CHAPTER 3

Underwater Physiology and Diving Disorders

3-1 INTRODUCTION

3-1.1 Purpose. This chapter provides basic information on the changes in human anatomy and physiology that occur while working in the underwater environment. It also discusses the diving disorders that result when these anatomical or physiological changes exceed the limits of adaptation.

3-1.2 Scope. Anatomy is the study of the structure of the organs of the body. Physiology is the study of the processes and functions of the body. This chapter explains the basic anatomical and physiological changes that occur when diver enters the water and is subject to increased ambient pressure. A diver's knowledge of these changes is as important as his knowledge of diving gear and procedures. When the changes in normal anatomy or physiology exceed the limits of adaptation, one or more pathological states may emerge. These pathological states are called diving disorders and are also discussed in this chapter. Safe diving is only possible when the diver fully understands the fundamental processes 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. Every part of the body is completely interwoven with intricate networks of extremely small blood vessels called capillaries. 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. 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. The two halves have no direct connection 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. 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

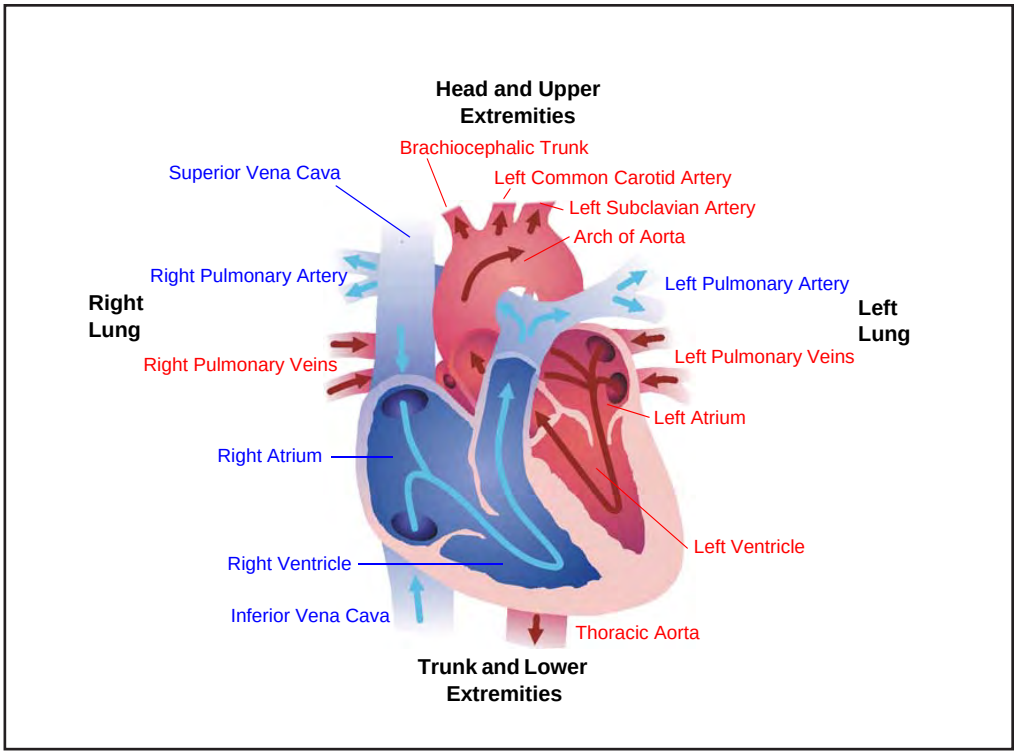

Figure 3-1. The Heart's Components and Blood Flow.

through the main veins into the right atrium and then through the tricuspid valve into the right ventricle.

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. Figure 3-2 shows how the pulmonary circulatory system is arranged.

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.

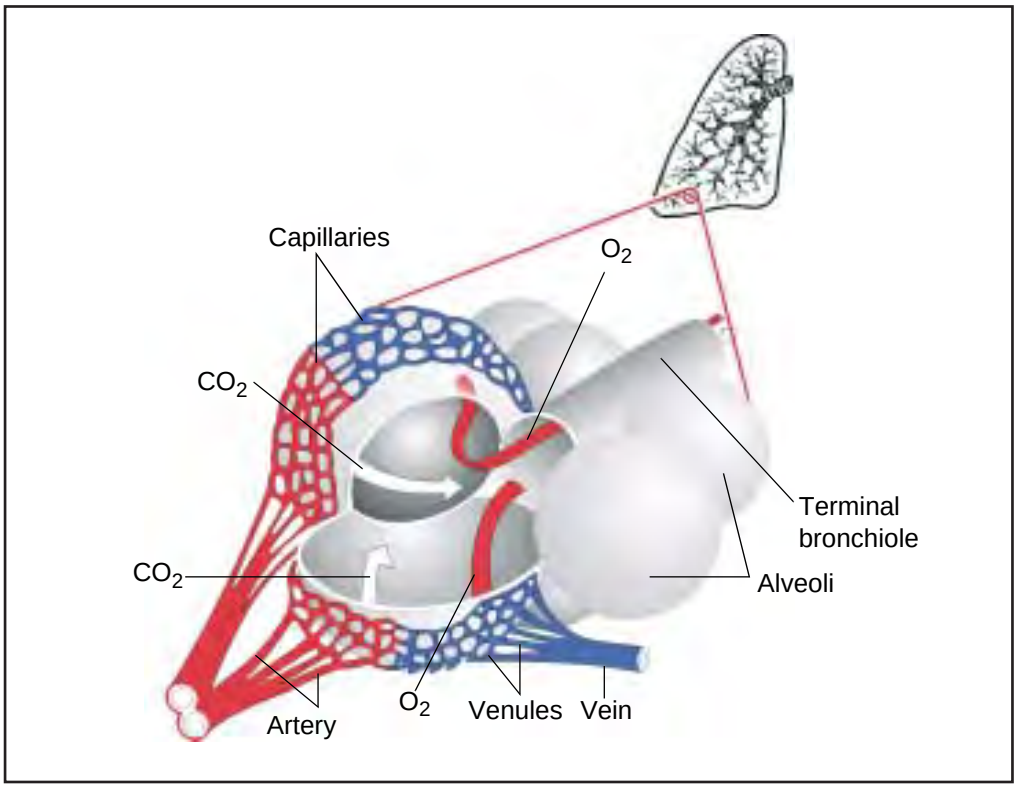

Figure 3-2. Respiration and Blood Circulation. The lung's gas exchange system is essentially three pumps. The thorax, a gas pump, moves air through the trachea and bronchi to the lung's air sacs. These sacs, the alveoli, are shown with and without their covering of pulmonary capillaries. The heart's 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 via the arterial system to the systemic capillaries which deliver oxygen to and collect carbon dioxide from the body's cells.

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 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 processes of breathing and the exchange of gases in the lungs are 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 surface area 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

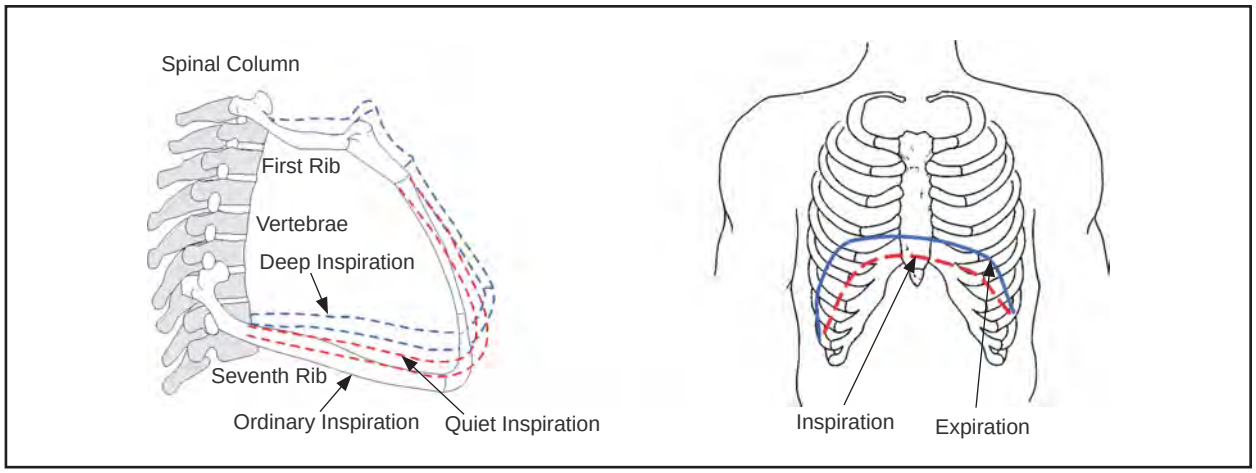

Figure 3-3. Inspiration Process. Inspiration involves both raising the rib cage (left panel) and lowering the diaphragm (right panel). Both movements enlarge the volume of the thoracic cavity and draw air into the lung.

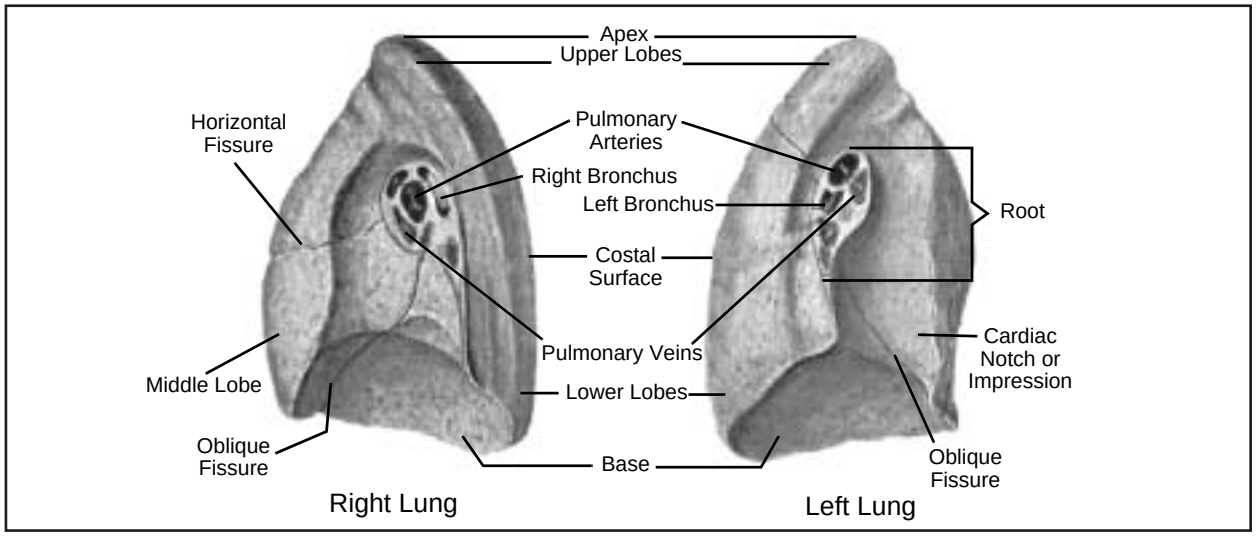

Figure 3-4. Lungs Viewed from Medical Aspect.

twigs of a tree. Air entering the main airways of the lungs gains access to the 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.

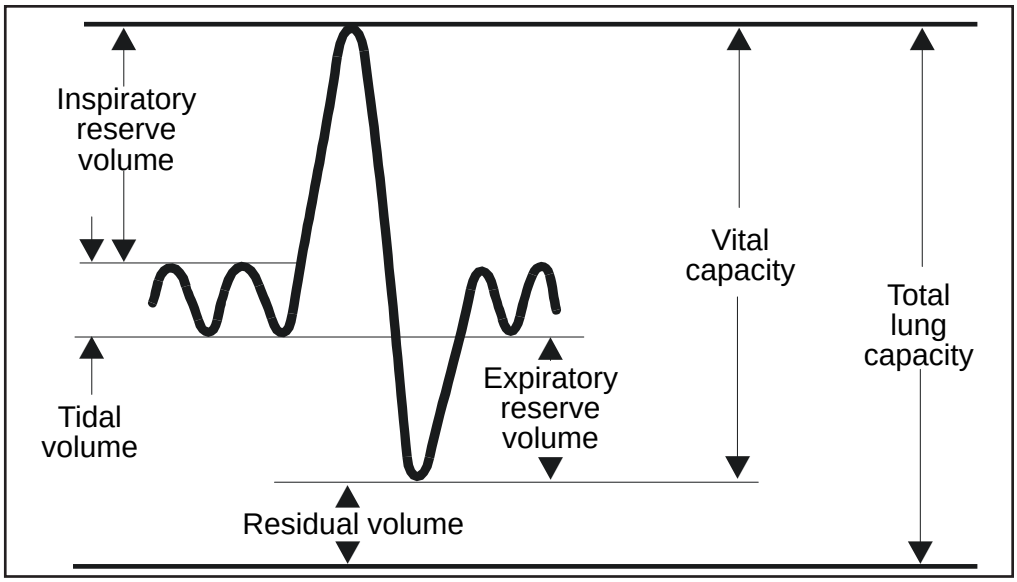

Figure 3-5. Lung Volumes. The heavy line is a tracing, derived from a subject breathing to and from a sealed recording bellows. Following several normal tidal breaths, the subject inhales maximally, then exhales maximally. The volume of air moved during this maximal effort is called the vital capacity. 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 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).

Respiratory Cycle. The *respiratory cycle* is one complete breath consisting of an inspiration and exhalation, including any pause between the movements.

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.

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.

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.

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 may be as high as 3 liters during severe work.

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 respiratory 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.

Maximal Breathing Capacity and Maximum Ventilatory Volume. The *maximum breathing capacity* (MBC) and *maximum voluntary ventilation* (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).

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.

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.

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 are again equalized.

Carbon dioxide diffuses from the blood into the alveolar air, lowering its partial pressure, and oxygen is absorbed by the blood from the alveolar air, increasing its partial 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 a diver inhales is depth dependent. At the surface, a diver swimming at 0.5 knot inhales 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 inhales 20 l/min at BTPS, but the gas is twice as dense; thus, the inhalation 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.

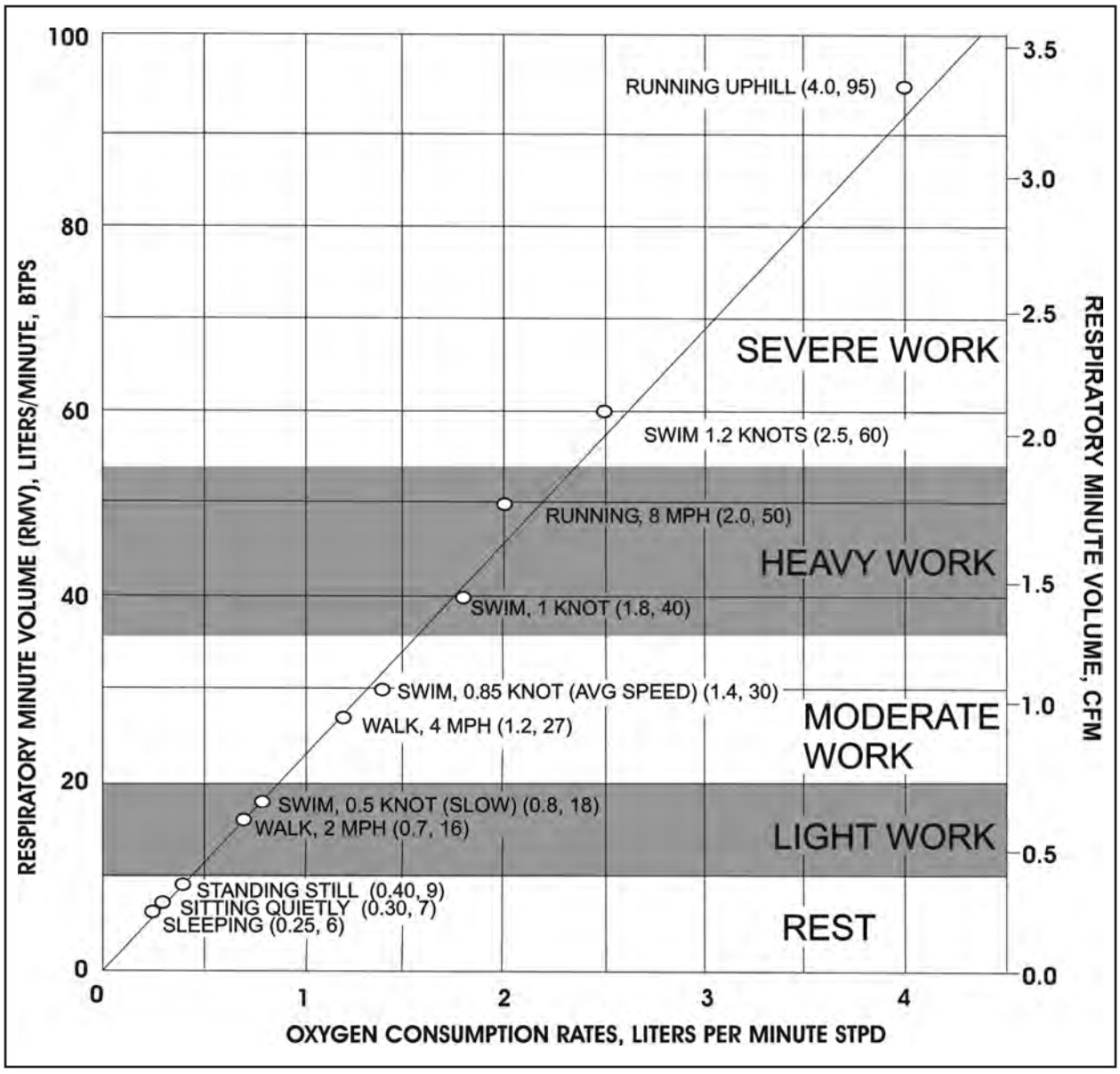

Figure 3-6. Oxygen Consumption and RMV at Different Work Rates.

3-5.1 Oxygen Deficiency (Hypoxia). Hypoxia, is an abnormal deficiency of oxygen in the arterial blood. Severe hypoxia will impede the normal function of cells and eventually kill them. The brain is the most vulnerable organ in the body to the effects of hypoxia.

The partial pressure of oxygen (ppO_2) determines whether the amount of oxygen in a breathing medium is adequate. Air contains approximately 21 percent oxygen and provides an ample ppO_2 of about 0.21 ata at the surface. A drop in ppO_2 below 0.16 ata causes the onset of hypoxic symptoms. Most individuals become hypoxic to the point of helplessness at a ppO_2 of 0.11 ata and unconscious at a ppO_2 of 0.10 ata. Below this level, permanent brain damage and eventually death will occur. In

diving, a lower percentage of oxygen will suffice as long as the total pressure is sufficient to maintain an adequate ppO_2. For example, 5 percent oxygen gives 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:

- Improper line up of breathing gases resulting in a low partial pressure of oxygen in the breathing gas supply.
- Partial or complete blockage of the fresh gas injection orifice in a semiclosed-circuit UBA. Failure of the oxygen addition valve in closed circuit rebreathers like the MK 16.
- Inadequate purging of breathing bags in closed-circuit oxygen rebreathers like the MK 25.
- Blockage of all or part of the air passages by vomitus, secretions, water, or foreign objects.
- Collapse of the lung due to pneumothorax.
- Paralysis of the respiratory muscles from spinal cord injury.
- Accumulation of fluid in the lung tissues (pulmonary edema) due to diving in cold water while overhydrated, negative pressure breathing, inhalation of water in a near drowning episode, or excessive accumulation of venous gas bubbles in the lung during decompression. The latter condition is referred to as “chokes”. Pulmonary edema causes a mismatch of alveolar ventilation and pulmonary blood flow and decreases the rate of transfer of oxygen across the alveolar capillary membrane.
- Carbon monoxide poisoning. Carbon monoxide interferes with the transport of oxygen by the hemoglobin in red blood cells and blocks oxygen utilization at the cellular level.
- Breathholding. During a breathhold the partial pressure of oxygen in the lung falls progressively as the body continues to consume oxygen. If the breathhold is long enough, hypoxia will occur.

3-5.1.2 **Symptoms of Hypoxia.** The symptoms of hypoxia include:

- Loss of judgment
- Lack of concentration
- Lack of muscle control

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

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.

3-5.1.3 **Treatment of Hypoxia.** A diver suffering from severe hypoxia must be rescued promptly. Treat with basic first aid and 100% oxygen. 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. Refer to Volume 4 for information on treatment of hypoxia arising in specific operational environments for dives involving semi-closed and closed-circuit rebreathers.

3-5.1.4 **Prevention of 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 semi-closed circuit rebreathers, a malfunction can cause hypoxia even though the proper gases are being used. Electronically controlled, fully closed-circuit Underwater Breathing Apparatus (UBAs), 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 a UBA malfunction. **To prevent hypoxia, oxygen sensors should be monitored closely throughout the dive. 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 Retention (Hypercapnia).** Hypercapnia is an abnormally high level of carbon dioxide in the blood and 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 an inadequate respiratory minute volume. The principal causes are:

- Excess carbon dioxide levels in compressed air supplies due to improper placement of the compressor inlet.
- Inadequate ventilation of surface-supplied helmets or UBAs.
- Failure of carbon dioxide absorbent canisters to absorb carbon dioxide or incorrect installation of breathing hoses in closed or semi-closed circuit UBAs.
- Inadequate lung ventilation in relation to exercise level. The latter may be caused by skip breathing, increased apparatus dead space, excessive breathing resistance, or increased oxygen partial pressure.

Excessive breathing resistance is an important cause of hypercapnia and arises from two sources: flow resistance and static lung load. Flow resistance results from the flow of dense gas through tubes, hoses, and orifices in the diving equipment and through the diver's own airways. 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 cannot occur. At this point, metabolically produced carbon dioxide is not adequately eliminated and increases in the blood and tissues, causing symptoms of hypercapnia. Symptoms of hypercapnia usually become apparent when divers attempt heavy work at depths deeper then 120 FSW on air or deeper than 850 FSW on helium-oxygen. At very great depths (1,600-2,000 FSW), shortness of breath and other signs of carbon dioxide toxicity may occur even at rest.

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 closed and semi-closed circuit underwater breathing apparatus such as the MK 25 and MK 16. When swimming horizontally with the MK 16, 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. Static lung load is an important contributor to hypercapnia.

Excessive breathing resistance may cause shortness of breath and a sensation of labored breathing (dyspnea) without any increase in blood carbon dioxide level. In this case, the sensation of shortness of breath is due to activation of pressure and stretch receptors in the airways, lungs, and chest wall rather than activation of the chemoreceptors in the brain stem and carotid and aortic bodies. Usually, both types of activation are present when breathing resistance is excessive.

3-5.2.2 **Symptoms of Hypercapnia.** Hypercapnia affects the brain differently than hypoxia does. However, it can result in similar symptoms. Symptoms of hypercapnia include:

- Increased breathing rate
- Shortness of breath, sensation of difficult breathing or suffocation (dyspnea)
- Confusion or feeling of euphoria
- Inability to concentrate
- Increased sweating
- Drowsiness
- Headache
- Loss of consciousness
- Convulsions

The increasing level of carbon dioxide in the blood stimulates the respiratory center to increase the breathing rate and volume. The pulse rate also often increases. On dry land, the increased breathing rate is easily noticed and uncomfortable enough to warn the victim before the rise in $ppCO_2$ becomes dangerous. This is usually not the case in diving. Factors such as water temperature, work rate, increased breathing resistance, and an elevated ppO_2 in the breathing mixture may produce changes in respiratory drive that mask changes caused by excess carbon dioxide. This is especially true in closed-circuit UBAs, particularly 100-percent oxygen rebreathers. In cases where the ppO_2 is above 0.5 ata, the shortness of breath usually associated with excess carbon dioxide may not be prominent 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. A similar situation can occur in cold water. Exposure to cold water often results in an increase in respiratory rate. This increase can make it difficult for the diver to detect an increase in respiratory rate related to a buildup of carbon dioxide.

Injury from hypercapnia is usually due to secondary effects such as drowning or injury caused by decreased mental function or unconsciousness. A diver who loses consciousness because of excess carbon dioxide in his breathing medium and does not inhale water generally revives rapidly when given fresh air and usually feels normal within 15 minutes. The after effects 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. If breathing resistance was high, the diver may note some respiratory muscle soreness post-dive.

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 CNS oxygen toxicity. Excess carbon dioxide during a dive is also believed to increase the likelihood of decompression sickness, but the reasons are less clear.

The effects of nitrogen narcosis and hypercapnia are additive. A diver under the influence of narcosis will probably not notice the warning signs of carbon dioxide intoxication. Hypercapnia in turn will intensify the symptoms of narcosis.

3-5.2.3 **Treatment of Hypercapnia.** Hypercapnia is treated by:

- Decreasing the level of exertion to reduce CO_2 production
- Increasing helmet and lung ventilation to wash out excess CO_2
- Shifting to an alternate breathing source or aborting the dive if defective equipment is the cause.

Because the first sign of hypercapnia may be unconsciousness and it may not be readily apparent whether the cause is hypoxia or hypercapnia. It is important to rule out hypoxia first because of the significant potential for brain damage in hypoxia. Hypercapnia may cause unconsciousness, but by itself will not injure the brain permanently.

3-5.2.4 **Prevention of Hypercapnia.** In surface-supplied diving, hypercapnia is prevented by ensuring that gas supplies do not contain excess carbon dioxide, by maintaining proper manifold pressure during the dive and by ventilating the helmet frequently with fresh gas. For dives deeper than 150 fsw, helium-oxygen mixtures should be used to reduce breathing resistance. In closed or semiclosed-circuit UBAs, hypercapnia is prevented by carefully filling the CO_2 absorbent canister and limiting dive duration to established canister duration limits. For dives deeper than 150 fsw, helium-oxygen mixtures should be used to reduce breathing resistance.

3-5.3 Asphyxia. *Asphyxia* is a condition where breathing stops and both hypoxia and hypercapnia occur simultaneously. Asphyxia will occur when there is no gas to breathe, when the airway is completely obstructed, when the respiratory muscles become paralyzed, or when the respiratory center fails to send out impulses to breathe. Running out of air is a common cause of asphyxia in SCUBA diving. Loss of the gas supply may also be due to equipment failure, for example regulator freeze up. Divers who become unconscious as a result of hypoxia, hypercapnia, or oxygen toxicity may lose the mouthpiece and suffer asphyxia. Obstruction of the airway can be caused by injury to the windpipe, the tongue falling back in the throat during unconsciousness, or the inhalation of water, saliva, vomitus or a foreign body. Paralysis of the respiratory muscles may occur with high cervical spinal cord injury due to trauma or decompression sickness. The respiratory center in the brain stem may become non-functional during a prolonged episode of hypoxia.

3-5.4 Drowning/Near Drowning. Drowning is fluid induced asphyxia. *Near drowning* is the term used when a victim is successfully resuscitated following a drowning episode.

3-5.4.1 Causes of Drowning. A swimmer or diver can fall victim to drowning because of overexertion, panic, inability to cope with rough water, exhaustion, or the effects of cold water or heat loss. Drowning in a hard-hat diving rig is rare. It can happen if the helmet is not properly secured and comes off, or if the diver is trapped in a head-down position with a water leak in the helmet. Normally, as long as the diver is in an upright position and has a supply of air, water can be kept out of the helmet regardless of the condition of the suit. Divers wearing lightweight or SCUBA gear can drown if they lose or ditch their mask or mouthpiece, run out of air, or inhale even small quantities of water. This could be the direct result of failure of the air supply, or panic in a hazardous situation. The SCUBA diver, because of direct exposure to the environment, can be affected by the same conditions that may cause a swimmer to drown.

3-5.4.2 **Symptoms of Near Drowning.**

- Unconsciousness
- Pulmonary edema
- Increased respiratory rate.

3-5.4.3 **Treatment of unconscious drowning victims.**

- In water rescue requires ventilation alone.
  1. Open/Maintain an airway.
  2. Check breathing
  3. Provide 5 rescue breaths if victim not breathing.
  4. DO NOT attempt chest compressions in water.
- The victim should be assumed to be in cardiac arrest if there is no response to rescue breaths.
- Once on a stable platform, the patient should be placed in the supine position
- It is possible that the patient may only need ventilation.

NOTE: It is important that we revert back to the ABC method for drowning, rather than the updated CAB.

A: Airway = Make sure airway is open

B: Breathing = Check for breathing; if victim is not breathing, give 2 rescue breaths (if not already done in water rescue).

C: Circulation = Check circulation by feeling for pulse; if pulse is absent, initiate chest compressions.

- Patient should be placed on 100% O2 and AED placed on chest – although a shockable rhythm is unlikely.
- Be prepared to turn patient on their side and suction their airway – vomiting is common.
- Even if AGE/DCS cannot be ruled out – immediately transport patient to nearest hospital for continued treatment of cardiac/respiratory arrest. The mildest cases of drowning will still require post rescue hospitalization and possibly intensive care.

3-5.4.4 **Prevention of Near Drowning.** Drowning is best prevented by thoroughly training divers in safe diving practices and carefully selecting diving personnel. A trained

diver should not easily fall victim to drowning. However, overconfidence can give a feeling of false security that might lead a diver to take dangerous risks.

3-5.5 **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. The demand to breathe is signaled by the respiratory center responding to the increasing levels of carbon dioxide in the arterial blood and peripheral chemoreceptors responding to the corresponding fall in arterial oxygen partial pressure. If the breathhold is preceded by a period of voluntary hyperventilation, the breathhold can be much longer. Voluntary hyperventilation lowers body stores of carbon dioxide below normal (a condition known as hypocapnia), without significantly increasing oxygen stores. During the breathhold, it takes an appreciable time for the body stores of carbon dioxide to return to the normal level then to rise to the point where breathing is stimulated. During this time the oxygen partial pressure may fall below the level necessary to maintain consciousness. This is a common cause of breathholding accidents in swimming pools. Extended breathholding after hyperventilation is not a safe procedure.

WARNING **Voluntary hyperventilation is dangerous and can lead to unconsciousness and death during breathhold dives.**

Another hazard of breathhold diving is the possible loss of consciousness from hypoxia during ascent. Air in the lungs is compressed during descent, 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 0.10 ata (10% sev), unconsciousness may result. This danger is further heightened when hyperventilation has eliminated normal body warning signs of carbon dioxide accumulation and allowed the diver to remain on the bottom for a longer period of time. Refer to Chapter 6 for breathhold diving restrictions.

3-5.6 **Involuntary Hyperventilation.** Hyperventilation is the term applied to breathing more than is necessary to keep the body's carbon dioxide tensions at proper level. Hyperventilation may be voluntary (for example, to increase breathholding time) or involuntary. In involuntary hyperventilation, the diver is either unaware that he is breathing excessively, or is unable to control his breathing.

3-5.6.1 **Causes of Involuntary Hyperventilation.** Involuntary hyperventilation can be triggered by fear experienced during stressful situations. It can also be initiated by the slight "smothering sensation" that accompanies an increase in equipment dead space, an increase in static lung loading, or an increase in 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.

3-5.6.2 **Symptoms of Involuntary Hyperventilation.** Hyperventilation may lead to a biochemical imbalance that gives rise to dizziness, tingling of the extremities, and

spasm of the small muscles of the hands and feet. Hyperventilating over a long period, produces additional symptoms such as weakness, headaches, numbness, faintness, and blurring of vision. The diver may experience a sensation of "air hunger" even though his ventilation is more than enough to eliminate carbon dioxide. All these symptoms can be easily confused with symptoms of CNS oxygen toxicity.

3-5.6.3 **Treatment of Involuntary Hyperventilation.** Hyperventilation victims should be encouraged to relax and slow their breathing rates. The body will correct hyperventilation naturally.

3-5.7 Overbreathing the Rig. "Overbreathing the Rig" is a special term divers apply to an episode of acute hypercapnia that develops when a diver works at a level greater than his UBA can support. When a diver starts work, or abruptly increases his workload, the increase in respiratory minute ventilation lags the increase in oxygen consumption and carbon dioxide production by several minutes. When the RMV demand for that workload finally catches up, the UBA may not be able to supply the gas necessary despite extreme respiratory efforts on the part of the diver. Acute hypercapnia with marked respiratory distress ensues. Even if the diver stops work to lower the production of carbon dioxide, the sensation of shortness of breath may persist or even increase for a short period of time. When this occurs, the inexperienced diver may panic and begin to hyperventilate. The situation can rapidly develop into a malicious cycle of severe shortness of breath and uncontrollable hyperventilation. In this situation, if even a small amount of water is inhaled, it can cause a spasm of the muscles of the larynx (voice box), called a laryngospasm, followed by asphyxia and possible drowning.

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 and pace their work accordingly. Always increase workloads gradually to insure that the UBA can match the demand for increased lung ventilation. If excessive breathing resistance is encountered, slow or stop the pace of work until a respiratory comfort level is achieved. If respiratory distress occurs following an abrupt increase in workload, stop work and take even controlled breaths until the sensation of respiratory distress subsides. If the situation does not improve, abort the dive.

3-5.8 Carbon Monoxide Poisoning. The body produces carbon monoxide as a part of the process of normal metabolism. Consequently, there is always a small amount of carbon monoxide present in the blood and tissues. Carbon monoxide poisoning occurs when levels of carbon monoxide in the blood and tissues rise above these normal values due to the presence of carbon monoxide in the diver's gas supply. Carbon monoxide not only blocks hemoglobin's ability to delivery oxygen to the cells, causing cellular hypoxia, but also poisons cellular metabolism directly.

3-5.8.1 **Causes of Carbon Monoxide Poisoning.** Carbon monoxide is not found in any significant quantity in fresh air. Carbon monoxide poisoning is usually caused by

a compressor's intake being too close to the exhaust of an internal combustion engine or malfunction of a oil lubricated compressor. Concentrations as low as 0.002 ata (2,000 ppm, or 0.2%) can prove fatal.

3-5.8.2 **Symptoms of Carbon Monoxide Poisoning.** The symptoms of carbon monoxide poisoning are almost identical to those of hypoxia. When toxicity develops gradually the symptoms are:

- Headache
- Dizziness
- Confusion
- Nausea
- Vomiting
- Tightness across the forehead

When carbon monoxide concentrations are high enough to cause rapid onset of poisoning, the victim may not be aware of any symptoms before he becomes unconscious.

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.

3-5.8.3 **Treatment of 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, shall 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. Divers with severe symptoms (i.e. severe headache, mental status changes, any neurological symptoms, rapid heart rate) should be treated using Treatment Table 6.

3-5.8.4 **Prevention of Carbon Monoxide Poisoning.** Locating compressor intakes away from engine exhausts and maintaining air compressors in the best possible mechanical condition can prevent carbon monoxide poisoning. When carbon monoxide poisoning is suspected, isolate the suspect breathing gas source, and forward gas samples for analysis as soon as possible.

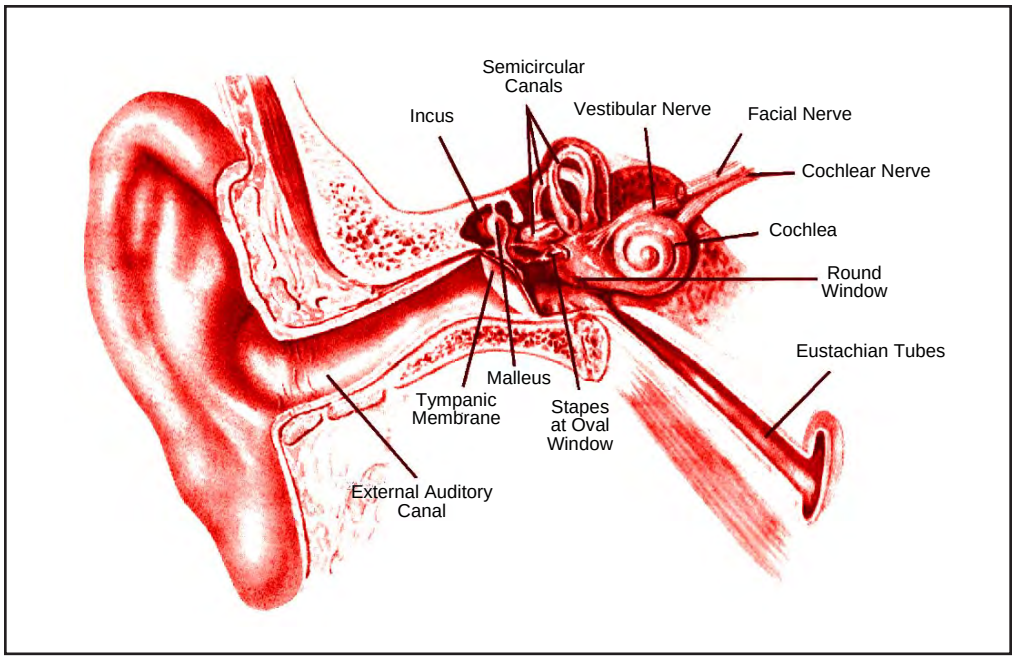

Figure 3-7. Gross Anatomy of the Ear in Frontal Section.

3-6 MECHANICAL EFFECTS OF PRESSURE ON THE HUMAN BODY-BAROTRAUMA DURING DESCENT

Barotrauma, or damage to body tissues from the mechanical effects of pressure, results when pressure differentials between body cavities and the hydrostatic pressure surrounding the body, or between the body and the diving equipment, are not equalized properly. Barotrauma most frequently occurs during descent, but may also occur during ascent. Barotrauma on descent is called squeeze. Barotrauma on ascent is called reverse squeeze.

3-6.1 Prerequisites for Squeeze.

For squeeze to occur during descent the following five conditions must be met:

- 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 gas-filled space must have rigid walls. If the walls are collapsible like a balloon, no damage will be done by compression.
- The gas-filled space must be enclosed. If gas or liquid can freely enter the space as the gas volume changes, no damage will occur.
- The space must have lining membrane with an arterial blood supply and venous drainage that penetrates the space from the outside. This allows blood to be forced into the space to compensate for the change in pressure.

- There must be a change in ambient pressure.

3-6.2 **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 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. When rupture occurs, this pain will diminish rapidly. 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. If eardrum rupture occurs, the dive shall be aborted. 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 disturbance 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. Suspected cases of eardrum rupture shall be referred to medical personnel.

3-6.2.1 **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. Medical personnel shall examine divers who have

trouble clearing their ears 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. A forceful Valsalva maneuver can result in alternobaric vertigo or barotrauma to the inner ear (see below).**

WARNING **If decongestants must be used, check with medical personnel trained in diving medicine to obtain medication that will not cause drowsiness and possibly add to symptoms caused by the narcotic effect of nitrogen.**

3-6.2.2 **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, the diver may have a bloody nose, the result of blood being forced out of the middle ear space and into the nasal cavity through the eustachian tube by expanding air in the middle ear. The diver shall report symptoms of middle ear squeeze to the diving supervisor and seek medical attention. Treatment consists of taking decongestants, pain medication if needed, and cessation of diving until the damage is healed. If the eardrum has ruptured antibiotics may be prescribed as well. Never administer medications directly into the external ear canal if a ruptured eardrum is suspected or confirmed unless done in direct consultation with an ear, nose, and throat (ENT) medical specialist.

3-6.3 **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.

3-6.3.1 **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

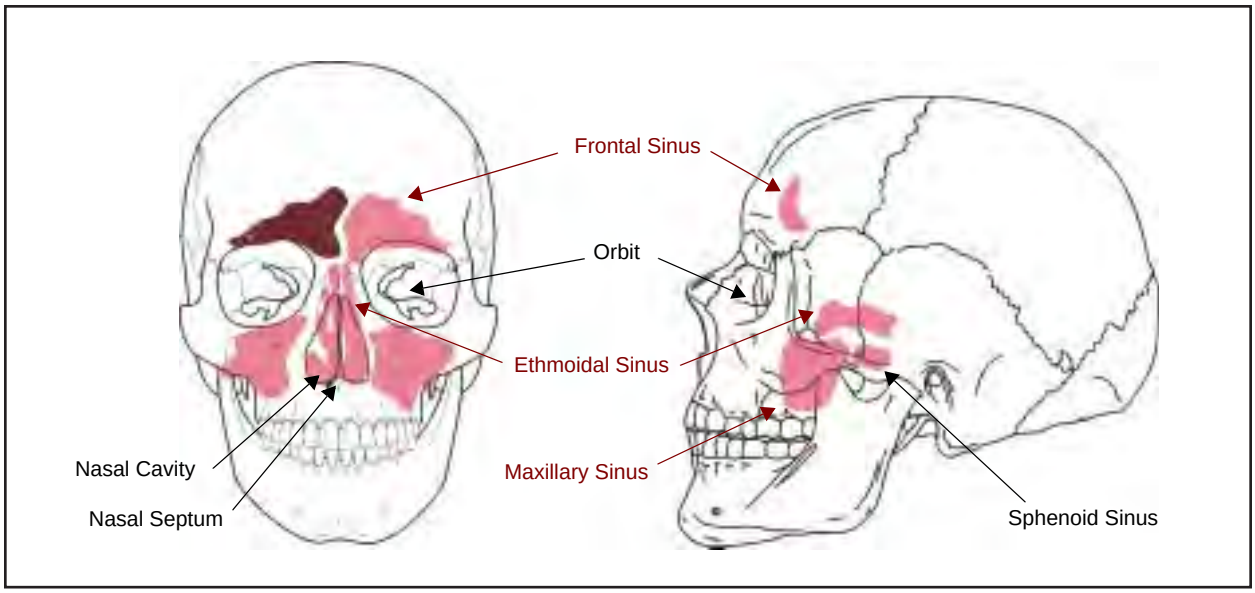

Figure 3-8. Location of the Sinuses in the Human Skull.

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. 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.

3-6.3.2 **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.

3-6.4 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.

3-6.5 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.

3-6.6 **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.

3-6.7 **Face or Body Squeeze.** SCUBA face masks, goggles, and certain types of exposure suits may cause squeeze under some conditions. Exhaling through the nose can usually equalize the pressure in a face mask, 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.

3-6.8 **Inner Ear Barotrauma.** The inner ear contains no gas and therefore cannot be "squeezed" in the same sense that the middle ear and sinuses can. However, the inner ear is located next to the middle ear cavity and is affected by the same conditions that lead to middle ear squeeze. To understand how the inner ear could be damaged as a result of pressure imbalances in the middle ear, it is first necessary to understand the anatomy of the middle and inner ear.

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

There are three bones in the middle ear: the malleus, the incus, and the stapes. They are also commonly referred to as the hammer, anvil, and stirrup, respectively (Figure 3-9). The malleus is connected to the eardrum (tympanic membrane) and

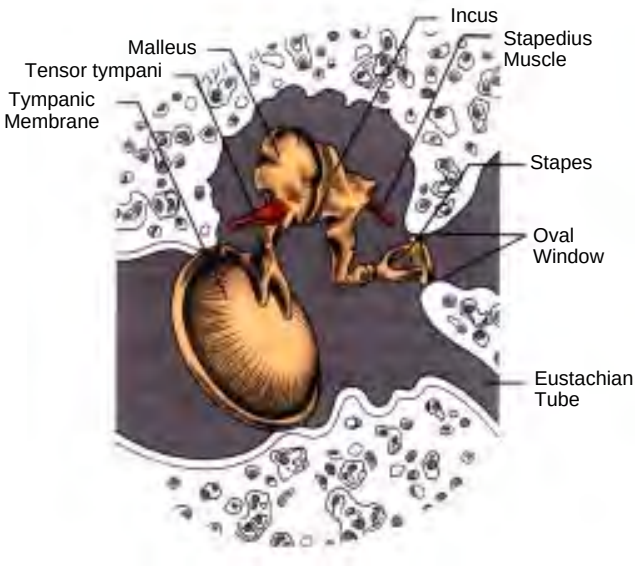

Figure 3-9. Components of the Middle Ear.

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. When the stapes drives the oval window inward, the round window bulges outward to compensate. The fluid-filled spaces of the inner ear are also connected to the fluid spaces surrounding the brain by a narrow passage called the cochlear aqueduct. The cochlear aqueduct can transmit increases in cerebrospinal fluid pressure to the inner ear. When Valsalva maneuvers are performed to equalize middle ear and sinus pressure, cerebrospinal fluid pressure increases.

If middle ear pressure is not equalized during descent, the inward bulge of the eardrum is transmitted to the oval window by the middle ear bones. The stapes pushes the oval window inward. Because the inner ear fluids are incompressible, the round window correspondingly bulges outward into the middle ear space. If this condition continues, the round window may rupture spilling inner ear fluids into the middle ear and leading to a condition know as *inner ear barotrauma with perilymph fistula*. Fistula is a medical term for a hole in a membrane; the fluid in the inner ear is called perilymph. Rupture of the oval or round windows may also occur when middle ear pressures are suddenly and forcibly equalized. When equalization is sudden and forceful, the eardrum moves rapidly from a position of bulging inward maximally to bulging outward maximally. The positions of the oval and round windows are suddenly reversed. Inner ear pressure is also increased by transmission of the Valsalva-induced increase in cerebrospinal fluid pressure. This puts additional stresses on these two membranes. Either the round or oval window may rupture. Rupture of the round window is by far the most common. The oval

window is a tougher membrane and is protected by the footplate of the stapes. Even if rupture of the round or oval window does not occur, the pressure waves induced in the inner ear during these window movements may lead to disruption of the delicate cells involved in hearing and balance. This condition is referred to *inner ear barotrauma without perilymph fistula*.

The primary symptoms of inner ear barotrauma are persistent vertigo and hearing loss. Vertigo is the false sensation of motion. The diver feels that he is moving with respect to his environment or that the environment is moving with respect to him, when in fact no motion is taking place. The vertigo of inner ear barotrauma is generally described as whirling, spinning, rotating, tilting, rocking, or undulating. This sensation is quite distinct from the more vague complaints of dizziness or lightheadedness caused by other conditions. The vertigo of inner ear barotrauma is often accompanied by symptoms that may or may not be noticed depending on the severity of the insult. These include nausea, vomiting, loss of balance, incoordination, and a rapid jerking movement of the eyes, called nystagmus. Vertigo may be accentuated when the head is placed in certain positions. The hearing loss of inner ear barotrauma may fluctuate in intensity and sounds may be distorted. Hearing loss is accompanied by ringing or roaring in the affected ear. The diver may also complain of a sensation of bubbling in the affected ear.

Symptoms of inner ear barotrauma usually appear abruptly during descent, often as the diver arrives on the bottom and performs his last equalization maneuver. However, the damage done by descent may not become apparent until the dive is over. A common scenario is for the diver to rupture a damaged round window while lifting heavy weights or having a bowel movement post dive. Both these activities increase cerebrospinal fluid pressure and this pressure increase is transmitted to the inner ear. The round window membrane, weakened by the trauma suffered during descent, bulges into the middle ear space under the influence of the increased cerebrospinal fluid pressure and ruptures.

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 with head elevation to exploratory surgery, depending on the severity of the symptoms and whether a perilymph fistula is suspected. Any hearing loss or vertigo occurring within 72 hours of a hyperbaric exposure should be evaluated as a possible case of inner ear barotrauma.

When either hearing loss or vertigo develop after the diver has surfaced, it may be impossible to tell whether the symptoms are caused by inner ear barotrauma, decompression sickness or arterial gas embolism. For the latter two conditions, recompression treatment is mandatory. Although it might be expected that recompression treatment would further damage to the inner ear in a case of barotrauma and should be avoided, experience has shown that recompression is generally not harmful provided a few simple precautions are followed. The diver should be placed in a head up position and compressed slowly to allow adequate time for middle ear equalization. Clearing maneuvers should be gentle. The diver should not be exposed to excessive positive or negative pressure when breathing

oxygen on the built-in breathing system (BIBS) mask. Recompress the diver if there is doubt about the cause of post-dive hearing loss or vertigo.

CAUTION When in doubt, always recompress.

Frequent oscillations in middle ear pressure associated with difficult clearing may lead to a transient vertigo. This condition is called *alternobaric vertigo of descent*. Vertigo usually follows a Valsalva maneuver, often with the final clearing episode just as the diver reaches the bottom. Symptoms typically last less than a minute but can cause significant disorientation during that period. Descent should be halted until the vertigo resolves. Once the vertigo resolves, the dive may be continued. Alternobaric vertigo is a mild form of inner ear barotrauma in which no lasting damage to the inner ear occurs.

3-7 MECHANICAL EFFECTS OF PRESSURE ON THE HUMAN BODY--BAROTRAUMA DURING ASCENT

During ascent gases expand according to Boyle's Law. If the excess gas is not vented from enclosed spaces, damage to those spaces may result.

3-7.1 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. If rupture occurs, the middle ear will equalize pressure with the surrounding water and the pain will disappear. However, there may be a transient episode of intense vertigo as cold water enters the middle ear space.

The increased pressure in the middle ear may also affect the inner ear balance mechanism, leading to a condition called *alternobaric vertigo of ascent*. Alternobaric vertigo occurs when the middle ear space on one side is overpressurized while the other side is equalizing normally. The onset of vertigo is usually sudden and may be preceded by pain in the ear that is not venting excess pressure. Alternobaric vertigo usually lasts for only a few minutes, but may be incapacitating during that time. Relief is usually abrupt and may be accompanied by a hissing sound in the affected ear as it equalizes. Alternobaric vertigo during ascent will disappear immediately if the diver halts his ascent and descends a few feet.

Increased pressure in the middle ear can also produce paralysis of the facial muscles, a condition known as *facial baroparesis*. In some individuals, the facial nerve is exposed to middle ear pressure as it traverses the temporal bone. If the middle ear fails to vent during ascent, the overpressure can shut off the blood supply to the nerve causing it to stop transmitting neural impulses to the facial muscles on the affected side. Generally, a 10 to 30 min period of overpressure is necessary for symptoms to occur. Full function of the facial muscles returns 5-10 min after the overpressure is relieved.

Increased pressure in the middle ear can also cause structural damage to the inner ear, a condition known as *inner ear barotrauma of ascent*. The bulging ear drum pulls the oval window outward into the middle ear space through the action of the middle ear bones. The round window correspondingly bulges inward. This inward deflection can be enhanced if the diver further increases middle ear pressure by performing a Valsalva maneuver. The round window may rupture causing inner ear fluids to spill into the middle ear space. The symptoms of marked hearing loss and sustained vertigo are identical to the symptoms experienced with inner ear barotrauma during descent.

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 or vertigo 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. If symptoms of sustained hearing loss or vertigo appear during ascent, or shortly after ascent, it may be impossible to tell whether the symptoms are arising from inner ear barotrauma or from decompression sickness or arterial gas embolism. Recompression therapy is indicated unless there is high confidence that the condition is inner ear barotrauma.

3-7.2 **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 titrate himself slowly to the surface in a series of ascents and descents just as with a reverse middle ear squeeze.

When overpressure occurs in the maxillary sinus, the blood supply to the infraorbital nerve may be reduced, leading to numbness of the lower eyelid, upper lip, side of the nose, and cheek on the affected side. This numbness will resolve spontaneously when the sinus overpressure is relieved.

3-7.3 **Gastrointestinal Distention.** Divers may occasionally experience abdominal pain during ascent because of gas expansion in the stomach or intestines. This condition is caused by gas being generated in the intestines during a dive, or by swallowing air (aerophagia). These pockets of gas will usually work their way out of the system through the mouth or anus. If not, distention will occur.

If the pain begins to pass the stage of mild discomfort, ascent should be halted and the diver should descend slightly to relieve the pain. The diver should then attempt to gently burp or release the gas anally. Overzealous attempts to belch should be

avoided as they may result in swallowing more air. Abdominal pain following fast ascents shall be evaluated by a Diving Medical Officer.

To avoid intestinal gas expansion:

- Do not dive with an upset stomach or bowel.
- Avoid eating foods that are likely to produce intestinal gas.
- Avoid a steep, head-down angle during descent to minimize the amount of air swallowed.

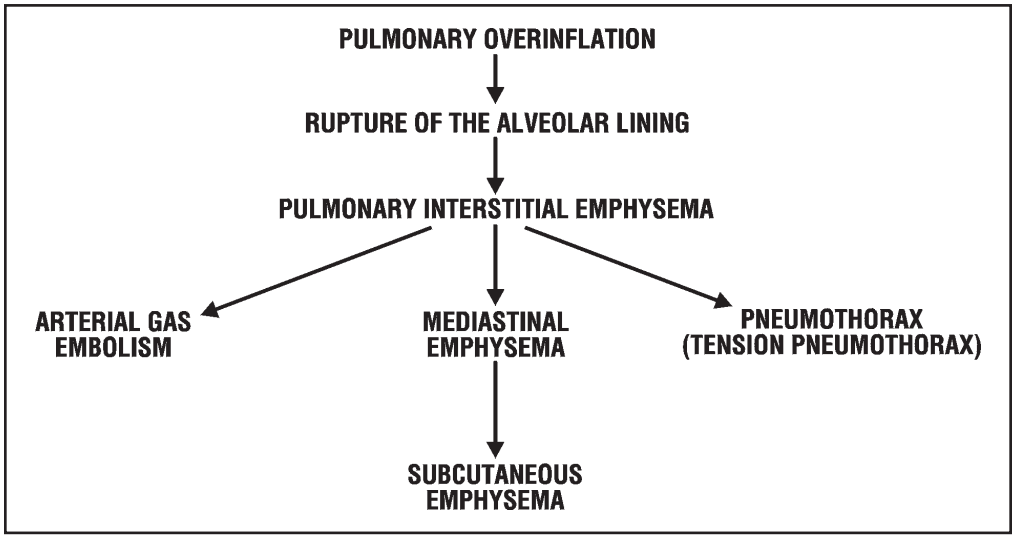

Figure 3-10. Pulmonary Overinflation Syndromes (POIS). Leaking of gas into the pulmonary interstitial tissue causes no symptoms unless further leaking occurs. If gas enters the arterial circulation, potentially fatal arterial gas embolism may occur. Pneumothorax occurs if gas accumulates between the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

3-8 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

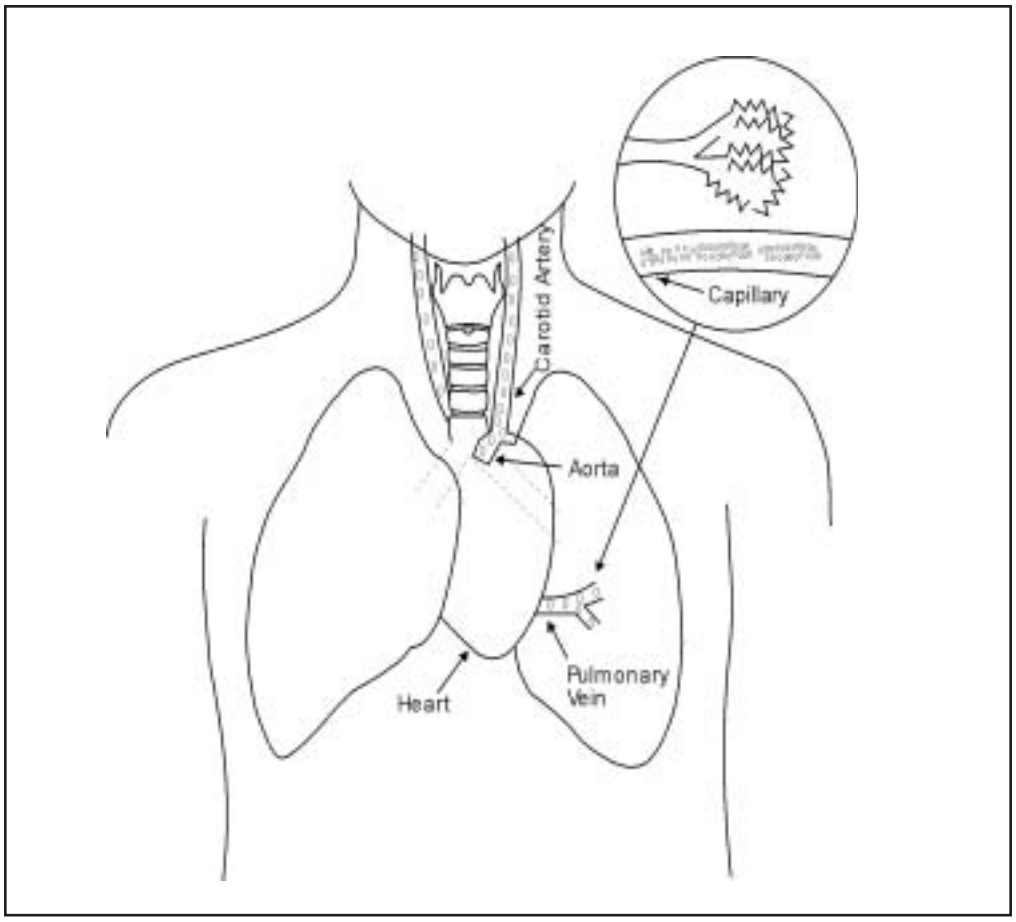

Figure 3-11. Arterial Gas Embolism.

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.

3-8.1 **Arterial Gas Embolism (AGE).** Arterial gas embolism (AGE), sometimes simply called gas embolism, is an obstruction of blood flow caused by gas bubbles (emboli) entering the arterial circulation. Obstruction of the arteries of the brain and heart can lead to death if not promptly relieved (see Figure 3-11).

3-8.1.1 **Causes of AGE.** AGE is caused by the expansion of gas taken into the lungs while breathing under pressure and held in the lungs during ascent. The gas might have been retained in the lungs by choice (voluntary breathholding) or by accident (blocked air passages), or by over pressurization of breathing gas. The gas could have become trapped in an obstructed portion of the lung that has been damaged from some previous disease or accident; or the diver, reacting with panic to a difficult situation, may breathhold without realizing it. If there is enough gas and if it expands sufficiently, the pressure will force gas through the alveolar walls into surrounding tissues and into the bloodstream. If the gas enters the arterial circulation, it will be dispersed to all organs of the body. The organs that are especially susceptible to arterial gas embolism and that are responsible for the life-threatening symptoms are the central nervous system (CNS) and the heart. In all cases of arterial gas embolism, associated pneumothorax is possible and should not be overlooked. Exhaustion of air supply and the need for an emergency ascent is the most common cause of AGE.

3-8.1.2 **Symptoms of AGE**

- Unconsciousness
- Paralysis
- Numbness
- Weakness
- Extreme fatigue
- Large areas of abnormal sensations (Paresthesias)
- Difficulty in thinking
- Vertigo
- Convulsions
- Vision abnormalities
- Loss of coordination
- Nausea and or vomiting
- Hearing abnormalities
- Sensation similar to that of a blow to the chest during ascent
- Bloody sputum
- Dizziness

- Personality changes
- Loss of control of bodily functions
- Tremors

Symptoms of subcutaneous/medistinal emphysema, pneumothorax and/or pneumopericardium may also be present (see below). In all cases of arterial gas embolism, the possible presence of these associated conditions should not be overlooked.

3-8.1.3 **Treatment of AGE.**

- Basic first aid (ABC)
- 100 percent oxygen
- Immediate recompression
- See Volume 5 for more specific information regarding treatment.

3-8.1.4 **Prevention of AGE.** The risk of arterial gas embolism can be substantially reduced or eliminated by paying careful attention to the following:

- Every diver must receive intensive training in diving physics and physiology, as well as instruction in the correct use of diving equipment. Particular attention must be given to the training of SCUBA divers, because SCUBA operations produce a comparatively high incidence of embolism accidents.
- A diver must never interrupt breathing during ascent from a dive in which compressed gas has been breathed.
- A diver must exhale continuously while making an emergency ascent. The rate of exhalation must match the rate of ascent. For a free ascent, where the diver uses natural buoyancy to be carried toward the surface, the rate of exhalation must be great enough to prevent embolism, but not so great that positive buoyancy is lost. In a uncontrolled or buoyant ascent, where a life preserver, dry suit or buoyancy compensator assists the diver, the rate of ascent may far exceed that of a free ascent. The exhalation must begin before the ascent and must be a strong, steady, and forceful. It is difficult for an untrained diver to execute an emergency ascent properly. It is also often dangerous to train a diver in the proper technique.
- The diver must not hesitate to report any illness, especially respiratory illness such as a cold, to the Diving Supervisor or Diving Medical Personnel prior to diving.

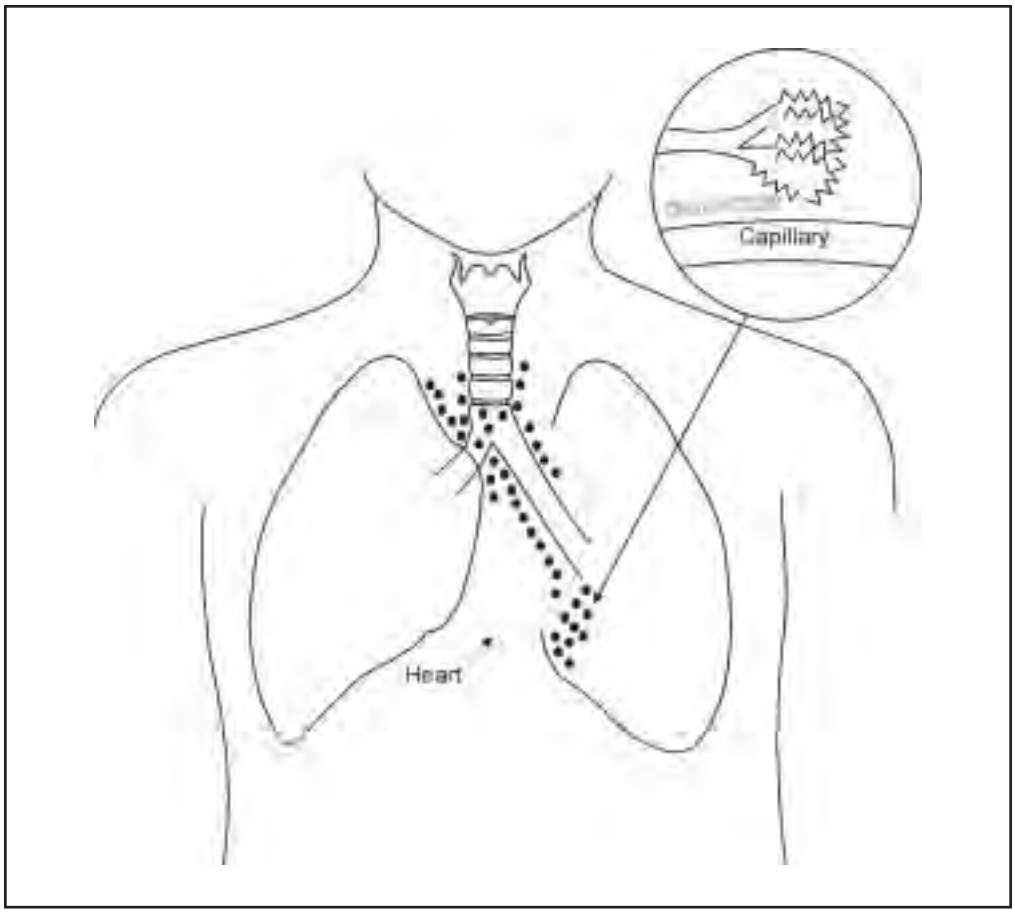

Figure 3-12. Mediastinal Emphysema.

3-8.2 **Mediastinal and Subcutaneous Emphysema.** Mediastinal emphysema, also called pneumomediastinum, occurs when gas is forced through torn lung tissue into the loose mediastinal tissues in the middle of the chest surrounding the heart, the trachea, and the major blood vessels (see Figure 3-12). Subcutaneous emphysema occurs when that gas subsequently migrates into the subcutaneous tissues of the neck (Figure 3-13). Mediastinal emphysema is a pre-requisite for subcutaneous emphysema.

3-8.2.1 **Causes of Mediastinal & Subcutaneous Emphysema.** Mediastinal/subcutaneous emphysema is caused by over inflation of the whole lung or parts of the lung due to:

- Breath holding during ascent
- Positive pressure breathing such as ditch and don exercises
- Drown proofing exercises
- Cough during surface swimming

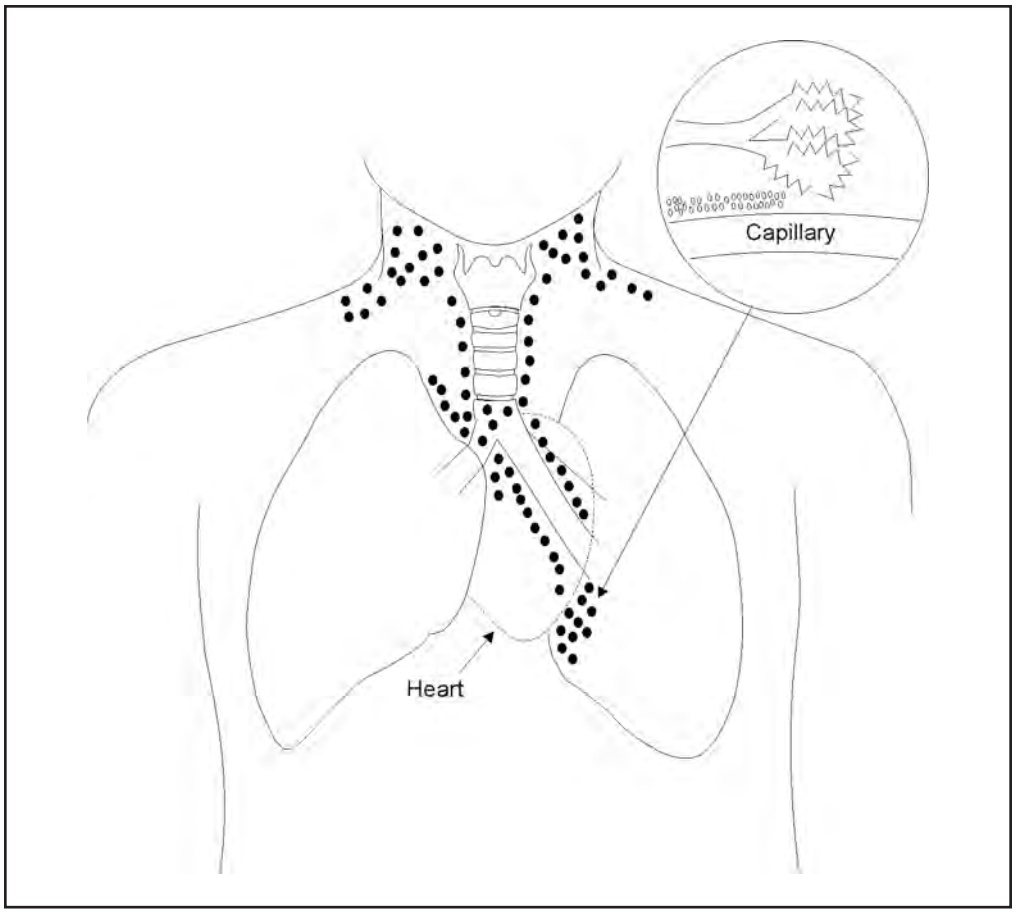

Figure 3-13. Subcutaneous Emphysema.

3-8.2.2 **Symptoms of Mediastinal & Subcutaneous Emphysema.** Mild cases are often unnoticed by the diver. In more severe cases, the diver may experience mild to moderate pain under the breastbone, often described as dull ache or feeling of tightness. The pain may radiate to the shoulder or back and may increase upon deep inspiration, coughing, or swallowing. The diver may have a feeling of fullness around the neck and may have difficulty in swallowing. His voice may change in pitch. An observer may note a swelling or apparent inflation of the diver's neck. Movement of the skin near the windpipe or about the collar bone may produce a cracking or crunching sound (crepitation).

3-8.2.3 **Treatment of Mediastinal & Subcutaneous Emphysema.** Suspicion of mediastinal or subcutaneous emphysema warrants prompt referral to medical personnel to rule out the coexistence of arterial gas embolism or pneumothorax. The latter two conditions require more aggressive treatment. Treatment of mediastinal or subcutaneous emphysema with mild symptoms consists of breathing 100 percent oxygen at the surface. If symptoms are severe, shallow recompression may be beneficial. Recompression should only be carried out upon the recommendation of a Diving Medical Officer who has ruled out the occurrence of pneumothorax. Recompression is performed with the diver breathing 100 percent oxygen and using the shallowest depth of relief (usually 5 or 10 feet). An hour of breathing oxygen should

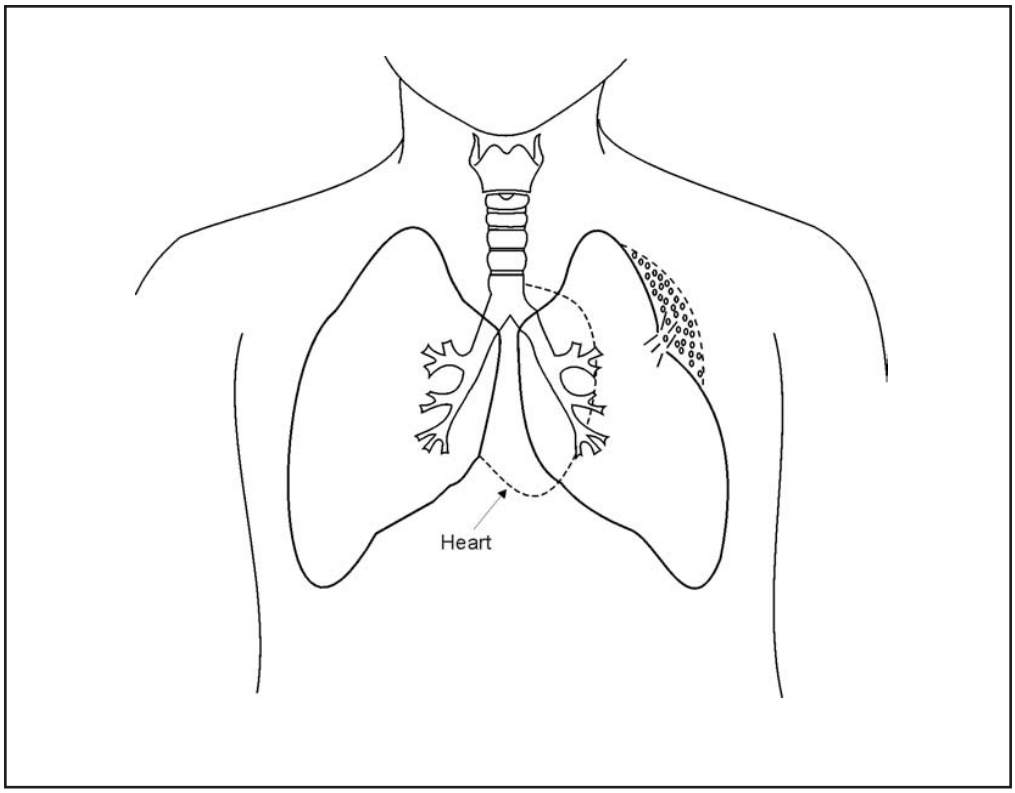

Figure 3-14. Pneumothorax.

be sufficient for resolution, but longer stays may be necessary. Decompression will be dictated by the tender's decompression obligation. The appropriate air table should be used, but the ascent rate should not exceed 1 foot per minute. In this specific case, the delay in ascent should be included in bottom time when choosing the proper decompression table.

3-8.2.4 **Prevention of Mediastinal & Subcutaneous Emphysema.** The strategies for preventing mediastinal/subcutaneous emphysema are identical to the strategies for preventing arterial gas embolism. Breathe normally during ascent. If emergency ascent is required, exhale continuously. Mediastinal/subcutaneous emphysema is particularly common after ditch and don exercises. Avoid positive pressure breathing situations during such exercises. The mediastinal/subcutaneous emphysema that is seen during drown proofing exercises and during surface swimming unfortunately is largely unavoidable.

3-8.3 Pneumothorax. A pneumothorax is air trapped in the pleural space between the lung and the chest wall (Figure 3-14).

3-8.3.1 **Causes of Pneumothorax.** A pneumothorax occurs when the lung surface ruptures and air spills into the space between the lung and chest wall. Lung rupture can result from a severe blow to the chest or from overpressurization of the lung. 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

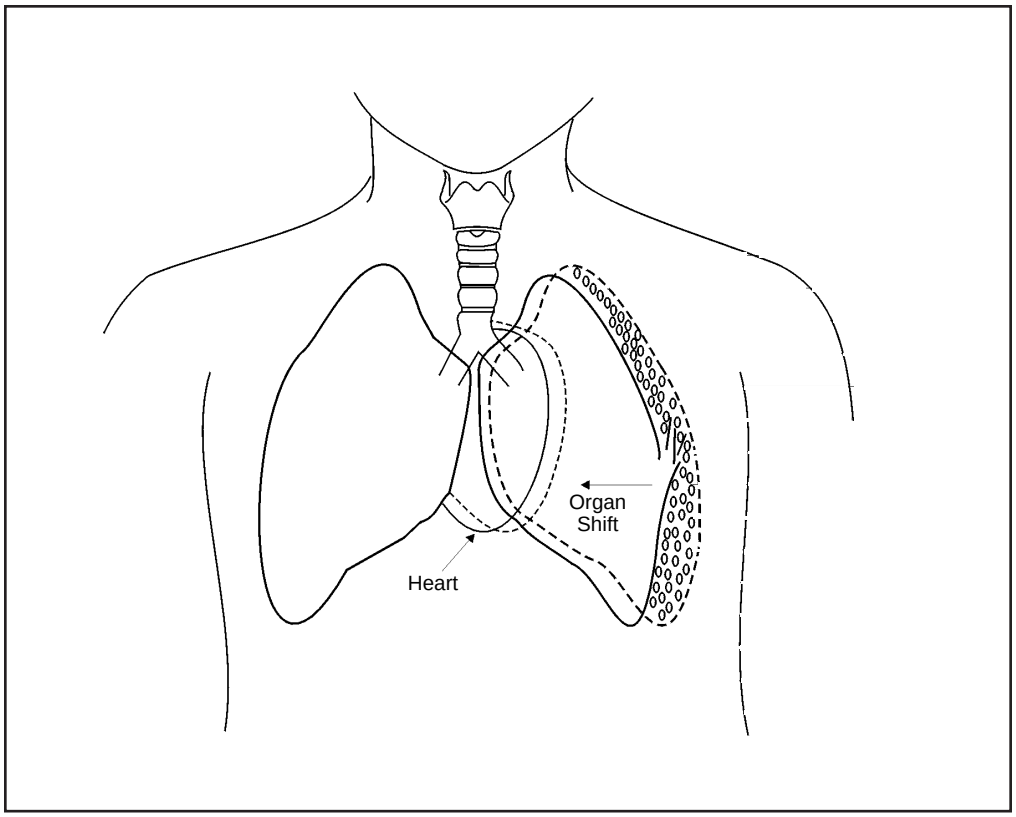

Figure 3-15. Tension Pneumothorax.

of respiratory distress. This condition normally improves with time as the air is reabsorbed. In severe cases of collapse, the air must be removed with the aid of a tube or catheter.

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) because of 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.

A simple pneumothorax that occurs while the diver is at depth can be converted to a tension pneumothorax by expansion of the gas pocket during ascent. Although a ball valve like mechanism that allows air to enter the pleural cavity but not escape is not present, the result is the same. The mounting tension collapses the lung on the affected side and pushes the heart and lung to the opposite side of the chest.

3-8.3.2 **Symptoms of Pneumothorax.** The onset of a simple pneumothorax is accompanied by a sudden, sharp chest pain, followed by shortness of breath, labored breathing, rapid heart rate, a weak pulse, and anxiety. The normal chest movements associated with respiration may be reduced on the affected side and breath sounds may be difficult to hear with a stethoscope.

The symptoms of tension pneumothorax are similar to simple pneumothorax, but become progressively more intense over time. As the heart and lungs are displaced to the opposite side of the chest, blood pressure falls along with the arterial oxygen partial pressure. Cyanosis (a bluish discoloration) of the skin appears. If left untreated, shock and death will ensue. Tension pneumothorax is a true medical emergency.

3-8.3.3 **Treatment of Pneumothorax.** A diver believed to be suffering from pneumothorax must be thoroughly examined for the possible co-existence of arterial gas embolism. This is covered more fully in Volume 5.

A small pneumothorax (less than 15%) normally will improve with time as the air in the pleural space is reabsorbed spontaneously. A larger pneumothorax may require active treatment. Mild pneumothorax can be treated by breathing 100 percent oxygen. Cases of pneumothorax that demonstrate cardio-respiratory compromise may require the insertion of a chest tube, largebore intravenous (IV) catheter, or other device designed to remove intrathoracic gas (gas around the lung). Only personnel trained in the use of these and the other accessory devices (one-way valves, underwater suction, etc.) necessary to safety decompress the thoracic cavity should insert them. Divers recompressed for treatment of arterial gas embolism or decompression sickness, who also have a pneumothorax, will experience relief upon recompression. A chest tube or other device with a one-way relief valve may need to be inserted at depth to prevent expansion of the trapped gas during subsequent ascent. A tension pneumothorax should always be suspected if the diver's condition deteriorates rapidly during ascent, especially if the symptoms are respiratory. If a tension pneumothorax is found, recompress to depth of relief until the thoracic cavity can be properly vented. Pneumothorax, if present in combination with arterial gas embolism or decompression sickness, should not prevent immediate recompression therapy. However, a pneumothorax may need to be vented as described before ascent from treatment depth. In cases of tension pneumothorax, this procedure may be lifesaving.

3-8.3.4 **Prevention of Pneumothorax.** The strategies for avoiding pneumothorax are the same as those for avoiding arterial gas embolism. Breathe normally during ascent. If forced to perform an emergency ascent, exhale continuously.

3-9 INDIRECT EFFECTS OF PRESSURE ON THE HUMAN BODY

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.

3-9.1 Nitrogen Narcosis.

Nitrogen narcosis is the state of euphoria and exhilaration that occurs when a diver breathes a gas mixture with a nitrogen partial pressure greater than approximately 4 ata.

3-9.1.1 **Causes of Nitrogen Narcosis.** Breathing nitrogen at high partial pressures has a narcotic effect on the central nervous system that causes euphoria and impairs the diver's ability to think clearly. The narcotic effect begins at a nitrogen partial pressure of approximately 4 ata and increases in severity as the partial pressure is increased beyond that point. A nitrogen partial pressure of 8 ata causes very marked impairment; partial pressures in excess of 10 ata may lead to hallucinations and unconsciousness. For a dive on air, narcosis usually appears at a depth of approximately 130 fsw, is very prominent at a depth of 200 fsw, and becomes disabling at deeper depths.

There is a wide range of individual susceptibility to narcosis. There is also some evidence that adaptation occurs on repeated exposures. Some divers, particularly those experienced in deep operations with air, can often work as deep as 200 fsw without serious difficulty. Others cannot.

3-9.1.2 **Symptoms of Nitrogen Narcosis.** The symptoms of nitrogen narcosis include:

- 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.

3-9.1.3 **Treatment of Nitrogen Narcosis.** The treatment for nitrogen narcosis is to bring the diver to a shallower depth where the effects are not felt. The narcotic effects will rapidly dissipate during the ascent. There is no hangover associated with nitrogen narcosis.

3-9.1.4 **Prevention of Nitrogen Narcosis.** 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 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 or dive with helium-oxygen mixtures.

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. Diving with helium-oxygen mixtures is the only way to prevent nitrogen narcosis. Helium-oxygen mixtures should be considered for any dive in excess of 150 fsw.

3-9.2 Oxygen Toxicity. Exposure to a partial pressure of oxygen above that encountered in normal daily living may be toxic to the body. The extent of the toxicity is dependent upon both the oxygen partial pressure and the exposure time. The higher the partial pressure and the longer the exposure, the more severe the toxicity. The two types of oxygen toxicity experienced by divers are pulmonary oxygen toxicity and central nervous system (CNS) oxygen toxicity.

3-9.2.1 **Pulmonary Oxygen Toxicity.** Pulmonary oxygen toxicity, sometimes called low pressure oxygen poisoning, can occur whenever the oxygen partial pressure exceeds 0.5 ata. A 12 hour exposure to a partial pressure of 1 ata will produce mild symptoms and measurable decreases in lung function. The same effect will occur with a 4 hour exposure at a partial pressure of 2 ata.

Long exposures to higher levels of oxygen, such as administered during Recompression Treatment Tables 4, 7, and 8, may produce 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. 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.

Return to normal pulmonary function gradually occurs after the exposure is terminated. There is no specific treatment for pulmonary oxygen toxicity.

The only way to avoid pulmonary oxygen toxicity completely is to avoid the long exposures to moderately elevated oxygen partial pressures that produce it. However, there is a way of extending tolerance. If the oxygen exposure is periodically interrupted by a short period of time at low oxygen partial pressure, the total exposure time needed to produce a given level of toxicity can be increased significantly.

3-9.2.2 **Central Nervous System (CNS) Oxygen Toxicity.** Central nervous system (CNS) oxygen toxicity, sometimes called high pressure oxygen poisoning, can occur whenever the oxygen partial pressure exceeds 1.3 ata in a wet diver or 2.4 ata in a dry diver. The reason for the marked increase in susceptibility in a wet diver is not completely understood. At partial pressures above the respective 1.3 ata wet and 2.4 ata dry thresholds, the risk of CNS toxicity is dependent on the oxygen partial pressure and the exposure time. The higher the partial pressure and the longer the

exposure time, the more likely CNS symptoms will occur. This gives rise to partial pressure of oxygen-exposure time limits for various types of diving.

3-9.2.2.1 ***Factors Affecting the Risk of CNS Oxygen Toxicity.*** A number of factors are known to influence the risk of CNS oxygen toxicity:

Individual Susceptibility. Susceptibility to CNS oxygen toxicity varies markedly from person to person. Individual susceptibility also varies markedly from time to time and for this reason divers may experience CNS oxygen toxicity at exposure times and pressures previously tolerated. Individual variability makes it difficult to set oxygen exposure limits that are both safe and practical.

CO_2 Retention. Hypercapnia greatly increases the risk of CNS toxicity probably through its effect on increasing brain blood flow and consequently brain oxygen levels. Hypercapnia may result from an accumulation of CO_2 in the inspired gas or from inadequate ventilation of the lungs. The latter is usually due to increased breathing resistance or a suppression of respiratory drive by high inspired ppO_2. Hypercapnia is most likely to occur on deep dives and in divers using closed and semi-closed circuit rebreathers.

Exercise. Exercise greatly increases the risk of CNS toxicity, probably by increasing the degree of CO_2 retention. Exposure limits must be much more conservative for exercising divers than for resting divers.

Immersion in Water. Immersion in water greatly increases the risk of CNS toxicity. The precise mechanism for the big increase in risk over comparable dry chamber exposures is unknown, but may involve a greater tendency for diver CO_2 retention during immersion. Exposure limits must be much more conservative for immersed divers than for dry divers.

Depth. Increasing depth is associated with an increased risk of CNS toxicity even though ppO_2 may remain unchanged. This is the situation with UBAs that control the oxygen partial pressure at a constant value, like the MK 16. The precise mechanism for this effect is unknown, but is probably more than just the increase in gas density and concomitant CO_2 retention. There is some evidence that the inert gas component of the gas mixture accelerates the formation of damaging oxygen free radicals. Exposure limits for mixed gas diving must be more conservative than for pure oxygen diving.

Intermittent Exposure. Periodic interruption of high ppO_2 exposure with a 5-15 min exposure to low ppO_2 will reduce the risk of CNS toxicity and extend the total allowable exposure time to high ppO_2. This technique is most often employed in hyperbaric treatments and surface decompression.

Because of these modifying influences, allowable oxygen exposure times vary from situation to situation and from diving system to diving system. In general, closed and semi-closed circuit rebreathing systems require the lowest partial pressure limits, whereas surface-supplied open-circuit systems permit slightly higher

limits. Allowable oxygen exposure limits for each system are discussed in later chapters.

3-9.2.2.2 ***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 convulsions that occur with little or no warning.

Warning symptoms may not always appear and most are not exclusively symptoms of oxygen toxicity. Muscle twitching is perhaps the clearest warning, but it may occur late, if at all. If any of these warning symptoms occur, the diver should take immediate action to lower the oxygen partial pressure.

A convulsion, 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.

3-9.2.2.3 ***Treatment of CNS Oxygen Toxicity.*** A diver who experiences the warning symptoms of oxygen toxicity shall inform the Diving Supervisor immediately. The following actions can be taken to lower the oxygen partial pressure:

- Ascend
- Shift to a breathing mixture with a lower oxygen percentage
- In a recompression chamber, remove the mask.

WARNING **Reducing the oxygen partial pressure does not instantaneously reverse the biochemical changes in the central nervous system caused by high oxygen partial pressures. 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.**

Despite its rather alarming appearance, the convulsion itself is usually not much more than a strenuous muscular workout for the victim. 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 diver convulses, the UBA should be ventilated immediately with a gas of lower oxygen content, if possible. If depth control is possible and the gas supply is secure (helmet or full face mask), the diver should be kept at depth until the convulsion subsides and normal breathing resumes. If an ascent must take place, it should be done as slowly as possible to reduce the risk of an arterial gas embolism. AGE should be considered in any diver surfacing unconscious due to an oxygen convulsion.

If the 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. Management of CNS oxygen toxicity during recompression therapy is discussed fully in Volume 5.

If a convulsing diver is prevented from drowning or causing other injury to himself, full recovery with no lasting effects can be expected within 24 hours. Susceptibility to oxygen toxicity does not increase as a result of a convulsion, although divers

may be more inclined to notice warning symptoms during subsequent exposures to oxygen.

3-9.2.2.4 ***Prevention of CNS Oxygen Toxicity.*** 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. Interruption of oxygen breathing with periodic "air" breaks can extend the exposure time to high oxygen partial pressures significantly. Air breaks are routinely incorporated into recompression treatment tables and some decompression tables.

3-9.3 Decompression Sickness (DCS). A diver's blood and tissues absorb additional nitrogen (or helium) from the lungs when at depth. If a diver ascends too fast this excess gas will separate from solution and form bubbles. These bubbles produce mechanical and biochemical effects that lead to a condition known as *decompression sickness*.

3-9.3.1 **Absorption and Elimination of Inert Gases.** The average human body at sea level contains about 1 liter of nitrogen. All of the body tissues are saturated with nitrogen at a partial pressure equal to the partial pressure in the alveoli, about 0.79 ata. If the partial pressure of nitrogen changes because of a change in the pressure or composition of the breathing mixture, the pressure of the nitrogen dissolved in the body gradually attains a matching level. Additional quantities of nitrogen are absorbed or eliminated, depending on the partial pressure gradient, until the partial pressure of the gas in the lungs and in the tissues is equal. If a diver breathes helium, a similar process occurs.

As described by Henry's Law, the amount of gas that dissolves in a liquid is almost directly proportional to the partial pressure of the 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 inert gas is called absorption or saturation. The process of giving up inert gas is called elimination or desaturation. The chain of events is essentially the same in both processes even though the direction of exchange is opposite.

Shading in diagram (Figure 3-16) 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

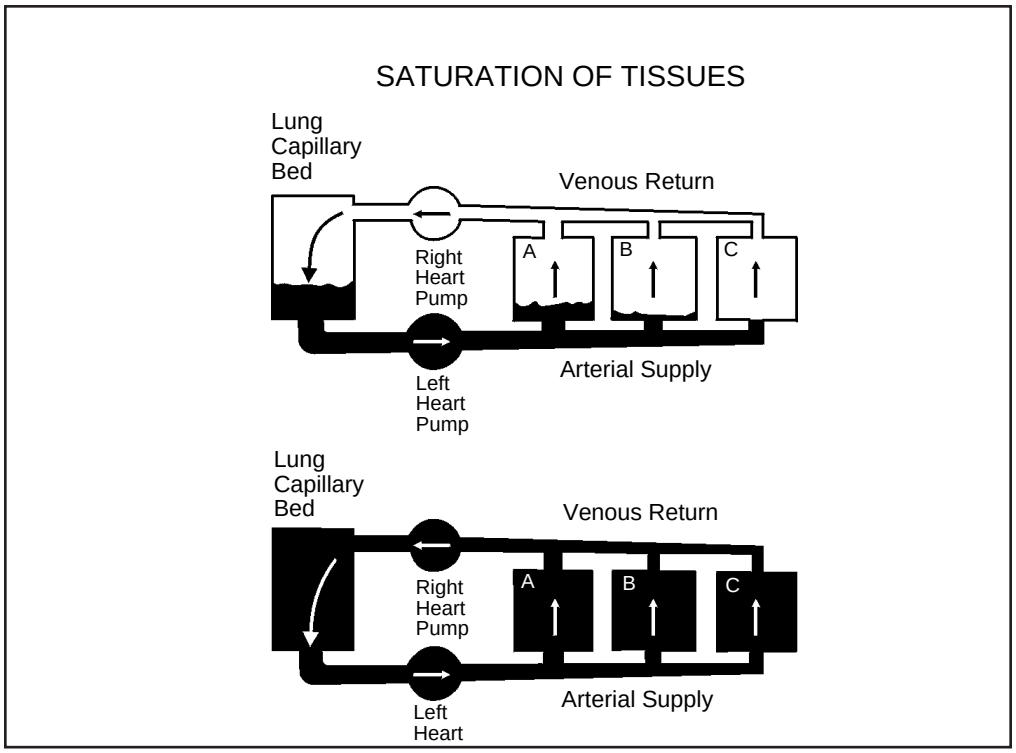

Figure 3-16. Saturation of Tissues. Shading in diagram indicates saturation with nitrogen or helium under increased pressure. Blood becomes saturated on passing through lungs, and tissues are saturated in turn via blood. Those with a large supply (as in A above) are saturated much more rapidly than those 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.

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.

3-9.3.1.1 ***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 3-16). 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 = 3.2 ata, while the blood and tissues remain temporarily at 0.8 ata. 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 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

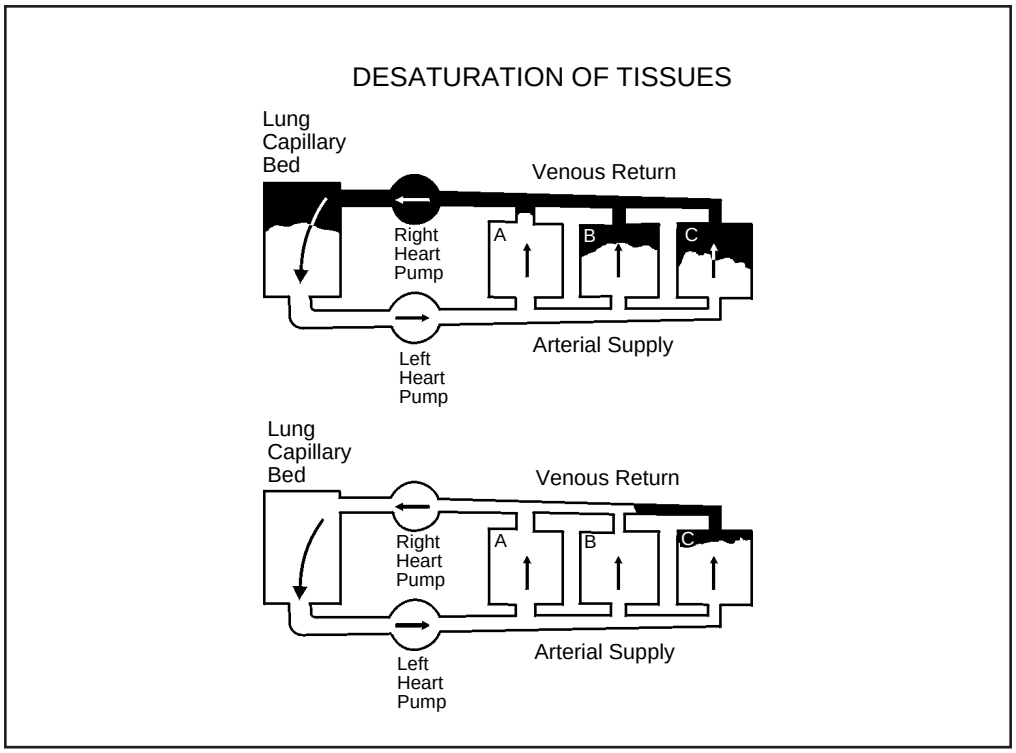

Figure 3-17. Desaturation of Tissues. The desaturation process is essentially the reverse of saturation. When pressure of inert gas is lowered, blood is cleared of excess gas as it goes through the lungs. Blood then removes gas from the tissues at rates depending on amount of blood that flows through 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).

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.

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. This is because the blood and tissue solubilities are different for the different inert gases. Helium, for example, is much less soluble in fat than nitrogen is.

3-9.3.1.2 ***Desaturation of Tissues.*** The process of desaturation is the reverse of saturation (Figure 3-17). If the partial pressure of the inert gas in the lungs is reduced, either through a reduction in the diver's depth 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

reason that they saturate more slowly: poor blood supply or a greater capacity to store inert gas. Washout of excess inert gas from these "slow" tissues will lag behind washout from the faster tissues.

3-9.3.2 **Bubble Formation.** Inert gas may separate from physical solution and form bubbles if the partial pressure of the inert gas in blood and tissues exceeds the ambient pressure by more than a critical amount. During descent and while the diver is on the bottom, blood and tissue inert gas partial pressures increase significantly as tissue saturation takes place, but the inert gas pressure always remains less than the ambient pressure surrounding the diver. Bubbles cannot form in this situation. During ascent the converse is true. Blood and tissue inert gas pressures fall as the tissues desaturate, but blood and tissue inert gas pressures can exceed the ambient pressure if the rate of ascent is faster than the rate at which tissues can equilibrate. Consider an air diver fully saturated with nitrogen at a depth of 100 fsw. All body tissues have a nitrogen partial pressure of 3.2 ata. If the diver were to quickly ascend to the surface, the ambient pressure surrounding his tissues would be reduced to 1 ata. Assuming that ascent was fast enough not to allow for any tissue desaturation, the nitrogen pressure in all the tissues would be 2.2 ata greater than the ambient pressure (3.2 ata - 1 ata). Under this circumstance bubbles can form.

Bubble formation can be avoided if the ascent is controlled in such a way that the tissue inert gas pressure never exceeds the ambient pressure by more than the critical amount. This critical amount, called the *allowable supersaturation*, varies from tissue to tissue and from one inert gas to another. A decompression table shows the time that must be spent at various decompression stops on the way to the surface to allow each tissue to desaturate to the point where its allowable supersaturation is not exceeded.

3-9.3.3 **Direct Bubble Effects.** Bubbles forming in the tissues (autochthonous bubbles) and in the bloodstream (circulating bubbles) may exert their effects directly in several ways:

- Autochthonous bubbles can put pressure on nerve endings, stretch and tear tissue leading to hemorrhage, and increase pressure in the tissue leading to slowing or cessation of incoming blood flow. These are thought to be the primary mechanisms for injury in Spinal Cord, Musculoskeletal, and Inner Ear DCS.

- Venous bubbles can partially or completely block the veins draining various organs leading to reduced organ blood flow (venous obstruction). Venous obstruction in turn leads to tissue hypoxia, cell injury and death. This is one of the secondary mechanisms of injury in Spinal Cord DCS.

- Venous bubbles carried to the lung as emboli (called venous gas emboli or VGE) can partially block the flow of blood through the lung leading to fluid build up (pulmonary edema) and decreased gas exchange. The result is systemic hypoxia and hypercarbia. This is the mechanism of damage in Pulmonary DCS.

- Arterial bubbles can act as emboli blocking the blood supply of almost any

tissue leading to hypoxia, cell injury and death. Arterial gas embolism and autochotonous bubble formation are thought be the primary mechanisms of injury in Cerebral (brain) DCS.

The damage done by the direct bubble effect occurs within a relatively short period of time (a few minutes to hours). The primary treatment for these effects is recompression. Recompression will compress the bubble to a smaller diameter, restore blood flow, decrease venous congestion, and improve gas exchange in the lungs and tissues. It also increases the speed at which the bubbles outgas and collapse.

3-9.3.4 **Indirect Bubble Effects.** Bubbles may also exert their effects indirectly because a bubble 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 eliminate the foreign body. Typical reactions include:

- Blood vessels become "leaky" due to damage to the endothelial lining cells and chemical release. Blood plasma leaks out while blood cells remain inside. The blood becomes thick and more difficult to pump. Organ blood flow is reduced.
- 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 fat clumps 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.

Indirect bubble effects take place over a longer period of time than the direct bubble 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.

3-9.3.5 **Symptoms of Decompression Sickness.** Decompression sickness is generally divided into two categories. Type I decompression sickness involves the skin, lymphatic system, muscles and joints and is not life threatening. Type II decompression sickness (also called serious decompression sickness) involves the nervous system, respiratory system, or circulatory system. Type II decompression sickness may become life threatening. Because the treatment of Type I and Type II decompression sickness may be different, it is important to distinguish between these two types. Symptoms of Type I and Type II decompression sickness may be present at the same time.

When the skin is involved, the symptoms are itching or burning usually accompanied by a rash. Involvement of the lymphatic system produces swelling of regional lymph nodes or an extremity. Involvement of the musculoskeletal system produces pain, which in some cases can be excruciating. 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 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 may prove fatal. A large number of bubbles in the circulation can lead to cardiovascular collapse and death. Unusual fatigue or exhaustion after a dive is probably due to bubbles in unusual locations and the biochemical changes they have induced. While not attributable to a specific organ system, unusual fatigue is a definite symptom of decompression sickness.

3-9.3.5.1 ***Time Course of Symptoms.*** Decompression sickness usually occurs after surfacing. If the dive is particularly arduous or decompression has been omitted, however, the diver may experience decompression sickness before reaching the surface.

After surfacing, there is a latency period before symptoms appear. This may be as short as several minutes to as long as several days. Long, shallow dives are generally associated with longer latencies than deep, short dives. For most dives, the onset of decompression sickness can be expected within several hours of surfacing.

3-9.3.6 **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.

3-9.3.7 **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 THERMAL PROBLEMS IN DIVING

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 environmental conditioning when external conditions tend toward cold or hot extremes.

Thermal problems, arising from exposure to various temperatures of water, pose a major consideration when planning operational dives and selecting equipment. Bottom time may be limited more by a diver's intolerance to heat or cold than his exposure to increased oxygen partial pressures or the amount of decompression required.

The diver's thermal status affects the rate of inert gas uptake and elimination. Divers who are warm on the bottom will absorb more inert gas than divers who are cold. No-decompression dives in warm water, therefore, may carry a greater risk of DCS than comparable dives in cold water. Given identical exposures on the bottom, divers who are warm during decompression stops will lose more inert gas and have a lower risk of DCS than divers who are cold.

3-10.1 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.

3-10.2 Excessive Heat Loss (Hypothermia). Hypothermia is a lowering of the core temperature of the body. Immersion hypothermia is a potential hazard whenever diving operations take place in cool to cold waters. A diver's response to immersion in cold water depends on the degree of thermal protection worn and water temperature. 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).

3-10.2.1 **Causes of Hypothermia.** Hypothermia in diving occurs when the difference between the water and body temperature is large enough for the body to lose more heat than it produces. 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.

3-10.2.2 **Symptoms of Hypothermia.** In mild cases, the victim will experience uncontrolled shivering, slurred speech, imbalance, and/or poor judgment. Severe cases of hypothermia are characterized by loss of shivering, impaired mental status, irregular heartbeat, and/or very shallow pulse or respirations. This is a medical emergency. The signs and symptoms of falling core temperature are given in Table 3-1, though individual responses to falling core temperature will vary. At extremely low temperatures or with prolonged immersion, body heat loss reaches a point at which death occurs.

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

3-10.2.3 **Treatment of Hypothermia.** To treat mild hypothermia, passive and active rewarming measures may be used and should be continued until the victim is sweating. Rewarming techniques include:

Passive:

- Remove all wet clothing.

- Wrap victim in a blanket (preferably wool).
- Place in an area protected from wind.
- If possible, place in a warm area (i.e. galley).

Active:

- Warm shower or bath.
- Place in a very warm space (i.e., engine room).

To treat severe hypothermia avoid any exercise, keep the victim lying down, initiate only passive rewarming, and immediately transport to the nearest medical treatment facility.

CAUTION **Do not institute active rewarming with severe cases of hypothermia.**

WARNING **CPR should not be initiated on a severely hypothermic diver unless it can be determined that the heart has stopped or is in ventricular fibrillation. CPR should not be initiated in a patient that is breathing.**

3-10.2.4 **Prevention of Hypothermia.** 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.

Hypothermia can be insidious and cause problems without the diver being aware of it. The diver should wear appropriate thermal protection based upon the water temperature and expected bottom time (See Chapter 6). 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. Acclimatization, adequate hydration, experience, and common sense all play a role in preventing hypothermia. Provide the diver and topside personnel adequate shelter from the elements. Adequate predive hydration is essential.

Heat loss through the respiratory tract becomes an increasingly significant factor in deeper diving. Inhaled gases are heated in the upper respiratory tract and more energy is required to heat the denser gases encountered at depth. In fact, a severe respiratory insult can develop if a diver breathes unheated gas while making a deep saturation dive in cold water. Respiratory gas heating is required in such situations.

3-10.3 Other Physiological Effects of Exposure to Cold Water. In addition to hypothermia, other responses to exposure to cold water create potential hazards for the diver.

3-10.3.1 **Caloric Vertigo.** The eardrum does not have to rupture for caloric vertigo to occur. Caloric vertigo can occur simply as the result of having water enter the external ear canal on one side but not the other. The usual cause is a tight fitting wet suit hood that allows cold water access to one ear, but not the other. It can also occur when one external canal is obstructed by wax. Caloric vertigo may occur suddenly upon entering cold water or when passing through thermoclines. The effect is usually short lived, but while present may cause significant disorientation and nausea.

3-10.3.2 **Diving Reflex.** Sudden exposure of the face to cold water or immersion of the whole body in cold water may cause an immediate slowing of the heart rate (bradycardia) and intense constriction of the peripheral blood vessels. Sometimes abnormal heart rhythms accompany the bradycardia. This response is known as the *diving reflex*. Removing or losing a facemask in cold water can trigger the diving reflex. It is still not known whether cardiac arrhythmias associated with the diving reflex contribute to diving casualties. Until this issue is resolved, it is prudent for divers to closely monitor each other when changing rigs underwater or buddy breathing.

3-10.3.3 **Uncontrolled Hyperventilation.** If a diver with little or no thermal protection is suddenly plunged into very cold water, the effects are immediate and 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. The lack of breathing control makes survival in rough water very unlikely.

3-10.4 Excessive Heat Gain (Hyperthermia). Hyperthermia is a raising of the core temperature of the body. Hyperthermia should be considered a potential risk any time air temperature exceeds 90°F or water temperature is above 82°F. 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 body core temperature should not exceed 102.2°F (39°C). By the time the diver's core temperature approaches 102°F noticeable mental confusion may be present.

3-10.4.1 **Causes of Hyperthermia.** Divers are susceptible to hyperthermia when they are unable to dissipate their body heat. This may result from high water temperatures,

protective garments, rate of work, and the duration of the dive. Predive heat exposure may lead to significant dehydration and put the diver at greater risk of hyperthermia.

3-10.4.2 **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. In severe cases of hyperthermia (severe heat exhaustion or heat stroke), the victim will experience disorientation, tremors, loss of consciousness and/or seizures.

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

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

3-10.4.3 **Treatment of Hyperthermia.** The treatment of all cases of hyperthermia shall include cooling of the victim to reduce the core temperature. In mild to moderate hyperthermia cooling should be started immediately by removing the victim's clothing, spraying him with a fine mist of lukewarm-to-cool water, and then fanning. This causes a large increase in evaporative cooling. Avoid whole body immersion in cold water or packing the body in ice as this will cause vasoconstriction which will decrease skin blood flow and may slow the loss of heat. Ice packs to the neck, armpit or groin may be used. Oral fluid replacement should begin as soon as the victim can drink and continue until he has urinated pale to clear urine several times. If the symptoms do not improve, the victim shall be transported to a medical treatment facility.

Severe hyperthermia is a medical emergency. Cooling measures shall be started and the victim shall be transported immediately to a medical treatment facility. Intravenous fluids should be administered during transport.

3-10.4.4 **Prevention of Hyperthermia.** 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. 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. Physically fit individuals and those with lower levels of body fat are less likely to develop hyperthermia. Guidelines for diving in warm water are contained in Chapter 6.

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. Acclimatization 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.

3-11 SPECIAL MEDICAL PROBLEMS ASSOCIATED WITH DEEP DIVING

3-11.1 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, depth of 1000 fsw may be achieved with relative freedom from HPNS. Beyond 1000 fsw, 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.

3-11.2 Compression Arthralgia. Most divers will experience pain in the joints during compression on deep dives. This condition is called *compression arthralgia*. The shoulders, knees, writs, and hips are the joints most commonly affected. The fingers, lower back, neck, and ribs may also be involved. The pain may be a constant deep ache similar to Type I decompression sickness, or a sudden, sharp, and intense but short-lived pain brought on my movement of the joint. These pains may be accompanied by “popping” or “cracking” of joints or a dry “gritty” feeling within the joint.

The incidence and intensity of compression arthralgia symptoms are dependent on the depth of the dive, the rate of compression, and individual susceptibility. While primarily a problem of deep saturation diving, mild symptoms may occur with rapid compression on air or helium-oxygen dives as shallow as 100 fsw. In deep helium saturation dives with slower compression rates, symptoms of compression arthralgia usually begins between 200 and 300 fsw, and increase in intensity as deeper depths are attained. Deeper than 600 fsw, compression pain may occur even with extremely slow rates of compression.

Compression joint pain may be severe enough to limit diver activity, travel rate, and depths attainable during downward excursion dives from saturation. Improvement is generally noted during the days spent at the saturation depth but, on occasion, these pains may last well into the decompression phase of the dive until shallower depths are reached. Compression pain can be distinguished from decompression sickness pain because it was present before decompression was started and does not increase in intensity with decreasing depth.

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

3-12 OTHER DIVING MEDICAL PROBLEMS

3-12.1 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.

3-12.1.1 **Causes of Dehydration.** Dehydration usually results from inadequate fluid intake and/or excessive perspiration in hot climates. Unless adequate attention is paid to hydration, there is a significant chance the diver in a hot climate will enter the water in a dehydrated state.

Immersion in water creates a special situation that can lead to dehydration in its own right. The water pressure almost exactly counterbalances the hydrostatic pressure gradient that exists from head to toe in the circulatory system. As a result, blood which is normally pooled in the leg veins is translocated to the chest, causing an increase central blood volume. The body mistakenly interprets the increase in central blood as a fluid excess. A reflex is triggered leading to an increase in urination, a condition called *immersion diuresis*. The increased urine flow leads to steady loss of water from the body and a concomitant reduction in blood volume during the dive. The effects of immersion diuresis are felt when the diver leaves the water. Blood pools once again in the leg veins. Because total blood volume is reduced, central blood volume falls dramatically. The heart may have difficulty getting enough blood to pump. The diver may experience lightheadness or faint while attempting to climb out of the water on a ladder or while standing on the stage. This is the result of a drop in blood pressure as the blood volume shifts to the legs. More commonly the diver will feel fatigued, less alert, and less able to think clearly than normal. His exercise tolerance will be reduced.

3-12.1.2 **Preventing Dehydration.** Dehydration is felt to increase the risk of decompression sickness. Divers should monitor their fluid intake and urine output during diving operations to insure that they keep themselves well hydrated. During the dive itself, there is nothing one can do to block the effects of immersion diuresis. Upon surfacing they should rehydrate themselves as soon as the opportunity presents itself.

3-12.2 Immersion Pulmonary Edema. Immersion in water can cause fluid to leak out of the circulation system and accumulate first in the interstitial tissues of the lungs then in the alveoli themselves. This condition is called *immersion pulmonary edema*. The exact mechanism of injury is not know, but the condition is probably related to the increase in central blood volume that occurs during immersion (see description above). Contributing factors include immersion in cold water, negative pressure breathing, and overhydration pre-dive, all of which enhance the increase in central blood volume with immersion. Heavy exercise is also a contributor.

Symptoms may begin on the bottom, during ascent, or shortly after surfacing and consist primarily of cough and shortness of breath. The diver may cough up blood tinged mucus. Chest pain is notably absent. A chest x-ray shows the classic pattern of pulmonary edema seen in heart failure.

A diver with immersion pulmonary edema should be placed on surface oxygen and transported immediately to a medical treatment facility. Signs and symptoms will usually resolve spontaneously over 24 hours with just bed rest and 100% oxygen.

Immersion pulmonary edema is a relatively rare condition, but the incidence appears to be increasing perhaps because of an over-emphasis on the need to hydrate before a dive. Adequate pre-dive hydration is essential, but overhydration is to be avoided. Beyond avoiding overhydration and negative pressure breathing situations, there is nothing the diver can do to prevent immersion pulmonary edema.

3-12.3 Carotid Sinus Reflex. External pressure on the carotid artery from a tight fitting neck dam, wet suit, or dry suit can activate receptors in the arterial wall, causing a decrease in heart rate with possible loss of consciousness. Using an extra-tight-fitting dry or wet suit or tight neck dams to decrease water leaks increase the chances of activation of the carotid reflex and the potential for problems.

3-12.4 Middle Ear Oxygen Absorption Syndrome. Middle ear oxygen absorption syndrome refers to the negative pressure that may develop in the middle ear following a long oxygen dive. Gas with a very high percentage of oxygen enters the middle ear cavity during an oxygen dive. Following the dive, the tissues of the middle ear slowly absorb the oxygen. If the eustachian tube does not open spontaneously, a negative pressure relative to ambient may result in the middle ear cavity. Symptoms are often noted the morning after a long oxygen dive. Middle ear oxygen absorption syndrome is difficult to avoid but usually does not pose a significant problem because symptoms are generally minor and easily eliminated. There may also be fluid (serous otitis media) present in the middle ear as a result of the differential pressure.

3-12.4.1 **Symptoms of Middle Ear Oxygen Absorption Syndrome.** The diver may notice mild discomfort and hearing loss in one or both ears. There may also be a sense of pressure and a moist, cracking sensation as a result of fluid in the middle ear.

3-12.4.2 **Treating Middle Ear Oxygen Absorption Syndrome.** Equalizing the pressure in the middle ear using a normal Valsalva maneuver or the diver's procedure of choice, such as swallowing or yawning, will usually relieve the symptoms. Discomfort and hearing loss resolve quickly, but the middle ear fluid is absorbed more slowly. If symptoms persist, a Diving Medical Technician or Diving Medical Officer shall be consulted.

3-12.5 Underwater Trauma. Underwater trauma is different from trauma that occurs at the surface because it may be complicated by the loss of the diver's gas supply and by the diver's decompression obligation. If possible, injured divers should be surfaced immediately and treated appropriately. If an injured diver is trapped, the first priority is to ensure sufficient breathing gas is available, then to stabilize the injury. At that point, a decision must be made as to whether surfacing is possible. If the decompression obligation is great, the injury will have to be stabilized until sufficient decompression can be accomplished. If an injured diver must be surfaced with missed decompression, the diver must be treated as soon as possible, realizing that the possible injury from decompression sickness may be as severe or more severe than that from the other injuries.

3-12.6 Blast Injury. 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.

3-12.7 Otitis Externa. Otitis externa (swimmer's ear) is an infection of the ear canal caused by repeated immersion. The water in which the dive is being performed does not have to be contaminated with bacteria for otitis externa to occur. The first symptom of otitis externa is an itching and/or wet feeling in the affected ear. This feeling will progress to local pain as the external ear canal becomes swollen and inflamed. Local lymph nodes (glands) may enlarge, making jaw movement painful. Fever may occur in severe cases. Once otitis externa develops, the diver should discontinue diving and be examined and treated by Diving Medical Personnel.

Unless preventive measures are taken, otitis externa is very likely to occur during diving operations, causing unnecessary discomfort and restriction from diving. *External ear prophylaxis*, a technique to prevent swimmer's ear, should be done each morning, after each wet dive, and each evening during diving operations. External ear prophylaxis is accomplished using a 2 percent acetic acid in aluminum acetate (e.g., Otic Domboro) solution. The head is tilted to one side and the external ear canal gently filled with the solution, which must remain in the canal for 5 minutes. The head is then tilted to the other side, the solution allowed to run out and the procedure repeated for the other ear. The 5-minute duration shall be timed with a watch. If the solution does not remain in the ear a full 5 minutes, the effectiveness of the procedure is greatly reduced.

During prolonged diving operations, the external ear canal may become occluded with wax (cerumen). When this happens, external ear prophylaxis is ineffective and the occurrence of otitis externa will become more likely. The external ear canal can be examined periodically with an otoscope to detect the presence of ear wax. If the eardrum cannot be seen during examination, the ear canal should be flushed gently with water, dilute hydrogen peroxide, or sodium bicarbonate solutions to remove the excess cerumen. Never use swabs or other instruments to remove cerumen; this is to be done only by trained medical personnel. Otitis externa is a particular problem in saturation diving if divers do not adhere to prophylactic measures.

3-12.8 Hypoglycemia. Hypoglycemia is an abnormally low blood sugar (glucose) level. Episodes of hypoglycemia are common in diabetics and pre-diabetics, but may also occur in normal individuals. Simply missing a meal tends to reduce blood sugar levels. 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 symptoms even in an individual who ordinarily has no abnormality in this respect.

Symptoms of hypoglycemia include unusual hunger, excessive sweating, numbness, chills, headache, trembling, dizziness, confusion, incoordination, anxiety, and in severe cases, loss of consciousness.

If hypoglycemia is present, giving sugar by mouth relieves the symptoms promptly and proves the diagnosis. If the victim is unconscious, glucose should be given intravenously.

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, attention to proper nutrition is required. 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.

3-12.9 Use of Medications while Diving. There are no hard and fast rules for deciding when a medication would preclude a diver from diving. In general, topical medications, antibiotics, birth control medication, and decongestants that do not cause drowsiness would not restrict diving. Diving medical personnel should be consulted to determine if any drugs preclude diving.

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