigations by Summitt and Reimers[89] and Murray[90] have demonstrated excessive noise levels ranging from 98 to 120 dBA in pressure chambers from the inflow of gases during compression from the ventilators and in diving helmets from the inflow of breathing gases. Acceptable damage risk criteria for noise-exposure limits at the surface suggest that these levels during diving in pressure chambers and in diving helmets may cause noise-induced hearing losses with exposures as brief as 15 min. It is not known whether the previously noted reversible and depth-related conductive hearing losses, secondary to decreased sound transmission by the eardrum and ossicles in compressed gases,[88–91] are sufficient to provide attenuation from excessive noise during diving. At the Naval Submarine Medical Research Laboratory in New London, Smith[92] indicated that auditory threshold shifts are smaller during noise exposures at depth than during comparable noise exposures on the surface. Thus, the temporary conductive hearing losses caused by the compressed gas environment may provide some protective attenuation. However, Summitt and Reimers[89] noted temporary threshold shifts in air helmet dives. This would indicate that these conductive hearing losses do not provide sufficient attenuation to protect divers from these noise levels.

In any case, the existing noise exposure limits for exposures at 1 ata would seem inappropriate for application to hyperbaric exposures. Until more data are available regarding the actual damage risk from excessive noise in diving, chambers and helmets should be designed to operate as quietly as possible.

PARANASAL SINUS BAROTRAUMA

Paranasal sinus barotrauma has been described in flyers[93,94] and in divers.[95,96] The mechanisms and pathophysiology are related to inadequate pressure equilibration between the air-containing paranasal sinus cavities during ascent, descent, or both. Adequate ventilation and pressure equilibration in the middle ear and paranasal sinuses largely depend on nasal function. Inflammation and congestion of the nasal mucosa, nasal structural deformities, or mass lesions can result in blockage of the paranasal sinus ostia. In the absence of ambient atmospheric pressure changes, such blockage leads to a series of changes within the sinuses consisting of absorption of preexisting air and decreased intrasinus pressure; swelling, engorgement, and inflammation of the sinus mucosa; and collection of transudate in the sinus cavity. When such blockage occurs during descent in diving or flying, the decrease in intrasinus pressure becomes greater and the resulting pathologic changes are more severe, with hemorrhage into the mucosa and often into the sinus cavity. Paranasal sinus barotrauma also occurs during ascent whereby the pathologic mechanism is frequently related to a one-way valve blockage of the sinus ostium by inflamed mucosa, cyst, or polyps located within the sinus. Thus, pressure equilibration may occur during descent but may be impaired during ascent.

In 1976, Fagan and colleagues[96] reported a series of 50 consecutive cases of documented paranasal sinus barotrauma in divers. In 68% of the divers, symptoms developed during or immediately after descent; symptoms occurred after ascent in 32%. Pain was the predominant symptom. The frontal sinus was most often involved, probably because the nasofrontal duct is longer and narrower whereas the communications between the maxillary, ethmoid, and sphenoid sinuses and the nasal cavity are short ostia. The second most common symptom was epistaxis, occurring in 58% of cases. Thirty-two percent of patients noted a history of previous paranasal sinus barotrauma, and 50% had a history of recent upper respiratory tract infections. A history of chronic nasal and sinus problems was also reported by 50% of the patients, and associated signs of middle ear barotrauma were noted in 48%. Symptoms in addition to pain and epistaxis include pain in the upper teeth, occasional paresthesia, and decreased sensation over the infraorbital nerve distribution, as reported by Idicula and coworkers in 1972,[95] Murrison and associates in 1991,[97] and Butler in 1999.[98] The presence of

purulent nasal discharge indicates secondary infection. Serious complications of sinus barotrauma, such as blindness and meningitis, have been reported in the literature.[99,100]

Chronic paranasal sinus disease can predispose to paranasal sinus and middle ear barotrauma during atmospheric pressure changes. The common underlying causes of such chronic diseases are

- Allergy, either intrinsic or extrinsic
- Chronic irritation from smoking, excessive or prolonged use of nose drops or nasal sprays, or exposure to toxic or irritating chemical vapors
- Mechanical obstruction from internal and external nasal deformities, polyps, or neoplasia
- Vasomotor causes from chronic tension, stress, or anxiety

In many patients with chronic paranasal sinus disease, more than one of these causative factors is involved. Exposure to cold dry air normally results in increased nasal blood flow with congestion. Thus, the underlying conditions frequently worsen during the winter months. Secondary bacterial infection is not uncommon and is indicated by the appearance of purulent discharge. Associated chronic inflammation of the lower respiratory tract often occurs, such as that seen in patients with allergies (asthma) or chronic irritation from smoking.

Treatment of paranasal sinus barotrauma includes the use of topical and systemic vasoconstrictor agents to promote nasal mucosal shrinkage and opening of the sinus ostia, with purulent nasal discharge. Cultures and appropriate antibiotics are also indicated. Future atmospheric pressure changes should be avoided until recovery, which usually requires 7 to 14 days. Most of the patients in the series reported by Fagan and colleagues[96] required no treatment; those who did usually responded to nasal decongestants alone, and only a few patients required antibiotics. No patient required sinus lavage or surgery. Patients with symptoms persisting for long periods, who have decreased transillumination of a maxillary or frontal sinus, or who have indications of systemic disease or a chronic underlying paranasal sinus disease should be referred for computed tomography of the sinus and otolaryngologic evaluation.

Systemic and topical adrenergic drugs can improve paranasal sinus and middle ear ventilation. However, cautious observation for rebound phenomenon is indicated, especially with topical nose drops, which can lead to greater nasal congestion and increased difficulty with middle ear and paranasal sinus cavity pressure equilibration. Topical nasal decongestants also cause varying degrees of paralysis in the mucosal microscopic cilia and dissolution of the protective mucous blanket. Thus, prolonged use of these agents can result in chronic nasal irritation and mucosal inflammation, with increased problems of middle ear and paranasal sinus pressure equilibration. Antihistamines, either in combination or singly, can unpredictably cause drowsiness. Also, systemic adrenergic drugs, either singly or in combination, can cause undesired adrenergic effects as well as excessive drying of the nasal and paranasal sinus mucosa and, thus, may be detrimental in some conditions.

Persons who must use systemic or topical decongestants or antihistamines to equilibrate middle ear pressure during diving should be warned that they are not ideal diving candidates, and they should consider undergoing an attempt to identify a chronic and possibly correctable underlying cause (as described earlier) before they return to diving. At the time of a prediving physical examination, persons with a history of possible chronic paranasal sinus or nasal disease—such as chronic nasal congestion or discharge, chronic purulent discharge, frequent upper respiratory infections, middle ear disease, or a condition that required nasal or paranasal sinus surgery—all occurring in the absence of altered atmospheric pressure changes, are certainly less likely to be able to adequately equilibrate paranasal sinus or middle ear pressure when exposed to the pressure changes encountered in diving and should be thoroughly evaluated before being cleared to dive.

OTOLARYNGOLOGIC GUIDELINES FOR THE MEDICAL EXAMINATION OF SPORTS SCUBA DIVERS

Neblett[102] well summarized the guidelines for the medical examination of sports scuba divers. Some of those related to otolaryngology have been reviewed in this chapter.

Ear

HISTORY

A history of ear drainage, middle ear effusions, or middle ear infections in the past 3 years indicates poor eustachian tubal function and an increased likelihood of middle ear barotrauma. The presence of frequent or chronic respiratory tract membrane disease, as noted earlier, also increases this likelihood.

Previous ear surgery should alert the physician to possible continuing borderline or poor eustachian tubal function. Persons who have undergone a previous simple repair of a tympanic membrane perforation can be considered for diving if the eardrum has remained healed and if the ear can easily be autoinflated or cleared at the surface. Velepic and colleagues reported a case series of three divers who returned to diving without incident after tympanoplasty once they had passed audiometric testing and a hyperbaric clearance test.[103] Those who have undergone a previous simple mastoidectomy that has healed well with adequate eustachian tubal function can also be considered for diving.

Those who have undergone a mastoidectomy, which involves removal of the bony posterior external auditory canal wall (a radical or modified radical mastoidectomy), should not dive. A caloric response with vertigo, nausea, and vomiting is more likely if water enters such a cavity. Also, such patients are more likely to have poor eustachian tubal function.

In the past, patients who have undergone stapedectomy or stapedotomy have been discouraged from diving because of the theoretical increased risk of an oval window fistula and inner ear injury with middle ear pressure changes. A retrospective questionnaire conducted by House and Toh[104] found no significant diving-related long-term effects in the hearing or vestibular systems. However, a small proportion of the divers experienced transient otalgia on descent, tinnitus, and transient vertigo. Harrill et al[105] recently reviewed otolaryngologists' recommendations regarding the return to diving after stapes surgery; these authors demonstrated that activity restrictions in terms of flying and water sports varied widely. No current consensus has been established because of the lack of adequate data.

Persons with a history suggestive of Meniere disease, characterized by recurrent bouts of vertigo or hearing loss with tinnitus, or another inner ear disease with recurrent bouts of vertigo, should not dive because vertigo (with possible nausea and vomiting) while one is underwater can result in drowning. Also, such pathologic changes in the inner ear may increase the likelihood of inner ear injury with diving. These are appropriate contraindications even if the audiometric and vestibular results at the time of the examination are not disqualifying.

Divers who have sustained inner ear barotrauma or decompression sickness have previously been advised to discontinue diving. In 1993, Parell and Becker[106] reported on 20 patients who suffered inner ear barotrauma while diving but continued to dive against medical advice. The divers were assessed intermittently for 1 to 12 years; no further deterioration of cochleovestibular function was demonstrated, and the recommendation to cease diving after inner ear injury may have been unnecessarily restrictive.

We recognize that a return to diving after ear injury and surgery remains controversial. The increasing numbers and diversity of recreational divers, many of who have had otologic injury or surgery, sparks debate. There are many anecdotal case reports of both professional and recreational divers who are asymptomatic while diving with cochlear implants, a middle ear prosthesis, and mastoidectomy. However, the number of divers with otologic concerns has not been carefully studied, and therefore safety cannot be accurately estimated. Until more accurate and complete data are available, a conservative approach regarding suitability for recreational diving seems prudent.

PHYSICAL EXAMINATION

The prerequisites for diving include an intact tympanic membrane and the ability to easily autoinflate each ear by a gentle modified Valsalva or Toynbee maneuver. Movement of the tympanic membrane should be visible. Contraindications to diving, in addition to a tympanic membrane perforation, would include the presence of or need for ventilation tubes, which indicates inadequate eustachian tubal function in the absence of significant atmospheric pressure changes.

Eardrums frequently exhibit whitish plaques of varying size. If the drum is intact, it moves well, and the patient can autoinflate, diving may be considered. A thin flaccid tympanic membrane (monomeric or dimeric eardrum) indicates poor eustachian tubal function and an increased likelihood of tympanic membrane perforation as well as middle ear barotrauma with diving. The presence of a cholesteatoma, or a skin-lined sac within the middle ear, indicates chronic middle ear disease and poor eustachian tubal function and should be a contraindication to diving. Also, water may enter and contaminate a cholesteatoma sac with infection and further bone erosion.

Stenosis or atresia of the ear canal as well as chronic or acute external otitis, until healed, should be contraindications to diving. Patients who have cerumen impactions should not dive until the cerumen is removed. Persons with marked narrowing of the canal from ear canal osteomas should be cautioned about diving.

LABORATORY INVESTIGATIONS

The presence (in either ear) of a pure-tone audiometric threshold worse or greater than 25 dB in the frequency range of 500 to 2000 Hz, a speech discrimination score of less than 90%, or electronystagmographic abnormalities indicating inner ear vestibular dysfunction should disqualify a person from diving. Such inner ears may be more susceptible to future inner ear barotrauma, inner ear decompression sickness, or both. Injury to the opposite inner ear during future diving could result in significant disabilities.

Nose and Paranasal Sinuses

Candidates being considered for suitability for scuba diving should have patent nasal airways and should not have acute or significant chronic nasal or sinus symptoms. Conditions that cause nasal obstruction, such as polyps, septal deviations, or chronically edematous nasal mucosa, as occurs with allergies or chronic irritations, increase the chance of middle ear and paranasal sinus barotrauma. Patients who require frequent or chronic use of oral and topical decongestants, antihistamines, or steroids should be carefully evaluated. Such persons are at increased risk for barotrauma. Many have underlying respiratory tract allergies that involve the lower as well as the upper respiratory tract; bronchial asthma would be a contraindication for diving. Persons who require decongestants in order to dive should be cautioned about rebound phenomena and adrenergic side effects; they should be advised to abort dives if adequate middle ear pressure equilibration does not easily occur every 2 feet of descent. The use of these drugs does not usually allow safe pressurization in the presence of an acute upper respiratory tract infection; diving should be delayed until the acute episode has subsided.

Larynx

Any patient who has intermittent and chronic aspiration suggesting an incompetent larynx should not be cleared for diving. The presence of a laryngocele should also disqualify a patient for diving until the problem is corrected. A tracheotomy or tracheostomy is an absolute contraindication to swimming as well as diving.

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CHAPTER 23 Neurologic Consequences of Diving

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NERVOUS SYSTEM

The nervous system as the principle target of diving injury was recognized in 1877.[1] The "bends" literature before 1975 describes "pain-only" versus "central nervous system" decompression sickness (DCS). The pathophysiology of spinal DCS was suggested by Hallenbeck[3] in 1976; however, he noted the rarity of brain involvement. In that same year he called attention to the rarity of brain DCS in divers.[2,3] At about the same time, Peters and colleagues[4] presented evidence that brain involvement does occur, often unrecognized. Others have written of divers being "punchy" after multiple exposures.[5,6]

BRAIN

Air embolism attacks the brain directly and immediately, presenting as an acute condition with focal vascular event brain injury. Seizure, aphasia, and hemiparesis are most common, although cortical blindness occurs disproportionately often. The frequent occurrence of cardiorespiratory arrest suggests embolization of the posterior circulation.[7–9]

Encephalopathy produced by hypoxemia is a grim feature of drowning or of air supply failure from any cause and is not specific to divers.

The common denominator is the vulnerability of nerve cells to ischemia, losing function after a few minutes of hypoxia; cell death occurs shortly thereafter. Because nerve cells have no anaerobic metabolism, they require a constant supply of oxygen and glucose from the blood to survive. The homeostatic limits are narrow. Loss of consciousness occurs when Pa_{O_2} falls to 20 to 30 mm Hg and when glucose falls to 20 to 30 mg%. The former limit is frequently approached in long breath-hold dives. The mechanisms of ischemia are different in DCS, air embolism, and asphyxia.

In DCS, brain dysfunction is often manifested by confusion, drowsiness, fatigue, and indifference. Pain and paraplegia due to spinal cord DCS occurs earlier and more often and attracts the most attention; however, examiners often call attention to decreased mentation. Retrospective analyses indicate that cognitive and psychological defects do occur from DCS.[10]

Peters[4] reported 10 divers who had definite neurologic DCS. Neuropsychological testing demonstrated evidence of cerebral injury in 7 of the 10 divers with a history of DCS.[4] None of the control divers showed such abnormalities. Vaernes and Eidsvik[5] reported similar results. Both studies suggest that cortical events can be present even in patients with only cord symptoms and signs. Therefore, all cortical symptoms must be evaluated.

The "punchy diver" hypothesis was tested in retrospective study of Australian abalone divers who commonly made long, shallow-water, air-compressed dives and were casual about decompression obligations.[6] However, divers did not describe a high incidence of definite DCS, were rarely treated for it, and did not recognize or admit to cognitive dysfunction. Testing with a battery of psychometric instruments, Edmonds[6] found evidence of intellectual impairment in 11 of 24 divers. Although half of the overall group had reported incidents of DCS, the recognized events did not correlate completely. Studies in Norway may also cause concern about the long-term effects of diving.[11,12]

In a Norwegian epidemiologic survey, 156 commercial air and saturation divers were examined, 23 of whom had performed

Case 1

The association of spinal cord and brain DCS is illustrated in this case history.

A 28-year-old commercial diver made a series of dives in 50°F water. On the fourth day of the job, during ascent from a 272-foot dive for 39 min, he experienced pain in both elbows, hips, ankles, shoulders, and knees. Standard surface decompression was altered to U. S. Navy (USN) Table 6, and symptoms resolved completely by the end of the treatment. He did not dive on the following day, but on the next day he made a similar dive to 272 feet. He completed in-water and surface decompression but an hour later experienced abdominal pain, lumbosacral pain, and weakness of his legs such that he had to drag himself back to the chamber.

He was treated according to USN Table 6 with two extensions at 60 feet. He walked from the chamber pain-free but exhausted. The next morning, he drove to visit friends and became lost in familiar surroundings. He ran his automobile off the road several times, fortunately without injury. On the following day, 2 days after completion of treatment, he flew home by commercial air. When he arrived, he was again sleepy and confused and had recurrent back pain. A long course of retreatment was carried out, with improvement in pain but with persistent ataxia, extensor toe signs, absent abdominal reflexes, absent reflexes in the left upper extremity, decreased pain, temperature, and vibratory sense below the T6 level, and with continuing complaints of decreased concentration, memory, and word finding. The neurologist who examined him at that time saw evidence of cord lesions at two levels. Psychometric testing indicated difficulties with concentration, word finding, arithmetic, and short-term memory. His family found him changed in personality.

air-only diving. The divers reported more symptoms from the nervous system than 100 controls.[11] The divers also had more clinical neurologic findings that correlated with diving exposure and DCS prevalence as well as more abnormal-appearing electroencephalograms but not more evoked-potential or magnetic resonance imaging abnormalities. In 40 commercial divers with deep dives (190 to 500 m), abnormal findings occurred in 18% compared with 5% of controls.[12]

This ongoing work generated the consensus conference on the long-term health effects of diving in June 1993. This work continues to raise issues surrounding nervous system involvement in symptomatic and asymptomatic divers.

Most of our knowledge in this area is based on clinical information. However, Calder and Palmer[13] have reported autopsy evidence of diffuse cerebral white matter disease in divers.

This case illustrates severe DCS involving multiple joints, the spinal cord, and the brain itself in a patient with a large gas burden. His course was complicated by flying after his initial treatment for DCS.

Brain injury from arterial gas embolism, principally a hazard to scuba divers, is anatomically discrete. The temporal profile is a sudden focal event usually in a known vascular distribution. The onset is commonly heralded by seizure or cardiorespiratory arrest as the expanding bubbles occlude major arterial supplies. Hemispheric lesions frequently cause seizures from cortical ischemia. Hemiparesis, aphasia, and cortical blindness all indicate emboli to the hemispheres. Cardiorespiratory arrest implies diffuse cortical hypoxia or focal brain stem involvement of the reticular activating system. Bilateral cortical blindness suggests that the posterior circulation is embolized.

Arterial gas embolism is a major cause of death in scuba diving.[14] At autopsy, unless the coroner is forewarned and performs the necropsy with tissues submerged, bubbles may not be seen. In addition, most patients have undergone prolonged arrest and resuscitation and have suffered whole brain hypoxia. Acute ischemic changes in the distribution of a specific artery are thus obscured. Some patients who succumb to typical events of arterial gas embolism are given a coroner's diagnosis of drowning.

However, many persons do well. There are a surprising number of accidents in which arterial gas embolism is manifested by seizure, hemiparesis, coma, and even respiratory arrest wherein the subject recovers spontaneously without recompression treatment. With recompression, however, the response is often dramatic and immediate.[9]

A patient in cardiac arrest may regain consciousness and alertness as the chamber is being pressurized, indicating that a simple reduction in bubble diameter is sufficient to restore circulation in the deficient area and to allow bubbles to pass through microcirculation.[15]

Immediate response to treatment is not always complete, and some patients are left with a focal neurologic deficit such as hemiparesis. Even in these cases, long-term disability is frequently less than that which follows ischemic stroke.

Two factors may contribute to the relatively good outlook for recovery in survivors of arterial gas embolism:

- Most diving accident victims are young in contrast with stroke victims. Cardiopulmonary function is healthier, collateral vessels are larger and more abundant, and, perhaps most important, more brain cells are available for retraining. The brain cell attrition that occurs with aging reduces plasticity of cortical function because it reduces the number of new synapses that can be established.
- The embolus is gas, not clot, and when it is eventually absorbed, with or without treatment, downstream flow may be restored. This is in contrast with thrombosis, in which the vessel is permanently lost. This likely occurs more often, more quickly, and more completely than in embolic events.

When arterial gas embolism and DCS occur together, injury can be severe. Scuba divers are particularly vulnerable to this combination because of the structure of the life support system. When the scuba diver close to the no-decompression limit in a deep dive runs out of air, the stage is set for a damaging chain of events. An out-of-air ascent from depth carries a high risk of pulmonary overpressure. If arterial gas embolism occurs, bubbles are introduced into an already supersaturated system and DCS is induced. Alternatively, the victim of arterial gas embolism who convulses may induce DCS from mechanical shear forces in a saturated system. The diver may convulse, be hemiparetic on surfacing, and then experience the abdominal pain and leg weakness of spinal cord DCS. This grievous combination may occur even without violation of safe diving practices. The following case history illustrates this situation.

Case 2

A scientific diver made an 85-foot, 25-min dive in the mid-Atlantic. He made a normal controlled ascent and stopped briefly at 10 feet to exhale before surfacing. Observers confirmed that his ascent rate was not rapid. As he climbed into the inflatable boat, he suddenly lost motor control and became unconscious. He awakened in a few minutes, quadriplegic and insensate in all four extremities. He then experienced a prolonged generalized seizure. When he next regained consciousness, he had excruciating pain across his chest. He was returned to the ship and treated with oxygen. The vessel got underway immediately and steamed for port, but it was 9 hours after the accident before helicopter evacuation could be accomplished. The diver was treated according to USN Table 6A and extended, but with little response. He was transferred to a saturation chamber and treated for 7 days. At the end of treatment, he had sensory loss to T4, weakness in both hands, flaccid paraplegia from the mid-thoracic region, and no sphincter control. There was gradual improvement during several months of rehabilitation. A year later, he was able to walk with forearm crutches, had moderate weakness of foot extensors, and had regained sphincter control.

Several years before this event, this diver had surfaced from a dive and had a brief episode of unilateral weakness. He was thought to have had an "undeserved" embolism from which he made a complete recovery. He had been treated for asthma in childhood but had not had symptomatic asthma as an adult.

Several points are illustrated here:

- Cerebral air embolism occurred in spite of good diving practice. The diver made a normal ascent, yet symptoms occurred immediately after surfacing; he lost consciousness and convulsed.
- Spinal cord DCS was precipitated by air embolism. The thoracic sensory level establishes the diagnosis as spinal cord disease. The introduction of air bubbles into the blood stream via the arterial circulation precipitated bubbling in a partially saturated solution. The dive did not exceed the no-decompression limits, but

dive time was sufficient to incur a considerable nitrogen load. Bubbling was further favored by the mechanical effect of the prolonged generalized seizure.
- Childhood asthma may be a continuing hazard to divers, even if apparently asymptomatic in adulthood. There are a number of documented cases of undeserved arterial gas embolism in this setting.
- The decompression illness precipitated by air embolism in a subject with large gas burden (type III DCS) is likely to be severe and resistant to treatment.

SPINAL CORD

The mechanical structure of the vertebral column shields the cord from most external injury. The redundant collateral arterial supply ensures that the cord will be nourished directly from the aorta; however, the anterior spinal artery is not from a single supply, so "watershed" areas exist in the vascular territories of the spinal cord.

The venous drainage of the cord, slowed and made pendular by respiratory pressure changes, makes it uniquely vulnerable to venous infarction.[3] Arterial insufficiency may play a part as well, particularly in the relative watershed areas, i.e., the dorsal root entry zone.[16]

This vulnerability causes a unique spinal cord disease, clinically different from any other neurologic syndrome. Spinal DCS presents a clinical picture of diffuse multilevel cord disease.[17] Extrinsic pressure on the cord, such as occurs in compression fracture or metastatic tumor, results in paraparesis with sensation loss to the level of the lesion. Likewise, a meningioma, compressing the cord from one side, may produce a Brown-Séquard lesion with loss of motor function, vibratory and joint sense on the compressed side, and loss of pain and temperature sense on the contralateral side. Anterior spinal artery thrombosis, most often following trauma to the thoracic aorta, causes injury to the anterior cord with resultant paraparesis, pain, and temperature loss, sparing the posterior columns that carry position and vibratory sense. This is most common in the mid-thoracic area, as a watershed area occurs at about T8 due to the supply of the artery of Adamkewitz. Spinal DCS, by contrast, attacks the cord at multiple levels and at random sites. Fiber tracts may be interrupted at different levels, and a nip may be taken out of the root entry zone here and there. The clinical result may be a painful transverse mid-thoracic myelopathy, but may as well be a combination of pain, sensory loss, and motor weakness at multiple sites all along the neuraxis.

Although the recovery from spinal DCS is often gratifying, there may be residual symptoms and findings that are unique among neurologic diseases.[18,19] For example, the sensory loss found in mid-lumbar DCS may be patchy. A physician accustomed to dealing with spinal cord trauma expects to find a level of sensory loss at or slightly below the level of the lesion. DCS may lead to patches of preserved sensory function interspersed between areas of dense anesthesia (Table 23–1).

During recompression treatment, an anesthetic patch may shrink in area, perhaps disappearing, only to reappear on decompression. This sequence is unlike any other neurologic disease.

Spinal DCS may also produce a chronic pain syndrome with paroxysmal features. We have seen several patients who have been treated for spinal DCS with nearly complete recovery but who have had recurrent paroxysmal bouts of pain in the distribution of the original insult.

Case 3

An experienced commercial diver made an heliox dive in the Gulf of Mexico and was decompressed on a proprietary 225/60 table. He shifted to air at 100 feet, then to 50/50 nitrox. After finishing surface decompression, he had pain in the left side of his chest, which increased and then subsided. Two days later, he made a second and similar dive and again experienced pain in the chest, worse than on the first occasion. He made a third and similar dive to the same profile on the next day. On descent, he became quite chilled when he shifted to heliox at 100 feet and remained so for the rest of the dive. On ascent, he felt warmer when he shifted back to air at 100 feet but became chilled again with the shift to 50/50 nitrox.

When he emerged from the surface decompression chamber, he had a wide band of pain across his chest and abdomen,

Table 23–1. Differentiating features between disorders of the spine and spinal cord decompression sickness

	Favoring Spine Disorder	Favoring Decompression Sickness
History	Prior lumbar or cervical radiculopathy, spine surgery	Absence of prior spine involvement
	Documented chronic sensory or motor deficits	No prior neurologic deficits (before dive)
Symptom onset	Prior to or during the dive (before ascent)	Post dive
Diving exposure	Benign: shallow depth, short duration, within No-D limits	Provocative: deep depth, long duration, at or beyond No-D limit
Physical		
Pain	Localized to specific dermatome, usually unilateral, commonly cervical or lumbar	Pain localized to a joint, bilateral, or involving multiple dermatomes, often trunk or abdomen
Paresethesia anesthesia	Dermatomal, usually cervical or lumbar, usually unilateral	Involves multiple cord levels, often bilateral
Cerebral/cerebrellar findings	Absent	May be present (with accompanying arterial gas embolism)
Tendon reflexes	Depressed or absent at level of involvement, often unilateral	Hyperreflexic, often bilateral
CT/MRI	Disk herniation, narrowed neuroforamina, no cord lesions demonstrated	Cord lesion demonstrated

extending from the neck to the groin. He felt weak and exhausted. He was evacuated by helicopter to a recompression facility, where he arrived about 12 hours after surfacing. Treatment according to USN Table 6A did not give relief, and he continued to complain of pain radiating from his mid-thorax into the right knee. Repeated treatments were carried out for 8 days, and the pain gradually subsided. During this hospitalization, neurologic examination showed minimal decrease in vibratory and position sense in the feet, with intact pain perception in the arms but decreased pain perception from T4 to T12 bilaterally. Motor function was intact and reflexes unremarkable. Auditory- and somatosensory-evoked potential responses were normal.

Pain had subsided by the time of discharge, 10 days after the dive. Altered sensation over the trunk persisted. Several days later, he began to experience numbness of the third and fourth fingers on the left hand and electric shock–like sensations shooting down his arm from the level of the mid-humerus into the fingers. These lasted for a few minutes, subsided, then returned. There was sharp pain in the elbow. He had similar sensation in the right arm with episodes occurring once or twice a day, or every other day, for 2 months. The pains lasted for minutes to hours.

On one occasion, while driving, he experienced shooting pains so severe that he became nauseated and vomited. His arm pain extended from the shoulder to the elbow, and below the elbow his arm felt as though it were being massaged. He returned to the hyperbaric facility, where his right hand was noted to be sweating profusely. This was confirmed with cornstarch testing, and the area of increased sweating extended to the elbow. He was treated with carbamazepine, which suppressed the pain but caused nausea and diarrhea. When he discontinued medication, the pain recurred. Codeine and meperidine were necessary for pain relief. For several days, his left arm and hand felt clumsy and he dropped things.

He was unsteady on his feet, particularly in diminished light, and had fallen on one occasion. He did not have vertigo or disturbance of bowel, bladder, or sexual function, and he did not complain of weakness. There was no previous history of DCS.

Neurologic examination 8 months after the injury demonstrated normal gait, reflexes, and strength. Abdominal reflexes were present and symmetrical. The anal reflex was decreased on the left. There was an unusual sensory loss extending from T4 on the right and T5 on the left down to T12 (right) and T10 (left). There was slight decrease of pain perception on the left side of the perineum. Vibratory and joint senses were normal (Fig. 23–1).

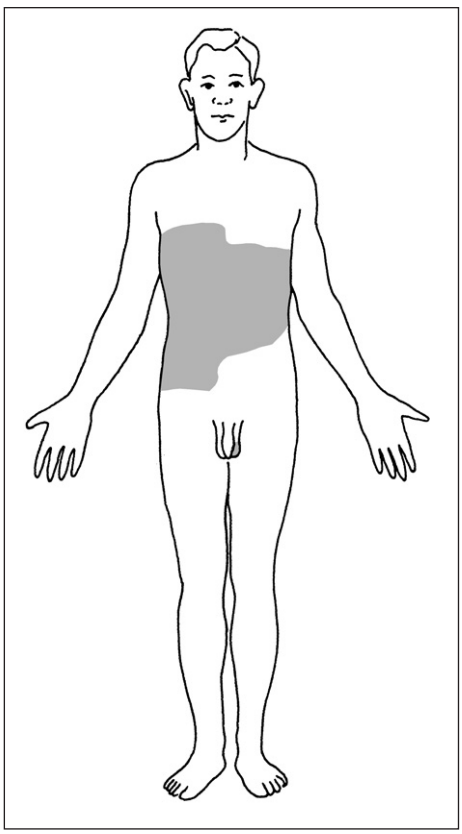

Figure 23–1. Pattern of pain and temperature loss in a diver with persistent paroxysmal limb pain following decompression sickness. The girdle-like anesthesia is consistent with multiple-level lesions in the dorsal root entry zone.

This case illustrates spinal DCS with bilateral thoracic myelopathy. The findings are remarkable in that motor function was entirely spared and disturbance in the posterior columns was minimal. The paroxysmal tic-like character of the pain, its relationship to the original site of injury, and its response to carbamazepine suggest a lesion in the dorsal root entry zone. This is clinically consistent with the experimental lesions produced by Palmer.[16] The condition most resembles the syndrome of "tonic painful seizures" described in multiple sclerosis, which is thought to represent multiple sclerosis plaque in the dorsal horn of the cervical spinal cord.[20]

PERIPHERAL NERVES

Peripheral nerve injuries are rare in diving. Those that occur in DCS usually involve nerves that traverse a tightly confined area in which a bubble may cause mechanical compression. These include the facial nerve in the facial canal,[21] the trigeminal nerve as it traverses the foramen ovale and rotundum, and the median nerve as it enters the hand beneath the carpal tunnel.[22] Scarcely any other nerves are affected.

Facial baroparesis may result from a single bubble hit but has also occurred in reverse sinus barotrauma on ascent. Both instances are benign and resolve in hours to weeks.[23,24] Peripheral mononeuropathy from DCS has been described in the oculomotor (third) nerve and in branches of the trigeminal nerve.[25] Traumatic peripheral neuropathy from a weight belt has been reported to cause "scuba diver's thigh" in the distribution of the lateral cutaneous nerve.[26] Any nerve predisposed to focal trauma (i.e., ulnar, median, radial, brachial plexus, peroneal) can likely also be traumatized in this setting.

The definition of peripheral nerve injury is important. It means nerve injury outside the spinal cord, peripheral to the dorsal root ganglion. Peripheral nerves are purely nerve fibers; no nerve cell bodies are present. Injury to the peripheral nerve causes pain, dysesthesia, sensory loss, and weakness only in the distribution of that nerve. These injuries in DCS are usually "neuropraxic." The axon fibers are preserved, the myelin is lost, and the outlook for recovery is good. Obviously, focal traumatic etiologies must be sought. Neurologic symptoms originating from other spine disorders may be confused with symptoms of spinal cord DCS (see Table 23–1).

Some attention should be devoted to the precise diagnosis of peripheral nerve injury and its differentiation from type I, or pain-only, DCS and type II, or spinal, DCS. The point here is that spinal DCS with pain,

dysesthesia, anesthesia, and weakness is common in the upper extremities as well as the lower. If the venerable concept of pain-only bends is to be honored, it must be strictly applied to pain only in a single joint. Multiple joint pain and bilateral pain must be regarded as serious DCS. If there are physical findings, such as weakness or loss of sensation, nerve tissue must be involved. This nearly always means the spinal cord. However, if physical findings prove injury to a single peripheral nerve, such as the median nerve in the carpal tunnel, the diagnosis reverts to type I DCS. Symptoms resulting from a single lesion in the limb, whether in the elbow, joint, or carpal tunnel, are of equal significance. Therefore, if one is satisfied that the weakness or sensory loss is anatomically located in a single nerve outside the cord, a lesser diagnosis is justified.

Finally, the anatomic diagnosis influences intensity of treatment. A physician requires some fortitude to change treatment and commit a patient and a chamber crew to additional hours of treatment. The more precise the anatomic diagnosis, the easier the decision becomes.

Is lesser treatment justified? Probably not. The diagnosis of DCS demands treatment. It has been common practice for diving supervisors, both naval and commercial, to treat type I with USN Table 5 and type II with USN Table 6 (see Chapter 10).[26,27] Table 5 takes 2 hours and 15 min; Table 6 takes 4 hours and 45 min, a difference of 2 hours and 30 min. A lesser diagnosis, therefore, shortens the task. Because inadequate treatment may allow recurrence of symptoms, many authorities, thinking that the use of Table 5 results in an unacceptable recurrence rate, have recommended that it be abandoned. It has not been. USN Table 5 is included in the latest edition of the *U.S. Navy Diving Manual* with the statement that it is to be used for pain-only DCS if symptoms resolve in 10 min at 60 feet. If symptoms persist beyond that time, treatment is extended to that described in Table 6.[27,28]

Therefore, why bother to determine the type of DCS? If the level of treatment is determined by response to treatment, is it really necessary to make fine anatomic distinction between spinal cord and peripheral nerve or even between type I and type II? Our view is that the diagnosis of DCS requires treatment according to USN Table 6 as a minimum. The cost of retreatment and the results of inadequate treatment far exceed the cost of 2.5 hours of additional chamber time. The distinction between type I and type II should not determine the choice of initial treatment. Physicians want to make the correct diagnosis, even if the treatment does not vary.

There are other considerations. Both the course of treatment and long-term management may be influenced by anatomic diagnosis. The patient who presents with spinal DCS requires maximal treatment directed at the prevention of permanent cord damage. If patients do not completely recover with standard treatment (e.g., single application of USN Table 6), they are permanently disqualified from diving.[29] If, however, patients have a median neuropathy or peripheral facial palsy, a complete recovery is likely in a matter of weeks and they may return to diving. The next case illustrates the difficulty in classifying symptoms into type I or type II.

Case 4

Contact was made from an oil rig off the Kenai Peninsula in Alaska. The diver (also the company owner) had made two decompression dives on the previous day. On day 2, he dove to 127 feet for 78 min on air. He decompressed using a 130/80 table and surface decompression with oxygen. He surfaced from the chamber without incident but awakened 2 hours and 20 min later with severe pain inside the left wrist, extending 6 inches above the wrist joint and into the fingers. He was treated according to USN Table 5 with relief before 60 feet. On conclusion of treatment, he went back to work on deck. After the shift, he retired only to awaken in 3 hours with recurrent pain and numbness in the first three fingers of the left hand. He was treated again, this time according to USN Table 6A, and again had immediate relief under pressure. Numbness waxed and waned. When he surfaced, the pain recurred somewhat.

Instructions for examination of the hand were given by radiotelephone. There was no weakness of intrinsic muscles of the hand, but there was decreased sensation in the second, third, and fourth fingers. He was treated yet again according to Table 6 and surfaced without recurrent pain. The numbness persisted. Final treatment

consisted of the schedule in Table 5 on the third day. The patient was evacuated to Anchorage and examined by a neurologist, who found weakness in the short abductor of the hand. Nerve conduction study of the median nerve yielded normal results. The diver's symptoms subsided without residual effects. He was ready to return to work in 1 week.

It was concluded that the diver had a discrete lesion in the median nerve, probably in the carpal tunnel at the wrist, and that this was resolved entirely with treatment. Type I DCS was the cause of a single lesion in one limb. Note that this did not meet the more limited definition of pain only; the diver had both sensory loss and weakness, and a distinction had to be made to exclude spinal cord disease.

VESTIBULAR DECOMPRESSION SICKNESS

Vestibular DCS is principally a heliox phenomenon, occurring almost exclusively when decompressing divers make the 170-foot switch from heliox to air. This probably represents a counterdiffusion phenomenon. At the moment the valve is turned, the diver is enormously supersaturated with helium, which off-gases rapidly across a 6 ata gradient. Even though helium has excellent diffusion characteristics, some bubbling may occur and DCS may result. The selectivity of this phenomenon for the vestibular system is explained by the anatomy of the semicircular canals. The tiny counterdiffusion bubbles confined in the semicircular canals bend the hair cells and cause vertigo. Although a similar phenomenon surely occurs in the spinal fluid, it causes no symptoms because the bubbles there have no such fragile receptors to stimulate.

PREEXISTING NEUROLOGIC DISEASE

Although the most important limiting conditions that pose problems for the physician interested in diving medicine relate to conditioning and cardiovascular and pulmonary functions, questions are occasionally raised about several neurologic problems which, if they exist in an individual, may affect their diving potential.

Epilepsy

About 0.8% of the population has epilepsy. It most often begins in childhood, but the risk continues indefinitely and increases again in late adulthood. A majority of patients with epilepsy are under treatment with medication. With good medical management, such patients are able to complete school, compete in the job market, participate in sports, and drive. Some want to dive.

Patients with uncontrolled seizures are obviously unsuited for diving, driving, or any other activity that exposes them to risk should they lose consciousness. A seizure underwater carries great hazards of drowning, uncontrolled ascent, and embolism. It exposes other members of the dive party and the rescue party to additional hazards. Should a serious accident not result, the diver's companions are still faced with a diagnostic dilemma. Conventional doctrine holds that a seizure in the water or after surfacing is presumptive evidence of air embolism. Treatment is obligatory. Any loss of consciousness (e.g., hypoglycemia or cardiac syncope) carries similar risk. Parenthetically, this is analogous to the dilemma that occurs when an insulin-dependent diabetic has altered consciousness after diving.

The fitness-to-dive decision is less clear-cut in patients with well-controlled epilepsy. Several considerations are important:

- Does diving increase the likelihood of seizures in an epileptic patient?
- Should a patient under treatment with anticonvulsant medication be allowed to dive?
- Should a person who has outgrown epilepsy be allowed to dive?

Each of these issues should be addressed.

Individuals with epilepsy are said to have a lower seizure threshold. This means that stimuli that induce seizures in some percentage of the population are more likely to do so in those who have epilepsy. Examples of such stimuli are prolonged sleep deprivation, alcohol and sedative withdrawal, visual stimulation with rapidly flashing lights, and hyperventilation. The latter is of particular interest to divers. Hyperventilation at atmospheric pressure is routinely used to test for

seizure susceptibility. Breath-hold divers regularly hyperventilate before a dive.

The effect of hyperbaric oxygen exposure on epileptic populations has not been studied. However, it is well established that high partial pressures of oxygen may induce seizures in normal persons. For many years, Navy diving standards required that candidates undergo an oxygen tolerance test in which they breathe pure oxygen in a chamber for 30 min at a depth equivalent to 60 fsw. This test was designed to screen out those candidates who are susceptible to oxygen toxicity. About 1% of healthy candidates were reported to have seizures under these conditions. Experience in civilian hyperbaric chambers indicates that the risk is actually much lower. The oxygen tolerance test has been discontinued for want of predictive value. It was based on the assumption that all divers will be exposed to high P_{O_2} in their work or in treatment and that the test might help to eliminate susceptible candidates.[30] Both assumptions are defensible. It is possible, although not proven, that epileptics are at increased risk for oxygen convulsions.

The patient with a medically controlled siezure disorder leads a fairly normal life, with no apparent physical handicap. In most jurisdictions, an epileptic can obtain a conditional driver's license if seizure-free for 1 year.[31,32] However, an epileptic cannot obtain a pilot's license or a commercial driver's license and does not meet the physical requirements for military or commercial diving.[27]

As sport divers, patients with seizures may choose to put their own life at risk. Should a seizure occur underwater, these persons may drown or suffer air embolism. Both of these accidents have occurred in nonepileptic patients with oxygen convulsions. These are personal risks. However, the diver who has trouble underwater also increases the risk of other members of the diving and search parties. As a student, a person with epilepsy imposes an unusual obligation and liability on the instructor. Value judgments must be made. The law takes notice of the epileptic driver, not because of the risk to the individual but because of the risk to the public; the public safety is greatly endangered by an automobile driver who has a seizure. With the diver, the public risk is less but not negligible.

The legal system generally accepts that a seizure-free interval of 2 years or less (under treatment) constitutes control that is sufficient to operate a motor vehicle. The risk of recurrent seizures in the controlled subject is nevertheless several times greater than in the general population.[33] Because the risk of seizures after 2 years of control are similar, public policy recognizes that driving has great social and economic importance and accepts the risk. Is this risk acceptable in a diver?

The controlled patient with a seizure disorder also pays a price in side effects of medication. Virtually all anticonvulsive medications have some sedative effect. There is great variation among individuals with respect to both dosage and susceptibility to sedation. The average dose of medication for epilepsy usually produces only mild sedation. This sedation is nevertheless measurable on performance testing. This has a bearing on divers. All sedative medications can be expected to increase the hazard of nitrogen narcosis in the manner as does alcohol.

About 20% of children with epilepsy outgrow the disorder by age 21. Those who have been seizure-free for 5 years without medication are generally regarded as cured for legal purposes, except for the special requirements of flying and military service. The risk of recurrent seizures in this population is nevertheless considerably greater than in the general population.[34] These considerations lead to the following recommendations:

- Persons with epilepsy are disqualified for military and commercial diving, without exception.
- In persons with epilepsy who have been seizure-free for 5 years and who take no medication, the statistical risk of recurrent seizures is smaller than during the first 5 years. There is no definite evidence that diving will increase the risk of recurrence, but these persons should be advised to avoid hyperventilation and cautioned that elevated partial pressures of oxygen may precipitate seizures. However, not diving at all is likely best.
- Persons with controlled epilepsy (taking medication, seizure-free for 2 years, therefore meeting the requirements of most driving jurisdictions) are nevertheless advised *not to dive*.[35] Although driving is important to livelihood, diving is not. The risk, to both individuals and their companions, is inconsistent with the pursuit of sport.[35,36]

Spinal Surgery

Although indications for spinal surgery continue to be a matter of dispute, many operations are performed. Since the advent of lumbar laminectomy and anterior interbody fusion, many thousands of Americans have had undergone one or more of these operations. Many want to dive. There are two considerations: one theoretical, the other practical.

- An operation that compromises the paravertebral venous plexus might be expected to increase the likelihood of DCS. Such compromise certainly occurs in lumbar laminectomy, in which coagulation of bleeders in the venous plexus is routine. Anterior interbody fusion, which approaches the spinal canal without laminectomy, has relatively less bearing on the paravertebral plexus.
- Spinal surgery causes spinal disability. Under the best circumstances, a patient recovers from spinal surgery with altered structure. If the operation is entirely successful and the patient is symptom-free, the patient nevertheless emerges with a skeleton that has been physiologically and structurally altered. In California Workers' Compensation Appeals Board parlance, a patient who has had such surgery is usually regarded as "disabled for heavy work." In the same parlance, this means that such persons have lost 30% of their previous capacity for lifting, pulling, and pushing.

Diving is hard work. A spinal structural disability is disqualifying for military and commercial divers. Whether a sport diver should be disqualified on this basis depends on how much pulling and hauling must be done on the surface. Someone else can lift bottles or dress the diver out, but the person will not necessarily have help to climb the ladder into the boat or to heave over the gunwale after surfacing. Table 23–1 lists some features that differentiate symptoms and signs between chronic spine disease and DCS.

Migraine

Queries about the history of migraine frequently appear on divers' medical evaluations, and candidates are sometimes turned away on this account. No data justify prohibiting diving in a person with migraine. Migraine is an exceedingly common problem. Perhaps more than half of the world's population will have some experience with migraine during their lives. Five percent will see a doctor about it.[37] Some of these people certainly dive. Even those with frequent migraine do not usually experience an increased incidence of headache related to diving, nor are they unusually vulnerable to DCS.

Vasodilating headache can be induced by elevated levels of carbon dioxide, just as it can be ameliorated by high Po_2. Decompression with falling Po_2 might provoke migraine, at least theoretically. Subjects with severe or complex migraine, in which the prodromal event is hemianopsia or hemiplegia, may present a diagnostic dilemma. Consideration of DCS or air embolism might even result in an unnecessary recompression treatment. However, there is little evidence to suggest that migraine poses a significant hazard to divers. Subjects with migraine of such severity as to interfere with diving will usually sort themselves out of a program. People with classic migraine (e.g., focal neurologic defects such as hemanopsia, aphasia, and hemiplegia with their migraines) could be at increased risk for problems.

Cerebral Palsy, Paraplegia, Multiple Sclerosis, and Muscular Dystrophy

These disparate illnesses are considered here because some patients with these conditions have learned diving and certainly more will do so. The issue here is principally physical stamina. Beyond that, each case must be considered individually. Patients with cerebral palsy, for instance, have an increased incidence of epilepsy. The previous discussion applies here. If they have not had seizures, are adequately conditioned, and can pass the swimming tests, there should be no blanket contraindication.

Paraplegic patients require special thought. In traumatic paraplegia from spinal cord injury (SCI), there is at least a theoretically increased risk of spinal cord DCS because of injury to the circulation of the spinal cord. Spinal cord disease, whether from trauma or demyelinization (multiple sclerosis), is evidence of structural abnormality. A person who undertakes diving with an altered spinal cord would seem to be at greater risk for DCS because some of the population of nerve fibers would already be lost. No data are available to confirm this opinion.

Multiple sclerosis is a distressingly common disease. Diving and exposure to a hyperbaric environment probably would not make it worse. The 1990s saw a brief flurry of interest in the use of hyperbaric oxygen to treat multiple sclerosis. Most studies have shown such treatment to have little effect.[38,39] Patients with multiple sclerosis, however, have limited stamina, and vigorous physical exercise usually serves them poorly. Fatigue is the most common symptom. Most neurologists advise such patients to avoid exhaustion, and this also means the avoidance of chilling or overheating. Since disease altering medications (i.e., interferons) cause flu-like symptoms when given as therapy, they could affect diving symptom interpretation in some situations.

Muscular dystrophy occurs in different forms. Those with early-onset generalized progressive dystrophy (Duchenne muscular dystrophy or even Becker muscular dystrophy) are severely handicapped and will not be divers. Some patients with limited disease (limb girdle, facioscapulohumeral dystrophy) may have sufficient strength to dive effectively when they are young; however, if they are symptomatic at rest it would be difficult. Diving will not make them worse. Patients with myotonic dystrophy (type I or II) are likely to do poorly when chilled, which increases the symptoms.[40] Many dystrophies have associated cardiac arrhythmias, especially myotonic dystrophy.

Risking repetition, the main criteria must be physical stamina and agility. Patients with post polio syndrome or previous acute immune demyelinating peripheral neuropathy (Landry Guillain-Barré) or chronic immune demyelinating neuropathy (CIDP) would experience this situation. Patients with preexisting neurologic disease or other handicaps need not be disqualified from diving if they are well conditioned and can function independently in the water. If their handicaps are so severe as to require regular assistance from other divers or instructors, common sense must prevail.[41]

DIVERS WITH CHRONIC SPINAL CORD INJURY*

Permanent spinal cord injury (SCI) produces physical handicaps that could preclude safe recreational diving, but medical opinion among rehabilitation professionals and the weight of experience argue that recreational diving for the paraplegic or tetraplegic patient can be a safe and gratifying endeavor. Although research on the effects of sport diving in this population is virtually nonexistent, several organizations have devised programs of instruction for disabled divers and diving professionals who seek to work with these people.

The Handicapped Scuba Association, organized in 1981, has developed a program of instruction for divers that stratifies them based on ability and prescribes protocols for safe recreational diving. Many young, healthy paraplegics can function at a level that requires few special accommodations. For divers with more significant impairments, diving is done in groups of three, with one diver being certified in rescue diving. Handicapped Scuba Association instruction materials are available through centers certified by this organization.

Several problems should considered by the physician evaluating a diver with SCI.

Thermoregulation

The individual with SCI experiences profound dysautonomia below the level of injury.[42,43] Impaired responses to temperature extremes include inability to shiver, aberrant vasoconstriction, and inability to perspire. The resulting poikilothermia demands adaptation, including careful choice of exposure suits and planning of dives to accommodate this impairment. Hyperthermia while above the water can be as serious as hypothermia while immersed.

Respiration

SCI produces a restrictive type of pulmonary dysfunction due to paralysis of accessory muscles of respiration and alteration in the mechanics of the diaphragm.[44,45] The severity of dysfunction is directly related to the level of injury, with quadraplegics more impaired than paraplegics. Forced vital capacity is reduced, and forced expiratory volume in 1 second is increased. Impaired pulmonary reserve and early fatigue of still-innervated respiratory muscles prompts individual consideration of equipment and

* This section contributed by Mark Fredrickson.

the dive plan. Regulators with decreased resistance are available. The increased work of breathing at depth requires caution when planning a dive.

Urologic Function

Neurogenic bladder dysfunction after SCI is almost universal.[46,47] Although several management strategies exist, the preferred method is intermittent catheterization, usually combined with anticholinergic therapy to moderate detrusor hyperreflexia. For the diver with SCI, immersion diuresis, common in divers without SCI, is especially troublesome. Exuberant diuresis, possibly exaggerated by the dependence on humeral mechanisms to control blood pressure after SCI, can lead to bladder distension and potentially lethal autonomic disturbance as described later. Some divers have used a Foley catheter to prevent bladder distension, but experience is anecdotal.

Autonomic Dysreflexia

The patient with SCI at T5 or above is susceptible to abrupt and potentially lethal disturbance of the autonomic nervous system.[47,48] Loss of descending inhibition over the sympathetic nervous system and acquired hypersensitivity to catecholamines can lead to initiation of unregulated sympathetic nervous system activity. Clinically, this presents as acute, dangerous hypertension with bradycardia, headache, and diaphoresis with associated nausea, vomiting, and vision changes. Seizures, intracranial hemorrhage, and fatal arrhythmias have been reported.

The trigger for this sympathetic storm is nociceptive input into the spinal cord triggering the reflex sympathetic nervous system response. Any stimulus that would be "painful" in the neurologically intact patient can trigger autonomic dysreflexia. The most common cause is bladder disturbance, especially a distended bladder. Divers with SCI must pay scrupulous attention to managing neurogenic bladder dysfunction and planning to accommodate immersion diuresis.

Sensory/Motor Function

Paralysis is the most obvious complication of SCI. Fortunately, its impact on function can be ameliorated. Because the diver with SCI depends on upper limb function, the choice of exposure suits is important. Avoid equipment that impairs shoulder and arm mobility. Training can accommodate impairment in somatosensation, including proprioception. Impairment of protective somatosensation (nociception) predisposes to injury.[49] Exposure suits provide skin protection in addition to their role in moderating poikilothermia.

Although the recreational diver with SCI presents certain problems, a physician with experience in managing SCI and an understanding of the demands imposed by scuba diving can counsel these patients and form a plan to minimize risk. Adherence to thoughtful, cautious safety protocol as devised by the Handicapped Scuba Association or other adaptive scuba programs is advised.

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CHAPTER 24 Pulmonary Disorders

Tom S. Neuman

The problem of advising patients with pulmonary disorders on the risk of diving is difficult. Clearly, the most important issue concerning pulmonary fitness to dive is the question of *whether the candidate has the ventilatory capacity required of the exercise load associated with diving.* In most cases, exercise capability is limited by cardiac output; exercise is rarely limited by pulmonary function on the surface. However, ventilatory function is affected by various facets of immersion, increased gas density, and the mechanical breathing resistance of diving equipment, all of which must be accounted for in determining whether a diver is fit to dive in terms of pulmonary function. Harries[1] observed the following relationship (for a respiratory quotient near 1 and for well-conditioned athletes):

$$\dot{V}O_{2max} = \dot{V}E_{max} \times 0.026 + 0.44$$

Thus, if one assumes that a scuba diver's required $\dot{V}O_2$ is approximately 2.5 L/min (the $\dot{V}O_2$ approximately required to swim at 1.2 knots), the minute ventilation required for such activity would be approximately 75 L/min. At the surface and in a dry environment, this would be little problem for most people; however, maximum voluntary ventilation (MVV) decreases with increasing gas density and decreases with immersion. In addition, most people can sustain a minute ventilation about equal to only 70% of their maximal ventilatory volume. Because immersion appears to decrease MVV by approximately 15% and increased gas density appears to reduce maximal breathing capacity (MBC) by 50% at 100 fsw, it should be clear that essentially normal pulmonary function is required to sustain a $\dot{V}O_2$ of 2.5 L at 100 fsw,[2] even without considering the added effects of the underwater breathing apparatus.

The other major issue concerning pulmonary fitness to dive is whether an underlying lung disease might predispose a diver to decompression sickness or an arterial gas embolism (AGE). It is easy to understand the risks of being unable to swim back to the boat if caught in a strong current or of not being able to equalize if there is trouble clearing the ears, but it is difficult to determine whether there is increased risk of a diving-related injury because of abnormal distribution of ventilation or altered airway function. This task is further hampered by a paucity of data for most conditions and the strong opinions of many in the field.

ASTHMA

Asthma is probably the most common pulmonary disorder that physicians are asked to evaluate in a diver or diving candidate. Fortunately, most physicians have a basic understanding of asthma, even though the participants of the CIBA Foundation study group on the identification of asthma concluded that "asthma could not be defined on the information at present available."[3] For practical purposes, it might be more useful to define asthma as a disorder "characterized by increased responsiveness of the airways (i.e., hyperactivity) to various stimuli and by resultant smooth muscle contraction and obstruction."[4] Yet asthma can still be subclassified, and most current classifications attempt to define syndromes based on specific precipitating factors and specific patterns of response. These classifications are important because the natural histories of these syndromes appear to be different, and therefore the recommendations of diving physicians may be different as well.

One of the more common asthma syndromes is atopic asthma. This is characterized by onset in childhood and by association with allergic rhinitis or allergic dermatitis. Attacks may be precipitated by various sensitizing agents. Children who experience this

asthma syndrome in the first few years of life do not necessarily have asthma that persists into adult life. In one study, only 15% of 10-year-old children whose asthma began before the age of 2 years had persistent wheezing.[5] If the precipitating cause of attack was infection, it was found that 50% of children were considered to be cured when reexamined 20 years later.[6] Adults do not seem to fare as well with asthma as children do. With adult-onset asthma, the percentage of patients who become free of disease with time is much lower than with children[7–9] and the proportion of severe to mild cases seems to rise steeply with age.[10]

In many patients, attacks cannot be ascribed to a specific precipitating agent or event. This syndrome is called *intrinsic asthma;* however, there is a great deal of overlap between groups, and many persons present with mixed symptoms.[11,12]

Another important syndrome is *exercise-induced asthma.* The cause of this syndrome, most frequent in the young, was for many years unclear. Some evidence now suggests that the underlying cause is not hyperventilation or hypocapnia but, rather, a cold stimulus to the tracheobronchial tree in susceptible persons.[13,14]

Other less common syndromes are the *aspirin-sensitivity triad* (nasal polyps, urticaria, and asthma following aspirin ingestion) and *occupational asthma.*

Thus, asthma is not a single disease, and there is great heterogeneity among asthmatic patients. As already mentioned, the factors that precipitate attacks vary tremendously. But equally importantly, the actual location of obstruction seems to vary with different patients depending on what the various pulmonary function tests are thought to measure.

Most importantly, there are considerable differences in the degrees of airway obstruction reversibility demonstrated by asthmatics. Some asthmatics demonstrate completely normal pulmonary function test results, including measurements of pulmonary mechanics and of regional ventilation distribution[15] between attacks. Others, although asymptomatic, continue to show evidence of airway obstruction even after vigorous therapy. Under such circumstances, the distinction between chronic obstructive pulmonary disease (COPD) and reactive airway disease (i.e., asthma) becomes blurred.

As a result of these marked differences in prognosis, intermorbid pulmonary function, and baseline pulmonary function, recommendations concerning diving should consider the individual patient's specific asthma syndrome and history.

The Case *Against* Diving for People with Asthma

The major concern of many practitioners in permitting an asthmatic person to dive is that such a person might be dangerously susceptible to pulmonary barotrauma and cerebral air embolism even during a normal ascent. This concern is based on an understanding of pulmonary physiology and on extrapolations derived from that understanding. Liebow and colleagues[16] have suggested that under certain conditions, partial pulmonary obstruction in large airways can lead to AGE. Similarly, Schaefer and colleagues[17] have demonstrated that overpressurization of the lung can lead to AGE, and Colebatch and colleagues[18] have shown that decreased compliance is associated with AGE. All of these factors are operative in asthmatics. Together with these factors, some asthmatics have significant noncommunicating air spaces as demonstrated by differences in measured lung volume compared by helium-dilution techniques and whole-body plethysmography.[19] Additionally, the acute asthmatic is usually hyperinflated, and as has been demonstrated, not only is overpressurization required to produce AGE, but overinflation is required as well.[17] A more sophisticated study on asthmatics revealed that even in supposedly asymptomatic persons, abnormalities of ventilation/perfusion ratios can be significant.[20] This experiment demonstrated that as many as half of the lung units were behind completely closed airways that were supplied only by collateral branches.

Epidemiologic data are hard to interpret because, according to most experts, asthma is considered to be a contraindication to diving. Nonetheless, in Australia and New Zealand,[21] asthma was present in 9% of scuba diving fatalities. A study by Divers Alert Network[22] revealed that as many as 12% of AGE victims have asthma. In another Divers Alert Network study,[23] asthmatic divers were found to have a fourfold increase in relative risk for decompression illness (decompres-

sion sickness and air embolism). Finally, other data suggest that as many as 50% of cases of air embolism are not associated with panic, out-of-air, or breath-holding ascents.[24] In other words, apparently half of the instances of AGE occur in divers ascending normally and not holding their breath. This implies that undetected underlying lung disease may play a role in these accidents. These concerns, however, deal only with barotrauma. Case reports also suggest that asthmatic divers died because their exercise tolerance was so poor that they drowned on the surface after their diving activity.[21]

Thus, a wealth of physiologic and epidemiologic data suggests that it would be unsafe for the active asthmatic to dive, and this explains the (until recently) almost universal recommendation that asthmatics not dive.

The Case *for* Diving for People with Asthma

From this discussion, it appears that there could be no rational argument for allowing asthmatics to dive. Closer analysis of the data, however, yields different conclusions. In the years that the University of Rhode Island recorded diving fatality statistics, only 1 of 2132 deaths was in a person with asthma; furthermore, no other information concerning the death is available; the exact cause of death is unknown, and the role played by asthma is uncertain.[25–34] Looking more closely at the data from Australia and New Zealand, Walker,[35] referring to the alleged overrepresentation of asthmatics[36] in diving fatalities, stated, "This appears to be untrue in relation to Australia and New Zealand." Reexamining all the combined Divers Alert Network data reveals that there is little evidence of excess risk[37]; indeed, the 95% confidence interval of the odds ratio (relative risk) for all asthmatics is 0.80 to 2.99 (not significant) and for current asthmatics is 0.65 to 5.33 (not significant).[38] Analysis of pulmonary function in approximately 40 cases of air embolism at the Marine Science Center on Catalina Island over a 5-year period revealed only one asthmatic,[39] and analysis of 18 consecutive fatalities at the Los Angeles County coroner's office between 1985 and 1990 failed to identify any asthmatic victims.[40] Finally, the Institute of Naval Medicine in England found no asthmatics in 140 cases of AGE they investigated from 1965 to 1985.[41] Importantly, in the few studies done in the United States,[42–44] it appears that asthmatics are represented in the diving population in much the same proportions as is seen in the nondiving population.

Interestingly, the international community does not uniformly agree that asthmatics should be prohibited from diving. The British SubAqua Club allows persons with extrinsic asthma to dive (although they prohibit those with exercise-induced asthma from doing so); however, persons needing bronchodilators regularly or those on steroids are cautioned against diving. The Club further recommends that asthmatics not dive within 48 hours of a wheezing episode. A study of asthmatic divers conducted via a questionnaire from a diving magazine reported that 104 asthmatic divers conducted more than 12,000 dives without any cases of AGE. Of this group, 9 persons wheezed daily, dived within 1 hour of wheezing, and logged more than 1200 dives without incident.[45]

Careful review of the papers historically quoted most often to support the recommendation that asthmatics not dive reveals interesting inconsistencies. Liebow and coworkers[16] hypothesized that a broncholith in a large airway was the cause of a fatal air embolism in an escape trainee; yet this cannot explain the bilateral evidence for barotrauma found at autopsy. Similarly, the paper by Wagner and colleagues,[20] often used to justify the exclusion of all asthmatics from diving because asymptomatic asthmatics have marked abnormalities of ventilation-perfusion ratios ($\dot{V}/\dot{Q}$ distribution),[46] becomes less compelling when examination of the data reveals that pulmonary function test results were profoundly abnormal in their patients. Finally, the consensus of the participants of two recent symposia (predominantly physicians and researchers interested in diving medicine) was that asthma did not predispose to diving-related pulmonary barotrauma[47,48] and that the limiting factor for asthmatics is adequate ventilatory capacity underwater.

What then are reasonable conclusions in light of these disparate data?

First, data are insufficient to uniformly reject asthmatics as diving candidates. Second, there is a group of asthmatic divers who dive with an acceptable safety record. Third, there *may* be an approximately

twofold risk in asthmatics. In light of these conclusions, it seems appropriate to make the following recommendations (pending the availability of additional data):

- Asthmatic persons whose pulmonary function test results (normal flow rates and static lung volumes), including functional reserve capacity and vital capacity, are normal (within 2 standard deviations of established norms) can be considered as candidates for diving.
- Persons with exercise-induced asthma or cold-induced asthma, in general, should be treated as any other asthmatic candidate. If pulmonary function test results are normal and if an appropriate exercise or cold challenge (at a level of exertion well above that expected during scuba diving) yields normal results, the candidate can be cleared for diving.
- Following an episode of asthma, a person should not dive until it has been determined (by appropriate pulmonary function tests, usually patient-assessed peak expiratory flow) that airway function has returned to normal.

In summary, it must be recognized that asthma is a heterogenous group of conditions. The stimuli that produce airway dysfunction differ from person to person, and the duration and severity of airway obstruction can vary tremendously even in the same individual. Thus, the decision as to whether an asthmatic should dive must be individualized; however, persons with normal airway function (whether or not on medication) appear to be at low risk for idiopathic pulmonary barotrauma and subsequent air embolism.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

The issue of whether a patient with COPD (chronic bronchitis and emphysema) should dive is in many ways the same as in the individual who has asthma. The same theoretical arguments apply to the individual with COPD concerning the increased risk of AGE, except that in the person with COPD, airway function never returns to normal. Thus, the diver or diver candidate with COPD may not be at increased theoretical risk when an attack takes place; rather, that individual may have an increased theoretical risk at all times.

From a practical point of view, by the time patients with chronic lung diseases become symptomatic they are usually incapable of sustaining the exercise capacity necessary to dive; as a result, it is extremely rare to see a diver with significant COPD. Additionally, COPD is generally a disease that develops after decades of exposure to tobacco smoke, and it is therefore a disease of older persons, which again makes it rare to encounter a diver with COPD in a diving medicine practice. Finally, by the time COPD can be detected clinically, such major physiologic alterations have occurred that there is little argument that such persons should be advised against diving merely on the basis of their exercise tolerance. Thus, the question of advising someone with COPD is really the question of advising someone who is asymptomatic but who has abnormal pulmonary function test results. Some patients with COPD may also have a component of reactive airway disease; that is, their pulmonary obstruction varies with external stimuli, and the obstruction is treated with bronchodilating drugs similar to those used for asthma.

If we are to be consistent and treat persons with COPD as we treat asthmatics, then persons with clear-cut laboratory evidence of COPD should be advised not to dive. In practice, this evidence is defined as pulmonary function test results that are more than 2 standard deviations from normal (Table 24–1). Unfortunately, the exact definition of normal is still unclear; as a result, persons with mild disease may have pulmonary function test values that overlap predicted normal values between 2 standard deviations and the mean.[49] As a result, persons whose isolated values may be at

Table 24–1. Approximate lower limits of normal at fifth percentile level

Parameter	Percent of Predicted
Vital capacity	Below 75
Functional reserve capacity	Below 70 or above 130
Residual volume	Below 80 or above 120
Forced expiratory volume in 1 second (FEV_1)	Below 80
Forced expiratory volume in 1 second/forced vital capacity % (FEV_1/FVC %)	Below 85
Forced expiratory flow ($FEF_{25–75}$)	Below 65

From Clausen J: Pulmonary function testing. *In* Bordow RA, Moser KM (eds): Manual of Clinical Problems in Pulmonary Medicine. 2nd ed. Boston, Little, Brown & Company, 1985.

the low end of the normal range should undergo more extensive testing if their clinical history suggests chronic lung disease. If, however, further studies confirm preliminary observations in submarine-escape trainees that pulmonary function tests lack useful predictive value for predicting pulmonary barotrauma, the use of such tests as criteria for diving (other than to assess exercise capability) will have to be reconsidered.[50–52]

Interestingly, a recent case report suggests a causal link between poorly ventilated areas of the lung and AGE. This case is fairly compelling because the AGE occurred in a hypobaric chamber and gas embolisms in a dry environment are extremely rare. Thus, the association between a pulmonary cyst and an AGE in this environment must be viewed with some concern.[53]

PNEUMOTHORAX

By definition, a pneumothorax is a collection of air within the pleural space. Pneumothorax can be classified in several ways, but for the purposes of advising a diving candidate who has had a pneumothorax, it is best to look at pneumothoraces as spontaneous, traumatic, or iatrogenic.

Spontaneous Pneumothorax

Spontaneous pneumothorax occurs in a patient without any antecedent trauma and without previous physician intervention. In a young, otherwise healthy person without any apparent underlying lung disease, the pneumothorax is usually caused by the rupture of congenital subapical blebs. This person is usually a man between 20 and 30 years of age who is tall, thin, and a cigarette smoker.[54] Another mechanism of pneumothorax is overdistention of distal air spaces by partial bronchial obstruction acting as a one-way valve. Eventually, this results in disruption of the air spaces and air dissects back along bronchovascular planes to the mediastinum. As the process continues, air can either dissect into the soft tissues of the neck (causing subcutaneous emphysema) or rupture into the pleural space, resulting in a pneumothorax.[55] The latter mechanism is thought to be the cause of subcutaneous emphysema in patients suffering from asthma.

Spontaneous pneumothorax can also occur because of more severe underlying lung disease. All of the diffuse interstitial lung diseases (eosinophilic granuloma, sarcoidosis, pneumoconiosis, interstitial pneumonitis) seem to predispose to a pneumothorax; spontaneous pneumothorax is also common in patients with COPD (especially bullous emphysema[56]).

Once a person suffers a spontaneous pneumothorax, recurrent pneumothoraces are likely. In one study,[57] approximately 40% of persons without apparent underlying lung disease who had one pneumothorax had a second. The average time between the first and second episodes was approximately 18 months (range, 4 to 35 months). Approximately one third of those who had a second pneumothorax went on to have a third. In another study[58] of persons (again without apparent underlying lung disease) treated with tube thoracostomy for spontaneous pneumothorax, approximately 50% had an ipsilateral recurrence. The average time span to recurrence was 2.3 years. Approximately two thirds of those who had a second pneumothorax went on to have a third.

Traumatic Pneumothorax

A traumatic pneumothorax can be due to either blunt or penetrating trauma. In the case of blunt trauma, rib fractures cause lacerations of the lung surface, although other mechanisms can produce pneumothorax. In penetrating trauma, the pneumothorax can be due to lung injury or to a direct leak from the chest wall.

Traumatic pneumothoraces caused by isolated injury to the chest wall should not pose any risk to a diver; however, most traumatic pneumothoraces are associated with underlying lung injury as well. If the injury was severe enough to lead to radiographic changes, it could have led to areas of air trapping. As a result, many authorities think that such persons should be advised not to dive, although extensive diagnostic and hyperbaric chamber testing might define those at greatest risk.[59]

Iatrogenic Pneumothorax

Iatrogenic pneumothoraces generally occur after invasive thoracic procedures such as

subclavian line placement, thoracentesis, transthoracic needle aspiration of the lung, and thoracotomy. Iatrogenic pneumothorax is also commonly due to positive pressure ventilation.

Iatrogenic pneumothoraces can be due to pulmonary surgery or nonpulmonary procedures. Pneumothoraces that are due to nonpulmonary procedures (such as subclavian line placement and cardiac or mediastinal surgery during which the pleura was opened) should not be a contraindication to diving. If pulmonary surgery was the cause of the pneumothorax, it is not the pneumothorax per se that may increase the diver's risk, but rather the underlying lung disease. Additionally, with the almost universal use of the stapling device for suture lines in pulmonary parenchyma, surgeons generally do not need to follow anatomic planes with precision. This in turn may lead to distortion of architecture and to areas of air trapping.[59] As a result, persons who have undergone previous pulmonary surgery are generally advised not to dive. Thus, any lung disease, procedure, or event that can result in air trapping is considered a contraindication to diving.[60–64]

Tension Pneumothorax

Any form of pneumothorax can be complicated by conversion to a tension pneumothorax. This entity occurs when the rent in the pleura acts as a one-way valve and air is continually introduced into the pleural space. This progressively leads to complete collapse of the involved lung and to shifting of the mediastinum to the uninvolved side. With compression of the contralateral lung and compromise of venous return to the heart, tension pneumothorax can rapidly result in death, even in otherwise normal persons.[65]

The information just given provides a firm *theoretical* basis for advising persons who have had previous spontaneous pneumothoraces against diving. Should a pneumothorax occur underwater, a reduction in ambient pressure while a diver is surfacing could cause a simple pneumothorax to become a tension pneumothorax as the pleural gas expands (see the discussion of Boyle's law in Chapter 2). This could result in death. The previous information also suggests that recurrences are possible after several years; the fact that a pneumothorax has not occurred for 2 to 3 years does not ensure that it will not recur. Finally and equally important, the presence of subpleural blebs (the cause of most spontaneous pneumothoraces) strongly suggests that areas of the lung are poorly ventilated and trap air and may be at risk for causing AGE. For these reasons, a history of spontaneous pneumothorax with or without underlying lung disease is generally considered a contraindication to diving.[60-64]

However, these arguments are theoretical. As with asthmatics and persons with COPD, the available United States diving fatality statistics have not shown a single reported death attributed to a tension pneumothorax or an AGE that was ascribed to a previous spontaneous pneumothorax or previous thoracic surgery.[25–34] As mentioned previously, underlying pulmonary problems have not been noted frequently in survivors of AGE.[39] As a result, these contraindications to diving must be considered in light of the diver's maturity, level of understanding, responsibility, and willingness to accept presumably increased risk.

RESTRICTIVE LUNG DISEASES

Restrictive lung diseases are much less common than asthma; thus, it is not surprising that there are not enough hard data to assess whether such processes increase a diver's risk of pulmonary barotrauma. That said, however, there are suggestions that such processes do increase a diver's risk. As noted earlier, Coltebatch and colleagues[18] noted an association between increased elastic recoil and pulmonary barotrauma. In addition, Benton and associates[66] noted a relationship between small lung volumes (a common finding in restrictive diseases) and AGE in submarine escape trainees. A 1999 case report[67] described an apparent AGE in a patient with asymptomatic sarcoidosis. This event also occurred in a dry hyperbaric chamber dive, again giving this association much more weight than would normally be the case for a single report.

SPECIAL TESTS

Methacholine or Histamine Challenge

The methacholine challenge is highly useful in evaluating a patient in whom the history

and diagnosis of asthma is in question. A small number of unusual asthmatics are unresponsive to methacholine,[19] but essentially all asthmatics show hyperactivity to nonspecific agents.[4] Thus, in a patient with a clear-cut history of asthma, bronchial provocation is pointless because that person will have a positive response. Indeed, in such a setting, bronchial provocation testing might even be dangerous. On the other hand, if the history is questionable and if episodes are not clearly bronchospastic, then methacholine challenge can be useful in identifying patients with asthma.

Methacholine challenge is probably not indicated in the diagnosis of exercise-induced asthma, however. In that setting, pre- and postexercise pulmonary function tests are more specific and certainly less dangerous. Unfortunately, although methacholine testing is extremely sensitive, it is not specific for bronchospastic asthma. In some patients with so-called *cough asthma,* pulmonary function test results are normal and there is no history of episodic wheezing, but methacholine challenge tests yield positive results. The sole manifestation of asthma in such persons is a relatively prolonged period of coughing following an upper respiratory infection.[68] In many other persons with so-called *twitchy airways,* pulmonary function test results are abnormal following upper respiratory infection and patients show a positive response to methacholine for as long as 2 months after the acute infection.[69] Thus, although useful, a positive bronchial provocation challenge is not equivalent to the diagnosis of asthma.

Finally, because the diagnosis of asthma per se does not appear to warrant diving proscription, a methacholine challenge test to establish the diagnosis of asthma in questionable cases does not appear to be warranted.

Specialized Pulmonary Function Testing

Because the results of routine pulmonary function tests show considerable overlap of mild disease with the low end of normal function, more sophisticated tests may be indicated on occasion. When the pulmonary function test results of divers or candidates are below the 5% probability of normalcy, such testing is probably not warranted because the diagnosis of lung disease is relatively secure; however, if results are low-normal and the person has a history of heavy smoking or another reason to suspect early COPD clinically, a more complete evaluation of static lung volumes can reveal a pattern of obstructive lung disease. Such evaluation may require helium dilution and body plethysmography and can, at times, detect significant noncommunicating air spaces.

Ventilation Scanning

Finally, on rare occasions, ventilation scanning with radioactive xenon or technetium-99m may be useful to determine whether areas of the lung are poorly ventilated and therefore at risk for pulmonary overinflation. Generally, a single breath scan in this setting is inadequate. A full study using wash in equilibrium and, most importantly, washout scanning is required to detect poorly ventilated areas. Typically, very poorly ventilated lung zones may fill apparently normally with radioactive gas during a single slow inhalation. This abnormality can be best detected by observing delayed clearance of radioactivity during the washout phase of the study.[70]

High-Resolution Computed Tomography

High-resolution computed tomography (CT) has become useful in defining abnormalities of pulmonary parenchyma that cannot be visualized using more conventional techniques. Tetzlaff and colleagues[71] studied 15 divers who sustained an AGE: 13 had abnormalities on CT scan, of which 8 were explained by the injury and 5 were lung cysts that did not clear in 6 to 12 months. The data from this study suggest that some divers who sustain an AGE may have lung disease that can be identified by CT. However, this small study is inadequate to allow one to conclude that CT is warranted in all patients with AGE. In the absence of epidemiologic studies that correlate abnormalities on high-resolution CT with any type of diving accident, CT cannot be recommended at present.

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CHAPTER 25 Cardiovascular Disorders and Diving

Alfred A. Bove

Sport diving involves more than 3 million divers in the United States. Although most participants receive their diving training in their local communities, many travel to tropical regions of the world to dive. Many diving candidates have chronic illnesses, and many have some form of cardiovascular disease. As the population ages,[1] more divers will be found to have cardiovascular diseases. Coronary artery disease, hypertension, and valvular disease are present in the sport diving population, and diving may result in problems for patients with specific types of heart disease, such as disorders of the atrial septum, long QT syndrome, and left-ventricular dysfunction. This chapter reviews cardiac disorders that can be affected by diving and discusses the assessment of patients with cardiovascular disease for diving fitness.

An important effect of diving on the cardiovascular system is related to the work and exercise involved. Exercise requires an increase in oxygen utilization to support the required skeletal muscle activity; therefore, the heart and circulation are always affected by diving. The cardiovascular system undergoes an immediate response to exercise.[2,3] From the cardiovascular standpoint, exercise is any activity that increases the oxygen consumption above basal levels (Fig. 25–1). Thus, the heart and cardiovascular system respond to swimming, walking with heavy gear, climbing ladders, and performing work relating to diving as forms of exercise that require increased cardiac work.[4] The principle that most tissues and organs contain a functional reserve is well accepted and applies also to the heart.[4,5] Thus, the heart at rest is working at a small percentage of its maximal capacity, and an accurate assessment of the limitations of heart disease may require measurement of maximal cardiac performance. The maximal capacity of the heart and circulation may be reduced early in heart disease but may be undetected for long periods unless the patient or physician tests the reserve and finds it diminished or unless cardiac impairment progresses to the point where the loss of reserve affects resting cardiac performance.

WORK LOADS RELATED TO DIVING

In assessing capability to dive, one must evaluate cardiovascular reserve capacity in the diving candidate. This can be done by formal testing or by determining that a diving candidate can exercise at a level appropriate for diving. Exercise testing may also be useful in detecting predictors of sudden death[6] and may provide insight into arrhythmias produced by abnormal conduction pathways.[7] The need to measure cardiovascular reserve is well accepted,[8,9] and exercise stress testing has become a useful clinical means of assessing cardiovascular reserve. Although exercise testing is often used to detect coronary disease,[10–12] its application in testing for cardiac reserve is also well documented.[7,8] With the addition of radionuclide techniques for measuring left-ventricular performance during exercise,[13,14] it is now possible to assess overall exercise capacity, to measure specific responses of the heart to exercise, and to identify abnormal cardiovascular responses.[15] The physical stress imposed by diving can be simulated by these standard clinical tests, and the results can be used to assess the capability to dive.

With diving candidates who have heart disease, it is important to understand the relationships among physical work, myocardial oxygen consumption, and blood flow to the myocardium. An understanding of these

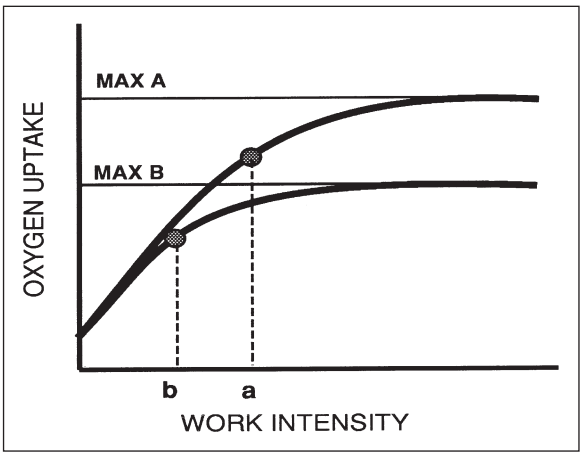

Figure 25–1. Comparison of two different curves of oxygen consumption vs. workload. The *upper curve* represents a conditioned person; the *lower curve* represents a less conditioned one. Workloads *a* and *b* represent 70% of maximum for each person. Note that the person with the higher maximum oxygen consumption can sustain greater workloads without reaching the anaerobic threshold.

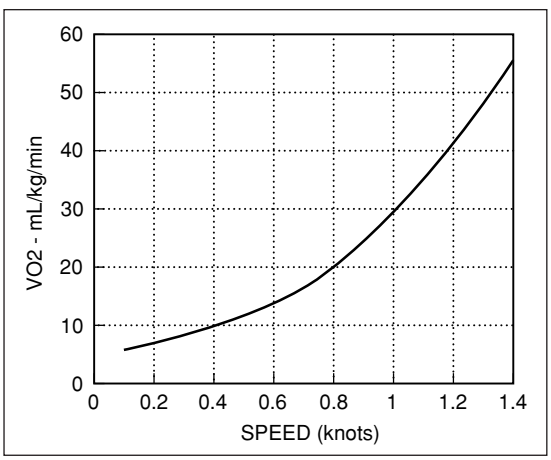

Figure 25–2. Oxygen consumption for underwater swimming. Curves are fitted to the data using a second order polynomial. (Data from United States Department of the Navy: U.S. Navy Diving Manual, revision 3, vol 1. NAVSEA 0994-LP-001-9010. Washington, D. C., Department of the Navy, 1993.)

relationships is the basis for assessing the performance of persons with heart disease and for determining their ability to dive.

Basal oxygen demand appears to increase in divers at rest underwater.[16] This is likely related to the need for increased heat generation, even in temperate water. Similar changes in metabolic need can be demonstrated in marine mammals.[17] Pendergast[18] expressed energy needed for swimming with full scuba gear in terms of oxygen used per kilometer. Tests of a group of divers in average condition yielded values of 30 L O_2/km for women and 50 L O_2/km for men. For an average-sized male swimming at 33 m/min (100 ft/min), oxygen consumption is about 24 mL/kg/min, which is similar to the value found in U.S. Navy studies (Fig. 25–2). This is a high workload that is uncommon in sport scuba diving and in most commercial diving.

Because swimming energy is proportional to the square of velocity, workloads for higher swimming speeds are tolerated only by extremely fit divers. A diver with a maximum oxygen consumption of 40 mL/min/kg can tolerate swimming at 1.3 knots for a few minutes, at which point the diver experiences extreme hyperventilation to compensate for lactate production, and fatigue develops rapidly. Swimming at 60% of maximum (about 24 mL/kg/min), a work level that is at or slightly below the anaerobic threshold, can be sustained for long periods because lactate does not accumulate in the blood.[18] For a diver with a maximum oxygen consumption of 40 mL/kg/min working at 60% to 65% of maximum capacity, sustainable swimming speed is about 100 ft/min; for the diver with a maximum oxygen consumption of 25 mL/kg/min, sustainable swimming speed is about 70 ft/min.

Safety considerations suggest that the sport diver should be able to tolerate a peak workload of about 40 mL/kg/min and a sustained workload of about 24 mL/kg/min (60% of maximum) to ensure safety when in an adverse diving environment, when another diver must be rescued, and when swimming a long distance becomes necessary. Oxygen consumption can be expressed in multiples of the resting value (3 to 3.5 mL/kg/min). This relative measure is expressed in metabolic equivalents (METS), where 1 MET is equivalent to resting oxygen consumption. A maximum capacity of 40 mL/kg/min is equivalent to about 13 METS. When divers cannot achieve this level of conditioning, they should be advised to avoid difficult diving

environments that might stress them beyond their physical capacity. The severe dyspnea that accompanies workloads above 65% of maximum aerobic capacity (the anaerobic threshold)[19] often leads to panic and increases the risk of drowning. This might be the case in elderly divers and in those with medical disorders that limit exercise capacity. Workloads for commercial and military divers are related to the specific task to be accomplished while diving. Conditioning requirements for these two populations are usually more stringent than those for sport divers.

CORONARY ARTERY DISEASE

Coronary artery disease is the most prevalent life-threatening disease in the United States. Nearly 3 million Americans per year are afflicted with the disease, and over 700,000 die from coronary artery disease each year.[20] From the physiologic standpoint, the basic abnormality of coronary disease is partial or complete obstruction of one or more epicardial coronary arteries (Fig. 25–3). Complete or partial occlusion of a coronary artery limits blood flow to the myocardium, and in the presence of increased myocardial demand or active coronary vasoconstriction, the myocardium becomes ischemic. If ischemia is prolonged or severe enough, myocardial cells die (i.e., myocardial infarction). For physicians, the basic principle in caring for patients with coronary disease who wish to dive is that each individual must be evaluated to ensure that myocardial ischemia is absent during diving. This principle can be applied to all patients with coronary disease.

Cardiac work is increased when either arterial pressure increases or blood flow (cardiac output) increases. Diving environments can produce both pressure and flow increases in the circulation. For example, isometric work associated with heavy lifting raises the arterial blood pressure and causes an increased pressure load on the heart, whereas the work associated with swimming increases flow demand on the heart and results in a volume load. A rise in blood pressure is generally more demanding in terms of myocardial oxygen consumption than an increase in blood flow.[21] A diver with hypertension experiences an excess load on the heart when exercising due to the combined workloads on the circulation. The heart

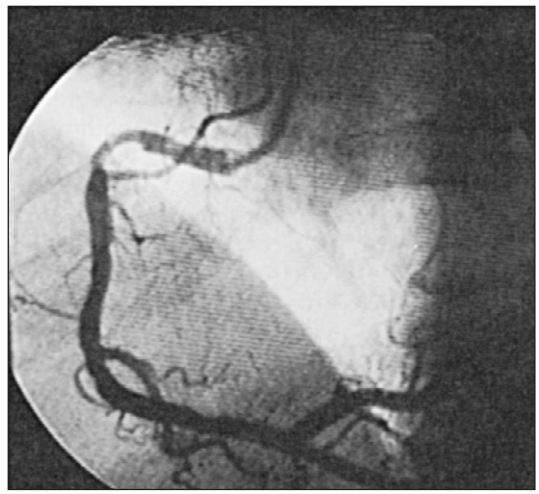

Figure 25–3. Coronary angiogram showing a narrowed segment of the right coronary artery.

does not greatly increase its extraction of oxygen as its work demands increase. Under increased work demands, increases in myocardial blood flow provide the increased oxygen demand.[22,23] Chronic hypertension, coronary artery disease, valvular heart disease, and congenital heart disease all may affect myocardial oxygen consumption, myocardial blood flow, and blood-flow distribution to the myocardium.[24,25] When flow restrictions are due to coronary artery narrowing, the myocardium cannot obtain adequate oxygen by increasing oxygen extraction and oxygen deficiency occurs during exercise.[24]

Coronary stenosis limits the blood flow that can pass through the narrowed segment of artery.[26] When a stenosis becomes significant (>50% reduction in cross-sectional area), resistance of the stenosis limits the ability of the peripheral coronary circulation to control flow.[27,28] A stenosis that causes 85% to 90% narrowing is the dominant factor controlling flow through the coronary artery.[27] When stenosis produces 90% narrowing, peripheral coronary regulation has little or no effect on blood flow under states of increased demand. In this situation, the maximal possible flow through the coronary artery may be adequate only to supply the myocardial demands at rest. Any amount of physical stress induces myocardial ischemia. The characteristics of stenosis versus flow in the coronary artery shown in Figure 25–4 show that stress testing is needed to demonstrate the imbalance between myocardial flow capability and myocardial oxygen

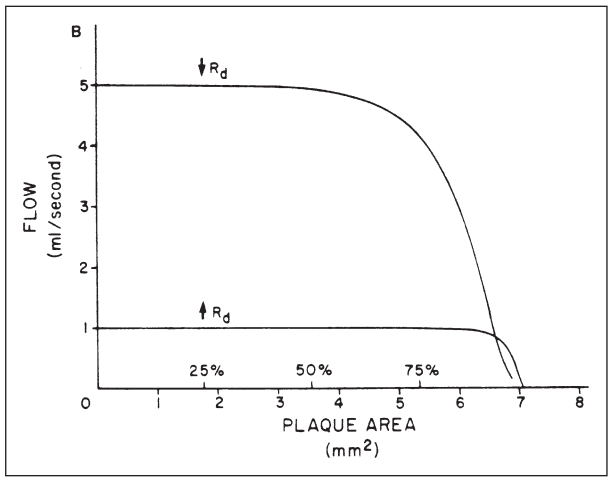

Figure 25–4. Plot of coronary flow reserve vs. percentage reduction in a cross-sectional area of a coronary artery. The reserve is not lost until the artery area is reduced by about 50%.

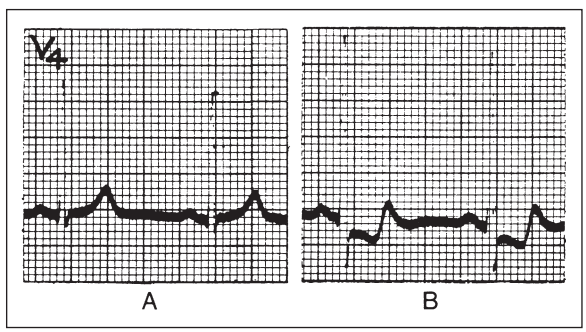

Figure 25–5. Typical electrocardiographic changes in exercise-induced ischemia. The normal ST segment *(A)* is depressed from the baselines with a down-sloping configuration *(B)*.

demand. Before approval for diving, the physician must be certain that diving-induced exercise stress will not produce ischemia (Fig. 25–5).

An imbalance between myocardial oxygen supply and demand during diving causes myocardial ischemia that may manifest as angina pectoris, serious ventricular arrhythmias, or left-ventricular dysfunction and heart failure. The signs and symptoms that accompany this latter response include onset of a third heart sound, development of marked dyspnea, development of basilar rales, a fall in blood pressure with exercise, and early fatigue. Coronary disease of such severity precludes diving.

An important variant of the usual pathophysiology of atherosclerotic coronary disease is coronary spasm that produces dynamic coronary narrowing under conditions that cause catecholamine release.[29] Coronary vasomotion of this type can produce signs and symptoms of unstable angina. Several studies have suggested that coronary vasomotion or coronary spasm may be induced by cold exposure or exercise.[30–32] Both stresses are common in diving, and patients demonstrating vasomotor-induced ischemia should not be approved for diving. Occasionally, electrocardiographic monitoring for 24 hours or longer, with an appropriate search for alterations in the S-T segment, may be needed to detect episodes of asymptomatic ischemia. Maseri and coworkers demonstrated that symptomless ischemia may develop in patients with coronary vasomotor instability (silent ischemia).[32,33] Some studies indicate that up to 80% of ischemic episodes may be asymptomatic.[32] Undetected ischemic episodes may coincide with exposures to physical stress or cold stress related to diving. Diving candidates with silent ischemia require careful evaluation of exercise capacity. Patients with asymptomatic ischemia may be at greater risk for sudden death because they experience no symptoms when myocardial ischemia occurs.

Evaluation of acute ischemia with serum enzymes can be confounded by similar enzyme changes caused by arterial air embolism without myocardial injury. Smith

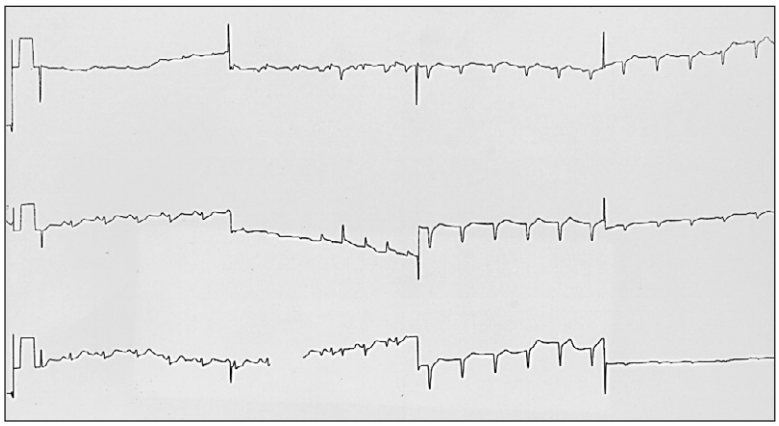

Figure 25–6. Electrocardiogram from a 26-year-old male with acute pulmonary barotrauma and arterial air embolism with neurologic impairment. A loss of R-wave amplitude occurs in all leads. This tracing can be interpreted as a massive myocardial infarction. The electrocardiogram reverted to normal, and the patient recovered with no residual symptoms after recompression treatment. In spite of markedly elevated serum creatine kinase associated with this electrocardiogram, this finding may not be indicative of a myocardial infarction. (Tracing courtesy T.S. Neuman, M.D.)

and Neuman reported elevated serum creatine kinase and electrocardiographic abnormalities in a series of patients with diving-related arterial gas embolism (see Chapter 9).[34] The most common rhythm in these patients was sinus tachycardia, likely related to the overall status. A few patients showed a striking loss of R-wave voltage in the precordial leads, a finding suggestive of acute myocardial infarction (Fig. 25–6). In the patients who demonstrated this change, however, post-treatment electrocardiograms (ECGs) appeared normal. The authors suggest that this change may be due to conduction abnormalities induced by air in the circulation and are not indicative of myocardial damage in spite of the elevated serum creatine kinase. The extremely high levels of this enzyme (10,000 to 20,000 units) suggest that the source of the enzyme is skeletal muscle, not myocardium.

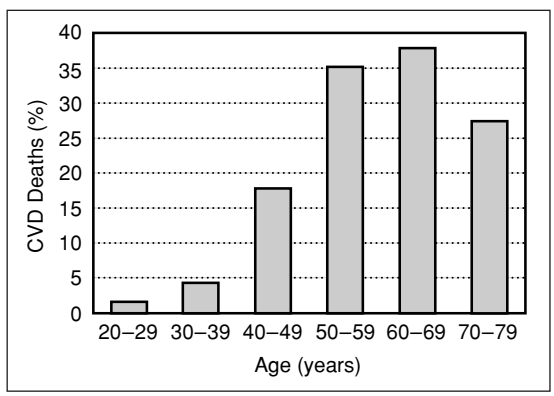

Figure 25–7. Incidence of cardiovascular deaths in sports diving related to age. The incidence follows the increasing incidence of coronary disease known to occur with age. (Data from Caruso JL, Bove AA, Uguccioni DM, et al: Recreational diving deaths associated with cardiovascular disease. Undersea Hyperbar Med 28[Suppl]:76, 2001.)

Assessing Coronary Risk in Divers

As the sport diving population ages, the incidence of coronary disease increases.[1] Unlike the commercial and military divers, sport divers are encouraged to continue diving into their seventies and even eighties if health and physical capacity allow. Because the number of divers older than 40 grows each year, there is also an increasing risk of coronary disease and acute coronary events in divers. Mebane and colleagues[35] reported the mortality experience from the Diving Alert Network. Of 33 cases of sudden death while diving, 31 were attributed to coronary disease, one was related to a cerebral vascular accident, and one was related to aortic stenosis. In a recent review of the Diving Alert Network registry, Caruso and associates[36] indicated that coronary events during sport diving increase with age and peak in the seventh decade of life (Fig. 25–7). Risk factors for coronary disease are well documented (Table 25–1) and can be used to assess risk of future coronary events.[37] Although risk factor analysis does not predict the incidence of acute events in divers, an assessment of risk factors can help to detect divers at risk and a plan for further risk assessment. If a diver or a diving candidate is positive for one or more of the factors in Table 25–1, further evaluation to determine the safety of exercise is warranted. This is best accomplished with a graded exercise stress test.

Table 25–1. Risk factors for coronary disease with range of high risk

Factor	High Risk
Age (male)	>40 years
Age (female)	>50 years
Family history of coronary artery disease	Siblings or parents
Smoking cigarettes	Any smoking
Hyperlipidemia	LDL > 100
Hypertension	BP > 140/90
Diabetes	HbA_{1C} > 7
Overweight	BMI >25

BMI, body mass index; BP, blood pressure; HbA_{1C}, hemoglobin A_{1C}; LDL, low-density lipoprotein.

CARDIOMYOPATHY AND CONGESTIVE HEART FAILURE

Patients with abnormal left-ventricular function due to cardiomyopathy often have clinical heart failure and are not eligible for diving. However, persons with some forms of cardiomyopathy are unaware of their condition and can experience manifestations of their illness while diving. Dilated cardiomyopathy causes a reduction in left-ventricular function that often manifests as a reduction in exercise tolerance. Patients with severe cardiomyopathy notice loss of exercise capacity, fatigue, and dyspnea. Left-ventricular ejection fraction below 35% usually causes physical disability and shortens life expectancy.[38] On the other hand, hypertrophic cardiomyopathy (HCM) may go undetected because left-ventricular function is usually preserved until late in the disease.

In both forms of cardiomyopathy, the more serious risk is sudden death due to lethal ventricular arrhythmias during exercise. Patients with these disorders are at risk during diving. HCM is the cause of 50% of sudden death events in high school and college athletes.[39] This disorder often causes enlargement of the ventricular septum and obstruction to outflow of the left ventricle. Mitral regurgitation is also present. A murmur of outflow obstruction intermingled with a mitral regurgitation murmur is characteristic of this disorder, which is often first suspected when a mixed murmur is detected on physical examination. Echocardiography is the best method for assessing ventricular function and hypertrophy, but screening echocardiography is impractically expensive. HCM and some forms of dilated cardiomyopathy are familial. Family history of sudden death should be investigated further to determine whether the events have been caused by cardiomyopathy. Persons with dilated cardiomyopathy and an ejection fraction of less than 50% should be advised against diving, although some sport divers with ejection fractions of 35% to 50% range and who are asymptomatic during moderate exercise appear to be diving safely. Candidates with HCM with an outflow gradient or with a history of arrhythmia or syncope should also be advised against diving. Less severe cases present a dilemma because the candidates usually have preserved physical capacity and an unknown risk for sudden death. Many patients with dilated cardiomyopathy and HCM undergo defibrillator implantation; they should be advised against diving.

Arrhythmogenic right-ventricular dysplasia is a rarer form of cardiomyopathy that involves the right ventricle and manifests as life-threatening ventricular arrhythmias.[40] This disorder is also familial, and patients with this disorder may have a history of unexplained sudden death in the family. Detection is less reliable because of the diffuse nature of the right-ventricular involvement and the nonspecific findings on echocardiogram.[40]

Decisions regarding diving are not difficult in patients with heart failure due to cardiomyopathy. Cardiomyopathies with adequate left-ventricular function for usual activity pose a greater threat from unpredictable sudden death. Every effort should be made to evaluate divers and diving candidates for the risk of sudden death. The costs of cardiovascular diagnostic procedures preclude testing all divers with echocardiography and electroencephalography; thus, it is essential to take a careful family history to learn of any unexplained syncope or sudden death.

Patients with reduced cardiac function may experience syncope while diving because of their inability to generate an appropriate cardiac output, vasodilation in skeletal muscle, and lowering of blood pressure. The response may be obscured by the absence of venous pooling during water immersion, and syncope occurs when exiting the water. A thorough analysis of cardiac performance is indicated for any diver candidate with suspected compromise of cardiac function. If the subject has reduced left-ventricular function (ejection fraction < 50%) or requires drug therapy for heart failure, the

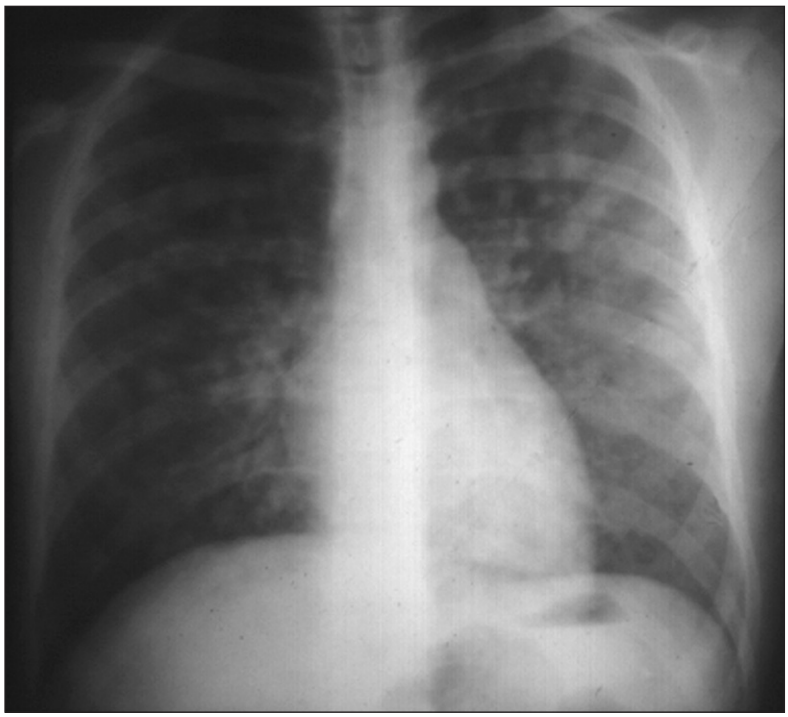

Figure 25–8. Chest radiograph of a 23-year-old healthy male who experienced dyspnea while diving at 60 ft. Diffuse infiltrates are typical of pulmonary edema.

loss of cardiac reserve is significant and the subject should not dive. A diver with undiagnosed heart failure may experience acute cardiac decompensation during a dive as an initial manifestation of disease.

IMMERSION PULMONARY EDEMA

Since the 1989 report of Wilmshurst and coworkers[41] describing a series of divers with acute pulmonary edema that occurred while diving, the entity of immersion pulmonary edema has become widely recognized.[42] Although rare in divers, acute respiratory distress with evidence of pulmonary congestion and arterial hypoxemia can cause serious consequences from panic ascent, with subsequent pulmonary barotrauma or drowning. The cases are typically described as a rapid onset of dyspnea while on the bottom. The diver ascends rapidly to find that the dyspnea does not resolve on the surface, and a cough with frothy sputum typical of pulmonary edema ensues. The dyspnea usually subsides over 1 to 2 hours. Patients who have been examined in emergency departments while still symptomatic show metabolic acidosis, arterial hypoxemia, and pulmonary congestion on chest radiograph (Fig. 25–8).

The cause of immersion pulmonary edema remains uncertain. Initial suggestions of cold-induced vasoconstriction causing left-ventricular overload are not in keeping with the patients described by Hampson and colleagues,[42] patients who were diving in warm water. Similar experience argues against the vasoconstriction being caused by chest restriction from a tight wet suit. Thorsen and associates[43] provided insight into one possible mechanism. In eight healthy male volunteers, the combination of immersion and an inspiratory resistive load caused a decrease in pulmonary diffusion capacity that was considered to be an indication of early pulmonary edema. Negative-pressure breathing causes acute pulmonary edema in 3 to 4 min when endotracheal extubation is complicated by laryngospasm.[44,45] Negative-pressure pulmonary hemorrhage can also occur.[46] Patients who experience negative-pressure pulmonary edema are usually in good health and exhibit rigorous chest activity against a closed glottis. Weiler-Ravell and colleagues[47] described pulmonary edema in Navy combat swimmers training in open-ocean swimming. All swimmers ingested about 5 L of water 2 hours before starting the swim. Eight swimmers experienced acute pulmonary edema, and all recovered within 24 hours, but two experienced a recurrence in a later swim.

Excess fluid loading contributed to the pulmonary edema in these healthy swimmers.

Divers who experience acute pulmonary edema while diving should be evaluated in a hospital to be certain that hypoxemia and pulmonary congestion resolve and to rule out other causes of pulmonary edema. Treatment with a rapid-acting diuretic (furosemide, 20 to 40 mg IV) is usually reverses symptoms and improves oxygenation, although some patients require intubation and positive-pressure ventilation for several hours. The lungs usually return to normal function within 24 hours. Hyperbaric oxygen therapy is not indicated because this syndrome is not caused by gas bubbles in blood or tissue. Cardiac evaluation should include ECG, cardiac enzymes to rule out ischemia, and echocardiogram to evaluate left-ventricular function, hypertrophy, and valvular integrity. In most cases, all cardiac study results are normal. Coronary angiography has been performed in isolated cases and yielded normal results (personal experience).

In otherwise healthy divers at low risk for coronary disease (see earlier), coronary angiography is not needed. Subjects should be advised to have their diving regulator checked for proper function, to avoid excessive fluid loading, and to avoid rapid deep breathing, which causes extremes of negative intrathoracic pressure while underwater. Most divers do not experience a recurrence, but the likelihood of recurrence is unpredictable.

CARDIAC TRANSPLANTATION

As the number of patients with solid-organ transplants increases, there will be persons who wish to dive after undergoing heart transplantation. The patient with a heart transplant has a number of problems that can interfere with diving. A few patients have successfully participated in limited sport diving after heart transplantation. Heart transplant recipients demonstrate lower maximum oxygen consumption than expected for age-matched unconditioned controls.[48,49] Average aerobic capacity is about 20 mL/kg/min, lower than the value needed to ensure safety in sport diving. This capacity level seems to persist for years after transplantation and may be related to abnormal skeletal muscle function acquired during the period of heart failure preceding transplantation.[49] The transplanted heart is denervated and lacks the usual exercise controls found in innervated hearts. Vagal tone is usually absent,[50] but sympathetic innervation may be partially restored. Cardiac performance can be correlated with the amount of sympathetic reinnervation that occurs after transplantation.[51]

Progressive atherosclerosis of the coronary arteries can occurs in the donor heart 2 to 3 years after transplantation.[52] Because the heart is denervated, most transplant recipients with ischemia from coronary disease do not experience angina, and their first symptom can be ventricular tachycardia or sudden death. When coronary disease is present, exercise tolerance may be reduced.[53] Evaluation of heart transplant recipients for ischemia involves annual exercise perfusion imaging and coronary angiography. Absence of ischemia is essential for any form of exercise in these patients.

Heart transplant recipients are treated with multiple medications. Besides immunosuppression medication, most patients are taking one or more antihypertensive medications, a lipid-lowering medication, and prophylactic antifungal medications. The immunosuppression necessary to avoid rejection of the transplanted heart also renders the patient prone to infections. Most commonly, the cellular immune suppression facilitates viral and fungal infections. This risk of infection is increased by exposure to contaminated water.

Sport diving should not be recommended for heart transplant recipients, but a few such patients who are already trained divers have returned to limited diving. The dependence of these patients on a heart transplant center, the frequency of minor and major medical events, the high incidence of infections, and the remoteness of many diving locations all weigh against diving for such patients. As noted earlier, most of these impediments can be managed in special cases that would allow limited diving for an occasional heart transplant recipient.

VALVULAR AND CONGENITAL HEART DISEASE

Valvular or congenital heart disease is not necessarily a contraindication to diving. Functional capacity of the diver and the nature of the lesion should dictate whether a

candidate can dive safely. In the case of atrial septal defects, significant stenotic valvular or vascular lesions, or cyanotic congenital heart disease, diving is contraindicated.

Pathophysiologic Principles

Overload lesions of the heart can be classed as either pressure or volume types.[54] *Pressure overload* lesions include the concentric left-ventricular hypertrophy that results from aortic stenosis, chronic hypertension, or aortic coarctation.[55] *Volume overload* of the left ventricle can occur from aortic or mitral regurgitation or in the right ventricle from an atrial septal defect.[56,57] The response of the myocardium to these overload states depends on the type of overload. The myocardium appears to adapt specifically to the type of load imposed.

In either type of hypertrophy the increased muscle mass that occurs in response to chronic overload demands an increased myocardial blood flow.[58] Thus, the blood flow to either the pressure or volume overloaded hypertrophied heart is increased above normal resting levels. Experimental evidence suggests that perfusion to the endocardium is inadequate in the hypertrophied heart under high load states.[59] The subendocardial ischemia found in aortic stenosis and in chronic hypertension with hypertrophy in the absence of coronary atherosclerotic narrowing is one example of abnormal flow distribution in hypertrophied myocardium. Such a response should also be expected under the stress of diving; demonstration of such a response by stress testing is a contraindication to diving. Some evidence suggests that regions of the volume-overloaded heart may also be underperfused when flow demand is high.

Early hypertrophy, which may be undetected by ECG, can be associated with evidence of subendocardial ischemia detected by exercise stress testing.[60] Echocardiography is a more sensitive method of detecting cardiac hypertrophy, but echocardiographic measurement of left-ventricular wall thickness does not provide evidence for or against exercise-induced ischemia. The changes induced in the endocardium by maldistribution of blood flow during exercise are best detected by the exercise stress test, which can be used to evaluate the presence or absence of ischemia in patients with volume or pressure overload. Although there are specific contraindications, it is possible to allow selected patients with congenital or valvular heart disease to dive (Table 25–2). The basic principle of simulating the diving exposure in the controlled environment of the exercise stress test with electrocardiographic and blood pressure monitoring should be followed. This information is then used to determine individual exercise capacity. Such an approach to the patient with valvular or congenital heart disease allows one to clear some candidates to dive if the lesion is small and there are no right-to-left shunts at the atrial level. The candidate should be able to reach 13 METS (oxygen consumption of about 40 mL/min/kg) at maximum capacity to dive safely.

Table 25–2. Congenital and valvular heart disease and diving

Condition	Diving Problem
Aortic stenosis	Exercise syncope, sudden death
Aortic insufficiency	None (heart failure if severe)
Mitral stenosis	Exercise induced pulmonary edema
Mitral insufficiency	None (heart failure if severe)
Pulmonic stenosis	None (reduced exercise tolerance if severe)
Pulmonic insufficiency	None
Tricuspid stenosis	None (reduced exercise tolerance if severe)
Tricuspid insufficiency	None (heart failure if severe)
Atrial septal defect	Paradoxical arterial gas embolism
Ventricular septal defect	None if small
Patent ductus arteriosus	None (heart failure if severe)
Idiopathic hypertrophic subaortic stenosis	Exercise syncope, sudden death
Mitral valve prolapse	None (arrhythmias may accompany)

Circulatory Considerations in Valvular and Congenital Disease

Certain abnormalities in valvular and congenital heart disease need special consideration in the evaluation of diving candidates. Exercise is limited in patients with circulatory obstruction (e.g., with aortic stenosis, mitral stenosis, aortic coarctation, or pulmonic stenosis) because of the narrowed segment of the circulation. When peripheral circulatory demand and cardiac output are out of balance, blood pressure falls and the patient experiences syncope. Indeed, this mechanism may be one of the causes of sudden death in patients with aortic stenosis. Patients with these abnormalities should not be approved for diving. This approach to the diving candidate is similar to that taken for competitive sports.[61] A useful guideline is to prohibit diving if aortic or mitral valve area is less than 2.5 sq. cm.

Patients with regurgitant or shunt lesions are generally less likely to experience syncope or hypotension while diving but are more likely to experience pulmonary congestion and show evidence of severe dyspnea from combined exercise and water immersion.

CONGENITAL HEART DISEASE

Ventricular septal defects are most common in the upper septum in its membranous portion.[62] Small defects do not cause significant right-to-left shunting and therefore are not likely to increase the risk of arterial emboli during diving. However, persons with ventricular septal defects should be counseled on the need for antibiotics during dental procedures due to the increased risk of endocarditis. A small membranous ventricular septal defect should not be a contraindication to diving.

If pressures in the central circulation are normal in patients with minimal or no symptoms and atrial or ventricular septal defects, the shunt is directed from left to right and no arterial desaturation occurs. Exercise capacity is usually severely limited in patients with a right-to-left shunt and arterial hypoxemia,[63] and they should not dive.

Diving candidates with atrial septal defects risk paradoxical embolism of gas bubbles that occur in the venous circulation during decompression.[64] Since intra-atrial shunt flow can change direction in different phases of the cardiac cycle[65] or with the Valsalva maneuver, an atrial septal defect is a contraindication to diving.

Mitral valve prolapse, found in 4% to 7% of the normal population,[66] is not a contraindication to diving. Valvular redundancy causes the mitral valve to prolapse into the left atrium during systole and produce a characteristic early systolic click. This phenomenon has no hemodynamic consequences and causes no dysfunction. Mitral regurgitation may occur in mid-systole if the leaflet edges separate. This finding also has no particular consequences on exercise capacity or diving unless the regurgitation becomes severe and compromises cardiac performance. In some patients, mitral prolapse is accompanied by arrhythmia. Whether the arrhythmia precludes diving depends on the nature of the arrhythmia and the response to treatment (see later). Mitral valve prolapse is not a contraindication to diving, and associated abnormalities should be considered independently.

Patent Foramen Ovale

Moon and colleagues[67] described 30 patients with a history of decompression sickness (DCS) who were studied with bubble contrast echocardiography for identification of a patent foramen ovale (PFO). Shunting was present in 61% of l8 patients with serious DCS, whereas the prevalence was 5% in normal volunteers. The authors conclude that in DCS, which is unpredictably severe, there seems to be an increased incidence of PFO. However, the results also indicate the inverse, that is, the risk of DCS in the presence of PFO could not be deduced from the data. Indeed, the number of patients was too small to allow these data to be used for risk prediction. Hagen and coworkers[68] studied 935 patients at autopsy and found a PFO in 263 (27%). The prevalence of PFO was 34% up to age 30 and declined to 25% in the fourth through eighth decades of life. In studies of normal volunteers, the incidence of Valsalva-induced right-to-left shunting across a PFO, demonstrated by echocardiography in nondiving normal subjects,[69] was 18.5%. A PFO was found in 40% of patients with a history of stroke.[70]

In seven divers with neurologic DCS, Walsh and associates[71] used transcatheter closure for a PFO in six and an atrial septal defect in one. All divers returned to diving.

Although this series is small and no conclusion can be drawn regarding long-term success, the early reduction in DCS in these divers suggests that closure of a PFO or atrial septal defect can reduce the incidence of DCS in persons prone to DCS who have a PFO. In a study of patients with stroke and PFO, Windecker and colleagues[72] found a reduced but continuing rate of neurologic events after catheter closure of the PFO. Their data indicate that PFO closure does not guarantee freedom from neurologic events in patients with strokes. These data suggest caution when recommending PFO closure for divers who have experienced neurologic DCS.

Moon and associates[73] evaluated 90 divers with previous DCS using bubble contrast echocardiography and color flow Doppler imaging to detect right-to-left shunting through a PFO. Fifty-nine of 90 had experienced serious decompression symptoms; 31 had experienced pain only or mild symptoms. An asymptomatic control group of divers was also studied. Forty-nine percent of the subjects who had experienced serious DCS had evidence of a right-to-left shunt during a Valsalva maneuver or at rest compared with 19.8% of controls. The odds ratio of 3.9 was significant at $P < .0002$. Interestingly, the Valsalva maneuver produced no significant difference between controls and patients who had suffered serious DCS whereas the resting echocardiogram showed significant differences ($P < .0002$; odds ratio, 4.9) compared with controls. With nonserious DCS, there was a trend toward increased incidence of PFO but no significant differences between the diving subjects and controls. Bubble contrast echocardiography appeared to be the most sensitive method for detecting a shunt, whereas color flow Doppler imaging appeared to be a poor means of detecting the shunt in a transthoracic echocardiogram. Wilmshurst and coworkers[74] compared 61 divers who had DCS (divided into four groups based on severity) with divers who had no DCS. There were no significant differences in terms of the incidence of PFO between the 15 of 63 controls who had right-to left shunting and the 24 divers who had experienced the onset of neurologic symptoms more than 30 min after surfacing or with joint pain only. In divers who had experienced neurologic symptoms *within* 30 min of surfacing, the prevalence of shunt was significantly higher than in controls.

Development of a skin rash soon after surfacing was also related to a higher incidence of shunting. Group 1A (29 divers) experienced neurologic symptoms in the first 20 min after diving; group 1B (24 divers) experienced neurologic symptoms more than 30 min after diving. The mean time to onset was 8.1 hours for group 1B; delays ranged from 0.5 to 48 hours. Six divers had joint pain only that developed 2 to 8 hours after surfacing. Two divers who had cutaneous DCS were also included in the study. One diver had PFO as detected by transthoracic contrast echocardiography. Studies were performed at rest and during repeated Valsalva maneuvers. In 25 of 61 persons, DCS followed what was considered to be safe decompression profiles. The prevalence of interatrial shunt was 65% in this group, significantly higher than with those who performed dives associated with risk factors for DCS. It should be noted that five divers experienced DCS without any provocative risk factors, including absence of a PFO.

The authors also note that interatrial shunts may be related to transient neurologic symptoms for which divers do not seek treatment. A number of unreported episodes may account for the higher-than-expected incidence of DCS. The authors point out that cranial dysfunction is usually thought to be due to cerebral gas embolism. However, in the presence of a PFO, cerebral gas embolism may occur and blur the distinction between spinal-cord DCS and air embolism. In the same study, Wilmshurst and coworkers noted that the severity of shunt varied with different tests and times.[74] They suggest that this variability may occur during diving, which would explain why divers with PFO may dive for many years without symptoms. Many symptomless divers also have shunts through a PFO. Thus, the presence of a PFO is only a contributing factor to the development of DCS. No recommendations can be made regarding screening for PFO; however, when DCS occurs following an apparently safe dive schedule, echocardiographic studies with bubble contrast and a Valsalva maneuver may be useful (Fig. 25–9).

Meta-Analysis

Figure 25–10 is derived from a logistic regression analysis of data from several papers that reported on PFO in divers.[75] The analysis includes a study by Cross and

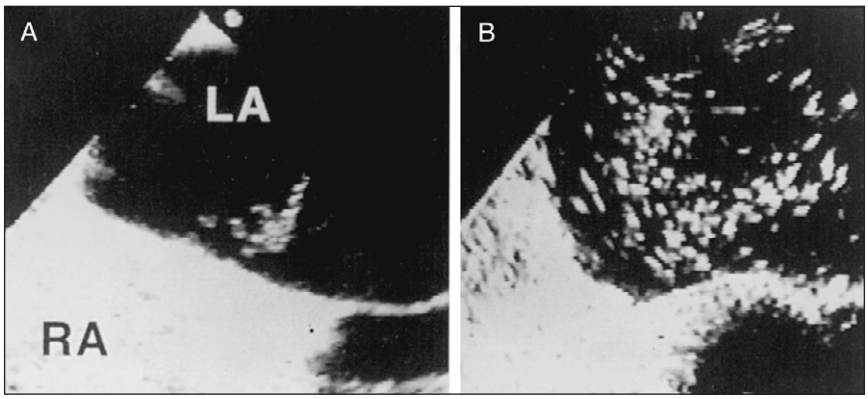

Figure 25–9. Echocardiogram showing mild shunt of a patent foramen ovale *(A)* and severe shunt *(B).* Bubble echoes are apparent in the left atrium in both cases, but the quantity is greater in the right panel. (From Housemann D, Mugge A, Daniel WG: Identification of patent foramen ovale permitting paradoxic embolism. J Am Coll Cardiol 26:1034–1038, 1995.)

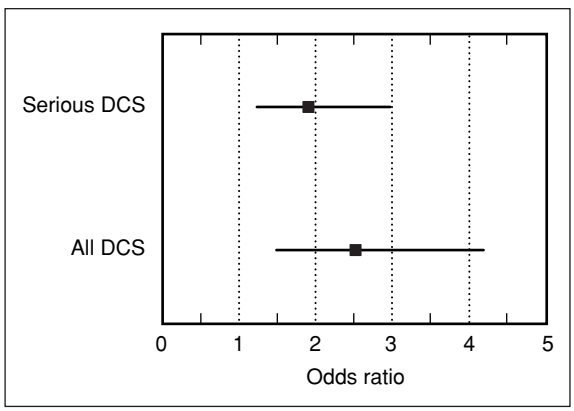

Figure 25–10. Odds ratio for developing any form of decompression sickness *(bottom line)* or serious (type II) decompression sickness *(top line)* based on a meta-analysis.[75] Odds ratio is shown with 95% confidence intervals. Data were analyzed using logistic regression. The odds ratios are significantly different from zero, suggesting that a patent foramen ovale increases the risk of decompression sickness.

colleagues,[65] who reported no DCS in a group of divers with PFO. The incidence of DCS in sport divers is used as a base for general incidence in the diving population. Data from the commercial diving population would presumably be similar to these results. The analysis indicates that the overall incidence of DCS should be about 0.05% in a diving population. The risk ratio for DCS is increased by a factor of about 2.5 for persons with a PFO and is reduced by a factor of about 2 in persons who do not have a PFO. In either case, the overall risk remains low.

Germonpré and associates[76] examined 37 divers with DCS and 36 controls using contrast transesophageal echocardiography. Their data (Table 25–3) show that divers with cerebral DCS have a high incidence of large PFO compared with control subjects and divers with spinal-cord DCS. These findings are similar to findings in patients with unexplained (cryptogenic) strokes and support the hypothesis that a large PFO increases the risk of cerebral gas or thrombotic emboli resulting from paradoxical embolization across the atrial septum.[77] A PFO can be evaluated with transthoracic or transesophageal echocardiography, although transthoracic echocardiography has been found to be less sensitive than transeso-

Table 25–3. Prevalence of shunting through a PFO in divers with cerebral and spinal decompression sickness

DCS Type	Mild Shunt	Severe Shunt
Cerebral n = 20	2 (10)	14 (70)
Control n = 20	2 (10)	3 (15)
Spinal n = 17	1 (6)	5 (29)
Control n = 16	2 (13)	6 (37)

Values are number of divers with a patent foramen ovale; numbers in parentheses are percentages.
Data from Germonpré P, Dendale P, Unger P, et al: Patent foramen ovale and decompression sickness in sports divers. J Appl Physiol 84:1622–1626, 1998.

phageal echocardiography.[78] Some investigators think that transcranial Doppler ultrasonography is also valuable for detecting bubbles entering the arterial circulation from the right heart,[79] but this method does not identify the location of the shunt.

CARDIAC ARRHYTHMIAS

During a dive, a variety of arrhythmias may develop in patients with or without heart disease. The importance of the arrhythmia varies with the type and the patient's history. Most arrhythmias are benign and cause no effects on the diver. Serious arrhythmias are a contraindication to diving.

Supraventricular Arrhythmias

Premature atrial beats, supraventricular tachycardia, and atrial fibrillation may be associated with diving. Episodic supraventricular tachycardia and atrial fibrillation in the young adult population is usually associated with a normal heart.[80] However, such arrhythmias are an indication for careful evaluation to rule out mitral stenosis, hyperthyroidism, and hypertension. Rarely, pulmonary emboli may produce atrial arrhythmias in this asymptomatic population, and this diagnosis should also be considered. Dietary supplements used for enhancing sports performance also contain stimulants that can induce cardiac arrhythmias.[81] Evaluation of all divers should include queries regarding use of dietary supplements. Generally, premature atrial contractions are of no consequence and are common in normal persons. Stress, alcohol, and caffeine—alone or in combination—are the usual cause of supraventricular arrhythmias. Catecholamine-based decongestants can also induce these arrhythmias. In the absence of organic heart disease, and when removal of these stimuli abolishes the arrhythmia, diving can be permitted.

In normal persons, therapy for the arrhythmia may produce more troublesome symptoms than the arrhythmia itself. Thus, one must be careful in selecting both therapy and the patient who requires it. After ruling out significant cardiac disease or systemic illness, such as hyperthyroidism or hypertension, and after a search for ingestion of cardiac excitatory agents, such as caffeine (coffee, cola drinks, and various over-the-counter analgesics), catecholamine-like drugs (such as those found in anti-allergy medications), alcohol, and nicotine, episodic tachycardia can be prevented with beta blockers, calcium channel blockers, or other specific antiarrhythmic medications. If the condition is adequately controlled, sport diving may be considered.

Ventricular Arrhythmias

Ventricular arrhythmias, which manifest as isolated premature ventricular contractions, are found in normal persons without heart disease. With diving candidates, such arrhythmias should be assessed for their behavior during exercise. Premature contractions that demonstrate a multifocal pattern, R on T phenomenon, or frequent coupling of sequential premature beats (nonsustained ventricular tachycardia) should be considered as serious and should disqualify the diving candidate. Patients with compromised left-ventricular function from ischemic or idiopathic cardiomyopathy are especially prone to sudden death from ventricular arrhythmias. The risk of sudden death is related to the presence and severity of cardiac dysfunction as well as the finding of nonsustained ventricular tachycardia.[82] Persons with compromised left-ventricular function, with or without ventricular arrhythmia, are at risk for sudden death with exercise and should be prohibited from diving. Patients with implanted cardioverter-defibrillators should be advised against diving.

Long QT Syndrome

This syndrome has been associated with in-water sudden death. In persons with cardiac electrical depolarization abnormalities known as the *long QT syndrome,* the risk of ventricular fibrillation and sudden death is high.[83] The torsades de pointes pattern of polymorphic ventricular tachycardia[84] is characteristic (Fig. 25–11). The first manifestation of the long QT syndrome is often syncope or cardiac arrest precipitated by emotional or physical stress. Patients are usually young and are found to have a

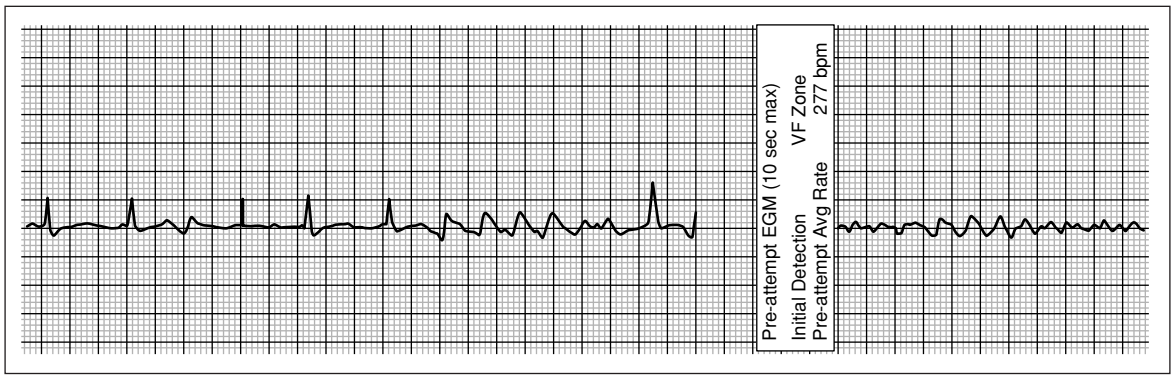

Figure 25–11. Ventricular electrogram recorded from an implanted cardioverter-defibrillator in a woman with a long QT syndrome who experienced cardiac arrest while swimming. The electrogram shows a sinus rhythm that breaks into ventricular tachycardia following a premature ventricular contraction, then deteriorates to ventricular fibrillation (VF) shown on the right. The panel preceding the VF is printed by the ICD and indicates the detection of the VF. The device fired and converted the VF to sinus rhythm (not shown). (From Ott P, Marcus FI, Moss AI: Ventricular fibrillation during swimming in a patient with long-QT syndrome. Circulation 106:521–522, 2002.)

Table 25–4. Causes of acquired long QT interval

Drug	Use
Sotalol	Antiarrhythmic
Diisopyramide	Antiarrhythmic
Procainamide	Antiarrhythmic
Flecainide	Antiarrhythmic
Dofetilide	Antiarrhythmic
Desipramine	Antidepresant
Erythromycin	Antibiotic
Halofantrine	Anti-malarial
Ibutilide	Antiarrhythmic
Imipramine	Antidepressant
Probucol	Lipid lowering
Sympathomimetics	Decongestants
Other causes	
Hypokalemia	
Hypomagnesemia	

corrected QT interval in excess of 440 msec on the ECG. Events that trigger the arrhythmias include exercise, loud sounds, electrolyte imbalance, and certain drugs (Table 25–4). Ackerman and coworkers[85] described a healthy woman who died suddenly while swimming in a pool and was found to have a genetic abnormality that causes the long QT syndrome. Her genetic defect was of the type that would make her prone to ventricular fibrillation during exercise. These authors published a second paper describing a series of patients with a similar history of swimming-induced sudden death.[86] These cases were first thought to be caused by drowning, but their identity as long QT syndrome indicated that the primary event was ventricular fibrillation.

Bradley and colleagues[87] reported a similar experience while studying the family members of several subjects who died suddenly while swimming. They found 35 of 78 family members who had unexplained syncope or near-drowning events. Sudden death with exercise is a known consequence of the long QT syndrome. Swimming and water immersion may add another stimulus. Yoshinaga and associates[88] found QT prolongation during face immersion in children with documented long QT syndrome. These authors suggested that immersion is an independent factor in causing ventricular arrhythmias in these subjects. Although many people with the long QT syndrome are aware of this problem from childhood,[89,90] the changes manifest only when other factors are present in other patients. Thus, exercise, water immersion, electrolyte imbalance, and medications may combine to cause sudden death in these patients.

The data from these clinical studies did not include divers; however, the circumstances of exposure that include immersion and exercise are typical of sport diving and would stimulate ventricular fibrillation in divers as well as swimmers. Beta-blockers are effective in preventing syncope in patients with long QT syndrome. However, 25% to 35% of patients with symptomatic long QT syndrome are likely to have another event within 5 years while on therapy.[91] Persons with documented, symptomatic long QT syndrome—particularly those who have experienced a sudden death

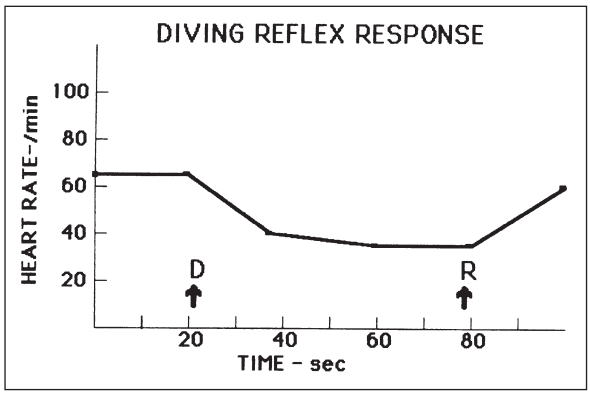

Figure 25–12. Heart-rate response to breath-held facial immersion in a diver. Note the rapid fall in rate immediately following facial immersion (D). Heart rate remains reduced until removing the face from water (R).

event—should not dive. Many patients have an implanted defibrillator, and such persons should not dive.

Vagotonic Arrhythmias

Well-conditioned candidates may have augmented vagal tone and resting bradycardia.[92] Vagal tone often is so high that resting heart rates range from 30 to 40 beats per minute. These rates are normally well tolerated because of the appropriately increased stroke volume, and athletes do not usually experience significant symptoms of bradycardia. Variants of vagotonic rhythms include first-degree heart block and Wenckebach-type second-degree heart block. Although these rhythms are often benign in a well-trained candidate, approval for diving should involve a test with exercise to show reversibility when vagal tone is released. Failure of these changes to reverse with exercise should raise suspicion of organic heart disease, and diving should not be approved. Diving bradycardia (Fig. 25–12) is a unique vagotonic response to water immersion that can result in heart rates of 40 to 50 beats per minute in some divers (see Chapter 5). Rarely, profound bradycardia and syncope occurs with every exposure to water immersion. These appear to be hypervagotonic syndromes and can be treated with anticholinergic medication. This rare but profound response to water immersion should be considered a contraindication to diving. Patients with this syndrome often avoid water immersion because of the severe reaction that follows and rarely show an interest in diving. High vagal tone also provides a substrate for atrial fibrillation.[92] In well-trained divers with high vagal tone who complain of palpitations while diving, paroxysmal atrial fibrillation should be considered in the differential diagnosis.

CONDUCTION ABNORMALITIES AND PACEMAKERS

Cardiac disease is usually the cause of abnormalities of the conduction system. Congenital heart disease, certain valvular heart diseases (aortic stenosis with valvular and A-V ring calcification), cardiomyopathy, and coronary heart disease all may be associated with chronic conduction system abnormalities. First-degree atrioventricular (AV) block is often induced by excess vagal tone (see earlier) but is sometimes due to cardiomyopathy or primary conduction system disease. One treatable cause is first-degree block due to Lyme disease. Variable second-degree AV block is caused by excess vagal tone and is discussed earlier. Second-degree fixed AV block is a pathologic delay and often leads to complete heart block. Fixed second-degree AV block is usually related to other cardiac disease (i.e., ischemic heart disease) that requires evaluation and should preclude diving. Complete right bundle branch block (RBBB) may be a normal variant and is usually benign. However, RBBB can be associated with certain forms of congenital heart disease. The history and physical examination of the heart can provide some assurance that the RBBB is benign. When in doubt, an echocardiogram can rule out anatomic cardiac abnormality as a cause of the RBBB.

Left bundle branch block (LBBB) is often associated with coronary heart disease or cardiomyopathy. In most cases, the primary cardiac problem is known and dictates

whether the candidate is capable of safe diving. In the absence of organic heart disease, LBBB is not a contraindication to diving. In the presence of LBBB, coronary disease and cardiomyopathy must be ruled out before a person can be cleared for diving. Most patients with acquired complete heart block are limited in their capacity to exercise because of inability to increase cardiac output. Patients with acquired complete heart block should be treated along standard clinical lines; usually, a permanent pacemaker is implanted to provide adequate cardiac output and heart rate. Diving candidates with pacemakers should not be permitted into commercial, military, and scientific diving. Sport diving must be individualized. If no other heart disease is present, if the pacemaker is tested against pressure up to 130 fsw, and if exercise tolerance is good, the candidate might be allowed to dive. The use of implantable defibrillators has become an established treatment for sudden death syndrome.[93] Patients with implantable defibrillators usually are at risk for ventricular fibrillation and should not dive.

PRE-EXCITATION SYNDROMES

Patients with short P-R intervals (Fig. 25–13), with and without QRS abnormalities, may experience rapid tachycardia at rest or during exercise.[94,95] However, many patients with short P-R intervals are asymptomatic. Sorbo and colleagues[96] surveyed a large population of high school students and found the prevalence of electrocardiographic findings indicating Wolfe-Parkinson-White syndrome to be 1.48 per 1000. Symptoms occurred in 34% of such subjects, for a prevalence of 0.51 per 1000. An interesting characteristic of these patients was the intermittent appearance of pre-excitation on the ECG. The finding of a short P-R interval on the ECG is not in itself a contraindication to diving. Patients with a history of paroxysmal tachycardia should be evaluated for the presence of the pre-excitation syndrome; if recurrent paroxysmal or exercise-induced tachycardia is a significant symptom, then appropriate diagnostic and therapeutic procedures should be followed. In some cases, a typical pattern of Wolfe-Parkinson-White syndrome emerges during exercise testing or during ambulatory monitoring. An isolated ECG in a diver with paroxysmal tachycardia therefore may not be adequate to rule out pre-excitation syndrome.[96] Sometimes, when the supraventricular tachycardia stimulates the ventricles to contract at rates exceeding 240 beats per minute, the subject is at risk for ventricular fibrillation and sudden death.[83]

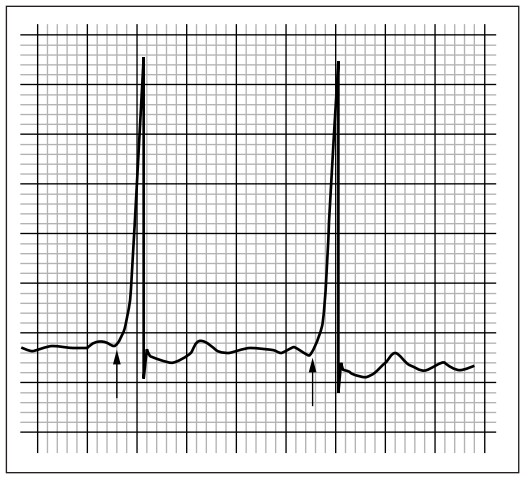

Figure 25–13. Electrocardiographic pattern of the Wolf-Parkinson-White type. The beginning of the QRS wave *(arrow)* is widened because of conduction over an accessory A-V pathway.

Symptomatic Wolfe-Parkinson-White syndrome is a contraindication to diving, but asymptomatic persons with evidence of pre-excitation on ECG are at low risk for arrhythmias[7] and can be approved for diving. If there is doubt about possible paroxysmal tachycardia, exercise testing or ambulatory monitoring can provide further insight.[83] Therapy of this disorder is now curative when a catheter ablation is performed. This procedure destroys the accessory pathway using local heat delivered through a catheter.[97,98] The cure rate with this procedure approaches 98%.[99] Because of the low complication rate and the high success rate of catheter ablation, divers with tachyarrhythmias caused by pre-excitation should undergo ablation to eliminate the arrhythmia.[97] When the procedure is successful, candidates have no contraindication to diving from this disorder.

CORONARY BYPASS SURGERY AND ANGIOPLASTY

Patients who have undergone successful coronary bypass surgery or angioplasty who wish to undertake sport diving need not be denied this activity, although commercial or

military diving should not be approved. Physicians should review coronary anatomy, degree of vascularization, and exercise capacity. A reasonable approach to the patient who has undergone bypass surgery is cardiac rehabilitation for 3 to 4 months after surgery, then a return to swimming and other conditioning activity, followed by a stress test at 6 months to rule out ischemia during exercise. If the diver can exercise to 13 METS with no ischemia or angina, with normal blood pressure response and with no serious arrhythmia, limited sport diving may be considered. The rate of restenosis within 6 months of the procedure is significant in patients undergoing percutaneous interventions for coronary disease (angioplasty, atherectomy, stent implantation). The restenosis rate approaches 50% in some procedures.[100] Patients who have undergone percutaneous revascularization should be advised to avoid diving for 6 months, regain physical conditioning through an exercise program, then undergo stress testing before returning to diving. The absence of ischemia during diving is an absolute requirement for divers who have coronary disease and have undergone revascularization. Most diving activity in warm water require activity at the level of 3 to 5 METS, with occasional excursions to 7 or 8 METS and a reserve capacity up to 13 METS. Divers who demonstrate ischemia, even when asymptomatic, on exercise testing at levels below 12 to 13 METS risk becoming ischemic when stressed in the water. Ischemia in the setting of exercise and immersion can lead to arrhythmias, myocardial infarction, or sudden death.

VALVULAR SURGERY

Prosthetic cardiac valves create two important problems in divers and potential divers. In high-output states related to exercise, the gradient across the valve may be significant; with high heart rates, the valve poppet may not open and close completely, thus aggravating the gradient or producing significant valve regurgitation. The use of center-opening valves, including valves with tissue leaflets that open with larger orifices and have less mass, provides the possibility for greater cardiac output and, therefore, greater exercise capacity.

The second consideration in divers with prosthetic cardiac valves is anticoagulation. Because diving often results in minor trauma, anticoagulation is likely to produce excess bleeding in situations that produce blunt trauma, injury, or bruising. Concern has been expressed for middle-ear barotrauma that might progress to severe hemorrhagic otitis in the presence of anticoagulation. Because of these limitations, patients who have prosthetic cardiac valves should not be routinely cleared for diving. However, individual exceptions for carefully constrained sport diving can be made. Heterograft and homograft valves provide a significant advantage in this regard because patients with heterograft and homograft aortic valves can be treated without anticoagulation after an initial period for recovery from surgery.[101] Patients with heterograft mitral valves often are maintained on anticoagulation. Human homograft valves in the aortic or pulmonary position leave the patient with normal valvular function and excellent exercise tolerance for diving. Divers and diving candidates with prosthetic heart valves must also be evaluated for cardiac function. Chronic valvular disease often results in decreased ventricular function and a state of chronic heart failure, which compromises exercise performance when diving. Exercise testing to 13 METS is a useful screening tool for resolving questions about exercise capacity in patients with prosthetic valves.

CARDIOVASCULAR DRUGS AND DIVING

Diving candidates may be taking a variety of prescription and nonprescription drugs. Although most drugs are not affected by the pressure, inert gas, or increased oxygen partial pressure of the diving environment, many cardiovascular drugs can alter exercise tolerance.

In patients on large doses of antihypertensive medication, significant inhibition of normal cardiovascular control mechanisms and poor exercise tolerance are possible. Medications that inhibit rises in blood pressure during exercise may result in exertional syncope or orthostatic hypotension. Diving candidates using antihypertensive drugs require careful assessment for evidence of an inadequate blood-pressure response during exercise. Limitations to moderate exercise due to dyspnea, weakness, dizziness, or palpitations should indicate exercise screening before diving can be approved.

Table 25–5. Perceived exertion scale used for subjective analysis of exercise capacity

6	
7	Very, very light
8	
9	Very light
10	
11	Farily light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very hard
18	
19	Very, very hard
20	

From Borg GAV: Perceived exertion: A note on history and methods. Med Sci Sports Exer 5:90–93, 1973.

The heart-rate response to exercise is somewhat limited in patients using beta-adrenergic blocking medication; therefore, the heart-rate response to exercise does not provide the same measure of exercise workload in patients with normal autonomic responsiveness.[102,103] Because of this difference, patients using beta-blocking drugs must be observed for subjective responses to exercise via perceived exercise scores (Table 25–5)[104] or direct measurement of maximum oxygen consumption. In stress-testing a patient who uses beta-blocking drugs, one can determine a relative maximum heart rate by comparing the perceived exercise score with that measured when the patient has significant fatigue. If the subject can achieve 13 METS of exercise under beta-blockade without severe fatigue, then diving can be approved. The blunted heart rate response to exercise does not indicate a poor physiologic response in patients on beta-blocking drugs.

Although beta-adrenergic blockade inhibits maximum exercise performance, this rarely interferes with diving because diving should not provoke maximum work demand. However, divers taking beta-blockers should be advised to avoid extreme exercise because their maximum capacity may be inhibited.

Stress testing should be done in the presence of beta-blockade when determining exercise capacity for a diver taking this medication. It is important to test exercise capacity in the presence of the usual therapeutic regimen. If the diver taking beta-blockers can sustain exercise to 13 METS, then diving should be safe.

Calcium channel blockers lower blood pressure by relaxing vascular smooth muscle. The inhibition of calcium flux into vascular smooth muscle cells allows these cells to relax, and blood pressure is reduced. These agents do not produce specific interactions with the diving environment, although they sometimes cause orthostasis after exercise when used in high doses and are known to cause peripheral edema not related to the heart.[105] Edema in a patient taking calcium channel blockers should not be interpreted as an indication of heart failure. Most patients who experience this side effect are taken off of the drugs, but sometimes the edema is intermittent and tolerated well. These drugs also relax the gastroesophageal sphincter and cause reflux of gastric contents. When immersed, divers lose the gravitational effects on gastric contents and may experience reflux with associated heartburn, laryngeal irritation, or cough. Moderate doses of these drugs used for control of blood pressure do not interfere with diving performance or safety.

Angiotensin-converting enzyme inhibitors and angiotensin-receptor blockers are commonly used for treatment of blood pressure in otherwise normal persons, physicians are likely to encounter diving candidates using these medications. Angiotensin-converting enzyme inhibitors are useful drugs in treatment of hypertension in younger persons because they interfere minimally with exercise ability. Like other antihypertensive drugs, these agents can cause orthostatic hypotension when used in large doses. When properly administered, they should not interfere with diving. An interesting side effect of these drugs is a chronic cough that develops 2 to 3 weeks the medication is begun. This cough sometimes requires withdrawal of the medication, but many patients tolerate the mildest form of the cough and continue to use the drug. This cough should not be confused with the chronic cough sometimes found in asthmatics. Patients using angiotensin-converting enzyme inhibitors do not experience typical airway reactivity, and use of the medication is in not itself a contraindication to diving. Angiotensin-receptor blockers do not usually cause a cough.

Diuretic agents may also be encountered in divers, particularly for treatment of hyperten-

Table 25–6. Important cardiovascular drugs and diving

Drug	Diving Problems
Angiotensin-converting enzyme inhibitors	No effect on exercise capacity; May produce hypotension
Angiotensin receptor blockers	No effect on exercise capacity; May produce hypotension
Antiarrhythmics	None (evaluate cause of drug use; caution on photosensitivity with amiodarone)
Antibiotics	None (caution on photosensitivity with tetracyclines)
Antihypertensives	Reduced exercise capacity; orthostatic hypotension
Aspirin	None
Beta blockers	Reduced exercise capacity; Raynaud phenomenon
Calcium channel blockers	May produce orthostasis or postexercise hypotension
Coumadin	Risk of bleeding with trauma
Digitalis	None (evaluate cause of drug use)
Diuretics	None (caution about hydration in hot climates)

sion. These agents do not interfere with diving but may augment fluid loss in hot climates and cause dehydration. Divers using diuretics for treatment of hypertension should be instructed to reduce the dose when they expect to be exposed to hot weather where excess sweating will occur. Loss of salt and water through sweating and evaporation affects blood pressure in a way similar to diuresis. The combination of both effects can cause excess fluid loss and hypotension.

Antiarrhythmic medications usually do not interfere with diving or exercise performance, but the arrhythmia for which treatment has been subscribed must be elucidated and the relation of diving to the arrhythmia should be questioned. Many antiarrhythmic drugs can cause prolonged Q-T syndrome with resultant ventricular fibrillation of the torsades des pointes type.[84] Of particular interest is the interaction with hypokalemia and the newer antihistamines in producing this effect. Sudden death has been described in a few patients taking terfenadine, an antihistamine known to prolong the Q-T interval, who were exercising excessively with associated sodium and potassium loss.[106]

Terfenadine was removed from sale. Even so, divers using such medications should be warned of this combination of events. Interaction with antiarrhythmic agents that have similar effects on the Q-T interval is also possible. Amiodarone is one agent that appears to be free of this complication,[107] although it is often used for life-threatening arrhythmias that would otherwise preclude diving. When used for control of atrial fibrillation in patients with no other heart disease, amiodarone should not interfere with diving. Amiodarone sensitizes the skin to sunlight, and divers using this drug should be cautioned about sun exposure. Pulmonary fibrosis, known to occur with higher doses of amiodarone, is unusual in the doses used for treatment of atrial fibrillation (100 to 200 mg/day).

Patients on nitrate medications generally have coronary disease that is severe enough to preclude diving. The nitrates per se do not have any specific interaction with the diving environment. There is no known interaction between diving and sildenafil, but divers should be cautioned regarding diving shortly (1 to 2 hours) after taking this drug because it may cause symptomatic hypotension. Table 25–6 summarizes the effect of common cardiac drugs in diving.

Anticoagulant medications are used increasingly in patients with cardiac disease because antiplatelet agents reduce the risk of coronary and other vascular disease as well as the risk of stroke in patients with continuous or paroxysmal atrial fibrillation.[108] Divers taking warfarin or aspirin should be cautioned about mechanical trauma and ear or sinus barotrauma. Either form of anticoagulation can aggravate bleeding from direct injury or barotrauma.

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CHAPTER 26 Diabetes and Diving

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ENERGY METABOLISM IN DIABETES MELLITUS

All of the processes of living cells are processes of energy transformation. These transformations can be divided into three phases:

- Derivation of energy from the oxidation of fuels
- Conversion of this energy into the biologically useful form found in the high-energy phosphate bonds of adenosine triphosphate (ATP)
- Utilization of ATP phosphate-bond energy to drive energy-requiring processes such as walking, running, and diving

The "fuels" of phase one include glucose, fatty acids, ketone bodies, and many amino acids derived from endogenous and dietary sources. The pathways of the oxidation of these fuels converge in the generation of the activated 2-carbon acetyl group in acetyl CoA, which, when completely oxidized in the tricarboxylic acid (TCA) cycle, allows collection of the energy of these reactions, mostly as the reduced forms of the electron-accepting coenzymes nicotinamide-adenine dinucleotide (NAD) or flavin adenine dinucleotide (FAD). This energy is then converted to the high-energy phosphate bonds of ATP by the process of oxidative phosphorylation, in which electrons are transferred from NADH and FAD (2H) to oxygen by the mitochondrial electron transport chain (Fig. 26–1).

All cells continuously use ATP and require a constant supply of fuels to provide energy for the generation of ATP. In a normal subject, the persistent access to fuels despite variations in dietary supply and rates of utilization is called *metabolic homeostasis.* This homeostasis is primarily accomplished by hormonal regulation of the pathways of fuel storage and fuel mobilization, principally by insulin and the insulin counter regulatory hormones: glucagon, epinephrine, and cortisol.

Glucose has a special role in metabolic homeostasis because the brain and many other tissues require glucose for all or part of their energy needs. As a consequence, in nondiabetic subjects, blood glucose levels are normally maintained in the range of 75 to 90 mg/dL. In patients with non–insulin-dependent (type 2) diabetes, however, a resistance to insulin's actions at multiple target cell levels, poorly timed or progressively inadequate secretion of insulin from the pancreatic beta cells, or both eventually leads to a diminished capacity to transport glucose from the bloodstream into the cytosol of insulin-sensitive tissues. The resulting hyperglycemia characterizes the increasingly common disorder of diabetes mellitus. Reduced entry of glucose into cells requires that alternative fuels be available for oxidation to meet cellular energy requirements. The release of fatty acids from adipose stores provides this alternative fuel to those cells that can oxidize fatty acids. This explains the increased level of free fatty acids in the blood as well as the weight loss in patients with poorly controlled diabetes mellitus. In patients with insulin-dependent (type 1) diabetes who have a severely diminished or absent insulin secretory capacity, the flow of large amounts of fatty acids to an "insulin-starved" liver may result in a relatively uncontrolled hepatic generation of ketone bodies such as beta hydroxybutyrate, leading to the potentially lethal complication of type 1 diabetes mellitus known as *diabetic ketoacidosis (DKA).*

Because of the aberrations in metabolic homeostasis, the bioenergetics of the cells in patients with poorly controlled diabetes mellitus are disordered. Thermogenesis (which is particularly important in cold-water immersion), active transmembrane transport of a variety of molecules and ions, the synthesis of enzymes and other proteins, and mechanical work are all compromised in proportion to the severity of decreased glucose oxidation.

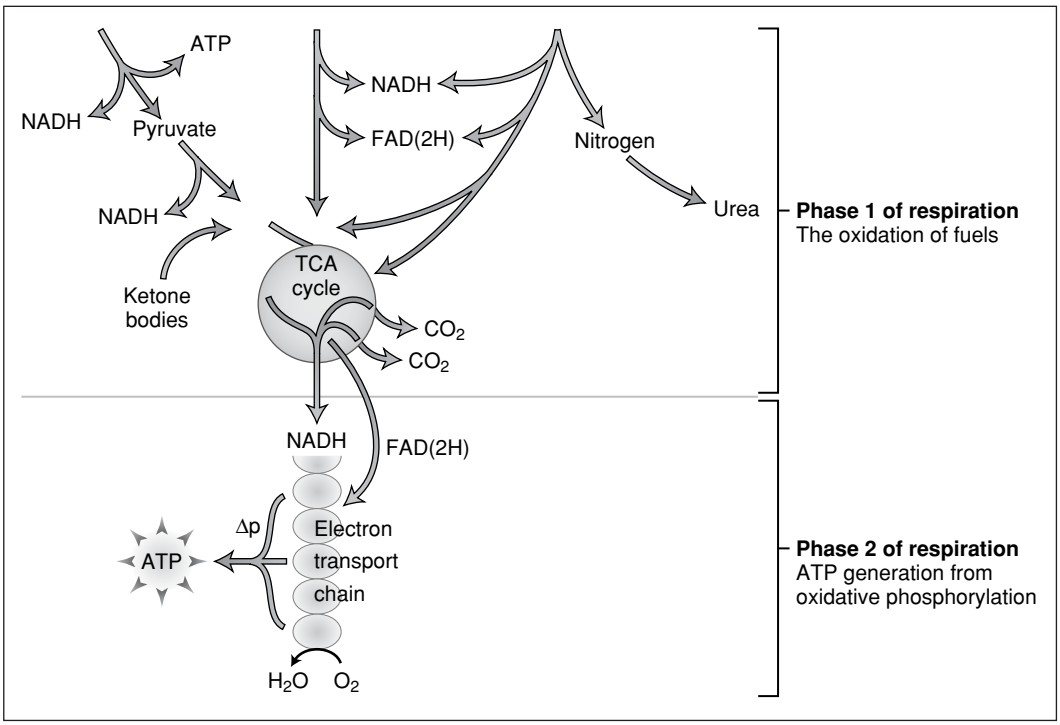

Figure 26–1. Cellular respiratory pathways. ATP, adenosine triphosphate; FAD(2H), reduced form of flavin adenine dinucleotide; NADH, reduced form of nicotinamide-adenine dinucleotide; TCA, tricarboxylic acid. (Adapted from Marks D, Marks A, Smith C: Basic Medical Biochemistry: A Clinical Approach. Baltimore, Williams & Wilkins, 1996, p 272.)

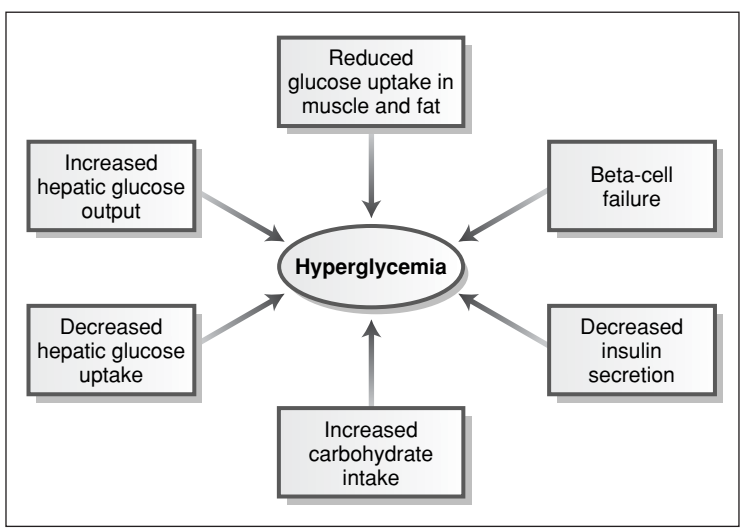

Figure 26–2. Metabolic and hormonal effects that lead to hyperglycemia. (Data from Florence JA, Yeager BF: Treatment of type 2 diabetes mellitus. Am Fam Physician 59:2835–2844, 2849–2850, 1999.)

Type 2 diabetes mellitus has become epidemic in this country, particularly in certain ethnic groups. The increasing incidence of obesity in the United States may also contribute to these alarming prevalence rates because adiposity, particularly visceral adiposity, reduces insulin sensitivity, not only in adipose tissues but in skeletal muscle and the liver.

Figure 26–2 depicts the potential metabolic defects that may contribute to hyperglycemia in patients with type 2 diabetes mellitus. There is no consensus as to when in the development of type 2 diabetes mellitus

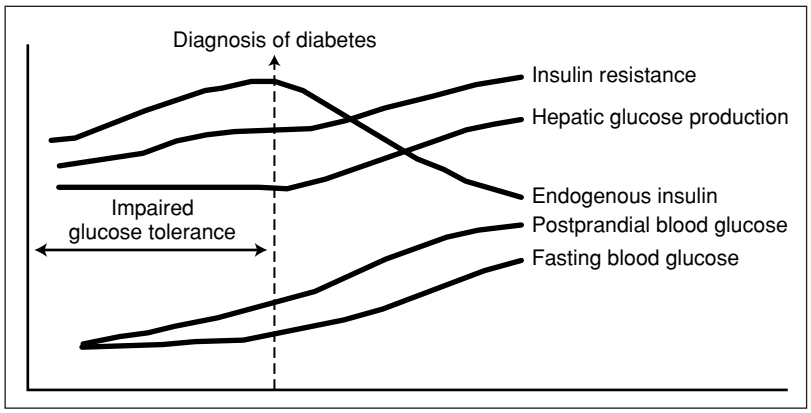

Figure 26–3. Progression of glucose and insulin status from impaired glucose tolerance to overt diabetes. The usual period of impaired glucose tolerance is 4 to 7 years. Microvascular changes progress from the time when diabetes is diagnosed. (Adapted from Ramlo-Halsted BA, Edelman SV: The natural history of type 2 diabetes. Implications for clinical practice. Primary Care 26:771–789, 1999.)

these various influences contribute to the onset of glucose intolerance. In general, however, hyperglycemia should not develop even in the presence of insulin resistance as long as the pancreatic beta cells can produce enough insulin to fully compensate for the insulin resistance. As shown in Figure 26–3, early in the progression toward glucose intolerance, the fasting blood glucose level often remains normal at a time when the postmeal serum glucose level is showing impaired glucose tolerance. Using the fasting blood glucose level to describe the presence or absence of glucose intolerance, the American Diabetes Association guidelines[1] state that a subject is euglycemic if the fasting glucose concentration is less than 110 mg/dL and that a fasting glucose level between 110 and 125 mg/dL represents "impaired fasting glucose tolerance." If the fasting level is 126 mg/dL or higher, diabetes mellitus is said to be present. The criteria used for blood glucose levels measured 2 hours post meal or 2 hours post glucose load are as follows[1]:

- A level less than 140 mg/dL is normal
- A level between 140 and 199 mg/dL indicates "impaired glucose tolerance"
- A level of 200 mg/dL or higher indicates overt diabetes mellitus

The selection of the postprandial guidelines versus the preprandial guidelines for diagnosis of abnormal glucose homeostasis has significant clinical implications. Clinical studies have demonstrated a significant association between the 2-hour postprandial glucose level and cardiovascular mortality.[2,3] On the other hand, no such relationship was demonstrated between cardiovascular death and fasting glucose levels in the blood. An understanding of the mechanisms responsible for postprandial hyperglycemia at a time when fasting glucose levels are still normal is relevant to the therapeutic measures required to prevent postmeal hyperglycemia and its complications.

One such mechanism is the loss of the first phase of insulin secretion in patients with type 2 diabetes mellitus following a meal. In normal subjects, insulin is released in a biphasic pattern postprandially (Fig. 26–4). The first phase of insulin release occurs within 10 min of a meal, reaches a sharp peak at about 4 min, and then rapidly returns to a low level. This short burst of insulin secretion probably suppresses hepatic glucose output, thereby inhibiting postprandial hyperglycemia.

The second phase of insulin release is more gradual and more sustained. It occurs within 30 to 60 min of a meal and continues thereafter for several hours. This more sustained phase of insulin release influences the transport of glucose into insulin-sensitive tissues. In type 2 diabetes, the initial phase is severely blunted; early on, the second phase may be exaggerated, at times resulting in postprandial "reactive" hypoglycemia.[4] As the disease progresses, however, the second phase of insulin release is also delayed and attenuated. These changes suggest that early in the course of diabetes, one might direct therapy toward reestablishing the first phase of insulin release. Rapid-acting and orally administered insulin secretagogues, such as the meglitinides or a formulation of rapidly

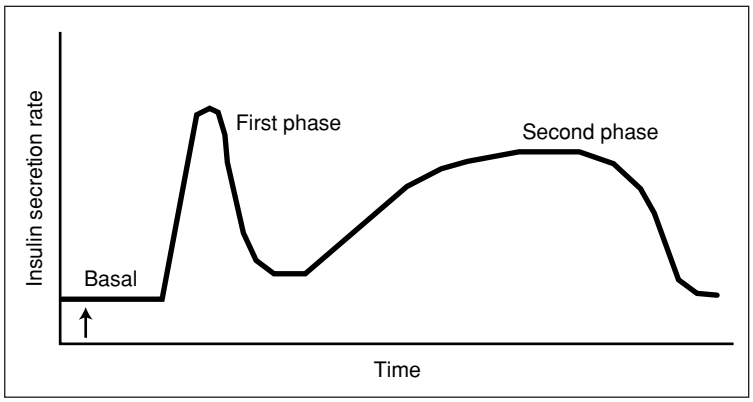

Figure 26–4. Insulin secretory response to a typical glucose meal ingested at the *arrow*. The first insulin peak occurs about 10 min after the meal is ingested; the second peak occurs 30 to 60 min later. (Adapted from Rorsman P, Eliasson L, Renstrom E, et al: The cell physiology of biphasic insulin secretion. News Physiol Sci 15:72–77, 2000.)

Table 26–1. Normal plasma glucose levels (mg/dL) and target levels for diabetic patients

Parameter	Normal	Goal	Action Suggested
Fasting or postprandial glucose	<110	<120	<80 or >140
Postprandial glucose	<140	<180	>180
Bedtime glucose	<120	100 to 140	<100 or >160
Hemoglobin A_{1c}	<6%	<7%	>8%

Data from American Diabetes Association: Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care 24(Suppl 1):5–20, 2002.

acting insulin with a short duration of action, would be an appropriate tailor-made therapeutic option in such patients.

Treatment of diabetes should be based on established blood glucose guidelines (Table 26–1). In all patients with chronic hyperglycemia, the initial approach to therapy is nonpharmacologic. This includes the application of an American Diabetes Association diet, whose daily calorie content is designed to help the patient reach and maintain "ideal" body weight. In this diet, refined sugars are severely limited and complex carbohydrates make up approximately 50% to 55% of total calories ingested daily. Protein should make up approximately 15% of total calories eaten, and fat approximately 30%. One third or less of fat intake should be composed of saturated fat, which is less than 10% of total calories ingested. This phase of diabetes is not associated with hypoglycemia or DKA and does not limit diving.

Aerobic exercises designed to gradually improve the patient's level of physical conditioning are also recommended to the maximum level tolerated by the patient. Weight loss, particularly a reduction in visceral fat, significantly increases insulin sensitivity, not only in adipose tissue but in skeletal muscle and liver cells as well. Dietary sodium restriction is recommended in diabetic patients with elevated blood pressure.

If nonpharmacologic measures fail to bring the fasting and postprandial blood glucose levels into the desired range, a pharmacologic approach is recommended. Because of the prothrombotic tendencies of poorly controlled diabetes, the American Diabetes Association recommends that, unless otherwise contraindicated, all diabetic patients should take aspirin in a dose of 81 to 162 mg/day.

The majority of type 2 diabetics are obese at the time of diagnosis. As a result, their increased adipose mass leads to varying degrees of insulin resistance. First-step therapy should include an insulin-sensitizing drug. Other medication choices depend on the degree of postprandial hyperglycemia. Sport divers on oral diabetes medication can

be permitted to dive, but commercial divers may be prohibited by specific regulations relevant to the work location. If the oral agents used alone or in combination fail to normalize blood glucose levels, then insulin is required. Oral agents are not affected by the diving environment, but they can result in hypoglycemia if the diver does not consume an adequate number of calories after the medications are taken.

It has become clear that, in most patients, both insulin resistance and a diminished insulin secretory capacity coexist once the disease is established and each progresses inexorably over time. Strict dietary compliance, significant weight loss, adherence to a vigorous aerobic exercise program, reduction in stress, and other measures delay the onset and the progression of this disease. However, oral antidiabetic agents eventually fail in the majority of patients, who at some point will require exogenous insulin.

How do these general principles of metabolic homeostasis, bioenergetics, the pathophysiologic mechanisms leading to glucose intolerance and the therapeutic paradigm relate to diving? Perhaps the greatest concern for the diabetic patient with an interest in diving relates to the acute and long-term complications of chronic hyperglycemia (glucose toxicity)[5] and elevated levels of free fatty acids in the blood (lipid toxicity).[6] Concern regarding hypoglycemia are readily allayed by training and good diabetes management (see later).

The most serious acute complication for type 1 diabetic patients that could profoundly influence the ability to perform the activities of diving is DKA. The severe metabolic acidosis and the severe volume depletion that result from vomiting and osmotic diuresis may compromise cardiac output and vascular tone. The resulting lactic acidosis adds to the existing metabolic acidosis, causing cognitive dysfunction, coma, and, if left untreated, death. The potential for DKA represents a very real risk in type 1 diabetic patients who are interested in diving but whose metabolic course has been unstable or who fail to take the prescribed insulin. Such events occur after several days of insulin withdrawal. A remote diving site with inadequate refrigeration to preserve insulin activity could lead to DKA.

A less frequent acute complication of diabetes mellitus is that known as the hyperglycemic hyperosmolar nonketotic state that usually occurs in type 2 diabetics with mild or occult diabetes. These patients are usually middle-aged to elderly. Underlying renal insufficiency or congestive heart failure is common and worsens the prognosis. The process can be precipitated by the use of thiazide diuretics, phenytoin, or glucocorticoids. Screening for these chronic illnesses and use of the above-mentioned medications is therefore essential in divers using oral hypoglycemic agents.

If the patient is unable to maintain adequate fluid intake due to an associated acute or chronic illness or has experienced excessive fluid losses from diuretic exposure or vomiting (e.g., from seasickness), marked dehydration results. As plasma volume contracts, renal insufficiency ensues, thereby limiting renal glucose excretion. These processes markedly increase blood glucose levels, and with that, increase serum osmolality. The hyperosmolar state causes cerebral dysfunction with confusion that begins when the serum osmolality reaches a range of 330 mOsm/L or higher. As the metabolic aberrations progress, coma eventually occurs. The insidious onset of this disorder, preceded for days or weeks by symptoms of weakness, polyuria, and polydipsia and signs of dehydration, make it necessary for physicians involved in diving activities to be aware of this potentially lethal complication of diabetes.

Hypoglycemic reactions are the most common complications in diabetic patients treated with either insulin secretagogues, such as the meglitinides and the sulfonylureas, or with exogenous insulin. Sulfonylurea-induced hypoglycemia is more likely in older patients or those with impaired liver or kidney function who are being treated with long-acting and highly potent oral antidiabetic agents, such as chlorpropamide or glyburide, or with long-acting insulin preparations. Hypoglycemia may also result from a delay in taking in a meal or from unusually heavy physical exertion without an increase in caloric intake or a decrease in insulin dosage.

The signs and symptoms of hypoglycemia are divided into those that induce autonomic hyperactivity (adrenergic symptoms include tachycardia, palpitations, sweating, and tremulousness, whereas parasympathetic symptoms include nausea and hunger) and those due to inadequate availability of glucose to the brain (mental confusion with impaired cognition leading to bizarre

antagonistic behavior and finally coma). Patients who have experienced hypoglycemia usually recognize its occurrence early in its course and, therefore, take the measures needed to rapidly restore euglycemia (such as ingesting a carbohydrate). However, it is known that, especially in patients with type 1 diabetes mellitus who experience frequent modest to severe hypoglycemia, the syndrome known as *hypoglycemic-associated autonomic failure* or "hypoglycemia unawareness" may develop. This syndrome is caused by a reduction in β-adrenergic sensitivity to the rising levels of catecholamines that accompany a significant and sudden drop in blood glucose levels. As a result, the patient may no longer receive the warning signs that blood glucose levels are rapidly decreasing. Physicians involved in diving programs in which sulfonylurea-treated or insulin-treated diabetic patients participate must recognize this syndrome.

Perhaps the most encouraging new information for subjects with a significant predisposition toward type 2 diabetes mellitus is the recent report from the Diabetes Prevention Study[7] showing that a healthy diet and aerobic exercise that reduces body weight to ideal levels actually can prevent the onset of diabetes mellitus in a significant percentage of predisposed subjects.

THE DIABETIC DIVER

For several decades, participation by diabetics in scuba diving has been one of the most controversial issues faced by the diving community. Type 1 diabetes was considered an absolute contraindication for participation in scuba by the Undersea and Hyperbaric Medical Society (UHMS), National Oceanic and Atmospheric Administration, Divers Alert Network (DAN), and all of the major recreational diving training agencies. Yet it was a well-known fact that many diabetics were diving successfully, with no apparent ill effects or evidence of increased diving accidents. Also, no significant body of scientific data supported a blanket ban on the participation of diabetics in recreational scuba. Subsequently, a joint UHMS/American Diabetes Association committee convened in June of 1994 to review established data on this question. Their conclusions are listed in Box 26–1.

In essence, the Diabetes and Diving Committee supported the concept that physically fit diabetics with well-controlled disease could participate in scuba diving. It established the first fitness-to-dive criteria for diabetics. The lack of scientific data concerning diabetes and diving was recognized. The danger of developing hypoglycemia under water was clearly defined as a major risk for the diabetic diver. Importantly, the committee's findings opened the door for various entities to further explore the issue of diving and diabetes.

The committee's findings were supported by surveys conducted by DAN in 1991 and 1995. A total of 129 divers with type 1 diabetes responded to a questionnaire concerning their diving activity.[8] Reportedly, these divers had performed over 27,000 dives without any adverse events. Type 1 and type 2 diabetics have been diving in Europe since 1991 under the supervision of the British Sub-Aqua Club, the Sub-Aqua Association, and the Scottish Sub-Aqua Club.[9] These divers are scrutinized carefully and must meet strict criteria for fitness to dive. A study of these divers revealed that they had logged over 1000 dives without any adverse events secondary to hypoglycemia. Four divers did report mild symptoms that they attributed to early hypoglycemia. Reportedly, all four of these divers' symptoms cleared when they ingested an oral glucose paste while underwater. All four continued their activity. None was confirmed to have glucose levels in the hypoglycemic range. Eighteen divers with type 1 diabetes responded to a questionnaire developed by Whitehouse and colleagues.[10] All of the divers denied having hypoglycemic episodes while diving. Based on these findings, these authors also concluded that fit diabetics could safely dive if they followed an appropriate diabetic management plan.

In 1997, Edge and associates[11,12] studied eight scuba divers with type 1 disease during a simulated dive to 100 ft in a hyperbaric chamber and surface activity of equal exertion and duration. All of the divers were certified and experienced. After a fasting blood sugar was drawn, subjects were allowed their usual predive breakfast and insulin dose. Predive blood glucose was measured 1 hour later. Each diver then performed vigorously for 16 min on an exercise bike. The total dive time for each subject was 23 min (18 min bottom time and 5 min ascend time). Blood glucose levels were monitored at regular intervals for a 5-hour period beginning with the onset of the dive.

Box 26–1. Summary of recommendations of the committee on diabetes and diving

1. Present data do not justify a blanket proscription to diving for diabetics. Clearly, additional research is necessary.
2. A significant number of diabetics treated with insulin or oral hypoglycemic agents are now diving.
3. Persons treated with insulin or oral hypoglycemic agents are at increased risk in diving, principally from hypoglycemia.
4. Diabetics treated with insulin or oral hypoglycemia agents who choose to dive should consider that others—including companions, instructors, and families—share this risk.
5. Until further data become available, it seems prudent to exclude the following groups from diving:
   a. Persons with a history of severe hypoglycemia (e.g., loss of consciousness, seizures, or requiring the assistance of others) within the preceding 12 months
   b. Persons with advanced secondary complications, such as proliferative retinopathy, neuropathy, or coronary artery disease
   c. Persons with hypoglycemia unawareness (lacking stress symptoms of mild hypoglycemia)
   d. Persons with inadequately controlled diabetes (as determined by their physician) or who do not have a good understanding of the relationship between diabetes and exercise
6. A remaining group of diabetics, who have well-controlled disease, are treated with insulin or oral hypoglycemic agents, and well understand their disease, may be considered for recreational diving with suitable training and by following a specially designed management protocol.

During that period, the divers were prohibited from taking any fluids, food, or insulin. Despite this, none of the subjects experienced a significant hypoglycemic event.

These data revealed several significant findings. First, no statistically significant differences were noted in corresponding blood glucose levels drawn at ambient pressure from those drawn at 4 atm of pressure. Therefore, the data suggest that, at least within the normal depth range for recreational scuba, increased pressure on the diver does not alter the blood glucose level. Second, none of the subjects experienced symptomatic hypoglycemia despite vigorous exercise and an unusual postdive period of deprivation.

In 1995, Burghan introduced a plan that met all the guidelines outlined by the Diabetes and Diving Committee.[13] It offered a diabetes management plan, general safety guidelines, measures for preventing hypoglycemia and recognizing hypoglycemia, and appropriate emergency care for hypoglycemia. Burghan's plan advocated frequent self-monitoring of blood glucose (SMBG) and ketone testing before and after diving. He empirically recommended specific predive blood glucose levels in order to prevent a hypoglycemic event. In 1995, Burghan and Winsett reported on a study of 32 divers with type 1 disease who followed Burghan's guidelines.[14] They performed a total of 146 dives without experiencing signs or symptoms of hypoglycemia. Lerch et al[9] confirmed these findings. They studied seven divers with type 1 disease who followed the Burghan guidelines. None of the divers experienced any adverse events or hypoglycemic episodes.

During this same period, Prosterman started a scuba camp for adult diabetics.[15,16] The divers at Camp DAVI (Diabetic Association of the Virgin Islands) follow a diabetic management plan that modified Burghan's original plan. Prosterman developed a flow sheet that assists the diabetic diver in managing diabetes before, during, and after diving activities. Camp DAVI is an excellent venue for diabetic divers to increase their skills and learn to dive safely. Diabetic divers attending this camp have not suffered any significant hypoglycemic episodes.

In early 1995, Scott introduced a protocol designed for training qualified diabetics to scuba dive.[17] It also follows the guidelines recommended by the Diabetes and Diving Committee. The plan includes guidelines for pool activities and open-water dives. It recommends that the diabetic student perform simulated open-water dives while in the

safety of the pool environment. These simulated dives include pre- and postdive SMBG, pre- and postdive meals, pre- and postdive adjustment of insulin dosages, participation of the informed buddy, and appropriate responses to a hypoglycemic event. During the 6 years of its existence, no adverse events have been reported with this protocol. YMCA scuba is the only training agency with a specific program for diabetic divers.

In 1997, DAN began an observational research project to study changes in blood glucose levels in divers with type 1 diabetes.[18] Their main objective was to determine whether and how often hypoglycemic episodes occur when accepted guidelines are followed. The guidelines developed by Burghan[13] and Prosterman[17] formed the basis of their dive management plan. Thirty-three divers with "moderately controlled" type 1 disease were studied while participating in "tropical dive vacations." The divers regulated their own diet, insulin doses, and dive activity. Immediately before a dive, blood glucose levels were required to be greater than 80 mg/dL. A total of 423 dives were performed over 5 days (each diver performed two to three dives daily). No complications secondary to hypoglycemia were reported during or after any of the dives. Two postdive blood glucose levels were in the hypoglycemic range (41 and 61 mg/dL), but the divers were asymptomatic and able to take corrective measures. In this preliminary report, DAN concluded "Results suggest that with careful attention, plasma glucose levels in [divers with type 1 diabetes] can be managed to avoid hypoglycemia during uncomplicated dives conducted under recreational diving conditions." In another preliminary report, DAN had reported that "Pre- to post-dive changes in blood glucose were similar with single or repetitive dives."[8]

Physiology

Hypoglycemia is considered the major risk for any physically fit diabetic who chooses to participate in scuba. Brain function is very sensitive to low concentrations of plasma glucose. Impaired judgment, lack of concentration, and, eventually, unconsciousness result from increasing severity of hypoglycemia. Such a scenario during a dive would endanger not only diabetic divers but also their companions. The potential for developing hypoglycemia is greater in diabetic divers with type 1 diabetes than in those with type 2. During exercise, the muscles increase their use of oxygen, glucose, liver glycogen, muscle triglycerides, and free fatty acids. In nondiabetics, the increased glucose utilization triggers a variety of homeostatic responses in order to maintain the blood glucose concentrations within normal ranges. Insulin declines and concentrations of counter-regulatory hormones (glucagon, growth hormones, catecholamine, and cortisol) rise, increasing hepatic glucogenesis. These normal metabolic responses are disrupted in type 1 diabetes. There is no endogenous source of insulin for self-regulation, and the counter-regulatory mechanisms may be impaired. Therefore, prudent regulation of the diabetic diver's exogenous insulin and carbohydrate intake are of the utmost importance to maintain reasonable metabolic homeostasis while avoiding the severe consequences of hypoglycemia.

In type 2 diabetes, the metabolic response to exercise may be impaired, but generally not to the degree seen in type 1 diabetes. Usually, plasma insulin does not decline and glucose production is slowed, but the resulting drop in blood sugar rarely approaches the point of hypoglycemia. Exceptions to this rule include patients with type 2 diabetes who take sulfonylureas or require exogenous insulin, or both. A physician may address this problem by variously adjusting the caloric intake, insulin, and hypoglycemic medication. It is recommended that diabetics who participate in scuba not be treated with sulfonylureas because of their unpredictability with regard to hypoglycemia.

Strategies for avoiding hypoglycemia during scuba include meal scheduling, decreasing the insulin dose, using the appropriate site and type of insulin, increasing caloric intake, and engaging in prudent pre- and postdive SMBG. Meticulous attention to the development of and adherence to a diabetic dive management plan is essential. This plan should include such issues as symptoms of hypoglycemia, emergency treatment of hypoglycemia, and the participation of an informed dive buddy. The informed dive buddy should be nondiabetic, understand diabetes, and be willing and able to respond to a hypoglycemic event.

Delayed hypoglycemia is another potential danger for the diabetic diver. During

exercise, the skeletal muscle cells become increasingly sensitive to insulin, allowing for increased glucose uptake. The associated increase in demand for glycogen depletes glycogen stores of skeletal muscle and liver. Following exercise, the diabetic's blood glucose concentration may be decreased while glycogen synthesis is increased. However, liver glycogen is replenished more slowly than skeletal muscle glycogen. The resulting delay in hepatic glucogenesis leaves the diabetic diver vulnerable to postdive hypoglycemia that may occur at night, 6 to 15 hours after the day's dives are completed. Delayed hypoglycemia may occur in diabetics who are in excellent metabolic control. This complication is possible when the diabetic diver performs multiple dives over several consecutive days, such as during a dive vacation, because of the prolonged period of increase exertion. Preventive measures include increased food intake, pre- and postdive consumption of complex carbohydrates, increased fluids, abstention from alcohol, reduction of non–dive-related exercise, careful monitoring of postdive blood glucose levels, and prudent reduction of insulin doses when indicated. Night dives should be avoided, unless the diabetic diver has developed a specific management plan and is trained for night diving.

Another potential risk for diabetic divers is complications arising from hyperglycemia. This is more likely when the diabetic is in poor metabolic balance. Unfortunately, some diabetics who participate in recreational scuba fit into this category. Some diabetic divers apparently initiate their diving activity in a self-imposed extreme hyperglycemic state. They reason that the elevated predive blood glucose will prevent hypoglycemia during the dive. Although this is a reasonable assumption, this behavior can lead to severe hyperinsulinemia. Observations conducted by DAN involving 16 recreational diabetic divers revealed that just prior to diving, random blood sugars were in the range of 233 ± 61 mg/dL before the first dive and 218 ± 64 mg/dL before repetitive dives. The divers were not monitored for urine or blood ketones. A predive blood glucose concentration of over 240 mg/dL may be dangerous. If a diabetic diver initiates a dive with significant hyperglycemia or preexisting mild ketosis, or both, the subsequent vigorous exercise may precipitate ketoacidosis. If hyperinsulinemia exists, the exercising muscle cell cannot utilize glucose effectively despite the state of increased insulin sensitivity. Glucagon-induced production of glucose from the liver is unopposed, and fatty acids are mobilized to supply the increased demand for fuel. This results in increasing hyperglycemia, ketosis, and acidosis.

To avoid these complications, SMBG and blood or urine ketone testing should be performed before and after every dive. Prevention should be aimed at keeping the diabetic diver under good metabolic control. Every diver with type 1 diabetes must learn to regulate insulin doses and ingest appropriate snacks in order to duplicate the system that functions automatically in nondiabetic divers. The YMCA scuba program's simulated (in the pool) open-water dives assist diabetic divers in obtaining that goal. In this program, we found that every diabetic diver is required to make adjustments to meet his or her own unique needs.

Fitness to Dive

Diabetics who participate in sport scuba diving must be held to a high physical standard. To qualify for diving, they should be physically fit, exercise regularly, and thoroughly understand diabetic management during exercise. They should chart their daily glucose patterns and know the effects of strenuous exercise on blood glucose levels. They should have no findings of significant systemic diabetes, such as retinopathy, peripheral neuropathy, nephropathy, microvascular disease, macrovascular disease, or diabetic foot. They must be in good metabolic balance as demonstrated by blood glucose levels in the acceptable range and hemoglobin A_{1c} levels consistently at 8% or lower (6.5% to 7.5% is desirable). They should have no recent history of episodes of hypoglycemia while at rest or during exercise, no history of hypoglycemic unawareness (e.g., nocturnal or asymptomatic hypoglycemia), and no recent history of DKA. The diabetic sport diver also must understand the importance of and be willing to accurately perform SMBG and ketone testing. Potential divers must demonstrate that they are mentally sound and mature enough to dive safely with diabetes. The diabetic's personal physician should have the final say in determining fitness to dive.

Diabetic Dive Management Planning

There is no evidence to declare any specific predive blood glucose range as ideal. Selecting an ideal blood glucose range for diving is further complicated by the fact that every diabetic is unique with regard to metabolic state, insulin, and hypoglycemic medication regimen, diet, exercise experience, and general health. All of these factors must be considered when one develops a diabetic dive management plan. Additionally, because of the rapid progress in diabetic therapy, such as the improved insulin pump and new forms of insulin, the various treatment regimens are in a constant state of flux. The underlying goal of management plans is to prevent hypoglycemia during the diving activity, especially while the diver is underwater.

The prototype for most diabetic management plans is that of Burghan and Winsett.[13,14] All of the plans recommend a self-imposed predive state of hyperglycemia. They do not recommend specific adjustments in predive insulin administration or diet in order to obtain this goal. Burghan recommends a "balanced meal" 1 to 2 hours before a dive. The diver is then instructed to perform three SMBG determinations and three tests for ketones: 1 hour before the dive, 30 min before the dive, and immediately before the dive. He emphasizes that the blood glucose level should be rising or at least not falling with each successive test. The recommended blood glucose range for each time frame is:

- 1 hour before diving, 80 to 250 mg/dL
- 30 min before diving, greater than previous blood glucose and between 120 and 250 mg/dL
- Immediately before diving, greater than previous blood glucose checks and between 150 and 250 mg/dL

If the blood glucose level is above 250 mg/dl or ketones are present in the urine (or blood), the diving activity should be canceled. The diabetic should seek advice and adjust the diabetic management plan to achieve better metabolic control. If the blood glucose level falls 20 mg/dL or more during the hour before the dive, Burghan recommends an appropriate snack and serial SMBG and ketone testing until the blood glucose level stabilizes and is within the acceptable range. At that point, the diver may proceed with the planned diving activity. These acceptable predive glucose levels are empirical but based on acceptable levels established for other strenuous sporting activities.

The YMCA SCUBA Diabetic Diver Protocol has similar guidelines for acceptable predive blood glucose levels.[17] The 1-hour, 30 min, and immediate predive SMBG and ketone tests are required. However, the protocol allows for a predive blood glucose range of 120 to 230 mg/dL. Physically fit diabetics with well-controlled disease who exercise regularly can be approved for participation in recreational scuba diving. Such persons should already understand how to adjust their diabetic management plan during exercise. The protocol offers specific instructions concerning the adjustment of predive insulin doses, meals, snacks, and fluids. A predive carbohydrate snack (20 to 30 g) is mandatory; dive bottom times are limited to 30 min or less and to a maximum dive depth of 60 to 100 ft. Most importantly, the YMCA scuba plan provides for monitored, simulated open-water dives in the safety of the pool. During these dives, diabetics can work out any flaws in the diabetic dive plan. If they discover that the predive glucose concentration is not high enough to ensure prevention of hypoglycemia, appropriate corrective measures may be taken.

Most diabetic divers can perform recreational dives safely with a predive blood glucose concentration of 120 to 180 mg/dL if they follow the protocol and their diabetes is well controlled. Therefore, the potential danger of extreme hyperglycemia, complicated by dehydration from diuresis, may be avoided and the diabetic diver may be maintained in a reasonable state of metabolic control. However, the predive blood glucose level should be increased (150 to 200 mg/dL or more) and the bottom time decreased (maximum, 20 min) when diving under adverse conditions, such as strong current, cold water, or excessive workload. It is recommended that all diabetic divers participate in simulated dives periodically, especially after changes in their diabetic regimen. The validity of these simulated dive activities is based on the study of Edge and coworkers,[17] which demonstrated that increasing atmospheric pressure does not alter an individual's blood glucose levels. Therefore, pool dives can be constructed to simulate a typical two-dive open-water experience, including varying degrees of workload and appropriate diabetic management during

surface interval time. We recommend that diabetics limit their diving activity to no more than two dives daily. SMBG and ketone testing should be performed immediately upon completion of the first dive. Appendix 4 provides a management protocol for diving with type 1 diabetes.

Responses to Hypoglycemia

The diabetic diver must be alert for any signs of hypoglycemia (Box 26–2) while underwater. We agree with other authors who recommend that the diabetic diver and an informed buddy carry a carbohydrate source during a dive such as Insta-Glucos honey in a squeeze bottle or a glucose paste that can be ingested underwater. If any signs of hypoglycemia develop, the diabetic diver and informed buddy should immediately terminate the dive and safely ascend to the surface. If the diver's blood glucose is in the hypoglycemic range, corrective measures should be taken and the diabetic's management plan reevaluated. If the blood glucose level is normal, other reasons for the symptoms should be considered, including barotrauma, decompression sickness, and other dive-related injuries. Diabetic divers and their informed buddies should practice responding to a hypoglycemic episode in the pool during a simulated dive. While underwater, diabetics should give an established hand signal (such as the letter "L" made with the thumb and index finger) to indicate the possibility of hypoglycemia. They should safely ascend to the surface, establish positive buoyancy, and move out of the pool. The informed buddy should then check the diabetic's blood glucose.

Box 26–2. Signs and Symptoms of Hypoglycemia

Early warning signs: unusual hunger, headache, alteration of mood, nervousness, fatigue
Mild reaction: tremors, pounding and/or rapid heart rate, sweating, clamminess of skin, extreme fatigue
Moderate reaction: severe head and/or neck pain, extreme alterations of mood, irritability, extreme fatigue
Severe reaction: decreased awareness or unresponsiveness, unconsciousness, convulsions

CONCLUSIONS

There is sufficient evidence to recommend that physically fit, well-controlled, properly trained diabetics may safely participate in recreational scuba diving. This recommendation does not apply to commercial or military diving. For safe diving, the diabetic must thoroughly understand the disease and the management of exercising with diabetes. Development of hypoglycemia while underwater is an inherent risk that can lead to drowning. Diabetics, their companions, and their families must recognize and assume the potential risk. This risk may be minimized by adherence to a meticulously developed diabetic dive management plan, participation of an informed dive buddy, and a commonsense approach. No definitive safe predive blood glucose range has been determined, but present data suggest that the empirical values recommended by existing management plans are adequate. Further research and data are necessary to establish absolute guidelines. Simulated dives in the pool are recommended to allow diabetics and their informed buddies to work out flaws in the dive management plan. Appropriate responses to a hypoglycemic event should also be practiced.

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CHAPTER 27 Medical Evaluation for Sport Diving

Alfred A. Bove

Whether diving is considered a recreation or an occupation, performance and safety require a reasonable level of physical conditioning. Recreational divers are required to swim, and safety may depend on the ability to deal with adverse surface conditions to return to a safe haven. Excellent health and adequate physical condition provide the needed safety margin for sport diving. Fitness considerations for commercial and for military divers are presented in Chapters 28 and 29.

All divers require some level of medical surveillance. Physicians who become involved with the care and evaluation of divers must know diving physiology and pathophysiology, the diving environment, and the diver in order to provide meaningful medical advice about diving fitness. Because of the unique environment involved in diving, physicians who support recreational divers should have training and experience in the operational aspects of diving. Most diving training programs for physicians offer not only didactic information but also provide hands-on training in the use of hyperbaric chambers and actual exposures to diving with a variety of equipment. The physician who wishes to work with recreational divers is encouraged to learn the details of diving equipment and experience some aspects of diving. In some countries, examiners must be certified in diving medicine. In the United States, a certification of added qualification in Diving and Hyperbaric Medicine is available to physicians who are board-certified through the American Board of Medical Specialties.

DIVING ENVIRONMENT

The recreational diving environment is usually comfortable, with few extremes of temperature or water conditions. Recreational divers, however, have been exposed to Arctic and Antarctic diving, mixed-gas diving, and diving to depths of 200 to 250 feet. Recreational diving agencies in the United States restrict recreational diving to 130 feet and use of open-circuit scuba. Many recreational divers, however, are exceeding these limits in the area of technical diving, wherein special gas mixtures (helium-oxygen and helium-nitrogen-oxygen) may be used (see Chapter 6). Recreational divers may spend 6 or 7 consecutive days performing five or six daily dives, thereby increasing the risk for excess gas supersaturation and decompression sickness (DCS).[1]

Training for recreational diving involves organized educational programs sponsored by dive training organizations. These programs may include 14 to 18 hours of didactic lectures and 20 to 30 hours of in-water training in a controlled underwater environment. Entry into a recreational diving program requires completion of a medical questionnaire but no physical examination in the United States. In Australia and New Zealand, a medical examination is required for recreational divers, which must be performed by a physician trained in diving medicine. In England, a medical examination must be completed by a physician and provided to the diving candidate before a recreational training program is begun. Once trained, a sport diver may never undergo a subsequent diving physical examination.

Physical requirements for recreational diving are minimal. Poor physical conditioning may not limit recreational diving if a diver uses appropriate judgment and avoids difficult diving conditions. Energy requirements for typical recreational diving are low. For a recreational diver of average size, oxygen consumption is about 10 mL/kg/min in a typical dive, but adverse conditions may require an oxygen consumption of 25 to 30 mL/kg/min

(Fig. 27–1). A poorly conditioned diver might require rescue in adverse conditions. Limitations in physical capacity of recreational divers may result from poor conditioning or chronic medical disorders. The medical practitioner evaluating a sport diver must determine the physical condition of the diver candidate and detect any complicating medical disorders. Divers with physical handicaps have participated in recreational diving, and many recreational divers with chronic illnesses can perform limited diving that does not compromise their health or safety.

There are important differences in the medical requirements for sport and commercial diving. Standards acceptable for sport diving are usually not stringent enough for commercial diving. Commercial divers are compensated by their employer to work toward the goals of the employer or the diving project, not toward the personal comfort of the diver. Although national and local regulations aim to protect the safety and health of commercial divers, the United States has no formal regulations that apply to recreational diving. Solo self-employed divers and diving instructors who are involved in teaching and training of sport divers are outside the jurisdiction of most commercial diving regulations. Medical requirements for sport diving therefore rest on the physician, the diver, and the training organization that reviews medical qualifications of candidates for sport diving training.

The following discussion regarding medical conditions that limit diving provides a basic conceptual framework into which each diving situation can be placed and a decision made regarding qualifications for diving.

MEDICAL DISORDERS THAT MAY LIMIT DIVING

There are no regulations or standards for sport divers except those set forth by training agencies for acceptance of students into scuba classes.[2] Several interested private agencies have created guidelines for evaluation of recreational divers.[3]

Sport scuba diving guidelines may vary among persons. A new diver may be disqualified if the medical history or physical examination suggests a risk from diving, whereas an experienced, certified sport diver might be prone to similar conditions and not prohibited from diving. For sport divers, the physician can only make recommendations regarding safe diving.

Neurologic Disorders

HEAD INJURIES

After head trauma, residual neurologic deficits may limit diving performance, or produce post-traumatic seizures. Significant risk factors for late seizures are brain contusion with subdural hematoma, skull fracture, loss of consciousness or amnesia for more than one day, and an age of 65 years or older.[4] Aviation medicine guidelines suggest that with even momentary amnesia or unconsciousness, there is brain damage, but if all studies are normal, including electroencephalogram (EEG) with no anticonvulsant medication, diving could be considered after six weeks.[5]

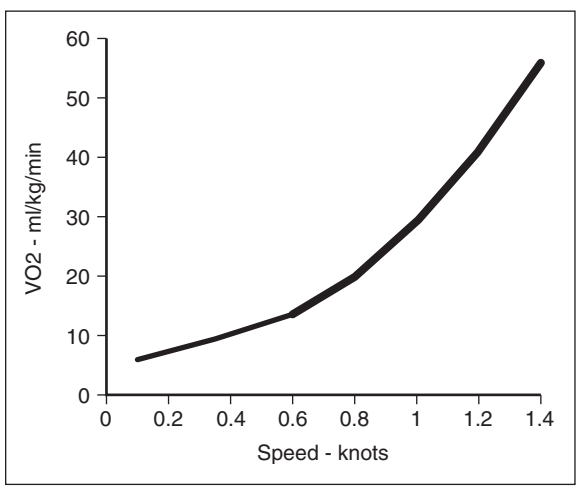

Figure 27–1. Oxygen consumption for swimming underwater with scuba equipment at different speeds. (Modified from Navy Department: U. S. Navy Diving Manual. Vol 1, revision 3: Air Diving. NAVSEA 0994-LP-001-9110. Washington, D. C., U. S. Government Printing Office, 1996.)

For the diving physician, the best course is to consult with the neurosurgeon or neurologist before providing a recommendation for the diver. Sport divers should dive in locations where appropriate medical support is available.

SEIZURE DISORDERS

Because of the risk of drowning and the risk to rescuers, seizure disorders are disqualifying for diving, regardless of control by anticonvulsant medication (see Chapter 23). A seizure may be preceded by an aura manifested by motor, sensory, or emotional changes. The typical periodic breath-holding that occurs during the tonic and clonic phases of a major seizure create a high risk for a pulmonary overpressure accident, with resulting pneumothorax or arterial gas embolism. A history of episodic unconsciousness early in life should also evoke evaluation.

The review by Berg and colleagues[6] provides a basis for accepting febrile seizures as benign. There is a 13% risk of subsequent nonfebrile seizures when two or more of the following are present with febrile seizures:

- Abnormal neurologic examination results
- Prolonged (>15 min) or focal seizure or one associated with transient or permanent neurologic deficit
- Family history of nonfebrile seizures

Other seizure disorders do not meet current United States Occupational Safety and Health Administration (OSHA) standards for commercial divers.[7] In sport divers, seizures secondary to sepsis, meningitis, or drug ingestion and post-traumatic seizures without subsequent neurologic sequelae are acceptable if the patient is not currently experiencing seizures. Completely normal neurologic examination results are mandatory for a patient with a history of any of the above-described seizures or febrile seizures of early childhood. In addition, an EEG and a neurologic consultation may be indicated. Syncope, feelings of faintness, sweating, and pallor can occur in otherwise normal subjects under conditions of emotional stress, repulsive sights, or excessive heat if combined with hypoglycemia or other physiologic stresses. Because maintenance of consciousness underwater is essential, persons who suffer unexplained or repeated episodes of syncope should be disqualified.

CHRONIC BACK AND NECK DISORDERS

Back and neck problems are common findings in most populations. If there are no neurologic or physical impairments, one can safely dive, although with caution about further injury. In sport diving, climbing and lifting can lead to further spine injury. With any neurologic manifestations, diving should be avoided until symptoms have resolved. After successful disc surgery, diving should not resume until the patient is symptom-free for 8 to 12 weeks. Treadmill stress testing can be used to determine functional status after back surgery. Patients should show significant improvement in ambulatory time and onset of pain after laminectomy.[8] Neurologic deficits related to radiculopathy may cause confusion in a symptomatic diver because of the similarity between signs of radiculopathy and signs of DCS. When residual neurologic deficits result from spinal radiculopathy, the neurologic examination should be carefully documented and the diver provided with the details of the neurologic findings. Many divers with previous low back symptoms and neurologic signs are diving. Knowledge of their permanent neurologic deficits and comparison of a current neurologic examination with previously documented findings often avoids the need for recompression treatment (also see Chapter 23).

STROKE

In most cases, a stroke is reason for disqualification because the resulting physical limitations would interfere with safe diving. Besides physical limitations, persons with stroke due to vascular disease may be at risk for subsequent cerebral or cardiac events. For persons with no further risk of cerebral events who have adapted to their functional limitations, limited sport diving may be possible. Each stroke case must be considered individually in terms of sport diving. Persons with carotid stenosis greater than 70% may be at risk for a stroke or transient ischemic attack while diving or performing other exercise, but no data are available to quantify the risk of stroke while diving. Everson and coworkers[9] studied males with mild carotid stenosis or evidence of nonobstructing plaque. They found that strenuous activity or elevated blood pressure caused the disease to progress significantly over 4 years. These

findings suggest that a diver with significant carotid stenosis is at risk for progression of the stenosis. If auscultation over the carotid arteries reveals a bruit, an ultrasound study will provide a quantitative measure of the severity of narrowing. A diver with a stenosis of greater than 70% should be prohibited from diving; those with lesser degrees of narrowing should be screened periodically to ensure that the narrowing is not progressing.

RETURN TO DIVING AFTER DECOMPRESSION ILLNESS

Data that directly bear on the advisability of further diving after a diving-related neurologic injury are sparse. A careful clinical evaluation aimed at elucidating the cause of the injury is particularly important. A neurologic syndrome due to arterial gas embolism caused by an anatomic lung abnormality requires different advice on return to diving than a DCS injury of the spinal cord from missed decompression. If the initial neurologic deficit completely clears within 24 hours with or without therapy, severe neurologic injury is unlikely to have occurred, although some reports suggest that microscopic damage has occurred.[10,11]

In focal brain ischemia, a state of neuronal paralysis can exist for hours while there is potential for complete recovery if normal blood flow can be restored.[10,12] This circumstance has been called the *ischemic penumbra,* and it is associated with reduced local blood flow to levels that inhibit cell function but preserve viability. Transient ischemic attacks, defined as focal neurologic symptoms with abrupt onset and rapid resolution lasting less than 24 hours and due to altered circulation to a limited region of the brain, are related to this state.[13] On this basis, one might be justified in viewing a person who suffered neurologic DCS and whose neurologic deficit cleared completely with or without recompression therapy in 24 hours or less as having minimal residual injury.

There are no guidelines for a return to diving after neurologic injury based on neurologic imaging. Disparities between clinical examination and imaging data are such that clinical evaluation should guide decisions on return to diving. Guidelines from the *U. S. Navy Diving Manual*[14] are representative: Divers who have pain-only DCS, meet the criteria for treatment with USN Table 5 (see Chapter 10), and have complete relief of symptoms can return to diving 48 hours after completing treatment; those who are treated for pain-only DCS using Table 6 (see Chapter 10) should wait 7 days before returning to diving. Following sensory deficits that completely resolve with treatment using Table 6, but in the absence of motor deficits, diving may be resumed after 14 days if all neurologic abnormalities have resolved. Divers with more severe neurologic symptoms or who have sustained neurologic injury from arterial gas embolism should not dive for at least 4 weeks, and only then after an examination shows that abnormal neurologic findings have resolved. When saturation treatments are needed to restore function, a diving medical officer should review the case and diving should not resume for a minimum of 3 months. Although they apply mainly to military divers, these guidelines can be reasonably applied to recreational divers as well.

A persistent neurologic deficit is considered disqualifying in many guidelines.[15] These policies are based on concern for more severe damage in already injured nervous tissue, but this concept has not been supported with clinical evidence to date. Blood flow in zones of ischemic central nervous system damage may remain at subnormal levels, with impaired ability of the vascular bed to adjust its resistance to changes in perfusion pressure; hence, the injured regions would be at increased risk during states that cause further lowering of local blood flow.

OTHER ABNORMALITIES OF THE CENTRAL NERVOUS SYSTEM

OSHA standards[7] require the disqualification of commercial divers with a tumor in any location, including the brain, until they are tumor-free for 5 years. Many sport divers have returned to diving in 6 to 12 months after successful therapy for cancer. This is particularly true for men with prostate cancer and women treated for breast cancer. Intracranial shunts disqualify a commercial diver. There are sport divers with ventriculovenous shunts who have been diving safely. These divers should explore the medical services available when they travel to remote diving sites because an acute shunt closure would require neurosurgical consultation. Huang and associates[16] studied several shunts in vitro and found no abnormalities of function at 1 and 4 ata.

PERIPHERAL NEUROPATHY

Diver candidates with peripheral neuropathy require evaluation of functional disability that would interfere with diving. Because of the inability to differentiate neuropathy from DCS, careful documentation of existing neurologic findings is essential. In its later stage, diabetes mellitus[17] is a common cause of peripheral neuropathy in the United States. Nerve injury from disease of the spine is also considered to be peripheral neuropathy. Neuropathies caused by chemical toxicity and vitamin deficiencies are rare. Persons with sensory deficits are prone to skin damage due to undetected trauma while diving, and motor neuropathies may cause functional impairment. Such cases require individual evaluation. For example, a commercial diver with a residual sensory deficit along L2 following repair of a herniated disk is usually not impaired, but the same diver with quadriceps atrophy might be disqualified.

Ophthalmic Disorders

Butler[18] provides a detailed, excellent review of ophthalmologic considerations in diving. A diver's visual acuity and visual fields must be adequate for safe sport diving. Near vision adequate to read the pressure gauge, watch, compass, dive computer, decompression tables, or depth gauge is necessary. The U. S. Navy permits qualified divers to undergo refractive laser surgery and return to diving. Visual correction may be accomplished by using contact lenses or by having the diving mask fitted with corrective lenses glued to the faceplate.

The existence of glaucoma or ocular hypertension does not preclude diving as long as visual acuity and visual fields are adequate. Retinal detachment that has been repaired or laser photocoagulation of the retina is not considered a contraindication to diving, nor is color-vision deficiency for the sport diver. Sport divers who have undergone cataract removal and artificial lens implantation are diving safely.

Cardiovascular Disorders

Chapter 25 provides a detailed discussion of cardiovascular disorders. Coronary artery disease is more common in persons over the age of 40; the incidence is particularly high in the elderly.[19] However, younger persons can also have coronary and other forms of heart disease. The population of those seeking medical clearance for diving may have a variety of congenital abnormalities of the heart, some of which are important in the diving environment. Of greatest importance is the need to identify those divers at risk for coronary artery disease. This disease may be silent until it manifests as sudden death. The nature of the progress of narrowing of a coronary artery is such that the threshold for symptoms may not be reached until the sudden occurrence of occlusion of an artery occurs and acute myocardial infarction; some cases result in sudden death.[20]

Patients with congenital heart disease may have no signs or symptoms of their disease until later in life. An atrial septal defect may be undetected until right-sided heart failure occurs in the fifth or sixth decade of life. An asymptomatic atrial septal defect allows right-to-left shunting and presents the risk of paradoxical embolization of bubbles or thrombus. Atrial septal defect is therefore a contraindication to diving.

A patent foramen ovale (PFO) is found in 17% to 35% of the population.[21] Following diving or altitude exposure, bubbles in the right atrium can travel through a PFO to form arterial emboli. A PFO cannot be detected by physical examination but can be detected by echocardiogram. The role of PFO in DCS remains unclear. No prospective studies have been performed to identify the actual risk of DCS in the presence of a PFO. Several studies indicate that a PFO is more prevalent in divers with severe DCS (see Chapter 25).

Routine echocardiographic screening of divers for a PFO is not indicated. However, the presence of severe DCS or unexplained cerebral injury indicates a more detailed evaluation.[22] The presence of a PFO in a diver who sustains neurologic injury is not a contraindication to further diving. The risk of DCS in divers with a PFO is quite small.[23] Recurrent DCS in the absence of obvious missed decompression should prompt a search for a PFO.

Ventricular septal defects are most common in the upper septum in its membranous portion.[24] Small defects do not cause right-to-left shunting and therefore are not likely to increase the risk of arterial emboli during diving.[25] Persons with such defects, however, should be counseled on the need

for antibiotics because of the increased risk of endocarditis during dental procedures. A small membranous ventricular septal defect is not a contraindication to diving.

Most valvular heart disease involves trivial abnormalities that produce no cardiac limitations. The most serious valve disease involves stenosis of the aortic or mitral valves (see Chapter 25). These two lesions can result in severe complications with diving. A diver with severe aortic stenosis may experience sudden death while diving. Mitral stenosis causes acute pulmonary congestion during exercise. The combination of physical activity plus central fluid shifts due to water immersion when diving cause fluid shifts into the lungs[26] that may progress to pulmonary edema in patients with mitral stenosis.

Regurgitant lesions of the aortic and mitral valve are less risky in terms of diving. Mild to moderate regurgitation is well tolerated during exercise and does not prohibit an person from diving. However, severe regurgitation of either valve produces congestive heart failure that can be aggravated by exercise and water immersion. Patients with mitral valve prolapse have no limitations to diving. Symptoms that are sometimes associated with mitral prolapse, such as chest pain or arrhythmias, are as frequent in patients without prolapse[27] and should be dealt with separately.

Lifetime risk of coronary artery disease at age 40 is 48% for males and 32% for females.[28] There is a significant risk of coronary disease appearing in male divers older than 40 (Fig. 27–2). Females older than 50 are also at increased risk. Screening tests can be used to identify persons at risk for a coronary event. Early disease may go undetected and is best evaluated by examining risk factors for coronary disease. Risk factors include increased blood lipids, diabetes, cigarette smoking, hypertension, age, and a family history of early coronary events. Persons older than 40 with more than one of these risk factors it is at increased risk for premature coronary disease.[29] Risk factor analysis should be used to screen sport diving candidates for coronary disease (Table 27–1). This can be done by physical examination, history, and simple blood studies. The cost of such screening is minimal and does not require more complex techniques. Occult coronary disease may be present for a considerable period before symptoms appear. Thus, the risk-factor screening procedure is essential in evaluating diving candidates. Diving

Table 27–1. Risk Factors for Coronary Disease

Factor	High Risk
Age (male)	> 40 years
Age (female)	> 50 years
Family history of CAD	Siblings or parents
Smoking cigarettes	Any smoking
Hyperlipidemia	Low-density lipoproteins > 100
Hypertension	Blood pressure > 140/90
Diabetes	Hemoglobin A_{1C} > 7
Overweight	Body mass index > 25

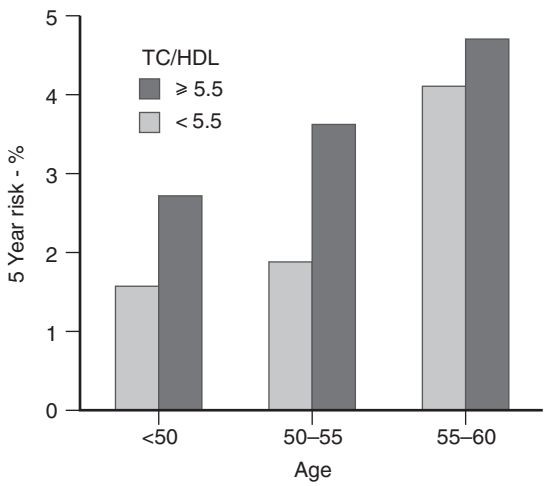

Figure 27–2. Risk of a coronary event (angina, myocardial infarction or sudden death) in 5 years based on age and ratio of total cholesterol to high-density lipoprotein cholesterol. Other risk factors act as multipliers. These include cigarette smoking, diabetes, and a diastolic blood pressure > 90 mm Hg. Each additional risk factor doubles the risk. (Data adapted from L'Italien G, Ford I, Norrie J, et al: The cardiovascular event reduction tool [CERT]: A simplified cardiovascular risk prediction model developed from the West of Scotland Coronary Prevention Study [WOSCOPS]. Am J Cardiol 85:720–724, 2000.)

deaths from coronary disease constitute the second most common cause of death in diving[30] and increase in incidence with each decade above the age of 40.[31]

Hypertrophic cardiomyopathy, particularly with obstruction, increases the risk of sudden death with exercise.[32] This disorder should be a contraindication to diving because of the risk of ventricular arrhythmias and the increased risk of sudden death. Hypertrophic cardiomyopathy with obstruction is considered to be the leading cause of sudden death in athletes[33] and is difficult to detect by clinical examination in asymptomatic subjects. Diagnosis can be confirmed by echocardiography. The presence of any outflow gradient, arrhythmias, or evidence of heart failure is cause for excluding patients from diving.

In a diving population, supraventricular (atrial) tachycardia is commonly due to external stimuli that provoke this rhythm. Alcohol, fatigue, decongestants, and caffeine can combine to excessively stimulate the heart. Performance-enhancing sports supplements that contain stimulants are also known to cause arrhythmias.[34] Avoiding cardiac stimulants is often enough to prevent any further arrhythmia. Subjects with recurrent supraventricular arrhythmias should be prohibited from diving until the cause is discovered and the arrhythmia is treated. Ventricular arrhythmias may also be stimulated by the same combination of agents. Cardiac disease also provokes ventricular arrhythmias that are not benign.[35] Subjects with the inherited long Q-T syndrome may be at risk for sudden death when diving.[36]

There are sport divers with atrial fibrillation who dive safely. Therapy may involve anticoagulation, and special precautions are needed to avoid trauma and ear or sinus squeeze because of the risk of excess bleeding. Chapter 25 presents other aspects of rhythm and conduction abnormalities that must be considered in sport diving.

Peripheral Vascular Disease

Peripheral arterial insufficiency may be associated with vascular disease in the coronary, renal, or cerebral circulation. Vascular insufficiency of the lower extremities impairs one's normal ability to walk or climb and limits diving performance. For recreational diving, persons with minimal claudication and no other organ involvement should be able to dive safely under ideal conditions. Exposure to cold water should be avoided. Poor wound healing is a common accompaniment of vascular insufficiency and would result in risk for chronic open wounds from minor diving injuries.

Pulmonary Disorders

Chapter 24 provides a detailed discussion of pulmonary disorders. Abnormal findings on the chest radiograph are unlikely in sport divers with no history of pulmonary disorders, and such radiographs are not required. Asthma has been controversial in diving because of possible air trapping and pulmonary overinflation during ascent. Active asthma with evidence of air trapping would prohibit diving. Elliott's report[37] provides a detailed discussion of issues related to asthma and diving. Even divers with a remote history of asthma but normal spirometric results are at little risk for an asthma-related problem during diving.

PNEUMOTHORAX

Clinical and operational experience suggests that patients with a history of spontaneous pneumothorax are at risk for lung collapse and a progressively worsening tension pneumothorax during ascent.[38] Treatment may require immediate decompression of the pleural space through an intercostal needle or chest tube and may be difficult in remote diving sites. Persons with a history of spontaneous pneumothorax are usually found to have defects on the pleural surface (blebs or bullae) that are prone to rupture under conditions of ambient pressure change. Newer thoracoscopic techniques of bleb removal without thoracotomy have reduced the frequency of recurrent spontaneous pneumothorax in affected persons.[39] Some sport divers have returned to diving after bleb removal.

Ear, Nose, and Throat Disorders

Middle ear barotrauma is the most common diving medical problem (see Chapter 22). Nasopharyngeal congestion usually results in inadequate eustachian tube function and

subsequent middle ear barotrauma. Attention should be paid to persons with evidence of middle ear or nasopharyngeal disease. Poor eustachian tube function is suggested by frequent ear infections or drainage, a history of middle ear surgery, a healed or persistent eardrum perforation, and cholesteatoma. Chronic paranasal sinus disease predisposes to sinus and middle ear barotrauma. Common causes are allergy, infection, smoking, overuse of nasal sprays, toxic or irritating chemical vapors, obstruction from nasal deformities, and vasomotor causes.

A history of previous ear surgery should alert the examiner to continuing poor eustachian tube function. After a tympanic membrane perforation is repaired, diving can be considered if the eardrum has remained healed and the ear can easily be cleared on the surface. Equipment is available to prevent water from entering the ear canal during diving and has allowed some divers with perforated ear drums to dive without contaminating the middle ear with the surrounding water. Simple mastoidectomy with adequate eustachian tube function does not prohibit diving. A radical mastoidectomy, which involves removal of the posterior external auditory canal, is a contraindication to diving. Patients who have undergone stapedectomy or stapedotomy should not dive because of the increased risk of an oval window fistula and inner ear injury. However, some divers have returned to diving successfully after stapedectomy. Persons who have Meniere disease (characterized by vertigo, tinnitus, and hearing loss) or another inner ear disease with recurrent vertigo should not dive. Nausea and vomiting associated with vertigo underwater can result in drowning.

Maxillofacial and Dental Considerations

Divers with full or partial dentures should be considered individually. Stability of a partial denture with a scuba mouthpiece may vary. Partial dentures must be retained securely enough that no loosening or loss will occur during diving. Advanced periodontal disease with loose teeth may be a problem with a scuba mouthpiece. Dental prostheses should be removed when a full helmet or face mask is used. Custom mouthpieces can be modified to prevent impingement on the orthodontic appliances. Teeth undergoing treatment (e.g., root canal) are susceptible to barotrauma due to retained gas pockets. Tooth replacement or a subperiosteal frame implant should withstand normal biting forces and present no problem in diving.

Endocrine Disorders

Hypothyroidism with thyroid replacement and hypopituitarism requiring replacement hormones are of no consequence to divers. Hypoadrenalism may cause hypotension with moderate trauma due to a lack of corticosteroids. Individual cases must be reviewed to determine the severity of these disorders, but the diver often can be qualified. Of more importance is insulin-dependent (type 1) diabetes mellitus (see Chapter 26). The most widespread recommendation of diving medicine physicians and certifying agencies is to disqualify persons with insulin-dependent diabetes from any type of diving. Hypoglycemic symptoms, including impaired judgment and seizures, can result in a serious accident if they occur underwater. Some patients with type 1 diabetes do dive successfully. The American Diabetic Association sports committee indicated that hypoglycemia is usually not a problem in insulin-dependent diabetics. The YMCA in the United States has instituted a diabetic diving program. More than 100 divers were trained in 1998 to 1999 with no accidents or injuries (see Chapter 26). All divers who use insulin must consider insulin availability while traveling. Insulin must be refrigerated and kept available for daily use. Remote sites that compromise the availability of insulin should be avoided. Non–insulin-dependent (type 2) diabetes that is controlled by weight reduction or oral hypoglycemic agents would not disqualify a sport diver. Coronary disease resulting from diabetes of any type should be considered, and proper screening should be carried out to rule out ischemic heart disease.

Gastrointestinal Disorders

When quiescent, chronic disease of the gastrointestinal tract is usually well tolerated in sport diving. Water immersion eliminates the

usual gravitational dependency of abdominal contents and permits gastric reflux even with normal function of the gastro-esophageal junction.[40] Gastro-esophageal reflux can result in aspiration and reflux of gastric contents into the diver's regulator. Hiatal hernia presents a risk of overdistention of the gastric remnant in the hernia with rupture on ascent. Gastric outlet obstruction presents a risk of overdistention of the stomach on ascent.[41]

An abdominal hernia that contains segments of intestine should be repaired before a person dives. Risk of incarceration is increased with heavy lifting, and air trapping in the contents of the hernia leads to strangulation when air expansion occurs during ascent.

A diver who has frequent acute bouts of diverticulitis would be at risk for serious infection if an episode occurred in a remote site without adequate medical therapy.

Hematologic Disorders

Anemia with a hemoglobin level below 11 g/dL should be corrected before diving is permitted.[42] Sickle cell trait is present in 6% to 8% of divers and diving candidates of African American descent.[43,44] Hemoglobin S levels can make up 30% to 50% of total hemoglobin. No data indicate that persons with sickle trait should be limited by the percentage of hemoglobin S. Diving is not contraindicated in candidates with sickle cell trait.[45]

Injuries and Inflammatory Conditions

A fracture, sprain, or dislocation, bursitis, tendinitis, or other inflammatory process limits range of motion and strength, an could compromise diving safety. No pain should be present that could impair the diver's ability to perform in diving emergencies or that could be confused with DCS. The examining physician must know the particular physical requirements of the diving equipment and the diving exposure to ensure that a local injury will not compromise safety or performance.

Amputation

The presence of an amputated limb is not in itself a contraindication to diving. Performance limits caused by the missing limb dictate capacity and whether the imposed limitations present risks. Amputees have been successfully trained to dive. Many unilateral upper- or lower-extremity amputees participate successfully in recreational diving with special equipment. Bilateral amputees have been trained to dive but require support by other divers.

Artificial Joints

With the proliferation of artificial joints to restore mobility of joints damaged by injury or arthritis, many persons with one or more artificial joints have sought training in sport diving. Artificial joints, plates, screws, or other internal fixation devices need not preclude diving if well healed and secure and if full range of motion and strength are present.

MEDICATIONS

Divers should not require chronic use of antihistamines, vasoconstrictors, and nasal sprays to clear the ears, and the use of such agents should be discouraged. However, occasional use is common and does not appear to cause serious complications during diving. There is little evidence that medications used for most common disorders interfere with diving. Sedative drugs cause problems of impaired judgment, as they do in other circumstances. The hyperbaric environment of sport diving (1 to 5 ata) does not affect the action of antibiotics, antihypertensive medications, cardiac antiarrhythmic medications, gastric acid inhibitors, gastric motility drugs, or anti-inflammatory drugs. In all cases, the nature of the disease being treated is of most importance. Medications pertinent to diving are those that impair physical capacity or mental judgment. Synergy of sedative drugs with nitrogen narcosis should be considered, and medications that are known to prolong the Q-T interval of the electrocardiogram are generally not recommended in situations that require work, exercise, or water immersion[36] because of the risk of arrhythmias. Orthostasis due to antihypertensive drugs is avoidable via proper medication management. Some sport divers are diving safely while taking warfarin. This medication is used for anticoagulation in patients with mechanical

heart valves, atrial fibrillation, intracardiac thrombus, and thrombophlebitis. A sport diver using warfarin should be warned about excess bleeding from ear and sinus squeeze. Bleeding from any type of injury is likely to be more severe in a person using warfarin. Table 27–2 summarizes commonly used drugs and their relation to diving.

SKIN

Continuous wetting of the skin can aggravate many skin disorders. Acute or chronic dermatitis that would be made worse by repeated wetting requires special attention. Allergic reactions to chemicals in wet suits or face masks can usually be managed via use of nonallergenic materials. Because diving often takes place in hot, sunny environments, sun-sensitive persons, including those taking photosensitizing medications, must be cautioned. All divers should be cognizant of the serious sunburn possible from prolonged exposure to the sun while on dive boats or other recreation sites.

OBSTETRICS AND GYNECOLOGY

Pregnancy

Chapter 19 provides details on pregnancy. To provide optimal health for the fetus, women who are pregnant should be advised against diving. Pregnancy should not be terminated for a woman who dives during pregnancy, but prenatal ultrasonography may be indicated to rule out fetal malformations.

Table 27–2. Commonly Used Medications and Interactions with Diving

Indication	Category of Drug	Typical Example	Diving Relation
Allergy	Antihistamine	Diphenhydramine	May cause sedation
		Loratadine	None
Anticoagulation	Anticoagulant	Warfarin	Contraindicated in commercial diving
Anxiety	Tranquilizer	Alprazolam	Anxiolytics may aggravate narcosis
		Diazapam	
		Fluoxetine	
Congestion	Decongestant	Pseudoephedrine	None
Fluid retention	Diuretics	Hydrochlorthiazide	Diuretics aggravate dehydration
		Furosamide	
		Triampterene	
Gastric upset	Antacid	Magnesium/aluminum hydroxide	None
	H_2 blocker	Cimetadine	None
Heart problems	Antiarrhythmics	Procainamide	None
		Mexilitine	None
		Amiodarone	Skin sensitivity — UV light
		Digoxin	None
		Verapamil	Gastric reflux
Hyperlipidemia	Statins	Atorvastatin	Muscle pain may be mistaken for decompression sickness
		Simvastatin	
Hypertension	Calcium blocker	Diltiazem	Gastric reflux
	Beta blocker	Atenolol	Cold intolerance
	Angiotensin-converting enzyme inhibitor	Enalapril	None
	Angiotensin receptor blocker	Losartan	None
Infection	Antibiotics	Penicillin	None
		Tetracycline	Skin sensitization — UV light
		Trimethaprim sulfa	None
		Erythromycin	None
		Ciprofloxacin	Skin sensitization — UV light
Motion sickness	Antimotion sickness	Scopolamine patch	Blurred vision
		Dimenhydrinate	Drowsiness
Musculoskeletal pain	Analgesic	Aspirin	None
		Ibuprofen	None
		Acetaminophen	None
Skin rashes	Steroids	Hydrocortisone	May aggravate oxygen toxicity

Menstruation

Effects of menstruation on decompression risk are uncertain. An abstract published by Dunford and Hampson[46] indicated that female hyperbaric chamber attendants were more susceptible to DCS when menstruating. They found 18 cases of DCS in 5222 exposures in women chamber attendants. Menstrual history was available in 9 of the 18, and 5 of the 9 were menstruating when they experienced DCS. Based on average timing of the menstrual cycle (4 days in 28), Dunford and Hampson[46] assumed that 1 in 7 women with DCS should have been menstruating. Comparing their chamber data to the calculated frequency of menses, these authors showed a significant increase in DCS during menses. No control subjects were included in the study, nor were the exposure histories of the women with DCS incorporated in the analysis.

Rudge[47] examined altitude chamber data from the U. S. Air Force School of Aerospace Medicine. Over the period 1978 to 1988, 81 female altitude DCS cases showed adequate menstrual histories. In this cohort, women were at higher risk for altitude-related DCS during menses, and risk decreased as the time since the last menstrual period increased. Female patients for whom a menstrual history was unavailable were not analyzed. Shirmer and Workman[48] obtained detailed menstrual and contraceptive histories from 508 altitude-training exposures in U. S. Air Force female flight trainees from 13 training sites. The authors found a uniform distribution of menses throughout the exposures and no cases of DCS. Dixon and colleagues[49] studied 30 women during 6-hour hypobaric exposure to 7.8 psia, breathing 50% oxygen in nitrogen while performing light exercise. Each subject was exposed daily for 3 days. Five subjects were considered to have DCS, and three received hyperbaric therapy. Forty-three percent of subjects had ultrasound-detected bubbles at least once during the 3-day study. Dixon and associates[49] compared their data to data on male flight crew exposed to the same protocol. Of interest is the finding that 73% of males had bubbles at least once but DCS was diagnosed in only one subject. In the study by Dixon and coworkers, all 5 women with DCS were menstruating at the time of the exposure, whereas 32% of the trainees without DCS were menstruating. The available data are inconclusive regarding the risk of DCS from diving or altitude exposure during menstruation.

RISK OF DECOMPRESSION SICKNESS IN WOMEN

Several studies suggest that women may be more susceptible to DCS than men. Bassett[50] reported that female flight crew trainees were more susceptible to DCS than their male counterparts exposed to the same hypobaric stress. Bassett's data, however, were confined to hypobaric exposure at one training site and were not supported by overall U. S. Air Force experience from other altitude training sites. A follow-up study by Bassett[51] showed results similar to the 1972 study. Operational exposure in the Air Force does not demonstrate an increased DCS risk for women exposed to altitude,[52] and women do not appear to have a higher incidence of DCS in simulated space flight exposures.[53] Zwingelburg and coworkers[54] reviewed the logs of 878 dives performed at the Navy Diving and Salvage Training Center and found no increase in DCS incidence among female Navy divers. The authors examined dives made by male-female diving pairs with similar exposures and showed no difference in incidence of DCS in females. With the exception of the experience of the U. S. Air Force School of Aerospace Medicine, no studies have shown an increased female susceptibility to DCS in diving or in altitude exposure.

PSYCHIATRY

The prospective diver who is comfortable in water is preferable for any diving program. Panic-prone persons and those with claustrophobia should be excluded. The examiner should seek evidence of past anxiety attacks, hyperventilation episodes, or fainting.

Psychotic Disorders

An actively psychotic patient is unlikely to apply for diving training, but affective psychosis may be difficult to detect. The medical history should include questions about psychiatric disorders. Inquiry should

seek the type of medications the candidate has taken as well as past psychiatric care. Because of the relapsing and recurring nature of psychoses, approval of such patients for sport diving during a period of remission may grant a permanent dive certification and may endanger a diving partner at some later date. Input from the patient's psychiatrist is helpful in establishing the prognosis for safe diving. As a general rule, patients requiring psychotropic medications should be disqualified, although sport divers have been diving while using serotonin reuptake inhibitors with no apparent problems.

Alcohol and Drugs

Many applicants and active divers use alcohol or other drugs in a limited recreational or social setting. When applicants are addicts or heavy users, there is no problem in the decision to exclude them from diving. In this population, chronic effects on mental function and organ damage would prohibit diving. In recreational or social use, the medical decision must rest on ability and willingness to control behavior. The recreational diver must abstain from drugs or alcohol during a day of diving.

Detection of chronic substance abuse includes drug screening of blood and urine, laboratory studies to determine liver function, evidence on physical examination of needle tracks over superficial veins, hepatic enlargement, jaundice, ascites, and muscle wasting. Cocaine effects include tachycardia, hypertension, ischemic chest pain, and a state of agitation.[55] Cocaine may cause acute myocardial infarction and acute pulmonary injury. Chronic cocaine users may have medical disorders (heart or lung disease) that are contraindications to diving.

DIVING FOR THE PHYSICALLY HANDICAPPED

Physically handicapped patients have been diving safely for many years. Limitations involve reduced mobility in diving environments, particularly on boats and along rough shorelines. A physically handicapped sport diver however can make choices of the diving environment that will facilitate diving. A trained diving partner who is willing to provide support when needed is an important asset for a physically handicapped diver.

HEALTH REGULATIONS FOR DIVERS

In most countries, sport diving is either minimally regulated or not regulated. In the United States, OSHA standards provide a list of contraindications to hyperbaric exposures (Table 27–3). For the physician

Table 27–3. Disorders that May Restrict or Limit Occupational Exposure to Hyperbaric Conditions Based on OSHA Standards

History of seizure disorder other than early febrile convulsions
Malignancies (active) unless treated and without recurrence for 5 yr
Chronic inability to equalize sinus and/or middle ear pressure
Cystic or cavitary disease of the lungs
Impaired organ function caused by alcohol or drug use
Conditions requiring continuous medication for control (e.g., antihistamines, steroids, barbiturates, mood altering drugs, or insulin)
Meniere's disease
Hemoglobinopathies
Obstructive or restrictive lung disease
Vestibular end-organ destruction
Pneumothorax
Cardiac abnormalities (e.g., pathologic heart block, valvular disease, intraventricular conduction defects other than isolated right bundle branch block, angina pectoris, arrhythmia, coronary artery disease)
Juxta-articular osteonecrosis

From Occupational and Health Administration: Examples of conditions which may restrict or limit exposure to hyperbaric conditions. Standard No. 1910, Subpart T, Appendix A. Washington, D. C., OSHA, 2000.

Table 27–4. Countries Requiring Medical Examinations for Sport Diving

Country	Diving Exam Required
Australia	Yes
Belgium	No
Canada	No
Denmark	No
France	Yes
Germany	No
Greece	No
Italy	No
Japan	No
Netherlands	No
Norway	No
Spain	No
Sweden	No
Switzerland	No
Turkey	No
United Kingdom	Yes
United States	No

working with divers in other countries, it is necessary to first understand the medical practice and medical licensing regulations, then inquire of the proper authority about current regulations for health and safety for sport divers. Table 27–4 provides a summary of countries that regulate sport diving.

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CHAPTER 28 Medical Evaluation of Working Divers

David H. Elliott

Working divers must be medically, mentally, and physically fit to perform their varied tasks in safety, yet it is not a simple matter to define the criteria of fitness and to pinpoint the threshold of any abnormality that should be disqualifying. Divers should undergo periodic examination to ensure that they maintain an adequate level of fitness; this is generally accepted by most categories of working divers. Divers are employed in a wide range of activities, and all share the unique hazards of their working environment. The commercial diving industry includes offshore, inshore, and inland divers trained in scuba, hose-supplied, and bell diving; divers undertake many tasks in the construction, inspection, maintenance, and repair of anything from deep sea structures to mountain dams. The working diver population, i.e., those who are paid to dive, also embraces divers who gather shellfish or coastal diamonds; marine biologists, geologists, and archeologists; police, fire department, and rescue divers; and, not least, professional guides and instructors for recreational divers. Military divers are also paid to dive but often do so in unusual conditions and with special equipment, requiring them to meet fitness specifications that are more demanding than ones used for commercial diving. For this reason, this discussion specifically excludes military divers (see Chapter 29 for military diving considerations), though much of it may be relevant to such persons.

Advice has been published on the physical examination of working divers.[1–4] Some of it is in the prescriptive form of lists of *absolute disqualifications, relative disqualifications,* and *temporary disqualifications.* Some advice is more comprehensive, but even the most meticulous fitness standard requires a wise interpretation by the examining physician, who must apply these standards to the wide variety of conditions found in a population of normal divers. A restricted certificate of fitness, perhaps during recovery from some illness or trauma, must also be based on a thorough understanding of the demands of an individual's specific diving duties.

The assessment needed for this unique workplace is best performed by medical examiners who are trained and experienced in diving and diving medicine. Training courses and continuing medical education can improve medical assessment such that the written standards used by examiners can become less prescriptive and more adjustable to the specific working conditions of the individual diver.

One must define the objectives of the medical examination of working divers before considering specific conditions that may lead to medical disqualification.

IS MEDICAL REVIEW NECESSARY?

The primary objective of the medical examination of a diver is to maximize his or her safety underwater. Examination can reduce the risk of fatality or serious injury due to some preexisting medical condition. Commercial diving is a team activity, and the diver who becomes ill underwater can jeopardize the safety of others. The success of medical screening may be judged by the anecdotal rarity of a medical condition being reported as a contributory factor in professional diving accidents.

Medical standards for in-water diver safety should also consider the diver's tasks in the bell or at the surface. These tasks include the ability to tend another diver with a hose and to respond to aural and visual stimuli.

The effect of increased environmental pressure is identical for all divers, but this does not mean that the same screening examination is valid for all. There are accepted differences between the standards for new-entry trainees and those for established and experienced divers. There are also differences in medical screening between different categories of divers—for instance, between the diver of a search-and-rescue helicopter team and a welder working in a habitat at a depth of around 1000 feet (300 m). Also, in terms of criteria that do not relate to immediate in-water safety, it is reasonable to have more stringent standards for saturation diving than for the other categories of diving. This higher standard pertains to disorders such as a healed peptic ulcer, which is not incompatible with in-water safety but may require access to treatment at short notice. In saturation diving, the patient can be isolated from treatment at normal atmospheric pressure by the need for a decompression that extends over several days. Such persons can be excluded from saturation diving while considered fit for other types of diving.

A secondary objective of the medical examination is the protection of divers from the adverse effects of diving on their future health by the early detection of presymptomatic occupational bone necrosis. As described in Chapter 21, however, this objective is difficult to meet.

A third objective of the medical examination of divers relates to the protection of the employer against future court action. Although such an objective may appear ethically dubious, the fact is that in many countries the employer has a right to determine the standards of fitness for employment. For example, at a pre-employment examination, a potential new employer need not accept a diver in whom radiography suggests existing osteonecrosis. This is an understandable policy but an example of incomplete logic: normal-appearing bone radiographs may be falsely negative during the lengthy latency between a causative exposure and the first detectable radiographic change.

An employer also can legitimately define additional vocational requirements such as eyesight standards for divers required to handle boats at sea. These standards do not relate to underwater safety and so need not be discussed further.

DEVELOPMENT OF THE MEDICAL REVIEW

Medical Standards

For many navies, the ideal selection process was to accept only "perfect specimens" from an already healthy group of young men. Any medical officer using a brief list of standards could conduct such an examination, which the candidate simply either passed or failed. The effects of aging were not a problem because most of the divers completed their naval service within a few years; those who stayed moved into more senior jobs that required little or no diving.

Early medical standards were based on a mix of theoretical considerations and practical experience. The method was successful for many years but, by setting standards that may have been unnecessarily high, navies probably eliminated many candidates who could have dived in safety. This was not important as long as there were plenty of new volunteers. Recreational divers have since demonstrated that safe diving can be compatible with some medical conditions that would have excluded them from joining the navy, let alone becoming a naval diver.

Working divers fall between these two extremes. Neither the rigidity of naval standards nor the relaxed approach of amateur assessment is appropriate for a person whose diving may become a regular activity until final retirement. In 1981, the Health and Safety Executive in the United Kingdom became the first government agency to issue detailed guidance on the medical examination of commercial divers. There was little scientific evidence to validate any medical safety standards because for many conditions, controlled underwater trials would be unethical. These fitness standards were first based on naval practice but now contain much more detail[4] and are subject to regular revision.

Similar prescribed standards now exist in many other countries. For an internationally mobile work force, harmonization of standards (or the mutual recognition of different national standards) is important. Standards of assessment are, at the time of printing, due to be ratified by the European Diving Technology Committee (EDTC) and recom-

mended for adoption by its 15 member nations. These standards can be found at www.edtc.org.

Limitations of Standards

An international review of the medical assessment of divers concluded that there is a limit to what can be achieved by the application of written standards.[5] Each medical examination for fitness to dive needs to be much more than a mere application of predetermined pass/fail criteria. If a diver has some disease or medical imperfection, the examining doctor must interpret the relevant standards in relation to the requirements and hazards of the diver's working environment and the need for underwater safety. No list of pass/fail criteria can ever provide complete guidance. An examining doctor needs not only the functional specifications of the diver's job but, as has become increasingly apparent, an understanding of the hazards and demands of each type of diving activity.

In many countries, only doctors who can demonstrate that they have acquired appropriate knowledge of diving can undertake the medical examination of divers. This is a good beginning but, until sufficient training and experience are available for all medical examiners, some basic guidance must still be provided. Prescribed guidance now tends to use standards that are somewhat more loosely written than the earlier absolute or relative restrictions. These new standards require intelligent interpretation to achieve their primary objective, the promotion of underwater safety.

The medical examiner of working divers should have attended at least an introductory course to learn about the effects of the environment on a diver's health and safety. The course needs to cover the physiologic aspects of diving, work procedures, types of personal equipment, and emergency procedures as well as the recognition and initial management of the occupational illnesses of divers. A working group from the EDTC and the European Committee for Hyperbaric Medicine has defined training objectives for diving doctors.[6] The EDTC's national representatives have since agreed on these objectives; as stated earlier, these objectives are in the process of adoption by member nations. In the United States, similar standards are promulgated by the Occupational Safety and Health Administration.[7] Periodic revision by medical examiners and audits of performance are also recommended.

HEALTH SURVEILLANCE

The purpose of health surveillance is to monitor the possible effects of diving on the diver. This is quite distinct from the medical assessment of diving safety. Whereas the medical objectives for safety remain the same for divers regardless of age, task, or depth or duration of dive, the requirements for health surveillance differ among divers because these are determined largely by exposure history.[8] The need to monitor selected divers for presymptomatic dysbaric osteonecrosis is well established (see Chapter 21), but for accurate diagnosis some pre-exposure radiographs need to be kept on file for life. No other baseline investigations are currently recommended. For many categories of working diver, there are no recognized diving illnesses for which regular health surveillance is appropriate, although divers may need to be monitored for the consequences of other environmental hazards such as noise.

FITNESS ASSESSMENT

Age

In many countries, employment legislation makes it unlikely that trainee commercial divers younger than 18 would present for a medical examination, but of course many persons younger than 18 do dive successfully. Concerns have been expressed about the immaturity of bone in young divers, but osteonecrosis shows no preference for the epiphyseal lines.

Divers age not only as individuals but as members of an aging population. In the North Sea, the average age of divers increased by 3.5 years over a 6-year period. This is partly because client companies are demanding that only experienced divers undertake their work. The technical requirements of underwater tasks are becoming increasingly more complex, and the diver may need 10 years of

experience in the industry to achieve the necessary competencies.

Because there is a marked difference between chronologic and biologic age, it seems unreasonable to define any statutory upper age limit for divers provided that they remain medically, mentally, and physically fit to continue.

The difficulties begin when one tries to account for age and the experience that comes with it in arriving at a standard of physical fitness. The ability to undertake physical work declines considerably between the ages of 18 and 65, and maintaining the necessary physical fitness becomes more difficult. For example, any diver may need to call on all reserves of effort in a life-threatening emergency. The required duration for that near-maximal effort is unpredictable, which makes it difficult to specify a required level of physical fitness. Functional goals should be independent of age and gender but should allow for the fact that the experience of older divers may make them more effective in some circumstances than fitter, younger divers.[9]

The significance of chronologic age also needs to be considered in relation to each body system. The concept of a routine annual medical examination that uses a prescribed pass/fail list must be replaced by that of functional assessment. The frequency with which the various components of the assessment should be tested may vary. For example, lung function, blood pressure, and audiometry are three parameters that would be assessed at an increasing frequency with increasing age. Indications for cardiovascular testing are discussed in Chapter 25.

Systematic History and Examination

Conventionally, the medical examiner first takes the diver's medical history, possibly using a checklist to ensure that the diving aspects of the history are complete. In some countries, the diver completes a questionnaire before seeing the doctor in order to achieve the same intended result. However, self-completed histories present the problem of a written question meaning something different to the person filling it in from the intended meaning of the person who wrote it.[10] Questionnaires need to be validated before they are used for acquiring medical information. It is also important that the examining doctor goes through the answers in the presence of the diver and before the diver signs it as being a true record. This declaration is witnessed by the doctor and is important because some working divers may be tempted to conceal symptoms or past diving incidents that might lead to medical disqualification. Such an action would be potentially dangerous for the diver and for others in the water.

The yield from the physical examination that follows may be low among young healthy divers but the repeated examination, year after year, is still regarded as an important factor in maintaining the medical safety of the aging diver. Physician compliance with required examination and supplementary tests is not always perfect; among nearly 500 working divers, an audit revealed the omission of a large number of significant and potentially disqualifying conditions.[11] Audits of medical performance should be broadened and can begin with simple checks such as the calibration and interpretation of pulmonary function testing equipment.

For each of the systems now to be reviewed briefly for commercial diving, guidelines differ somewhat from the standards for military diving and the more relaxed approach of recreational diving. Fitness standards for young candidates for commercial diving tend to be more stringent than military requirements in some ways because these divers are planning a career for which they may expect to remain fit to dive for some 40 years. The annual reexamination of established working divers is certainly more restrictive than for recreational divers, especially for conditions that may compromise in-water safety. Some countries require that professional instructors of recreational divers adhere to the same standards as other working divers. This is considered important for the novices whose underwater safety is the instructor's responsibility. This principle has not yet been acknowledged in all parts of the world.

The following clinical section is based on published guidance[4] and on the proceedings of the Edinburgh conference,[5] which present a fuller account and other references. Some of the following statements may seem obvious and are included for a reader unfamiliar with professional diving. The specific clinical requirements for rating a person as fit to dive cannot be detailed here because

these still differ widely among national authorities.

Respiratory System

The primary purpose of lung assessment is to exclude, as far as is practical, the presence of any respiratory condition that would be a hazard to underwater safety. Two hazards dominate this part of the assessment. One is insufficient respiratory capacity to maintain hard physical exercise in the water. The other is the failure of the lungs to vent all the expanding gases during decompression.

Most of the existing pulmonary guidelines seem logical, and there is no evidence that any condition that may compromise safety is being overlooked. Indeed, the guidelines are probably too restrictive and may be excluding potential divers who would be fit to dive. The respiratory conditions that preclude diving are generally listed as any acute or chronic respiratory infection; a history of spontaneous pneumothorax; the presence of lung cysts, blebs, or bullae; chronic bronchitis; emphysema; pleural effusion; lung fistula; bronchiectasis; fibrosis; and neoplasm. Conditions requiring individual assessment but that may preclude diving include a history of pneumothorax that was not spontaneous but was provoked by unusual respiratory stress or following surgery, with at least 3 months having elapsed since the rupture for healing to occur. In these circumstances, detailed investigation by a specialist laboratory must show normal lung function and no evidence of local or generalized airflow obstruction.

A history of asthma or bronchoconstriction after early childhood remains controversial. Contrary to theoretical predictions, there appears to be no firm evidence that asthma predisposes to pulmonary barotrauma and gas embolism. Stable asthmatics whose asthma is not triggered by provocation testing (histamine or methacholine) could be considered fit provided they demonstrate a less than 20% reduction of peak flow or forced expired volume in 1 second (FEV_1) after a few minutes of hard exercise. Indeed, it has been stated[12] that the use of inhaled steroids to maintain stability in a person with good peak flow is not per se an absolute contraindication to diving. The Americans with Disabilities Act and similar legislation in other countries now suggest that some asthmatics must be allowed to dive professionally. A meticulous collection and analysis of relevant data is needed to confirm the wisdom of this policy because firm data show that this practice would be safe or unsafe.

Many authorities still require full-sized postero-anterior chest radiographs at full inspiration, at least at the initial medical examination of a candidate before the start of diver training. Also, to aid detection of lucencies, some agencies have recommended that a second film be taken at full expiration, but there is some evidence that the bullae detected can be benign. After the initial examination, chest radiography is required only at the discretion of the examining doctor. Although spiral computed tomography of the lungs is considered a superior investigation, its limited availability is likely to preclude it from routine use in screening.

Because of the low yield of abnormal findings among diving candidates and the need to minimize lifelong x-ray exposure, radiographs are likely to be phased out from routine screening in the next few years.

The forced vital capacity (FVC) and the FEV_1 should be recorded, but the use of the FEV_1/FVC ratio to provide a numeric threshold for pass/fail decisions is not appropriate. Nevertheless, this ratio remains an important indicator for specialist referral. The shape of the maximal flow volume loop is another important consideration. From a series of annual examinations, the examining doctor must look for individual trends, particularly those that are still within the range of normal for the general population but that are revealed as significant when compared with that individual's previous records.

A secondary purpose is to review the effects of diving on pulmonary function, but only some divers may need pulmonary health surveillance. The effects of diving on the lungs have been shown to include increased total lung capacity, reduced small-airway conductance,[13] and reduced gas transfer capacity,[14] but this would probably be detected only if referral to a pulmonary specialist were indicated.

Cardiovascular System

There is consensus that any organic heart disease should be grounds for rejection of diving clearance unless a cardiologist considers the disease to be hemodynamically

unimportant. Similarly, any rhythm disturbance that might cause in-water incapacity should be assessed and would probably disqualify a diver. Except for sinus arrhythmia and ventricular extrasystoles, these rhythm disturbances should be referred to a cardiologist and are usually grounds for rejection. Diving candidates with electrocardiogram (ECG) abnormalities should be referred to a cardiologist. Such abnormalities are usually a cause for rejection unless they are demonstrably benign, such as an isolated right bundle branch block. The variants of the Wolff-Parkinson-White syndrome are such that general rules cannot be applied: Some are benign, others can be ablated, but a few are severe and a cause for rejection.

Any evidence of coronary insufficiency or myocardial ischemia, whether clinical or electrocardiographic, is a cause for rejection, but the occurrence of false-positive exercise test results in young fit people needs to be recognized. Coronary artery bypass surgery does not render a person fit to work as a diver, although percutaneous transluminal coronary angioplasty, if it produces revascularization, may not be a contraindication.

The resting blood pressure at the initial examination should not exceed 140 mm Hg systolic or 90 mm Hg diastolic. The EDTC consensus standards have proposed a cutoff of 140/80 mm Hg at the initial examination of a candidate for diving, but a cardiologist's opinion on individual prognosis is more valuable than a numeric threshold.

No published data show the ECG to be of value in the evaluation of diver fitness, and most authorities have discontinued the annual resting ECG. Cardiomegaly should be assessed fully. It is usually be a cause for rejection except in those divers with "athletic hearts" that are confirmed by a cardiologist.

Screening all divers by echocardiography for a right-to-left shunt because of its association with the risk of a decompression injury is not appropriate. The presence of a patent foramen ovale is a natural variant found in approximately one third of all divers. The majority of them have never had decompression sickness or arterial gas embolism and never will. Echocardiography should be considered only after one or more "undeserved" episodes of neurologic decompression sickness, especially if it was associated with cutaneous manifestations.[15]

Exercise Testing

It has been suggested that an exercise tolerance be carried out at each annual examination. An Army physical fitness test is an example of a test in common use. The diver has to step at a rate of 30 times per minute with both feet to a height of 43 cm for 5 min, after which a sum of pulse counts is made by adding the 30 sec pulse counts taken at 1, 2, and 3 min post exercise. The total should be less than 190. The results of different simple tests of this type are unreliable to the extent that a subject might fail one of them but then pass another. These tests should be used for gross guidance only and perhaps retained to encourage divers to keep themselves fit.

A bicycle or treadmill ergometer is of greater value. The heart rate can be monitored against the exercise load and can provide an indirect measure of physical fitness. If the rate of oxygen uptake can also be monitored, aerobic capacity can be assessed more directly and accurately. The direct methods of measuring oxygen consumption are possibly too complex or costly to be considered as an annual routine. That the treadmill test gives results 10% greater than those from bicycle ergometry is considered relatively unimportant. Exercise at up to 13 mets (about 40 mL/kg/min O_2 consumption) seems to be generally considered a useful measure of individual fitness that should be assessed at least every 3 years.

There is merit in considering a more functional test, such as the time taken to swim 1 km, as an annual measure of continuing fitness; in practice, however, this would not be easy for an examining doctor to verify personally. There is also considerable difficulty in interpreting the results from exercise tests in practical terms because, in a given situation, an older but more skilled diver may need to use less effort than a younger, fitter person.

Obesity is reputed to predispose to decompression sickness but is certainly inversely related to fitness. Body mass index (BMI) is weight (kg) ÷ height $(m)^2$ and is a convenient though relatively arbitrary way to measure obesity. Some degree of fatness can protect against the cold, but exercise tolerance testing seems to be more appropriate as an indicator of fitness. Details of cardiovascular assessment can be found in Chapter 25.

Ear, Nose, and Throat

The tympanic membranes should be examined annually because a person can perforate an eardrum and be unaware of it. Ninety-five percent of traumatic perforations heal spontaneously; for the remainder, the drum can be repaired. Once healed, the injury does not prevent someone from diving. However, the presence of attic or posterior marginal perforations of the drum indicates middle ear disease and contraindicates diving. Complications of otitis media such as glue ear, deafness, perforation, and persistent discharge are causes for rejection. Atticotomy or radical mastoidectomy also disqualifies a patient, but a simple mastoidectomy does not. Chronic otitis media contraindicates diving. Stapes surgery is a contraindication to diving because subsequent barotrauma may drive the prothesis inward, causing total deafness. Meniere's disease is incompatible with diving. After successful repair of a round window rupture, the diver may resume diving provided there is no difficulty in clearing the ears.[16] Nasal septal deformity may result in difficulties in eustachian tube function. Many cases have been rectified easily by a simple operation.[17]

The diver must be able to hear and understand normal conversation. Pre-employment audiometry is advised. Subsequently, audiometric screening need be carried out only as part of a hearing protection program in which hazardous noise levels have been identified. Although this may be common in some types of work, many divers are not exposed to this risk. Benton[18] reported no greater loss of hearing threshold associated with diving, except in the few individuals with evidence of significant barotrauma.

Eyes

Published guidelines state that corrected vision should be 6/9 or better (binocular), and yet many divers work in zero-visibility water. The diver should be able to see well enough to accomplish the necessary functions, and a person who can read a car license plate at 25 m (80 feet) probably has adequate vision for diving. The diver should also be able to read diving tables, gauges, and watches accurately, so corrected vision should be adequate for reading. To accomplish this, modern gas-permeable contact lenses are acceptable for diving. An experienced diver who loses one eye may sometimes be able to return to work.

Color vision should be assessed at the initial medical examination and any deficiency communicated to the diver. Even though color vision is not essential for diving safety, it may be a vocational requirement in some diving tasks such as nondestructive testing. Visual acuity and color vision may be essential for other tasks such as boat navigation, but these tasks are outside the scope of the diving assessment.

The orb is incompressible, so there should be no reason why corneal laser surgery or any other surgery on the eye should be a contraindication to diving once any gas pocket retained in the eye after surgery has been reabsorbed.

Endocrine System

A gross and possibly unstable endocrine disorder may be an obvious contraindication to diving, but early or marginal cases may be difficult to diagnose. After treatment, some conditions either become stable or are completely eliminated and therefore are no longer a barrier to diving.[19]

Gross thyroid disease is one obvious contraindication to diving, but after replacement therapy, hypothyroidism can be compatible with professional diving even when a dose or two of thyroxine is missed. Similarly, although thyrotoxicosis may bar a person from diving, after treatment with radioiodine and replacement therapy, the candidate's fitness should be considered favorably, although persons with retrosternal goiter should be assessed with care.

Hypopituitarism after replacement therapy might be thought to follow the same principle as hypothyroidism, but when injured or stressed, divers on cortisol replacement therapy may collapse, a certain contraindication to diving. Therefore, each individual with an endocrinologic disorder must be assessed for fitness jointly by an endocrinologist and a diving medical specialist.

If diabetes is found at the time of initial assessment before diver training, the candidate should not become a professional diver because of the likelihood of later disqualification owing to complications. He or she

should be advised to train for some other job. Once diabetes is diagnosed in a commercial diver, an automatic disqualification may seem wise but is not always an acceptable option. The two determining factors are the nature of the work and the degree of control achieved by treatment. What is appropriate for a diving scientist working in a laboratory wave tank may not be appropriate for a construction diver at sea. It should be noted that persons on oral medication who become hypoglycemic may be more difficult to treat than those who become hypoglycemic on insulin. Continuing advances in the management of diabetes suggest that policies on diabetic divers need regular review and update. In addition to the acute problems of diabetics, the longer-term complications are a matter of concern. The following should disqualify a diver: atherosclerosis, cardiomyopathy, retinal changes, peripheral vascular disease, diabetic foot syndrome, nephropathy, and neuropathy. Diabetes is discussed further in Chapter 26.

Musculoskeletal System

For a new diving candidate, the musculoskeletal standards are much higher than those in somebody who has 10 years' experience and has a good work record; in such a person, the standards can become much more pragmatic.

To become a commercial diver, the candidate must have unimpeded mobility and dexterity and must be physically robust enough to meet the demands of the proposed work. In particular, for personal safety and the safety of others, all joints should have a normal range of functional mobility in relation to work tasks and emergency procedures. Nevertheless, a number of experienced divers can be assessed as fit to return to work after a minor amputation.

Divers with a history of back pain should be carefully assessed because a diver may be required to do heavy lifting. Recurrent episodes of incapacitating back pain can be a cause for medical disqualification. A candidate who has had successful spinal surgery is acceptable if the neurologic examination results are normal and full agility is regained.

Dysbaric osteonecrosis is the one established occupational health hazard of diving for which health surveillance is recommended (see Chapter 21). The use of magnetic resonance imaging as a screening tool may become more commonplace subject to cost and availability, but until then the traditional special radiographs remain important for epidemiologic studies and for medicolegal prognosis. Until magnetic resonance imaging becomes routinely available, specific radiographs of the hips and shoulders need to be taken before the first significant exposure and retained as a baseline diagnostic reference for the diver's lifetime. Baseline imaging and subsequent screening is not needed for such persons as police divers whose dive profiles are considered not to put them at special risk.

Hematology

The number of available biochemical, immunologic, and endocrinologic tests is increasing, and vast quantities of data can be derived from a small sample of blood. Caution must be exercised when applying the results of these tests to apparently healthy persons unless the conclusions that need to be drawn from a positive test result have been clearly defined.

At the initial medical examination, a complete blood count should be performed. A hematocrit of 40% and a hemoglobin level of 12 g/dL in men and 10.5 g/dL in women are the minimum acceptable levels. The presence of the sickle cell trait is not a cause for rejection, but testing for thalassemia minor may be judged appropriate in candidates from the Mediterranean area.

Gastrointestinal System

Candidates' teeth and gums should be healthy. Dentures should be removed while diving to prevent possible inhalation, but partial dentures can be worn if they are secured to the remaining teeth.

Dyspepsia requires investigation, and the association of reflux esophagitis with a predisposition for duodenal ulceration could compromise in-water safety. Symptomatic hiatus hernia and active peptic ulceration disqualifies persons from diving until they are symptom-free and without treatment for at least 1 year. A past history of peptic ulceration leading to bleeding or perforation or requiring emergency surgical treatment

may also disqualify a candidate. One should consider the difficulties of a surgical emergency arising while a saturation diver is isolated from sea-level care by some days of decompression.

Recurring episodes of abdominal pain should be investigated and may be disqualifying because of the likelihood of confusing the diagnosis with the abdominal pain of "spinal" decompression sickness. Chronic inflammatory intestinal disease is cause for rejection. Acute distal colitis or proctitis should await the outcome of investigation and treatment. The presence of an intestinal stoma does not affect safety and should not disqualify a candidate from short-duration diving. In saturation diving, the problem may be one of social acceptance, but disqualification on medical grounds is not appropriate. Symptomatic hemorrhoids should lead to referral for surgical treatment and should contraindicate diving only until they have been successfully treated.

Abdominal wall herniation must be a cause for temporary disqualification until it is repaired because of the risk of encapsulation, particularly during decompression.

Evidence of acute or chronic hepatic disease renders a diver unfit. Once a diver with viral hepatitis is over the initial illness and is no longer antigen-positive, a return to diving is permissible. Regardless of known hepatic disease, all divers must be trained to have immaculate personal and environmental hygiene at all times, particularly within the close confines of a diving chamber.

Genitourinary System

A history of renal disease or of urinary tract infection needs careful assessment. Those with active genitourinary infections, including herpes, should not dive until they have been adequately treated and are symptom-free. A patient with recurrent herpes infection might be advised against saturation diving.

The presence of renal stones and other genitourinary diseases is usually a cause for rejection. However, renal stones may be asymptomatic, and some divers have returned to restricted surface-orientated diving without problem. A commercial diver in a saturation dive treated for renal colic subsequently underwent decompression and was referred for further investigation.[20] However, a case of renal colic occurring after a dive or saturation excursion does pose a diagnostic dilemma.

Skin

The skin is very vulnerable to the repeated and sometimes constant wetness experienced by divers and to the high humidity and reduced temperature tolerance experienced in the closed environment of saturation chambers.

In relation to diving, skin diseases may be divided into those in which the integument is compromised (e.g., eczema, psoriasis, pityriasis rosea, lichen planus) and those in which there is some disorder of thermoregulation.[21] Some diseases such as eczema can fall within either category. The disorders that can affect thermoregulation can be divided into those in which there is a hemodynamically based disturbance (severe eczema or psoriasis, urticaria, mastocytosis) and those in which there is a disturbance of the sweat gland apparatus. The latter can be occlusive (psoriasis), destructive (scleroderma), or congenital (ichthyosis). Neurologic causes of impaired sweating also occur.

Urticaria is a transient condition, and provided that it affects only the skin and not the mucous membranes, it does not appear to be a contraindication.

Any acute or chronic skin infections, whether fungal, bacterial, parasitic, or viral, must be controlled before diving is allowed. Recurrent herpes simplex virus infection poses very little risk to others and no risk to the diver. Hand warts are not a contraindication to diving. Verrucae of the feet probably should be a reason for temporary unfitness, although on the general grounds of hygiene and not because of any diving hazard.

Occupationally acquired skin diseases need to be considered. Neoprene contains antioxidants, and the glues used for the nylon backing can contain allergens. Drilling muds are complex materials with many varied constituents but do not appear to constitute a major sensitization problem. The alkalis are primary irritants and can give rise to serious skin reaction. The oil-based muds are also irritants. Occasionally reactions to the tannins and to chromium have been reported. Persons identified as suffering from allergic contact dermatitis must avoid all future contact with that allergen, but the

majority of persons are found to have an irritant dermatitis that should respond in time to rest and can thereafter return to diving.

The so-called *diver's hand*[22] affects only nonpigmented skin with epidermal peeling on the palmar surface. It is noninflammatory and usually resolves in 2 to 3 weeks, although a few divers have been rendered permanently unfit.

Mental Fitness

Important but almost ignored in published guidance is the need for mental fitness, particularly in the interests of safety. Is the candidate a person with whom one would trust one's life underwater? Mental fitness for diving should be considered in three areas.[23] Psychiatric and psychological disorders that are unrelated to diving but are thought to influence the safety of the diver in the water constitute the first area to be considered. Then there are those psychiatric and psychological disorders related to or arising from diving. The third area is that of the need for research in diver selection and the assessment of diver fitness, but these are not yet ready to be presented as guidance.

At the initial examination, the primary concern is to detect disorders that might affect future diving safety. A statement for guidance that "any evidence of past or present psychiatric or psychological disorder (including alcohol or drug abuse) should be cause for rejection unless the examining doctor can be confident that it is of a minor nature and unlikely to recur" is of little practical use to the medical examiner.

Psychosis, such as schizophrenia and bipolar affective disorder, should be an absolute diving disqualification because both the diver and the other divers may be incorporated into the delusions. There is also a disruption of logical thought that impairs the diver's ability to cope with the underwater environment. Risks are therefore present for individual divers and for all those diving with them. Depression is of many types and requires accurate psychiatric diagnosis. An acute grief reaction, for example, is likely to have a good prognosis. Overarousal leads to a rapid deterioration in performance, and any anxiety disorder should disqualify because it may precipitate a panic attack. Phobias, particularly claustrophobia, may be exacerbated in diving and can lead to a decrement of performance and should be contraindications. Use of so-called recreational drugs can certainly affect safety performance, but detection of such use, which is likely to lead to disqualification, is not always easy.

There may be value in establishing baseline neuropsychological data for all commercial divers in order to be able to later screen those who have had some dive-related incident.

Neurologic Assessment

There are a few obvious contraindications to diving such as any unprovoked loss of consciousness, recurring fainting episodes, or epilepsy (other than febrile convulsions occurring up to the age of 5 years). In contrast, and less certain, is the traditional rejection of those who suffer migraines with visual, speech, motor, or sensory disturbances. These manifestations may cause diagnosable post-dive confusion but do not necessarily affect in-water safety.

Any history of an intracranial surgical procedure, depressed skull fracture, or penetrating head injury needs careful assessment because of the increased risk of subsequent epilepsy, especially in the subsequent 12 months. A history of head injury is acceptable if there was a loss of consciousness of less than 30 min without focal localizing signs and if the period of post-traumatic amnesia (defined as the duration until the restored memory becomes continuous) is less than 1 hour. Minor linear skull fractures are acceptable if these criteria are met. Other cases of mild to moderate head injury, especially if recurrent, require full neurologic and psychometric assessment.

Examination of the cranial nerves, motor system, sensation, reflexes, and coordination should be detailed, and a permanent record of the examination needs to be retained as a baseline and made available to others.

RESUMPTION OF DIVING AFTER UNFITNESS

The purpose of assessing fitness to return to diving after illness, surgery, or injury is primarily to determine any factors that may affect subsequent safety. Such assessment

follows conventional principles but with some additional considerations.

A hand injury, for example, may affect a bellman's ability to handle another diver's hose in an emergency. The physician must know the tasks that the diver is expected to perform and the hazards to be met after the diver's return to diving. If there is any doubt, the physician should seek advice from someone such as a diving supervisor.

A different consideration applies, for example, after an apparent recovery from a splenectomy following an accident. The body's natural defenses may have been compromised, and the long-term risk of overwhelming postsplenectomy infection needs to be assessed. Such persons may be fit to undertake bounce dives, but even after appropriate immunization and while permanently on antibiotics they should not risk the relative isolation of a saturation dive.

The examiner may impose medical restrictions on a person's diving, but these restrictions do not always have a logical basis in safety. Too often, maximum depth limit is restricted, but this is rarely meaningful. An example of a practical restriction is one prescribed for a working diver who became a paraplegic from a car accident. His particular work was as a diving scientist, and although not many other divers would find this economically viable, his restriction is that he can dive only in company of a dedicated buddy whose sole task is safety and potential rescue. The diver's own view that he has "less spinal cord to get hit than anyone else" was not a consideration but has been correct now for some 25 years. The value of that example is that it was a decision that could not have been made using pass/fail criteria but could only be made on the basis of his previous experience as a military and scientific diver, his excellent upper body strength, and the doctor's understanding of his work and diving procedures.

Decompression Injuries

Some divers in the past have continued to work in spite of having muscle wasting and other residua of neurologic decompression sickness, but for some years the general opinion has been to allow a resumption of diving only after all clinical signs have resolved. The assessment of fitness to return to diving after an episode of neurologic decompression sickness should be concerned with more than a simple confirmation that there are no clinical residua.

Each diver needs an assessment of the possible contributory factors. Was the incident deserved or undeserved? In some cases, an omission of an appropriate decompression procedure is a sufficient explanation for the illness, but many cases occur after adherence to accepted tables. It is not always possible to make a retrospective differential diagnosis between gas embolism and decompression sickness as being the underlying pathology in a case of illness following decompression, but in practice this distinction is not necessarily critical.

Before a person returns to diving, the nature of the decompression sickness and its response to treatment should also be considered. Concern has been expressed about the possibility of permanent scarring of the spinal cord, leading to a diminished reserve of neurons that might reduce the chance of recovery from some future spinal decompression sickness. This cannot be assessed quantitatively and is a theoretical and ethical issue. The examiner depends on clinical judgment. In the assessment of an adequate recovery, the seriousness of the original incident should be assessed. This includes the speed of onset, the extent of the manifestations, and the rapidity of a complete response to recompression. Sensory manifestations in the limbs that lasted for only minutes would seem in retrospect to be less significant than a motor deficit that required prolonged treatment. A serious case with residua that persist for days before there is a full recovery constitutes a significant injury. The decision by some diving physicians that a diver should be disqualified after two neurologic episodes has been applied to a few cases with no more than transient pins-and-needles that resolved as soon as recompression began. The implied threat of consequent unemployment could encourage dangerous nonreporting by divers at the time of their more serious symptoms.

The ultimate answer about what advice to give the diver after a neurologic incident needs to be based on good data. There must be accurate descriptive diagnoses, clear medical records, detailed clinical follow-up, and a recorded history of all subsequent occupational exposures. Such surveillance becomes even more important if the right of divers to return to work, even though they

may be "disabled" by minor neurologic residua, is tested in the courts.

Echocardiography for patent foramen ovale needs to be considered, particularly for those who have had undeserved neurologic manifestations after a relatively trivial dive or who have had more than one incident. The place for neuropsychometric assessment, brain scans, and evoked potentials in assessing the completeness of a return to normality is uncertain and also needs further study.

Impaired Consciousness

There are many causes of impaired consciousness underwater; after such an incident, the cause, as well as any sequelae, must be assessed. The cause may be factors such as hypoxia from the accidental supply of a hypoxic breathing gas or oxygen toxicity from using too high an oxygen mixture at depth. Once the diver has recovered from a near drowning, possibly with cerebral edema or some pulmonary complication, the only remaining assessment is that of future in-water safety. A return to diving is possible with all of these conditions. If unconsciousness is medical in origin or unexplained, then a safe return is improbable.

Post-Traumatic Stress Disorder

Post-traumatic stress disorder is a result of a traumatic event with a perception of potential danger to life and with an intense activation of the autonomic nervous system. The balance between coping and not coping is critical. Post-traumatic stress disorder with fear of the water can follow an in-water incident that has no physical sequelae. In contrast, another diver whose underwater incident was the traumatic amputation of an arm has striven to return to diving. Many subtle variants lie between these extremes. However, an apparent full recovery may disintegrate during some subsequent underwater stress.

CONCLUSIONS

Fitness examinations are essential to diving safety. They can reveal medical conditions that not only are incompatible with an individual's safety in the water but that also put the safety of other divers at risk. The aspects of the medical examination that assess the effects of diving on the health of the diver, but have no effect on in-water safety, should be considered as a separate activity. Nevertheless, the possible long-term effects of diving on the integrity of the hip and shoulder joints should be assessed. Any components of the examination that relate only to the nondiving vocational aspects of employment should also be considered separately.

The medical history and examination of a candidate before entry into training for a career as a commercial diver should be especially stringent because, at this stage, the consequences of rejection are relatively straightforward, whereas medical disqualification shortly after training may create large financial problems for the candidate.

Subsequent periodic assessments of the healthy young diver need not be as detailed as the initial examination. These assessments need not be an annual requirement, although this is preferred for ease of administration. The examination can become more frequent and certainly more detailed with increasing age.

Each medical examination for fitness to dive needs to be much more than mere application of predetermined pass/fail standards. If physicians are to carefully assess the individual diver in relation to the requirements and hazards of a particular working environment, they must have a good knowledge of the tasks and the risks of the job. The training objectives and minimal experience for the medical examiners of divers need to be implemented. The course content needs to include the physiologic aspects of diving, work procedures, types of equipment, and emergency procedures. The medical examiner requires some experience with active working scuba, hose, and bell divers and periodic refresher training. Assessments of knowledge and audits of performance of the examining physician are important contributions to the safety of divers in the water.

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CHAPTER 29 U.S. Navy Diving Equipment and Techniques

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This chapter reviews the various missions, diver qualifications, types of diving equipment, and operational diving techniques used by the U.S. Navy. The chapter also discusses fitness-to-dive considerations unique to military divers. The material in this chapter is not intended to be generally inclusive but to emphasize aspects unique to military diving.

MISSION

Military divers comprise a diverse group of occupations whose equipment and techniques depend on the particular mission. Traditionally, Navy divers have been grouped by training into various specialties. Conventional basic diver training commences at the Navy Diving and Salvage Training Center in Panama City, Florida, where students learn to perform diving missions such as ship husbandry, salvage, underwater construction and repair, and underwater explosive ordnance disposal (EOD). Fleet divers also are trained to conduct rescue operations for personnel trapped underwater, such as submariners stranded in a disabled submarine. The Navy maintains rapidly deployable teams of divers ready to travel anywhere in the world to provide deep salvage expertise for recovery of airplanes, munitions, and naval vessels. During search operations, divers are frequently required to help characterize underwater objects and geologic formations. Divers routinely conduct "security" swims, inspecting hulls for damage or signs of covert action, and are trained in the use of explosives underwater for applications such as clearing transportation routes. Some divers specialize in EOD. The principal mission of the EOD force is to identify, render safe, recover, and dispose of an ever-increasing array of explosive ordnance. Underwater construction divers (Seabee divers) are specifically trained in construction techniques used to build or repair harbors, piers, waterfronts, dams, and ocean structures.

The Navy has an extensive training and qualification program to maintain fleet operational diving capabilities. For a particular qualification, a diver is required to complete specified training and meet a defined level of competence. Subsequently, divers must maintain these qualifications by meeting prescribed requirements, which include requalification dives and fitness testing. The U.S. Navy professional qualification categories include (1) Basic Scuba Diver, (2) Second Class Diver, (3) First Class Diver, (4) Diving Medical Technician, (5) Master Diver, (6) Basic Diving Officer, and (7) Diving Medical Officer (DMO), as well as secondary qualifications such as Saturation Diver.

Initial dive training for Navy Sea, Air, Land (SEAL) team combat swimmers takes place at the Basic Underwater Demolition/SEAL training course taught at the Naval Special Warfare Center in Coronado, California. The Navy SEAL mission includes a wide variety of combat swimmer operations, including ship attacks, harbor penetrations, agent infiltration and exfiltration, and hydrographic reconnaissance. SEAL operations may be conducted with the divers either swimming free in the water or piloting open submersible SEAL Delivery Vehicles (SDVs).

The views expressed in this article are those of the authors and do not reflect official policy or position of the Department of the Navy, the Department of Defense, or the U.S. Government.

DIVING EQUIPMENT

The diverse missions of military divers require an assortment of diving equipment. Either scuba or surface-supplied equipment may be used, depending on the operational setting. The choice of equipment normally depends on the mission environment and the task requirements (duration and depth of the dive, type of work, and work constraints), along with the capabilities of the individual diver and personal choice. This discussion includes a brief review of open-circuit diving and a detailed discussion of closed-circuit scuba, followed by an overview of surface-supplied equipment available for Navy use.

Scuba Diving

The principal advantages of scuba diving are simplicity (particularly with open-circuit air scuba), transportability, and good mobility. Its primary disadvantages include limited personal protection (particularly head protection), limited gas duration (especially in open-circuit air scuba), limited depth range, restricted communications, and difficulty operating in strong currents.

Scuba can be divided functionally into open-circuit and closed-circuit equipment. Both systems use high-pressure gas stored in cylinders. Open-circuit scuba uses a demand-activated, two-stage reduction system, which reduces the gas pressure to that of the ambient environment. The first-stage regulator reduces the stored gas to approximately 110 to 150 psi more than ambient pressure, and the second-stage regulator reduces the pressure to the ambient level.[1] In open-circuit systems, the gas is exhaled and lost to the environment. There is no recirculation of the expired gas, and only a small fraction of the oxygen contained in each tidal volume is metabolized.[2] Closed-circuit systems reuse the expired breathing gas by collecting the exhaled gas, removing carbon dioxide, and replacing metabolized oxygen as required, thus significantly extending the operating time of the underwater breathing apparatus (UBA).

CONVENTIONAL OPEN-CIRCUIT AIR SCUBA DIVING

Applications and Equipment

Diving with open-circuit air scuba is the most common type of diving performed by the U.S. Navy, and all Navy divers are trained to use this type of equipment. It is portable and relatively uncomplicated to use. In addition, the equipment is designed to allow neutral buoyancy in the water, permitting the diver to work at multiple levels in the water column easily. It is commonly used for inspections, search and recovery tasks, and light maintenance and repair. Scuba equipment is discussed in Chapter 3.

Navy divers who use open-circuit scuba utilize commercially available equipment that meets specifications established by the Naval Sea Systems Command (NAVSEA). Many of the specifications are designed to maximize the performance characteristics and minimize the physiologic demands of the UBA, such as breathing resistance and dead space. Prior to being approved for Navy use, equipment is tested to ensure that it meets these standards. The Navy primarily employs a standard single-hose regulator in which the second-stage regulator is at the diver's mouth. Double-hose regulators are used in some situations, such as for operations in very cold water and marine-life photography. The following equipment is required as a minimum during Navy scuba diving: facemask, life preserver or buoyancy compensator, weight belt, knife, swim fins, submersible wristwatch, and depth gauge.[1]

Navy divers are required to observe the no-decompression limits in the U.S. Navy Standard Air Decompression Tables when using air scuba. These tables are specifically designed for Navy use, although many other groups use them as well. As discussed in Chapter 7, the current tables are based on Haldanian principles and have been extensively tested and modified with experience. The U.S. Navy has also pioneered the development of the maximum likelihood probabilistic model, a new method of computing decompression that is statistically based.[3] Weathersby and his colleagues at the Naval Medical Research Institute (NMRI) first described this new model, which allows the calculation of tables with varying predicted incidences of decompression sickness (DCS). Probabilistic models are somewhat more realistic than the Haldanian tables because they acknowledge that the risk of DCS after an air or mixed-gas dive increases gradually as a function of increasing decompression stress encountered on the dive rather than beginning abruptly at some arbitrary level of decompression stress. Probabilistic models are also useful in estimating comparative

decompression risk for proposed Haldanian schedules.

Operational Considerations

Standard open-circuit scuba diving is restricted to no-decompression dives, and planned decompression dives are not permitted, except in emergencies. In addition, the normal working limit for open-circuit scuba is 130 fsw,[4] although descent down to 190 fsw is permitted if dictated by operational necessity. The diver must be tended if direct ascent to the surface is not possible. The duration of the dive is often limited by the volume of gas carried.

Medical Considerations

There are no diving-related illnesses to which an open-circuit scuba diver is uniquely predisposed. However, one of the principal disadvantages of this method of diving is the limited head and body protection. A diver who becomes unconscious or debilitated as a result of nitrogen narcosis or other dysbaric disorders is not protected from drowning. The U.S. Navy depth limits for air diving are based primarily on the physiologic effects of nitrogen narcosis. However, as the depth increases, the time required for safe decompression increases rapidly, which further complicates the dive.

Skip-breathing is defined as voluntary hypoventilation. Open-circuit scuba divers commonly skip-breathe to reduce air consumption. Skip-breathing is discouraged because the resulting alveolar hypoventilation increases alveolar and blood carbon dioxide levels.

As with most other types of diving, DCS (see Chapter 8) and pulmonary barotrauma (see Chapter 9) are considerations in open-circuit scuba diving. These conditions may be encountered with any type of diving equipment.

CLOSED-CIRCUIT SCUBA DIVING

In closed-circuit scuba, the expired gas is exhaled into a breathing bag and retained in the breathing loop so that the oxygen remaining in the exhaled air may be recycled. The exhaled gas travels through a canister filled with a chemical designed to absorb the carbon dioxide. Once the carbon dioxide has been removed, the gas can be rebreathed. Oxygen is added to the breathing loop as needed to replace that consumed by metabolism. The movement of the gas through the system is powered by the positive and negative pressures generated by the diver's lungs during respiration. The flow of gas in one direction is achieved by the use of one-way valves in the circuit.

Application and Equipment

The primary groups in the Navy that use closed-circuit UBAs are SEALs and EOD divers. Closed-circuit scuba has several advantages over open-circuit scuba:

- Operating times are increased because all of the oxygen carried is available for metabolism.
- Exhaled gas is recirculated, which allows clandestine diving operations to be conducted without the threat of compromise from bubbles cascading to the surface.
- Acoustic and magnetic signatures, qualities that may be critical in EOD operations, can be designed to be very low.

Disadvantages include:

- Greatly increased cost of equipment, both in acquisition and in maintenance
- Increased diving complexity, with a resultant increase in training requirements
- Increased risk of diving accidents

The two main types of closed-circuit scuba are those with closed-circuit oxygen, in which only a single bottle of compressed oxygen is used, and those with closed-circuit mixed gas, which employ one bottle of pure oxygen and another of oxygen mixed with either nitrogen or helium.

Canister duration, the length of time that the canister can effectively remove carbon dioxide from the breathing loop, may be a key determinant of the operating duration of a closed-circuit UBA. Box 29–1 lists the factors that affect canister duration.[5–14] Although the operating limits of closed-circuit UBA canisters are tested under relatively strenuous conditions, divers who use closed-circuit UBAs must remain alert for the symptoms of carbon dioxide buildup. Improper packing of absorbent into the canister, leaking of the canister, or channeling of the gas in the canister may cause hypercarbia at any point in the dive. Channeling may occur despite meticulous care in filling the canister with absorbent.

A second important determinant of closed-circuit UBA operating duration is the *oxygen supply.* Excessive leaking of gas from the facemask, frequent depth changes, or

Box 29–1. Factors Affecting the Operating Time of Closed-circuit UBA CO_2-absorbent Canisters

1. Size of canister
2. Insulation of the canister
3. Gas flow design of the canister
4. Operating depth
5. Water temperature
6. Diver oxygen consumption
7. Diver breathing pattern
8. Absorbent chemical used
9. Storage time and temperatures

swimming at a very high rate of oxygen consumption may significantly decrease the duration of the oxygen supply. A diver using a closed-circuit system obviously realizes no benefit from voluntarily reducing the ventilation rate (skip-breathing). When skip-breathing is practiced in a closed-circuit UBA, the hazard is increased over that in open-circuit scuba because the resultant hypercapnia may predispose to central nervous system (CNS) oxygen toxicity. The best techniques for extending the duration of oxygen supply are to avoid frequent depth changes, minimize leaks from the facemask, and avoid a high exercise rate because strenuous swimming consumes proportionately more oxygen for the distance traveled. Canister function is also less efficient with large tidal volumes, which may result in incomplete carbon dioxide scrubbing (canister blowby).

Pulmonary oxygen toxicity has not been a limiting factor in closed-circuit oxygen diving until recently. In the past, CO_2-absorbent canister operating limits were based on the oxygen consumption and carbon dioxide production appropriate for working divers. These canister limits resulted in closed-circuit oxygen dives being shorter than the 4 hours a day allowed by the CNS oxygen toxicity limits. Canister operating limits for a diver at rest for the majority of the operation (SDV operations) would be expected to be much longer. There are three reasons for this increased duration: decreased CO_2 production at rest, longer canister dwell time as the respiratory rate decreases at rest, and reduced tidal volumes. The reduced exercise rate might also be expected to allow for longer dives at shallow depths without CNS toxicity. Studies requested by the Naval Special Warfare Command and performed by Marineau and her colleagues at the Navy Experimental Diving Unit (NEDU)[15] found that the MK 25 Mod 2 canister will reliably support a diver for an 8-hour profile during which the diver spends 7 hours resting and 1 hour exercising. However, the report notes that divers performing such profiles may experience pulmonary oxygen toxicity, and the current MK 25 MOD 2 operating limits for SDV operations have pulmonary oxygen toxicity considerations as one of the limiting factors.

Another physiologic factor encountered in closed-circuit scuba diving that is not present with open-circuit scuba diving is *static lung loading.* Static lung loading occurs in closed-circuit UBAs because the breathing bag may be at a slightly different level in the water column than the mouth. With open-circuit scuba, the regulator is designed to provide the diver with air under pressure once a slight inspiratory pressure is generated. Open-circuit regulators are designed to ensure that these pressures are only minimally affected by changes in diver position. The breathing bag in a closed-circuit UBA is exposed to the ambient pressure in the water column. If it is located slightly below the diver, as would be the case with a UBA worn on the chest of a diver swimming in a prone position, the increased pressure at the breathing bag compared with the mouth helps force gas into the diver's lungs on inhalation but resists the diver's effort to exhale the gas back into the breathing bag. If the breathing bag is located above the lungs, as is the case with a back-mounted UBA in this situation, exhalation is facilitated but inspiration is more difficult.

The movement of gas through the breathing loop of the UBA is powered by the diver's respiratory efforts, and the design of a closed-circuit UBA must carefully consider breathing resistance. A poorly designed canister may result in excessive resistance as the gas flows across the canister bed. The breathing hoses, which connect the mouthpiece to the breathing bag, must be large enough in diameter so that gas flow is not impeded. The hoses must be flexible but resistant to crimping or collapse. One-way valves at the mouthpiece and canister fittings must ensure that the gas flows in the correct direction in the breathing loop, but they must also not cause undue resistance to the flow of gas.

In the 1970s and 1980s, extensive testing of closed-circuit UBAs at NEDU in Panama

City, Florida led to the development of performance goals for UBA design that attempt to address many of the issues already outlined.[16]

CLOSED-CIRCUIT OXYGEN SCUBA DIVING

The closed-circuit oxygen UBA used by the United States military is the Draeger LAR V. This rig is worn over the chest and abdomen of the diver, with the breathing bag and the canister contained in a fiberglass shell and the oxygen bottle attached to the bottom of the UBA (Fig. 29–1).

The newest version of the Draeger LAR V UBA currently in use by U.S. combat swimmers has been designated the MK 25 Mod 2.[17] This UBA offers the advantages of being light (25 lbs) and mechanically simple. It can withstand the rigors of high-speed transport in small boats and still function reliably. The MK 25 Mod 2 is currently replacing the older Mod 0. The Mod 2 has a larger oxygen bottle (1.9 L floodable volume that holds 410 L of oxygen at its working pressure of 207 bar) and a larger, better insulated canister.[17] The Mod 2 canister has also been redesigned to use smaller-mesh CO_2 absorbent. The MK 25 Mod 1 is similar to the Mod 2 except that is has been designed to have a lower magnetic and acoustic signature for EOD use. Because divers swimming at a relaxed, moderate pace consume approximately 1.3 L/min of oxygen,[1] the oxygen supply might be expected to last over 4 hours if the bottle is fully charged and gas loss from the UBA is minimized. The carbon dioxide–absorbent canister duration depends on water temperature and diver activity. The MK 25 operating limits are currently classified.[18] As noted earlier, no bubbles escape from the UBA, which allows the diver to approach targets undetected.

The primary disadvantage of diving with closed-circuit oxygen is the depth limitation imposed by the risk of CNS oxygen toxicity. This is usually not a significant problem in SEAL diving operations because many combat swimmer missions require a depth only deep enough to provide concealment at night. The MK 25 Mod 2 UBA is also considerably more expensive than open-circuit scuba.

The operation of the MK 25 Mod 2 UBA is exceedingly simple. Gas is supplied from a breathing bag via the inhalation hose. If the gas in the breathing bag is insufficient for the diver's tidal volume, a demand valve is activated and oxygen is added from the high-pressure bottle. A manual bypass valve is present so that the diver may add gas to the breathing loop while descending or in the event of malfunction of the demand valve or canister. The gas flow path of the MK 25 Mod 2 UBA is shown in Figure 29–2.

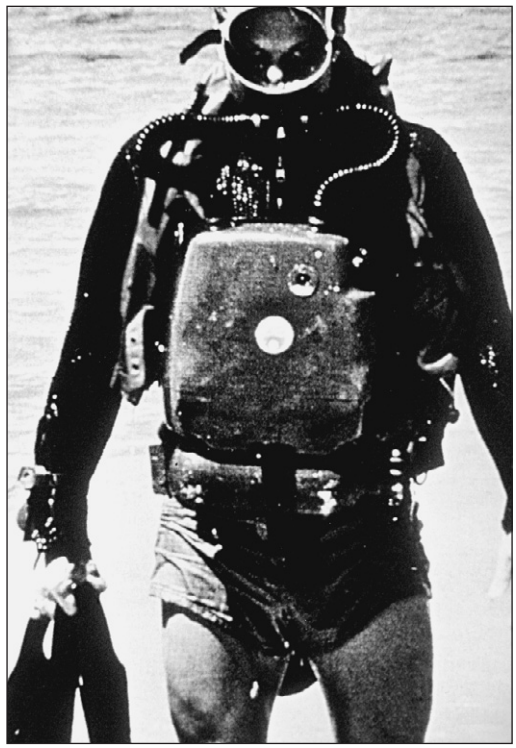

Figure 29–1. Diver wearing a Draeger LAR V UBA. (Photograph courtesy of U.S. Navy.)

A predive purge of the nitrogen in the rig and in the diver's lungs must be performed for any closed-circuit oxygen UBA that is not configured to monitor the oxygen concentration in the breathing mix. Although the UBA gas bottle contains only oxygen, nitrogen is present in the diver's lungs and in the UBA breathing loop before the dive. If a large quantity of this inert gas is retained in the UBA, the breathing bag volume may not be small enough to trigger the addition of oxygen, even though the oxygen fraction in the breathing mix may have dropped to a dangerously low level. To prevent such an occurrence, NEDU has developed purging procedures for the MK 25 Mod 2 UBA.[19–21] The goal of the predive purge is to remove enough nitrogen to eliminate the risk of

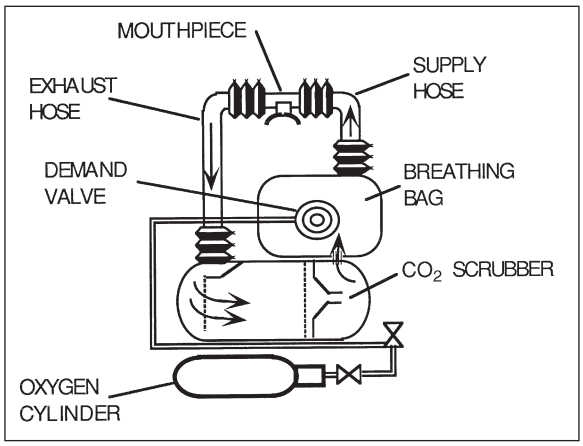

Figure 29–2. Gas flow path within the Draeger LAR V UBA.

hypoxia; elimination of all the nitrogen from the UBA is not necessary. Excessive purging of the UBA beyond the prescribed procedure serves only to reduce the amount of oxygen available for diving and to increase the risk of CNS oxygen toxicity.

Statistical analysis of episodes of toxicity resulting from experimental dives with oxygen has shown that relatively small decreases in the oxygen fraction of a UBA are important in reducing the risk of CNS oxygen toxicity.[22] The fraction of oxygen in the UBA breathing loop required to prevent hypoxia depends on both the gas volume in the breathing loop and the design of the oxygen addition mechanism. Because these factors may vary somewhat among UBAs, it is important that the diver use a purge procedure developed for the specific closed-circuit oxygen UBA used.

The oxygen fraction in a closed-circuit UBA changes throughout the dive. As the diver descends from the surface to the desired dive depth, additional oxygen needs to be added to the breathing bag to compensate for the increased pressure. This has been found to cause the oxygen fraction in the Draeger LAR V to increase from a mean of 71% at the surface to 82% at a depth of 20 fsw.[20] Theoretically, the oxygen fraction in a closed-circuit UBA after the initial descent might be expected to decrease during the dive because of off-gassing of nitrogen from body tissues. This consideration resulted in the previous Navy practice of repurging the UBA every 30 min during the dive. This procedure was discontinued when no significant decrease of the oxygen fraction in the Draeger LAR V UBA was found on 2-hour experimental dives without periodic repurging.[20] In fact, the oxygen fraction might actually increase during the dive if a leak from the facemask is significant or if the diver changes depth frequently because the volume of nitrogen-oxygen gas mixture lost from the breathing loop during these events is replaced with 100% oxygen from the cylinder.

To prevent CNS oxygen toxicity, the U.S. Navy publishes oxygen exposure limits for closed-circuit oxygen scuba diving. The current limits[23] were developed by Butler and Thalmann[24–26] at NEDU in the early 1980s after analysis of three experimental dive series that together produced almost 700 dives. The divers in these series were immersed and were working because combat swimmers would be expected to use closed-circuit UBAs under these conditions. Earlier researchers had found both of these factors to increase the risk of CNS oxygen toxicity.[27–29] The water temperatures for these studies were chosen to induce mild to moderate cold stress, and carbon dioxide levels in the UBAs were monitored to ensure that the divers' breathing mix had a carbon dioxide partial pressure less than 3.8 mm Hg (0.5% surface equivalent). The single-depth oxygen exposure limits currently used by the U.S. Navy are shown in Table 29–1. Brief downward excursions are permitted provided the diver remains at a depth of 20 fsw or shallower for the remainder of the dive.[19,23] These limits have now been in use by United States military combat swimmers since 1983. Walters and colleagues[30] reviewed data from the Naval Safety Center and found records of 157,930 closed-circuit oxygen dives with only a single convulsion reported.

Table 29–1. U.S. Navy closed-circuit scuba oxygen limits

Depth	Length of Exposure (min)
25 fsw or less	240 min
30 fsw	80 min
35 fsw	25 min
40 fsw	15 min
50 fsw	10 min

CLOSED-CIRCUIT MIXED-GAS SCUBA DIVING

The MK 16 UBA is a closed-circuit mixed-gas UBA that uses a microprocessor to control the partial pressure of oxygen at 0.75 ata.[31] Note that this is slightly higher than the more conservative 0.7 ata that was used to calculate the decompression tables for the MK 16 and is intended to give the diver an extra margin of safety. It contains two high-pressure gas bottles, one for oxygen and one for a diluent gas (either air or a helium-oxygen mixture). As oxygen is consumed, three sensors in the breathing loop detect the falling partial pressure of oxygen and activate the oxygen addition valve. The diver can monitor the oxygen partial pressure on a facemask-mounted primary display and on a secondary display. The two gas bottles, the microprocessor, the breathing bag, and the large, circular carbon dioxide–absorbent canister are enclosed in a fiberglass casing that is worn on the diver's back. Manual bypass valves provide additional oxygen or diluent gas to the UBA, if necessary. Figure 29–3 shows a diver wearing a MK 16 UBA.

The large, well-insulated carbon dioxide–absorbent canister on the MK 16 UBA achieves operating durations of up to 300 min, depending on the depth and water temperature of the dive.[31] Because the MK 16 UBA monitors and controls the oxygen levels in the apparatus, there is no need to purge the rig before diving. The higher partial pressure of oxygen breathed by the diver at shallow depths provides a distinct advantage over air breathing during shallow dives and decompression stops. The U.S. Navy has approved the use of a computer program that allows customized computation of decompression for combination air/0.7 ata nitrox dives, and the reduction in decompression time realized by using the MK 16 instead of air at shallow decompression stops is approximately 60%.[32]

Figure 29–3. Diver wearing a MK 16 UBA. (Photograph courtesy of U.S. Navy.)

The MK 16 UBA has a very low magnetic and acoustic signature. This was a requirement during its development because of its intended use by Navy EOD units to disarm mines that might have acoustic or magnetic firing devices. Because it can use heliox, the MK 16 UBA can achieve depths of 300 fsw and deeper with helium as the diluent gas. The present U.S. Navy working limits are 150 fsw for nitrox dives and 200 fsw for heliox dives.[31] The primary disadvantage of the MK 16 UBA is its high price tag (approximately $34,000). In addition, preparation and use of the MK 16 UBA requires a significant amount of training; even more technical sophistication is required to maintain and repair it. Another disadvantage is that when the UBA is used in the nitrox mode, the higher partial pressure of nitrogen at depths greater than 85 fsw may cause nitrogen narcosis at depths shallower than would be the case with air dives.

The EOD community has requested that decompression tables be developed for the MK 16 that use a 1.3 ata partial pressure of oxygen in nitrogen and helium in order to reduce the decompression obligation for deep diving with this UBA. NEDU has completed development of these tables, but the

new tables have not yet been published in the U.S. Navy *Diving Manual*.

MEDICAL CONSIDERATIONS IN CLOSED-CIRCUIT SCUBA DIVING

Central Nervous System Oxygen Toxicity. CNS oxygen toxicity (see Chapter 12) is one of the greatest hazards with closed-circuit oxygen UBAs. The likelihood of CNS oxygen toxicity increases with increasing time of exposure and partial pressure of oxygen. Because the physiologic effects of an inspired gas are a function of its partial pressure, increasing either the absolute pressure or the fractional percentage of oxygen increases the effects. Other factors that increase susceptibility to CNS oxygen toxicity are immersion,[23,27–29] exercise,[23,28,29] elevated levels of inspired carbon dioxide,[23,33] and cold stress.[23,28] Although the limits shown in Table 29–1 have been extensively tested and proven safe in field use, toxicity episodes, including convulsions, have occurred within the times allowed by these limits during controlled experimental trials. Other possible signs and symptoms of CNS oxygen toxicity include muscle twitching, nausea, visual disturbances, tinnitus, confusion, and dysphoria. Should any of these symptoms occur during a closed-circuit scuba dive, it is important to make a controlled ascent to the surface at once in case the episode should progress to a convulsion. However, a convulsion may occur without any preceding warning symptoms. If a diver experiences a convulsion while in the water, the dive buddy should follow the sequence of steps outlined in Box 29–2 to try to get the diver safely to the surface.[23] A number of measures can be taken before and during the course of the dive to minimize the risk of oxygen toxicity. These measures are listed in Box 29–3.[19]

Theoretically, CNS oxygen toxicity should not be a problem with the MK 16 UBA. Divers using this UBA must be familiar with the signs and symptoms of oxygen toxicity, however, because a rig malfunction—such as sensor malfunctions, microprocessor failure, or a mechanical sticking of the oxygen addition valve—may cause the oxygen level to rise to potentially toxic levels.

For many years, candidates for combat swimmer training programs in the Navy were required to pass an oxygen tolerance test (OTT) consisting of a 30 min exposure to 100% oxygen at 2.8 ata (60 fsw) while seated in a dry chamber at rest. Candidates who exhibited CNS oxygen toxicity during the OTT were disqualified from combat swimmer training. This test was discontinued in 1999 after a study conducted by Walters and coworkers.[30] The authors recommended discontinuation of the OTT because:

- The failure rate for the OTT as it was administered in Naval Special Warfare was found to be very small (0.096%).
- The logistical burden of administering the OTT caused testing to be conducted after

Box 29–2. Management of an underwater convulsion

1. Assume a position behind the convulsing diver. The weight belt should be left in place to prevent the diver from assuming a face down position on the surface. Release the victim's weight belt only if progress to the surface is significantly impeded.
2. Leave the victim's mouthpiece in his mouth. If it is not in his mouth, do not attempt to replace it; however, if time permits, ensure that the mouthpiece is switched to the SURFACE position.
3. Grasp the victim around his chest above the UBA or between the UBA and his body. If difficulty is encountered with gaining control of the victim in this manner, the rescuer should use the best method possible to obtain control of the victim. The UBA waist strap or neck straps may be grasped if necessary.
4. Make a controlled ascent to the surface, maintaining a slight pressure on the diver's chest to assist exhalation.
5. If additional buoyancy is required, activate the victim's life jacket. The rescuer should not release his weight belt or inflate his life jacket.
6. Upon reaching the surface, inflate the victim's life jacket if not previously done.
7. Remove the victim's mouthpiece and switch the valve to the SURFACE position to prevent the possibility of the rig flooding and weighing down the victim.
8. Signal for emergency pick-up.
9. Once the convulsion has subsided, open the victim's airway by tilting his head back slightly.
10. Ensure that the victim is breathing. Mouth-to-mouth breathing may be initiated if necessary.
11. If an upward excursion occurred during the actual convulsion, transport to the nearest chamber and have the victim evaluated by an individual trained to recognize and treat diving-related illness.

the SEAL students had completed the most rigorous 9 weeks of SEAL training and class size was much smaller. Disqualification of a SEAL candidate at that point in training should be based on clear and compelling evidence that he is unfit to continue training. The OTT was not thought to meet that standard.

- Even if a more stringent OTT were to be developed, individual variability would prevent any single screening test from being a reliable indicator of increased oxygen sensitivity.
- Factors such as a high exercise rate, diver hypoventilation, canister failure, inadvertent depth excursions, inadequate thermal protection, or excessive purging of the UBA may be equally or more important than individual oxygen sensitivity as modifiers of the risk of oxygen toxicity.

Pulmonary Oxygen Toxicity. Unlike CNS oxygen toxicity, pulmonary oxygen toxicity has an insidious onset with a slow, steady progression of chest pain, cough, and breathing difficulty if the diver continues to breathe hyperbaric oxygen. Because of this potential for pulmonary oxygen toxicity, NEDU has recommended that any dive on which the diver breathes from the LAR V for more than 240 min be considered an *extended* oxygen dive.[15] NEDU recommends that divers perform no oxygen dives for at least 24 hours before an extended oxygen dive and that they not perform any MK 25 Mod 2 or MK 16 dives for at least 2 weeks after an extended oxygen dive. NEDU further recommends that extended oxygen dives not be performed any closer than 45 days apart. The extended MK 25 Mod 2 UBA operating limits for SDV operations are currently classified.[18]

Hypoxia. Hypoxia in closed-circuit scuba diving can cause loss of consciousness without warning. As described previously, hypoxia can occur with a closed-circuit oxygen UBA if a sufficiently large volume of inert gas remains in the breathing loop to keep the demand valve from being activated as oxygen in the breathing loop is consumed. Both forgetting to purge the UBA and procedural mistakes such as mistakenly exhaling into the breathing bag during the emptying phase of the procedure may cause the diver to become hypoxic. Using oxygen bottles that have inadvertently been filled with air is another potential cause of hypoxia.

The risk of hypoxia is greatest at the start of the dive when the diver is breathing from the UBA on the surface. As soon as the diver descends in the water column, the absolute pressure increases, thereby increasing the partial pressure of oxygen. With the MK 25 Mod 2 UBA, additional oxygen is added to the breathing bag during descent to compensate for the pressure-induced decrease in volume. This additional oxygen increases the fraction of oxygen in the breathing mix as well.[19] If unconsciousness occurs at the start of a dive, before the diver has left the surface, neither CNS oxygen toxicity nor arterial gas embolism is possible and hypoxia should be suspected. Divers should be treated by removing the mouthpiece, opening the airway, and allowing them to breathe surface air with the expectation of a rapid recovery. Divers must carefully monitor UBA oxygen levels during ascent from deep dives when using a closed-circuit

Box 29–3. Avoidance of oxygen toxicity

1. Observe the oxygen exposure limits in Table 29–1.
2. Swim as shallow as possible within the mission constraints of the dive.
3. Swim at a comfortable, relaxed pace.
4. Observe the canister operating limits of the UBA.
5. Avoid skip breathing done from habit or a misguided attept to conserve gas.
6. Never swim with a reduced volume in the breathing bag as a method of bouyancy control.
7. Wear adequate thermal protection for the ambient water temperature to avoid shivering and the tendency to swim faster to stay warm.
8. Minimize gas loss from the UBA.
9. Minimize depth changes as allowed by mission constraints.
10. Use an accurate depth gauge with a large scale face to allow precise depth control in the shallow depth range.
11. If a dive pair is using a single depth gauge, the diver with the depth gauge should always be the deepest of the pair.
12. Use the prescribed purge procedure without additional purging.

mixed gas UBA. The partial pressure of oxygen in the breathing mix falls because of the decreased absolute pressure, and they may need to slow their ascent or manually add extra oxygen to speed up the restoration of the desired oxygen partial pressure in the rig.

Divers using the MK 16 UBA must be alert for equipment malfunctions that may cause hypoxia, such as battery flooding or microprocessor failure. They should be aware of the low levels of oxygen in their UBAs from the warning signal provided by the primary display and should immediately add oxygen to the breathing loop and return to the surface.

Hypercarbia. Channeling, canister leaks, excessive exertion, or exceeding the operating duration limit of the canister may result in a carbon dioxide buildup. Channeling and leaks are possible despite careful canister preparation and strict adherence to operating guidelines. The symptoms of hypercarbia are generally progressive: Once a canister has started to fail, its performance continues to deteriorate. If the failure stems from a leaking canister, hypercarbia may worsen very rapidly over the course of several minutes and may be followed by inhalation of a caustic alkaline solution resulting from the mixture of water and the hydroxide compounds in the carbon dioxide absorbent.

The first symptom of hypercarbia that a diver usually notices is deep and rapid breathing as the partial pressure of carbon dioxide exceeds the 3% surface equivalent level. An inspired P_{CO_2} of 30 mm Hg was found to produce a significant increase in minute ventilation at several exercise rates.[34] This sign may be misinterpreted as resulting from external factors such as currents or increased exercise rate if the diver is not thinking of the possibility of hypercarbia. If hypercarbia continues to increase to the 5% to 10% range, increased minute ventilation may progress to frank dyspnea, and a severe, throbbing headache may ensue. A 5 min exposure of resting subjects to 8% inspired CO_2 was found to produce a 25% incidence of headache.[35] Further increases may lead to confusion and unconsciousness. When a closed-circuit oxygen UBA is used, the first symptom of hypercarbia may be a convulsion resulting from the potentiation of oxygen toxicity by hypercarbia.[33]

Hypercarbia suspected during a dive with the MK 25 Mod 2 UBA should be managed by immediately activating the oxygen bypass and by exhaling through the nose to allow gas to escape from the facemask. This lowers the fraction of carbon dioxide in the breathing loop. At the same time, the diver should begin a controlled ascent to the surface, which also lowers the partial pressure of the carbon dioxide remaining in the breathing loop. The diver should maintain a vertical position in the water column during ascent to minimize the possibility of inhaling a caustic solution if the canister is leaking.[19,23] Once at the surface, the diver should immediately discontinue breathing from the UBA and begin breathing surface air. The diver should be alert for symptoms of oxygen toxicity both during the ascent and after reaching the surface.

Inhalation of Caustic Solution (Caustic Cocktail). Carbon dioxide absorbents used in the MK 25 Mod 2 UBA and other closed-circuit UBAs are generally composed of hydroxide compounds that produce caustic alkaline solutions if water gains access to the canister. As mentioned previously, a leaking canister generally produces symptoms of hypercarbia initially as the absorbent material loses its ability to remove carbon dioxide. The symptoms of hypercarbia progress at a rate dependent on the magnitude of the leak. At some point, as the canister fills, water may enter the inhalation hose while the diver is attempting to inhale. This caustic mix may then enter the pharynx, esophagus, and trachea and cause immediate pain and choking. Management of a caustic cocktail is essentially the same as that for hypercarbia and consists of immediate activation of the bypass valve, exhaling through the nose, and a controlled ascent to the surface.[19,23] Assumption of a vertical position in the water is critical because it may prevent further aspiration of the alkaline solution from the canister and breathing bag. A diver who inhales a caustic solution is at significant risk for arterial gas embolism; the symptoms, because of their severity, are highly likely to lead to an uncontrolled ascent. If fresh water is available on the surface, several mouthfuls should be swallowed; no attempt should be made to neutralize the alkaline solution with administration of acidic solutions. Any altered state of consciousness should raise the question of an arterial gas embolism, although it may also be due to carbon dioxide intoxication or near-drowning.

Middle-Ear Oxygen Absorption Syndrome (Draeger Ear). Divers who use closed-circuit UBAs with their higher partial pressures of oxygen may notice auditory symptoms after the dive.[19,23] Symptoms often are first noted upon waking the morning after the dive. The usual presentation is a painless decrease in hearing, but mild discomfort may also be noted, as may a fullness in the ear resulting from serous otitis media. The cause of this disorder is not well defined, but one proposed mechanism is related to middle-ear oxygen absorption. As the diver breathes gas with a very high fraction of oxygen, the air in the middle-ear cavity is gradually replaced with a gas mixture having a much higher oxygen content. After the dive, this oxygen is slowly absorbed by the tissues of the middle ear and is subsequently metabolized. If the diver's eustachian tube does not open spontaneously, the absorbed gas is not replaced with air and a relative negative pressure may develop. Treatment consists of simply equalizing the pressure in the middle ear with a Valsalva maneuver. This procedure typically needs to be repeated several times as additional oxygen is absorbed.

Hyperoxic Myopia. Progressive myopic changes as a result of hyperoxic exposures have been reported in patients undergoing hyperbaric chamber therapy.[36–41] This phenomenon has recently been reported to occur in a closed-circuit scuba diver using a 1.3 ata P_{O_2} in a nitrox mix.[42] After several weeks of diving, he began to experience a progressive decrease in distance vision in both eyes with preservation of near visual acuity. The clinical findings in this diver indicated that he was suffering from hyperoxic myopia, which is a form of ocular oxygen toxicity that is thought to result from alteration of the refractive properties of the crystalline lens of the eye.[38] Additional unpublished reports of suspected hyperoxic myopia in scuba divers have been encountered, and the potential for hyperoxic myopia should be recognized by anyone conducting intensive, multiday diving operations with hyperoxic gas mixes at or above 1.3 ata.[42]

Nitrogen Narcosis. Use of the MK 16 UBA in the nitrox mode leads to a greater likelihood of nitrogen narcosis on deep dives. Table 29–2 displays the equivalent air depths for various depths while using the MK 16 UBA. The U.S. Navy limits nitrox dives on the MK 16 UBA to 150 fsw.

Table 29–2. Equivalent air depth (EAD) for the MK 16 (assuming oxygen set point of 0.75 ata)

Depth of MK 16 UBA (fsw)	EAD (fsw)
10	−10
20	3
30	16
40	28
50	41
60	53
70	66
80	79
85	85
90	91
100	104
110	117
120	129
130	146
140	155
150	167

Operational Considerations in Closed-Circuit Scuba Diving

The MK 25 Mod 2 UBA is currently used in the United States military by Navy SEAL teams, Army Special Forces scuba-capable teams, Air Force Special Tactics groups, and Marine Reconnaissance units. This UBA has proven to be highly suitable for a wide variety of combat swimmer operations, as described previously. Closed-circuit oxygen has several disadvantages in SDV (Figs. 29–4 and 29–5) operations: (1) The depth limitations imposed by the current oxygen exposure limits do not allow enough operational flexibility for some SDV missions, and (2) although the MK 25 Mod 2 UBA performs well for swimmers who are in a horizontal position in the water column, divers in a sitting position navigating an SDV (see Fig. 29–5) often find that the increased pressure on the breathing bag, compared with that on the mouth, causes an uncomfortable tendency for the rig to off-gas. Despite these disadvantages, the simplicity, small size, and reliability of the MK 25 have led to its use on many SDV operations.

Another operational problem that may be encountered with closed-circuit UBAs is the tendency for divers to experience buoyancy changes as the gas in the breathing bag expands on ascent. This may result in the diver's making an uncontrolled ascent in the last few feet to the surface. This event may be very hazardous, especially when it occurs near a hostile warship. The change in

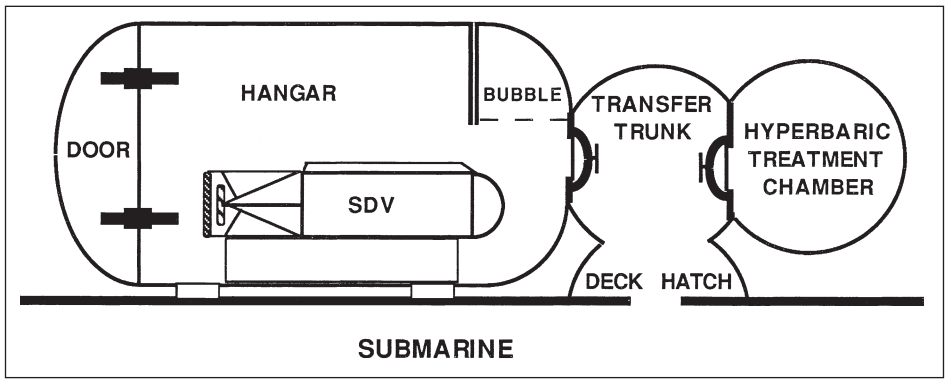

Figure 29–4. Schematic of a Dry Deck Shelter with SDV (SEAL Delivery Vehicle).

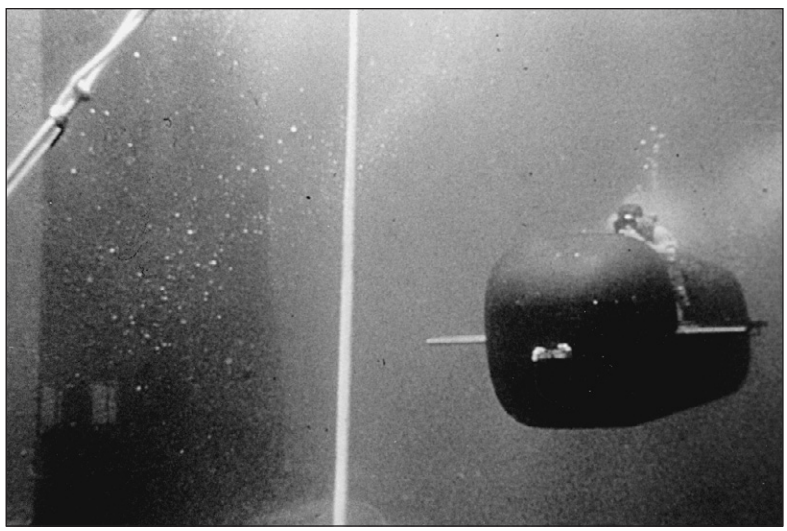

Figure 29–5. SDV and host submarine. (Photograph courtesy of U.S. Navy.)

buoyancy may be prevented by the diver's exhaling through the nose on ascent, thus allowing gas to escape from the breathing loop, but this maneuver allows potentially compromising bubbles to appear on the surface. The best technique for avoiding this problem is to avoid sudden ascents when there is a need to avoid off-gassing from the UBA.

The MK 16 UBA has been successfully used by EOD divers for many years for mine countermeasure operations. The rig is nonmagnetic and has a low acoustic signal. These characteristics, coupled with its long operating duration and relatively deep operating depth, make the MK 16 UBA very well suited for EOD missions.

The MK 16 UBA is used by Navy SEAL teams in SDV operations. This UBA lends itself well to these operations because of its long operational duration and increased depth capability. The static lung load of the back-mounted UBA is also near-optimal for divers in a seated position in the swimmer delivery vehicle and allows full facemasks to be used without significant overpressurization of the mask. The use of a full facemask allows the diver to avoid mouthpiece fatigue and to use diver communication systems within the SDV.

Decompression Considerations in SEAL Delivery Vehicle Operations

SDVs are often launched from either a fast-attack or a converted ballistic missile submarine. These submarines are fitted with a special hyperbaric complex called a *dry deck shelter* (see Fig. 29–4; Figs. 29–6 and 29–7). SDV operations often entail very long dive times, with most of the dive spent at a relatively shallow transit depth but with brief excursions to a greater depth. Use of the standard air tables to calculate decompres-

Figure 29–6. Launching the SDV from the Dry Deck Shelter. (Photograph courtesy of U.S. Navy.)

sion schedules for such profiles results in decompression obligations that are both tactically impractical and physiologically unnecessary. In addition, many SDV dives require the use of both air and nitrox mixtures on the same dive profile. In these circumstances, decompression schedules may be calculated in several ways:

- The *Combat Swimmer Multi-Level Dive (CSMD)* procedures were developed by Thalmann and Butler[43] at NEDU in 1983 and have since been used extensively by Navy SEAL teams for SDV operations. The CSMD procedures allow calculation of decompression times for these very long multilevel dives by dividing the dive into transit periods at 30 fsw and shallower and downward excursions deeper than 30 fsw. The procedures are based on the 1957 Navy air tables and assume that the diver is breathing air at shallow depths. For depths greater than 70 fsw with the MK 16 UBA, the equivalent air depth is calculated and used. The CSMD procedures do not allow the diver to receive decompression credit for the higher oxygen partial pressure of the MK 16 UBA during decompression stops but still result in substantial decompression time savings over the Standard Air Tables because the diver is not required to undergo decompression as though the entire bottom time was spent at the greatest depth attained during the dive.
- The *Naval Special Warfare Dive Planner* computer program was developed at NMRI in 1993.[32] This program uses a laptop computer version of the NMRI maximum-likelihood nitrox decompression algorithm and has been approved by NAVSEA for use in SDV operations. The divers follow the CSMD procedures until they return to the submarine. At that point, they provide the DMO and the diving supervisor with their dive profile.

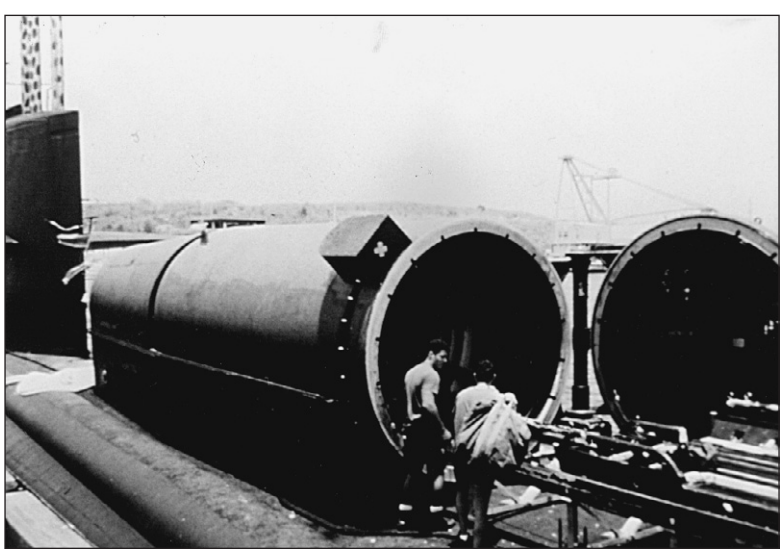

Figure 29–7. Dry Deck Shelter being loaded at pierside. (Photograph courtesy of U.S. Navy.)

The profile is entered into the Dive Planner program, and a customized decompression schedule is calculated. The two primary benefits of the Dive Planner over the CSMD procedures are the ability to give the divers credit for the periods of time spent using the MK 16 at shallow depths and to provide schedules for decompression breathing either mixed gas from the MK 16 or air.

- In January 2001, the U.S. Navy began using the first Navy-approved diver-worn decompression computer, the *Cochran NAVY Decompression Computer*. The computer uses the VVAL 18 decompression algorithm developed by Captain Ed Thalmann during over two decades of testing at both NEDU and the NMRI.[44] The first version of the Cochran NAVY decompression computer uses a nitrox algorithm customized for SEAL SDV operations. The computer assumes that the diver is breathing air at 78 fsw and shallower and nitrox with a constant oxygen partial pressure of 0.7 ata at 79 ft and deeper. This allows SEAL divers to breathe from either an open-circuit air source or from the MK 16 and still ensure that decompression will be safe.

Tables produced by VVAL 18 result in no-decompression limits that are somewhat more conservative than the current Navy no-decompression limits in the shallow range, similar in the 60 to 80 ft range and less conservative at deeper depths. Like the NMRI probabilistic model, this model becomes much more conservative than the current Navy air tables as total decompression time increases. Very long bottom time profiles commonly require decompression times three or four times as long as those found in the Standard Navy Air Tables.[44]

On 20 October 2000, NEDU recommended approval of the Cochran NAVY Decompression Computer for SEAL use.[45] On 25 January 2001, the Supervisor of Diving and Salvage for the U.S. Navy authorized the use of this computer by selected SEAL units.[46] The Navy's first decompression computer dive was conducted by SDV Team One on 31 January 2001 in the waters off Barber's Point in Hawaii.

Is the Cochran NAVY Decompression Computer suitable for use by sport divers? Because most recreational divers do not routinely make decompression dives, the extra safety incorporated into those areas of the VVAL 18 model will not benefit them. The air no-decompression limits found in this model are less conservative than those in most, if not all, civilian dive computers. However, Navy divers have used less conservative shallow no-decompression limits for many years with a very low incidence of DCS. As outlined in Captain Thalmann's NEDU Report 8-85,[47] additional testing of the deeper no-decompression limits in his model resulted in no cases of DCS in 107 experimental dives. These trials were performed under worst-case conditions, with divers immersed in cold water and exercising strenuously on the bottom. The 3 to 5 min safety stop that has become common in recreational diving practice would further increase the safety of the VVAL 18 no-decompression limits.

Two additional factors lower the decompression risk of the Cochran NAVY computer as it is used by SEAL teams. Because the computer assumes that the diver is breathing the gas mix with the highest possible partial pressure of nitrogen for the depth sensed, in many cases, the decompression calculations provided will be much more conservative than those required had the diver's breathing mix been recorded precisely. In addition, because SEAL diving operations entail multiple divers, all divers undergoing decompression as a group will do so on the computer that displays the longest decompression time, providing an extra measure of safety for the other divers on the profile.

Approval of the Cochran NAVY computer heralds the beginning of a new era in Navy diving. Use of this computer offers the opportunity to accurately capture research-grade data about operational dive profiles. This data will be collected by NEDU and archived there. It will then be available to decompression researchers. If and when episodes of DCS occur, the profiles that caused the episodes will have been recorded precisely, rather than having to rely on possibly inaccurate data supplied by the diver. Should clusters of bends cases occur on similar profiles, this may be addressed by retesting of the VVAL 18 algorithm in the targeted areas. NEDU has established a standing oversight panel on decompression-computer diving to oversee these efforts and to recommend changes to the decompression algorithm or the hardware as needed. The Cochran NAVY is shown in Figure 29–8.

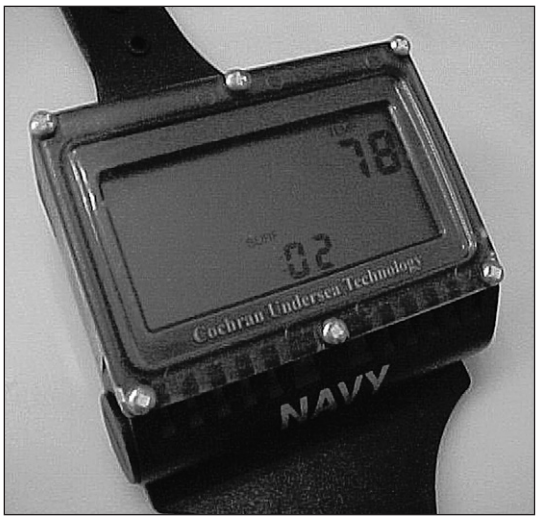

Figure 29–8. The Cochran NAVY decompression computer.

Decompression Considerations—Advanced Seal Delivery Vehicle Operations

The Advanced Seal Delivery Vehicle (ASDS) is a 65 ft dry submersible craft that has been developed by Naval Special Warfare (Fig. 29–9). Compared with the SDV, the ASDS has the advantages of enhanced environmental protection, increased range, augmented mission equipment carrying capacity, and an improved medical evacuation space. The ASDS has a greatly enhanced operating capability compared with the SDV but also entails a new set of decompression issues. ASDS operational doctrine is evolving as this chapter is being written, but it appears at this time that diver operations from this craft will entail either (1) compression, lock-out, and ascent on air with a subsequent shift to closed-circuit oxygen or (2) compression on air, closed-circuit mixed-gas breathing, and

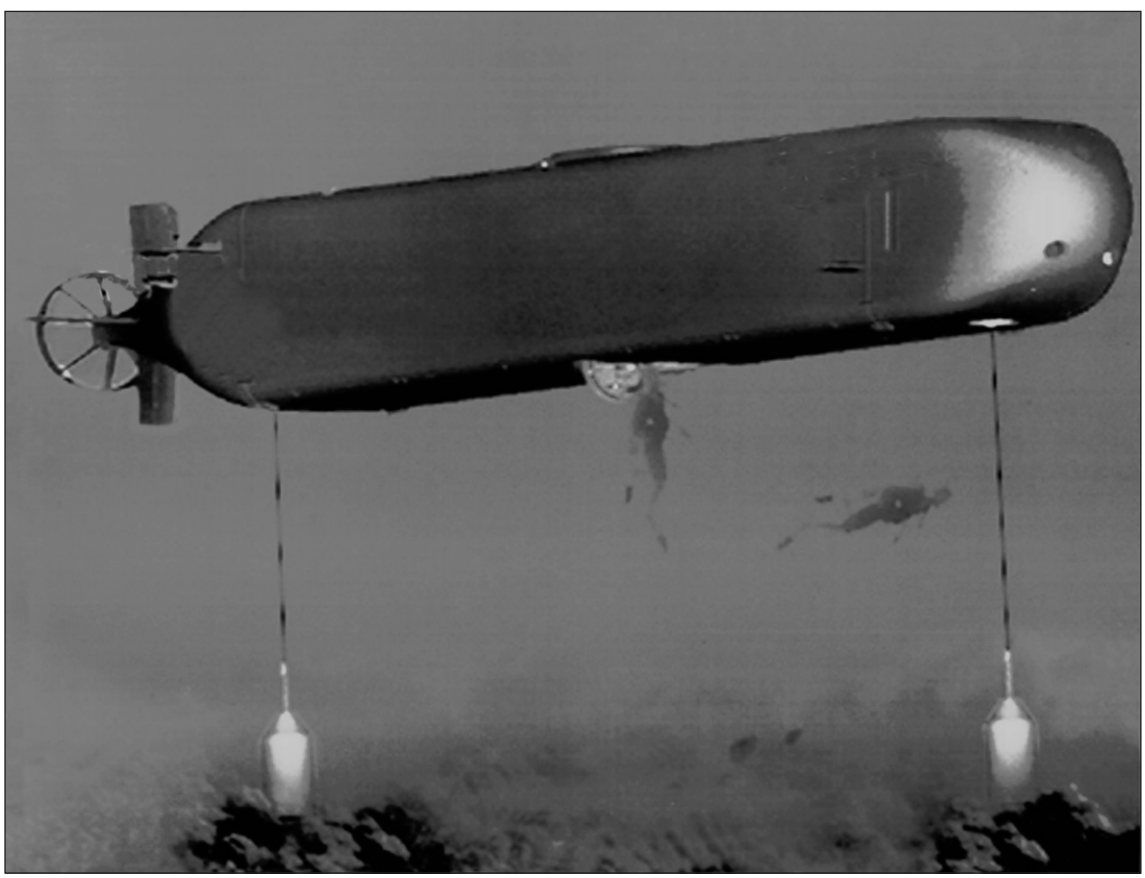

Figure 29–9. The Advanced SEAL Delivery System

decompression on air if necessary. Divers will be in a dry transport compartment during the transit to their launch area. At their launch site, they will lock out of a "moon-pool" hatch in the middle of the three compartments. There will also be a pilot and copilot in a 1 atm forward compartment, but because there will be no Master Diver, dive supervisor, or DMO on board the ASDS to calculate decompression, use of the decompression computer will be very important for these operations.

Decompression Consideration in Deep Explosive Ordnance Disposal MK 16 Diving

For free-swimming dives in which decompression is planned, such as deep EOD dives, other considerations apply. How will a diver complete the decompression if a UBA fails during a decompression stop? This eventuality requires that during decompression dives, alternative breathing sources be provided at the planned decompression stops, consisting either of additional scuba rigs or of surface-supplied gas. The MK 16 UBA contains a connector for a surface-supplied umbilical hose for this purpose. These and other operational considerations are discussed in the MK 16 UBA deployment procedures.[31,48]

Surface-Supplied Diving

Surface-supplied diving overcomes many of the disadvantages of scuba by providing an almost unlimited gas supply, increased head protection, better buoyancy control, hardwired communications, and a greater depth capability. The full facemask or helmet also provide better protection from drowning if the diver becomes unconscious. Surface-supplied diving, however, is more complex and expensive and requires considerably more support in both equipment and personnel. In addition, the diver's mobility in the water and comfort on the surface are decreased by the umbilical connection and the bulkiness of the equipment.

Three surface-supplied diving systems are currently approved for U.S. Navy surface-supplied diving: the Mark 20 Mod 0, the Mark 21 Mod 0 and Mod 1, and the Exo-26 BR systems. The Mark 20 Mod 0 UBA is a lightweight surface-supplied full-face mask (Fig. 29–10). The one-piece rubber mask includes a wide-view polycarbonate face-

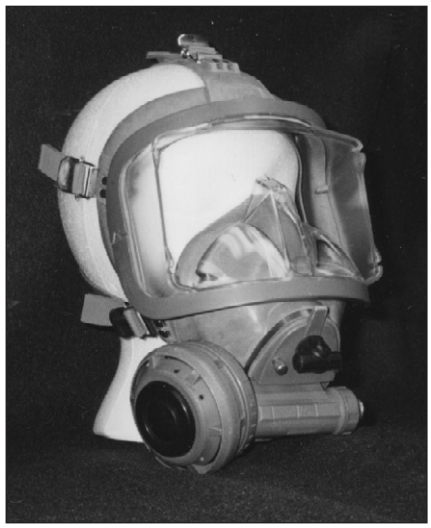

Figure 29–10. MK 20 lightweight diving mask. (Photograph courtesy of M. Knafelc.)

mask, regulator, communication system, and oronasal mask. A small positive pressure is maintained in the mask to prevent water leakage. No head protection is provided. The principal application for the Mark 20 UBA is for work in enclosed spaces, such as inspection, repair, and cleaning of ballast or mud tanks. The working depth limit for this rig is 60 fsw.[49]

The Exo-26 BR (balanced regulator) is a commercially available full-face mask manufactured by Kirby-Morgan Dive Systems. It can be configured for use in either surface-supplied or scuba modes.[49] This mask is authorized for use to depths of 190 ft with air. It has an exoskeleton of thermoplastic with a neoprene face seal. This mask's name comes from the so-called EXOthermic exhaust system that isolates the intake and exhaust chambers from each other and uses the diver's breath to warm portions of the regulator.[50] These features are designed to reduce the risk of regulator freeze-ups in cold water.

The Mark 21 system is the Navy version of the commercially available Kirby Morgan Superlite 17 system (Fig. 29–11). It consists of a rigid helmet that attaches to a neck dam. This combination keeps the head protected and dry and prevents the helmet from separating from the diver. It can be used for either air or mixed-gas (helium-oxygen) diving. Surface-supplied gas is provided to a second-stage demand regulator that has a valve to adjust demand pressure. Gas is also routed to an operator-controlled port that allows

Figure 29–11. Diver wearing a MK 21 Mod 0 UBA. (Photograph courtesy of R. Ball.)

clearing of the faceplate. The helmet is fitted with an oronasal mask to reduce gas consumption, decrease noise, and decrease dead space, thereby preventing carbon dioxide buildup. The helmet also contains a two-way communication system consisting of a microphone and a bone-conduction earphone. Line-pull communications serve as the emergency backup if primary communications fail. Both rigs require a surface supply umbilical connection that consists of an air supply hose, a communication line, a strength cable, and a pneumofathometer for depth determination. The maximum working depth for this system configured for air is 190 fsw.[49]

APPLICATIONS

Air Diving

The Mark 21 is the principal surface-supplied diving rig used in the U.S. Navy because of its increased physical and thermal protection compared with the Mark 20 system. As noted previously, surface-supplied diving requires substantially more personnel and more logistical support than does scuba diving. Common applications of surface-supplied diving include inspection and salvage, major in-water ship repairs, work in polluted waters, and underwater construction. When adequately weighted, a diver wearing a Mark 21 system can function in currents of up to 2.5 knots.[4] The air supply system for the unit must maintain a gas flow of 1.4 actual ft^3/min and must be capable of supplying 3.2 actual ft^3/min for short periods of heavy work. An emergency air supply is required when the Mark 21 is used deeper than 60 fsw and for dives on wrecks. The standard U.S. Navy air decompression tables are used for surface-supplied air diving.

Helium-Oxygen Diving

A helium-oxygen gas mixture (heliox) is employed to avoid nitrogen narcosis during dives deeper than 190 ft. In addition, the work of breathing is significantly less with helium than with air at greater depths. At greater depths, however, regardless of which inert gas is breathed, decompression obligations become significant and surface-support requirements increase. Careful planning is required to ensure an adequate gas supply for all likely contingencies during the operation. Applications for this mode of diving include deep search, inspection, salvage, and repair. The maximum working depth for standard Mark 21 helium-oxygen diving is 300 fsw, but exceptional exposure dives to 380 fsw may be performed if operational needs dictate and if the Chief of Naval Operations authorizes the dive. In general, saturation diving techniques are employed for dives deeper than 300 fsw or dives with very long bottom times requiring extensive decompression. All divers using heliox are equipped with an emergency gas supply. The U.S. Navy requires on-site recompression chamber facilities for surface-supplied mixed-gas diving.[51]

MEDICAL CONSIDERATIONS

Air Diving

As with open-circuit scuba diving, there are no diving-related illnesses for which a surface-supplied diver using the Mark 20, Mark 21, or EXO-26 BR rigs is at particularly increased risk. In older air-supplied helmet designs, carbon dioxide buildup was common because of inadequate helmet ventilation, either because of heavy work or, frequently, because of diver choice in order to reduce noise and facilitate communications. The inclusion of an oronasal mask has greatly reduced the risk of carbon dioxide buildup. Importantly, the risk of head injury is substantially reduced in the Mark 21

compared with scuba, and because of the integrated regulator design, drowning is also less likely if the diver should become unconscious.

U.S. Navy standards for air quality require compressed-air sources to be checked every 6 months at a minimum (sooner, if circumstances warrant). If compressors are not properly maintained, however, or if air inlets are not protected from exhausts, carbon monoxide poisoning or other gas contamination can occur.

Mixed-Gas Diving

Decompression Sickness. Decompression from U.S. Navy helium-oxygen dives is accomplished by having divers ascend breathing the bottom mixture up to a depth of 90 fsw. They are then switched to a mixture of 50% helium, 50% oxygen. Upon arrival at the 30 ft stop, divers are shifted to 100% oxygen. Decompression at the 30 fsw and 20 fsw stops is accomplished by having divers breathe 100% oxygen for 30 min periods separated by 5 min air breaks. The air breaks do not count toward required decompression time. For all dives, surface decompression may be used after completing the 40 fsw stop.[51]

The onset of DCS during in-water decompression is a rare event that increases in incidence with deeper or longer dives.[52] If in-water DCS occurs, procedures are available to treat the diver in the water when a better alternative, such as transfer under pressure to a saturation diving system, is not on-site. In general, the diver undergoes recompression in 10 ft increments and the partial pressure of oxygen in the diver's gas supply is optimized. If resolution is achieved with in-water recompression, the in-water decompression stops are then increased in duration.[51] Surface decompression procedures are subsequently employed, and the diver is removed from the water and treated in the chamber. If in-water DCS occurs during a saturation dive, the diver can be transferred under pressure to the chamber for definitive treatment.

Hypoxia. The most common cause of hypoxia during mixed-gas surface-supplied diving is use of a breathing mixture with an inadequate partial pressure of oxygen. For deep dives, the gas mixture used at the worksite (on the bottom) will cause hypoxia on the surface. Dives requiring a gas mixture of less than 16% oxygen in helium on the bottom are begun on air and shifted over to heliox at 20 fsw to avoid hypoxia while on the surface. The dive team must remember to shift the gas to a nonhypoxic mix on ascent if decompression does not require the shift.

Hypercarbia. Although less likely than in closed-circuit UBAs, hypercarbia may occur during mixed-gas diving, particularly at great depths. Contributing factors may include a heavy work rate, pulmonary hypoventilation as a result of the increased breathing resistance from dense gas, high oxygen partial pressures that suppress respiratory drive, and dead space in the diving rig. Dead space has been significantly reduced in the Mark 21 rig with the incorporation of the oronasal mask and demand regulator.

Contaminated Gas. Gas contamination with materials such as carbon monoxide, carbon dioxide, and oils is unusual in mixed-gas diving because gas comes from high-pressure storage tanks, not from a compressor. Careful monitoring of gas purity and meticulous maintenance of storage cylinders reduce the probability of contamination. The diver is equipped with an emergency gas supply as a backup, which is employed if contamination of the gas is suspected.

Central Nervous System Oxygen Toxicity. CNS oxygen toxicity may occur in mixed-gas diving, but it is less catastrophic than in closed-circuit scuba diving because the diver has a secure gas supply and is tethered to the surface. Oxygen toxicity is most likely during decompression while enhanced oxygen mixtures are breathed. Whenever the diver experiences convulsions or other symptoms of oxygen toxicity, emergency procedures to reduce the partial pressure of oxygen are used. In order to avoid pulmonary barotrauma, the diver should not be brought up in the water column until the tonic-clonic phase of the convulsion has subsided.

The surface-supplied HeO_2 tables were revised in 1991. Because of a relatively high incidence of DCS on some schedules, one of the changes introduced was to switch the diver from bottom mix to a 40% oxygen, 60% helium mixture at 100 fsw during ascent on dives deeper than 200 fsw. Upon arrival at 50 fsw, the divers would shift from 40% to 100% oxygen, as with the old tables, then undergo surface decompression when eligible. The 1991 revised tables were little used until about 1997 when diving intensity in-

creased in conjunction with the operational evaluation of the new Fly-Away Mixed Gas Diving System. At least five oxygen seizures were observed either at the 40 fsw water stop on oxygen or right after surfacing during those operational evaluation dives.

The tables were revised once more in 1999. The principal change in the 1999 revision was the substitution of a 50/50 heliox mix for 100% oxygen at 50 and 40 fsw to eliminate the possibility of CNS oxygen toxicity at those stops prior to surface decompression. In order to avoid a significant increase in decompression time, the switch to 50/50 is now made at 90 fsw for all dives. From the pure gas-loading perspective, this was an imperfect fix. On dives 200 fsw and deeper, the divers were already shifting to 40% oxygen at 100 fsw in the 1991 procedures, so this new procedure of shifting to 50% at 90 fsw was only a marginal gain. However, the probabilistic models (LEMDEP and LEMGEN) showed that the compensation was nearly perfect (i.e., the predicted bends rate with the 50/50 shift at 90 fsw was nearly identical to the predicted bends rate with the 1991 procedures). Both of these models contain oxygen correction factors. The net result was that although washout of inert gas was slower at 50 and 40 fsw, the influence of these oxygen factors was also less, so the end result was about the same.

The new procedures were tested operationally on the Monitor 2000 and 2001 operations with no CNS events and three cases of bends in 600 person-dives. The second major change was provision of an in-water decompression capability with a reduced risk of oxygen seizures. This procedure is used when the standard practice of removing divers from the water when eligible and finishing their decompression in a surface chamber while breathing 100% oxygen (surface decompression) is not feasible. When eligible for surface decompression, divers are brought up to 30 fsw and shifted from 50/50 to 100% oxygen rather than traveling to the surface. The 40 fsw oxygen time from the 1991 procedures is then divided into thirds. One third of the old 40 fsw time was taken on oxygen at 30 fsw and the remaining two thirds at 20 fsw, then the diver surfaced. This procedure was not tested. The third major change was lengthening the oxygen time in the chamber during surface decompression for most of the exceptional exposure dives. For this, the oxygen times of the Oceaneering International Alpha table were taken as a guide (personal communication, Dr. E. Flynn).

Military Breath-Hold Diving

Civilian breath-hold divers comprise three main groups: recreational breath-hold divers, commercial underwater harvest divers (classically, the Ama woman divers of Korea and Japan), and the extreme breath-hold divers who attempt to establish new records for dive depth. Breath-hold diving has historically been limited primarily by the break point, that is, the point during the breath-hold at which the diver can no longer voluntarily hold his or her breath. The primary hazard entailed in this activity is hypoxic loss of consciousness (HLOC). HLOC has often been called *shallow-water blackout* because divers are at increased risk for HLOC as they ascend at the end of the dive and the partial pressure of oxygen decreases, but HLOC may also occur without a change in depth if divers ignore the urge to breathe and oxygen in the lungs is depleted.

Military combat swimmers have been trained in breath-hold diving largely for the mission of attaching explosive charges to underwater beach obstacles designed to prevent landing craft from assaulting a beach. No mission-related requirement for breath-holds of a specific duration have been identified.

Other military missions entail underwater breath-holding after breathing from compressed gas sources. These include responding to scuba emergencies and underwater UBA switches during SDV operations. These are different physiologically in that the divers begin their breath-hold with a greater amount of oxygen molecules in their lungs but are also now susceptible to arterial gas embolism during the ascent if they do not exhale.

Much of the research done in this area has focused on the effect of various factors to break point. Time to break point has been found to be increased by hyperventilation,[53,54] repetitive breath-hold dives,[55] training,[53,56] rest versus exercise,[54] competition,[53] task focus,[53] warm versus cold water,[57] and oxygen breathing.[54] Although understandably few studies examine time to HLOC, the available literature suggests that breath-hold dive times of 60 sec or less are safe for immersed, exercising divers. Vann and Pollack found that exercising divers holding

their breath after a 60 sec hyperventilation had a mean breath-hold time of 86 sec.[54] Stanek and colleagues found that Ama divers had no significant decrease in hemoglobin oxygen saturation from a predive level of 98% in 92 routine dives lasting from 15 to 44 seconds.[58] Lanphier and Rahn noted no HLOC in seven trials of immersed, working divers with breath-hold durations of 60 sec or during six exposures to 80 sec.[59]

Breath-hold dives longer than 60 sec have been found to result in arterial hemoglobin desaturation and symptoms of hypoxia. Stanek and coworkers found that arterial HbO_2 saturation decreased to 73% in 15 dives by Ama divers that averaged 69 sec.[58] Vann and Pollack reported that one diver who held his breath for 164 sec on an immersed, resting dive after hyperventilating for 60 sec had "mental grayness, anxiety, and lost concentration." The diver's Pao_2 was 36 mm Hg.[54] Other authors have stated that the approximate Pao_2 at HLOC is 20 to 30 mm Hg,[59,60] which corresponds to a hemoglobin oxygen saturation of 50% to 60%. Two of Lanphier and Rahn's breath-hold immersed, working divers making 80 sec breath-holds were "confused" at the end of the exposure. The lowest surfacing HbO_2 saturation was 58% and the lowest surfacing PAo_2 was 24 mm Hg.[59]

Wong studied the pearl divers of the Tuamoto Archipelago near Tahiti.[60] These divers hyperventilate 3 to 10 min before diving and make a weighted descent. He found that their average dive time was 90 sec and the maximum breath-hold time was 155 sec. He reported that 5 of the 235 divers experienced HLOC during a 6-hour working day. A fatality occurred during this study.[60] Ferrigno reported immersed, post-hyperventilation breath-hold times of 130, 110, and 107 sec in a family of elite breath-hold divers.[61] These divers made weighted descents and pulled themselves back up the ascent line until wet-suit buoyancy took over. They were noted to have arrhythmias—junctional rhythms, premature ventricular complexes, and bigeminy—at end breath hold.

THERMAL PROTECTION IN COLD-WATER DIVING

As discussed in Chapter 13, maintaining body temperature while underwater is a physiologic challenge. Immersion removes the normally present air insulation layer. Water has a coefficient of thermal conductivity that is approximately 25 times greater than that of air, which results in a rate of heat loss in an unprotected diver in water at 27°C (80°F) that is similar to that in an unprotected person in air at 6°C (42°F).[62] To maintain thermoneutrality in an unprotected diver, the water temperature must be 35°C (91°F).[63]

Because of the diversity of its diving missions, the Navy uses several different types of thermal protection depending on the task. Neoprene wet suits are widely used, and these meet the needs of many missions. Because neoprene is compressible, it loses insulation capability with increasing depth. Variable-volume dry suits, on the other hand, permit inflation with pressurized gas to compensate for depth-related compression, thereby allowing the air insulation layer to be maintained. As a result, dry suits offer thermal protection that is superior to that afforded by wet suits, and dry suits are employed frequently in scuba operations when long or deep exposures are required. Dry suits, however, require more training in correct use and have the following additional disadvantages:

- Increase in work secondary to bulk
- Possible leakage or malfunction of inlet or outlet valves
- Need for additional weighting to maintain neutral buoyancy

Air can migrate to various parts of the suit depending on the diver's orientation in the water; for example, if the head is dependent, gas can flow up into the legs of the suit, causing loss of buoyancy control. In 1986, NMRI developed a urinary overboard dump system for dry suits, thereby solving a major drawback to the use of dry suits for extended missions.

Hot-water suits surround the diver with warm water and are employed for very cold dives or for deep diving. However, hot-water suits require more technical support because of the heating requirements. At greater depths (>100 m), heating of the respiratory gas is also required because of the significant heat loss that results from the combination of the high coefficient of heat transfer of helium and the increased density of the gas.[63–65] This respiratory loss may not be sensed and may therefore cause asymptomatic hypothermia if it is not prevented.

The U.S. Navy *Diving Manual* provides a water temperature/thermal protection chart that lists exposure times that will "challenge the average diver wearing the thermal pro-

tection listed" but that will lead to "a minimal chance of producing significant hypothermia." These times are not rules or limits. A diver wearing a wet suit has a listed time of 5 hours at 55°F, 3 hours at 45°F, and 1 hour at 35°F. A diver in a dry suit has listed times of 5 hours at 45°F and 3 hours at 35°F.[4]

Thermal protection in SDV/dry deck shelter operations has been a major operational concern for many years. Well-documented thermal protection limits are currently not available for conventional or special warfare applications. Thermal protection issues become very important in SDV operations because of the very long dive times involved and because the divers are not exercising while piloting and navigating the SDVs. The Navy has recently tested a battery-powered resistive heating suit for use by SDV crews who often make extended dives in very cold water.[66] This suit has been found not only to protect the diver from hypothermia in 35° water for an 8-hour exposure but also to prevent the performance decrements that typically result from cold stress of this magnitude.[67] This suit uses resistive-heating elements woven into a diving undergarment that can be worn under either a wet or dry suit. The large power supply required to warm two divers for 8 hours entails the use of a large, expensive SDV battery, which unfortunately restricts the use of this suit to SDV operations. Valaik's 1996 report on this topic[68] is an excellent review of thermal protection.

Thermal Factors in Warm-Water Diving

On some occasions, the diver may be exposed to water temperatures higher than 91°F. These very warm water temperatures are common in Southwest Asian waters in the summer. Heat stress is a significant concern during diving operations in this setting. In 1990, NMRI was tasked by the Commander in Chief of the U.S. Pacific Fleet to conduct studies on the feasibility of warm-water diving in anticipation of combat swimmer operations in support of Operation Desert Storm. NMRI addressed this issue in a 30-dive series in 1990. A 4-hour exposure to 100°F dry heat followed by 3 hours of mild to moderate exercise in 94°F water at 20 fsw on 100% oxygen did not result in episodes of heat injury or oxygen toxicity.[69] The results of this study were used to provide the Navy's initial guidelines on warm-water diving.[70] Based on this study and additional work done during field studies in the Persian Gulf[71] and laboratory studies done at NEDU,[72] the Naval Sea Systems Command has now established Navy-wide Interim Guidelines for Warm-Water Diving.[73] These guidelines are found in Table 29–3.

Table 29–3. Interim Guidance for Warm-water Diving Operations

Combat Swimmer (swimming at 0.8 kts or less)	
88°–94°F	Limited to canister/O_2 bottle duration
94°–97°F	Limited to 3 hours based on physiologic limits
97°–99°F	Limited to 1 hour based on physiologic limits
SDV (Resting Diver)	
88°–94°F	Limited to canister/O_2 bottle duration
94°–97°F	Limited to canister/O_2 bottle duration
97°–99°F	Limited to 2 hours based on physiologic limits

Notes

Diving in water temperatures above 99°F should not be attempted without first contacting NAVSEA OOC.
Weight losses of up to 15 lbs (or 6%–8% of body weight) due to fluid loss may occur and may affect mental and physical performance.
Divers should hydrate fully (approximately 500 mL or 17 oz) 2 hours before diving.
Fluid loading in excess of the recommended 500 mL may cause life-threatening pulmonary edema and should not be attempted.
Hydrating with water or a glucose/electrolyte beverage should occur as soon as possible after diving.
Approximately 500 mL should be replaced for each hour of diving.
Exposure limits represent maximum ccumulative exposure over a 12-hour period.
Divers should be hydrated and calorically replete to baseline weight, rested, and kept in a cool environment for at least 12 hours before a repeat exposure to warm water is deemed safe.
Until further guidance is developed regarding the measurable effects of these factors, the above limits shall serve as maximum levels of exposure.

In air, the danger of hyperthermia is closely related to the level of exercise.[74] Exertional hyperthermia is an inevitable consequence of prolonged, intense exercise in a warm environment. In elite marathoners, rectal temperatures of 39° to 40°C are common after a race. Similar considerations apply in the water, and Table 29–3 contains more permissive guidelines for divers piloting an SDV than for free-swimming divers.

Fluid and electrolyte status during warm-water dives is another area of concern. Immersion in water causes diuresis because of the redistribution of blood volume to the central circulation, resulting in increased renal blood flow and urine output. In one study, the magnitude of this diuresis was approximately 440 mL/hour of diving.[75] Fluid loading prior to a dive in an attempt to offset this diuresis is not indicated because of the possible development of pulmonary edema. Although this disorder is usually encountered in cold-water scuba divers,[76] it may also be encountered in warm-water diving and in surface swimmers. This was illustrated dramatically by the development of pulmonary edema in 8 of 30 Israeli combat swimmers after drinking approximately 5 L of fluid each prior to a swim in an effort to avoid becoming dehydrated.[77] Fluids lost during a dive should be replaced following the dive. In addition to increased susceptibility to heat injury, dehydrated persons may experience orthostatic hypotension and syncope as well as decreased aerobic capacity. Approximately 500 mL of water or a glucose/electrolyte beverage should be replaced for each hour of diving. This is especially important if an overland segment of the mission entails substantial heat stress. Salt tablets should not be used.

Additional factors to be considered during warm-water diving operations include[70,73]:

- Diver education: closed-circuit oxygen divers should not attempt to swim at high exercise levels. This is emphasized in the U.S. Navy *Diving Manual,* which notes that strenuous exercise is a potentiating factor for CNS oxygen toxicity and recommends that closed-circuit oxygen divers swim at a relaxed, comfortable pace.[23] The U.S. Navy oxygen exposure limits were established for a diver swimming at 1.3 L/min oxygen consumption. This is the exercise level attained by an experienced diver swimming underwater at a comfortable pace. Closed-circuit UBA canister duration testing uses similar work rates. Exercising at a high work rate on closed-circuit oxygen dives exposes the diver to the twin risks of CO_2 buildup and CNS oxygen toxicity, no matter what the water temperature is. On warm-water dives, the risk of hyperthermia is added.
- Conduct training dives at night, dusk, or dawn to reduce radiant heat stress that may be encountered while surface swimming or diving at shallow depths.
- Consider swimming without dive skins, if feasible, because they retain heat to some extent.
- Conduct approximately 1 week of reduced intensity diving as an acclimatization period when first diving in warm water.
- Should very heavy exercise rates be required by operational exigencies, the resulting increased risk of oxygen toxicity can be reduced by swimming at a shallower depth (10 to 15 fsw) if feasible until a normal swim pace can be resumed. This is true at any water temperature.
- Recognition and management of heat injuries should be added to dive planning and briefing.

MILITARY FITNESS-TO-DIVE STANDARDS

Medical Surveillance

The U.S. Navy requires that medical surveillance of divers be carried out or reviewed by a DMO because examiners must understand the physiologic stresses routinely imposed on divers. The DMO has a thorough understanding of diving physiology and its associated stresses and of the specific workplace hazards potentially encountered by divers. Divers must meet the standards required for military service in general, as well as additional physical qualifications required for diving duty. These standards are contained in Chapter 15 of the *Manual of the Medical Department.*[78] Examinations are required before training is initiated and at intervals thereafter. Any person who does not meet these standards is disqualified and not permitted to dive either until the condition resolves or until a waiver is granted by the Head of Undersea Medicine at the U.S. Navy Bureau of Medicine and Surgery. Reexamination by a DMO must be completed after

any significant illness or injury, particularly diving-related injuries.

Military fitness-to-dive standards are based first on medical safety considerations for the patient:

- Does the condition endanger the individual in the hazardous hyperbaric environment (e.g., symptomatic coronary artery disease)?
- Can the condition be exacerbated by hyperbaric exposures (e.g., neurologic residua from DCS)?
- Would hyperbaric exposures possibly result in complications from a preexisting condition that might not otherwise cause the individual any problems (e.g., inability to equalize middle-ear pressures)?

The other major determinant of physical standards for divers is whether they can meet the physical demands of the very specialized tasks required of divers in groups such as SEAL and EOD units. For example, good color vision is not necessary for safe diving, but the ability to discriminate red from green would be crucial for an EOD diver who proposes to disarm an explosive device.

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APPENDIX I
Pressure Conversion Table

Multiply This Unit → To obtain ↓	**kg/cm²**	**ata***	**atm†**	**Bar**	**mm Hg**	**lb/in²**	**Meters Seawater (msw)**	**Feet Seawater (fsw)**	**Pascal‡ (Pa)**
kg/cm²	1.00	1.00	1.0033	1.020	1.36×10^{-3}	7.031×10^{-2}	0.1026	3.124×10^{-2}	1.02×10^{-5}
ata	1.00	1.00	1.033	0.9807	1.36×10^{-3}	7.031×10^{-2}	0.1026	3.124×10^{-2}	1.02×10^{-5}
atm	0.9678	0.9678	1.00	1.013	1.316×10^{-3}	6.805×10^{-2}	9.921×10^{-2}	3.024×10^{-2}	0.987×10^{-5}
Bar	0.9807	0.9807	1.013	1.00	1.333×10^{-3}	6.895×10^{-2}	1.0×10^{-1}	3.064×10^{-2}	1.040×10^{-5}
mm Hg	735.5	735.5	760	750.1	1.00	51.71	75.40	23.00	7.502×10^{-3}
lb/in²	14.22	14.22	14.70	14.50	1.934×10^{-2}	1.00	1.458	0.4445	1.45×10^{-4}
Meters seawater (msw)	9.76	9.76	10.08	10.00	1.33×10^{-2}	0.6859	1.00	0.3064	0.898×10^{-4}
Feet seawater (fsw)	32.01	32.01	33.07	32.64	4.38×10^{-2}	2.250	3.264	1.00	3.264×10^{-4}
Pascal (Pa)	9.807×10^4	9.807×10^4	1.01×10^5	1×10^5	1.333×10^2	6.895×10^3	1.005×10^4	3.064×10^3	1.00

* A technical atmosphere is equal to 1 kg/cm².
† A secondary atmosphere is equal to pressure of a 760 mm column of Hg of density 13.5951 g/cm³.
‡ A pascal is defined as 1 newton/m².

APPENDIX 2

Medical Examination Form

MEDICAL HISTORY

(To be completed by applicant)

Name _____ Age _____ Sex _____ Date _____
Address _____ Phone _____

1. Have you had previous experience in diving?
   Yes _____ No _____
2. When driving through mountains or flying do you have trouble equalizing pressure in your ears or sinuses?
   Yes _____ No _____
3. Have you ever been rejected for service, employment, or insurance for medical reasons?
   Yes _____ No _____
   (If yes, explain under remarks or discuss with doctor.)
4. When was your last physical examination?
   Date _____ Results _____
5. When was your last chest x-ray examination?
   Date _____ Results _____
6. Have you ever had an electrocardiogram?
   Yes _____ No _____
   Date _____ Results _____
7. Have you ever had an electroencephalogram (brain wave study)?
   Yes _____ No _____
   Date _____ Results _____
8. Do you smoke?
   Yes _____ No _____
   If so, how much? _____
9. What sports or exercise do you regularly engage in? _____
   _____

(Check the blank if you have, or ever have had, any of the following conditions. Explain under Remarks, giving dates and other pertinent information or discuss with doctor.)

10. _____ Frequent colds or sore throat
11. _____ Hay fever or sinus trouble
12. _____ Trouble breathing through nose (other than during colds)
13. _____ Painful or running ear, mastoid trouble, broken eardrum
14. _____ Hardness of hearing
15. _____ Asthma or bronchitis
16. _____ Shortness of breath after moderate exercise
17. _____ History of pleurisy
18. _____ Collapsed lung (pneumothorax)
19. _____ Chest pain or persistent cough
20. _____ Periods of tiring easily
21. _____ Spells of fast, irregular, or pounding heartbeat
22. _____ High or low blood pressure
23. _____ Any kind of "heart trouble"
24. _____ Frequent upset stomach, heartburn or indigestion, peptic ulcer
25. _____ Frequent diarrhea or blood in stool
26. _____ Anemia or (women) heavy menstruation
27. _____ Bellyache or backache lasting more than a day or two
28. _____ Kidney or bladder disease; blood, sugar, or albumin in urine

29. _____ Broken bone, serious sprain or strain, dislocated joint
30. _____ Rheumatism, arthritis, or other joint trouble
31. _____ Severe or frequent headaches
32. _____ Head injury causing unconsciousness
33. _____ Dizzy spells, fainting spells, or fits
34. _____ Trouble sleeping, frequent nightmares, or sleepwalking episodes
35. _____ Nervous breakdown or periods of marked nervousness or depression
36. _____ A phobia for closed-in spaces, large open places, or high places
37. _____ Any neurologic or psychologic condition
38. _____ Motion sickness or nausea
39. _____ Alcoholism or any drug or narcotic habit
40. _____ Recent gain or loss of weight or appetite
41. _____ Jaundice or hepatitis
42. _____ Tuberculosis
43. _____ Diabetes
44. _____ Rheumatic fever
45. _____ Any serious accident, injury, or illness not mentioned above (describe under Remarks and give dates)
46. _____ Dental bridgework or plates
47. _____ Susceptibility to panic
48. _____ Pain from altitude or flying
49. _____ Regular use of medication (List medication under Remarks)

Remarks: _____
_____
_____
_____

_____
Signature of applicant

MEDICAL EXAMINATION

(This form and the medical history form are retained by the physician for his/her records)

A. Height _____ (in) Weight _____ (lb)
Blood pressure _____ Pulse rate _____

Vision:	Right eye	Left eye
uncorrected	_____	_____
corrected	_____	_____

B. Medical History: Is there a significant past history that would disqualify the applicant from scuba diving? (See medical history form.)
Yes _____ No _____
Remarks: _____
_____

C. Examination: (Check following items. If abnormal, give details below.)

	Normal	Abnormal
1. General appearance (including obesity, gross defects, postural abnormalities)	_____	_____
2. Head and neck	_____	_____
3. Eyes	_____	_____
4. Nose and sinuses	_____	_____
5. Ears (including otitis and perforation)	_____	_____
6. Mouth and throat	_____	_____
7. Spine	_____	_____
8. Lungs and chest	_____	_____
9. Heart	_____	_____
10. Abdomen	_____	_____
11. Inguinal ring (males)	_____	_____
12. Genitalia (males)	_____	_____
13. Anus and rectum	_____	_____
14. Extremities	_____	_____

Form continued on following page

	Normal	Abnormal
15. Skin reactions or eruptions	_____	_____
16. Nervous system	_____	_____
17. Psychiatric (including apparent motivation for diving, emotional stability, claustrophobia)	_____	_____

Explanation of abnormal findings: _____
_____
_____

D. Test Results:

All applicants:
Chest radiograph(s) _____

As indicated:
ECG _____
VC and FEV_1* _____
Audiogram _____
Hematocrit _____
Urinalysis _____
Other _____

E. Final Impression (circle one):

Approval: I find no defects that I consider incompatible with diving.

Conditional approval: I do not consider diving to be in this person's best interests but find no defects that present marked risk. I have discussed my impression with him/her.

Disapproval: This applicant has defects that, in my opinion, constitute unacceptable hazards to his/her health and safety in diving.

_____ _____
Date *Signature of physician*

(*The form below is to be completed and returned to the examinee if evidence of medical examination is required.*)

Impression (circle one):

I have examined _____ and reached the following conclusion concerning his/her fitness for diving:

Approval: I find no defect that I consider incompatible with diving.

Conditional approval: I do not consider diving to be in this person's best interests but find no defects that present marked risk. I have discussed my impression with him/her.

Disapproval: This applicant has defects that, in my opinion, constitute unacceptable hazards to his/her health and safety in diving.

Signature of physician: _____
Address: _____
Date: _____

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* VC, Vital capacity; FEV_1, forced expiratory volume at 1 second.

APPENDIX 3

Diving Medicine Physician's Kit for Remote Locations

Equipment
Sphygmomanometer
Stethoscope
Otoophthalmoscope
Oropharyngeal airway
Endotracheal tubes, scope, blade
Foley catheter, 18–22 gauge
Syringes and needles
Venous cannula
Tourniquet
Intravenous infusion sets
Scissors, disposable scalpels
Bandage materials
Ace bandages
Sterile gloves

Drugs and Fluids
Normal saline and lactated Ringer's solution
Injectable dexamethasone
Sodium bicarbonate ampules
Local anesthetic injection
Aspirin tablets
Otic Domeboro solution
Cortisporin Otic solution
Opthalmic antibiotic solution
Afrin nasal spray
Benadryl for injection and Benadryl capsules
Topical steroid cream
Topical antibiotic ointment
Hibiclens surgical soap
Antacid tablets
Ciprofloxacin 500 mg tablets
Diazepam injection
Baby ear syringe
White vinegar

APPENDIX 4

Recommended Protocol for Diabetic Management During Recreational Scuba Diving

Pre-dive Plan – General Comments

1. **Drugs:** Absolutely no alcohol or recreational drugs on the day before or the day of the dive. Diabetic divers should not be on blood sugar reducing drugs such as beta blockers.
2. **Insulin administration:** Errors must be avoided, such as reversal of AM and PM doses of insulin, reversal of regular and NPH insulin units, excessive insulin dose, improper timing of insulin administration with regard to meals.
3. **Insulin injection:** Insulin injections of the pre-dive day should be performed in the abdominal area. (Insulin absorption from an exercising limb is faster and more erratic than normal.) The injection site should be an area of the skin that is not scarred or thickened (hypertrophic fatty tissue) from previous injections.
4. **Food:** Do not alter meal and snack schedule unless directed to do so by the Protocol. Do not skip any meals or decrease daily calories; avoid fatty foods; avoid foods or fluids containing caffeine, such as coffee, tea, chocolate, and soft drinks. Multiple small snacks are recommended over a single larger one.
5. **Exercise:** Avoid strenuous activity or strenuous exercise during day or evening prior to the scheduled scuba diving activity. If the diabetic diver has a daily aerobic exercise program in place, he/she should reduce the amount of exercise by at least one-third.
6. **Fluids:** Increase normal fluid intake significantly for 24 hours prior to scheduled dive. A minimum of 8 ounces of fluid should be consumed by the diabetic diver during the pre-dive period. A 1:3 dilution with water of an electrolyte beverage such as Gatorade is recommended.
7. **Rest:** Adequate rest the night before the scheduled dive trip is essential. Eight hours of sleep is recommended.

Evening of Pre-dive Day

1. **Insulin:** Reduce evening dose of intermediate or long-acting insulin by 10%, if under loose control or 20%, if under tight control. The diabetic diver should seek the advice of his/her physician to determine the degree of diabetic control and recommended insulin management.
2. **Meal:** Normal evening meal – avoid fatty foods and caffeine.

Morning of Dive Day: Pre-dive Plan

1. **Meals:** Eat meal approximately 2 hours before planned dive. Increase meal by 200 calories or as directed by personal physician. A mixture of complex carbohydrates and proteins is recommended for these additional calories.
2. **Fluids:** Drink 16 ounces of non-caloric fluids (or more) during pre-dive period. A 1:3 solution diluted with water of electrolytic beverage, such as Gatorade, is recommended.
3. **Insulin:** Adjust AM insulin dose and/or diabetic medication for planned degree of exercise. The following recommendations may be considered: If the FBG is between 80 mg/dl and 240 mg/dl, administer AM insulin according to the following schedule:
   a. Reduce usual AM dosage of regular insulin by 50% if under tight control;
   b. Reduce usual AM dosage of regular insulin by 25% if under loose control;
   c. Reduce usual AM daily dose of intermediate or long-acting NPH insulin by 20%.

4. **SMBG (self-measured blood glucose) and Ketone Testing** (blood or urine):
   a) *Pre-meal*. Perform fasting blood glucose (FBG) just before meal.
      I. If FBG value is between 80 mg/dl and 240 mg/dl, proceed with pre-dive plans.
      II. If FBG value is below 80 mg/dl or above 240 mg/dl and/or ketone testing is positive, the planned dive activity should be terminated. Seek medical advice.
   b) *Pre-dive*. Perform 3 SMBG determinations and 3 tests for ketones during the hour prior to the dive – 1 hour pre-dive, 30 minutes pre-dive, and immediately pre-dive. If the 1-hour ketone test is negative, the 30-minute pre-dive and immediate pre-dive tests may be omitted, unless extreme hyperglycemia develops (BG above 240 mg/dl).
      I. The random blood glucose (RBG) value should be rising or stable with each successive test.
      II. The recommended blood glucose range for each time frame is:
         a. 1 hour pre-dive, 80-240 mg/dl
         b. 30 minutes pre-dive, greater or equal to previous RBG and between 120 and 240 mg/dl.
         c. Immediately before diving, greater or equal to previous RBG and between 120 and 240 mg/dl.
      III. The recommended pre-dive range is 120 to 240 mg/dl, depending on degree of exercise planned and diabetic's previous exercise experience.
      IV. If the RBG level is above 240 mg/dl and/or ketones are present, the diving activity should be cancelled. The diabetic should seek medical advice and adjust diabetic management plan accordingly.
      V. If the RBG level is decreasing during successive SMBG tests, appropriate snacks and SMBG should be continued until the RBG stabilizes within the acceptable range. If doubt exists, terminate the dive activity and adjust diabetic management plan.

First Dive of the Day

1. **Dive Plan:** The dive should comply with accepted standards for no-decompression recreational scuba diving, except that the bottom time for any dive (independent of depth) should not exceed 25 to 30 minutes. Under adverse conditions (such as strong current, cold waters, or increased work), or if unexpected physical exertion is required, then the bottom time should not exceed 20 to 24 minutes. Maximum depth should not exceed 100 fsw.
2. **Hypoglycemia Precautions:** The diabetic diver and the "informed buddy" should carry a glucose paste or honey in a squeeze bottle. If the diabetic diver should experience a hypoglycemic event underwater, one of these should be ingested after the diver and the informed buddy have made a safe ascent and established positive buoyancy on the surface.
3. **Post-dive Plan**
   a) **SMBG:** Perform random blood sugar (RBS) immediately upon completing the dive.
      I. If the RBS value is below 80 mg/dl a carbohydrate snack should be eaten immediately. Repeat RBS every 30 minutes and take carbohydrate snacks until TBS is 80 mg/dl or above. (See section on hypoglycemia below.)
      II. If RBS value is 80 mg/dl or above a small protein and/or complex carbohydrate snack is appropriate.
   b) **Fluids:** Drink at least two eight-ounce glasses of a non-caloric fluid.
   c) **Other:** Remove tight-fitting wet suit. If cold, get warm. If hot, attempt to cool down.

Surface Interval Time

1. Rest for the first 30 minutes of surface interval time.
2. Perform no strenuous activity during the entire surface interval time.
3. An SMBG should be performed if any symptoms of hypoglycemia develop and appropriate action should be taken. (See section on hypoglycemia below.)
4. Appropriate meals and snacks should be taken.

5. Omit any scheduled insulin administration between the first and second dive unless otherwise directed by personal physician.
6. Insulin pump should be reconnected after the dive unless hypoglycemia is present on SMBG. If hypoglycemia is not present, pump should be set at 50% of usual rate during the surface interval, and SMBG should be performed periodically.

Second Dive of the Day

1. Repeat steps for First Dive of the Day, and steps 3-6 listed under Surface Interval Time.
2. The diabetic diver should limit his/her scuba diving to two dives per day.

HYPOGLYCEMIA

Signs and Symptoms of Hypoglycemia

1. **Early warning signs:** Unusual hunger, headache, alteration of mood, nervousness, and/or unusual fatigue.
2. **Mild reaction:** Tremors, pounding and/or rapid heart rate, sweating, clamminess of skin, and/or extreme fatigue.
3. **Moderate Reaction:** Severe head and/or neck pain, extreme alterations of mood, irritability, and/or extreme fatigue.
4. **Severe Reaction:** Decreased awareness or responsiveness, unconsciousness, and/or convulsions.

Management of Hypoglycemia

1. If only early warning signs of hypoglycemia are observed and the SMBG is 80 mg/dl or above, look for other causes of diabetic diver's symptoms, including anxiety, sea sickness, dehydration, heat exhaustion, and/or early signs of decompression sickness (DCS).
   a) Eat an appropriate complex carbohydrate-protein snack,.
   b) Repeat SMBG in about 30 minutes.
   c) If symptoms clear and SMBG is 80 mg/dl or above, and no other contraindications are determined, then the diabetic diver may continue with his/her scuba diving activity in accordance with the Protocol.
2. If only early warning signs of hypoglycemia are evident, but the SMBG is below 80 mg/dl, the diabetic diver should:
   a) Take 10 to 15 grams of sugar, such as 4 to 6 ounces of fruit juice, 6 lifesavers, or 4 teaspoonfuls of sugar.
   b) Repeat SMBG at 30 minute intervals as needed.
   c) Continue this process until blood sugar value is 80 mg/dl or above and symptoms have cleared.
   d) Watch carefully for relapses.
   e) Eat a complex carbohydrate-protein snack. Cease all scuba diving activity and seek medical advice.
   f) Hydration is a priority.
3. If mild signs of hypoglycemia develop and SMBG is below 80 mg/dl, the diabetic diver should:
   a) Initiate same treatment as for early warning signs (Section 2.b. above).
   b) 4 glucose tablets may be substituted for sugar snack (easier to carry).
4. If moderate hypoglycemia develops and SMBG is below 60 mg/dl, the diabetic diver should:
   a) Immediately take 4 to 6 glucose tablets or large sugar snack, or teawspoonfuls of sugar.
   b) Repeat SMBG at 30 minute intervals as needed.
   c) Continue treatment with glucose tablets and interval monitoring until symptoms clear and SMBG value is 80 mg/dl or above.

   d) Watch carefully for signs of relapse. Recovery is usually longer.
   e) Cease all scuba diving activity and seek medical advice.
   f) If the diabetic diver is unable to take sugar snack or glucose tablets, then Glucagon 1 mg should be given subcutaneously or intramuscularly in the shoulder or anterior thigh. A positive response should be noted in 10 to 15 minutes. Proceed with sugar snack, etc.
   g) Observe SMBG carefully for rebound hyperglycemia.
   h) Do not give the diabetic diver food or fluids by mouth until he/she is alert enough to swallow, in order to avoid possible aspiration into the lung and/or choking.
5. If severe hypoglycemia develops (the SMBG value will generally be below 60 mg/dl), the diabetic diver's informed dive partner should:
   a) Immediately administer Glucagon 1 mg subcutaneously or intramuscularly to the diabetic diver, as described above.
   b) If available, and a trained medical person is present, then intravenous glucose and fluid should be given.
   c) Seek emergency medical service and advice.
   d) Cease all scuba diving activity.

DIVE KIT FOR THE DIABETIC SCUBA DIVER

1. Watertight container to hold the kit, clearly marked.
2. The Protocol.
3. Personal medical history.
4. Personal physician name and phone number.
5. SMBG monitor and glucose oxidase sticks, with instructions.
6. Glucose tablets (or substitute).
7. Glucagon for subcutaneous or intramuscular injection.
8. Instructions and supplies for administering Glucagon.
9. Glucose paste or honey in squeeze bottle.
10. Diabetic identification tag or bracelet. Identification should be worn during diving activity.

DIVE LOG FOR THE DIABETIC SCUBA DIVER

1. Log all dives.
2. Keep details of insulin administration, SMBG, environmental conditions, and any adverse reaction.

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