C H A P T E R 3
Underwater Physiology

3-1 INTRODUCTION

3-1.1 **Purpose.** This chapter provides basic information on human physiology and anatomy as it relates to working in the underwater environment. Physiology is the study of the processes and functions of the body. Anatomy is the study of the structure of the organs of the body.

3-1.2 **Scope.** This chapter contains basic information intended to provide a fundamental understanding of the physiological processes and functions that are affected when humans are exposed to the underwater environment. A diver's knowledge of underwater physiology is as important as a knowledge of diving gear and procedures. Safe diving is only possible when the diver fully understands the fundamental physiological processes and limitations at work on the human body in the underwater environment.

3-1.3 **General.** A body at work requires coordinated functioning of all organs and systems. The heart pumps blood to all parts of the body, the tissue fluids exchange dissolved materials with the blood, and the lungs keep the blood supplied with oxygen and cleared of excess carbon dioxide. Most of these processes are controlled directly by the brain, nervous system, and various glands. The individual is generally unaware that these functions are taking place.

As efficient as it is, the human body lacks effective ways of compensating for many of the effects of increased pressure at depth and can do little to keep its internal environment from being upset. Such external effects set definite limits on what a diver can do and, if not understood, can give rise to serious accidents.

3-2 THE NERVOUS SYSTEM

The nervous system coordinates all body functions and activities. The nervous system comprises the brain, spinal cord, and a complex network of nerves that course through the body. The brain and spinal cord are collectively referred to as the *central nervous system* (CNS). Nerves originating in the brain and spinal cord and traveling to peripheral parts of the body form the *peripheral nervous system* (PNS). The peripheral nervous system consists of the cranial nerves, the spinal nerves, and the sympathetic nervous system. The peripheral nervous system is involved in regulating cardiovascular, respiratory, and other automatic body functions. These nerve trunks also transmit nerve impulses associated with sight, hearing, balance, taste, touch, pain, and temperature between peripheral sensors and the spinal cord and brain.

3-3 THE CIRCULATORY SYSTEM

The circulatory system consists of the heart, arteries, veins, and capillaries. The circulatory system carries oxygen, nutrients, and hormones to every cell of the body, and carries away carbon dioxide, waste chemicals, and heat. Blood circulates through a closed system of tubes that includes the lung and tissue capillaries, heart, arteries, and veins.

3-3.1 **Anatomy.** The very large surface areas required for ample diffusion of gases in the lungs and tissues are provided by the thin walls of the capillaries. Every part of the body is completely interwoven with intricate networks of extremely small blood vessels called capillaries. In the lungs, capillaries surround the tiny air sacs (alveoli) so that the blood they carry can exchange gases with air.

3-3.1.1 **The Heart.** The heart (Figure 3-1) is the muscular pump that propels the blood throughout the system. It is about the size of a closed fist, hollow, and made up almost entirely of muscle tissue that forms its walls and provides the pumping action. The heart is located in the front and center of the chest cavity between the lungs, directly behind the breastbone (sternum).

The interior of the heart is divided lengthwise into halves, separated by a wall of tissue called a septum, that have no direct conduit to each other. Each half is divided into an upper chamber (the atrium), which receives blood from the veins of its circuit and a lower chamber (the ventricle) which takes blood from the atrium and pumps it away via the main artery. Because the ventricles do most of the pumping, they have the thickest, most muscular walls. The arteries carry blood from the heart to the capillaries; the veins return blood from the capillaries to the heart. Arteries and veins branch and rebranch many times, very much like a tree. Trunks near the heart are approximately the diameter of a human thumb, while the smallest arterial and venous twigs are microscopic. Capillaries provide the connections that let blood flow from the smallest branch arteries (arterioles) into the smallest veins (venules).

3-3.1.2 **The Pulmonary and Systemic Circuits.** The circulatory system consists of two circuits with the same blood flowing through the body. The pulmonary circuit serves the lung capillaries; the systemic circuit serves the tissue capillaries. Each circuit has its own arteries and veins and its own half of the heart as a pump. Figure 3-2 shows how the circulatory system is arranged. In complete circulation, blood first passes through one circuit and then the other, going through the heart twice in each complete circuit.

3-3.2 **Circulatory Function.** Blood follows a continuous circuit through the human body. Blood leaving a muscle or organ capillary has lost most of its oxygen and is loaded with carbon dioxide. The blood flows through the body's veins to the main veins in the upper chest (the superior and inferior vena cava). The superior vena cava receives blood from the upper half of the body; the inferior vena cava receives blood from areas of the body below the diaphragm. The blood flows through the main veins into the right atrium and then through the tricuspid valve into the right ventricle.
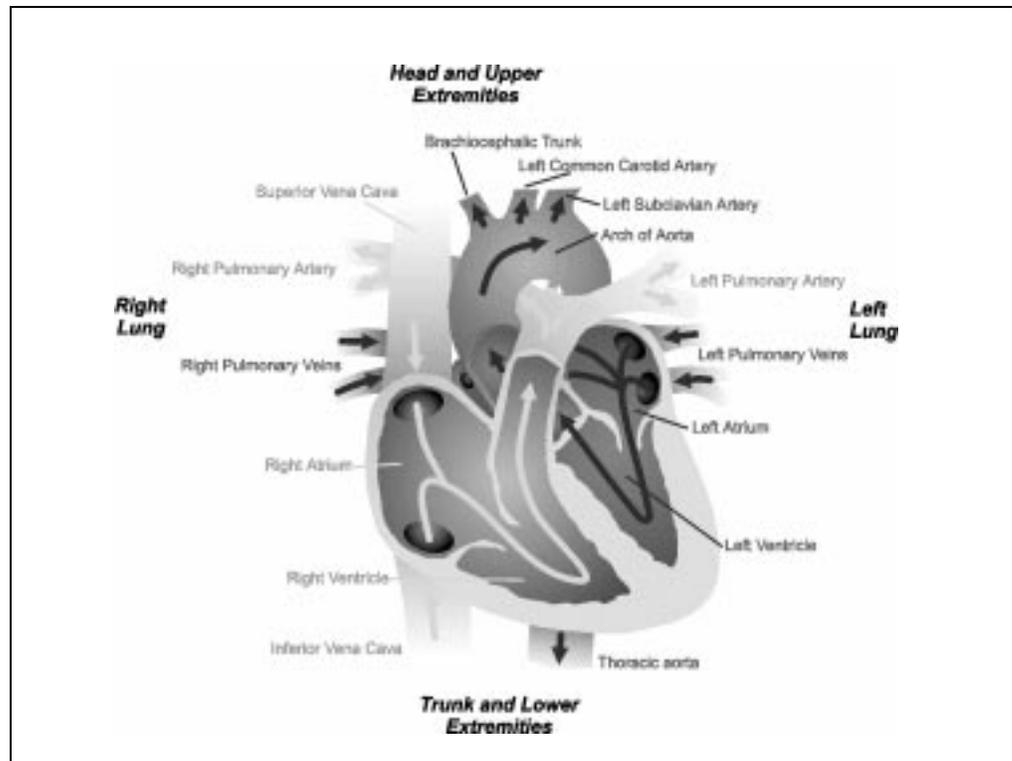
Figure 3-1. The Heart's Components and Blood Flow.

The next heart contraction forces the blood through the pulmonic valve into the pulmonary artery. The blood then passes through the arterial branchings of the lungs into the pulmonary capillaries, where gas transfer with air takes place. By diffusion, the blood exchanges inert gas as well as carbon dioxide and oxygen with the air in the lungs. The blood then returns to the heart via the pulmonary venous system and enters the left atrium.

The next relaxation finds it going through the mitral valve into the left ventricle to be pumped through the aortic valve into the main artery (aorta) of the systemic circuit. The blood then flows through the arteries branching from the aorta, into successively smaller vessels until reaching the capillaries, where oxygen is exchanged for carbon dioxide. The blood is now ready for another trip to the lungs and back again.

The larger blood vessels are somewhat elastic and have muscular walls. They stretch and contract as blood is pumped from the heart, maintaining a slow but adequate flow (perfusion) through the capillaries.

3-3.3 **Blood Components.** The average human body contains approximately five liters of blood. Oxygen is carried mainly in the red corpuscles (red blood cells). There are approximately 300 million red corpuscles in an average-sized drop of blood. These corpuscles are small, disc-shaped cells that contain hemoglobin to carry oxygen. Hemoglobin is a complex chemical compound containing iron. It can form a loose chemical combination with oxygen, soaking it up almost as a sponge
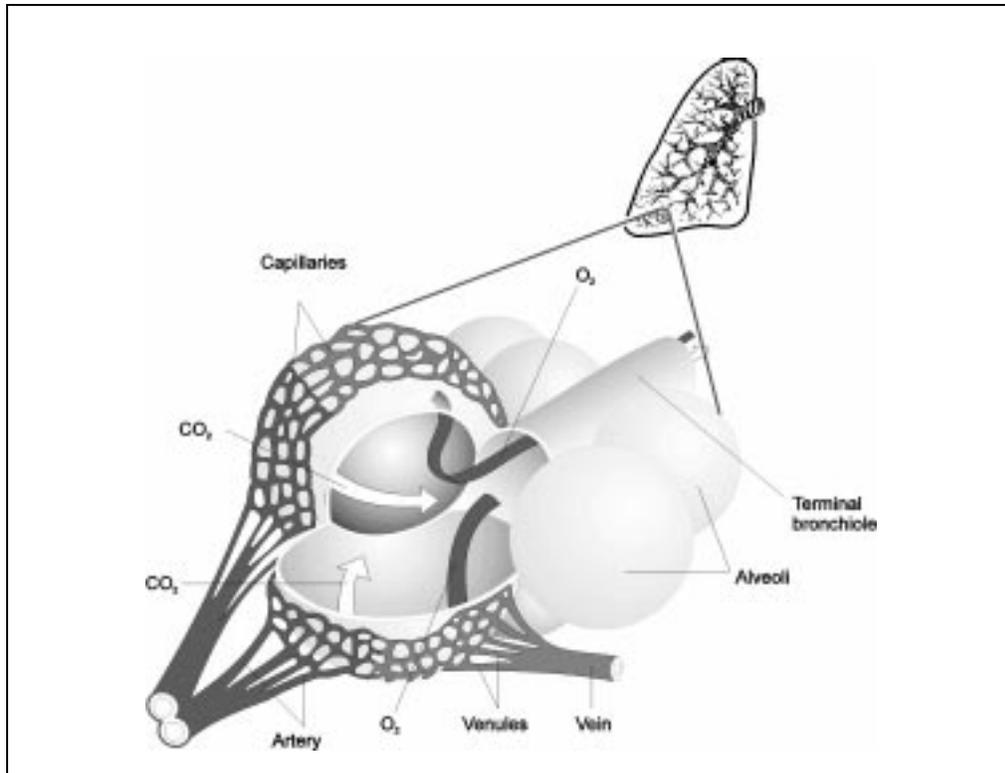
Figure 3-2. Respiration and Blood Circulation. The lung's gas exchange system is essentially three pumps. The thorax, a gas pump, moves air through the trachea and bronchi to the lung's air sacs. These sacs, the alveoli, are shown with and without their covering of pulmonary capillaries. The heart's right ventricle, a fluid pump, moves blood that is low in oxygen and high in carbon dioxide into the pulmonary capillaries. Oxygen from the air diffuses into the blood while carbon dioxide diffuses from the blood into the air in the lungs. The oxygenated blood moves to the left ventricle, another fluid pump, which sends the blood to the systemic capillaries which deliver oxygen to and collect carbon dioxide from the body's cells.

soaks up liquid. Hemoglobin is bright red when it is oxygen-rich; it becomes increasingly dark as it loses oxygen. Hemoglobin gains or loses oxygen depending upon the partial pressure of oxygen to which it is exposed. Hemoglobin takes up about 98 percent of the oxygen it can carry when it is exposed to the normal partial pressure of oxygen in the lungs. Because the tissue cells are using oxygen, the partial pressure (tension) in the tissues is much lower and the hemoglobin gives up much of its oxygen in the tissue capillaries.

Acids form as the carbon dioxide dissolves in the blood. Buffers in the blood neutralize the acids and permit large amounts of carbon dioxide to be carried away to prevent excess acidity. Hemoglobin also plays an important part in transporting carbon dioxide. The uptake or loss of carbon dioxide by blood depends mainly upon the partial pressure (or tension) of the gas in the area where the blood is exposed. For example, in the peripheral tissues, carbon dioxide diffuses into the blood and oxygen diffuses into the tissues.

Blood also contains infection-fighting white blood cells, and platelets, which are cells essential in blood coagulation. Plasma is the colorless, watery portion of the blood. It contains a large amount of dissolved material essential to life. The blood also contains several substances, such as fibrinogen, associated with blood clotting. Without the clotting ability, even the slightest bodily injury could cause death.

3-4   THE RESPIRATORY SYSTEM

Every cell in the body must obtain energy to maintain its life, growth, and function. Cells obtain their energy from oxidation, which is a slow, controlled burning of food materials. Oxidation requires fuel and oxygen. Respiration is the process of exchanging oxygen and carbon dioxide during oxidation and releasing energy and water.

3-4.1   **Gas Exchange.** Few body cells are close enough to the surface to have any chance of obtaining oxygen and expelling carbon dioxide by direct air diffusion. Instead, the gas exchange takes place via the circulating blood. The blood is exposed to air over a large diffusing surface as it passes through the lungs. When the blood reaches the tissues, the small capillary vessels provide another large surface where the blood and tissue fluids are in close contact. Gases diffuse readily at both ends of the circuit and the blood has the remarkable ability to carry both oxygen and carbon dioxide. This system normally works so well that even the deepest cells of the body can obtain oxygen and get rid of excess carbon dioxide almost as readily as if they were completely surrounded by air.

If the membrane surface in the lung, where blood and air come close together, were just an exposed sheet of tissue like the skin, natural air currents would keep fresh air in contact with it. Actually, this lung membrane surface is many times larger than the skin area and is folded and compressed into the small space of the lungs that are protected inside the bony cage of the chest. This makes it necessary to continually move air in and out of the space. The process of breathing and the exchange of gases in the lungs is referred to as *ventilation* and *pulmonary gas exchange*, respectively.

3-4.2   **Respiration Phases.** The complete process of respiration includes six important phases:

1.   Ventilation of the lungs with fresh air

2.   Exchange of gases between blood and air in lungs

3.   Transport of gases by blood

4.   Exchange of gases between blood and tissue fluids

5.   Exchange of gases between the tissue fluids and cells

6.   Use and production of gases by cells

If any one of the processes stops or is seriously hindered, the affected cells cannot function normally or survive for any length of time. Brain tissue cells, for example, stop working almost immediately and will either die or be permanently injured in a few minutes if their oxygen supply is completely cut off.

The respiratory system is a complex of organs and structures that performs the pulmonary ventilation of the body and the exchange of oxygen and carbon dioxide between the ambient air and the blood circulating through the lungs. It also warms the air passing into the body and assists in speech production by providing air to the larynx and the vocal chords. The respiratory tract is divided into upper and lower tracts.

3-4.3 **Upper and Lower Respiratory Tract.** The upper respiratory tract consists of the nose, nasal cavity, frontal sinuses, maxillary sinuses, larynx, and trachea. The upper respiratory tract carries air to and from the lungs and filters, moistens and warms air during each inhalation.

The lower respiratory tract consists of the left and right bronchi and the lungs, where the exchange of oxygen and carbon dioxide occurs during the respiratory cycle. The bronchi divide into smaller bronchioles in the lungs, the bronchioles divide into alveolar ducts, the ducts into alveolar sacs, and the sacs into alveoli. The alveolar sacs and the alveoli present about 850 square feet of space for the exchange of oxygen and carbon dioxide that occurs between the internal alveolar surface and the tiny capillaries surrounding the external alveolar wall.

3-4.4 **The Respiratory Apparatus.** The mechanics of taking fresh air into the lungs (inspiration or inhalation) and expelling used air from the lungs (expiration or exhalation) is diagrammed in Figure 3-3. By elevating the ribs and lowering the diaphragm, the volume of the lung is increased. Thus, according to Boyle's Law, a lower pressure is created within the lungs and fresh air rushes in to equalize this lowered pressure. When the ribs are lowered again and the diaphragm rises to its original position, a higher pressure is created within the lungs, expelling the used air.

3-4.4.1 **The Chest Cavity.** The chest cavity does not have space between the outer lung surfaces and the surrounding chest wall and diaphragm. Both surfaces are covered by membranes; the visceral pleura covers the lung and the parietal pleura lines the chest wall. These pleurae are separated from each other by a small amount of fluid that acts as a lubricant to allow the membranes to slide freely over themselves as the lungs expand and contract during respiration.

3-4.4.2 **The Lungs.** The lungs are a pair of light, spongy organs in the chest and are the main component of the respiratory system (see Figure 3-4). The highly elastic lungs are the main mechanism in the body for inspiring air from which oxygen is extracted for the arterial blood system and for exhaling carbon dioxide dispersed from the venous system. The lungs are composed of lobes that are smooth and shiny on their surface. The lungs contain millions of small expandable air sacs (alveoli) connected to air passages. These passages branch and rebranch like the twigs of a tree. Air entering the main airways of the lungs gains access to the
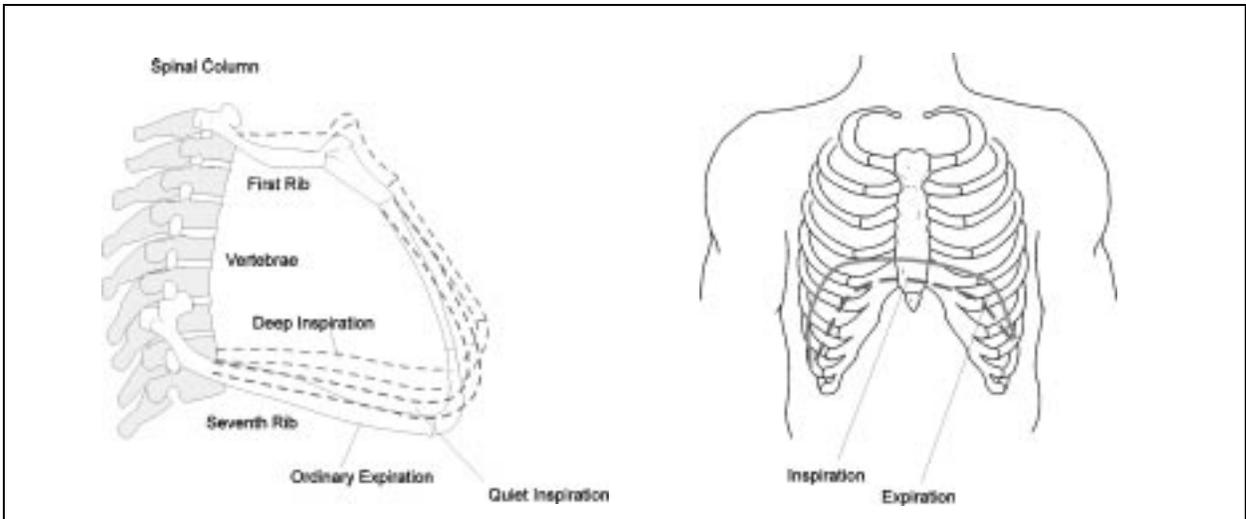
Figure 3-3. Inspiration Process. Inspiration involves both raising the rib cage (left panel) and lowering the diaphragm (right panel). Both movements enlarge the volume of the thoracic cavity and draw air into the lung.
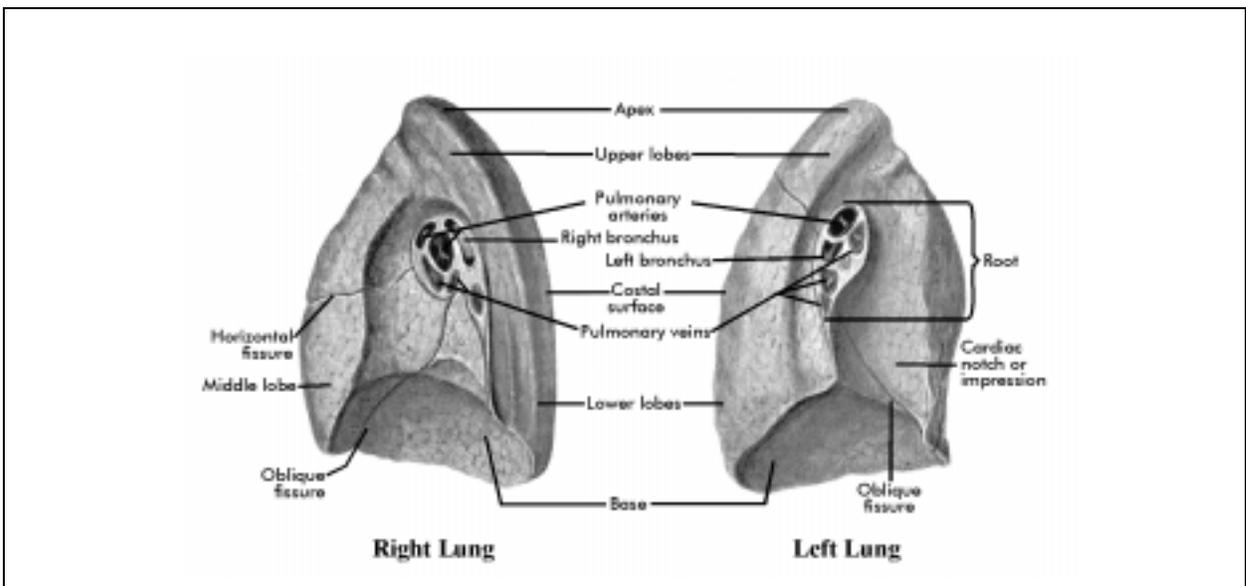

Figure 3-4. Lungs Viewed from Medial Aspect.

entire surface of these alveoli. Each alveolus is lined with a thin membrane and is surrounded by a network of very small vessels that make up the capillary bed of the lungs. Most of the lung membrane has air on one side of it and blood on the other; diffusion of gases takes place freely in either direction.

3-4.5 **Respiratory Tract Ventilation Definitions.** Ventilation of the respiratory system establishes the proper composition of gases in the alveoli for exchange with the blood. The following definitions help in understanding respiration (Figure 3-5).

3-4.5.1 **Respiratory Cycle.** The *respiratory cycle* is one complete breath consisting of an inspiration and exhalation, including any pause between the movements.
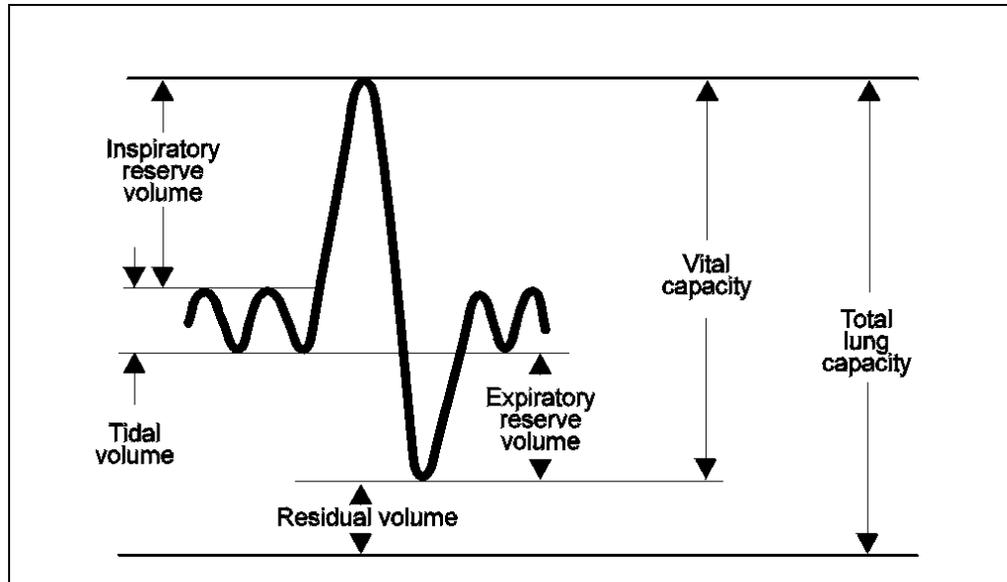
Figure 3-5. Lung Volumes. The heavy line is a tracing, derived from a subject breathing to and from a sealed recording bellows. Following several normal tidal breaths, the subject inhales maximally, then exhales maximally. The volume of air moved during this maximal effort is called the vital capacity. During exercise, the tidal volume increases, using part of the inspiratory and expiratory reserve volumes. The tidal volume, however, can never exceed the vital capacity. The residual volume is the amount of air remaining in the lung after the most forceful expiration. The sum of the vital capacity and the residual volume is the total lung capacity.

3-4.5.2 **Respiratory Rate.** The number of complete respiratory cycles that take place in 1 minute is the *respiratory rate*. An adult at rest normally has a respiratory rate of approximately 12 to 16 breaths per minute.

3-4.5.3 **Total Lung Capacity.** The *total lung capacity* (TLC) is the total volume of air that the lungs can hold when filled to capacity. TLC is normally between five and six liters.

3-4.5.4 **Vital Capacity.** *Vital capacity* is the volume of air that can be expelled from the lungs after a full inspiration. The average vital capacity is between four and five liters.

3-4.5.5 **Tidal Volume.** *Tidal volume* is the volume of air moved in or out of the lungs during a single normal respiratory cycle. The tidal volume generally averages about one-half liter for an adult at rest. Tidal volume increases considerably during physical exertion, and cannot exceed the vital capacity.

3-4.5.6 **Respiratory Minute Volume.** The *respiratory minute volume* (RMV) is the total amount of air moved in or out of the lungs in a minute. The respiratory minute volume is calculated by multiplying the tidal volume by the rate. RMV varies greatly with the body's activity. It is about 6 to 10 liters per minute at complete rest and may be over 100 liters per minute during severe work.

3-4.5.7   **Maximal Breathing Capacity and Maximum Ventilatory Volume.**   The *maximal breathing capacity* (MBC) and *maximum ventilatory volume* (MVV) are the greatest respiratory minute volumes that a person can produce during a short period of extremely forceful breathing. In a healthy young man, they may average as much as 180 liters per minute (the range is 140 to 240 liters per minute).

3-4.5.8   **Maximum Inspiratory Flow Rate and Maximum Expiratory Flow Rate.** The *maximum inspiratory flow rate* (MIFR) and *maximum expiratory flow rate* (MEFR) are the fastest rates at which the body can move gases in and out of the lungs. These rates are important in designing breathing equipment and computing gas use under various workloads. Flow rates are usually expressed in liters per second.

3-4.5.9   **Respiratory Quotient.** *Respiratory quotient* (RQ) is the ratio of the amount of carbon dioxide produced to the amount of oxygen consumed during cellular processes per unit time. This value ranges from 0.7 to 1.0 depending on diet and physical exertion and is usually assumed to be 0.9 for calculations. This ratio is significant when calculating the amount of carbon dioxide produced as oxygen is used at various workloads while using a closed-circuit breathing apparatus. The duration of the carbon dioxide absorbent canister can then be compared to the duration of the oxygen supply.

3-4.5.10   **Respiratory Dead Space.** *Respiratory dead space* refers to the part of the respiratory system that has no alveoli, and in which little or no exchange of gas between air and blood takes place. It normally amounts to less than 0.2 liter. Air occupying the dead space at the end of expiration is rebreathed in the following inspiration. Parts of a diver's breathing apparatus can add to the volume of the dead space and thus reduce the proportion of the tidal volume that serves the purpose of respiration. To compensate, the diver must increase his tidal volume. The problem can best be visualized by using a breathing tube as an example. If the tube contains one liter of air, a normal exhalation of about one liter will leave the tube filled with used air from the lungs. At inhalation, the used air will be drawn right back into the lungs. The tidal volume must be increased by more than a liter to draw in the needed fresh supply, because any fresh air is diluted by the air in the dead space. Thus, the air that is taken into the lungs (inspired air) is a mixture of fresh and dead space gases.

3-4.6   **Alveolar/Capillary Gas Exchange.** Within the alveolar air spaces, the composition of the air (alveolar air) is changed by the elimination of carbon dioxide from the blood, the absorption of oxygen by the blood, and the addition of water vapor. The air that is exhaled is a mixture of alveolar air and the inspired air that remained in the dead space.

The blood in the capillary bed of the lungs is exposed to the gas pressures of alveolar air through the thin membranes of the air sacs and the capillary walls. With this exposure taking place over a vast surface area, the gas pressure of the blood leaving the lungs is approximately equal to that present in alveolar air.

When arterial blood passes through the capillary network surrounding the cells in the body tissues it is exposed to and equalizes with the gas pressure of the tissues.

Some of the blood's oxygen is absorbed by the cells and carbon dioxide is picked up from these cells. When the blood returns to the pulmonary capillaries and is exposed to the alveolar air, the partial pressures of gases between the blood and the alveolar air is again equalized.

Carbon dioxide diffuses from the blood into the alveolar air, lowering its pressure, and oxygen is absorbed by the blood from the alveolar air, increasing its pressure. With each complete round of circulation, the blood is the medium through which this process of gas exchange occurs. Each cycle normally requires approximately 20 seconds.

3-4.7 **Breathing Control.** The amount of oxygen consumed and carbon dioxide produced increases markedly when a diver is working. The amount of blood pumped through the tissues and the lungs per minute increases in proportion to the rate at which these gases must be transported. As a result, more oxygen is taken up from the alveolar air and more carbon dioxide is delivered to the lungs for disposal. To maintain proper blood levels, the respiratory minute volume must also change in proportion to oxygen consumption and carbon dioxide output.

Changes in the partial pressure (concentration) of oxygen and carbon dioxide (ppO_2 and $ppCO_2$) in the arterial circulation activate central and peripheral chemoreceptors. These chemoreceptors are attached to important arteries. The most important are the carotid bodies in the neck and aortic bodies near the heart. The chemoreceptor in the carotid artery is activated by the $ppCO_2$ in the blood and signals the respiratory center in the brain stem to increase or decrease respiration. The chemoreceptor in the aorta causes the aortic body reflex. This is a normal chemical reflex initiated by decreased oxygen concentration and increased carbon dioxide concentration in the blood. These changes result in nerve impulses that increase respiratory activity. Low oxygen tension alone does not increase breathing markedly until dangerous levels are reached. The part played by chemoreceptors is evident in normal processes such as breathholding.

As a result of the regulatory process and the adjustments they cause, the blood leaving the lungs usually has about the same oxygen and carbon dioxide levels during work that it did at rest. The maximum pumping capacity of the heart (blood circulation) and respiratory system (ventilation) largely determines the amount of work a person can do.

3-4.8 **Oxygen Consumption.** A diver's oxygen consumption is an important factor when determining how long breathing gas will last, the ventilation rates required to maintain proper helmet oxygen level, and the length of time a canister will absorb carbon dioxide. Oxygen consumption is a measure of energy expenditure and is closely linked to the respiratory processes of ventilation and carbon dioxide production.

Oxygen consumption is measured in liters per minute (l/min) at Standard Temperature (0°C, 32°F) and Pressure (14.7 psia, 1 ata), Dry Gas (STPD). These rates of oxygen consumption are not depth dependent. This means that a fully charged MK

16 oxygen bottle containing 360 standard liters (3.96 scf) of usable gas will last 225 minutes at an oxygen consumption rate of 1.6 liters per minute at any depth, provided no gas leaks from the rig.

Minute ventilation, or respiratory minute volume (RMV), is measured at BTPS (body temperature 37°C/98.6°F, ambient barometric pressure, saturated with water vapor at body temperature) and varies depending on a person's activity level, as shown in Figure 3-6. Surface RMV can be approximated by multiplying the oxygen consumption rate by 25. Although this 25:1 ratio decreases with increasing gas density and high inhaled oxygen concentrations, it is a good rule-of-thumb approximation for computing how long the breathing gas will last.

Unlike oxygen consumption, the amount of gas exhaled by the lungs is depth dependent. At the surface, a diver swimming at 0.5 knot exhales 20 l/min of gas. A scuba cylinder containing 71.2 standard cubic feet (scf) of air (approximately 2,000 standard liters) lasts approximately 100 minutes. At 33 fsw, the diver still exhales 20 l/min at BTPS, but the gas is twice as dense; thus, the exhalation would be approximately 40 standard l/min and the cylinder would last only half as long, or 50 minutes. At three atmospheres, the same cylinder would last only one-third as long as at the surface.

Carbon dioxide production depends only on the level of exertion and can be assumed to be independent of depth. Carbon dioxide production and RQ are used to compute ventilation rates for chambers and free-flow diving helmets. These factors may also be used to determine whether the oxygen supply or the duration of the CO_2 absorbent will limit a diver's time in a closed or semi-closed system.

3-5 RESPIRATORY PROBLEMS IN DIVING

Physiological problems often occur when divers are exposed to the pressures of depth. However, some of the difficulties related to respiratory processes can occur at any time because of an inadequate supply of oxygen or inadequate removal of carbon dioxide from the tissue cells. Depth may modify these problems for the diver, but the basic difficulties remain the same. Fortunately, the diver has normal physiological reserves to adapt to environmental changes and is only marginally aware of small changes. The extra work of breathing reduces the diver's ability to do heavy work at depth, but moderate work can be done with adequate equipment at the maximum depths currently achieved in diving.

3-5.1 **Oxygen Deficiency (Hypoxia).** Oxygen deficiency, or *hypoxia*, is an abnormal deficiency of oxygen in the arterial blood that causes the tissue cells to be unable to receive sufficient oxygen to maintain normal function. Severe hypoxia will stop the normal function of any tissue cell in the body and will eventually kill it, but the cells of the brain tissue are by far the most susceptible to its effects.

The partial pressure of oxygen determines whether the amount of oxygen in a breathing medium is adequate. For example, air contains about 21 percent oxygen and thus provides an oxygen partial pressure of about 0.21 ata at the surface. This

Figure 3-6. Oxygen Consumption and RMV at Different Work Rates.
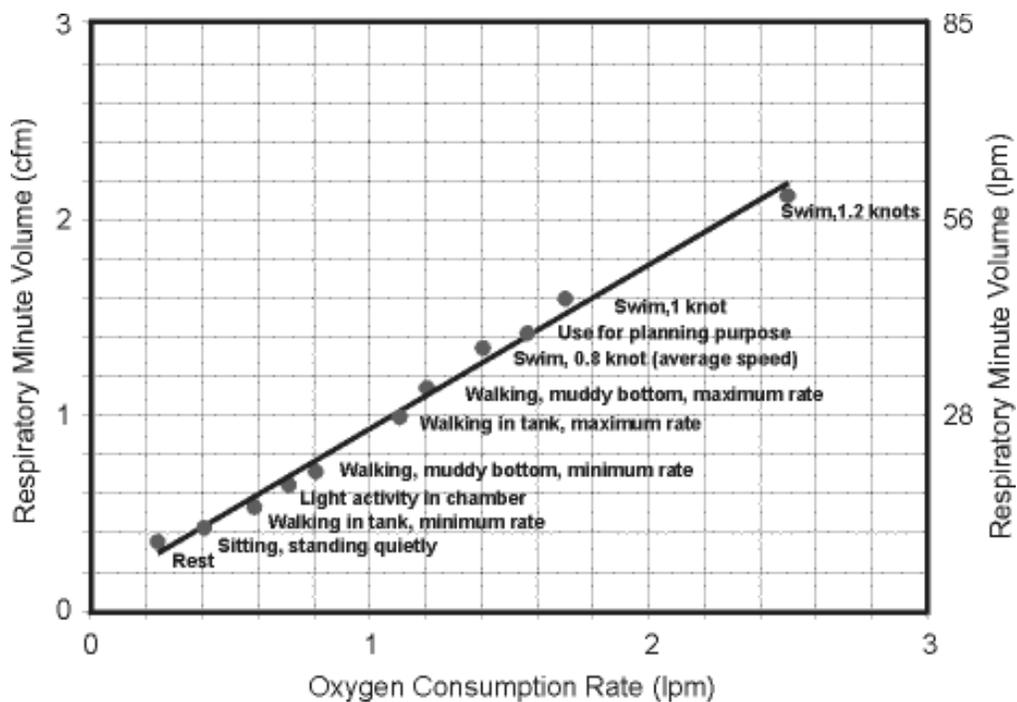
Work	VO$_2$ (lpm)	RMV (acfm)	RMV (lpm)	Work Level
Rest	0.24	0.35	10	—
Sitting, standing quietly	0.40	0.42	12	Light
Walking in tank, minimum rate	0.58	0.53	15	Light
Light activity in chamber	0.70	0.64	18	Light
Walking, muddy bottom, minimum rate	0.80	0.71	20	Moderate
Walking in tank, maximum rate	1.10	0.99	28	Moderate
Walking, muddy bottom, maximum rate	1.20	1.14	32	Moderate
Swim, 0.8 knot (average speed) (use for planning purposes, round up to 1.4)	1.40	1.34	38	Moderate
Swim, 1 knot	1.70	1.59	45	Heavy
Swim, 1.2 knot	2.50	2.12	60	Severe

is ample, but a drop to 0.14 ata causes the onset of hypoxic symptoms on the surface. If the ppO_2 goes as low as 0.11 ata at the surface, most individuals become hypoxic to the point of being nearly helpless. Consciousness is usually lost at about 0.10 ata and at much below this level, permanent brain damage and death will probably occur. In diving, a lower percentage will suffice as long as the total pressure is sufficient to maintain an adequate ppO_2. For example, 5 percent oxygen would render a ppO_2 of 0.20 ata for a diver at 100 fsw. On ascent, however, the diver would rapidly experience hypoxia if the oxygen percentage were not increased.

3-5.1.1 **Causes of Hypoxia.** The causes of hypoxia vary, but all interfere with the normal oxygen supply to the body. For divers, interference of oxygen delivery can be caused by:

- Equipment problems such as low partial pressure of oxygen in the breathing mix, inadequate gas flow, inadequate purging of breathing bags in a closed oxygen UBA like the LAR V, or blockage of the fresh gas injection orifice in a semiclosed-circuit UBA.

- Blockage of all or part of the pulmonary system air passages by vomitus, secretions, water, foreign objects, or pneumomediastinum.

- Pneumothorax or paralysis of the respiratory muscles from spinal cord injury.

- Decreased oxygen exchange at the alveoli/capillary membrane caused by accumulation of fluid in the tissues (edema), a mismatch of blood flow and alveolar ventilation, lung damage from near-drowning or smoke inhalation, or "chokes" or bronchospasm from lung irritation due to showers of bubbles in the circulation.

- Physiological problems such as anemia and inadequate blood flow that interfere with blood transportation of oxygen. Edema can interfere with gas exchange at the capillary/tissue areas, and carbon monoxide poisoning can interfere with oxygen utilization at the cellular level.

- Hyperventilation followed by breathholding, which can lead to severe hypoxia. Hyperventilation lowers the carbon dioxide level in the body below normal (a condition known as hypocapnia) and may prevent the control mechanism that stimulates breathing from responding until oxygen tension has fallen below the level necessary to maintain consciousness. Extended breathholding after hyperventilation is not a safe procedure. Refer to paragraph 3-7 for more information on hyperventilation and its hazards.

3-5.1.2 **Symptoms of Hypoxia.** Brain tissue is by far the most susceptible to the effects of hypoxia. Unconsciousness and death can occur from brain hypoxia before the effects on other tissues become very prominent.

There is no reliable warning of the onset of hypoxia. It can occur unexpectedly, making it a particularly serious hazard. A diver who loses his air supply is in

danger of hypoxia, but he immediately knows he is in danger and usually has time to do something about it. He is much more fortunate than a diver who gradually uses up the oxygen in a closed-circuit rebreathing rig and has no warning of impending unconsciousness.

When hypoxia develops, pulse rate and blood pressure increase as the body tries to offset the hypoxia by circulating more blood. A small increase in breathing may also occur. A general blueness (cyanosis) of the lips, nail beds and skin may occur with hypoxia. This may not be noticed by the diver and often is not a reliable indicator of hypoxia, even for the trained observer at the surface. The same signs could be caused by prolonged exposure to cold water.

If hypoxia develops gradually, symptoms of interference with brain function will appear. None of these symptoms, however, are sufficient warning and very few people are able to recognize the mental effects of hypoxia in time to take corrective action.

Symptoms of hypoxia include:

- Lack of concentration
- Lack of muscle control
- Inability to perform delicate or skill-requiring tasks
- Drowsiness
- Weakness
- Agitation
- Euphoria
- Loss of consciousness

3-5.1.3   **Treating Hypoxia.** A diver suffering from severe hypoxia must be rescued promptly. Hypoxia's interference with brain functions produces not only unconsciousness but also failure of the breathing control centers. If a victim of hypoxia is given gas with adequate oxygen content before his breathing stops, he usually regains consciousness shortly and recovers completely. For scuba divers, this usually involves bringing the diver to the surface. For surface-supplied mixed-gas divers, it involves shifting the gas supply to alternative banks and ventilating the helmet or chamber with the new gas. Details of treatment are covered in volume 3.

3-5.1.4   **Preventing Hypoxia.** Because of its insidious nature and potentially fatal outcome, preventing hypoxia is essential. In open-circuit scuba and helmets, hypoxia is unlikely unless the supply gas has too low an oxygen content. On mixed-gas operations, strict attention must be paid to gas analysis, cylinder lineups and predive checkout procedures. In closed- and semiclosed-circuit Underwater Breathing Apparatus (UBA), a malfunction can cause hypoxia even though the proper gases are being used. Electronically controlled, fully closed-circuit UBA like the MK 16 have oxygen sensors to read out oxygen partial pressure, but divers must be constantly alert to the possibility of hypoxia from UBA malfunction. **Oxygen sensors should be monitored closely throughout the dive in closed-circuit mixed gas MK 16 UBA. MK 25 UBA breathing bags should**

be purged in accordance with Operating Procedures (OPs). Recently surfaced mixed-gas chambers should not be entered until after they are thoroughly ventilated with air.

3-5.2   Carbon Dioxide Toxicity (Hypercapnia). Carbon dioxide toxicity, or *hypercapnia,* is an abnormally high level of carbon dioxide in the body tissues.

3-5.2.1   **Causes of Hypercapnia.** In diving operations, hypercapnia is generally the result of a buildup of carbon dioxide in the breathing supply or in the body caused by:

- Inadequate ventilation of surface-supplied helmets

- Excess carbon dioxide in helmet supply gas (failure of CO_2 absorbent canister) in mixed-gas diving

- Failure of carbon dioxide absorbent canisters in closed- or semiclosed-circuit UBA

- Inadequate lung ventilation in relation to exercise level (caused by controlled breathing, excessive apparatus breathing resistance, increased oxygen partial pressure, or increased gas density)

- Any cause of increased dead space, such as shallow and rapid breathing through a snorkel

3-5.2.2   **Symptoms of Hypercapnia.** Underwater breathing equipment is designed to keep the carbon dioxide below 1.5 percent during heavy work. The most common cause of hypercapnia is failure to ventilate helmets adequately. This can occur through improper breathing techniques or excessive breathing resistance; a diver can poison himself by inadequately ventilating his lungs. This happens primarily when a scuba diver tries to conserve his breathing supply by reducing his breathing rate below a safe level (skip-breathing). Inadequate lung ventilation is more common in diving than in surface activities for two reasons. First, some divers have a lower drive to increase lung ventilation in the face of increased blood carbon dioxide levels. Second, the usually high ppO_2 encountered in diving takes away some of the uncomfortable shortness of breath that accompanies inadequate lung ventilation.

Hypercapnia affects the brain differently than hypoxia does. However, it can result in similar symptoms such as confusion, inability to concentrate, drowsiness, loss of consciousness, and convulsions. Such effects become more severe as the degree of excess increases. A diver breathing a gas with as much as 10 percent carbon dioxide generally loses consciousness after a few minutes. Breathing 15 percent carbon dioxide for any length of time causes muscular spasms and rigidity.

A diver who loses consciousness because of excess carbon dioxide in his breathing medium and does not aspirate water generally revives rapidly when given fresh air. He usually feels normal within 15 minutes and the aftereffects rarely include symptoms more serious than headache, nausea, and dizziness.

Permanent brain damage and death are much less likely than in the case of hypoxia.

3-5.2.2.1 ***Effects of Increasing Carbon Dioxide Levels.*** The increasing level of carbon dioxide in the blood stimulates the respiratory center to increase the breathing rate and volume, and the heartbeat rate is often increased. Ordinarily, increased breathing is definite and uncomfortable enough to warn a diver before the $ppCO_2$ becomes very dangerous. However, variables such as work rate, depth, and the composition of the breathing mixture may produce changes in breathing and blood mixture that could mask any changes caused by excess carbon dioxide.

This is especially true in closed-circuit UBA (especially 100-percent oxygen rebreathers) when failure or expenditure of the carbon dioxide absorbent material allows a carbon dioxide buildup while the amount of oxygen increases. In cases where the ppO_2 is above 0.5 ata, the shortness of breath usually associated with excess carbon dioxide may not be excessive and may go unnoticed by the diver, especially if he is breathing hard because of exertion. In these cases the diver may become confused and even slightly euphoric before losing consciousness. For this reason, a diver must be particularly alert for any marked change in his breathing comfort or cycle (such as shortness of breath or hyperventilation) as a warning of hypercapnia.

3-5.2.2.2 ***Effects of Excess Carbon Dioxide.*** Excess carbon dioxide also dilates the arteries of the brain. This may partially explain the headaches often associated with carbon dioxide intoxication, though these headaches are more likely to occur following the exposure than during it. The increase in blood flow through the brain, which results from dilation of the arteries, is thought to explain why carbon dioxide excess speeds the onset of oxygen toxicity or possibly convulsions. Excess carbon dioxide during a dive is also believed to increase the likelihood of decompression sickness, but the reasons are less clear. Headache, cyanosis, unusual sweating, fatigue, and a general feeling of discomfort may warn a diver if they occur and are recognized, but they are not very reliable as warnings.

Hypothermia also can mask the buildup of carbon dioxide because the respiration rate increases initially on exposure to cold water. Additionally, nitrogen narcosis can mask the condition because a diver under the effects of narcosis would not notice any difference in his breathing rate. During surface-supplied air dives deeper than 100 fsw (30.5 meters), the Diving Supervisor must ensure the divers maintain sufficient ventilation rates.

3-5.2.3 **Treating Hypercapnia.** Hypercapnia is treated by relieving the excess partial pressure of carbon dioxide. This is accomplished in surface-supplied diving by ventilating the helmet with fresh air in an air diving apparatus, bypassing the carbon dioxide absorbent in a mixed-gas diving apparatus, or ascending. Any method used to decrease the partial pressure removes the problems encountered with excess carbon dioxide.

3-5.3 **Asphyxia.** *Asphyxia* indicates the existence of both hypoxia and carbon dioxide excess in the body. Asphyxia occurs when breathing stops. Breathing stoppage can

be due to injury to the windpipe (trachea), the lodging of an inhaled object, the tongue falling back in the throat during unconsciousness, or the inhalation of water, saliva, or vomitus.

In many situations, hypoxia and carbon dioxide excess occur separately. True asphyxia occurs when hypoxia is severe or prolonged enough to stop a diver's breathing and carbon dioxide toxicity develops rapidly. At this point the diver can no longer breathe.

3-5.4 **Breathing Resistance and Dyspnea.** The ability to perform useful work underwater depends on the diver's ability to move enough gas in and out of his lungs to provide sufficient oxygen to the muscles and to eliminate metabolically produced carbon dioxide. Increased gas density and breathing apparatus resistance are the two main factors that impede this ability. Even in a dry hyperbaric chamber without a breathing apparatus, the increased gas density may cause divers to experience shortness of breath (*dyspnea*). Dyspnea usually becomes apparent at very heavy workloads at depths below 120 fsw when a diver is breathing air. If a diver is breathing helium-oxygen, dyspnea usually becomes a problem at heavy workloads in the 850-1,000 fsw range. At great depths (1,600-1,800 fsw), dyspnea may occur even at rest.

3-5.4.1 **Causes of Breathing Resistance.** Flow resistance and static lung load are the two main causes of the breathing limitations imposed by the underwater breathing apparatus. Flow resistance is due to a flow of dense gas through tubes, hoses, and orifices in the diving equipment. As gas density increases, a larger driving pressure must be applied to keep gas flowing at the same rate. The diver has to exert higher negative pressures to inhale and higher positive pressures to exhale. As ventilation increases with increasing levels of exercise, the necessary driving pressures increase. Because the respiratory muscles can only exert so much effort to inhale and exhale, a point is reached when further increases can not occur. At this point, metabolically produced carbon dioxide is not adequately eliminated and increases in the blood, causing symptoms of hypercapnia.

Static lung load is the result of breathing gas being supplied at a different pressure than the hydrostatic pressure surrounding the lungs. For example, when swimming horizontally with a single-hose regulator, the regulator diaphragm is lower than the mouth and the regulator supplies gas at a slight positive pressure once the demand valve has opened. If the diver flips onto his back, the regulator diaphragm is shallower than his mouth and the regulator supplies gas at a slightly negative pressure. Inhalation is harder but exhalation is easier because the exhaust ports are above the mouth and at a slightly lower pressure.

Static lung loading is more apparent in semiclosed- and closed-circuit underwater breathing apparatus such as the MK 25 and MK 16. When swimming horizontally, the diaphragm on the diver's back is shallower than the lungs and the diver feels a negative pressure at the mouth. Exhalation is easier than inhalation. If the diver flips onto his back, the diaphragm is below the lungs and the diver feels a positive pressure at the mouth. Inhalation becomes easier than exhalation. At high work

rates, excessively high or low static lung loads may cause dyspnea without any increase in blood carbon dioxide level.

3-5.4.2 **Preventing Dyspnea.** The U.S. Navy makes every effort to ensure that UBA meet adequate breathing standards to minimize flow resistance and static lung loading problems. However, all UBA have their limitations and divers must have sufficient experience to recognize those limitations. If the UBA does not impede ventilation, the diver's own pulmonary system may limit his ability to ventilate. Whether due to limitations of the equipment or limitations imposed by the diver's own respiratory system, the end result may be symptoms of hypercapnia or dyspnea without increased carbon dioxide blood levels. This is commonly referred to as "overbreathing the rig."

Most divers decrease their level of exertion when they begin to experience dyspnea, but in some cases, depending on the depth and type of UBA, the dyspnea may continue to increase for a period of time after stopping exercise. When this occurs, the inexperienced diver may panic and begin to hyperventilate (breathe faster than is necessary for the exchange of respiratory gases), which increases the dyspnea. The situation rapidly develops into one of severe dyspnea and uncontrollable hyperventilation. In this situation, if even a small amount of water is inhaled, it can cause a spasm of the muscles in the larynx (voice box) called a laryngospasm, followed by asphyxia and possible drowning. The proper reaction to the dyspnea is to stop exercising, ventilate the UBA if possible, take even, controlled breaths until the dyspnea subsides, evaluate the situation and then proceed carefully. Generally, soreness of the respiratory muscles is the only prominent aftereffect of a dive in which breathing resistance is high.

3-5.5 **Carbon Monoxide Poisoning.** Carbon monoxide in a diver's air supply is dangerous. Carbon monoxide displaces oxygen from hemoglobin and interferes with cellular metabolism, rendering the cells hypoxic. Carbon monoxide is not found in any significant quantity in fresh air; carbon monoxide pollution of a breathing supply is usually caused by the exhaust of an internal combustion engine being too close to a compressor intake. Concentrations as low as 0.002 ata can prove fatal. Carbon monoxide poisoning is particularly treacherous because conspicuous symptoms may be delayed until the diver begins to ascend.

While at depth, the greater partial pressure of oxygen in the breathing supply forces more oxygen into solution in the blood plasma. Some of this additional oxygen reaches the cells and helps to offset the hypoxia. In addition, the increased partial pressure of oxygen forcibly displaces some carbon monoxide from the hemoglobin. During ascent, however, as the partial pressure of oxygen diminishes, the full effect of carbon monoxide poisoning is felt.

3-5.5.1 **Symptoms of Carbon Monoxide Poisoning.** The symptoms of carbon monoxide poisoning are almost identical to those of other types of hypoxia. The greatest danger is that unconsciousness can occur without reliable warning signs. When carbon monoxide concentration is high enough to cause rapid onset of poisoning, the victim may not be aware of weakness, dizziness, or confusion before he

becomes unconscious. When toxicity develops gradually, tightness across the forehead, headache and pounding at the temples, or nausea and vomiting may be warning symptoms.

3-5.5.2 **Treating Carbon Monoxide Poisoning.** The immediate treatment of carbon monoxide poisoning consists of getting the diver to fresh air and seeking medical attention. Oxygen, if available, should be administered immediately and while transporting the patient to a hyperbaric or medical treatment facility. Hyperbaric oxygen therapy is the definitive treatment of choice and transportation for recompression should not be delayed except to stabilize the serious patient prior to transport. The air supply of a diver suspected of suffering carbon monoxide poisoning must be secured to prevent anyone else from breathing it and the air must be analyzed.

3-5.5.3 **Preventing Carbon Monoxide Poisoning.** Carbon monoxide poisoning can be prevented by locating compressor intakes away from engine exhausts and maintaining air compressors in the best possible mechanical condition.

3-6 BREATHHOLDING AND UNCONSCIOUSNESS.

Most people can hold their breath approximately 1 minute, but usually not much longer without training or special preparation. At some time during a breathholding attempt, the desire to breathe becomes uncontrollable. This demand is signaled by the respiratory center responding to the increasing levels of carbon dioxide and acids in the arterial blood and chemoreceptors responding to the corresponding rise in arterial carbon dioxide.

3-6.1 **Breathhold Diving Restrictions.** Breathhold diving shall be confined to tactical and work situations that cannot be effectively accomplished by the use of underwater breathing apparatus and applicable diver training situations such as scuba pool phase and shallow water obstacle/ordnance clearance. Breathhold diving includes the practice of taking two or three deep breaths prior to the dive. The diver shall terminate the dive and surface at the first sign of the urge to breath. Hyperventilation (excessive rate and depth of breathing prior to a dive, as differentiated from two or three deep breaths prior to a dive) shall not be practiced because of the high possibility of causing unconsciousness under water.

3-6.2 **Hazards of Breathhold Diving.** One of the greatest hazards of breathhold diving is the possible loss of consciousness during ascent. Air in the lungs during descent is compressed, raising the oxygen partial pressure. The increased ppO_2 readily satisfies the body's oxygen demand during descent and while on the bottom, even though a portion is being consumed by the body. During ascent, the partial pressure of the remaining oxygen is reduced rapidly as the hydrostatic pressure on the body lessens. If the ppO_2 falls below 11 percent (83.6 mmHg), unconsciousness may result with its attendant danger. This danger is further heightened when hyperventilation has eliminated normal body warning signs of carbon dioxide accumulation.

3-7 HYPERVENTILATION

Hyperventilation is the term applied to breathing more than is necessary to keep the body's carbon dioxide tensions at proper level. Hyperventilation (whether voluntary or involuntary) has little effect on the body's oxygen levels, but abnormally lowers the partial pressure of carbon dioxide in the blood and delays the normal urge to breathe. If the carbon dioxide stores are ventilated below the stimulus level, there will be little urge to breathe until late in the breathhold. The oxygen partial pressure falls progressively as oxygen is consumed continuously. Exertion causes oxygen to be consumed faster, and decreases sensitivity of the carbon dioxide breakpoint mechanism. This permits the oxygen level to go lower than it would otherwise. When the diver ascends, the drop in oxygen partial pressure in the lungs may be sufficient to stop further uptake of oxygen completely. At the same time, the partial pressure of carbon dioxide in the lungs also drops, giving the diver the false impression that he need not breathe. Low levels of oxygen do not cause a powerful demand to resume breathing; thus, the level of oxygen in the blood may reach the point at which the diver loses consciousness before he feels a demand to breathe.

WARNING **Hyperventilation is dangerous and can lead to unconsciousness and death.**

3-7.1 **Unintentional Hyperventilation.** Unintentional hyperventilation can be triggered by fear experienced during stressful situations. It can be partly initiated by the slight "smothering sensation" that accompanies an increase in dead space, abnormal static loading, and increased breathing resistance. Cold water exposure can add to the sensation of needing to breathe faster and deeper. Divers using scuba equipment for the first few times are likely to hyperventilate to some extent because of anxiety.

3-7.2 **Voluntary Hyperventilation.** Voluntary hyperventilation (taking a number of deep breaths in a short period of time) can produce symptoms of abnormally low carbon dioxide tension (hypocapnia). Under these circumstances, one may develop a lightheadedness and tingling sensations. Hyperventilating over a long period, produces additional symptoms such as weakness, headaches, numbness, faintness, and blurring of vision. The anxiety caused by the sensation of suffocation that often initiates hyperventilation and continues in spite of adequate ventilation, may lead to a further increase in breathing and a vicious cycle develops. Severe hypocapnia with muscular spasms, loss of consciousness and shock may be the end result. The diver must pay attention to his breathing rate and, in the event of fear-induced hyperventilation, take steps to remain calm and control his breathing.

3-8 EFFECTS OF BAROTRAUMA AND PRESSURE ON THE HUMAN BODY

The tissues of the body can withstand tremendous pressure. Divers have made open-sea dives in excess of 1,000 fsw (445 psi) and, in experimental situations, have been exposed to a depth of 2,250 fsw (1001.3 psi). Despite these pressures, it is somewhat ironic that divers make the greatest number of medical complaints

during the shallowest part of a dive. The cause is *barotrauma*, which is the damage done to tissues when there is a change in ambient pressure. Barotrauma on descent is called *squeeze*. Barotrauma on ascent is called *reverse squeeze*.

3-8.1   **Conditions Leading to Barotrauma.** Barotrauma does not normally occur in divers who have normal anatomy and physiology, and who are using properly functioning equipment and correct diving procedures. Barotrauma can occur in body areas subject to all five of the following conditions:

■ There must be a gas-filled space. Any gas-filled space within the body (such as a sinus cavity) or next to the body (such as a face mask) can damage the body tissues when the gas volume changes because of increased pressure.

■ The space must have rigid walls. When the walls are elastic like a balloon, there is no damage done by gas compression or expansion until the volume change surpasses the elasticity of the walls or vessels.

■ The space must be enclosed. If any substance (with the exception of blood in the vessels lining the space) were allowed to enter or leave the space as the gas volume changes, no damage would occur.

■ The space must have vascular penetration (arteries and veins) and a membrane lining the space. This allows the blood to be forced into the space and exceed the elasticity of the vessels to compensate for the change in pressure.

■ There must be a change in ambient pressure.

3-8.2   **General Symptoms of Barotrauma.** The predominant symptom of barotrauma is pain. Other symptoms such as vertigo, numbness, or facial paralysis may be produced depending on the specific anatomy. Pulmonary Overinflation Syndrome is a potentially serious form of barotrauma and is discussed in detail later in this chapter. In all diving situations, arterial gas embolism and decompression sickness must be ruled out before the diagnosis of squeeze can be accepted.

3-8.3   **Middle Ear Squeeze.** Middle ear squeeze is the most common type of barotrauma. The anatomy of the ear is illustrated in Figure 3-7. The eardrum completely seals off the outer ear canal from the middle ear space. As a diver descends, water pressure increases on the external surface of the drum. To counterbalance this pressure, the air pressure must reach the inner surface of the eardrum. This is accomplished by the passage of air through the narrow eustachian tube that leads from the nasal passages to the middle ear space. When the eustachian tube is blocked by mucous, the middle ear meets four of the requirements for barotrauma to occur (gas filled space, rigid walls, enclosed space, penetrating blood vessels).

As the diver continues his descent, the fifth requirement (change in ambient pressure) is attained. As the pressure increases, the eardrum bows inward and initially equalizes the pressure by compressing the middle ear gas. There is a limit to this stretching capability and soon the middle ear pressure becomes lower than the external water pressure, creating a relative vacuum in the middle ear space. This
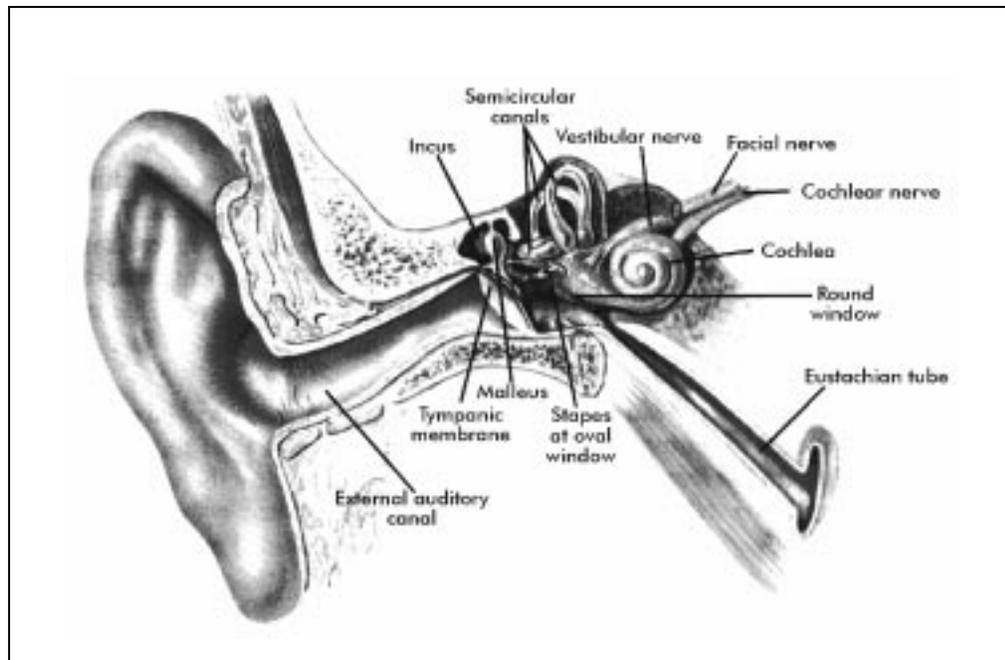
Figure 3-7. Gross Anatomy of the Ear in Frontal Section.

negative pressure causes the blood vessels of the eardrum and lining of the middle ear to first expand, then leak and finally burst. If descent continues, either the eardrum ruptures, allowing air or water to enter the middle ear and equalize the pressure, or blood vessels rupture and cause sufficient bleeding into the middle ear to equalize the pressure. The latter usually happens.

The hallmark of middle ear squeeze is sharp pain caused by stretching of the eardrum. The pain produced before rupture of the eardrum often becomes intense enough to prevent further descent. Simply stopping the descent and ascending a few feet usually brings about immediate relief.

If descent continues in spite of the pain, the eardrum may rupture. Unless the diver is in hard hat diving dress, the middle ear cavity may be exposed to water when the ear drum ruptures. This exposes the diver to a possible middle ear infection and, in any case, prevents the diver from diving until the damage is healed. At the time of the rupture, the diver may experience the sudden onset of a brief but violent episode of vertigo (a sensation of spinning). This can completely disorient the diver and cause nausea and vomiting. This vertigo is caused by violent distur- bance of the malleus, incus, and stapes, or by cold water stimulating the balance mechanism of the inner ear. The latter situation is referred to as caloric vertigo and may occur from simply having cold or warm water enter one ear and not the other. The eardrum does not have to rupture for caloric vertigo to occur. It can occur as the result of having water enter one ear canal when swimming or diving in cold water. Fortunately, these symptoms quickly pass when the water reaching the middle ear is warmed by the body.

3-8.3.1   **Preventing Middle Ear Squeeze.** Diving with a partially blocked eustachian tube increases the likelihood of middle ear squeeze. Divers who cannot clear their ears on the surface should not dive. Divers who have trouble clearing their ears shall be examined by medical personnel before diving.

The possibility of barotrauma can be virtually eliminated if certain precautions are taken. While descending, stay ahead of the pressure. To avoid collapse of the eustachian tube and to clear the ears, frequent adjustments of middle ear pressure must be made by adding gas through the eustachian tubes from the back of the nose. If too large a pressure difference develops between the middle ear pressure and the external pressure, the eustachian tube collapses as it becomes swollen and blocked. For some divers, the eustachian tube is open all the time so no conscious effort is necessary to clear their ears. For the majority, however, the eustachian tube is normally closed and some action must be taken to clear the ears. Many divers can clear by yawning, swallowing, or moving the jaw around.

Some divers must gently force gas up the eustachian tube by closing their mouth, pinching their nose and exhaling. This is called a Valsalva maneuver. If too large a relative vacuum exists in the middle ear, the eustachian tube collapses and no amount of forceful clearing will open it. If a squeeze is noticed during descent, the diver shall stop, ascend a few feet and gently perform a Valsalva maneuver. If clearing cannot be accomplished as described above, abort the dive.

WARNING   **Never do a forceful Valsalva maneuver during descent or ascent. During descent, this action can result in alternobaric vertigo or a round or oval window rupture. During ascent, this action can result in a pulmonary overinflation syndrome.**

3-8.3.2   **Treating Middle Ear Squeeze.** Upon surfacing after a middle ear squeeze, the diver may complain of pain, fullness in the ear, hearing loss or even mild vertigo. Occasionally, blood may be in the nostrils as the result of blood being forced through the eustachian tube by expanding air in the middle ear. The diver shall report this to the diving supervisor and seek medical attention. Treatment consists of taking decongestants and cessation of diving until the damage is healed.

3-8.4   **Sinus Squeeze.** Sinuses are located within hollow spaces of the skull bones and are lined with a mucous membrane continuous with that of the nasal cavity (Figure 3-8). The sinuses are small air pockets connected to the nasal cavity by narrow passages. If pressure is applied to the body and the passages to any of these sinuses are blocked by mucous or tissue growths, pain will soon be experienced in the affected area. The situation is very much like that described for the middle ear.

3-8.4.1   **Causes of Sinus Squeeze.** When the air pressure in these sinuses is less than the pressure applied to the tissues surrounding these incompressible spaces, the same relative effect is produced as if a vacuum were created within the sinuses: the lining membranes swell and, if severe enough, hemorrhage into the sinus spaces. This process represents nature's effort to balance the relative negative air pressure by filling the space with swollen tissue, fluid, and blood. The sinus is actually squeezed. The pain produced may be intense enough to halt the diver's descent.
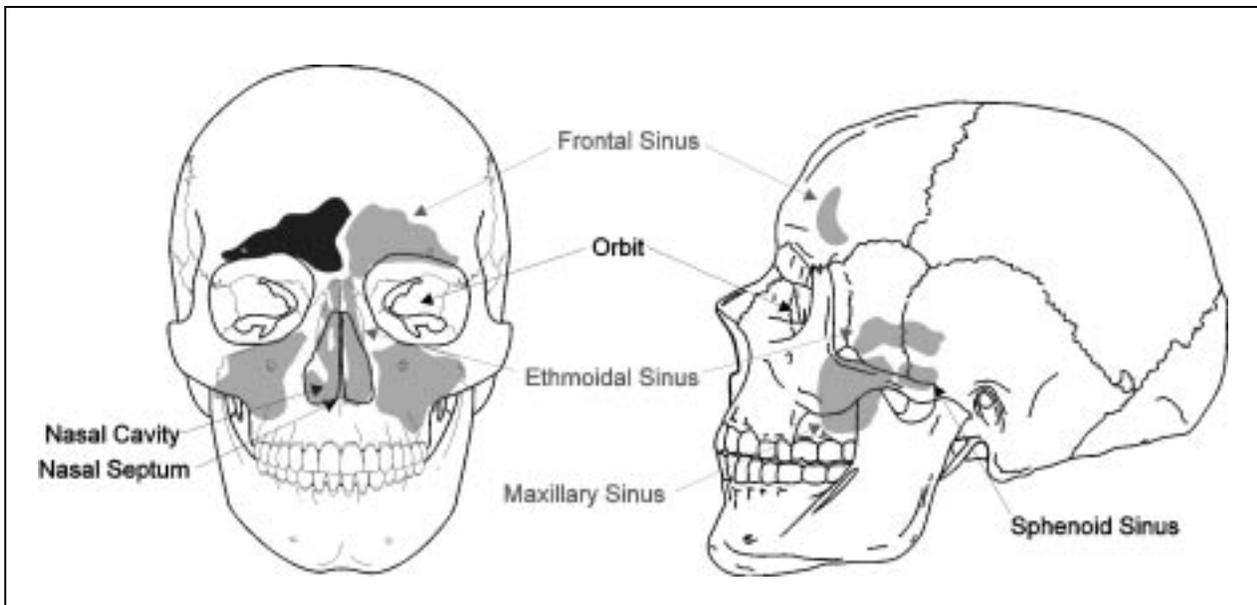
Figure 3-8. Location of the Sinuses in the Human Skull.

Unless damage has already occurred, a return to normal pressure will bring about immediate relief. If such difficulty has been encountered during a dive, the diver may often notice a small amount of bloody nasal discharge on reaching the surface.

3-8.4.2 **Preventing Sinus Squeeze.** Divers should not dive if any signs of nasal congestion or a head cold are evident. The effects of squeeze can be limited during a dive by halting the descent and ascending a few feet to restore the pressure balance. If the space cannot be equalized by swallowing or blowing against a pinched-off nose, the dive must be aborted.

3-8.5 **Tooth Squeeze (Barodontalgia).** Tooth squeeze occurs when a small pocket of gas, generated by decay, is lodged under a poorly fitted or cracked filling. If this pocket of gas is completely isolated, the pulp of the tooth or the tissues in the tooth socket can be sucked into the space causing pain. If additional gas enters the tooth during descent and does not vent during ascent, it can cause the tooth to crack or the filling to be dislodged. Prior to any dental work, personnel shall identify themselves as divers to the dentist.

3-8.6 **External Ear Squeeze.** A diver who wears ear plugs, has an infected external ear (external otitis), has a wax-impacted ear canal, or wears a tight-fitting wet suit hood, can develop an external ear squeeze. The squeeze occurs when gas trapped in the external ear canal remains at atmospheric pressure while the external water pressure increases during descent. In this case, the eardrum bows outward (opposite of middle ear squeeze) in an attempt to equalize the pressure difference and may rupture. The skin of the canal swells and hemorrhages, causing considerable pain.

Ear plugs must never be worn while diving. In addition to creating the squeeze, they may be forced deep into the ear canal. When a hooded suit must be worn, air (or water in some types) must be allowed to enter the hood to equalize pressure in the ear canal.

3-8.7   **Thoracic (Lung) Squeeze.** When making a breathhold dive, it is possible to reach a depth at which the air held in the lungs is compressed to a volume somewhat smaller than the normal residual volume of the lungs. At this volume, the chest wall becomes stiff and incompressible. If the diver descends further, the additional pressure is unable to compress the chest walls, force additional blood into the blood vessels in the chest, or elevate the diaphragm further. The pressure in the lung becomes negative with respect to the external water pressure. Injury takes the form of squeeze. Blood and tissue fluids are forced into the lung alveoli and air passages where the air is under less pressure than the blood in the surrounding vessels. This amounts to an attempt to relieve the negative pressure within the lungs by partially filling the air space with swollen tissue, fluid, and blood. Considerable lung damage results and, if severe enough, may prove fatal. If the diver descends still further, death will occur as a result of the collapse of the chest. Breathhold diving shall be limited to controlled, training situations or special operational situations involving well-trained personnel at shallow depths.

A surface-supplied diver who suffers a loss of gas pressure or hose rupture with failure of the nonreturn valve may suffer a lung squeeze, if his depth is great enough, as the surrounding water pressure compresses his chest.

3-8.8   **Face or Body Squeeze.** Scuba face masks, goggles, and certain types of exposure suits may cause squeeze under some conditions. The pressure in a face mask can usually be equalized by exhaling through the nose, but this is not possible with goggles. Goggles shall only be used for surface swimming. The eye and the eye socket tissues are the most seriously affected tissues in an instance of face mask or goggle squeeze. When using exposure suits, air may be trapped in a fold in the garment and may lead to some discomfort and possibly a minor case of hemorrhage into the skin from pinching.

3-8.9   **Middle Ear Overpressure (Reverse Middle Ear Squeeze).** Expanding gas in the middle ear space during ascent ordinarily vents out through the eustachian tube. If the tube becomes blocked, pressure in the middle ear relative to the external water pressure increases. To relieve this pressure, the eardrum bows outward causing pain. If the overpressure is significant, the eardrum may rupture and the diver may experience the same symptoms that occur with an eardrum rupture during descent (squeeze).

The increased pressure in the middle ear may also affect nearby structures and produce symptoms of vertigo and inner ear damage. It is extremely important to rule out arterial gas embolism or decompression sickness when these unusual symptoms of reverse middle ear squeeze occur during ascent or upon surfacing.

A diver who has a cold or is unable to equalize the ears is more likely to develop reverse middle ear squeeze. There is no uniformly effective way to clear the ears

on ascent. Do not perform a Valsalva maneuver on ascent, as this will increase the pressure in the middle ear, which is the direct opposite of what is required. The Valsalva maneuver can also lead to the possibility of an arterial gas embolism. If pain in the ear develops on ascent, the diver should halt the ascent, descend a few feet to relieve the symptoms and then continue his ascent at a slower rate. Several such attempts may be necessary as the diver gradually works his way to the surface.

3-8.10   **Sinus Overpressure (Reverse Sinus Squeeze).** Overpressure is caused when gas is trapped within the sinus cavity. A fold in the sinus-lining membrane, a cyst, or an outgrowth of the sinus membrane (polyp) may act as a check valve and prevent gas from leaving the sinus during ascent. Sharp pain in the area of the affected sinus results from the increased pressure. The pain is usually sufficient to stop the diver from ascending. Pain is immediately relieved by descending a few feet. From that point, the diver should slowly ascend until he gradually reaches the surface.

3-8.11   **Overexpansion of the Stomach and Intestine.** While a diver is under pressure, gas may form within his intestines or gas may be swallowed and trapped in the stomach. On ascent, this trapped gas expands and occasionally causes enough discomfort to require the diver to stop and expel the gas. Continuing ascent in spite of marked discomfort may result in actual harm.

3-8.12   **Inner Ear Dysfunction.** The inner ear contains no gas and is not subject to barotrauma. However, the inner ear is located next to the middle ear cavity and is affected by the same conditions that produce middle ear barotrauma. As the gas in the middle ear is compressed or expands without the relief normally provided by the eustachian tube, the fluid and membranes of the delicate inner ear will be functionally disturbed. The membranes may tear as the pressure gradient increases.

The inner ear contains two important organs, the cochlea and the vestibular apparatus. The cochlea is the hearing sense organ; damage to the cochlea can result in symptoms of hearing loss and ringing in the ear (tinnitus).

3-8.12.1   **Vertigo.** The vestibular apparatus senses balance and motion; damage to the vestibular apparatus may cause vertigo, which is the false sensation of a spinning type of motion. The diver will feel that he or the surrounding area is spinning while in fact there is no motion. One can usually tell this distinct sensation from the more vague complaints of dizziness or lightheadedness caused by other conditions. Vertigo is usually specific to the inner ear or that part of the brain that analyzes inner ear input. Vertigo has associated symptoms that may or may not be noticed. These include nausea, vomiting, loss of balance, incoordination, and a rapid jerking movement of the eyes (nystagmus). Vertigo may also be caused by arterial gas embolism or Type II decompression sickness, which are described in volume 5.

Frequent oscillations in middle ear pressure associated with difficult clearing may lead to a condition of transient vertigo called alternobaric vertigo of descent. This vertigo usually follows a Valsalva maneuver, often with the final clearing episode

just as the diver reaches the bottom. The vertigo is short-lived but may cause significant disorientation.

Alternobaric vertigo may also occur during ascent in association with middle ear overpressurization. In this case, the vertigo is often preceded by pain in the ear that is not venting excess pressure. The vertigo usually lasts for only a few minutes, but may be incapacitating during that time. Relief is abrupt and may be accompanied by a hissing sound in the affected ear. Alternobaric vertigo during ascent disappears immediately when the diver halts his ascent and descends a few feet.

3-8.12.2   **Inner Ear Barotrauma.** A pressure imbalance between the middle ear and external environment may cause lasting damage to the inner ear if the imbalance is sudden or large. This type of inner ear barotrauma is often associated with round or oval window rupture.

There are three bones in the middle ear: the malleus, the incus, and the stapes. They are commonly referred to as the hammer, anvil, and stirrup, respectively (Figure 3-9). The malleus is connected to the eardrum (tympanic membrane) and transmits sound vibrations to the incus, which in turn transmits these vibrations to the stapes, which relays them to the inner ear. The stapes transmits these vibrations to the inner ear fluid through a membrane-covered hole called the oval window. Another membrane-covered hole called the round window connects the inner ear with the middle ear and relieves pressure waves in the inner ear caused by movement of the stapes.
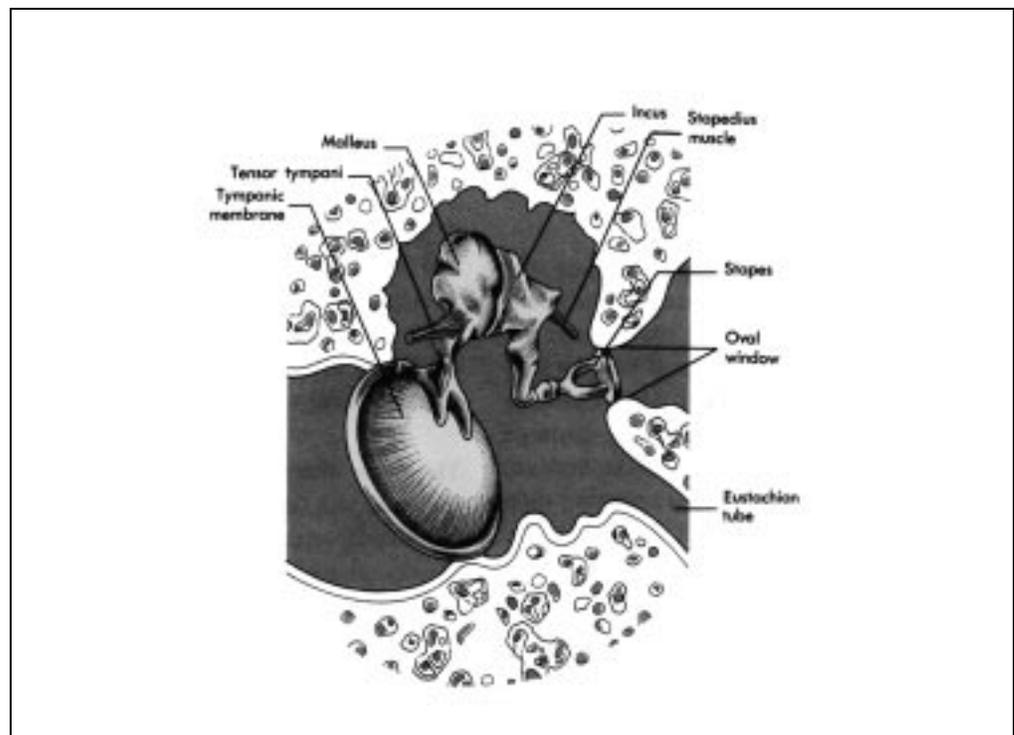
Figure 3-9. Impedance Matching Components of Inner Ear.

Barotrauma can rupture the round window membrane, causing the inner ear fluid (perilymphatic fluid) to leak. A persistent opening following barotrauma that drains perilymphatic fluid from the inner ear into the middle ear is referred to as a perilymph fistula. Perilymph fistula can occur when the diver exerts himself, causing an increase in intracranial pressure. If great enough, this pressure can be transmitted to the inner ear, causing severe damage to the round window membrane. The oval window is very rarely affected by barotrauma because it is protected by the foot of the stapes. Inner ear damage can also result from overpressurization of the middle ear by a too-forceful Valsalva maneuver. The maneuver, in addition to its desired effect of forcing gas up the eustachian tube, increases the pressure of fluid within the inner ear. Symptoms of this inner ear dysfunction include ringing or roaring in the affected ear, vertigo, disorientation, nystagmus, unsteadiness, and marked hearing loss.

The diagnosis of inner ear barotrauma should be considered whenever any inner ear symptoms occur during compression or after a shallow dive where decompression sickness is unlikely. In some cases it is difficult to distinguish between symptoms of inner ear barotrauma and decompression sickness or arterial gas embolism. Recompression is not harmful if it turns out barotrauma was the cause of the symptoms, provided the simple precautions outlined in volume 5 are followed. When in doubt, recompress. All cases of suspected inner ear barotrauma should be referred to an ear, nose and throat (ENT) physician as soon as possible. Treatment of inner ear barotrauma ranges from bed rest to exploratory surgery, depending on the severity of the symptoms.

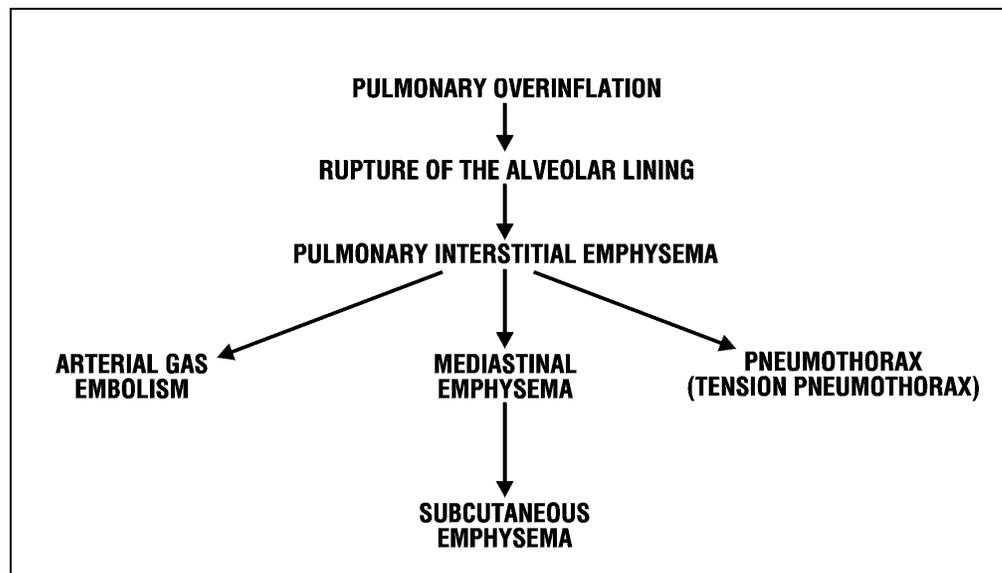## 3-9 PULMONARY OVERINFLATION SYNDROMES

Pulmonary overinflation syndromes are a group of barotrauma-related diseases caused by the expansion of gas trapped in the lung during ascent (reverse squeeze) or overpressurization of the lung with subsequent overexpansion and rupture of the alveolar air sacs. Excess pressure inside the lung can also occur when a diver presses the purge button on a single-hose regulator while taking a breath. The two main causes of alveolar rupture are:

■ Excessive pressure inside the lung caused by positive pressure
■ Failure of expanding gas to escape from the lung during ascent

Pulmonary overinflation from expanding gas failing to escape from the lung during ascent can occur when a diver voluntarily or involuntarily holds his breath during ascent. Localized pulmonary obstructions that can cause air trapping, such as asthma or thick secretions from pneumonia or a severe cold, are other causes. The conditions that bring about these incidents are different from those that produce lung squeeze and they most frequently occur during free and buoyant ascent training or emergency ascent from dives made with lightweight diving equipment or scuba.

The clinical manifestations of pulmonary overinflation depend on the location where the free air collects. In all cases, the first step is rupture of the alveolus with

a collection of air in the lung tissues, a condition known as interstitial emphysema. Interstitial emphysema causes no symptoms unless further distribution of the air occurs. Gas may find its way into the chest cavity or arterial circulation. These conditions are depicted in Figure 3-10.

PULMONARY OVERINFLATION

↓

RUPTURE OF THE ALVEOLAR LINING

↓

PULMONARY INTERSTITIAL EMPHYSEMA

ARTERIAL GAS EMBOLISM   **MEDIASTINAL EMPHYSEMA**   **PNEUMOTHORAX (TENSION PNEUMOTHORAX)**

↓

SUBCUTANEOUS EMPHYSEMA

Figure 3-10. Pulmonary Overinflation Consequences. Leaking of gas into the pulmonary interstital tissue causes no symptoms unless further leaking occurs. If gas enters the arterial circulation, potentially fatal arterial gas embolism may occur. Pneumothorax occurs if gas accumulates between the lung and chest wall and if accumulation continues without venting, then tension pneumothorax may result.

3-9.1   **Arterial Gas Embolism.** Arterial gas embolism is the most serious potential complication of diving and is caused by an excess pressure inside the lungs that fails to vent during ascent (Figure 3-11). For example, if a diver ascends to the surface from a depth of 100 fsw, the air within his lungs expands to four times its original volume. If this expanding air is not allowed to escape, pressure builds up within the lungs, overexpanding them and rupturing their air sacs and blood vessels. Air is then forced into the pulmonary capillary bed and bubbles are carried to the left chambers of the heart, where they are then pumped out into the arteries. Any bubble that is too large to go through an artery lodges and forms a plug (embolus). The tissues beyond the plug are then deprived of their blood supply and their oxygen. The consequences depend upon the area or organ where the blockage occurs. When the brain is involved, the symptoms are usually extremely serious. Unless the victim is recompressed promptly to reduce the size of the bubble and permit blood to flow again, death may follow. The symptoms and treatment of arterial gas embolism are discussed more fully in volume 5.

A diver shall never hold his breath on ascent. A diver who does may feel a sensation of discomfort behind the breast bone (sternum) and a stretching of the lungs. Fear and inhalation of water can also trigger a spasm of the laryngeal muscles (laryngospasm) that seals the main lung passageway and thus brings about the
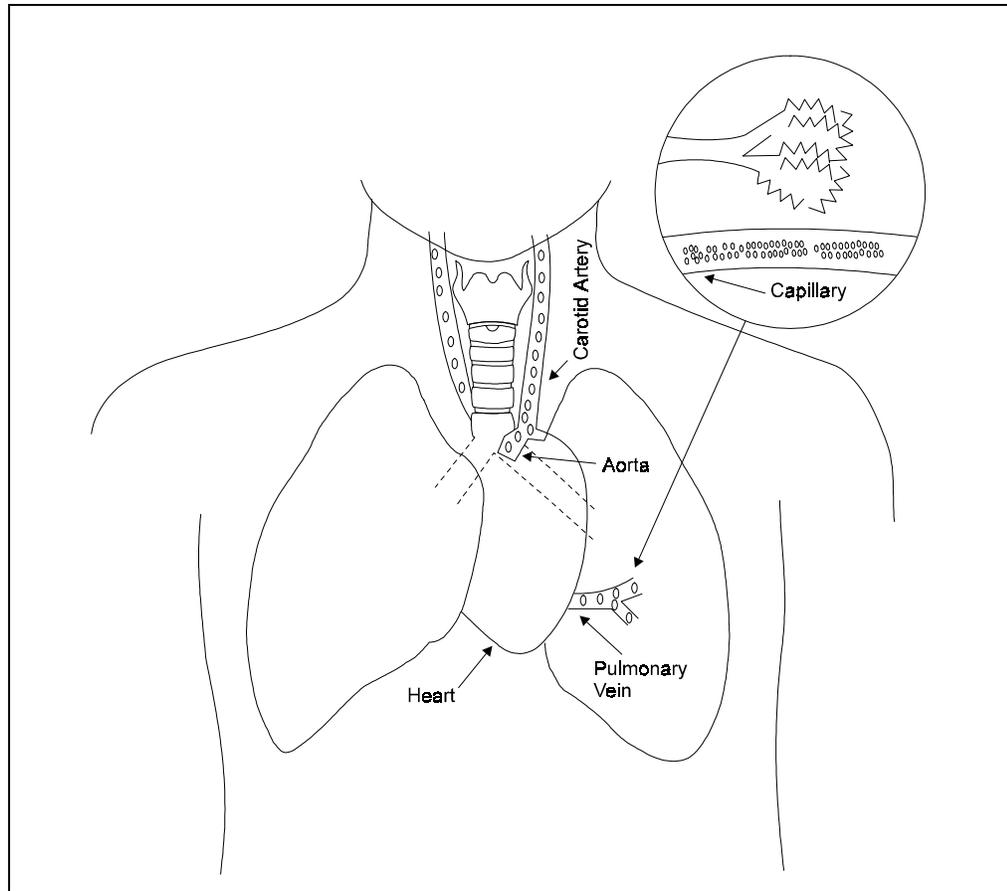
Figure 3-11. Arterial Gas Embolism.

overexpansion of the lungs. Under these circumstances, death has occurred during ascent from depths of only a few feet. Every diver shall make it an absolute rule to breathe normally and continually during ascent. However, a diver who cannot breathe because he is out of air or because his gear is not working must exhale during ascent.

3-9.2　**Mediastinal and Subcutaneous Emphysema.** Mediastinal emphysema (Figure 3-12) occurs when gas has been forced through torn lung tissue into the loose mediastinal tissues in the middle of the chest, around the heart, the trachea, and the major blood vessels. Subcutaneous emphysema (Figure 3-13) results from the expansion of gas that has leaked from the mediastinum into the subcutaneous tissues of the neck. These types of emphysema, including interstitial emphysema, should not be confused with the emphysema brought on by the aging process or by smoking.

3-9.3　**Pneumothorax.** Pneumothorax is the result of air entering the potential space between the lung covering and the lining of the chest wall (Figure 3-14). In its usual manifestation, called a simple pneumothorax, a one-time leakage of air from the lung into the chest partially collapses the lung, causing varying degrees of respiratory distress. This condition normally improves with time as the air is reab-
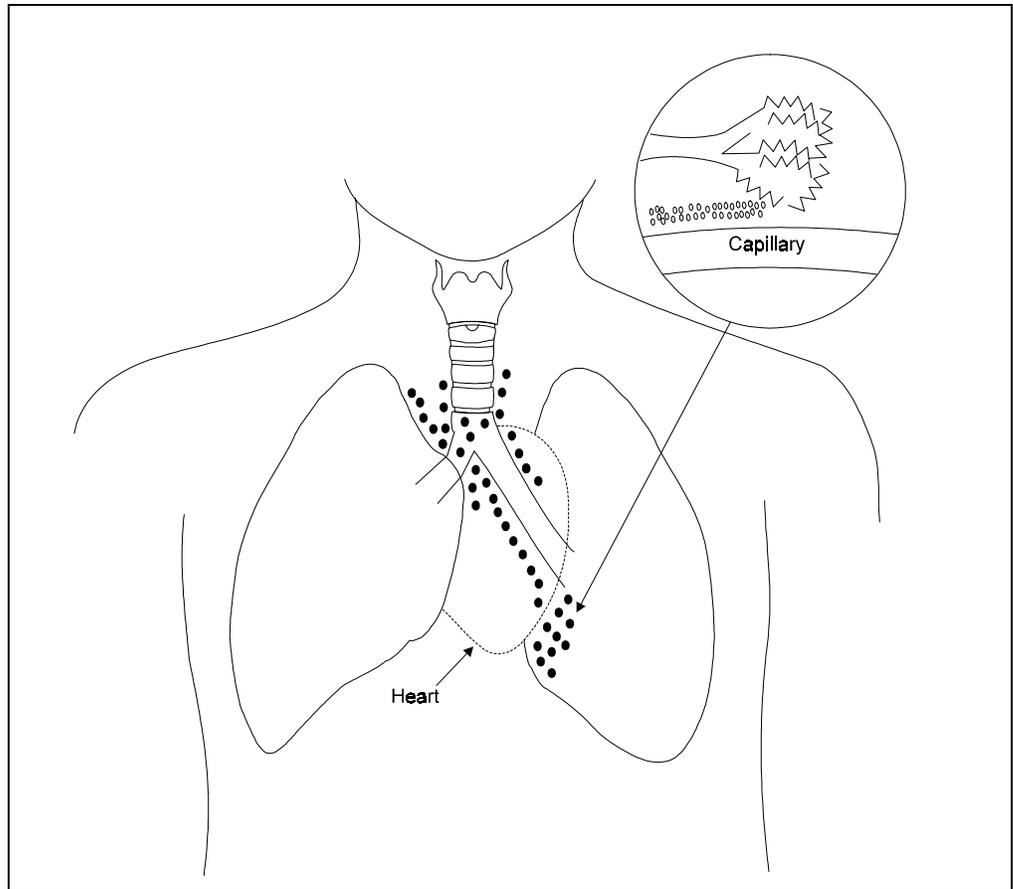
Figure 3-12. Mediastinal Emphysema.

sorbed. In severe cases of collapse, the air must be removed with the aid of a tube or catheter. The onset of pneumothorax is accompanied by a sudden, sharp chest pain, followed by difficult, rapid breathing, cessation of normal chest movements on the affected side, tachycardia, a weak pulse, and anxiety. A diver believed to be suffering from pneumothorax shall be thoroughly examined for the presence of arterial gas embolism. This is covered more fully in volume 5.

In certain instances, the damaged lung may allow air to enter but not exit the pleural space. Successive breathing gradually enlarges the air pocket. This is called a tension pneumothorax (Figure 3-15) due to the progressively increasing tension or pressure exerted on the lung and heart by the expanding gas. If uncorrected, this force presses on the involved lung, causing it to completely collapse. The lung, and then the heart, are pushed toward the opposite side of the chest, which impairs both respiration and circulation. The symptoms become progressively more serious, beginning with rapid breathing and ending in cyanosis (a bluish skin color), hypotension (low blood pressure), shock and, unless corrected, death.

If a simple pneumothorax occurs in a diver under pressure, the air will expand during ascent, according to Boyle's law, creating a tension pneumothorax. The
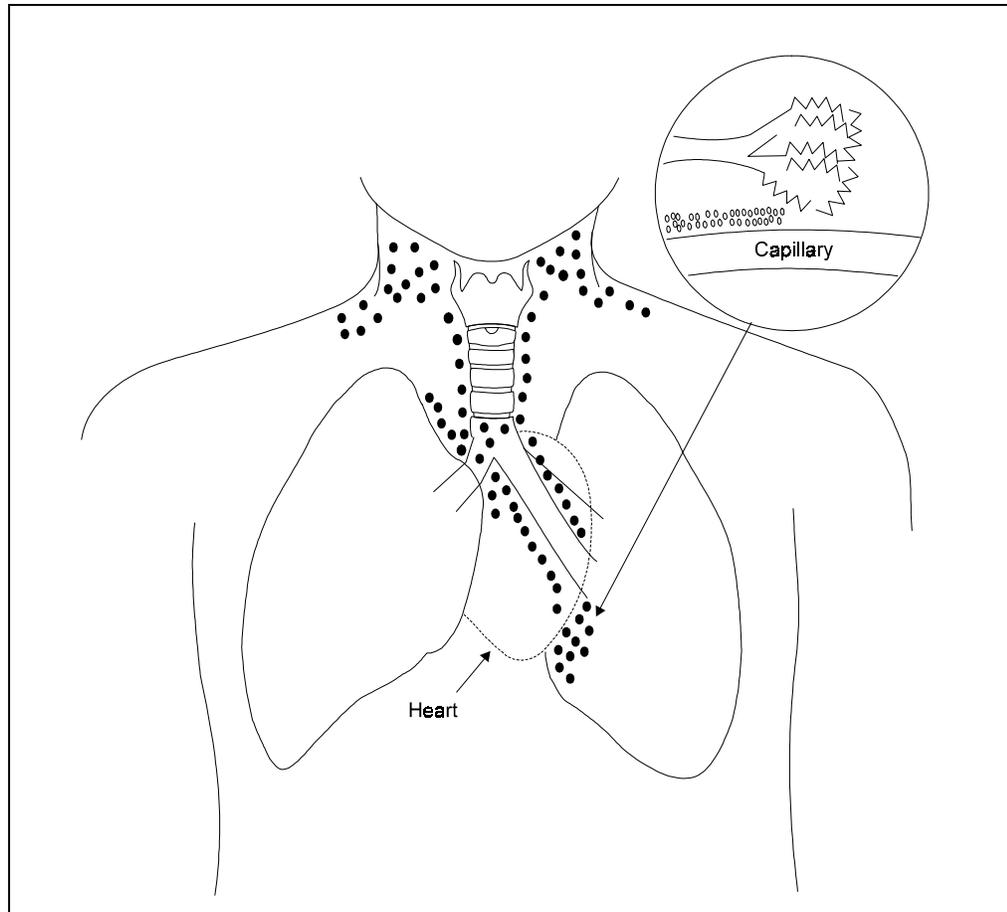
Figure 3-13. Subcutaneous Emphysema.

volume of air initially leaked into the pleural cavity and the remaining ascent distance will determine the diver's condition upon surfacing.

All cases of pneumothorax must be treated. This is sometimes done by removing the air with a catheter or tube inserted into the chest cavity. In cases of tension pneumothorax, this procedure may be lifesaving. Volume 5 fully discusses the treatment of simple and tension pneumothorax.

3-10   INDIRECT EFFECTS OF PRESSURE

The conditions previously described occur because of differences in pressure that damage body structures in a direct, mechanical manner. The indirect or secondary effects of pressure are the result of changes in the partial pressure of individual gases in the diver's breathing medium. The mechanisms of these effects include saturation and desaturation of body tissues with dissolved gas and the modification of body functions by abnormal gas partial pressures.

3-10.1   **Nitrogen Narcosis.** In diving, inert gas narcosis impairs the diver's ability to think clearly. The most common form, nitrogen narcosis, is caused by breathing compressed air at depth.
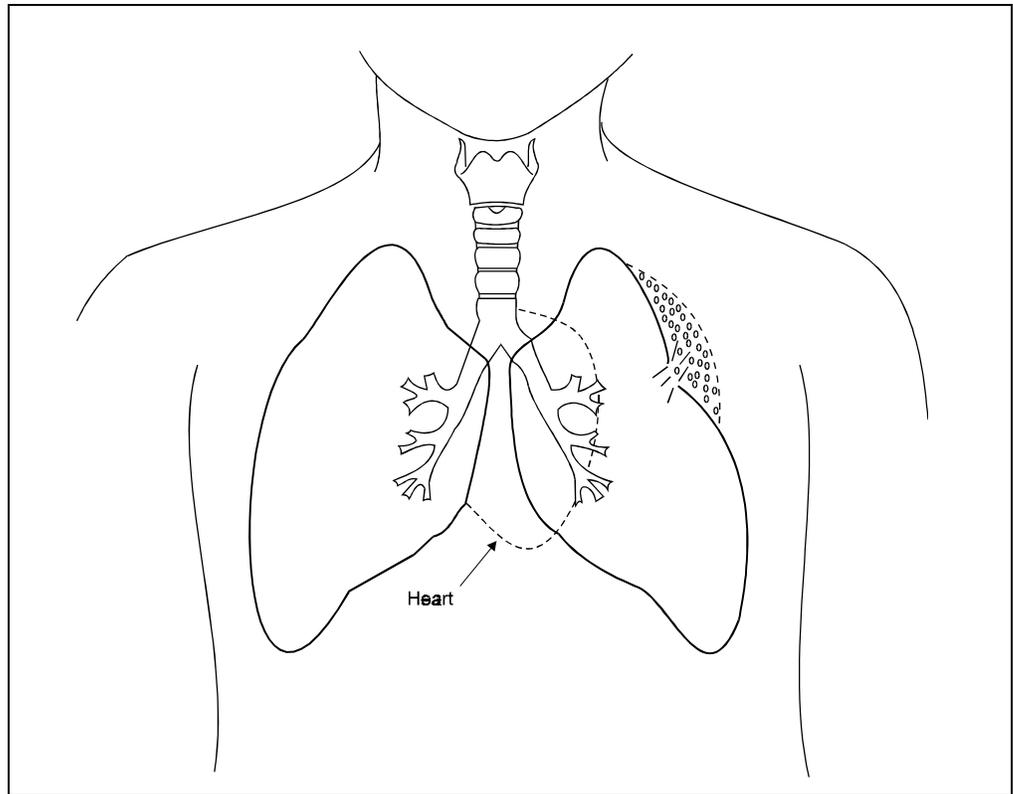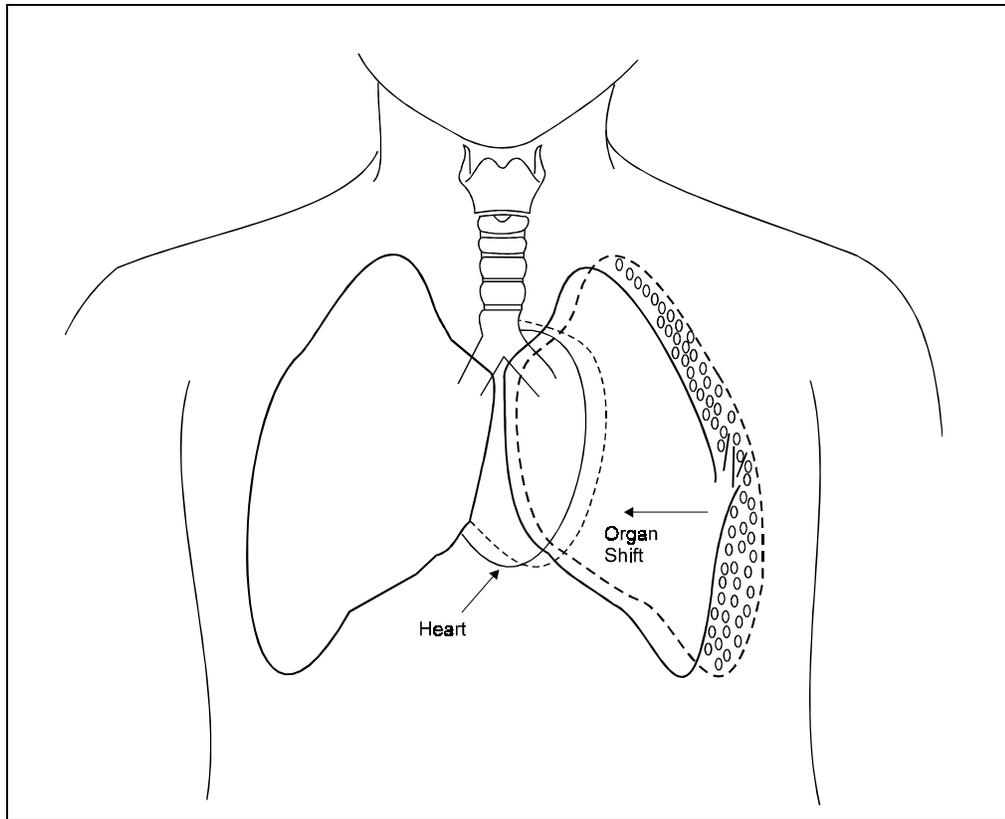
Figure 3-14. Pneumothorax.

3-10.1.1   **Symptoms of Narcosis.** The signs of narcosis are:

- Loss of judgment or skill
- A false feeling of well-being
- Lack of concern for job or safety
- Apparent stupidity
- Inappropriate laughter
- Tingling and vague numbness of the lips, gums, and legs

Disregard for personal safety is the greatest hazard of nitrogen narcosis. Divers may display abnormal behavior such as removing the regulator mouthpiece or swimming to unsafe depths without regard to decompression sickness or air supply. There is no specific treatment for nitrogen narcosis; the diver must be brought to shallower depths where the effects are not felt.

3-10.1.2   **Susceptibility to Narcosis.** Inert gases vary in their narcotic potency. The effects from nitrogen may first become noticeable at depths exceeding 100 fsw, but become more pronounced at depths greater than 150 fsw. There is a wide range of individual susceptibility and some divers, particularly those experienced in deep operations with air, can often work as deep as 200 fsw without serious difficulty.

Experienced and stable divers may be reasonably productive and safe at depths where others fail. They are familiar with the extent to which nitrogen narcosis

Figure 3-15. Tension Pneumothorax.

impairs performance. They know that a strong conscious effort to continue the dive requires unusual care, time, and effort to make even the simplest observations and decisions. Any relaxation of conscious effort can lead to failure or a fatal blunder.

Experience, frequent exposure to deep diving, and training may enable divers to perform air dives as deep as 180-200 fsw, but novices and susceptible individuals should remain at shallower depths. The performance or efficiency of divers breathing compressed air is impaired at depths greater than 180 fsw. At 300 fsw or deeper, the signs and symptoms are severe and the diver may hallucinate, exhibit bizarre behavior, or lose consciousness. Furthermore, the associated increase in oxygen partial pressure at such depths may produce oxygen convulsions. (Helium is widely used in mixed-gas diving as a substitute for nitrogen to prevent narcosis. Helium has not demonstrated narcotic effects at any depth tested by the U.S. Navy.) Figure 3-16 shows the narcotic effects of compressed air diving.

3-10.2   **Oxygen Toxicity.** Partial pressure of oxygen in excess of that encountered at normal atmospheric conditions may be toxic to the body. Oxygen toxicity is dependent upon both partial pressure and exposure time. The two types of oxygen toxicity experienced by divers are pulmonary oxygen toxicity and central nervous system (CNS) oxygen toxicity.

Nitrogen Narcosis

Depth (fsw)	Symptoms Include
100	Intoxicating effect similar to that of alcohol
	Slowing of mental activity
150	Slowing of reaction time and reflexes
	General euphoria
	Fixation of ideas
200	Difficulty in concentrating or reasoning
	Difficulty in remembering what to do or what has already been done
240	Observations often inaccurate
	Likely to make incorrect decision about what to do
	Diver may not care about job or safety

Figure 3-16. Nitrogen Narcosis.

3-10.2.1   **Pulmonary Oxygen Toxicity.** Low pressure oxygen poisoning, or pulmonary oxygen toxicity, can begin to occur if more than 60 percent oxygen is breathed at one atmosphere for 24 hours or more. While diving, this can occur after a 24-hour exposure to ppO_2 of 0.6 ata (e.g., 60 fsw breathing air). Long exposures to higher levels of oxygen, such as administered during Recompression Treatment Tables 4, 7, and 8, may quickly lead to pulmonary oxygen toxicity. The symptoms of pulmonary oxygen toxicity may begin with a burning sensation on inspiration and progress to pain on inspiration. During recompression treatments, pulmonary oxygen toxicity may have to be tolerated in patients with severe neurological symptoms to effect adequate treatment. In conscious patients, the pain and coughing experienced with inspiration eventually limit further exposure to oxygen. Return to normal pulmonary function gradually occurs after the exposure is terminated. Unconscious patients who receive oxygen treatments do not feel pain and it is possible to subject them to exposures resulting in permanent lung damage or pneumonia. For this reason, care must be taken when administering 100 percent oxygen to unconscious patients even at surface pressure.

3-10.2.2   **Central Nervous System (CNS) Oxygen Toxicity.** High pressure oxygen poisoning, or central nervous system (CNS) oxygen toxicity, is most likely to occur when divers are exposed to more than 1.6 atmospheres of oxygen.

Susceptibility to central nervous system oxygen toxicity varies from person to person. Individual susceptibility varies from time to time and for this reason divers may experience CNS oxygen toxicity at exposure times and pressures previously tolerated. Because it is the partial pressure of oxygen itself that causes toxicity, the problem can occur when mixtures of oxygen with nitrogen or helium are breathed at depth. Oxygen toxicity is influenced by the density of the breathing gas and the characteristics of the diving system used. Thus, allowable limits for oxygen partial pressures differ to some degree for specific diving systems (which are discussed in

later chapters). In general, oxygen partial pressures at or below 1.4 ata are unlikely to produce CNS toxicity. Closed-system oxygen rebreathing systems require the lowest partial pressure limits, whereas surface-supplied helium-oxygen systems permit slightly higher limits.

3-10.2.2.1 ***Factors Contributing to CNS Oxygen Toxicity.*** Three major external factors contributing to the development of oxygen toxicity are the presence of a high level of carbon dioxide in the breathing mixture resulting from CO_2 absorbent failure, carbon dioxide in the helmet supply gas, or inadequate ventilation during heavy exertion.

3-10.2.2.2 ***Symptoms of CNS Oxygen Toxicity.*** The most serious direct consequence of oxygen toxicity is convulsions. Sometimes recognition of early symptoms may provide sufficient warning to permit reduction in oxygen partial pressure and prevent the onset of more serious symptoms. The warning symptoms most often encountered also may be remembered by the mnemonic VENTIDC:

V: Visual symptoms: Tunnel vision, a decrease in diver's peripheral vision, and other symptoms, such as blurred vision, may occur.

E: Ear symptoms. Tinnitus, any sound perceived by the ears but not resulting from an external stimulus, may resemble bells ringing, roaring, or a machinery-like pulsing sound.

N: Nausea or spasmodic vomiting. These symptoms may be intermittent.

T: Twitching and tingling symptoms. Any of the small facial muscles, lips, or muscles of the extremities may be affected. These are the most frequent and clearest symptoms.

I: Irritability: Any change in the diver's mental status including confusion, agitation, and anxiety.

D: Dizziness. Symptoms include clumsiness, incoordination, and unusual fatigue.

C: Convulsions. The first sign of CNS oxygen toxicity may be a convulsion that occurs with little or no warning.

Symptoms may not always appear and most are not exclusively symptoms of oxygen toxicity. Twitching is perhaps the clearest warning of oxygen toxicity, but it may occur late, if at all. The appearance of any one of these symptoms usually represents a bodily signal of distress of some kind and should be heeded.

3-10.2.3 **CNS Convulsions.** Convulsions, the most serious direct consequence of CNS oxygen toxicity, may occur suddenly without being preceded by any other symptom. During a convulsion, the individual loses consciousness and his brain sends out uncontrolled nerve impulses to his muscles. At the height of the seizure, all of the muscles are stimulated at once and lock the body into a state of rigidity.

This is referred to as the *tonic phase* of the convulsion. The brain soon fatigues and the number of impulses slows. This is the *clonic phase* and the random impulses to various muscles may cause violent thrashing and jerking for a minute or so.

After the convulsive phase, brain activity is depressed and a postconvulsive (postictal) depression follows. During this phase, the patient is usually unconscious and quiet for a while, then semiconscious and very restless. He will then usually sleep on and off, waking up occasionally though still not fully rational. The depression phase sometimes lasts as little as 15 minutes, but an hour or more is not uncommon. At the end of this phase, the patient often becomes suddenly alert and complains of no more than fatigue, muscular soreness, and possibly a headache. After an oxygen-toxicity convulsion, the diver usually remembers clearly the events up to the moment when consciousness was lost, but remembers nothing of the convulsion itself and little of the postictal phase.

3-10.2.3.1   **Recommended Actions.** Despite its rather alarming appearance, the convulsion itself is usually not much more than a strenuous muscular workout for the victim. In an oxygen convulsion, the possible danger of hypoxia during breathholding in the tonic phase is greatly reduced because of the high partial pressure of oxygen in the tissues and brain. If a convulsion occurs in a recompression chamber, it is important to keep the individual from thrashing against hard objects and being injured. Complete restraint of the individual's movements is neither necessary nor desirable. The oxygen mask shall be removed immediately. It is not necessary to force the mouth open to insert a bite block while a convulsion is taking place. After the convulsion subsides and the mouth relaxes, keep the jaw up and forward to maintain a clear airway until the diver regains consciousness. Breathing almost invariably resumes spontaneously.

Convulsions may lead to squeeze while surface-supplied helmet diving if the diver falls to a greater depth, but bruises and a chewed tongue are more likely the only consequences. Bringing a diver up rapidly during the height of a convulsion could possibly lead to gas embolism. When using scuba, the most serious consequence of convulsions is drowning. In this situation, using the buddy system can mean the difference between life and death.

The biochemical changes in the central nervous system caused by high oxygen partial pressures are not instantaneously reversed by reducing the oxygen partial pressure. If one of the early symptoms of oxygen toxicity occurs, the diver may still convulse up to a minute or two after being removed from the high oxygen breathing gas. One should not assume that an oxygen convulsion will not occur unless the diver has been off oxygen for 2 or 3 minutes.

If a diver with oxygen convulsions is prevented from drowning or causing other injury to himself, full recovery with no lasting effects occurs within 24 hours. Susceptibility to oxygen toxicity does not increase, although divers may be more inclined to notice warning symptoms during subsequent exposures to oxygen. However, this is most likely a psychological matter.

3-10.2.3.2 **_Prevention._** The actual mechanism of CNS oxygen toxicity remains unknown in spite of many theories and much research. Preventing oxygen toxicity is important to divers. When use of high pressures of oxygen is advantageous or necessary, divers should take sensible precautions, such as being sure the breathing apparatus is in good order, observing depth-time limits, avoiding excessive exertion, and heeding abnormal symptoms that may appear.

3-10.3 **Absorption of Inert Gases.** The average human body at sea level contains about one liter of dissolved nitrogen. All of the body tissues are saturated with nitrogen at a partial pressure equal to the partial pressure in the alveoli, about 570 mmHg (0.75 ata). If the partial pressure of nitrogen changes because of a change in the pressure of the composition of the breathing mixture, the pressure of the nitrogen dissolved in the body gradually attains a matching level. Additional quantities are absorbed or some of the gas is eliminated, depending on the partial pressure gradient, until the nitrogen partial pressures in the lungs and in the tissues are in balance.

As described in Henry's law, the amount of gas that dissolves in a liquid is almost directly proportional to the partial pressure of that gas. If one liter of inert gas is absorbed at a pressure of one atmosphere, then two liters are absorbed at two atmospheres and three liters at three atmospheres, etc.

The process of taking up more nitrogen is called absorption or saturation. The process of giving up nitrogen is called _elimination_ or _desaturation_. The chain of events is essentially the same in both of these processes even though the direction of exchange is opposite. In diving, both saturation (when the diver is exposed to an increased partial pressure of nitrogen at depth) and desaturation (when he returns to the surface) are important. The same processes occur with helium and other inert gases.

3-10.4 **Saturation of Tissues.** The sequence of events in the process of saturation can be illustrated by considering what happens in the body of a diver taken rapidly from the surface to a depth of 100 fsw (Figure 3-17). To simplify matters, we can say that the partial pressure of nitrogen in his blood and tissues on leaving the surface is roughly 0.8 ata. When the diver reaches 100 fsw, the alveolar nitrogen pressure in his lungs will be about 0.8×4 ata or 3.2 ata, while the blood and tissues remain temporarily at 0.8 ata.

3-10.4.1 **Nitrogen Saturation Process.** The partial pressure difference or gradient between the alveolar air and the blood and tissues is thus 3.2 minus 0.8, or 2.4 ata. This gradient is the driving force that makes the molecules of nitrogen move by diffusion from one place to another. Consider the following 10 events and factors in the diver at 100 fsw:

1. As blood passes through the alveolar capillaries, nitrogen molecules move from the alveolar air into the blood. By the time the blood leaves the lungs, it has reached equilibrium with the new alveolar nitrogen pressure. It now has a nitrogen tension (partial pressure) of 3.2 ata and contains about four times as much nitrogen as before. When this blood reaches the tissues, there is a similar

SATURATION OF TISSUES
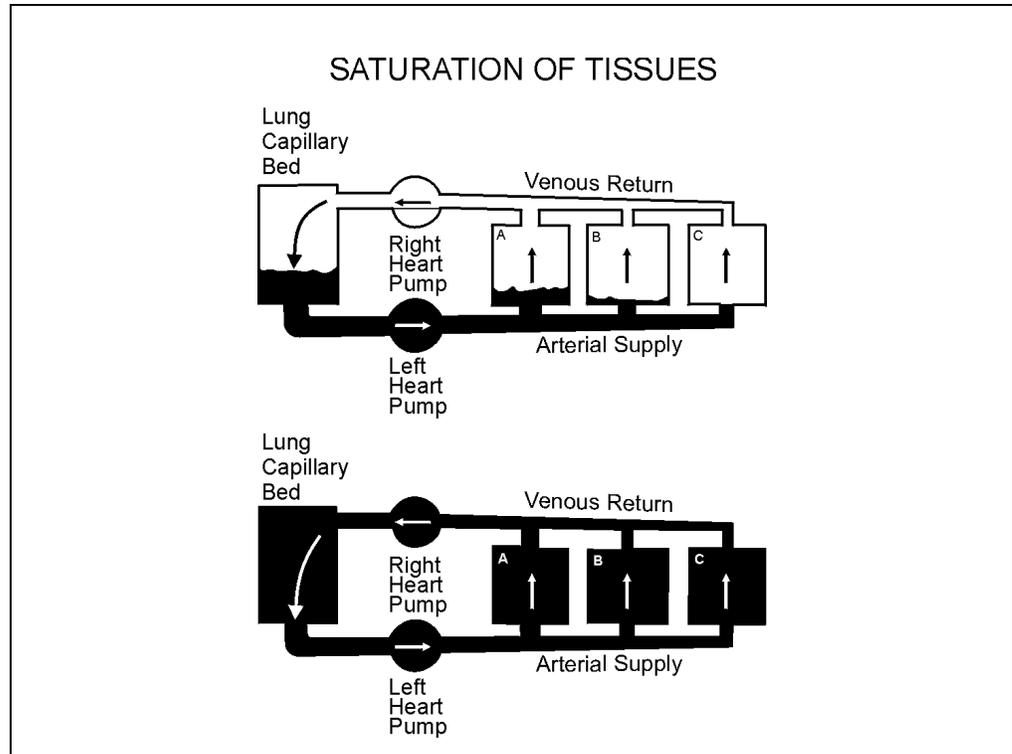
Figure 3-17. Saturation of Tissues. Shading in diagram indicates saturation with nitrogen or helium under increased pressure. Blood becomes saturated on passing through lungs, and tissues are saturated in turn via blood. Those with a large supply (as in A above) are saturated much more rapidly than those with poor blood supply (C) or an unusually large capacity for gas, as fatty tissues have for nitrogen. In very abrupt ascent from depth, bubbles may form in arterial blood or in "fast" tissue (A) even through the body as a whole is far from saturation. If enough time elapses at depth, all tissues will become equally saturated, as shown in lower diagram.

gradient and nitrogen molecules move from the blood into the tissues until equilibrium is reached.

2. The volume of blood in a tissue is relatively small compared to the volume of the tissue and the blood can carry only a limited amount of nitrogen. Because of this, the volume of blood that reaches a tissue over a short period of time loses its excess nitrogen to the tissue without greatly increasing the tissue nitrogen pressure.

3. When the blood leaves the tissue, the venous blood nitrogen pressure is equal to the new tissue nitrogen pressure. When this blood goes through the lungs, it again reaches equilibrium at 3.2 ata.

4. When the blood returns to the tissue, it again loses nitrogen until a new equilibrium is reached.

5. As the tissue nitrogen pressure rises, the blood-tissue gradient decreases, slowing the rate of nitrogen exchange. The rate at which the tissue nitrogen partial pressure increases, therefore, slows as the process proceeds. However, each volume of blood that reaches the tissue gives up some nitrogen which

increases the tissue partial pressure until complete saturation, in this case at 3.2 ata of nitrogen, is reached.

6. Tissues that have a large blood supply in proportion to their own volume have more nitrogen delivered to them in a certain amount of time and therefore approach complete saturation more rapidly than tissues that have a poor blood supply.

7. All body tissues are composed of lean and fatty components. If a tissue has an unusually large capacity for nitrogen, it takes the blood longer to deliver enough nitrogen to saturate it completely. Nitrogen is about five times as soluble (capable of being dissolved) in fat as in water. Therefore, fatty tissues require much more nitrogen and much more time to saturate them completely than lean (watery) tissues do, even if the blood supply is ample. Adipose tissue (fat) has a poor blood supply and therefore saturates very slowly.

8. At 100 fsw, the diver's blood continues to take up more nitrogen in the lungs and to deliver more nitrogen to tissues, until all tissues have reached saturation at a pressure of 3.2 ata of nitrogen. A few watery tissues that have an excellent blood supply will be almost completely saturated in a few minutes. Others, like fat with a poor blood supply, may not be completely saturated unless the diver is kept at 100 fsw for 72 hours or longer.

9. If kept at a depth of 100 fsw until saturation is complete, the diver's body contains about four times as much nitrogen as it did at the surface. Divers of average size and fatness have about one liter of dissolved nitrogen at the surface and about four liters at 100 fsw. Because fat holds about five times as much nitrogen as lean tissues, much of a diver's nitrogen content is in his fatty tissue.

10. An important fact about nitrogen saturation is that the process requires the same length of time regardless of the nitrogen pressure involved. For example, if the diver had been taken to 33 fsw instead of 100, it would have taken just as long to saturate him completely and to bring his nitrogen pressures to equilibrium. In this case, the original gradient between alveolar air and the tissues would have been only 0.8 ata instead of 2.4 ata. Because of this, the amount of nitrogen delivered to tissues by each round of blood circulation would have been smaller from the beginning. Less nitrogen would have to be delivered to saturate him at 33 fsw, but the slower rate of delivery would cause the total time required to be the same.

3-10.4.2 **Other Inert Gases.** When any other inert gas, such as helium, is used in the breathing mixture, the body tissues become saturated with that gas in the same process as for nitrogen. However, the time required to reach saturation is different for each gas.

The actual total pressure of gases in a tissue may achieve significant supersaturation or subsaturation during the gas exchange when one gas replaces another in body tissues without a change in ambient pressure (isobaric gas exchange).

3-10.5   **Desaturation of Tissues.** The process of desaturation is the reverse of saturation (Figure 3-18). If the arterial pressure of the gas in the lungs is reduced, either through a change in pressure or a change in the breathing medium, the new pressure gradient induces the nitrogen to diffuse from the tissues to the blood, from the blood to the gas in the lungs, and then out of the body with the expired breath. Some parts of the body desaturate more slowly than others for the same reasons that they saturate more slowly: poor blood supply or a greater capacity to store gas.
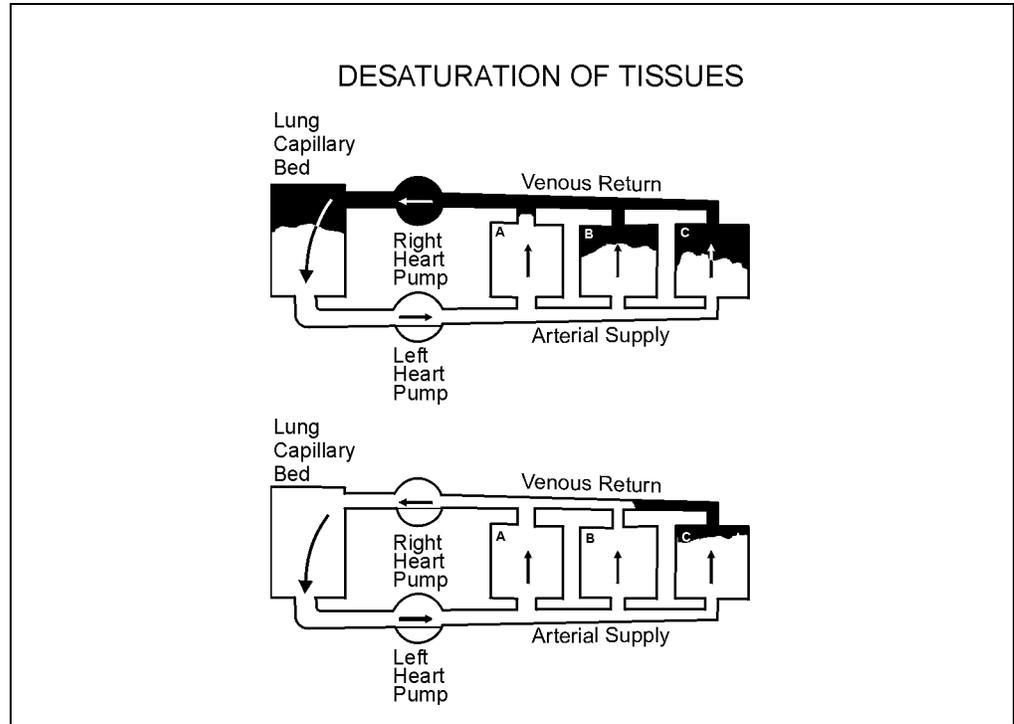
Figure 3-18. Desaturation of Tissues. The desaturation process is essentially the reverse of saturation. When pressure of inert gas is lowered, blood is cleared of excess gas as it goes through the lungs. Blood then removes gas from the tissues at rates depending on amount of blood that flows through them each minute. Tissues with poor blood supply (as in C in upper sketch) or large gas capacity will lag behind and may remain partially saturated after others have cleared (see lower diagram). If dive is long enough to saturate each tissue, long decompression stops are required to desaturate the tissue enough so that bubbles will not form in it on ascent.

3-10.5.1   **Saturation/Desaturation Differences.** There is a major difference between saturation and desaturation. The body accommodates large and relatively sudden increases in the partial pressure of the inspired gas without ill effect. The same is not true for desaturation, however, where a high pressure gradient (toward the outside) can lead to serious problems.

A diver working at a depth of 100 fsw is under a total pressure of 4 ata. The partial pressure of the nitrogen in the air he is breathing is approximately 3.2 ata (80 percent of 4 ata). If his body is saturated with nitrogen, the partial pressure of the

nitrogen in his tissues is also 3.2 ata. If the diver were to quickly ascend to the surface, the total hydrostatic pressure on his tissues would be reduced to 1 ata, whereas the tissue nitrogen tension would remain momentarily at 3.2 ata.

3-10.5.2 **Bubble Formation.** A dissolved gas can have a tension higher than the total pressure in the body. If a tissue is supersaturated with gas to this degree, the gas eventually separates from solution in the form of bubbles. Bubbles of nitrogen forming in the tissues and blood result in the condition known as decompression sickness. These bubbles can put pressure on nerves, damage delicate tissues, block the flow of blood to vital organs and induce biochemical changes and blood clotting. Symptoms may range from skin rash to mild discomfort and pain in the joints and muscles, paralysis, numbness, hearing loss, vertigo, unconsciousness, and in extreme cases, death.

Fortunately, the blood and tissues can hold gas in supersaturated solution to some degree without serious formation of bubbles. This permits a diver to ascend a few feet without experiencing decompression sickness, while allowing some of the excess gas to diffuse out of the tissues and be passed out of his body. By progressively ascending in increments and then waiting for a period of time at each level, the diver eventually reaches the surface without experiencing decompression sickness.

3-10.6 **Decompression Sickness.** As has been discussed, when a diver's blood and tissues have taken up nitrogen or helium in solution at depth, reducing the external pressure on ascent can produce a state of supersaturation, as has been discussed. If the elimination of dissolved gas, via the circulation and the lungs, fails to keep up with the reduction of external pressure, the degree of supersaturation may reach the point at which the gas can no longer stay in solution. The situation then resembles what happens when a bottle of carbonated beverage is uncapped.

3-10.6.1 **Direct Bubble Effects.** Supersaturated tissues may result in bubble formation in tissue or in the bloodstream. Also, bubbles may arise from the lung and enter the bloodstream from pulmonary overinflation (arterial gas embolism). Once in the bloodstream these bubbles will cause symptoms depending only on where they end up, not on their source. These bubbles may exert their effects directly in several ways:

■ Direct blockage of arterial blood supply leading to tissue hypoxia, tissue injury, and death. This is called embolism and may occur from pulmonary damage (arterial gas embolism) or from the bubbles reaching the arterial circulation during decompression. The mechanism usually causes cerebral (brain) symptoms.

■ Venous congestion from bubbles or slow blood flow and sludging, which leads to increased back pressure. This increased back pressure leads to hypoxia, tissue injury, and death. This is one of the mechanisms of injury in Spinal Cord DCS.

- ■ Direct pressure on surrounding tissue (autochthonous bubbles) causing stretching, pressure on nerve endings, or direct mechanical damage. This is another mechanism for Spinal Cord DCS and may be a mechanism for Musculoskeletal DCS.

- ■ Bubbles blocking blood flow in the lungs that leads to decreased gas exchange, hypoxia, and hypercarbia. This is the mechanism of damage in Pulmonary DCS.

The time course for these Direct Bubble Effects is short (a few minutes to hours). The only necessary treatment for Direct Bubble Effects is recompression. This will compress the bubble to a smaller diameter. This restores blood flow, decreases venous congestion and improves gas exchange in the lungs and tissues. It also increases the speed at which the bubbles outgas and collapse.

3-10.6.2 **Indirect Bubble Effects.** Bubbles may also exert their effects indirectly because a bubble present in a blood vessel acts like a foreign body. The body reacts as it would if there were a cinder in the eye or a splinter in the hand. The body's defense mechanisms become alerted and try to reject the foreign body. This try at rejection includes the following:

- ■ Blood vessels become "leaky" (due to chemical release). Blood plasma leaks out while blood cells remain inside. The blood becomes thick and causes sludging and decreased pressure downstream, with possible shock.

- ■ The platelet system becomes active and the platelets gather at the site of the bubble causing a clot to form.

- ■ The injured tissue releases fats that clump together in the bloodstream. These act as emboli, causing tissue hypoxia.

- ■ Injured tissues release histamine and histamine-like substances, causing edema, which leads to allergic-type problems of shock and respiratory distress.

Bubble reaction takes place in a longer period (up to 30 minutes or more) than the direct effects. Because the non-compressible clot replaces a compressible bubble, recompression alone is not enough. To restore blood flow and relieve hypoxia, hyperbaric treatment and other therapies are often required.

3-10.6.3 **Symptoms of Decompression Sickness.** The resulting symptoms depend on the location and size of the bubble or bubbles. Symptoms include pain in joints, muscles or bones when a bubble is in one of these structures. Bubble formation in the brain can produce blindness, dizziness, paralysis and even unconsciousness and convulsion. When the spinal cord is involved, paralysis and/or loss of feeling can occur. Bubbles in the inner ear produce hearing loss and vertigo. Bubbles in the lungs can cause coughing, shortness of breath, and hypoxia, a condition referred to as "the chokes." This condition often proves fatal. Skin bubbles produce itching or rash or both. Unusual fatigue or exhaustion after a dive is prob-

ably also due to bubbles in unusual locations and the biochemical changes they have induced. Decompression sickness that affects the central nervous system (brain or spinal cord) or lungs can produce serious disabilities and may even threaten life if not treated promptly and properly. When other areas such as joints are affected, the condition may produce excruciating pain and lead to local damage if not treated, but life is seldom threatened.

3-10.6.4 **Treating Decompression Sickness.** Treatment of decompression sickness is accomplished by recompression. This involves putting the victim back under pressure to reduce the size of the bubbles to cause them to go back into solution and to supply extra oxygen to the hypoxic tissues. Treatment is done in a recompression chamber, but can sometimes be accomplished in the water if a chamber cannot be reached in a reasonable period of time. Recompression in the water is not recommended, but if undertaken, must be done following specified procedures. Further discussion of the symptoms of decompression sickness and a complete discussion of treatment are presented in volume 5.

Modern research has shown that the symptoms caused by bubbles depend on their ultimate location and not their source. Bubbles entering the arterial circulation from the lung (pulmonary overinflation syndrome) have exactly the same effects as those arising from body tissues and cells (decompression sickness) that find their way into the arterial circulation. This means that the treatment of diseases caused by bubbles is dependent on the ultimate symptoms and symptom severity and not on the source of the bubbles.

This finding has lead to new treatment protocols in which the initial treatment for arterial gas embolism and decompression sickness is the same, recompression to 60 fsw. After that, treatment proceeds according to the patient's condition and response to therapy. Many agree with the opinion that Direct Bubble Effects are the cause of symptoms occurring early after surfacing. These cases usually respond to recompression alone. However, the longer after surfacing that symptoms appear, the more likely it is that the effect of the bubbles is responsible for symptoms, rather than the bubbles themselves. In this situation, recompression alone will be less effective.

3-10.6.5 **Preventing Decompression Sickness.** Prevention of decompression sickness is generally accomplished by following the decompression tables. However, individual susceptibility or unusual conditions, either in the diver or in connection with the dive, produces a small percentage of cases even when proper dive procedures are followed meticulously. To be absolutely free of decompression sickness under all possible circumstances, the decompression time specified would have to be far in excess of that normally needed. On the other hand, under ideal circumstances, some individuals can ascend safely in less time than the tables specify. This must not be taken to mean that the tables contain an unnecessarily large safety factor. The tables represent the minimum workable decompression time that permits average divers to surface safely from normal working dives without an unacceptable incidence of decompression sickness.

3-10.7   **High Pressure Nervous Syndrome (HPNS).** High Pressure Nervous Syndrome (HPNS) is a derangement of central nervous system function that occurs during deep helium-oxygen dives, particularly saturation dives. The cause is unknown. The clinical manifestations include nausea, fine tremor, imbalance, incoordination, loss of manual dexterity and loss of alertness. Abdominal cramps and diarrhea develop occasionally. In severe cases a diver may develop vertigo, extreme indifference to his surroundings and marked confusion such as inability to tell the right hand from the left hand. HPNS is first noted between 400 and 500 fsw and the severity appears to be both depth and compression rate dependent. With slow compression, depths of 1000 fsw may be achieved with relative freedom from HPNS. Beyond that, some HPNS may be present regardless of the compression rate. Attempts to block the appearance of the syndrome have included the addition of nitrogen or hydrogen to the breathing mixture and the use of various drugs. No method appears to be entirely satisfactory.

3-10.8   **Compression Pains.** Compression pains (referred to as compression arthralgia) result from increases in external pressure surrounding the body. These pains affect the joints and may occur in almost any diver. They have been experienced in the knees, shoulders, fingers, back, hips, neck, and ribs. Compression pains are often deep aching pains, similar to those of Type I decompression sickness. However, the pains may be relatively sudden in onset and initially intense. These pains may be accompanied by "popping" of joints or a dry, "gritty" feeling within the joint.

Symptoms are dependent on depth, rate of compression, and individual susceptibility. While primarily a problem encountered in saturation diving, symptoms may occur as shallow as 100 fsw at rapid compression rates, such as seen in air diving. In deep, helium saturation dives with slower compression rates, symptoms are more commonly seen deeper than 300 fsw. Deeper than 600 fsw, compression pains may occur even at very slow rates of compression. These pains may be severe enough to limit diver activity, travel rate and depths during downward excursions. Improvement is generally noted as time is spent at depth but, on occasion, these pains may last well into the decompression phase of the dive until shallower depths are reached. They can be distinguished from decompression sickness pain because they were present before decompression was started and do not increase in intensity with decreasing depth.

The mechanism of compression pain is unknown, but is thought to result from the sudden increase in tissue gas tension surrounding the joints causing fluid shifts and interfering with joint lubrication.

3-11   PHYSIOLOGICAL HAZARDS FROM MUNITIONS

Divers frequently work with explosive material or are involved in combat swimming and therefore may be subject to the hazards of underwater explosions. An explosion is the violent expansion of a substance caused by the gases released during rapid combustion. One effect of an explosion is a shock wave that travels outward from the center, somewhat like the spread of ripples produced by dropping a stone into a pool of water. This shock wave moving through the

surrounding medium (whether air or water) passes along some of the force of the blast.

A shock wave moves more quickly and is more pronounced in water than in air because of the relative incompressibility of liquids. Because the human body is mostly water and incompressible, an underwater shock wave passes through the body with little or no damage to the solid tissues. However, the air spaces of the body, even though they may be in pressure balance with the ambient pressure, do not readily transmit the overpressure of the shock wave. As a result, the tissues that line the air spaces are subject to a violent fragmenting force at the interface between the tissues and the gas.

The amount of damage to the body is influenced by a number of factors. These include the size of the explosion, the distance from the site, and the type of explosive (because of the difference in the way the expansion progresses in different types of explosives). In general, larger, closer, and slower-developing explosions are more hazardous. The depth of water and the type of bottom (which can reflect and amplify the shock wave) may also have an effect. Under average conditions, a shock wave of 500 psi or greater will cause injury to the lungs and intestinal tract.

The extent of injury is also determined in part by the degree to which the diver's body is submerged. For an underwater blast, any part of the body that is out of the water is not affected. Conversely, for an air blast, greater depth provides more protection. The maximum shock pressure to which a diver should be exposed is 50 psi. The safest and recommended procedure is to have all divers leave the water if an underwater explosion is planned or anticipated. A diver who anticipates a nearby underwater explosion should try to get all or as much of his body as possible out of the water. If in the water, the diver's best course of action is to float face up, presenting the thicker tissues of the back to the explosion.

3-12 THERMAL PROBLEMS AND OTHER PHYSIOLOGICAL PROBLEMS IN DIVING

Thermal problems arising from exposure to cold water pose the major consideration when planning operational dives and selecting equipment. The working diver commonly experiences heat loss during immersion and often expects to be uncomfortably chilled at the end of a dive. Bottom time limits may be determined by the diver's cold tolerance rather than by decompression considerations.

The human body functions effectively within a relatively narrow range of internal temperature. The average, or normal, core temperature of 98.6°F (37°C) is maintained by natural mechanisms of the body, aided by artificial measures such as the use of protective clothing or air conditioning when external conditions tend toward cold or hot extremes. Rewarming before a repetitive dive is as important as allowing for residual nitrogen levels.

When the body temperature is reduced below normal, gas absorption increases. This requires modification of decompression procedures by selecting a decompression table appropriate for the next longer or deeper dive schedule.

3-12.1	**Regulating Body Temperature.** The metabolic processes of the body constantly generate heat. If heat is allowed to build up inside the body, damage to the cells can occur. To maintain internal temperature at the proper level, the body must lose heat equal to the amount it produces.

Heat transfer is accomplished in several ways. The blood, while circulating through the body, picks up excess heat and carries it to the lungs, where some of it is lost with the exhaled breath. Heat is also transferred to the surface of the skin, where much of it is dissipated through a combination of conduction, convection, and radiation. Moisture released by the sweat glands cools the surface of the body as it evaporates and speeds the transfer of heat from the blood to the surrounding air. If the body is working hard and generating greater than normal quantities of heat, the blood vessels nearest the skin dilate to permit more of the heated blood to reach the body surfaces, and the sweat glands increase their activity.

Maintaining proper body temperature is particularly difficult for a diver working underwater. The principal temperature control problem encountered by divers is keeping the body warm. The high thermal conductivity of water, coupled with the normally cool-to-cold waters in which divers operate, can result in rapid and excessive heat loss.

3-12.2	**Excessive Heat Loss (Hypothermia).** When cold water enters a dry suit or a wet suit, the diver experiences a sudden drop in skin temperature. If a diver with no thermal protection is suddenly plunged into very cold water, the effects are immediate and rapidly disabling. The diver gasps and his respiratory rate and tidal volume increase. His breathing becomes so rapid and uncontrolled that he cannot coordinate his breathing and swimming movements. This lack of breathing control makes survival in rough, cold water very unlikely.

A water temperature of approximately 91°F (33°C) is required to keep an unprotected, resting man at a stable temperature. The unprotected diver will be affected by excessive heat loss and become chilled within a short period of time in water temperatures below 72°F (23°C). As his body temperature falls, the diver first feels uncomfortable and then, as his body tries to increase heat production in the muscles, shivering begins. If cooling continues, his ability to perform useful work becomes seriously impaired; his sense of touch is dulled and his hands lose dexterity. As shivering intensifies, it brings on a general lack of coordination and a scuba diver may experience difficulty keeping his mouthpiece in place. He soon loses his ability to think clearly and finds it increasingly difficult to concentrate.

At extremely low temperatures or with prolonged immersion, body heat loss reaches a point at which death occurs. Appropriate dress can greatly reduce the effects of heat loss and a diver with proper dress can work in very cold water for reasonable periods of time.

Inhaled gases are heated in the upper respiratory tract. More energy is required to heat the denser gases encountered at depth. Thus, heat loss through the respiratory tract becomes an increasingly significant factor in deeper diving. In fact, respira-

tory shock can develop if a diver breathes unheated gas while making deep saturation dives at normal water temperature.

3-12.2.1   **Internal Temperature Regulation.** The body's ability to tolerate cold environments is due to natural insulation and a built-in means of heat regulation. Temperature is not uniform throughout the body. It is more accurate to consider the body in terms of an inner core where a constant or uniform temperature prevails and a superficial region through which a temperature gradient exists from the core to the body surface. Over the trunk of the body, the thickness of the superficial layer may be 1 inch (2.5 cm). The extremities become a superficial insulating layer when their blood flow is reduced to protect the core.

Once in the water, heat loss through the superficial layer is lessened by the reduction of blood flow to the skin. The automatic, cold-induced vasoconstriction (narrowing of the blood vessels) lowers the heat conductance of the superficial layer and acts to maintain the heat of the body core. Unfortunately, vasoconstrictive regulation of heat loss has only a narrow range of protection. When the extremities are initially put into very cold water, vasoconstriction occurs and the blood flow is reduced to preserve body heat. After a short time, the blood flow increases and fluctuates up and down for as long as the extremities are in cold water. As circulation and heat loss increase, the body temperature falls and may continue falling, even though heat production is increased by shivering.

Much of the heat loss in the trunk area is transferred over the short distance from the deep organs to the body surface by physical conduction, which is not under any physiological control. Most of the heat lost from the body in moderately cold water is from the trunk and not the limbs.

3-12.2.2   **Effects of Exercise on Hypothermia.** Exercise normally increases heat production and body temperature in dry conditions. Paradoxically, exercise in cold water may cause the body temperature to fall more rapidly. Any movement that stirs the water in contact with the skin creates turbulence that carries off heat (convection). Heat loss is caused not only by convection at the limbs, but also by increased blood flow into the limbs during exercise. Continual movement causes the limbs to resemble the internal body core rather than the insulating superficial layer. These two conflicting effects result in the core temperature being maintained or increased in warm water and decreased in cold water.

Increased heat production requires an equivalent increase in oxygen consumption. The respiratory minute volume of the lungs must increase by the same magnitude. If a diver is breathing nine liters of air per minute at rest in the water and becomes chilled, his heat production may increase three times to compensate for chilling. His respiratory ventilation then increases to 36 liters per minute. In this example, the diver would have the same air consumption at rest keeping warm as when performing moderate work in warm water.

3-12.2.3   **Symptoms of Hypothermia.** All of these factors work against the diver. Even his body's natural insulation and protective function give way to cold water. The diver's thinking ability becomes impaired and the effect of this impairment on the

use of his hands and other motor functions may prevent him from choosing and executing the best procedures to complete a task. In some cases, his survival may be at stake.

The signs and symptoms of dropping body core temperature, from the first noticeable effects to death, are listed in Table 3-1. The treatment for hypothermia is discussed in Volume 5.

Table 3-1. Signs and Symptoms of Dropping Core Temperature.

Core Temperature		
°F	°C	Symptoms
98	37	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	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, shivering impaired
90	32	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

3-12.3   Excessive Heat (Hyperthermia). Diving in tropical areas, such as the Middle East, may expose a diver to heat stress both in and out of the water. Predive heat exposure may lead to significant dehydration putting the diver at risk once he enters the water. This is especially true if a protective suit has to be worn because of marine life or contamination. Specific guidelines based on temperature/time exposures are not available at this time but hyperthermia should be considered a potential risk any time air temperature exceeds 90°F and water temperature is above 82°F.

3-12.3.1   **Heat Stress Factors.** The magnitude of heat stress imposed on a diver depends on water temperature, duration of the dive, thermal protection garment, and the rate at which the diver is working. Heat stress is related to a rise in the body core temperature. An individual is considered to have developed hyperthermia when core

temperature rises 1.8°F (1°C) above normal (98.6°F, 37°C). The maximum safe body core temperature is 102.2°F (39°C). A diver wearing a wet suit in cool water while performing hard work can reach this upper limit, as can a diver wearing no thermal protection in warmer water and working at a lower rate. If during work a diver feels hot and uncomfortable, then he should consider decreasing his work rate or limiting his exposure.

Individual differences affect development of hyperthermia. Physically fit individuals and those with lower levels of body fat are less likely to develop hyperthermia. Drinking adequate amounts of fluid reduces the risk, compared to dehydrated divers. Alcohol or caffeine beverages should be avoided since they can produce dehydration. Medications containing antihistamines or aspirin should not be used in warm water diving. Age (if 20-40 years old), sex, and race do not alter the risk of hyperthermia. Risk of acute oxygen toxicity may increase in warm water diving where 100 percent oxygen is breathed, so precautions should be heightened. There is no evidence that warm water diving increases the risk of decompression sickness.

3-12.3.2 **Acclimatization.** Acclimatization is the process where repeated exposures to heat will reduce (but not eliminate) the rise in core temperature. At least 5 consecutive days of acclimatization to warm water diving are needed to see an increased tolerance to heat. Exercise training is essential for acclimation to heat. Where possible, acclimatization should be completed before attempting long duration working dives. Acclimation should begin with short exposures and light workloads. All support personnel should also be heat acclimatized. Fully acclimatized divers can still develop hyperthermia, however. Benefits of acclimatization begin to disappear in 3 to 5 days after stopping exposure to warm water. Acclimatization can be maintained by diving or swimming in warm water on days when a diver is not scheduled for a working dive.

3-12.3.3 **Symptoms of Hyperthermia.** Signs and symptoms of hyperthermia can vary among individuals. Since a diver might have been in water that may not be considered hot, support personnel must not rely solely on classical signs and symptoms of heat stress for land exposures. Table 3-2 lists commonly encountered signs and symptoms of heat stress in diving.

A breathing rate higher than normally expected for the rate of work is an early warning of hyperthermia. Excessive breathing rates maintained for more than 1-2 minutes can produce light-headedness, muscle twitching, headache, or unconsciousness.

Mental abilities begin to deteriorate with core temperatures greater than 100.5°F. The ability to learn and retain new information will be impaired. Swimming patterns or work behavior may become progressively more erratic. By the time the diver's core temperature approaches 102°F noticeable mental confusion may be present.

3-12.3.4 **Impact of Dive Time on Hyperthermia.** The likelihood of hyperthermia increases with dive time. Dehydration may occur mainly through sweating; urination may

Table 3-2. *Signs of Heat Stress.*

Least Severe	High breathing rate
	Feeling of being hot, uncomfortable
	Low urine output
	Inability to think clearly
	Erratic swim or work pattern
	Fatigue
	Light-headedness or headache
	Nausea
	Muscle cramps
	Sudden rapid increase in pulse rate
	Disorientation, confusion
	Exhaustion
	Collapse
Most Severe	Death

be absent. A two-pound weight loss after a dive indicates a loss of one quart of body water. Losses greater than four pounds indicate marked dehydration. Muscle cramps may develop. A rapid increase in pulse rate can indicate severe hyperthermia. Divers may experience sensation of being hot, fatigued, disoriented, or nauseated before they collapse. Collapse may occur suddenly without prior warning signs.

Divers may appear physically functional in the water, but collapse when they exit the water. Therefore, all divers who have been in the water for more than an hour should be assisted out of the water and monitored carefully. If they feel light-headed when standing, they should lie down, receive adequate rehydration, and cease diving until the next day.

3-12.3.5　**Preventing Hyperthermia.** Like hypothermia, hyperthermia can be insidious and cause problems without the diver being aware of it. Acclimatization, adequate hydration, experience, and common sense all play a role in preventing hyperthermia. Shelter personnel from the sun and keep the amount of clothing worn to a minimum. Adequate predive hydration is essential. Urinating 1-2 times per hour, where urine is pale and clear, usually indicates adequate hydration. Standby divers and support personnel should consume about one quart of fluid per hour in hot environments. Frequent rest periods are advised.

3-12.4　**Dehydration.** Dehydration is a concern to divers, particularly in tropical zones. It is defined as an excessive loss of water from the body tissues and is accompanied

by a disturbance in the balance of essential electrolytes, particularly sodium, potassium, and chloride.

3-12.4.1 **Causes of Dehydration.** Dehydration can occur through excessive perspiration or long periods of breathing dry gases.

Immersion in water creates a condition resembling a gravity-free state. The weight of the body and the hydrostatic gradient in the circulatory system are almost exactly counterbalanced by the ambient water pressure. This reduces the volume of pooled blood in the leg veins and results in an increase in central blood volume, leading to an increase in urination (immersion diuresis). The increased urine flow leads to increasing loss of water from the body during the dive.

3-12.4.2 **Preventing Dehydration.** Dehydration is felt to increase the incidence of decompression sickness. Prevention is the best medicine. Divers should increase their fluid intake during diving operations to keep themselves well hydrated.

3-12.5 **Hypoglycemia.** Hypoglycemia is an abnormally low blood sugar (glucose) level. It is a condition that is not due to respiratory difficulties, but can complicate or be confused with them. Sugar, derived from food, is the body's main fuel. It is carried to the tissues by the blood and if the blood level falls, tissue function is affected.

3-12.5.1 **Symptoms of Hypoglycemia.** The brain is especially sensitive to lack of glucose. The highly variable symptoms can sometimes closely resemble those of other conditions in which brain function is affected, including carbon dioxide intoxication, hypoxia, carbon monoxide poisoning, oxygen toxicity, and arterial gas embolism. Some of the more common symptoms are unusual hunger, excessive sweating, numbness, chills, headache, trembling, dizziness, confusion, incoordination, anxiety, and fainting. In severe cases, loss of consciousness and convulsions may occur.

3-12.5.2 **Causes of Hypoglycemia.** There are several possible causes of hypoglycemia. Simply missing a meal tends to reduce the blood sugar level, but the body normally can draw on its stored supplies to keep the level close to normal for a long time. A few individuals who are otherwise in good health will develop some degree of hypoglycemia if they do not eat frequently. Severe exercise on an empty stomach will occasionally bring on the symptoms even in a person who ordinarily has no abnormality in this respect. Normally, the body secretes insulin which promotes the use and storage of glucose. People with diabetes do not secrete enough insulin and have an excess of glucose in their blood. They must take insulin by injection to avoid the symptoms of the disease and to keep their blood sugar at a normal level. If they take too much, or if some factor such as unexpectedly hard work reduces the amount needed, serious hypoglycemia can develop rapidly. For this reason, diabetics are considered bad risks in diving.

3-12.5.3 **Preventing Hypoglycemia.** The possibility of hypoglycemia increases during long, drawn out diving operations. Personnel have a tendency to skip meals or eat haphazardly during the operation. For this reason, prevention through proper nutrition is the best medicine. Prior to long, cold, arduous dives, divers should be

encouraged to load up on carbohydrates. For more information, see Naval Medical Research Institute (NMRI) Report 89-94. A diver who often experiences definite weakness (or other symptoms mentioned) when he misses meals, should have a physical examination to determine whether hypoglycemia is the cause and if he is particularly susceptible to it. If hypoglycemia is present, giving sugar by mouth (or if the victim is unconscious, intravenous glucose) relieves the symptoms promptly and proves the diagnosis.

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