

Diving Physiology



SECTION	PAGE	SECTION	PAGE
3.0	GENERAL		3- 1
3.1	SYSTEMS OF THE BODY		3- 1
3.1.1	Musculoskeletal System		3- 1
3.1.2	Nervous System		3- 1
3.1.3	Digestive System.....		3- 2
3.2	RESPIRATION AND CIRCULATION		3- 2
3.2.1	Process of Respiration		3- 2
3.2.2	Mechanics of Respiration		3- 3
3.2.3	Control of Respiration.....		3- 4
3.2.4	Circulation		3- 4
3.2.4.1	Blood Transport of Oxygen and Carbon Dioxide		3- 5
3.2.4.2	Tissue Gas Exchange.....		3- 6
3.2.4.3	Tissue Use of Oxygen		3- 6
3.2.5	Summary of Respiration and Circulation Processes		3- 8
3.2.6	Respiratory Problems		3- 8
3.2.6.1	Hypoxia		3- 8
3.2.6.2	Carbon Dioxide Toxicity.....		3- 9
3.2.6.3	Hyperventilation		3-10
3.2.6.4	Shallow Water Blackout.....		3-11
3.2.6.5	Carbon Monoxide Poisoning		3-11
3.2.6.6	Excessive Resistance to Breathing		3-12
3.2.6.7	Lipoid Pneumonia		3-12
3.3	EFFECTS OF PRESSURE		3-12
3.3.1	Direct Effects of Pressure During Descent		3-13
3.3.1.1	Ears		3-13
3.3.1.2	Sinuses		3-15
3.3.1.3	Lungs		3-15
3.3.1.4	Eyes		3-16
3.3.2	Direct Effects of Pressure During Ascent.....		3-16
3.3.2.1	Lungs—Pneumothorax		3-16
3.3.2.2	Lungs—Mediastinal Emphysema		3-17
3.3.2.3	Lungs—Subcutaneous Emphysema		3-18
3.3.2.4	Arterial Gas Embolism		3-18
3.3.2.5	Stomach and Intestine		3-19
3.3.2.6	Teeth		3-19
3.3.2.7	Contact Lenses		3-19
3.3.3	Indirect Effects of Pressure During Descent		3-20
3.3.3.1	Inert Gas Narcosis		3-20
3.3.3.2	High Pressure Nervous Syndrome (HPNS)		3-21
3.3.3.3	Oxygen Toxicity		3-21
3.3.3.3.1	CNS: Central Nervous System		3-21
3.3.3.3.2	Lung and “Whole Body”		3-21
3.3.3.3.3	Variations In Tolerance		3-22
3.3.3.3.4	Benefits of Intermittent Exposure.....		3-22
3.3.3.3.5	Concepts of Oxygen Exposure Management		3-22
3.3.3.3.6	Prevention of CNS Poisoning		3-22
3.3.3.3.7	The “Oxygen Clock” or “O ₂ Limit Fraction”		3-22
3.3.3.3.8	Prevention of Lung or Whole-Body Toxicity		3-24
3.3.4	Indirect Effects of Pressure During Ascent		3-24
3.3.4.1	Inert Gas Elimination		3-24
3.3.4.2	Decompression Sickness		3-26
3.3.4.3	Treatment Tables		3-28
3.3.4.4	Failures of Treatment.....		3-28
3.3.4.5	Counterdiffusion.....		3-29
3.3.4.6	Aseptic Bone Necrosis (Dysbaric Osteonecrosis).....		3-29
3.3.4.7	Patent Foramen Ovale		3-29
3.3.4.8	Pregnancy and Diving		3-30
3.4	HYPOTHERMIA/HYPERTHERMIA		3-30
3.4.1	Effects of Cold		3-30
3.4.2	First Aid for Hypothermia.....		3-30
3.4.3	Thermal Protection		3-31
3.4.4	Thermal Stress Irrespective of Ambient Temperature.....		3-32
3.4.5	Survival in Cold Water		3-32
3.4.6	Overheating and Hyperthermia		3-33
3.4.7	Types of Heat Stress		3-33
3.5	DRUGS AND DIVING		3-34
3.5.1	Prescription Drugs		3-34
3.5.2	Smoking		3-34
3.5.3	Illicit Drugs and Alcohol		3-35

Diving Physiology

3

3.0 GENERAL

This section provides an overview of how the human body responds to the varied conditions of diving. Diving physics, explained in the previous chapter, does not directly determine how the body reacts to forces on it. Despite many external physical forces, the body normally maintains internal functions within healthy ranges. Past a point, however, the body cannot maintain healthy physiology, which may result in medical problems. A knowledge of diving physiology contributes to diving safety and enables a diver to describe diving-related medical symptoms when problems occur.

3.1 SYSTEMS OF THE BODY

The body tissues and organs are organized into various systems, each with a specific job. These systems are as follows:

3.1.1 Musculoskeletal System

Bones provide the basic structure around which the body is formed (see Figure 3.1). They give strength to the

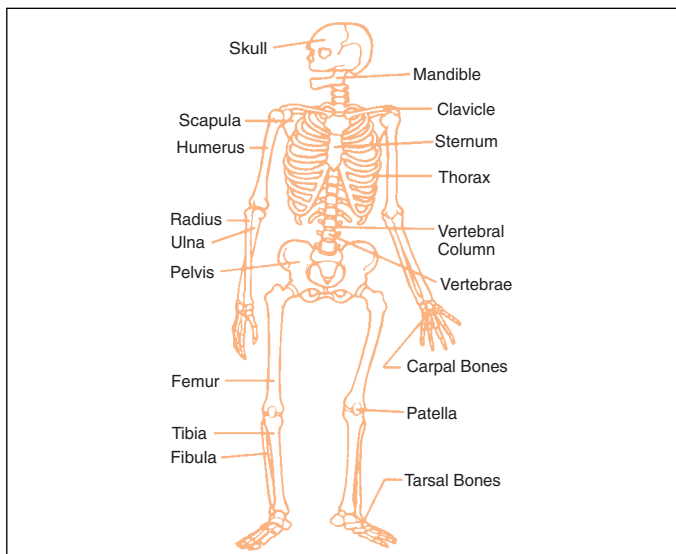


FIGURE 3.1
Skeletal System

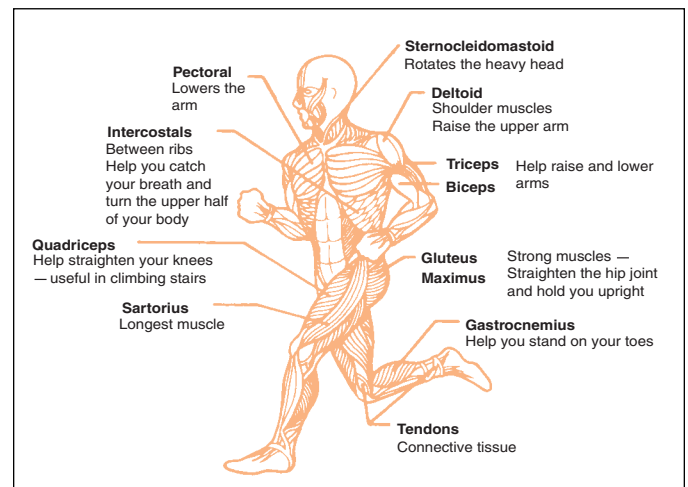


FIGURE 3.2
Muscular System

body and protection to the organs. Bones are the last tissues to become saturated with inert gases. The muscles make the body move — every movement from the blinking of an eyelid to breathing (see Figure 3.2). Additionally, muscles offer protection to the vital organs. Some muscles are controlled consciously, while others, like the heart, function automatically.

3.1.2 Nervous System

The nervous system includes the brain, spinal cord, and a complex network of nerves. Collectively, the brain and spinal cord are called the central nervous system (CNS). All nerves originate in the brain or spinal cord. The basic unit of the nervous system is the neuron (see Figure 3.3), which has the ability to transmit electrochemical signals as quickly as 350 feet per second. There are over ten billion nerve cells in the body, the largest of which has fibers that reach all the way from the spinal cord to the big toe (three feet or more). The brain uses approximately 20% of the available oxygen supply in the blood, at a rate ten times faster than other tissues, and its cells will begin to die within four to six minutes if deprived of that oxygen supply.

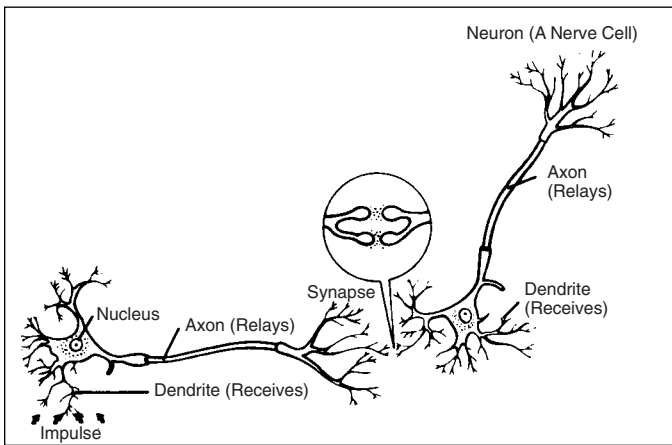


FIGURE 3.3
A Nerve Cell

3.1.3 Digestive System

The digestive system consists of the stomach, small and large intestine, the salivary glands, pancreas, liver, and gall bladder (see Figure 3.4). The digestive system converts food to a form that can be transported to and utilized by the cells. Through a combination of mechanical, chemical, and bacteriological actions, the digestive system reduces food into soluble basic materials such as amino acids, fatty acids, sugars, and water. These materials diffuse into the blood and are carried by the circulatory system to all of the cells in the body. Non-digested material passes out of the body as feces.

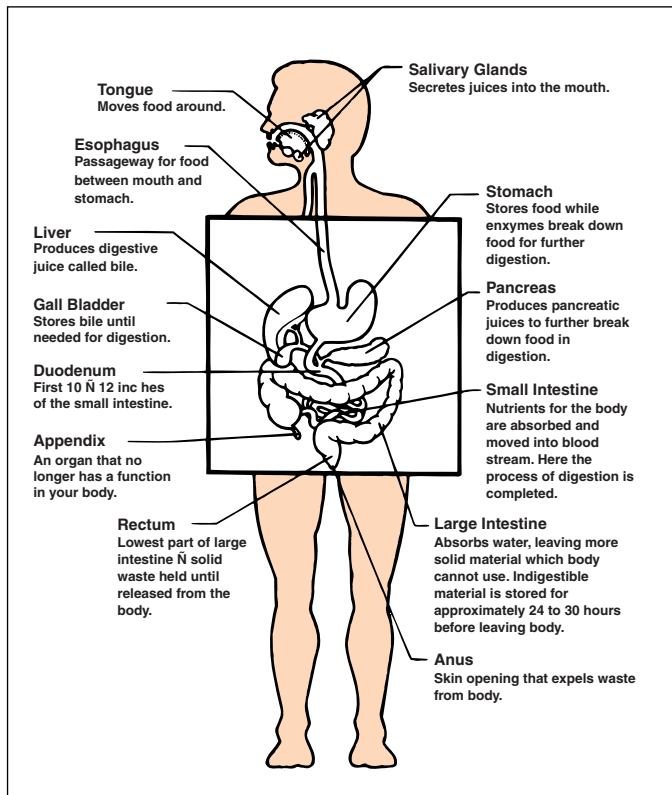


FIGURE 3.4
Digestive System

3.2 RESPIRATION AND CIRCULATION

Two body processes most noticeably affected during diving are respiration and circulation (see Figure 3.5).

3.2.1 Process of Respiration

Respiration is the process of getting oxygen (O₂) into the body, and carbon dioxide (CO₂) out. Inspired air is warmed as it passes through the nose, mouth, and throat. This warm air continues down the trachea, into two bronchi at the top of each lung. These bronchi divide and re-divide into ten bronchopulmonary branches which make up the five lobes of the lungs: three for the right lung; the left lung has only two lobes to allow room for the heart. In each lobe, the branches divide into even smaller tubes called bronchioles. The purpose of all these branches is to provide a large amount of gas-transfer tissue in a small area. Unfolded, the bronchio-pulmonary branches would be enormous—between 750 and 860 square feet each (70 and 80 square meters).

The larger bronchioles have a muscular lining that can squeeze or relax to regulate how much air can pass. Special cells lining the bronchioles secrete mucus to lubricate and moisten the lungs so that breathing doesn't dry them, and to trap dust and other particles. Trapped particles are then removed by coughing or swallowing. Irritating stimuli trigger the secretion of too much mucus into the bronchioles; this congests air passages, creating respiratory conditions that cause problems when diving. Other stimuli can trigger bronchiole-muscle spasms, reducing the amount of air breathed in a given time. When spasms occur frequently, asthma is suspected.

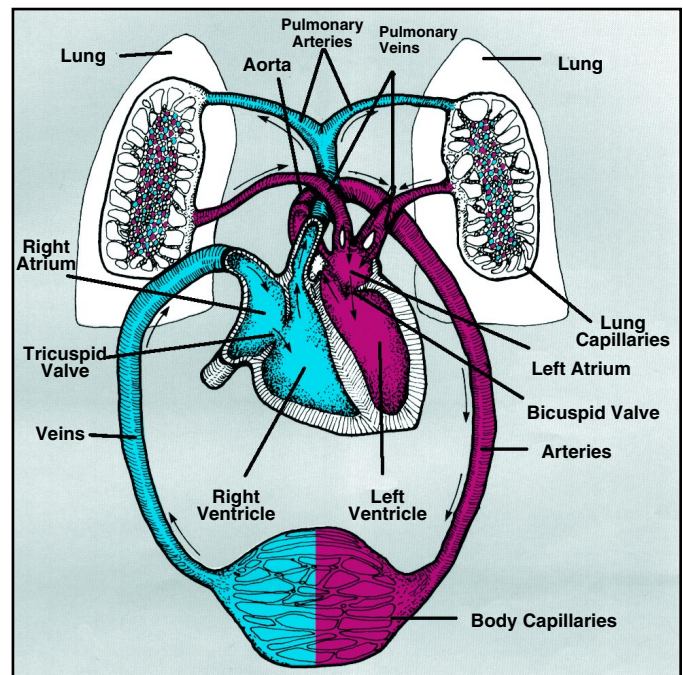


FIGURE 3.5
Respiratory and Circulatory System

The bronchioles are honeycombed with pouches, each containing a cluster of tiny air sacs called alveoli. Each alveolus is less than .04 inch (1mm) wide. Surrounding each alveolus is a network of tiny blood vessels called capillaries. It is in the capillaries that dissolved oxygen and carbon dioxide are exchanged between the lungs and the bloodstream. The walls of alveoli and their capillaries are only one cell thick, semi-permeable, and close together so gas transfers easily. There are about 300 million alveoli in each lung, so gas transfers quickly. This process is shown in Figures 3.6 and 3.7.

3.2.2 Mechanics of Respiration

The volume of air breathed in and out is called tidal volume; like the tide, it comes in and goes out. Tidal volume at rest averages about 0.5 liter.

Normal inhalation requires the contraction of the inspiratory rib muscles (external intercostals) and the diaphragm muscle below the lungs. As the chest cavity enlarges, it pulls on the double membrane around the lungs called the pleura. In turn, the pleura pulls on the lungs, enlarging them. As lung volume increases, pressure within decreases allowing air to flow into the lungs to equalize pressure. To exhale, the diaphragm and inspiratory muscles relax, pushing on the lungs by elastic recoil and pushing air out.

Normal inspiration can be increased by adding contraction of some of the neck muscles (accessory muscles), and more rib muscles. Exhalation can be increased by contracting the abdominal wall and the expiratory muscles of the chest (internal intercostals).

Vital capacity refers to the largest volume exhaled after maximum inhalation. This volume is usually determined by size and age; larger individuals usually have higher vital capacity. Vital capacity alone does not determine capacity for exercise, the ability to breathe adequately during exertion, or the ability to deliver oxygen to the blood.

Additional air that can be inhaled after a normal inspiration is the inspiratory reserve. Inspiratory reserve averages three liters. After exhaling normally, one can forcefully exhale another liter or so of air, called the expiratory reserve. Even after forcefully expelling all the air possible, there is still just over a liter in the lungs. This residual volume keeps the lungs from collapsing.

Besides exchanging oxygen and carbon dioxide, lungs have several other interesting functions, including filtering. Lungs are directly exposed to all the pollutants, dust, smoke, bacteria, and viruses in the air. Particles not trapped by bronchiole mucus enter the alveoli. There, special cells called alveolar macrophages engulf or destroy them. Lungs also filter the blood supply, removing harmful particles, such as fat globules and small blood clots. Special cells and enzymes break down and remove the trapped particles. The lungs even filter gas bubbles generated during diving ascents, preventing bubbles, in most cases, from going back to the heart and being pumped from there to the rest of the body. However, too many bubbles will overwhelm this pulmonary filter.

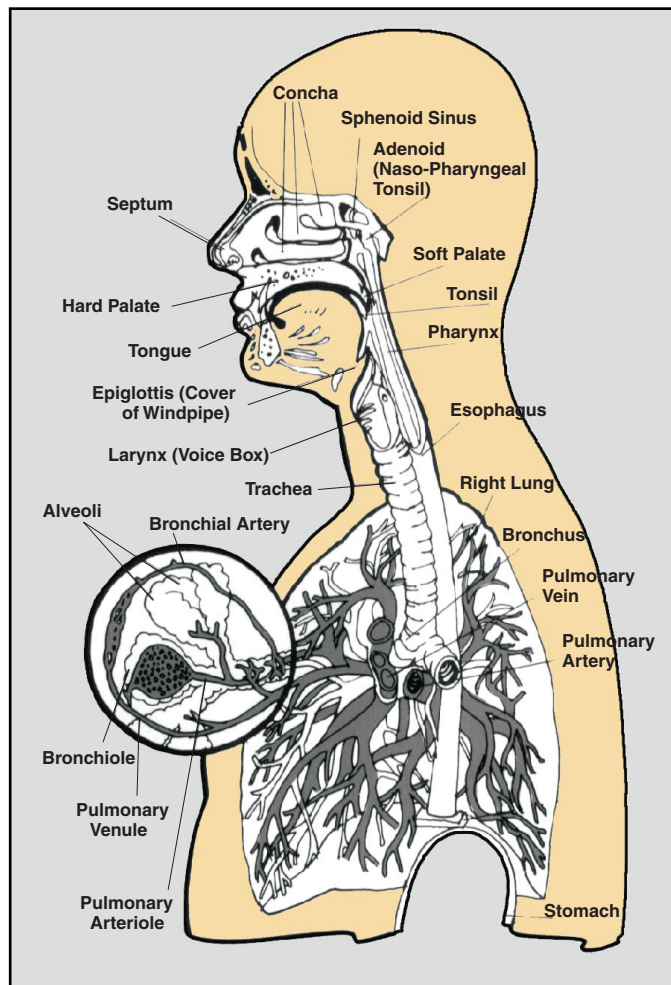


FIGURE 3.6
Process of Respiration

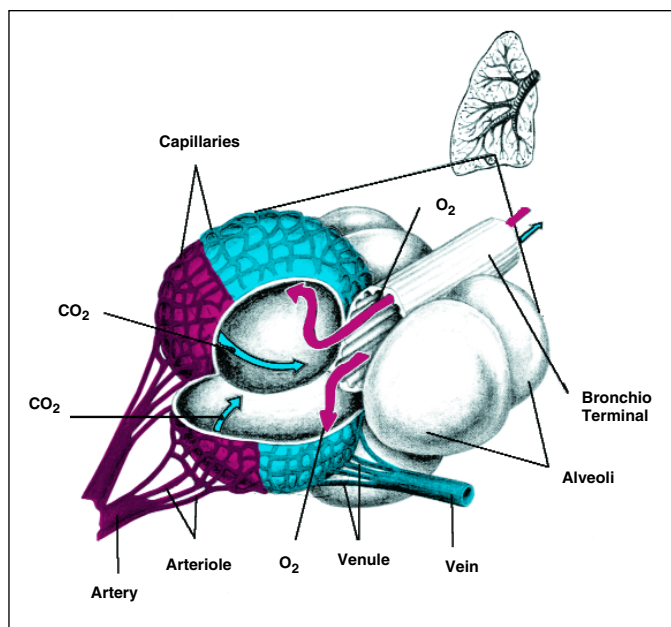


FIGURE 3.7
Lung Air Sacs (Aveoli)

3.2.3 Control of Respiration

At rest, a person normally breathes about a 0.5 liter of air, 12 to 20 times a minute. During exertion or emotional stress, rate and volume increase many times. The rate slows during rest and deep relaxation.

The body has many self-regulatory mechanisms to keep internal levels of oxygen and carbon dioxide the same, even during heavy exercise. Although tissues use oxygen during exertion, net blood levels do not fall. Although the body produces carbon dioxide during exercise, levels do not ordinarily rise. The body makes the necessary adjustments by changing breathing patterns.

What is called “the respiratory center” is several separate groups of nerve cells in the brain stem, each regulating different respiratory events. Every few seconds, bursts of impulses from these main nerve centers signal the respiratory muscles, and separately determine rate, pattern, and depth of inspiration and expiration.

As the primary stimulus during exercise, rising production of CO₂ stimulates receptors in the respiratory center, resulting in greatly increased inspiratory and expiratory signals to the respiratory muscles. Ventilation increases to remove (“blow off”) CO₂; this immediately restores the blood CO₂ level to normal and keeps it there throughout exercise.

Oxygen, as the secondary stimulus, does not directly affect the respiratory center to any great degree. Oxygen acts on cells called chemoreceptors in two places in the heart. These chemoreceptors transmit signals to the brain’s respiratory controls.

An excessive ventilatory rate during emotional stress such as fear, or during deliberate hyperventilation, can lower CO₂ too far. Low CO₂ reduces the drive to breathe, sometimes so low that one can become oxygen deficient (hypoxia), or even unconscious (see Section 3.2.6.3). An insufficient ventilatory rate may occur when breathing resistance is high or there is a high partial pressure of oxygen, both found in certain diving situations. These can contribute to carbon dioxide toxicity (hypercapnia) (see Section 3.2.6.2).

3.2.4 Circulation

Oxygen from air in the lungs needs to get to the tissue, and carbon dioxide from the tissue needs to get back to the lungs. Oxygen in the alveoli dissolves and transfers into the blood through the millions of alveolar capillaries. These capillaries join, forming fewer but larger venules; the venules join, forming the large pulmonary vein; and the pulmonary vein carries the oxygenated blood to the left side of the heart.

The left side of the heart pumps blood into the aorta, and through a series of large muscular blood vessels called arteries (see Figure 3.8). Arteries branch into many progressively smaller arterioles. The muscular arteriole walls squeeze or relax to regulate how much blood can pass. Arterial constriction and dilation is useful to direct blood

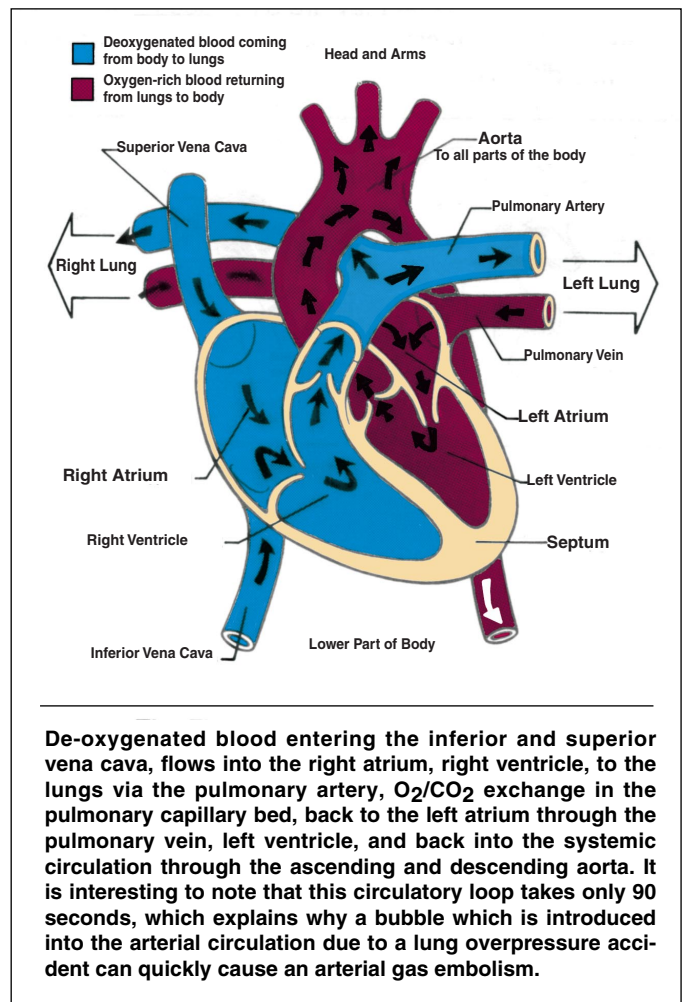


FIGURE 3.8
Flow of Blood Through the Heart

into needed areas, away from others, and to increase and decrease resistance to blood flow, which is a factor in controlling blood pressure. Arterial pressure also contributes to the force that distributes blood through the body.

Arterioles increase in number and decrease in size until they become capillaries—the human body has nearly 60,000 miles (100,000 km) of them. Capillaries are so narrow that blood cells can only go through them single file. The number of capillaries in any particular part of the body depends on how metabolically active that part is. Muscles may have approximately 240,000 capillaries per square centimeter. The lens of the eye has none. Only about five to ten percent of capillaries flow with blood at any given time. The body contains a finite amount of blood, therefore it must be regulated to meet the body’s varying needs. When there is insufficient blood to meet the body’s needs, problems arise. For example, if blood fluid volume depletes from dehydration or can’t keep up with the competing demands of exercise and cooling in the heat, the body is adversely affected.

Dissolved oxygen transfers easily through the capillary walls to the cells, and carbon dioxide transfers from cells to capillaries. The CO₂-loaded blood continues through all the capillaries, onward to venules, then veins, and back to the heart. The heart pumps the blood to the lungs where CO₂ is removed and more oxygen is received. A small amount of oxygen and nutrient-rich blood reaches the lungs directly from the left side of the heart; the lungs, like all other tissues, need oxygen to function.

Another part of the circulatory system is the lymph system. As blood passes through capillary networks, pressure inside capillaries pushes fluid out of the capillaries. About one percent of the liquid is not resorbed and remains in the spaces between capillaries and cells. The lymph system drains this extra fluid so it can return to the blood vessels to maintain proper blood volume. The lymph system also filters cell debris and foreign substances in the blood, and makes and stores infection-fighting white cells (lymphocytes) in bean-shaped storage bodies called lymph nodes. Whenever lymphocytes collect to fight invaders, the swollen piles of them can be felt in the lymph nodes.

3.2.4.1 Blood Transport of Oxygen and Carbon Dioxide

Blood transports food, water, disease-fighting cells, chemicals, messages, waste, and repair kits throughout the body. This section focuses on the blood's role in bringing oxygen to the body and carbon dioxide back to the lungs.

Blood is mostly water. Oxygen and carbon dioxide don't dissolve well in water, particularly in warm water, as in the body. As a result, at sea level pressure, only a small amount of oxygen dissolves in blood plasma (the part of blood without cells). The oxygen-carrying problem is solved with a red protein molecule called hemoglobin found inside red blood cells. Red blood cells carry far more oxygen with hemoglobin than they could without it. Up to four oxygen molecules loosely attach to each hemoglobin molecule to form oxyhemoglobin. At sea level, about 98 percent of the oxygen in blood is carried by hemoglobin.

A hemoglobin molecule with four oxygen molecules bound to it looks red, while hemoglobin without bound oxygen is so dark-red that it looks blue. This is why oxygenated (arterial) blood looks red, and deoxygenated (venous) blood looks blue. It is also why, if all of the blood is deoxygenating from a serious injury or disease process, the victim can look blue; this is called cyanosis, from the word root *cyan*, meaning blue.

Carbon dioxide is easier to transport in the blood than oxygen; it can be transported in higher quantity, and in more ways (see Figure 3.9). Dissolved CO₂ diffuses out of cells into capillary blood. A small amount stays in the dissolved state in blood plasma all the way to the lung. Hemoglobin can loosely bond a small amount, and when combined, it is called carbaminohemoglobin. An even smaller amount of CO₂ can bond with plasma proteins. These three ways are minor and slow.

The bulk of CO₂ (about 70%) reacts quickly with water inside red blood cells to form first the weak, unstable

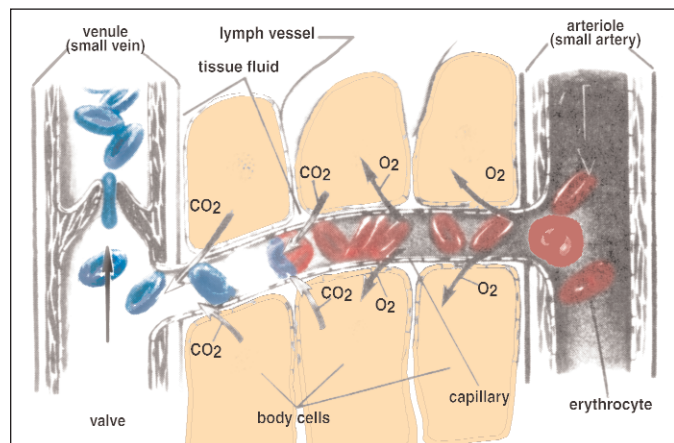


FIGURE 3.9
Carbon Dioxide Exchange

carbonic acid (H₂CO₃), and then, just as quickly (another small fraction of a second) loses hydrogens to become bicarbonate ions (HCO₃⁻), many of which diffuse into the plasma where it is transported to the lungs. Bicarbonate is alkaline, and so it is a buffering agent in the blood against acids, such as carbonic acid. Hemoglobin also functions as a powerful acid-base buffer and scavenges the acidic hydrogen ions. These are useful reactions in the body. Acid from carbon dioxide and its reactions may form in great quantities, yet still not build to unhealthy levels.

Ordinarily, the reaction of changing carbonic acid to bicarbonate ions would take seconds to minutes—too slow to be useful, so an enzyme called carbonic anhydrase inside red blood cells decreases the reaction time by a factor of 5,000 times so that great amounts of CO₂ can react with water, even before blood leaves the capillaries on the way back to the lung. Drugs called carbonic anhydrase inhibitors block the reaction of carbonic anhydrase, slowing CO₂ transport so that tissue levels rise. Carbonic anhydrase inhibitors are used to combat glaucoma, fluid retention, and altitude sickness.

Carbonic acid is used to carbonate soft drinks. Just as bicarbonate in soda releases carbon dioxide gas when a pop can is opened, bicarbonate in blood becomes carbonic acid again, releasing carbon dioxide into the alveoli so that CO₂ can be exhaled. The difference between the soft drink and the body is that the reaction to release carbon dioxide in soda has no catalyst to speed it up. Though seemingly fast, it is far too slow to keep one alive if it occurred at the same rate in the body. The lungs have enzymes to speed the reaction.

Carbon dioxide is also released in the lung by hemoglobin. When hemoglobin arrives in the alveolar capillaries with excess carbon dioxide, it first wants to pick up new oxygen. The oxygen makes the hemoglobin a stronger acid. Having just become more acidic, hemoglobin does not want the existing acid from the acidic carbon dioxide any more, so it releases it. This effect, called the Haldane Effect, means that picking up oxygen in the lung

promotes releasing carbon dioxide. The reverse is also true—as hemoglobin picks up carbon dioxide in the body, it makes the hemoglobin more acid, so it wants to release its stores of oxygen right then, which is an important factor in oxygen delivery to the cells. The Haldane Effect is named for Scottish-born British physiologist John Scott Haldane, who also co-developed the first algorithm to estimate amounts of inert gas absorbed and released by the body. Many modern decompression tables are based on his work.

3.2.4.2 Tissue Gas Exchange

Blood flow is not the only determinant of how much oxygen reaches the body. How much oxygen the blood releases to cells, and how much carbon dioxide it removes, is determined by variable, yet tightly regulated processes.

Cells withdraw oxygen from the blood. By the time blood returns to the lungs, oxygen pressure is low. Oxygen in the air in the lungs travels toward the blood through a simple gradient of higher to lower pressure. Now it is blood with higher oxygen pressure. Oxygenated blood travels back to oxygen-depleted tissues. Gas transfers via that pressure gradient to the lower pressure areas of the body.

Meanwhile, cells have been producing carbon dioxide. Body CO₂ concentration is higher than blood concentration. CO₂ travels from tissue to blood, then blood to lungs, down its own gradient. Gas exchange of carbon dioxide and oxygen occurs quickly and easily, so that tissue levels remain in set ranges, even though blood rushes through the body, and even with the high demands of exercise.

The body also controls oxygen delivery; it does not simply accept all the oxygen provided by the gradient. One regulation mechanism involves the small blood vessels. Oxygen is a vasoconstrictor. With high oxygen pressures during diving, small blood vessels constrict, thus reducing the oxygen delivered through vascular beds.

Another control mechanism is the hemoglobin-oxygen buffer system. Hemoglobin does not just carry oxygen and blindly deliver it to the cells. Hemoglobin regulates how much oxygen it releases. With low surrounding oxygen partial pressure, at altitude or other low oxygen states, for example, hemoglobin releases more than usual. With increased oxygen pressure, as during diving, hemoglobin releases less. Within limits (though one breathes higher or lower than normal pressure oxygen), hemoglobin still delivers oxygen to the body tissues at almost normal pressure. The lungs get exposed to too much or too little oxygen, but the rest of the body does not.

However, above and below a range of about half normal pressure at moderate altitude to many times normal at depth, the body can't compensate. See Section 3.3.3.3 on Oxygen Toxicity for effects of excess oxygen.

3.2.4.3 Tissue Use of Oxygen

The body uses some of the oxygen supplied to it, but not all, even during heavy exercise. At rest, the body inhales approximately 21 percent oxygen, and exhales about 16 percent. This is why mouth-to-mouth resuscitation can work. Exhaled air has sufficient O₂ to benefit the hypoxic victim. During exercise, working muscles need more oxygen; so, the blood vessels redistribute blood flow, the blood releases more oxygen, and the working cells extract more of the oxygen from the blood supply (see Figure 3.10). The better shape one is in, the more oxygen the body can deliver and extract. The amount of oxygen taken up by the body, the oxygen consumption, is a means of measuring the body's metabolism and energy production. Usually about 25% of the oxygen used by the body is available for muscular activity; the balance produces heat and supports other metabolic functions.

During exercise, heart rate and the force of the heart beat increase. Blood pressure rises. Hemoglobin distributes nitric oxide, which controls the width of the blood vessels. Blood vessels constrict in areas of the body not using as much, such as the digestive tract, spleen, liver, and non-working muscles. Contraction of arteriolar muscles constricts the arteriole, reducing the amount of blood entering the capillary bed. Arteriolar smooth muscle cells form sphincters, called precapillary sphincters, at selected places in the capillary bed to shut off blood flow. Every capillary bed has one capillary with no sphincter, called the thoroughfare channel. It stays open all the time, allowing some blood passage to maintain normal functioning.

Blood expelled from low-demand areas increases blood flow to areas with high demand for oxygen supply and for carbon dioxide and waste removal. In these areas, the arteriolar muscular lining relaxes to allow more blood to enter.

Unlike other areas of the body with varying blood supply, the brain always needs a steady supply of oxygen. If circulation slows or stops, consciousness may be lost in seconds, and irreparable brain damage may occur within four to six minutes (see Section 3.2.6.1).

Aerobic fitness is the ability of lungs, heart, and blood vessels to deliver oxygen, and the ability of the muscles and other cells to extract and use it. Aerobically fit people can deliver, extract, and use more oxygen when exercising and are able to do more aerobic exercise. Average exercise increases the amount of oxygen needed by the active tissues by about ten times. Heavy exercise can increase it to around twenty times, depending on the aerobic fitness. The better aerobic shape one is in, the more work the body can do without reaching its own maximum oxygen-processing ability. World-class athletes have reached over 30 times their resting rate. Merely breathing in more oxygen does not affect how much one can use for exercise. One has to increase their ability to deliver, extract, and use oxygen. Supplying more oxygen does not improve one's fitness. Only regular aerobic exercise will make the necessary changes in the body.

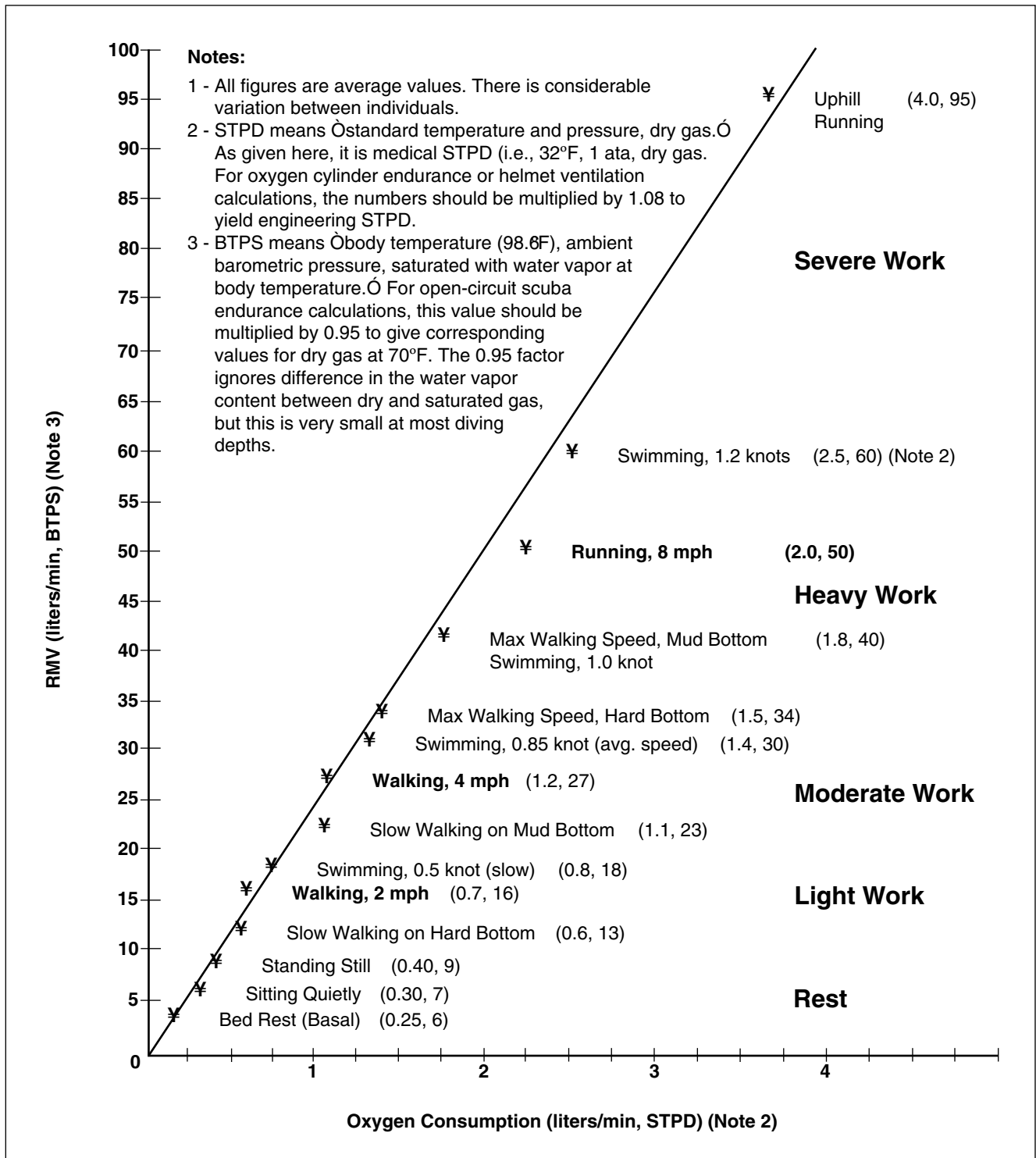


FIGURE 3.10
Oxygen Consumption and RMV at Different Work Rates

Aerobic fitness is not the only fitness needed for life activities. In rapid-onset, short duration, and intense activity, the body uses special stored fuel and glucose, not oxygen. Because these two fuels are not oxygen-using (aerobic) systems, they are called anaerobic. These two anaerobic systems are utilized for breath-hold diving, swimming against strong currents, sprints, hauling out of the water in full gear, or rescuing a heavy buddy. Regularly exercising at high speed and intensity for short bouts improves one's anaerobic capacity.

3.2.5 Summary of Respiration and Circulation Processes

The processes of respiration and circulation include six important, continuous phases:

1. Breathing air into the lungs (ventilation)
2. Oxygen and carbon dioxide exchange between air in the lung alveoli and blood
3. Oxygen transport by blood to the body tissue
4. Releasing oxygen by blood to cells, and extraction by body cells
5. Use of oxygen in the cells by combining oxygen with fat and carbohydrates to generate energy and produce waste products including carbon dioxide
6. Carbon dioxide transport by blood back to the heart, then lungs, where it diffuses into the lungs and is breathed out of the body

3.2.6 Respiratory Problems

3.2.6.1 Hypoxia

The brain requires constant oxygen to maintain consciousness, and ultimately, life. The brain is subject to damage when it is deprived of oxygen for more than four to six minutes, as can happen in heart failure when the blood supply to the brain is interrupted, in drowning, asphyxia, if breathing stops and the lungs receive no oxygen, or if the oxygen partial pressure in the lungs is insufficient. An inadequate supply of oxygen is known as hypoxia, which means low oxygen and can mean any situation where cells have insufficient oxygen. Hypoxia may result from several situations:

- Breathing mixtures that may be low in oxygen such as in seafloor or surface-based saturation systems or rebreathers
- Ascending to high elevation
- Convulsing under water from an oxygen-toxicity event
- Breathing the wrong gas; for example, mistaking the argon supply for dry suits for a breathing gas supply
- Breathing gas from a scuba cylinder that has been stored with a little water in it for long periods — the oxidation reaction (misting) can, over time, consume nearly all of the oxygen in the cylinder
- Inadequate purging of breathing bags in closed or semiclosed breathing apparatus

In terms of inspired oxygen percentage at one atmosphere or at equivalent oxygen partial pressures, there are usually no perceptible effects down to about 16% oxygen (PO₂ of 0.16 ata). At 12-14%, most people will not notice the first symptoms of tingling, numb lips, and tunnel vision. These symptoms become more prominent at 9-10%, with the onset of dizziness; collapse is imminent for some. At levels much below this, some people can stay conscious with great effort but most will become unconscious. There is a significant variation between individuals in susceptibility and symptoms; an adaptation to altitude can greatly increase one's tolerance to hypoxia. Fitness helps, but individual physiology is a more prominent factor. Typical responses are included in Table 3.1, which shows both the range of hypoxic effects and higher ranges of oxygen uses.

Hypoxia decreases the ability to think, orient, see properly, or perform tasks. Of all the cells in the body, brain cells are the most vulnerable to hypoxia. Unconsciousness and death can occur in brain cells before the

TABLE 3.1
Effects of Different Levels
of Oxygen Partial Pressure

PO ₂ (atm)	Application and Effect
<0.08	Coma to ultimate death
<0.08-0.10	Unconsciousness in most people
0.09-0.10	Serious signs/symptoms of hypoxia
0.14-0.16	Initial signs/symptoms of hypoxia
0.21	Normal environment oxygen (sea level air)
0.35-0.40	Normal saturation dive PO ₂ level
0.50	Threshold for whole-body effects; maximum saturation dive exposure
1.6	NOAA limit for maximum exposure for a working diver
2.2	Commercial/military "Sur-D" chamber surface decompression, 100% O ₂ at 40 fsw pressure
2.4	60% N ₂ /40% O ₂ nitrox recompression treatment gas at six ata (165 fsw)
2.8	100% O ₂ recompression treatment gas at 2.8 ata (60 fsw)
3.0	50/50 nitrox recompression treatment gas for use in the chamber at six ata

effects of hypoxia are apparent in other cells. The victims of hypoxia do not usually understand what is occurring, and they may even experience a feeling of well-being.

Signs and Symptoms:

- Frequently none (the diver may simply lapse into sudden unconsciousness)
- Mental changes similar to those of alcohol intoxication
- Confusion, clumsiness, slowing of response
- Foolish behavior
- Cyanosis (bluish discoloration of the lips, nailbeds, and skin)
- In severe cases, cessation of breathing

Prevention:

- Avoid excessive hyperventilation before a breath-hold dive.
- When diving with a rebreather, flush the breathing circuit with fresh gas mixture before ascending.
- Always know the amount of oxygen in gas mixtures being breathed.

Treatment:

- Get the victim to the surface and into fresh air.
- If under water and using a rebreather, manually add oxygen to the breathing circuit and begin ascent immediately; if manual adjustments are made incorrectly, oxygen toxicity may result.
- If the victim is still breathing, supplying a breathing gas with sufficient oxygen usually causes a rapid reversal of symptoms.
- An unconscious victim should be treated as if he is suffering from gas embolism.
- Cardiopulmonary resuscitation should be administered if necessary and should be continued after the victim is in the recompression chamber.

3.2.6.2 Carbon Dioxide Toxicity

In diving, carbon dioxide excess (hypercapnia) occurs either from too much carbon dioxide in the breathing medium or because carbon dioxide produced by the body is not eliminated properly by the equipment or by the diver.

The breathing mixture itself may contain a higher than normal level of CO₂, or the equipment may allow exhaled CO₂ to be rebreathed.

Failure of the carbon dioxide absorption system of closed or semi-closed circuit breathing systems allows the build up of high CO₂ levels in any space where exhaled air accumulates and can be re-inhaled. Too much of this “dead space” in diving helmets or masks and in overly-large snorkels allows exhaled CO₂ to collect and be rebreathed.

Some full-face masks have as much as 0.5 liter of dead space. Free-flow helmets generally do not have dead space problems unless the flow rate is maintained at a low volume for an extended period. Oral-nasal masks inside full-face masks/helmets are also effective in reducing the amount of dead space.

A well-designed system has little dead space. Dead space volume cannot be determined by visual examination; special equipment is needed to determine how much exhaled gas is rebreathed.

Normally, the body keeps arterial CO₂ levels the same (within 3 mmHg), even with heavy exercise. With exercise, the body produces more CO₂, but the breathing rate automatically increases to eliminate the excess. The more production, the greater the sensation of shortness of breath occurs, and the greater the ventilatory effort. However, it is also true that large differences exist in individual responses to increases in carbon dioxide.

It is unknown why some divers do not increase ventilation sufficiently. Other divers deliberately breathe slowly, or they skip breathe—pausing after each breath to conserve cylinder air. In these cases, CO₂ may not be removed in a normal fashion, and carbon dioxide levels may rise (hypercapnia).

Another factor elevating CO₂ is the increased effort of breathing at depth. To a smaller extent, high oxygen partial pressure decreases ventilation in some situations; the body has enough oxygen and does not need to breathe as much, so it does not get rid of CO₂ as fast.

WARNING

SKIP-BREATHING IS NOT A SAFE PROCEDURE. CARBON DIOXIDE TOXICITY OCCURS WITH LITTLE OR NO WARNING.

Signs and Symptoms:

Occasionally, CO₂ poisoning produces no symptoms, although it is usually accompanied by an overwhelming urge to breathe and noticeable air starvation. There may be headache, dizziness, weakness, perspiration, nausea, a slowing of responses, confusion, clumsiness, flushed skin, and unconsciousness. In extreme cases, muscle twitching and convulsions may occur. The progressive nature of CO₂ poisoning is shown in Figure 3.11.

Zone I: At these concentrations and durations, no perceptible physiological effects are observed.

Zone II: Small threshold hearing losses have been found and there is a perceptible doubling in the depth of respiration.

Zone III: Discomfort, mental depression, headache, dizziness, nausea, “air hunger,” and decrease in visual discrimination.

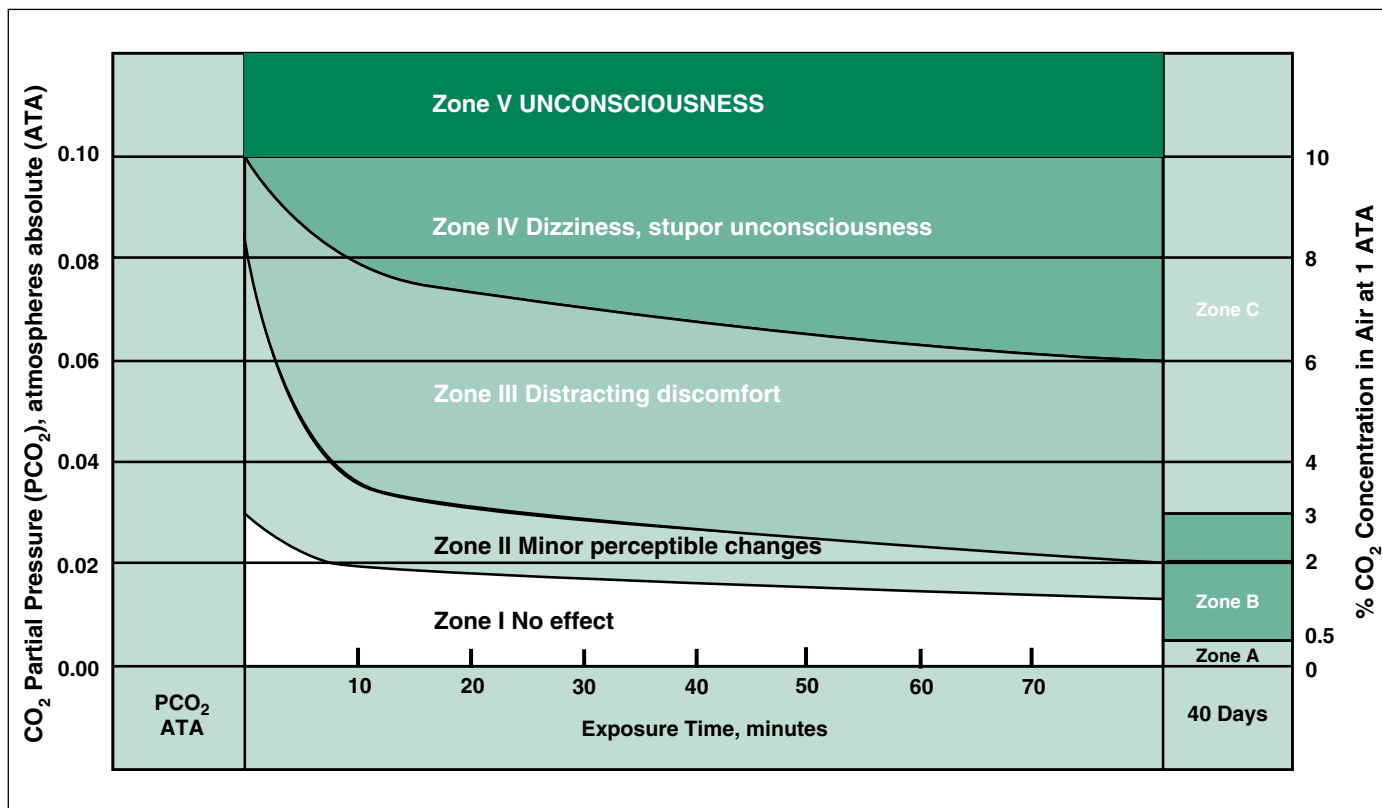


FIGURE 3.11
The Relationship of Physiological Effects in Carbon Dioxide Concentration and Exposure Periods

Zone IV: Marked physical distress, dizziness, stupor, inability to take steps for self-preservation.

Zone V: Unconsciousness. Above a CO₂ partial pressure (PCO₂) of 0.15 ata, muscle spasms, rigidity, and death can occur. The bar graph at the right of Figure 3.11 extends the period of exposure to 40 days.

Zone A: Concentrations between 0.5 and 3.0% (0.005-0.03 ata partial pressure), no biochemical or other effects.

Zone B: Above 3% (0.03 ata partial pressure). Adaptive biochemical changes, which may be considered a mild physiological strain.

Zone C: Pathological changes in basic physiological functions. For normal diving operations, ventilation rates should be maintained so that carbon dioxide partial pressures are maintained in Zones I and II for short-term exposures and in Zones A and B for long-term exposures.

Treatment:

Divers who are aware that they are experiencing carbon dioxide buildup should stop, rest, breathe deeply, and ventilate themselves and their apparatus. Fresh breathing gas usually relieves all symptoms quickly, although any

headache caused by the buildup may persist even after surfacing. If a diver becomes unconscious, he should be treated in accordance with the procedure described in Chapter 21.

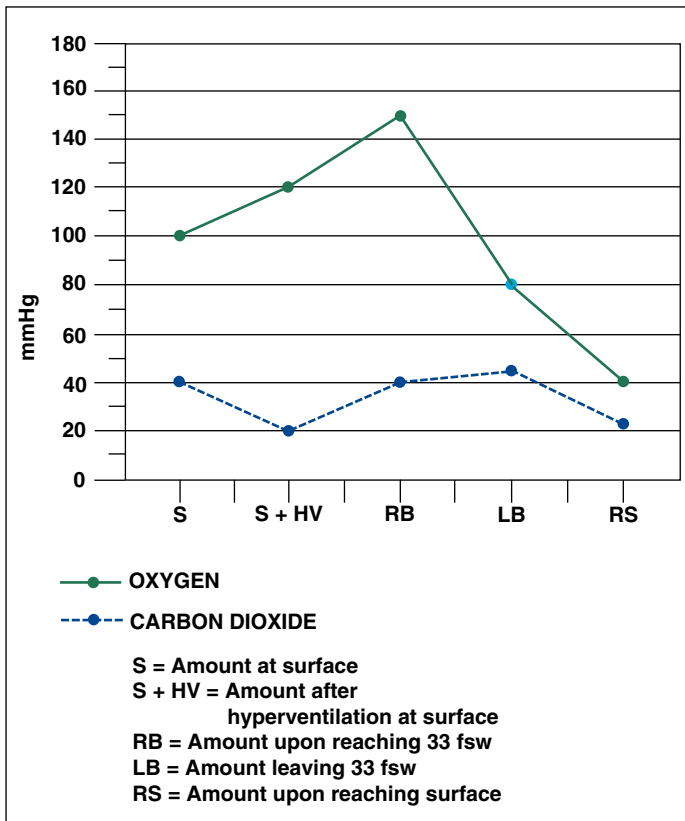
3.2.6.3 Hyperventilation

Hyperventilation includes several conditions that have the end result of lowering the blood carbon dioxide levels through overbreathing. In diving, hyperventilation means short-term, rapid, deep breathing beyond the amount needed for the activity. Divers may hyperventilate unintentionally during high-stress situations, from various health problems, or intentionally to extend breath-holding time.

Hyperventilation lowers CO₂ levels below normal, a condition known alternately as hypocapnia or hypocarbia. Without enough CO₂, normal, needed carbonic acid levels are not achieved, pushing body chemistry to the alkaline. The resulting alkalosis initially produces tingling fingers and limbs and lightheadedness. Over a longer period, it may produce weakness, faintness, headache, and blurred vision.

Slowing breathing will correct this, but divers may not be aware of why symptoms are occurring and not take corrective measures.

Divers who notice that they are excessively hyperventilating should take immediate steps to slow their breathing



Adapted from Robert Bumgarner, MD, "Diving Medicine - Shallow Water Blackout," *Skin Diver*, November 1990.

FIGURE 3.12
Partial Pressure \bar{D} mmHg

rate, notify their buddies, and, if feasible, ascend promptly. After reaching the surface, they should establish positive buoyancy inflating their buoyancy compensators or variable-volume dry suits. Hyperventilating divers should not attempt to swim to a boat or the shore unaided because they may lose consciousness in the attempt.

During surface-supplied diving, the tender should continuously monitor the sound of diver's breathing for signs of hyperventilation. Divers starting to hyperventilate should be instructed to stop work, rest, try to control their breathing rate, and ventilate their mask/helmet. Once on the surface, slowing breathing, breathing into a paper bag to rebreathe air with a higher level of carbon dioxide, or even holding the breath for short periods will restore CO_2 to normal levels.

3.2.6.4 Shallow Water Blackout

Hyperventilation prior to a surface dive used to be popular with free divers to extend their breath-hold time. It is still used today by some who have the distorted view that it will improve their diving capabilities. Unfortunately, upon ascending, the process may also lead to unconsciousness on or before returning to the surface.

It is important to understand that for a human being to remain conscious there has to be a certain concentration of

oxygen in the blood. Expressed as partial pressure of oxygen in the arterial blood, it is below a level of approximately 40–50 mmHg that an individual is exposed to the risk of sudden loss of consciousness (syncope).

Figure 3.12 depicts the changes in partial pressures of oxygen and carbon dioxide in the arterial blood on a free dive to 33 fsw (2 ata) and subsequent return to the surface. At "S" (surface), the chart illustrates that the arterial tension of CO_2 is 40 mmHg and O_2 at 100 mmHg. These are normal values for a healthy human at sea level. After hyperventilation (S + HV) the carbon dioxide is decreased to 1/2 its normal value (20 mmHg) and partial pressure of oxygen (120 mmHg) increases slightly.

In the healthy individual, it is the carbon dioxide level that stimulates one to breathe. At 33 fsw (RB) oxygen has been used up and there has been little or no real increase in CO_2 . However, due to the increase in ambient pressure to 2 ata, the actual partial pressure of the carbon dioxide has doubled to 40 mmHg. Oxygen, though consumed to the equivalent of 80 mmHg at sea level, is 150 mmHg at the 33 fsw, so there is no oxygen-deprivation distress at depth.

Eventually, the carbon dioxide rises slightly to 44 mmHg (LB), a level sufficient to stimulate the diver to want to breathe or in this case return to the surface. While at depth (2 ata) the partial pressure of the oxygen (80 mmHg) is sufficient to maintain consciousness. However, a significant amount of oxygen has been utilized. When the diver returns to the surface (RS), the partial pressures of the gases are cut in half by the reduction in ambient pressure to 1 ata. The net effect is that O_2 partial pressure falls to 40 mmHg, or below the level necessary to remain conscious and the diver "blacks out."

3.2.6.5 Carbon Monoxide Poisoning

Carbon monoxide (CO) is a poisonous gas, directly toxic to the body. It used to be popular to describe carbon-monoxide toxicity as simply a matter of carbon monoxide combining with hemoglobin to make carboxyhemoglobin, which blocked hemoglobin from carrying oxygen and produced hypoxia (oxygen deficiency). Although that is one of the effects, the situation is far more serious.

Carbon monoxide combines strongly with myoglobin, the oxygen-transporting and storage protein of muscle, and with the respiratory enzymes necessary for oxygen use in cells, directly stopping vital cellular functions. The entire oxygen process of transport, uptake, and utilization is disrupted. Carbon monoxide also blocks hemoglobin from removing carbon dioxide. Effects of CO increase with depth. The increased pressure of oxygen at depth does not offset carbon monoxide toxicity. Because of cellular toxicity, hypoxia occurs even if the air being breathed has sufficient oxygen.

CO exposure can result in pounding headache, nausea, and vomiting. High concentrations may cause sudden loss of consciousness.

TABLE 3.2
Carboxyhemoglobin Relative to CO Exposure

Continuous Exposure Level of CO	HbCO in Blood
50 ppm	8.4 %
40 ppm	6.7 %
30 ppm	5.0 %
20 ppm	3.3 %
10 ppm	1.7 %
–	0.5 % (non-smoker)

Hemoglobin binds with CO 200 to 300 times more readily than O₂, rendering it difficult and time consuming to eliminate from the body once inhaled. CO behaves in half-time fashion, like nitrogen. At sea level breathing regular air, 5 1/2 hours pass before half the CO leaves the body; another 5 1/2 hours must pass for the next 25 percent to leave, and so on. Breathing 100 percent oxygen at the surface reduces the half-time to just under 1 1/2 hours. At three ata (66 ft.) in a hyperbaric chamber, the half-time is about 23 minutes.

Contamination of a scuba cylinder of air with CO can come from fumes drawn into the compressor intake. These fumes can come from the exhaust of an internal combustion engine or from partial combustion of lubricating oil in a compressor not properly operated or maintained. Air intakes on compressors must be protected to avoid carbon monoxide contamination. Use oil with an appropriate flash point if using an oil-lubricated compressor.

Smoking cigarettes creates carboxyhemoglobin in the blood. The exhaled breath of a smoker can contain more carbon monoxide than NOAA allows in its compressed air for diving (see Table 3.2). Diving-industry standards call for air suppliers to adhere to air-quality standards.

Signs and Symptoms:

Carbon monoxide poisoning usually produces no symptoms until the victim loses consciousness. Some victims experience headache, nausea, dizziness, weakness, a feeling of tightness in the head, confusion, or clumsiness, while others may be unresponsive or display poor judgement. Rapid deep breathing may progress to cessation of breathing. There may be abnormal redness or blueness of lips, nailbeds, or skin. The classic sign of CO poisoning, “cherry-red” lips, may or may not occur and is therefore not a reliable diagnostic aid.

Treatment:

The victim should be given fresh air and, if available, oxygen. Some effects, such as headache or nausea, may persist after the exposure has ended. An unconscious victim should be treated in accordance with the procedures

outlined in Chapter 21. The treatment of choice is hyperbaric oxygen therapy in a recompression chamber.

3.2.6.6 Excessive Resistance to Breathing

“Work-of-breathing” is the amount of effort involved with inhaling against the elastic resistance of the chest walls, the resistive forces from restrictions in and lengths of the airways, and those of supplemental breathing apparatuses. If breathing resistance is high, it makes breathing more difficult, particularly during hard work.

Work-of-breathing increases with high resistance to gas flow in poorly tuned scuba regulators, valves, and hoses, and from tight equipment and exposure suits, or from an air supply in which the supply valve is not wide open. Breathing resistance can increase with gas density, which increases with depth. Some regulators are not designed to handle deep depths or high breathing needs. Well-designed, fitting, and maintained equipment minimizes resistance to the flow of breathing gas. Resistance increases to the square of the increased flow rate. That is, doubling the flow rate increases breathing resistance by four times. Rapid breathing patterns increase turbulence which, past a point, can increase breathing resistance. Small-bore snorkels, small-diameter exhaust valves, breathing hoses, and mouthpieces may increase turbulent flow to the point of increasing work-of-breathing.

The body compensates for high breathing resistance by reducing ventilation—easily demonstrated by breathing through a narrow tube. As work-of-breathing increases, the body reaches a limit; it will accept increased carbon dioxide rather than perform the increased respiratory work required to maintain a normal CO₂ level in the tissues. Excess breathing resistance has been implicated in some diving accidents.

To reduce work-of-breathing, breathe normally. Keep equipment well tuned and serviced regularly.

3.2.6.7 Lipoid Pneumonia

Lipoid pneumonia can result if a diver breathes gas containing suspended petroleum vapor. Once petroleum particles enter the lungs they remain there for a long time. This condition is commonly known as “black lung” and is prevented by not allowing oil vapor in the diver’s breathing gas and by ensuring that only approved oil is used in diving compressors.

NOTE

Oil of any kind is dangerous if breathed into the diver’s lungs.

3.3 EFFECTS OF PRESSURE

Effects of pressure may be arbitrarily divided into two main categories:

1. Direct, mechanical effects during descent and ascent.
2. Effects from changes in the partial pressure of inspired gases during descent and ascent.

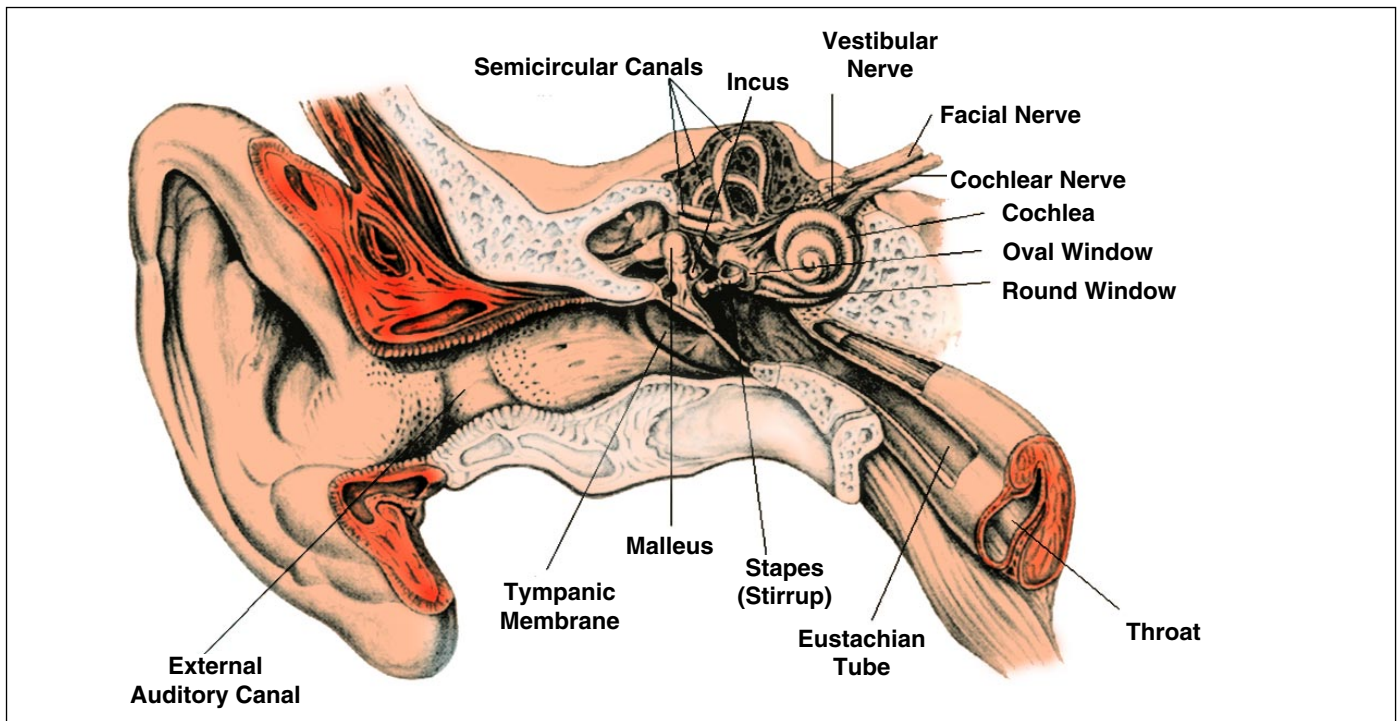


FIGURE 3.13
Principle Parts of the Ear

3.3.1 Direct Effects of Pressure During Descent

The body can withstand great hydrostatic pressure. Divers have made open-sea dives to over 1,500 fsw (682 psia); in experimental situations, divers have been exposed to pressures equivalent to 2,250 fsw (1,016 psia). Pressure increases do not affect the liquid areas of the body which are essentially incompressible and do not change shape or distort. Air spaces are not affected as long as pressure inside the air space is the same as pressure outside. If openings to the air space are blocked on descent or ascent preventing pressure equalization, the air spaces can be mechanically distorted and injured. Injury from pressure change is called barotrauma. The Greek word *baros*, means weight or pressure, and *trauma* means injury.

This section covers barotrauma during descent, as pressure increases on the air spaces of the ears, sinuses, lungs, and in certain pieces of diving equipment.

3.3.1.1 Ears

The ear has three divisions: outer, middle, and inner (see Figures 3.13). Each division functions separately to convert sound waves into nerve impulses going to the brain.

The outer ear is comprised of the outer projecting portion and the ear canal. The outer ear collects sound waves and channels them through the ear canal to the ear drum which begins the middle ear (see Figure 3.14). The middle ear is a tiny air cavity in the temporal bone on both sides of the skull. It is a closed air space; when pressure increases volume decreases, making the middle

ear susceptible to pressure problems. The middle ear has three small bones (malleus, incus, stapes) which intensify sound waves from the ear drum and transforms them into mechanical vibrations going to the inner ear through a small, delicate, oval membrane called the oval window. The last of the three middle ear bones, the stirrup,

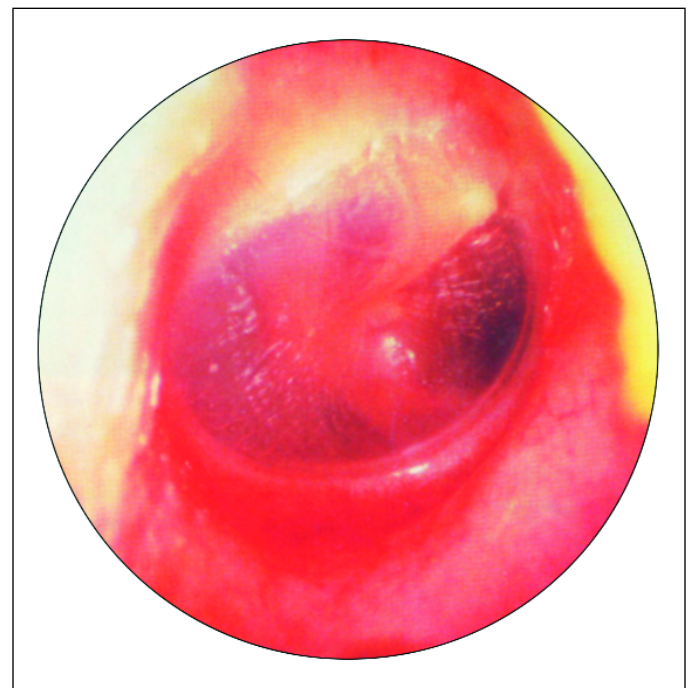


FIGURE 3.14
Tympanic Membrane (Ear Drum)

attaches directly to the oval window. The oval window is 20 times smaller than the ear drum, so pressure changes on the ear drum from sound waves or diving amplify greatly on the oval window. Another delicate structure separating middle and inner ear is the round window.

WARNING

MIDDLE EAR SQUEEZE, OR TOO VIGOROUS AUTO INFLATION OF THE MIDDLE EAR SPACE USING A VALSALVA MANEUVER (ATTEMPTING TO EXHALE THROUGH CLOSED NOSTRILS), CAN TRANSFER PRESSURE AGAINST OVAL OR ROUND WINDOWS, OR EVEN RUPTURE THEM, LEADING TO VERTIGO AND SOMETIMES PERMANENT HEARING LOSS.

The middle ears connect to the throat by the eustachian tubes, allowing air to enter the middle ears for pressure equalization. The eustachian tube is about 1.5 inches (3.8 cm) long in the adult. Pressure equalization of the middle ear is ongoing, whether diving, flying, or while on dry land. The eustachian tubes open as middle-ear volume changes when swallowing and chewing, allowing air to pass and equalize pressure.

The closed, fluid-filled inner ear has two parts. Both have intricate shapes, called labyrinths. Receptor cells in the inner ear receive mechanical vibrations, change them to neural impulses, and transmit them to the brain. The second, separate function of the inner ear pertains to location, motion, and balance.

During descent, water pressure increases against the ear drum. The ear drum bows inward, compressing air in the middle ear, initially equalizing pressure; however, the ear drum can stretch only so far. As water pressure increases relative to middle ear pressure, it creates an uncomfortable relative vacuum in the middle ear. This is “middle-ear squeeze,” or barotitis media. Ear squeeze is common among divers, but easily preventable.

Successful methods of equalizing middle-ear pressure are swallowing, yawning, or gently blowing against a closed mouth and nostrils, which allows air from the throat to enter the middle ear through the eustachian tube. If one does not equalize pressure, negative pressure in the middle ear continues to stress the eardrum, expanding the blood vessels of the eardrum and middle-ear lining. Either the ear drum will rupture, allowing air or water to enter the middle ear and equalize the pressure, or the blood vessels will leak and rupture to allow enough bleeding in the middle ear to equalize pressure. Inner-ear injury may occur from rupture of the round window. Round-window rupture requires surgical repair. Injuries to the eardrum or inner ear may occur with as little as three pounds of pressure differential and can happen at any depth. It’s usually more difficult to equalize during descent than ascent because the air passes out of the middle ear through the eustachian tube more easily than into the middle ear.

WARNING

BECAUSE OF THE DANGER OF EAR DAMAGE, DO NOT DO A FORCEFUL VALSALVA MANEUVER BEFORE OR DURING DESCENT OR ASCENT.

Upper respiratory infection (URI) may reduce or prevent equalization. Conditions contributing to stuffiness include acute or chronic inflammatory illness, allergy, irritation from smoking, and prolonged use of nasal spray. If one has difficulty equalizing on the surface, don’t dive. If one has an upper respiratory infection of any kind, don’t dive until the infection has cleared.

Systemic and topical drugs may improve nasal and sinus function and middle-ear equalization. Use them cautiously because a rebound phenomenon can occur, particularly with nasal spray. When the drug wears off, greater congestion and equalization problems can reoccur.

If you have chronic nasal obstruction, frequent upper-respiratory infections, nasal allergies, mastoid or ear disease, or chronic sinus trouble, see an ear, nose, and throat specialist.

Signs and Symptoms:

- Fullness or pressure in region of the external ear canals
- Squeaking sound
- Pain
- Blood or fluid from external ear
- Rupture of ear drum

Prevention:

- Use of solid ear plugs are prohibited in diving.
- Fit of diving hoods and earphones should be adjusted so that they do not completely cover or seal the external ear canal during ascent or descent.
- Accumulated wax that can obstruct the ear canal should be removed by gently irrigating the canal with a lukewarm water solution, using a rubber bulb syringe. Care should be taken before irrigation to guarantee that there is no ear drum perforation behind the obstructing wax.
- Descend feet first, preferably down the anchor line or descent line. Membranes line the airways in the head, and gravity affects blood in the vessels within the membranes. When you are upside down in water, the membranes of the air passages swell and narrow.
- Don’t wait for ear pain to start before equalizing. Start equalizing as soon as pressure is felt, or before.
- Equalize gently. Avoid forceful blowing.
- Stop descent if ear blockage or fullness develops. Ascend until symptoms resolve, even if you must return to the surface.

Treatment:

Ear drum rupture should be treated according to the procedures for treating middle ear barotrauma. See a physician.

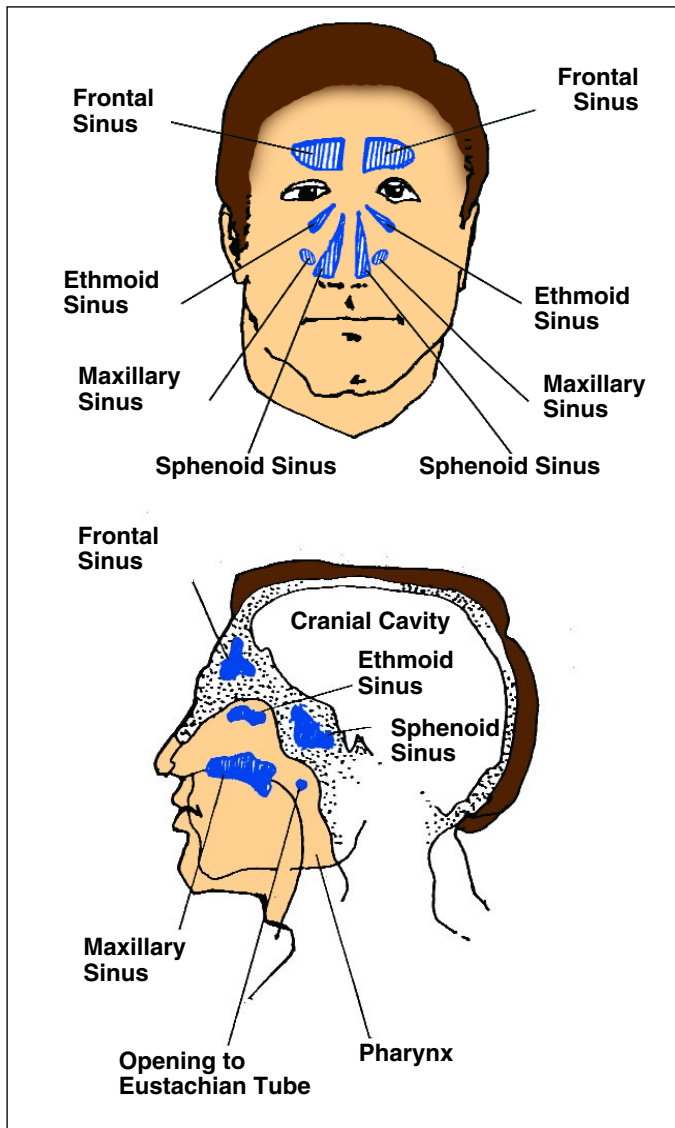


FIGURE 3.15
Sinus Cavities in the Head

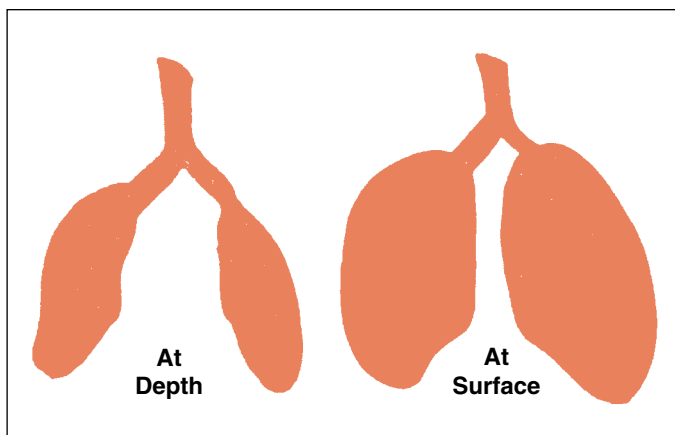


FIGURE 3.16
Effect of Descent/Ascent on Lungs

3.3.1.2 Sinuses

The term “sinus” can mean any channel, hollow space, or cavity in a bone, or a dilated area in a blood vessel or soft tissue; most often sinus refers to the four, paired, mucus-lined, air cavities in the facial bones of the head. Sinus cavities are shown in Figure 3.15.

The same kind of membrane lines the sinuses and nose, so nasal infections spread easily to the sinuses. In sinusitis, mucous membranes inflame and swell, closing sinus openings and preventing infected material from draining. If nasal inflammation, congestion, deformities, or masses block sinus openings, the sinus lining swells and inflames, absorbing pre-existing gas which forms negative pressure. When blockage occurs during descent, the relative vacuum in the sinus increases the risk of damage. Hemorrhage into the sinus may occur.

Don't dive if you have congested sinuses. Various over-the-counter and prescription medications open sinus passages. If a decongestant wears off during your dive, the sinuses become closed spaces (rebound effect) containing high pressure air. This can lead to a reverse block on ascent. Sinus barotrauma can also occur during ascent if blockage of a one-way valve of the sinus, by inflamed mucosa, cysts, or polyps, allows equalization on descent, but impairs it on ascent.

Signs and Symptoms:

- Sensation of fullness or pain in the vicinity of the involved sinus or in the upper teeth
- Numbness of the front of the face
- Bleeding from the nose

Treatment:

The treatment of sinus squeeze may involve the use of nasal decongestants, vasoconstrictors, and antihistamines taken by mouth. These medications will promote nasal mucosal shrinkage and opening of the sinus. Most of the symptoms of paranasal sinus barotrauma disappear within five to ten days without serious complications. Divers who have symptoms for longer periods should see a specialist. If severe pain and nasal bleeding are present or if there is a yellow or greenish nasal discharge, with or without fever, a specialist should be seen promptly. Individuals with a history of nasal or sinus problems should have a complete otolaryngologic evaluation before beginning to dive.

3.3.1.3 Lungs

On a breath-hold dive, there is no compressed air supply. So lung pressure cannot be equalized with ambient pressure. Lung spaces compress with increasing depth (see Figure 3.16). It used to be thought that the lungs compress by the simple pressure-volume relationship of Boyle's Law; that is at five times surface pressure (132 ft. or 40 m) lung volume would compress to 1/5 volume, which can become less than residual volume, depending on the size of the lungs. At such low volume, a condition called thoracic squeeze would develop, including pulmonary congestion, swelling, and hemorrhage of the lung tissue.

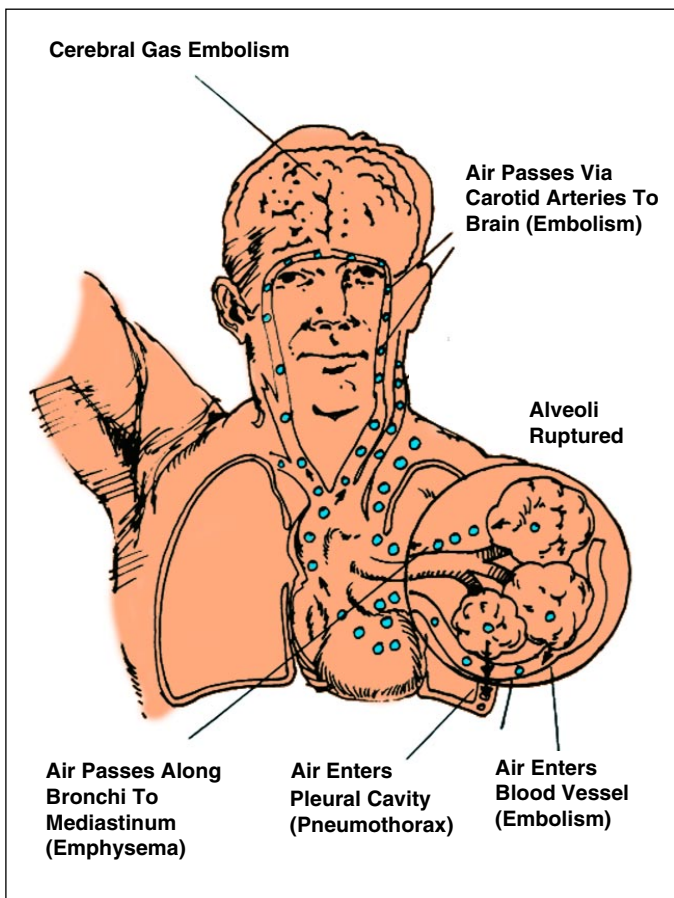


FIGURE 3.17
Consequences of Overinflation of the Lungs

However, the body has many self-regulatory abilities. Thoracic squeeze does not readily occur, even at far greater depths. Compression during descent shifts blood from the extremities and abdomen into thoracic blood vessels, maintaining a larger than predicted lung volume. Lung volume can fall below residual volume without the damage previously thought. Record free dives to over 400 ft. (122 m) have been successful. Such dives are not without other dangers; they should not be attempted without extraordinary preparation and training.

Signs and Symptoms:

- Feeling of chest compression during descent
- Pain in the chest
- Difficulty in breathing on return to the surface
- Bloody sputum

Treatment:

In severe cases of lung squeeze, the diver requires assistance to the surface. The diver should be placed face down, and blood should be cleared from the mouth. If breathing has ceased, cardiopulmonary resuscitation with oxygen should be administered. Attendants should be alert

for symptoms of shock, and treatment for shock should be instituted, if necessary. The dive accident management plan should be initiated.

3.3.1.4 Eyes

Non-compressible fluids fill the eyes, protecting them against direct water pressure. Use of a mask preserves underwater vision, but introduces an air-space around the eyes that must be equalized with ambient pressure during descent. Without equalization, negative pressure in the mask space creates suction on the eyes and lids. Swelling, bruising, and bleeding can occur in the mucous membrane lining the eyelid (conjunctiva). A more serious injury can also occur — blood in the anterior chamber of the eye, called hyphema. To prevent mask squeeze, mask pressure is equalized by exhaling through the nose during descent. During ascent, air vents harmlessly out of the mask.

Signs and Symptoms:

- Sensation of suction on the face, or of mask being forced into face
- Pain or a squeezing sensation
- Face swollen or bruised
- Whites of eyes bright red

Treatment:

Ice packs should be applied to the damaged tissues and pain relievers may be administered if required. In serious cases, the services of a physician knowledgeable in diving medicine should be obtained.

3.3.2 Direct Effects of Pressure During Ascent

During ascent, ambient pressure decreases and air in the body's air spaces expands. When this air vents freely, there is no problem. When expanding air is blocked from venting, overinflation occurs and increases the possibility of overpressurization injury.

This section covers barotrauma during ascent, as decreasing pressure affects the lungs, gastrointestinal tract, teeth, and the space behind contact lenses.

3.3.2.1 Lungs—Pneumothorax

If breathing is normal and there are not any lung lesions or conditions that obstruct air flow, lungs will vent expanding air on ascent without problem. If expanding air is blocked from exiting, the lungs will overinflate, thus damaging the alveoli and bronchial passages. Breath-holding or insufficient exhalation can create general lung barotrauma; obstruction from chronic or acute respiratory diseases, or bronchospasm with asthma, can result in localized lung overpressure and barotrauma. If expanding air ruptures the lung, air escapes into the small, normally airless area between lungs and chest wall (see Figure 3.17). This injury is called pneumothorax.

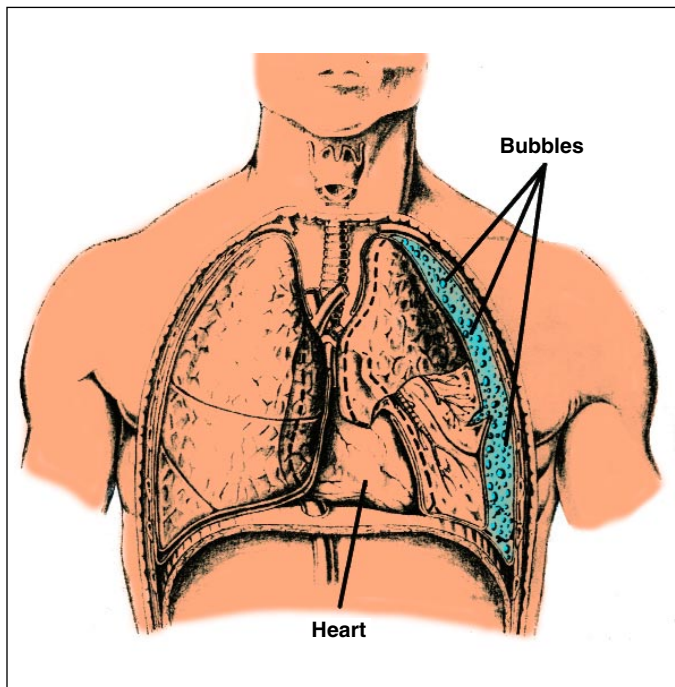


FIGURE 3.18
Lungs Ñ Pneumothorax

The lungs are attached to the chest wall by a thin, paired membrane called the pleura. The two pleural membranes lie so close to each other that they touch. A watery fluid lubricates the layer between them, making a suction between the layers, which holds open the lungs. Air rupturing the lung walls vents air into the pleural cavity, breaking the suction. There are two types of pneumothorax; simple and tension. A simple pneumothorax is a one time leaking of air into the pleura cavity. A tension pneumothorax is a repeated leaking of air from the lungs into the pleural cavity with each successive breath, thus progressively enlarging the air pocket. A large amount of air between pleural membranes prevents the lungs from expanding. Trapped intrapleural gas expands as ascent continues, increasing pressure in the chest cavity. A lung may collapse, and the heart may push out of normal position, causing sudden severe pain, difficulty breathing, and, rarely, coughing frothy blood or death from shock (see Figure 3.18).

Signs and Symptoms:

- Difficulty or rapid breathing
- Leaning toward affected side
- Hypotension
- Cyanosis and shock
- Chest pain (deep breath hurts)
- Shortness of breath
- Decreased or absent lung sounds on affected side
- Rapid, shallow breathing
- Death

Treatment:

Simple Pneumothorax

- Normally improves with time as air is reabsorbed.
- Monitor for signs of tension pneumothorax.
- Monitor ABC (airway, breathing, and circulation) and administer 100 percent oxygen.
- Transport to nearest medical facility.

Tension Pneumothorax

- Position patient on injured side.
- ABC.
- Treat for shock and administer 100 percent oxygen.
- Transport immediately to nearest medical facility (air must be vented from chest cavity).

WARNING

DO NOT HOLD BREATH WHEN ASCENDING USING SCUBA OR SURFACE-SUPPLIED EQUIPMENT.

WARNING

A DIVER WHO HAS HAD AN OVERPRESSURE ACCIDENT MUST BE EXAMINED IMMEDIATELY BY A DIVING MEDICAL DOCTOR.

3.3.2.2 Lungs—Mediastinal Emphysema

Emphysema, in general, means an abnormal distention of body tissues from retention of air. The most familiar type of emphysema usually results from smoking or other lung pollution, and permanently overexpands and damages alveoli. In mediastinal emphysema, air escapes from a lung overpressurization into tissues around the heart, major blood vessels, and trachea (windpipe). This gas expands on ascent, causing pain under the sternum (breast-bone), shortness of breath, or, in extreme cases, fainting from impaired blood return to the heart (see Figure 3.19).

Signs and Symptoms:

- Pain under the breastbone that may radiate to the neck, collarbone, or shoulder
- Shortness of breath
- Faintness
- Blueness (cyanosis) of the skin, lips, or nailbeds
- Difficulty in breathing
- Shock
- Swelling around the neck
- A brassy quality to the voice
- A sensation of pressure on the windpipe
- Cough
- Deviation of adams apple to affected side

Treatment:

- ABC
- Administer oxygen and monitor for shock
- Examine diver for other signs of pulmonary barotrauma

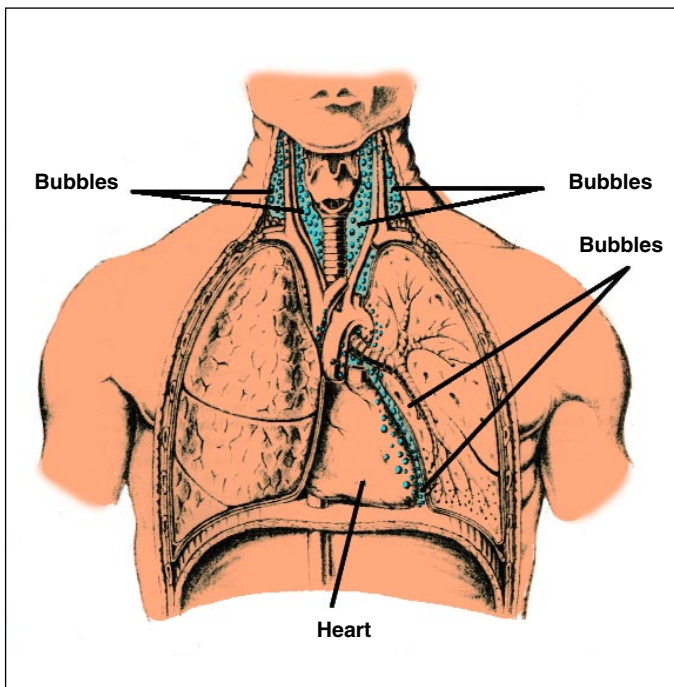


FIGURE 3.19
Lungs Ñ Mediastinal Emphysema and Subcutaneous Emphysema

- Mediastinal emphysema causing respiratory or circulatory impairment may require recompression.
- Transport to the nearest medical facility.

3.3.2.3 Lungs—Subcutaneous Emphysema

Subcutaneous emphysema results from air forced into tissues beneath the skin of the neck. It can be associated with mediastinal emphysema or can occur alone (see Figure 3.19).

Signs and Symptoms:

- Feeling of fullness in the neck area
- Swelling or inflation around the neck and upper chest
- Crackling sensation when skin is palpated
- Change in sound of voice
- Cough

Treatment:

Unless complicated by gas embolism, recompression is not normally required. The services of a physician should be obtained and oxygen should be administered if breathing is impaired.

3.3.2.4 Arterial Gas Embolism

An arterial gas embolism occurs when a bubble of gas (or air) causes a blockage of the blood supply to the heart, brain, or other vital tissue (see Figure 3.20). Arterial gas embolism may be abbreviated AGE. A cerebral (brain) arterial gas embolism is abbreviated CAGE.

The bubble tends to increase in size as the pressure decreases (Boyle's Law), which makes the blockage worse.

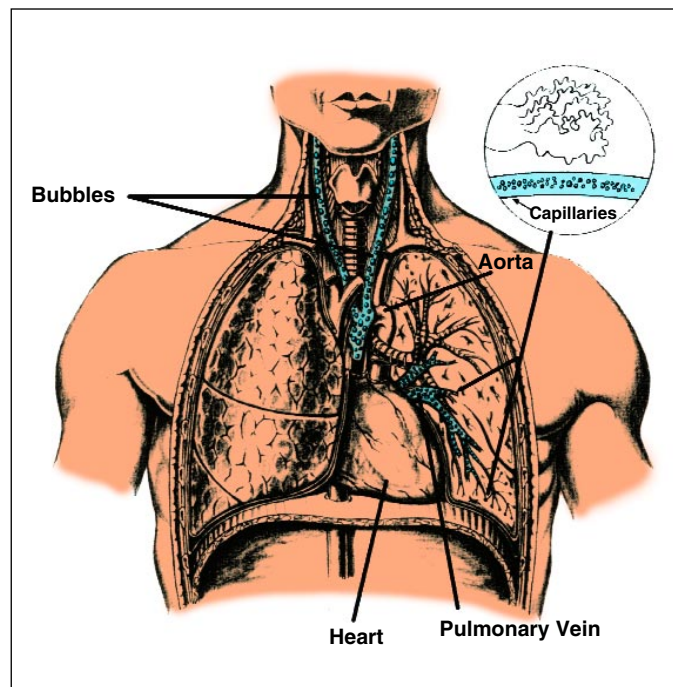


FIGURE 3.20
Arterial Gas Embolism

When a diver holds his breath or has local air trapped in his lungs during ascent, the volume of gas in the lungs increases due to a reduction in ambient pressure. Alveoli can rupture or air can be forced across apparently intact alveoli. If air bubbles enter the pulmonary veins, they travel to the left side of the heart muscle, and then commonly on through the carotid arteries to embolize the brain. As the bubbles pass into smaller arteries, they reach a point where they can move no further, and here they stop circulation. Deprived of oxygen, those tissues die.

Arterial gas embolism may occur quickly after surfacing with damage depending on the area involved. There is no way to predict which area will be affected. Symptoms of arterial gas embolism usually occur immediately or within five minutes of surfacing. Prompt recompression is the only treatment for gas embolism.

One, a few, or all of the symptoms listed below may be present.

Signs and Symptoms:

- Chest pain
- Cough or shortness of breath
- Bloody, frothy sputum
- Headache
- Visual disturbances including blindness, partial or complete
- Numbness or tingling (paresthesias)
- Weakness or paralysis
- Loss of, or change in, sensation over part of body
- Dizziness

- Confusion
- Sudden unconsciousness (usually immediately after surfacing, but sometimes before surfacing)
- Respiratory arrest
- Death

WARNING

ARTERIAL GAS EMBOLISM IS LIFE THREATENING AND REQUIRES IMMEDIATE TREATMENT.

Prevention:

- Never hold your breath when diving with compressed gases
- Ascend slowly (30 feet per minute)
- Do not dive with a chest cold or obstructed air passages
- Maintain good physical fitness, nutrition, and hydration
- Carry sufficient quantities of gas to complete the dive

Treatment:

- Establish and maintain ABC, and initiate cardiopulmonary resuscitation, if necessary
- Administer 100 percent oxygen with the injured diver supine or in recovery position
- Transport to nearest medical facility and initiate recompression treatment ASAP
- Perform a physical examination, including neurological examination, as soon as situation permits
- Provide additional life support measures
- Reassess diver's condition regularly

Rescuers and attendants must be aware that many embolism patients are also near-drowning victims. Position the injured diver in a supine or the recovery position. Injured diver position should not be allowed to interfere with the immediate administration of CPR. Administer 100 percent oxygen with a tight-fitting oronasal mask by demand/positive-pressure valve or non-rebreather mask at 15-lpm, and transport the patient as rapidly as possible to a medical facility for recompression treatment. A gas embolism case is a minute-to-minute emergency transfer. The chances of full recovery decrease with each minute lost in returning the patient to pressure. If air transportation is required, the patient must not be exposed to decreased cabin pressure during transit; consequently, aircraft capable of being pressurized to sea level must be used. A helicopter or unpressurized aircraft must be flown as low as is safely possible. Despite the decreased chance of recovery if therapy is delayed, patients have responded even after several hours delay. Victims should not be taken back into the water for treatment.

3.3.2.5 Stomach and Intestine

Only a small amount of gas is normally present in the small intestine at any time. Although gas enters or forms in larger quantities, most is usually absorbed back through the intestinal mucosa. Any air remaining in the stomach and large intestine compresses with descent and returns to normal volume on ascent. The intestines are surrounded by soft tissue so compression and expansion are, normally, neither hazardous nor noticeable.

If you add enough gas to the system while under water, the gas will expand on ascent. These gases can be generated by swallowing air, or within the intestine from carbon dioxide liberated by reactions between gastric and pancreatic juices, or a prior gas-producing meal. Ambient pressure pushing on a stomach full of gas can cause belching or back flow of stomach contents (heartburn). Severe injury is rare. With a hernia, expanding gas trapped in a loop of bowel could make the hernia irreducible.

To prevent gastrointestinal (GI) barotrauma, breathe normally, don't swallow air, and avoid large meals and gas-producing food and drink before diving. Should GI distress occur on ascent, descend to relieve discomfort, and slowly re-ascend. It may help to keep the legs moving. If surfacing is necessary before relieving pressure, try various over-the-counter, anti-gas preparations. In extreme cases, get medical attention.

3.3.2.6 Teeth

Barodontalgia means "tooth pain." It occurs when a small pocket of gas collected in a tooth during the dive expands on ascent. Tooth pain has been reported during air travel as well.

The air space may be generated by decay resulting in an area for gas to collect under a filling. Other causes of tooth squeeze include recent extractions, gum infections that have formed periodontal pockets, large areas of decay where the pulp is infected, abscesses, recent fillings, and recent root canal therapy. Part of the root canal procedure is to dry and temporarily seal the canal between treatments with a material designed for pressure of one atmosphere. Exposure to higher pressures can produce small leaks that cannot release air fast enough during ascent. Trapped air can shatter full porcelain crowns in teeth where the cement bond is failing. Gas accumulated slowly during a saturation dive has been known to (rarely) cause tooth cracking and even explosion.

Tooth squeeze is not common, but prevention is worth keeping in mind. Keep teeth clean, have cavities filled and ill-fitting crowns replaced. Complete endodontic therapy before diving. Before undergoing any dental work, inform the dentist of diving status.

3.3.2.7 Contact Lenses

Bubbles have been found in the precorneal film of tears beneath hard contact lenses after ascent. Affected divers

TABLE 3.3
Narcotic Effects of Compressed Air Diving

Guideline Depths		
Feet	Meters	Effect
0-100	0-30.5	Mild impairment of performance on unpracticed tasks. Mild euphoria.
100	30.5	Reasoning and immediate memory affected more than motor coordination and choice reactions. Delayed response to visual and auditory stimuli.
100-165	30.5-50.3	Laughter and loquacity may be overcome by self control. Idea fixation and overconfidence. Calculation errors.
165	50.3	Sleepiness, hallucinations, impaired judgment.
165-230	50.3-70.1	Convivial group atmosphere. May be terror reaction in some. Talkative. Dizziness reported occasionally. Uncontrolled laughter approaching hysteria in some.
230	70.1	Severe impairment of intellectual performance. Manual dexterity less affected.
230-300	70.1-91.5	Gross delay in response to stimuli. Diminished concentration. Mental confusion. Increased auditory sensitivity, i.e. sounds seem louder.
300	91.5	Stupefaction. Severe impairment of practical activity and judgment. Mental abnormalities and memory defects. Deterioration in handwriting, euphoria, hyperexcitability. Almost total loss of intellectual and perceptive faculties.
300	91.5	Hallucinations (similar to those caused by hallucinogenic drugs rather than alcohol).

experienced soreness, decreased visual acuity, and the appearance of halos around lights for about two hours after ascent. Divers who wear contact lenses should use either soft lenses or hard fenestrated lenses (hard lenses with a special hole drilled). Consult with an ophthalmologist.

3.3.3 Indirect Effects of Pressure During Descent

Indirect effects of pressure occur from changes in the partial pressure of the gases in the breathing mix. This section covers inert gas narcosis, high pressure nervous syndrome (HPNS), and oxygen toxicity.

3.3.3.1 Inert Gas Narcosis

Inert gas narcosis is a condition of confusion or stupor resulting from increased pressure of dissolved inert gas. The most common inert gas narcosis is nitrogen narcosis.

The gases producing narcosis have no effect if they are not breathed under pressure. High pressure dissolves gas in the protein coverings of nerve cell membranes, depressing nerve excitability and interfering with signals. Of course, there are other factors involved in this complex and incompletely understood phenomenon.

Although often portrayed as such, narcosis is not always rapturous or intoxicating. Effects can be unpleasant

or frightening, particularly in limited visibility or cold water. Even if pleasant, narcosis impairs intellectual capacities, short-term memory, time perception, orientation, judgment, reasoning, and the ability to perform mental functions, making it difficult to monitor time, depth, air supply, or the location of a buddy. Dive plan information may be forgotten. Spatial orientation may become a matter of complete indifference. Severe narcosis can produce hallucinations, bizarre behavior, or loss of consciousness. Physical problems include decreased motor ability and slowed reaction time. Because it decreases perceptions of cold and decreases heat production, narcosis may play an important role in diving hypothermia. Despite the popular belief, narcosis does not slow respiration. Narcosis is dangerous because it increases the risk of an accident while diminishing the ability to cope with one.

Impairment increases with depth. Narcosis is often first noticed at approximately 100 feet (31 m) when breathing compressed air. Wide variations in susceptibility occur; although, at greater depths, most compressed-air divers are affected (see Table 3.3).

Helium causes minimal narcosis, making it useful at depths where nitrogen narcosis would incapacitate a diver. Two other inert gases used in experimental diving are

neon and argon. Neon is not narcotic; however, argon is narcotic at deeper depths. Interestingly, it appears that too high an oxygen level can leave some oxygen in the tissues unmetabolized. To the extent that it is present in certain tissues, oxygen may also act as an inert gas and produce narcosis (Bennett and Elliott 1993).

Several factors can compound the effects of narcosis: CO₂, fatigue, anxiety, cold, alcohol, and hangovers. Medications that might cause drowsiness or reduce alertness, such as motion sickness remedies and sedatives, or sedating recreational drugs, also contribute to narcosis. This is consistent with the view that narcosis depresses the central nervous system (CNS). Narcosis rapidly reverses with ascent, though divers who have experienced narcosis may not remember events occurring at depth.

Signs and Symptoms:

- Loss of judgment and skill
- A false feeling of well being
- Lack of concern for job or safety
- Inappropriate laughter
- Euphoria

Prevention and Treatment:

There is no specific treatment for nitrogen narcosis. A diver experiencing narcosis must be brought to a shallower depth, where the effects will be reversed.

3.3.3.2 High Pressure Nervous Syndrome (HPNS)

High pressure nervous syndrome (HPNS) occurs at depths greater than 400 fsw (123 msw). First noted in the 1960s, HPNS was initially thought to be an effect of breathing helium, so it was called helium tremors. At that time, helium was the most commonly used diluent gas for diving at deeper depths. HPNS becomes worse with increasing pressure and rate of compression.

HPNS is characterized by dizziness, nausea, vomiting, postural and intention tremors, fatigue and somnolence, sudden muscle twitching (called myoclonic jerking), stomach cramps, intellectual and psychomotor performance decrements, and poor sleep with nightmares.

Adding a small amount (5-10%) of nitrogen into the breathing mix reduces HPNS. At high pressure, nitrogen is a neural depressant. Other methods of preventing or reducing HPNS include slow, steady compression, stage compression with long intervals, exponential compression rates, and careful personnel selection.

3.3.3.3 Oxygen Toxicity

Given oxygen’s metabolic effects, it should be no great surprise that in excess it can be toxic. In fact, all living things have enzymes and other mechanisms that protect against oxygen’s toxicities. There are two types of oxygen poisoning for which divers must be concerned: those affecting the central nervous system (CNS), and those affecting many other parts of the body more generally, particularly the lungs.

Con	– Convulsion
V	– Visual disturbances, including tunnel vision
E	– Ear ringing
N	– Nausea
T	– Tingling, twitching or muscle spasms, especially of the face and lips
I	– Irritability, restlessness, euphoria, anxiety
D	– Dizziness, dyspnea

FIGURE 3.21
CNS Oxygen Toxicity Signs and Symptoms

3.3.3.3.1 CNS: Central Nervous System

CNS oxygen toxicity can occur at the high end of PO₂ levels, even after short exposures. Typically, it can develop within a few to many minutes on exposure to partial pressures of oxygen above 1.6 atm (roughly 5 to 50 min, but this is highly variable) (Lambertsen 1978). The end result may be an epileptic-like convulsion that is not damaging in itself, but can result in drowning or physical injury. The acronym *CONVENTID* (see Figure 3.21) is a simple way to remember all the signs and symptoms of CNS oxygen toxicity. It is important to note that these symptoms may come in any order.

There are other signs and symptoms of CNS toxicity. Not onerous in themselves, they are justification to stop a dive. They include twitching of lips and facial muscles, visual or hearing disturbances, nausea, dizziness, difficulty in breathing (dyspnea), anxiety, confusion, poor coordination, and unusual fatigue. These may warn of an impending convulsion; however, a convulsion is just as likely to occur without any warning. Divers have been known to “black out” or go unconscious without a convulsion; this may be a manifestation of oxygen toxicity.

3.3.3.3.2 Lung and “Whole-Body”

Slower developing oxygen toxicities may follow exposure to lower levels of oxygen for longer times. The lung is the principal organ affected, but many other parts of the body can be affected as well. Therefore, the term “whole-body” toxicity is used to include the affected parts of the body other than the CNS.

A classical symptom of whole-body toxicity is pulmonary irritation, the result of oxygen’s effect on the lung. Such a symptom usually takes hours or longer to develop from exposure levels that may be lower than those that cause CNS symptoms. Whole-body oxygen toxicity is generally of little concern to divers doing no-stop dives, even when breathing oxygen-enriched mixtures, but it may be seen during intensive diving operations or during long oxygen treatments for decompression sickness in a hyperbaric chamber. Symptoms are

chest pain or discomfort, coughing, inability to take a deep breath without pain or coughing, a development of fluid in the lungs, and a reduction in vital capacity. Non-pulmonary symptoms of “whole-body” oxygen toxicity include skin numbness and itching, headache, dizziness, nausea, effects on the eyes, and a dramatic reduction of aerobic capacity during exercise.

3.3.3.3.3 Variations in Tolerance

There is wide variation in susceptibility to oxygen toxicity among individuals, and a significant variation in a single individual at different times. Part of this latter variation is due to unknown causes, but a large part can be attributed to known environmental and physiological circumstances. Susceptibility to CNS toxicity is increased by certain factors, particularly those that cause an increase in internal PCO_2 , such as exercise, breathing dense gas, or breathing against a resistance. Immersion, dramatic changes of temperature, and physical exertion also increases one's susceptibility to CNS oxygen toxicity. These differences make it difficult to predict the occurrence of CNS oxygen toxicity.

3.3.3.3.4 Benefits of Intermittent Exposure

Oxygen poisoning can be reduced or postponed by interrupting the exposure time (U.S. Navy Diving Manual 1999). If “breaks” in periods of low oxygen are taken during oxygen breathing, tolerance is greatly improved. In the U.S.N. tables for treatment of decompression sickness using oxygen, breaks of five minutes of air breathing are taken every 20 or 30 minutes of oxygen breathing at high PO_2 levels. This avoids oxygen convulsions in all but very rare cases and also postpones pulmonary toxicity. In situations where supplemental oxygen or high oxygen content mixtures are used for decompression, it is strongly recommended that a five minute “air” break be taken every 20 minutes to minimize the risk of oxygen poisoning.

3.3.3.3.5 Concepts of Oxygen Exposure Management

The traditional method used for prevention of CNS oxygen toxicity is to stay within exposure durations that are based on the oxygen level, or PO_2 , to which the diver is exposed (U.S. Navy Diving Manual 1999). These limits allow a certain time at each PO_2 range. Such an approach has been practiced by the U.S. Navy and by NOAA for many years in their procedures for mixed gas and oxygen diving.

As with decompression, a limit appears to be implemented as if it were a solid line dividing “no problems” from “guaranteed problems.” Actually, a limit is a solid line drawn through a wide gray area of gradually increasing risk. The limits given here and in other limit-based algorithms (such as a decompression table) are recommended guidelines for use under normal conditions. They have been proven in practice. They work for most

people most of the time, but they are not guaranteed to work for all people all of the time under all circumstances. They may need to be more conservative when conditions are more stressful.

Diving with procedures described in this chapter imposes a relatively low risk of oxygen toxicity. The exposures are short and outside the limits that are expected to cause problems.

3.3.3.3.6 Prevention of CNS Poisoning

With the help of experts, NOAA developed estimated oxygen exposure limits that were published in the 1991 version of the *NOAA Diving Manual*. These limits are shown in Table 3.4. They are intended for a diver doing dives for research, sampling, inspection, observation, and light to moderate work at the higher PO_2 levels. The lower levels can be used for heavier and more stressful types of work.

For each level of oxygen, the chart shows an allowable time for a single exposure and also an accumulated time at that level over a full day.

If more than one dive is made to the maximum exposure of a PO_2 of 1.6 ata, a suggested surface interval of at least 90 minutes is advised between dives (three dives of 45 minutes each would theoretically be possible within the 150-minute daily total allowed at 1.6 ata PO_2). This helps lower the accumulated oxygen dose. This only applies to the exposure at 1.6 ata, because only one maximal dive can be done in a single day with lower oxygen exposure levels.

If, however, one or more dives in a 24-hour period have reached or exceeded the limits for a normal single exposure, the diver should spend a minimum of two hours at a normoxic PO_2 (such as on the surface breathing air) before resuming diving. If diving in a 24-hour period reaches the Maximum 24-hour Limit, the diver must spend a minimum of 12 hours at normoxic PO_2 before diving again.

3.3.3.3.7 The “Oxygen Clock” or “ O_2 Limit Fraction”

These exposure limits are sometimes referred to as the “oxygen clock” in percentage of the allowable limit, or the “ O_2 limit fraction” as a decimal fraction of the limit (Hamilton 1988). For single dives to a single depth (square profile), calculating the percentage of oxygen exposure is as simple as dividing the minutes of the exposure by the maximum allowable exposure time at a given PO_2 . However, it is rare that a diver is ever at one depth for the entire dive. Although the principle has not been verified experimentally, it is customary to add the percentages or fractions of exposure for different parts of the dive to calculate an estimated total oxygen exposure for a given dive.

It is not necessary to have a dive computer to track these exposures if the dive can be separated into segments

TABLE 3.4
NOAA Oxygen Exposure Limits

PO ₂ (atm)	Maximum Single Exposure (minutes)	Maximum per 24 hr (minutes)
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

that have a predominant or average level. The times spent at each depth or exposure level can be assigned a fraction or percentage of the “allowable” limit, and these can simply be added together. Table 3.5 allows these segments to be determined from a chart.

For multilevel dives or more than one dive of less than maximum allowed duration, it is possible to interpolate the limit values. That is to say, at any level the full limit on the oxygen clock is 100 percent of the limit, or an O₂ limit fraction of 1.0. Exposures at all levels are totaled. For example, at 1.4 atm the allowable exposure time is 150 minutes (see Figure 3.22). If a diver has an exposure to that level for 75 minutes, half the allowable time, this would run the oxygen clock to 50 percent of the limit or the limit fraction to 0.5. If there is additional exposure on the same dive, for example, 60 minutes at 1.3 PO₂, for which the allowable time is 180 minutes, an additional one-third, 33 percent or 0.33 is added, giving an oxygen clock now of 83 percent or a limit fraction of 0.83. When the total reaches 100 percent or 1.0, the diver is considered to have reached the allowable limit, and further exposure to elevated oxygen is at increased risk. Diving beyond the limit is not recommended.

Although there has been no specific laboratory validation of this technique of interpolating the exposure times, it appears to work in practice. The NOAA oxygen exposure limits have been shown to be reasonable limits through extensive use.

TABLE 3.5
CNS Oxygen Exposure Table

Oxygen PO ₂ (atm)	Single Dive Limit (minutes)	Bottom Time Values (minutes)											
		5	10	15	20	25	30	35	40	45	50	55	60
1.20	210	2%	5%	7%	10%	12%	14%	17%	19%	21%	24%	26%	29%
1.25	195	3%	5%	8%	10%	13%	15%	18%	21%	23%	26%	28%	31%
1.30	180	3%	6%	8%	11%	14%	17%	19%	22%	25%	28%	31%	33%
1.35	165	3%	6%	9%	12%	15%	18%	21%	24%	27%	30%	33%	36%
1.40	150	3%	7%	10%	13%	17%	20%	23%	27%	30%	33%	37%	40%
1.45	135	4%	7%	11%	15%	19%	22%	26%	30%	33%	37%	41%	44%
1.50	120	4%	8%	13%	17%	21%	25%	29%	33%	38%	42%	46%	50%
1.55	82	6%	12%	18%	24%	30%	36%	42%	48%	55%	61%	67%	73%
1.60	45	11%	22%	33%	44%	56%	67%	78%	89%	100%	111%	122%	133%

Note: Oxygen exposure is a percentage of NOAA's allowable limits. The 1.60 atm PO₂ level; the oxygen clock runs more than three times as fast at 1.60 atm than at a PO₂ level of 1.40 atm. Values for intermediate PO₂ values are linearly interpolated. Values in table are normally rounded. Highlighted percentages indicate times exceeding the NOAA oxygen exposure limits.

Oxygen PO ₂	Single Dive Limit	Actual Dive Time	% Limit
1.40 atm	150 min	75 min	50%
1.30 atm	180 min	60 min	33%
	Totals:	135 min	88%

FIGURE 3.22
Multi-Level Dive Oxygen Exposure

3.3.3.3.8 Prevention of Lung or Whole-Body Toxicity

Other parts of the body are sensitive to excess oxygen, especially the lungs. Pulmonary oxygen toxicity, and in due course other whole-body aspects, can become a problem in extended or repeated oxygen-based decompressions, and in treatments in a recompression chamber. These conditions are unlikely to be encountered in nitrox diving; in fact, they are not significantly more likely than in ordinary scuba diving with air. However, procedures have been developed for managing this toxicity, and it is helpful for the diver to be acquainted with the general methods and terminology (U.S.Navy Diving Manual 1999).

On continued exposure to above normal PO₂, generally at levels below those causing CNS toxicity but above a PO₂ of 0.5 atm, the lungs may show symptoms and a reduction in vital capacity. Vital capacity is the maximum amount of gas that a person can exhale after taking a full inspiration. Although it takes training to get reproducible data, vital capacity is relatively easy to measure; it has been used as the primary indicator for pulmonary toxicity. At the laboratory of Dr. C.J. Lambertsen at the University of Pennsylvania, empirical methods were developed in the early 1970s to use vital capacity as a monitor for pulmonary effects of oxygen exposure. Among the developments was a “unit” for measuring and tracking oxygen exposure, the UPTD or Unit Pulmonary Toxicity Dose, as a function of PO₂ and time.

The dose measure was conceived around a basic unit of exposure equivalent to one minute of breathing 100 percent oxygen at a pressure of one atm. At PO₂ levels above this, the dose increases more rapidly as the PO₂ increases. This toxicity appears to have a threshold at 0.5 atm PO₂ below which toxicity development is insignificant. The unit dose for different exposure levels was determined by fitting a curve to empirical data, then deriving an equation to describe the curve. This equation is available in several references, including the *Underwater Handbook*, (Shilling et al. 1976, p. 158), which includes “look-up” tables for deriving doses from exposure data. The method also used an additional dose term, CPTD, a measure of the Cumulative Pulmonary Toxicity Dose. The method does not include a means of calculating recovery when exposure is below 0.5 atm PO₂.

A more recent approach, designated the Repex method, allows doses to be calculated or tabulated the same way using the same equation but calls the single dose unit, OTU or Oxygen Tolerance Unit (see Table 3.6).

The Repex method provides procedures for avoiding toxic effects during extended operational exposures and takes recovery into account.

Tracking OTUs is not of great importance when the dives are of a no-stop nature. It is when the diver will be conducting many dives over more than three days, and where the exposures get lengthy, that OTU tracking will be of significant value.

Table 3.6 gives the empirically determined Repex limits for whole-body oxygen exposure. The Repex limits allow a greater exposure for a diver who has not been exposed recently, but the allowable daily dose decreases as exposure days increase. The total for a given “mission” or exposure period is given in the third column. Table 3.7 facilitates calculating OTU or UPTD per minute for a range of PO₂s.

3.3.4 Indirect Effects of Pressure During Ascent

This section covers inert gas elimination, decompression sickness, counterdiffusion, and aseptic bone necrosis (dysbaric osteonecrosis).

3.3.4.1 Inert Gas Elimination

Even on land there is pressure on the body. This pressure comes from the atmosphere and dissolves nitrogen everywhere in the body until the internal nitrogen pressure reaches about the same as nitrogen pressure in the blood. It is not exactly the same as the ambient nitrogen pressure because water vapor and carbon dioxide from the body “dilutes” the air breathed. Subtracting the small water vapor pressure and arterial CO₂ values gives the blood nitrogen tension. This, more or less, is the starting nitrogen tension.

At depth, water pressure increases the nitrogen dissolved in the body. Upon ascent, or at increased elevation on land, extra nitrogen begins coming back out of the body. Ascend slowly enough and the nitrogen passes into the bloodstream, still dissolved, then travels to the lungs where it is exhaled. This process continues until the internal nitrogen pressure is again equal to ambient. Come up too fast and nitrogen can’t stay dissolved. It begins to become a gas again before it can be exhaled and forms bubbles inside the body. This triggers a cascade of problems that become decompression sickness (see Section 3.3.4.2).

Taking up inert gas by the body is called absorption or on-gassing. Giving up gas is called elimination or off-gassing. Nitrogen and carbon monoxide enter and leave the body in real and measurable units of time. The units are called half-times. Half-times refer to the time in minutes necessary to uptake or eliminate enough nitrogen to fill or empty half the area with gas. A half-time is the same as a half-life of radioactivity, which is the time needed for half the nuclei in a specific isotopic to decay.

Half-times describe real biological processes, not just theoretical numbers. How fast the body areas equilibrate with ambient pressure depends on the volume of blood flow and the capacity of the area to absorb the dissolved gas. Different areas of the body are made of different materials

TABLE 3.6
REPEX Oxygen Exposure Chart for
Tolerable Multiple Day Exposures

Exposure Days	OTU Average Dose	OTU Total Dose
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

and have varying blood supplies, so some take up nitrogen slowly, while others do it faster. These are called the slow and fast compartments, or tissues. In decompression, the term “tissue” or “compartment” means the different body areas that on-gas and off-gas at the same rate. The areas that are grouped into each compartment designation might be scattered all over the body. For example, fatty tissues hold more gas than watery tissues, and take longer than watery tissues to absorb and eliminate inert gas; these are called “slow” compartments. Fast compartments usually build higher amounts of nitrogen after a dive than slower ones because they on-gas more in the same period of time.

TABLE 3.7
OTU Calculation Table

PO ₂ (atm)	OTU Per Minute
0.50	0
0.55	0.15
0.60	0.27
0.65	0.37
0.70	0.47
0.75	0.56
0.80	0.65
0.85	0.74
0.90	0.83
0.95	0.92
1.00	1.00
1.05	1.08
1.10	1.16
1.15	1.24
1.20	1.32
1.25	1.40
1.30	1.48
1.35	1.55
1.40	1.63
1.45	1.70
1.50	1.78
1.55	1.85
1.60	1.92
1.65	2.00
1.70	2.07
1.75	2.14
1.80	2.21
1.85	2.28
1.90	2.35
1.95	2.42
2.00	2.49

When a compartment fills to capacity, it is called saturated. Given enough time, the pressure of nitrogen in all the different compartments will eventually equal ambient pressure, and thus, the entire body is saturated. On most dives, there is not enough time for total saturation. Faster compartments may be saturated, while slow compartments may be practically empty, while other compartments attain middling pressure.

Differences in solubility and rates of gas diffusion give different gases different half-times. Helium is much less soluble in tissues than nitrogen, but it diffuses faster, so helium equilibration occurs faster than for nitrogen. Still, the basic principles of absorption and elimination apply for any inert gas breathed. On ascent, for example, the diver's tissues, especially the slow compartments, may continue to absorb nitrogen. During most dives, there isn't time for slower compartments to equilibrate with ambient pressure; these compartments have a lower pressure than the surrounding water. During ascent, ambient pressure can drive nitrogen into slow tissues, even as higher pressure, fast compartments off-gas. Not all nitrogen passes directly back into the blood stream for direct off-gassing by exhalation. Nitrogen may pass from the higher pressure in one part of the body to the lower pressure in an adjacent one (Hamilton, pers. com. 2000).

Fast tissues not only on-gas quickly, they also off-gas quickly. Decompression or safety stops taken near the surface on a recreational-type dive are favorable. They allow some extra gas to be taken up by the slow tissues, but allow more gas to be given off by the faster tissues, while holding at a pressure slightly greater than the surface (Hamilton, pers. com. 2000).

After ascending to the surface (or to a shallower level), equilibration at the new level may require 24 hours or so, even though the dive was far shorter in duration. Half-time gas elimination is the reason. It takes six half-times before a compartment can fill or empty. No matter how much gas a compartment starts with, it takes six half-times to empty. A 60-minute compartment will half fill (or empty) with nitrogen in 60 minutes. After another 60 minutes, or two hours total, the compartment will be 3/4 or 75 percent full (or depleted). It will take another 60 minutes for the remaining 1/4 to move, making the compartment 7/8 or 87.5 percent full (or empty) in three hours ($1/2 + 1/4 + 1/8 = 7/8$). In four hours, the compartment will be 93.8 percent exchanged and in five hours it will be 97.0 percent. It takes six half-times for any compartment to become about 99 percent full or empty. For practical purposes 99 percent is completely saturated or de-saturated. This means a 60-minute compartment is full or empty in six hours, since six half-times x 60 minutes = 360 minutes or six hours. A fast compartment like a five-minute compartment fills or empties in only 30 minutes (6 half-times x 5 minutes = 30 minutes). The slow 120-minute compartment fills and empties in 12 hours (6 half-times x 120 minutes = 720 minutes or 12 hours).

Several complicated factors can slow the release of nitrogen from the body. However, for practical applications like calculating decompression tables, off-gassing is considered to proceed at the same half-time rate as on-gassing. This means that after any dive, it takes six hours for the 60-minute compartment to return to its starting amount of nitrogen, and 12 hours for the 120-minute half-time compartment to return to starting pressure—equilibration with ambient pressure on land.

From another perspective, oxygen can significantly enhance decompression. Decompression requirements are dictated by the on-gassing of inert gases. By breathing 100 percent oxygen, the inert gas gradient is significantly increased, thus increasing inert gas elimination from the body. For example, pure oxygen can be used to shorten decompression on the 20 and 10 fsw stops. In addition, high oxygen content mixtures can also be used to shorten decompression from the 30 fsw stop and deeper. Mixes rich in oxygen have proven to substantially improve decompression outcome when used as a supplemental decompression gas from both air and nitrox dives.

3.3.4.2 Decompression Sickness

Decompression sickness (DCS, also known as “the bends”) is the result of inadequate decompression following exposure to increased pressure. During a dive, the diver's body tissues absorb nitrogen from the breathing gas in proportion to the surrounding pressure. If the diver ascends too quickly, the nitrogen may come out of solution and form bubbles in the body's fluids and tissues.

WARNING

ALTHOUGH DECOMPRESSION SICKNESS MAY OCCUR AS A RESULT OF VIOLATING ACCEPTED SAFE DIVING PRACTICES, IT CAN ALSO OCCUR EVEN WHEN THE ACCEPTED GUIDELINES ARE BEING FOLLOWED PRECISELY.

Bubbles form after many dives, often with no symptoms; these are called silent bubbles. It's probably not true that asymptomatic bubbles form after every dive; however, they are not uncommon. Bubbles cause damage in several ways: they can block blood and lymph circulation, depriving vital areas of oxygen and waste removal; extravascular bubbles can compress and stretch blood vessels and nerves creating pain, damage, and disability; as foreign invaders to the body, bubbles can provoke a cascade of defenses including blood coagulation, release of vasoactive substances from cells lining the blood vessels, and the body's immune system reacts by coating the bubbles with lipoproteins, which then denature and release circulating fat emboli. Bubbles do not pass from body tissues into veins, unless the veins are already torn. Bubbles, even though tiny, are too big to

pass directly through blood vessel walls. They may redissolve for passage through vessel walls then reform into bubbles, but they do not drain into vessels intact. Although bubbles are a good explanation for many decompression problems, they may not be the sole precursor of decompression problems. Pressure may have direct effects of its own on blood cells and other body areas (Bookspan 1995).

It is not easy to detect bubbles in tissue, but they can be detected in circulating blood because they are moving. This is done with a device called a Doppler ultrasonic bubble detector. Ultrasonic sound waves at too high a frequency to be heard are used in various ways in medical diagnosis. Using Doppler electronics, only waves reflected from moving objects are detected. Bubbles can be “heard” moving through the circulation on the way to the lungs. Doppler bubble detectors have shown that normal and otherwise benign dives may create a few circulating bubbles in some divers. These are called “silent bubbles” because they do not cause overt symptoms. In fact, the bubbles detected in the venous blood are “on their way out” and are not likely to be involved in decompression sickness. Doppler bubble detection in venous blood has not proven to be useful for predicting DCS in a given diver, but dive profiles that cause a lot of bubbles also tend to cause a substantial number of DCS cases.

Major determinants of risk of DCS are depth, time at depth, ascent rate, and multiple dives. Individual variation is also a factor. The same depth and time profile, or “dose” of nitrogen, varies in effect on different people, just as the same dose of medication can vary in effect. Individual factors have been explored but are not well understood, leaving these variables open to sometimes wild conjecture. Other factors that may predispose to DCS include fatigue, dehydration, smoking, alcohol consumption, and carbon dioxide retention. Environmental factors include chilling at the end of a dive, heavy work, and the use of heated suits.

WARNING

DECOMPRESSION SICKNESS MAY OCCUR EVEN IF DECOMPRESSION TABLES OR COMPUTERS ARE PROPERLY USED. ALTHOUGH IT IS UNCOMMON FOR DCS TO OCCUR ON NO-DECOMPRESSION DIVES, IT CAN HAPPEN.

There was early speculation, now dismissed, that birth control pills or menstruation might increase risk for women. Given the dearth of comparative DCS studies, there is no substantive evidence that gender plays a role in DCS (Bookspan 1995). Most medical experts today agree that decompression sickness is the result of complex individual, not sex specific, factors. However, we still do not have definitive answers and additional research is needed.

WARNING

THE MAJOR DETERMINANTS OF THE RISK OF DECOMPRESSION SICKNESS ARE DEPTH, TIME AT DEPTH, ASCENT RATE, AND MULTIPLE DIVES.

Decompression sickness was formerly divided into Type I, Type II, and Type III. Type I DCS included skin itching or marbling; brief, mild pain called “niggles,” which resolved typically within ten minutes; joint pain; and lymphatic swelling. Extreme fatigue was sometimes grouped into Type I. Type II DCS was considered to be respiratory symptoms, hypo-volemic shock, cardiopulmonary problems, and central or peripheral nervous system involvement. Type III grouped DCS and arterial gas embolism together, also called decompression illness (DCI). Arterial gas embolism is covered in Section 3.3.2.4. It is now more common to categorize decompression sickness by area involved and severity of symptom.

Limb Bends. A common symptom of DCS is pain, usually in the elbow, shoulder, hip, or knee. DCS pain is often described as dull, throbbing, and deep in the joint or tissue. Pain onset is usually gradual and, in the early stages, the diver may not recognize the pain as DCS. Pain slowly intensifies, however, and, in severe cases, interferes with limb strength. In divers, upper limbs are affected about three times as often as lower limbs. In caisson workers, lower limbs are more often affected.

Central Nervous System (CNS) Decompression Sickness may cause muscular weakness, numbness, “pins and needles,” paralysis, loss of sensation, loss of sphincter control, and, in extreme cases, death. Often, the symptoms do not follow typical nerve distribution and are unstable in position and type during the early stages—different from the usual history of traumatic nerve injuries. Strange neurological complaints or findings should not be dismissed as imaginary.

Cerebral Decompression Sickness is decompression sickness occurring in the brain. It may produce almost any symptom: headache or visual disturbance, dizziness, tunnel vision, tinnitus (buzzing or ringing in the ears), partial deafness, confusion, disorientation, emotional, even psychotic symptoms, paralysis, and unconsciousness. Cerebral DCS is more common than previously thought and may account for a portion of symptoms formerly attributed to spinal-cord DCS. There is some discussion whether, and to what extent, long-term brain changes occur with repeated exposure to decompression stress, even decompression stress that does not result in known decompression sickness.

Pulmonary DCS, or “Chokes” occurs in about two percent of DCS cases. It is characterized by pain under the breastbone (sternum) on inhalation, coughing that can become paroxysmal, and severe respiratory distress that can end in death.

Skin Bends come in two forms: harmless simple itchy skin after hyperbaric chamber exposure, or rashy marbling on the torso, called cutis marmorata, that may warn of serious decompression sickness.

Inner-Ear Decompression Sickness (vestibular decompression sickness, or labyrinthine decompression sickness) produces vertigo, ringing in the ears, nausea, or vomiting. Inner ear DCS is also called “staggers” because of difficulty maintaining balance. Vestibular decompression sickness occurs more after deep helium-oxygen dives, particularly after switching to air in the later stages of decompression, although it also has occurred in shallower air diving.

It should be assumed that any diver with ear symptoms during descent is experiencing inner ear barotrauma, including possible rupture of the oval and round windows; *this diver should not be recompressed*. Recompression would again subject the diver to unequal middle-ear pressures. Even without inner ear DCS or barotrauma, hearing impairment can result from diving. Divers should have periodic audiometric examinations.

First Aid. Secure the victim’s ABC (airway, breathing and circulation). Give 100 percent O₂ through a demand/positive-pressure type mask. If necessary, CPR should begin immediately. Make the victim comfortable and place him in a supine position, take notes and record vital signs every fifteen minutes, continually monitor the victim’s level of consciousness, check for neurological deficits, conduct an interview with both the diver and his buddy regarding the cause of the accident including the diver’s profile within the last 24 hours and any pertinent medical information, collect the patient’s diving equipment and send it with the diver to the medical facility/recompression chamber. Also, the diver should only be given fluids by mouth if he is fully conscious, able to tolerate liquids, and intravenous (IV) therapy is not available. The treatment of choice for re-hydration is Ringers[®] Solution administered by IV. Mild hypovolemia may be more common in diving than generally realized. DCS treatment is less effective if hypovolemia is uncorrected. Transport without delay to a hyperbaric treatment facility. If transporting by air, ascend to an altitude or equivalent pressure of no greater than 1,000 feet. Prompt recompression treatment increases the likelihood of a favorable outcome. Several treatments may be needed. Complete resolution of symptoms is not guaranteed.

Do not treat by returning the diver to the water. In-water recompression can be hazardous and should not be attempted unless circumstances are extraordinary, for example, in isolated areas with no medical treatment options, and with specific equipment and rehearsed procedures in place for a system of surplus air, oxygen cylinders, added personnel, extra thermal protection, and land support.

NOTE

Taking vital signs and/or interviewing the injured diver must not interrupt oxygen breathing. NOAA requires that an oxygen kit capable of ventilating an unconscious victim be on site during all diving operations.

Prevention:

- Make safety stops (when conditions permit)
- Ascend slowly (30 feet per minute)
- Use longer surface intervals
- Plan the dive well and have a backup plan
- Maintain good physical fitness, nutrition, and hydration

Treatment:

- If condition permits, perform quick neurological examination before recompression to ensure that case is pain only.
- Administer 100 percent oxygen, if possible, via tight-fitting oronasal mask by demand/positive-pressure valve or non-rebreather mask at 15-lpm constant flow with the injured diver positioned supine or in recover position.
- Enter chamber, put diver on oxygen, pressurize chamber to 60 fsw, and initiate recompression on appropriate treatment table.
- Reassess diver regularly.

Remember, Divers Alert Network (DAN) provides 24 hour emergency services to assist injured divers. If assistance is required, a DAN medical professional (hyperbarically-trained physician or paramedic) can be reached at (919) 684-8111 (24 hrs).

WARNING

WHEN PLANNING DIVES, CHECK AVAILABILITY OF EMERGENCY OXYGEN/FIRST AID EQUIPMENT, CONTACT INFORMATION FOR LOCAL/REGIONAL EMERGENCY MEDICAL ASSISTANCE AND TREATMENT FACILITY, AND DEVELOP AN EMERGENCY ASSISTANCE PLAN. ANY DELAY IN SYMPTOM RECOGNITION, FIRST AID, AND TREATMENT CAN RESULT IN PERMANENT INJURY.

3.3.4.3 Treatment Tables

The primary treatment for decompression sickness is recompression. Hyperbaric oxygen therapy treatment tables include U.S.N. Treatment Tables 1, 2A, 3, 4, 5, 6, 6A, 7, 8, and 9. These tables are shown in Appendix VI, along with Accident Treatment Flow Charts to be followed when selecting a treatment strategy. The first step in any treatment involves diagnosing the condition properly. The Accident Treatment Flow Charts are diagnostic aids designed to ensure the selection of an appropriate table. Once a treatment table has been chosen, treatment is conducted in accordance with the recompression procedures specified for that table. If complications occur during or after treatment, the procedures shown in the Accident Treatment Flow Charts will help determine the appropriate course of action.

3.3.4.4 Failures of Treatment

Four major complications may affect the recompression treatment of a patient. These are:

- Worsening of the patient's condition during treatment
- Recurrence of the patient's original symptoms or development of new symptoms *during* treatment
- Recurrence of the patient's original symptoms or development of new symptoms *after* treatment
- Failure of symptoms of decompression sickness or gas embolism to resolve despite all efforts using standard treatment procedures

Alternative treatment procedures have been developed and used successfully when standard treatment procedures have failed. These special procedures may involve the use of saturation diving decompression schedules; cases of this type occur more frequently when a significant period of time has elapsed between the onset of symptoms and the initial recompression. Although it is important to know that alternative procedures are available, it is equally important to note that they have not been standardized. The use of an oxygen-nitrogen saturation therapy may be the only course of action when the situation involves a paralyzed diver already at depth whose condition is deteriorating. It is therefore essential that the advice of experts in the field of hyperbaric medicine, such as Divers Alert Network (DAN), be obtained early in the treatment process.

3.3.4.5 Counterdiffusion

Divers breathing one gas mixture while surrounded by another can develop serious skin lesions, nausea, vomiting, and vestibular problems, even with no change in ambient pressure. Problems can also occur after switching from breathing nitrogen-oxygen mix to breathing heliox while still under pressure. Different gases have different diffusion rates. Helium, for example, diffuses faster than nitrogen; in other words, helium moves into tissues from blood faster than nitrogen moves out. Total inert gas partial pressure in the body increases even though depth has not changed. This increased inert gas partial pressure can result in bubble formation. The inner ear seems particularly susceptible, resulting in vestibular symptoms. Because two different gases can go in opposite directions in the body at the same ambient pressure, it is termed isobaric counterdiffusion or isobaric counterexchange.

Interestingly, cases of inner-ear DCS have occurred after diving heliox, then switching to air. Although it would be expected that helium in tissues moves into blood faster than nitrogen in breathing air moves into tissues, which would reduce gas load and risk of DCS, it's possible that the middle ear (and other structures) fill with heliox during the dive. During the switch to air during decompression, partial pressure of helium in blood falls quickly, but the middle ear and other structures remain full of heliox, and the total inner ear inert-gas partial pressure rises.

3.3.4.6 Aseptic Bone Necrosis (Dysbaric Osteonecrosis)

Months to years after prolonged pressure exposure, joint surfaces of the long-bone ends can die. The hip and

shoulder are most often affected, resulting in pain, spasm around the joint, and finally, disabling arthritis. This condition is called avascular necrosis of bone, caisson disease of bone, aseptic bone necrosis, or dysbaric bone necrosis. The word "necrosis" means death of cells in an area. Bone necrosis, and its crippling effects, was first noted in 1888 in caisson workers (Kindwall 1972).

Aseptic bone necrosis seems to involve several mechanisms of damage: bubbles formed during decompression obstruct blood vessels in the bone ends, platelets, fat, and blood cells clump and obstruct blood flow, and blood vessels themselves narrow in reaction to bubble damage. Bone ends seem to be vulnerable because supersaturation in fatty bone marrow may generate fat emboli that occlude vessels, the surrounding bone tissue is not elastic with minimal margin for foreign body accumulation, and bone collects uranium 238 which might promote nucleation and subsequent gas bubble formation.

Bone necrosis seems to be a significant occupational hazard of professional divers, caisson workers, and others who spend great amounts of time compressing and decompressing at depth. There seems to be a definite relationship between length of time exposed to depth and bone lesions, although cases have occurred with minimal exposure. Other factors may include cases of bends, and the adequacy and promptness of recompression treatment.

Bone necrosis is seldom seen in the elbows, wrists, or ankles, and lesions that occur in the shafts of the long bones rarely cause symptoms or disability. Lesions that occur in the head of the femur (long leg bone) or humerus (upper arm bone) weaken bone underlying the cartilage covering the joint, causing the joint surface to break down. Lesions often are bilateral, resulting in the collapse of both femoral heads. Severe disability is the result. The only treatment known to have any degree of success is surgical repair or replacement of the joint.

3.3.4.7 Patent Foramen Ovale

The foramen ovale is a flap-like opening in the septum wall which separates the right and left atria of the heart. The foramen ovale is normally open in a developing fetus, because the fetus derives its oxygen and nutritional supply directly from its mother via the umbilical circulation. Upon birth, when the neonate's lungs become functional the foramen ovale functionally closes. Within a year after birth, the foramen ovale structurally closes. However, in an estimated 20 percent to 30 percent of the general population, the foramen ovale remains partially or fully open (patent "PFO"). In normal activities at sea level, a PFO does not induce any detrimental effects, and most people with PFOs are not aware of the anomaly. PFOs can be detected by means of a specialized echocardiogram, but this is an expensive and complex test which is not recommended for the general population. However, PFO can cause severe problems for divers (Bove 1997). In divers with a partially or fully open foramen ovale, performing a forceful valsalva maneuver may shift the

pressure gradient so as to open the foramen ovale and allow bubbles to shunt. If the bubbles accumulated during a dive bypass the lungs and are shunted directly into the diver's systemic circulation, they can block carotid or coronary arteries, leading to arterial gas embolism. Depending on where these shunted bubbles lodge, they may also induce DCS. PFOs have been implicated in a number of otherwise unexplained cases of decompression illness.

3.3.4.8 Pregnancy and Diving

The consensus of diving medical experts agrees that women should not dive during pregnancy. Given the limited existing data, however, it is difficult to extract specific safety guidelines. This is because animal studies may not accurately enough simulate human physiology, and anecdotal diving surveys of pregnant female divers, whose data relies on subjective reporting, is often not scientifically accurate. Additionally, because of the potential dire consequences, it is unethical and illegal to conduct experiments which purposely induce decompression illness in pregnant women and their fetuses. Nevertheless, from the existing experiences of humans and animal studies, there are risks associated with diving during pregnancy, both for the mother and her fetus (Bove 1997).

3.4 HYPOTHERMIA/HYPERTHERMIA

The body maintains internal temperature well, despite functioning in a wide range of cold environments. The body produces and loses heat several ways. By itself, the heat loss process is not a problem as long as heat is restored. Otherwise chilling results.

The body's inner, or core, temperature is the familiar 37C/98.6°F. Skin temperature is usually much lower, close to ambient temperature, and variable. It used to be popular to refer to any downward variation of body temperature as hypothermia. However, core temperature normally falls several degrees during sleep, for example, and is not hypothermia, and skin temperature drops dramatically with falling ambient temperature to protect the core against hypothermia. True clinical hypothermia is reduction of core temperature (not skin temperature) below 35C/95°F. Hypothermia is not the most common danger of cold. A diver can become incapacitated by chilling without ever becoming hypothermic.

3.4.1 Effects of Cold

Chilling, even if not life-threatening in itself, increases fatigue and reduces dexterity and sense of touch, making it difficult to do useful work or to control diving equipment such as weight belts and buoyancy compensators. Short-term memory and ability to think clearly may be seriously affected. Shivering further reduces coordination and may make it difficult to hold the mouthpiece. By the time shivering becomes uncontrollable, oxygen consumption has increased greatly. A diver may become helpless even before reaching moderate hypothermia (see Table 3.8).

Many factors interact in susceptibility to chilling. Water temperature and duration of exposure are obvious factors. Thermal protection by protective garments and the body's heat-producing and heat-saving abilities are covered in the following Section 3.4.3. Nitrogen narcosis reduces perception of cold and inhibits central neural structures involved in temperature regulation and heat production. Narcosis seems to be a large contributor to hypothermia in compressed-air divers. Susceptibility to chilling increases with dehydration, fatigue, hunger, and illness. If a diver is out of shape, underweight, a smoker, or has been using drugs or alcohol, he is at risk of chilling.

Gradual heat loss over a long period, such as multiple dives in warm water over days, often will not cause shivering; however, the accumulated slow cooling can result in impaired performance and fatigue similar to that accompanying cold water chilling.

Terminate a dive and begin rewarming if any of the following signs and symptoms are present:

Signs and Symptoms:

- Loss of dexterity and grip strength
- Difficulty performing routine tasks, confusion, or repeating tasks or procedures
- Intermittent shivering, even though routine tasks can still be performed
- Behavioral changes in a buddy that may indicate existing or approaching hypothermia

3.4.2 First Aid for Hypothermia

Treatment:

The best help that fellow divers can render at the scene of the accident is:

- ABC (airway, breathing, and circulation)
- Handle the victim extremely gently
- Prevent further heat loss
- Activate the EMS system immediately

WARNING

SEVERE HYPOTHERMIA IS A LIFE-THREATENING CONDITION AND NEEDS TO BE TREATED BY TRAINED MEDICAL PERSONNEL.

ABC. As in any medical emergency, protecting the ABC is the utmost priority. In addition to securing and monitoring the victim's airway, breathing, and circulation, it is also important to determine the victim's temperature.

Treat the Victim Gently. A victim of severe hypothermia must be carefully removed from the water in as horizontal a position as possible to reduce the possibility of hypotension (reduced blood pressure) and shock. The victim should be removed from the cold environment, and sheltered. The victim should be kept lying down in a supine

position, but not in direct contact with the cold ground or metal objects, and should always be handled very gently.

Prevent Further Heat Loss. To prevent further heat loss, rescuers should remove the victim’s wet clothes, and cover him with blankets, particularly around the areas of highest heat loss—the head, neck, armpits, chest, and groin. Never attempt to rewarm a severely hypothermic diver in

the field. Ideally, hypothermia victims should be stabilized in a hospital setting and carefully rewarmed under medical supervision.

It is widely debated whether and by which method even trained medics should attempt field rewarming, because rapid or aggressive rewarming may precipitate a phenomenon known as “afterdrop,” in which the core temperature continues to drop even when rewarming has begun. Afterdrop is believed to occur when cold blood in the periphery circulates to the central core as vessels in the skin dilate from the warm environment. The heart of a severely hypothermic person is extremely vulnerable, and afterdrop can induce ventricular fibrillation (uncontrolled, irregular heart beats). Well-intentioned “remedies” such as rigorous rubbing of the victim’s extremities, heat packs, hot drinks, hot baths, alcoholic beverages, even a cigarette, therefore, can be lethal.

Activate the EMS System. Divers should always have a dive accident management plan, which includes information and equipment for contacting the local EMS and U.S. Coast Guard.

Fortunately, divers rarely have to deal with severe hypothermia. It is more likely that a diver will appear cold or complain of being cold, will be shivering and/or have slightly impaired speech and dexterity. Other divers or the diving supervisor should remove this diver from the water and wind immediately, remove wet clothing, and dry him off. As long as the diver is not shivering uncontrollably, is conscious, has a core temperature of 95°F (35°C) or more, and can swallow, he can be given warm drinks that contain no alcohol or caffeine, and should be wrapped in warm blankets or an exposure bag. A chilled person can warm up by taking a warm bath or shower.

Often a lay rescuer cannot distinguish between the various categories of hypothermia, as signs and symptoms may overlap. Good indicators are the diver’s level of consciousness, temperature, and intensity of shivering (or lack of shivering). If unsure, a lay rescuer should refrain from aggressive rewarming.

WARNING

DO NOT TAKE HOT BATHS OR SHOWERS AFTER COMPLETING DECOMPRESSION DIVES (OR DIVES NEAR DECOMPRESSION LIMITS). HEAT MAY STIMULATE BUBBLE FORMATION.

3.4.3 Thermal Protection

A variety of diving suits are available, from standard foam neoprene wet suits and dry suits to specially heated suits.

NOTE

A wet suit does not stop heat loss, it merely slows it.

Diving in water temperature below 50°F usually requires a dry suit, which provides insulation by maintaining a dry air space between the suit and the diver’s skin.

TABLE 3.8
Signs and Symptoms of
Dropping Core Temperature

Core Temperature		Symptoms
°F	C	
Below 98.6	37	CHILLING Cold sensations, skin vasoconstriction, increased muscle tension, increased oxygen consumption
97	36	Sporadic shivering suppressed by voluntary movements, gross shivering in bouts, further increase in oxygen consumption, uncontrollable shivering
95	35	MODERATE HYPOTHERMIA Voluntary tolerance limit in laboratory experiments; mental confusion, impairment of rational thought, possible drowning, decreased will to struggle
93	34	Loss of memory, speech impairment, sensory function impairment, motor performance impairment
91	33	Hallucinations, delusions, partial loss of consciousness; in shipwrecks and survival history, 50% do not survive; shivering impaired
90	32	SEVERE HYPOTHERMIA Heart rhythm irregularities, motor performance grossly impaired
88	31	Shivering stopped, failure to recognize familiar people
86	30	Muscles rigid, no response to pain
84	29	Loss of consciousness
80	27	Ventricular fibrillation (ineffective heartbeat), muscles flaccid
79	26	Death

However, if flooded, the suit loses its insulating value and can become a severe thermal hazard. Protective suits create an interesting complication. The body's defense is reduced by the thermal barrier of the clothing. This complication, long known, is only just being recognized as an important contributor to designing protective systems.

Body fat, ability to generate heat, ability to constrict blood vessels in the limbs to shunt and save heat for the core, physical conditioning, and regular cold exposure are important contributors to cold tolerance and protection.

There is evidence that vasoconstriction, a heat-preservation response, may be highly efficient in women. During vasoconstriction, blood vessels in the shell narrow and restrict cutaneous blood flow, thereby decreasing convective heat transfer from core to skin and subsequent loss to the environment and shunt the warm blood to the vital organs.

Vasoconstriction, however, is only one of many factors involved in thermal stress. Each diver will respond to the cold water environment based on his own specific physiological makeup, level of training and conditioning, and the environmental factors in that particular situation.

Prevention of Hypothermia:

- Check air and water temperature conditions before the dive.
- Wear adequate thermal protection for the dive.
- After a dive, get out of wet clothes.
- Move to a warm, protected area.
- Dry your hair.
- Wear a hat.
- Drink warm liquids in between dives.
- When considering adequate thermal protection, factor in the duration of decompression or safety stops.
- Be adequately nourished, stay well hydrated, and avoid alcohol and caffeine.
- Repetitive dives should not be made until diver is completely rewarmed.
- For maximum cold water performance, divers should swim in cold water on a regular basis to improve cold tolerance.

3.4.4 Thermal Stress Irrespective of Ambient Temperature

Hypothermia is not a problem only in frigid environments and can occur irrespective of ambient temperature. Similarly, divers may also suffer extremes of hot and cold thermal stress simultaneously during the same dive. There have been documented cases of severe heat exhaustion in Arctic waters by commercial divers as a result of wearing thick, occlusive dry suits, aggravated by dehydration from breathing dry compressed gas and perspiring from prolonged underwater swimming or heavy underwater work. Perspiration from excessive or from pre-dive overheating can also cause the diver's dry suit underwear to lose insulation, thus predisposing him to hypothermia.

Warm Water Hypothermia. Divers also have to be wary of hypothermia in warm environments. A phenomenon called "warm water hypothermia" can occur even in the tropics, especially during long dives and repetitive dives made without adequate rewarming between dives. In warm water hypothermia, long slow cooling can take place in water temperatures as warm as 82°–91°F (27–33°C). Although warm water hypothermia is not as easily recognized as its cold water counterpart, it definitely warrants attention. The physiological mechanisms of warm water hypothermia have been demonstrated in various medical studies, but they still are not clearly understood. The victim in this situation may not shiver, because the drop in core temperature may not be rapid enough to activate the body's thermoregulator defense mechanism. There may be a discrepancy between the input of the receptors in the body's shell and core, making the diver's skin feel warm while his core is cooling. Warm water hypothermia can cause confusion, fatigue, apathy, incoordination, delayed reaction time, and sudden anxiety. These mental and physical disabilities, especially when concurrent with any problems during the dive itself, can result in panic, embolization, and drowning.

3.4.5 Survival in Cold Water

When diving, wear thermal protection appropriate for the water temperature. Although exercise increases heat transfer to the water, it is not always the case that the only outcome of swimming or other movement is net heat loss. Heavy exercise can generate enough heat to match heat loss in cold water. Because it is more common to chill in very cold water even with exercise, the common recommendation is to remain still, not swim in very cold water.

If ship abandonment is necessary, specific procedures increase chance of survival. Ship sinkings, even in worst cases, usually require, at minimum, 15 to 30 minutes, affording valuable preparation time. Being prepared and practiced makes best use of this time:

- Don a personal flotation device immediately.
- Wear several layers of clothing because the trapped air provides insulation. Even in the water, extra layers of clothing reduce the rate of body heat loss.
- Board a lifeboat or raft as soon as possible to avoid wetting insulating clothing and losing body heat.
- If it is necessary to enter the water, enter slowly to minimize likelihood of increasing breathing rate, swallowing water, wetting the face and head, shock, and death.
- Once in the water, orient to lifeboats, floating objects, etc. Button up, turn on signal lights immediately, before manual dexterity is lost.
- Keep head and neck out of the water. Protect head, neck, groin, and the sides of the chest—these are areas of rapid heat loss.
- In extremely cold water, do not attempt to swim except to a nearby craft, fellow survivor, or floating object.

- To conserve body heat, hold knees against chest, arms around the side of chest. This is called the Heat Escape Lessening Position (HELP). If others are nearby, huddle together and maintain maximum body contact.
- Keep a positive attitude. Will to live makes a difference.

3.4.6 Overheating and Hyperthermia

The body's adaptation to overheating involves complex integrations between the circulatory, neurologic, endocrine, and exocrine functions. As it does in response to cold stress, when exposed to ambient heat, the body's core temperature is regulated by the control center in the hypothalamus, which reacts to changes in the temperature of the circulating blood and to impulses from thermal receptors in the body's shell. Whenever core temperature rises above normal, the heat-promoting center in the hypothalamus is inhibited. Concurrently, the heat-loss center in the brain is stimulated, resulting in vasodilation. Through vasodilation, heat is dissipated from the shell through conduction, convection, and radiation. If the external environment is so hot or the body is so overheated that heat cannot be lost by conduction, the sudorific (sweating) mechanism is activated, allowing heat to escape as the sweat evaporates. At high ambient temperatures and during exercise, sweating provides the major physiologic defense against overheating. As sweating causes the body to lose fluid and electrolytes, hormonal adjustments begin. Vasopressin or antidiuretic hormone is released by the pituitary gland and the hormone aldosterone, which helps conserve sodium, is released from the adrenal cortex.

As core temperature continues to rise beyond the homeostatic range (100°F/37.8°C), the body's natural heat loss processes become ineffective, the hypothalamus is depressed, biochemical reactions are impaired, and proteins begin to degrade. At 106°F (41°C), most people go into convulsions. The outside limit for human life is 108°F (43°C).

3.4.7 Types of Heat Stress

Heat syncope is the sudden loss of consciousness due to heat. It is usually experienced by individuals undergoing prolonged exposure to a hot environment to which they are not acclimatized, or by individuals who have been moving about in extreme heat while dressed in heavy garments (i.e., tenders on long duty, fully dressed scuba divers).

Heat cramps are a mild response to heat stress, and are manifested by muscle cramps. Cramping usually occurs in the legs, arms, or abdomen, and may occur several hours after exercise. If unaccompanied by serious complications, heat cramps are best treated by rest, oral fluids, cooling down, ice, and stretching and massaging the muscles. For severe cramping, electrolyte replacement drinks or salt tablets may be indicated.

Heat exhaustion is a serious problem in which hypovolemia (low blood volume) develops as a result of fluid loss. Heat exhaustion often develops in unacclimatized people and is evidenced by profuse sweating, nausea, vomiting, a weak and rapid pulse, ataxia, low blood pressure, headache, dizziness, altered mental state, and general weakness, and may require medical attention. Victims of severe heat exhaustion should be given IV fluids, cooled aggressively (e.g. with an ice bath), and possibly transported to an emergency medical facility.

Heat stroke, the most serious and complex heat disorder, is a serious, life-threatening medical emergency. When hyperthermia has progressed to heat stroke, the body's thermoregulatory mechanism, or capacity to cool itself by sweating, has failed and core temperatures can soar to above 105°F, leading to convulsions, delirium, and coma. The skin becomes hot and dry. As the temperature spirals upward, permanent brain damage may occur. If left untreated, heat stroke can result in death due to circulatory collapse and damage to the central nervous system. Victims of heat stroke must be stabilized, removed from the hot environment immediately, cooled aggressively, put in the shock position (legs slightly elevated), given IV fluid replacement, and be transported to an emergency medical facility.

Unlike chilling, overheating rarely results from immersion in water. However, if water temperature is high, around 86°F (29.4°C), there is little or no difference between the skin and water temperature; heat has no gradient to transfer to the water. Any exercise under such conditions can end in overheating. Even in cooler water, heavy exercise can generate more heat than is lost, and the diver can become warm. However, hyperthermia under water has only recently been a subject of attention, and primarily by military and commercial dive operations that put divers in waters near the Equator, the Persian Gulf, etc., or in hazardous warm environments (nuclear reactor coolant pools) that require dry suits for protection.

To reduce the risk of overheating, drink water and juices liberally. Drink before thirsty. Avoid alcohol, coffee, and other fluids which act as diuretics. Avoid drugs that increase susceptibility to overheating. To acclimate to the climatic conditions, gradually and regularly increase heat exposure. Get into good physical condition—it will greatly extend heat tolerance.

The prevention of hyperthermia in these specialized dive situations involves a number of strategies, including heat acclimatization, specialized equipment (i.e. suit-under-suit or SUS), ice vests, pre-cooling, etc. Other suggestions to add to those for preventing hyperthermia are:

- Dive buddy teams should suit up in sync, particularly on a hot day, to minimize time spent above the water enclosed in a dry suit or wet suit. If divers

- cannot suit up together, the first buddy to get dressed should wait in the water and cool off.
- Wear a hat or visor, and use a high SPF broad-spectrum sunscreen before and after diving.
- Ingest salt only if needed. Individuals who tend to sweat copiously can use salt with meals, but should avoid salt tablets, which can cause excessive body salt levels.

Protective dress required for diving in contaminated water can lead to overheating and/or hypothermia in warm water situations (see Chapter 13, Polluted-Water Diving).

3.5 DRUGS AND DIVING

The use of prescription and over-the-counter medications while diving is a complex issue. Drug interaction is an enormous topic; it is difficult to know all the variables, all the possible drugs, and effects or changes caused by diving. Individual variability, existing medical and physical conditions, and the mental and physical requirements of the specific dive are additional variables.

3.5.1 Prescription Drugs

The hyperbaric environment of diving may change how some drugs act in the body. Specific concerns include:

- How the body absorbs, metabolizes, and excretes the drug.
- Possible physical effects of the type of breathing gas, increased density of the gases, water temperature, and other environmental factors.
- Side effects; on the surface, side effects, like drowsiness from antihistamines, may be acceptable. Under water, as with the operation of machinery on land, any impairment of cognitive function, neuromuscular strength, coordination, or integration of thought and action may lead to accidents.

There are several commonly used drugs that may affect diver safety, performance, and the diver's ability to thermoregulate. These drugs include beta blockers, motion sickness remedies, antihistamines, amphetamines, tranquilizers, sedatives, hypertensive drugs, and decongestants. Before diving, consult with your physician and ask the following questions:

- What is the underlying condition/illness/disease? Is it relative or absolute contraindication to diving?
- What is the half-life of the drug, and how long before or after use would it be prudent to avoid a high-pressure environment?
- What are any side effects that might increase risk of diving?
- Does the drug interfere with physical performance or exercise tolerance?

- Does the drug interfere with consciousness or cause alteration in decision-making ability?
- Does the drug produce rebound phenomena?

Divers and their physicians have an obligation to communicate with each other. The clinician has the responsibility to explain the nature of treatment to the diver, and the diver has the responsibility to inform the physician that diving exposure is anticipated, and what other drugs they may be taking.

3.5.2 Smoking

Cigarette smoke contains poisons in gas and particulate form, including hydrogen cyanide, nitrogen oxides, and carbon monoxide. Smoking directly affects the respiratory and cardiovascular systems; it creates toxic effects throughout the body ranging from bone-cell destruction to cancerous changes. Additionally, smoking is addictive.

In the respiratory system, poisons deposit on the mucous lining of the airways and lungs. Over time, they irritate the air spaces, scar the lungs, and damage the cilia (thousands of microscopic hairs lining the airways). Cilia normally move mucus, and the pollutants that accumulate in the mucus, out of the lungs to the throat. The mucus is usually swallowed or blown out the nose. Smoking paralyzes the cilia; pollutants stay in the lungs, increasing the smoker's risk of bronchitis, influenza, and other respiratory infections. The accumulation of secretions can make equalizing ear and sinus pressure difficult. Smoking eventually produces structural weakness in the lung, such as irreversibly enlarged and useless alveoli, leading to a lung disease called emphysema.

Smoking affects the cardiovascular system, accelerating atherosclerotic changes in blood vessels, damaging heart tissue, and limiting the oxygen-carrying ability of red blood cells. Inhaled nicotine and carbon monoxide increase stickiness of blood platelets, causing clumping that can block blood flow in the small vessels. It is speculated that increased clumping increases susceptibility to decompression sickness. Cigarette smoke is directly toxic to bone cells and the discs in the back, increasing risk of back pain and disc degeneration.

The dose of carbon monoxide a smoker receives from smoking is toxic; it causes fatigue, headache, irritability, dizziness, and disturbed sleep, as well as changes in neurologic reflexes, psychomotor test results, sensory discrimination, and electrocardiograms. Carbon monoxide concentration inhaled from smoking one cigarette averages 400 to 500 ppm, producing up to ten percent carboxyhemoglobin (HbCO) (see Table 3.9). The level in non-smokers is generally 0.5 percent.

The HbCO level in the blood of divers who smoke is higher than it would be if they were exposed to 20 ppm carbon monoxide for 12 hours (maximum carbon monoxide level allowed in divers' breathing air by NOAA).

A heavy smoker takes approximately eight hours to eliminate 75 percent of the carbon monoxide inhaled.

TABLE 3.9
Carboxyhemoglobin
as a Function of Smoking

Smoking Habits	Median HbCO Level, %	Expired CO, ppm
Light smoker (less than 1/2 pack/day)	3.8	17.1
Moderate smoker (more than 1/2 pack/day and less than 2 packs/day)	5.9	27.5
Heavy smoker (2 packs or more/day)	6.9	32.4

The HbCO level, even for a light smoker diving eight hours after the last cigarette (0.95%), is almost twice that of a non-smoker (0.50%). The carboxyhemoglobin level of a person who does not smoke, but is exposed to the smoke of others (passive smoke), can rise to five percent after exposure.

Epidemiologists have discovered that smoking is implicated not only in fatal lung disorders and coronary artery disease, but also in strokes, bladder cancer, cervical cancer, hearing deficits, and is a serious risk factor during pregnancy.

Each cubic centimeter of tobacco smoke contains over five million particles, including chemicals which are so dangerous that they are on the Environmental Protection Agency's list of substances which are illegal to dispose of in the environment. According to the American Lung Association, "cigarette smoke in its gaseous and particulate phases contains 4,700 compounds, including 43 known carcinogens, which can damage tissues and cause disease."

For divers, the respiratory deficits caused by smoking can be especially dangerous if the diving activities involve deep exposures which create breathing resistance, thermal stress, swimming against strong currents or a number of factors which necessitate optimal aerobic capacity and increased supply of oxygen. Intuitively, it seems likely that carboxyhemoglobin may not be able to carry as much CO₂ as normal hemoglobin does. If so, carbon dioxide toxicity and decompression sickness (CO₂ is believed to be a predisposing factor to DCS) cannot be ruled out. There is yet another hazard associated with carbon monoxide, one which pathologists do not yet fully understand. Long term exposure to CO results in the CO binding to the cellular enzyme cytochrome oxidase, an enzyme necessary for the transfer of oxygen from the blood to the inside of the cells.

Even a moderate smoker will have about six to eight percent of his hemoglobin tied up with carbon monoxide, and therefore will have the oxygen-carrying capacity of the blood reduced by that amount. Carbon monoxide has a 220 to 290-fold greater affinity for hemoglobin than oxygen does, and therefore readily combines with this vital component of the blood to produce carboxyhemoglobin. For every molecule of carbon monoxide in the blood, the blood can carry one less molecule of oxygen. For heavy chronic smokers, the oxygen-carrying capacity of blood can be reduced by as much as ten percent. Anyone with a history of unconsciousness, or anyone exhibiting confusion or other neurological signs, no matter how good they look upon admission, must be treated with hyperbaric oxygen (Kindwall 1999).

3.5.3 Illicit Drugs and Alcohol

Psychoactive agents impair cognitive and motor performance, the very basis of their use.

Alcohol, barbiturates, and marijuana are commonly abused nervous system depressants. Depressed motor function is hazardous under water. Risk of cold injury and nitrogen narcosis increases, and, as blood glucose falls, which is another side effect of these drugs, weakness and confusion increase. Because of its diuretic action, alcohol can contribute to dehydration, particularly in the tropics. Drugs take time to leave the system, meaning their hazards may persist even days after taking them.

Cocaine and the many other commonly abused central nervous system stimulants render a diver incapable of responding properly to life-threatening emergencies. These drugs are often combined with alcohol or marijuana, thus compounding problems. Cocaine increases the likelihood of an oxygen toxicity seizure; it can trigger abnormal heart beats, sudden heart attack, even in a young person, and heart illness.

Other Important Facts About Alcohol. Alcohol interferes with the body's ability to replenish the energy the body needs after diving, and it causes a dramatic drop in blood-glucose level, leading to hypoglycemia which can cause weakness, confusion, irritability, interference with temperature maintenance, and fainting. According to the American College of Sports Medicine, because alcohol is a depressant of the central nervous system, even a small amount can disrupt a wide variety of psychomotor skills, reaction time, hand-eye coordination, alertness, accuracy, balance, and complex coordination. Because of these detriments, alcohol is banned by various federations within the International Olympic Committee. Diving under these circumstances can be even more hazardous.

Alcohol by itself causes acutely diminished mental and physical faculties; alcohol consumption combined with breathing compressed gas may accelerate and multiply the effects of nitrogen narcosis.

Alcohol can enhance exercise fatigue by increasing lactic acid production, which will make even non-stressful swimming and diving much more difficult.

The decreased strength, power, local muscular, and overall cardiovascular endurance caused by alcohol may become life-threatening detriments in an emergency diving situation. For a diver who drinks and dives, an unexpected problem (i.e., having to swim a great distance on the surface, struggling against a strong current, rescuing another diver, etc.) may intensify into a diving accident or fatality.

Alcohol is considered a factor in many drownings and diving accidents. In analysis of large numbers of drownings in the U.S. and Australia, about 80 percent of the adult victims had elevated blood alcohol levels (BAL). According to DAN Accident Report data, approximately one-third of the reported diving accident victims had consumed alcohol within 12 hours before or after diving. Tests have also shown that some individuals still have a BAL above the legal limit for driving 24 hours after their last drink (Plueckhahn 1984). There are some divers, therefore, who ideally should refrain from drinking alcohol 24 hours before and after a dive.

It's important to drink a lot of fluids before and after diving, but those fluids should be water and fruit juices, not alcoholic beverages. Alcohol is a diuretic, i.e., a substance that causes greater loss of fluids than it contains. Not only fluid, but essential minerals and electrolytes are lost through diuresis (urination). Alcohol inhibits the brain hormone ADH (Antidiuretic Hormone), creating a vicious cycle whereby the more alcohol a person drinks, the more he urinates, which leads to further dehydration. When the brain becomes

dehydrated, the individual experiences dizziness, headache, and a "hangover" feeling.

For divers, dehydration resulting from alcohol consumption poses a number of problems. Dehydration creates hypovolemia (thicker blood volume), resulting in slower off-gassing of nitrogen. This makes alcohol a major risk factor in decompression sickness, particularly bends with serious neurological deficits. According to DAN, there appears to be a relationship between an increased number of drinks and the severity of decompression illness. In a study on alcohol and bends, it was reported that alcohol can reduce the surface tension, a force which limits bubble growth, and therefore may encourage bubble formation (Edmonds, Lowry, and Penefather 1992). Additionally, the vasodilation of capillaries caused by alcohol may allow nitrogen to escape too rapidly, increasing chances of decompression sickness (DCS) even more.

Alcohol also predisposes a diver to thermal stress. As alcohol dilates the peripheral blood vessels, circulation is diverted to the skin and heat escapes. In cold climates, this impairment in thermoregulation may deteriorate into a life-threatening state of hypothermia.

One of the first signs of hypothermia is shivering, which concurrently promotes a faster rate of nitrogen elimination. In hot environments, alcohol can increase sweating, which leads to further dehydration, and precipitates hyperthermia, a state of elevated body core temperature, which can progress to heat stroke.

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