C H A P T E R 4 3

Aviation, High-Altitude, and Space Physiology

As we have ascended to higher and higher altitudes in aviation, mountain climbing, and space vehicles, it has become progressively more important to understand the effects of altitude and low gas pressures (as well as several other factors—acceleratory forces, weightlessness, and so forth) on the human body. This chapter deals with these problems.

Effects of Low Oxygen Pressure on the Body

Barometric Pressures at Different Altitudes. Table 43–1 gives the approximate *barometric* and *oxygen pressures* at different altitudes, showing that at sea level, the barometric pressure is 760 mm Hg; at 10,000 feet, only 523 mm Hg; and at 50,000 feet, 87 mm Hg. This decrease in barometric pressure is the basic cause of all the hypoxia problems in high-altitude physiology because, as the barometric pressure decreases, the atmospheric oxygen partial pressure decreases proportionately, remaining at all times slightly less than 21 per cent of the total barometric pressure—P_{O_2} at sea level about 159 mm Hg, but at 50,000 feet only 18 mm Hg.

Alveolar P_{O_2} at Different Elevations

Carbon Dioxide and Water Vapor Decrease the Alveolar Oxygen. Even at high altitudes, carbon dioxide is continually excreted from the pulmonary blood into the alveoli. Also, water vaporizes into the inspired air from the respiratory surfaces. These two gases dilute the oxygen in the alveoli, thus reducing the oxygen concentration. Water vapor pressure in the alveoli remains 47 mm Hg as long as the body temperature is normal, regardless of altitude.

In the case of carbon dioxide, during exposure to very high altitudes, the alveolar P_{CO_2} falls from the sea-level value of 40 mm Hg to lower values. In the *acclimatized* person, who increases his or her ventilation about fivefold, the P_{CO_2} falls to about 7 mm Hg because of increased respiration.

Now let us see how the pressures of these two gases affect the alveolar oxygen. For instance, assume that the barometric pressure falls from the normal sea-level value of 760 mm Hg to 253 mm Hg, which is the usual measured value at the top of 29,028–foot Mount Everest. Forty-seven millimeters of mercury of this must be water vapor, leaving only 206 mm Hg for all the other gases. In the *acclimatized* person, 7 mm of the 206 mm Hg must be carbon dioxide, leaving only 199 mm Hg. If there were no use of oxygen by the body, one fifth of this 199 mm Hg would be oxygen and four fifths would be nitrogen; that is, the P_{O_2} in the alveoli would be 40 mm Hg. However, some of this remaining alveolar oxygen is continually being absorbed into the blood, leaving about 35 mm Hg oxygen pressure in the alveoli. At the summit of Mount Everest, only the best of acclimatized people can barely survive when breathing air. But the effect is very different when the person is breathing pure oxygen, as we see in the following discussions.

Table 43–1

Effects of Acute Exposure to Low Atmospheric Pressures on Alveolar Gas Concentrations and Arterial Oxygen Saturation*

			Breathing Air			Breathing Pure Oxygen		
Altitude (ft)	Barometric Pressure (mm Hg)	P_{O_2} in Air (mm Hg)	*P_{CO_2} in Alveoli (mm Hg)*	*P_{O_2} in Alveoli (mm Hg)*	*Arterial Oxygen Saturation (%)*	*P_{CO_2} in Alveoli (mm Hg)*	*P_{O_2} in Alveoli (mm Hg)*	*Arterial Oxygen Saturation (%)*
0	760	159	40 (40)	104 (104)	97 (97)	40	673	100
10,000	523	110	36 (23)	67 (77)	90 (92)	40	436	100
20,000	349	73	24 (10)	40 (53)	73 (85)	40	262	100
30,000	226	47	24 (7)	18 (30)	24 (38)	40	139	99
40,000	141	29				36	58	84
50,000	87	18				24	16	15

* Numbers in parentheses are acclimatized values.

Alveolar P_{O_2} at Different Altitudes. The fifth column of Table 43–1 shows the approximate P_{O_2}s in the alveoli at different altitudes when one is breathing air for both the *unacclimatized* and the *acclimatized* person. At sea level, the alveolar P_{O_2} is 104 mm Hg; at 20,000 feet altitude, it falls to about 40 mm Hg in the unacclimatized person but only to 53 mm Hg in the acclimatized. The difference between these two is that alveolar ventilation increases much more in the acclimatized person than in the unacclimatized person, as we discuss later.

Saturation of Hemoglobin with Oxygen at Different Altitudes. Figure 43–1 shows arterial blood oxygen saturation at different altitudes while a person is breathing air and while breathing oxygen. Up to an altitude of about 10,000 feet, even when air is breathed, the arterial oxygen saturation remains at least as high as 90 per cent. Above 10,000 feet, the arterial oxygen saturation falls rapidly, as shown by the blue curve of the figure, until it is slightly less than 70 per cent at 20,000 feet and much less at still higher altitudes.

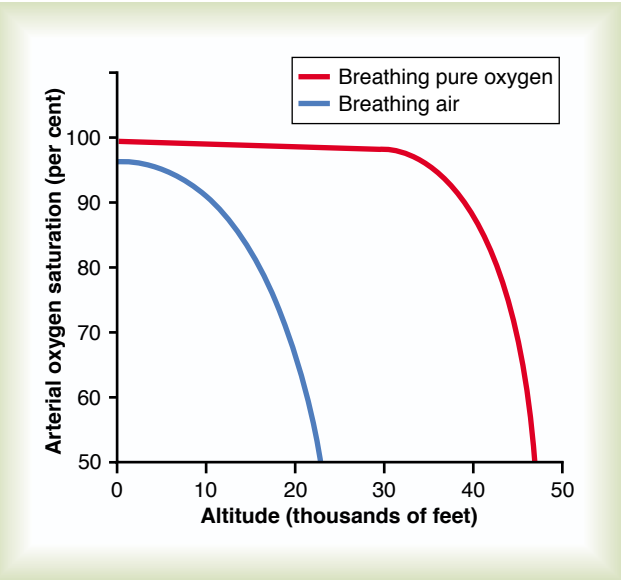

Figure 43–1

Effect of high altitude on arterial oxygen saturation when breathing air and when breathing pure oxygen.

Effect of Breathing Pure Oxygen on Alveolar P_{O_2} at Different Altitudes

When a person breathes pure oxygen instead of air, most of the space in the alveoli formerly occupied by nitrogen becomes occupied by oxygen. At 30,000 feet, an aviator could have an alveolar P_{O_2} as high as 139 mm Hg instead of the 18 mm Hg when breathing air (see Table 43–1).

The red curve of Figure 43–1 shows arterial blood hemoglobin oxygen saturation at different altitudes when one is breathing pure oxygen. Note that the saturation remains above 90 per cent until the aviator ascends to about 39,000 feet; then it falls rapidly to about 50 per cent at about 47,000 feet.

The "Ceiling" When Breathing Air and When Breathing Oxygen in an Unpressurized Airplane

Comparing the two arterial blood oxygen saturation curves in Figure 43–1, one notes that an aviator breathing pure oxygen in an unpressurized airplane can ascend to far higher altitudes than one breathing air. For instance, the arterial saturation at 47,000 feet when one is breathing oxygen is about 50 per cent and is equivalent to the arterial oxygen saturation at 23,000 feet when one is breathing air. In addition, because an unacclimatized person usually can remain conscious until the arterial oxygen saturation falls to 50 per cent, for short exposure times the ceiling for an aviator in an unpressurized airplane when breathing air is about 23,000 feet and when breathing pure oxygen is about 47,000 feet, provided the oxygen-supplying equipment operates perfectly.

Acute Effects of Hypoxia

Some of the important acute effects of hypoxia in the unacclimatized person breathing air, beginning at an

altitude of about 12,000 feet, are drowsiness, lassitude, mental and muscle fatigue, sometimes headache, occasionally nausea, and sometimes euphoria. These effects progress to a stage of twitchings or seizures above 18,000 feet and end, above 23,000 feet in the unacclimatized person, in coma, followed shortly thereafter by death.

One of the most important effects of hypoxia is decreased mental proficiency, which decreases judgment, memory, and performance of discrete motor movements. For instance, if an unacclimatized aviator stays at 15,000 feet for 1 hour, mental proficiency ordinarily falls to about 50 per cent of normal, and after 18 hours at this level it falls to about 20 per cent of normal.

Acclimatization to Low P_{O_2}

A person remaining at high altitudes for days, weeks, or years becomes more and more *acclimatized* to the low P_{O_2}, so that it causes fewer deleterious effects on the body. And it becomes possible for the person to work harder without hypoxic effects or to ascend to still higher altitudes.

The principal means by which acclimatization comes about are (1) a great increase in pulmonary ventilation, (2) increased numbers of red blood cells, (3) increased diffusing capacity of the lungs, (4) increased vascularity of the peripheral tissues, and (5) increased ability of the tissue cells to use oxygen despite low P_{O_2}.

Increased Pulmonary Ventilation—Role of Arterial Chemoreceptors. Immediate exposure to low P_{O_2} stimulates the arterial chemoreceptors, and this increases alveolar ventilation to a maximum of about 1.65 times normal. Therefore, compensation occurs within seconds for the high altitude, and it alone allows the person to rise several thousand feet higher than would be possible without the increased ventilation. Then, if the person remains at very high altitude for several days, the chemoreceptors increase ventilation still more, up to about five times normal.

The immediate increase in pulmonary ventilation on rising to a high altitude blows off large quantities of carbon dioxide, reducing the P_{CO_2} and increasing the pH of the body fluids. These changes *inhibit* the brain stem respiratory center and thereby *oppose the effect of low P_{O_2} to stimulate respiration by way of the peripheral arterial chemoreceptors in the carotid and aortic bodies*. But during the ensuing 2 to 5 days, this inhibition fades away, allowing the respiratory center to respond with full force to the peripheral chemoreceptor stimulus from hypoxia, and ventilation increases to about five times normal.

The cause of this fading inhibition is believed to be mainly a reduction of bicarbonate ion concentration in the cerebrospinal fluid as well as in the brain tissues. This in turn decreases the pH in the fluids surrounding the chemosensitive neurons of the respiratory center, thus increasing the respiratory stimulatory activity of the center.

An important mechanism for the gradual decrease in bicarbonate concentration is compensation by the kidneys for the respiratory alkalosis, as discussed in Chapter 30. The kidneys respond to decreased P_{CO_2} by reducing hydrogen ion secretion and increasing bicarbonate excretion. This metabolic compensation for the respiratory alkalosis gradually reduces plasma and cerebrospinal fluid bicarbonate concentration and pH toward normal and removes part of the inhibitory effect on respiration of low hydrogen ion concentration. Thus, the respiratory centers are much more responsive to the peripheral chemoreceptor stimulus caused by the hypoxia after the kidneys compensate for the alkalosis.

Increase in Red Blood Cells and Hemoglobin Concentration During Acclimatization. As discussed in Chapter 32, hypoxia is the principal stimulus for causing an increase in red blood cell production. Ordinarily, when a person remains exposed to low oxygen for weeks at a time, the hematocrit rises slowly from a normal value of 40 to 45 to an average of about 60, with an average increase in whole blood hemoglobin concentration from normal of 15 g/dl to about 20 g/dl.

In addition, the blood volume also increases, often by 20 to 30 per cent, and this increase times the increased blood hemoglobin concentration gives an increase in total body hemoglobin of 50 or more per cent.

Increased Diffusing Capacity After Acclimatization. It will be recalled that the normal diffusing capacity for oxygen through the pulmonary membrane is about 21 ml/mm Hg/min, and this diffusing capacity can increase as much as threefold during exercise. A similar increase in diffusing capacity occurs at high altitude.

Part of the increase results from increased pulmonary capillary blood volume, which expands the capillaries and increases the surface area through which oxygen can diffuse into the blood. Another part results from an increase in lung air volume, which expands the surface area of the alveolar-capillary interface still more. A final part results from an increase in pulmonary arterial blood pressure; this forces blood into greater numbers of alveolar capillaries than normally—especially in the upper parts of the lungs, which are poorly perfused under usual conditions.

Peripheral Circulatory System Changes During Acclimatization—Increased Tissue Capillarity. The cardiac output often increases as much as 30 per cent immediately after a person ascends to high altitude but then decreases back toward normal *over a period of weeks as the blood hematocrit increases*, so that the amount of oxygen transported to the peripheral body tissues remains about normal.

Another circulatory adaptation is *growth of increased numbers of systemic circulatory capillaries* in the nonpulmonary tissues, which is called *increased tissue capillarity* (or *angiogenesis*). This occurs

especially in animals born and bred at high altitudes but less so in animals that later in life become exposed to high altitude.

In active tissues exposed to chronic hypoxia, the increase in capillarity is especially marked. For instance, capillary density in right ventricular muscle increases markedly because of the combined effects of hypoxia and excess workload on the right ventricle caused by pulmonary hypertension at high altitude.

Cellular Acclimatization. In animals native to altitudes of 13,000 to 17,000 feet, cell mitochondria and cellular oxidative enzyme systems are slightly more plentiful than in sea-level inhabitants. Therefore, it is presumed that the tissue cells of high altitude–acclimatized human beings also can use oxygen more effectively than can their sea-level counterparts.

Natural Acclimatization of Native Human Beings Living at High Altitudes

Many native human beings in the Andes and in the Himalayas live at altitudes above 13,000 feet—one group in the Peruvian Andes lives at an altitude of 17,500 feet and works a mine at an altitude of 19,000 feet. Many of these natives are born at these altitudes and live there all their lives. In all aspects of acclimatization, the natives are superior to even the best-acclimatized lowlanders, even though the lowlanders might also have lived at high altitudes for 10 or more years. Acclimatization of the natives begins in infancy. The chest size, especially, is greatly increased, whereas the body size is somewhat decreased, giving a high ratio of ventilatory capacity to body mass. In addition, their hearts, which from birth onward pump extra amounts of cardiac output, are considerably larger than the hearts of lowlanders.

Delivery of oxygen by the blood to the tissues is also highly facilitated in these natives. For instance, Figure 43–2 shows oxygen-hemoglobin dissociation curves for natives who live at sea level and for their counterparts who live at 15,000 feet. Note that the arterial oxygen Po_2 in the natives at high altitude is only 40 mm Hg, but because of the greater quantity of hemoglobin, the quantity of oxygen in their arterial blood is greater than that in the blood of the natives at the lower altitude. Note also that the venous Po_2 in the high-altitude natives is only 15 mm Hg less than the venous Po_2 for the lowlanders, despite the very low arterial Po_2, indicating that oxygen transport to the tissues is exceedingly effective in the naturally acclimatized high-altitude natives.

Reduced Work Capacity at High Altitudes and Positive Effect of Acclimatization

In addition to the mental depression caused by hypoxia, as discussed earlier, the work capacity of all muscles is greatly decreased in hypoxia. This includes not only skeletal muscles but also cardiac muscles.

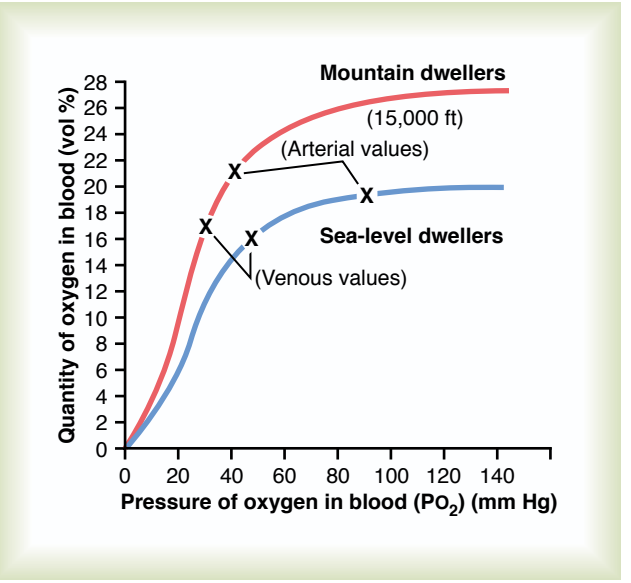

Figure 43–2

Oxygen-hemoglobin dissociation curves for blood of high-altitude residents (*red curve*) and sea-level residents (*blue curve*), showing the respective arterial and venous Po_2 levels and oxygen contents as recorded in their native surroundings. (Data from Oxygen-dissociation curves for bloods of high-altitude and sea-level residents. PAHO Scientific Publication No. 140, Life at High Altitudes, 1966.)

In general, work capacity is reduced in direct proportion to the decrease in maximum rate of oxygen uptake that the body can achieve.

To give an idea of the importance of acclimatization in increasing work capacity, consider this: The work capacities as per cent of normal for unacclimatized and acclimatized people at an altitude of 17,000 feet are as follows:

	Work capacity (per cent of normal)
Unacclimatized	50
Acclimatized for 2 months	68
Native living at 13,200 feet but working at 17,000 feet	87

Thus, naturally acclimatized native persons can achieve a daily work output even at high altitude almost equal to that of a lowlander at sea level, but even well-acclimatized lowlanders can almost never achieve this result.

Acute Mountain Sickness and High-Altitude Pulmonary Edema

A small percentage of people who ascend rapidly to high altitudes become acutely sick and can die if not

given oxygen or removed to a low altitude. The sickness begins from a few hours up to about 2 days after ascent. Two events frequently occur:

1. *Acute cerebral edema*. This is believed to result from local vasodilation of the cerebral blood vessels, caused by the hypoxia. Dilation of the arterioles increases blood flow into the capillaries, thus increasing capillary pressure, which in turn causes fluid to leak into the cerebral tissues. The cerebral edema can then lead to severe disorientation and other effects related to cerebral dysfunction.
2. *Acute pulmonary edema*. The cause of this is still unknown, but a suggested answer is the following: The severe hypoxia causes the pulmonary arterioles to constrict potently, but the constriction is much greater in some parts of the lungs than in other parts, so that more and more of the pulmonary blood flow is forced through fewer and fewer still unconstricted pulmonary vessels. The postulated result is that the capillary pressure in these areas of the lungs becomes especially high and local edema occurs. Extension of the process to progressively more areas of the lungs leads to spreading pulmonary edema and severe pulmonary dysfunction that can be lethal. Allowing the person to breathe oxygen usually reverses the process within hours.

Chronic Mountain Sickness

Occasionally, a person who remains at high altitude too long develops *chronic mountain sickness*, in which the following effects occur: (1) the red cell mass and hematocrit become exceptionally high, (2) the pulmonary arterial pressure becomes elevated even more than the normal elevation that occurs during acclimatization, (3) the right side of the heart becomes greatly enlarged, (4) the peripheral arterial pressure begins to fall, (5) congestive heart failure ensues, and (6) death often follows unless the person is removed to a lower altitude.

The causes of this sequence of events are probably threefold: First, the red cell mass becomes so great that the blood viscosity increases severalfold; this increased viscosity tends to *decrease* tissue blood flow so that oxygen delivery also begins to decrease. Second, the pulmonary arterioles become vasoconstricted because of the lung hypoxia. This results from the hypoxic vascular constrictor effect that normally operates to divert blood flow from low-oxygen to high-oxygen alveoli, as explained in Chapter 38. But because *all* the alveoli are now in the low-oxygen state, all the arterioles become constricted, the pulmonary arterial pressure rises excessively, and the right side of the heart fails. Third, the alveolar arteriolar spasm diverts much of the blood flow through nonalveolar pulmonary vessels, thus causing an excess of pulmonary shunt blood flow where the blood is poorly oxygenated; this further compounds the problem. Most of these people recover within days or weeks when they are moved to a lower altitude.

Effects of Acceleratory Forces on the Body in Aviation and Space Physiology

Because of rapid changes in velocity and direction of motion in airplanes or spacecraft, several types of acceleratory forces affect the body during flight. At the beginning of flight, simple linear acceleration occurs; at the end of flight, deceleration; and every time the vehicle turns, centrifugal acceleration.

Centrifugal Acceleratory Forces

When an airplane makes a turn, the force of centrifugal acceleration is determined by the following relation:

$$f = \frac{mv^2}{r}$$

in which f is centrifugal acceleratory force, m is the mass of the object, v is velocity of travel, and r is radius of curvature of the turn. From this formula, it is obvious that as the velocity increases, the *force of centrifugal acceleration increases in proportion to the square of the velocity*. It is also obvious that the force of acceleration is *directly proportional to the sharpness of the turn* (*the less the radius*).

Measurement of Acceleratory Force—"G." When an aviator is simply sitting in his seat, the force with which he is pressing against the seat results from the pull of gravity and is equal to his weight. The intensity of this force is said to be +1 G because it is equal to the pull of gravity. If the force with which he presses against the seat becomes five times his normal weight during pull-out from a dive, the force acting on the seat is +5 G.

If the airplane goes through an outside loop so that the person is held down by his seat belt, *negative G* is applied to his body; if the force with which he is held down by his belt is equal to the weight of his body, the negative force is –1 G.

Effects of Centrifugal Acceleratory Force on the Body—(Positive G)

Effects on the Circulatory System. The most important effect of centrifugal acceleration is on the circulatory system, because blood is mobile and can be translocated by centrifugal forces.

When an aviator is subjected to *positive G,* blood is centrifuged toward the lowermost part of the body. Thus, if the centrifugal acceleratory force is +5 G and the person is in an immobilized standing position, the pressure in the veins of the feet becomes greatly increased (to about 450 mm Hg). In the sitting position, the pressure becomes nearly 300 mm Hg. And, as pressure in the vessels of the lower body increases, these vessels passively dilate so that a major portion of the blood from the upper body is translocated into the lower vessels. Because the heart cannot pump

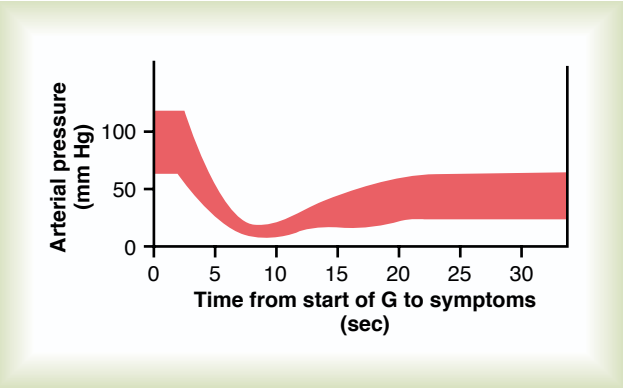

Figure 43–3

Changes in systolic (*top of curve*) and diastolic (*bottom of curve*) arterial pressures after abrupt and continuing exposure of a sitting person to an acceleratory force from top to bottom of 3.3. G. (Data from Martin EE, Henry JP: Effects of time and temperature upon tolerance to positive acceleration. J Aviation Med 22:382, 1951.)

unless blood returns to it, the greater the quantity of blood "pooled" in this way in the lower body, the less that is available for the cardiac output.

Figure 43–3 shows the changes in systolic and diastolic arterial pressures (top and bottom curves, respectively) in the upper body when a centrifugal acceleratory force of +3.3 G is suddenly applied to a sitting person. Note that both these pressures fall below 22 mm Hg for the first few seconds after the acceleration begins but then return to a systolic pressure of about 55 mm Hg and a diastolic pressure of 20 mm Hg within another 10 to 15 seconds. This secondary recovery is caused mainly by activation of the baroreceptor reflexes.

Acceleration greater than 4 to 6 G causes "blackout" of vision within a few seconds and unconsciousness shortly thereafter. If this great degree of acceleration is continued, the person will die.

Effects on the Vertebrae. Extremely high acceleratory forces for even a fraction of a second can fracture the vertebrae. The degree of positive acceleration that the average person can withstand in the sitting position before vertebral fracture occurs is about 20 G.

Negative G. The effects of negative G on the body are less dramatic acutely but possibly more damaging permanently than the effects of positive G. An aviator can usually go through outside loops up to negative acceleratory forces of –4 to –5 G without causing permanent harm, although causing intense momentary hyperemia of the head. Occasionally, psychotic disturbances lasting for 15 to 20 minutes occur as a result of brain edema.

Occasionally, negative G forces can be so great (–20 G, for instance) and centrifugation of the blood into the head is so great that the cerebral blood pressure reaches 300 to 400 mm Hg, sometimes causing small vessels on the surface of the head and in the brain to rupture. However, the vessels inside the cranium show less tendency for rupture than would be expected for the following reason: The cerebrospinal fluid is centrifuged toward the head at the same time that blood is centrifuged toward the cranial vessels, and the greatly increased pressure of the cerebrospinal fluid acts as a cushioning buffer on the outside of the brain to prevent intracerebral vascular rupture.

Because the eyes are not protected by the cranium, intense hyperemia occurs in them during strong negative G. As a result, the eyes often become temporarily blinded with "red-out."

Protection of the Body Against Centrifugal Acceleratory Forces. Specific procedures and apparatus have been developed to protect aviators against the circulatory collapse that might occur during positive G. First, if the aviator tightens his or her abdominal muscles to an extreme degree and leans forward to compress the abdomen, some of the pooling of blood in the large vessels of the abdomen can be prevented, thereby delaying the onset of blackout. Also, special "anti-G" suits have been devised to prevent pooling of blood in the lower abdomen and legs. The simplest of these applies positive pressure to the legs and abdomen by inflating compression bags as the G increases. Theoretically, a pilot submerged in a tank or suit of water might experience little effect of G forces on the circulation because the pressures developed in the water pressing on the outside of the body during centrifugal acceleration would almost exactly balance the forces acting in the body. However, the presence of air in the lungs still allows displacement of the heart, lung tissues, and diaphragm into seriously abnormal positions despite submersion in water. Therefore, even if this procedure were used, the limit of safety almost certainly would still be less than 10 G.

Effects of Linear Acceleratory Forces on the Body

Acceleratory Forces in Space Travel. Unlike an airplane, a spacecraft cannot make rapid turns; therefore, centrifugal acceleration is of little importance except when the spacecraft goes into abnormal gyrations. However, blast-off acceleration and landing deceleration can be tremendous; both of these are types of *linear acceleration*, one positive and the other negative.

Figure 43–4 shows an approximate profile of acceleration during blast-off in a three-stage spacecraft, demonstrating that the first-stage booster causes acceleration as high as 9 G, and the second-stage booster as high as 8 G. In the standing position, the human body could not withstand this much acceleration, but in a semireclining position *transverse to the axis of acceleration*, this amount of acceleration can be withstood with ease despite the fact that the acceleratory forces continue for as long as several minutes at a time. Therefore, we see the reason for the reclining seats used by astronauts.

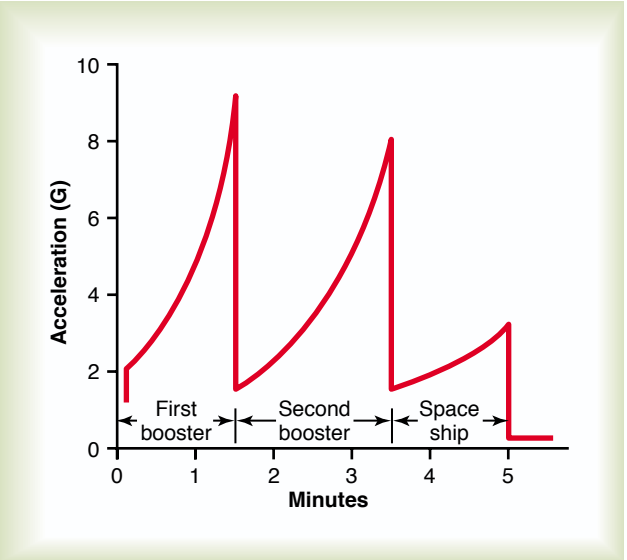

Figure 43–4

Acceleratory forces during takeoff of a spacecraft.

Problems also occur during deceleration when the spacecraft re-enters the atmosphere. A person traveling at Mach 1 (the speed of sound and of fast airplanes) can be safely decelerated in a distance of about 0.12 mile, whereas a person traveling at a speed of Mach 100 (a speed possible in interplanetary space travel) would require a distance of about 10,000 miles for safe deceleration. The principal reason for this difference is that the total amount of energy that must be dispelled during deceleration is proportional to the *square* of the velocity, which alone increases the required distance for decelerations between Mach 1 versus Mach 100 about 10,000-fold. But in addition to this, a human being can withstand far less deceleration if the period of deceleration lasts for a long time than for a short time. Therefore, deceleration must be accomplished much more slowly from high velocities than is necessary at lower velocities.

Deceleratory Forces Associated with Parachute Jumps. When the parachuting aviator leaves the airplane, his velocity of fall is at first exactly 0 feet per second. However, because of the acceleratory force of gravity, within 1 second his velocity of fall is 32 feet per second (if there is no air resistance); in 2 seconds it is 64 feet per second; and so on. As the velocity of fall increases, the air resistance tending to slow the fall also increases. Finally, the deceleratory force of the air resistance exactly balances the acceleratory force of gravity, so that after falling for about 12 seconds, the person will be falling at a "terminal velocity" of 109 to 119 miles per hour (175 feet per second). If the parachutist has already reached terminal velocity before opening his parachute, an "opening shock load" of up to 1200 pounds can occur on the parachute shrouds.

The usual-sized parachute slows the fall of the parachutist to about one ninth the terminal velocity. In other words, the speed of landing is about 20 feet per second, and the force of impact against the earth is 1/81 the impact force without a parachute. Even so, the force of impact is still great enough to cause considerable damage to the body unless the parachutist is properly trained in landing. Actually, the force of impact with the earth is about the same as that which would be experienced by jumping without a parachute from a height of about 6 feet. Unless forewarned, the parachutist will be tricked by his senses into striking the earth with extended legs, and this will result in tremendous deceleratory forces along the skeletal axis of the body, resulting in fracture of his pelvis, vertebrae, or leg. Consequently, the trained parachutist strikes the earth with knees bent but muscles taut to cushion the shock of landing.

"Artificial Climate" in the Sealed Spacecraft

Because there is no atmosphere in outer space, an artificial atmosphere and climate must be produced in a spacecraft. Most important, the oxygen concentration must remain high enough and the carbon dioxide concentration low enough to prevent suffocation. In some earlier space missions, a capsule atmosphere containing pure oxygen at about 260 mm Hg pressure was used, but in the modern space shuttle, gases about equal to those in normal air are used, with four times as much nitrogen as oxygen and a total pressure of 760 mm Hg. The presence of nitrogen in the mixture greatly diminishes the likelihood of fire and explosion. It also protects against development of local patches of lung atelectasis that often occur when breathing pure oxygen because oxygen is absorbed rapidly when small bronchi are temporarily blocked by mucous plugs.

For space travel lasting more than several months, it is impractical to carry along an adequate oxygen supply. For this reason, recycling techniques have been proposed for use of the same oxygen over and over again. Some recycling processes depend on purely physical procedures, such as electrolysis of water to release oxygen. Others depend on biological methods, such as use of algae with their large store of chlorophyll to release oxygen from carbon dioxide by the process of photosynthesis. A completely satisfactory system for recycling has yet to be achieved.

Weightlessness in Space

A person in an orbiting satellite or a nonpropelled spacecraft experiences *weightlessness*, or a state of near-zero G force, which is sometimes called *microgravity*. That is, the person is not drawn toward the bottom, sides, or top of the spacecraft but simply floats inside its chambers. The cause of this is not failure of gravity to pull on the body, because gravity from any nearby heavenly body is still active. However, the gravity acts on both the spacecraft and the person at

the same time, so that both are pulled with exactly the same acceleratory forces and in the same direction. For this reason, the person simply is not attracted toward any specific wall of the spacecraft.

Physiologic Problems of Weightlessness (Microgravity). The physiologic problems of weightlessness have not proved to be of much significance, as long as the period of weightlessness is not too long. Most of the problems that do occur are related to three effects of the weightlessness: (1) motion sickness during the first few days of travel, (2) translocation of fluids within the body because of failure of gravity to cause normal hydrostatic pressures, and (3) diminished physical activity because no strength of muscle contraction is required to oppose the force of gravity.

Almost 50 per cent of astronauts experience motion sickness, with nausea and sometimes vomiting, during the first 2 to 5 days of space travel. This probably results from an unfamiliar pattern of motion signals arriving in the equilibrium centers of the brain, and at the same time lack of gravitational signals.

The observed effects of prolonged stay in space are the following: (1) decrease in blood volume, (2) decrease in red blood cell mass, (3) decrease in muscle strength and work capacity, (4) decrease in maximum cardiac output, and (5) loss of calcium and phosphate from the bones, as well as loss of bone mass. Most of these same effects also occur in people who lie in bed for an extended period of time. For this reason, exercise programs are carried out by astronauts during prolonged space missions.

In previous space laboratory expeditions in which the exercise program had been less vigorous, the astronauts had severely decreased work capacities for the first few days after returning to earth. They also had a tendency to faint (and still do, to some extent) when they stood up during the first day or so after return to gravity because of diminished blood volume and diminished responses of the arterial pressure control mechanisms.

Cardiovascular, Muscle, and Bone "Deconditioning" During Prolonged Exposure to Weightlessness. During very long space flights and prolonged exposure to microgravity, gradual "deconditioning" effects occur on the cardiovascular system, skeletal muscles, and bone despite rigorous exercise during the flight. Studies of astronauts on space flights lasting several months have shown that they may lose as much 1.0 percent of their bone mass each month even though they continue to exercise. Substantial atrophy of cardiac and skeletal muscles also occurs during prolonged exposure to a microgravity environment.

One of the most serious effects is cardiovascular "deconditioning", which includes decreased work capacity, reduced blood volume, impaired baroreceptor reflexes, and reduced orthostatic tolerance. These changes greatly limit the astronauts' ability to stand upright or perform normal daily activities after returning to the full gravity of Earth. Astronauts returning from space flights lasting 4 to 6 months are also susceptible to bone fractures and may require several weeks before they return to pre-flight cardiovascular, bone, and muscle fitness. As space flights become longer in preparation for possible human exploration of other planets, such as Mars, the effects of prolonged microgravity could pose a very serious threat to astronauts after they land, especially in the event of an emergency landing. Therefore, considerable research effort has been directed toward developing countermeasures, in addition to exercise, that can prevent or more effectively attenuate these changes. One such countermeasure that is being tested is the application of intermittent "artificial gravity" caused by short periods (e.g., 1 hour each day) of centrifugal acceleration of the astronauts while they sit in specially designed short-arm centrifuges that create forces of up to 2 to 3 G.

References

Adams GR, Caiozzo VJ, Baldwin KM: Skeletal muscle unweighting: spaceflight and ground-based models. J Appl Physiol 95:2185, 2003.

Alfrey CP, Udden MM, Leach-Huntoon C, et al: Control of red blood cell mass in spaceflight. J Appl Physiol 81:98, 1996.

Basnyat B, Murdoch DR: High-altitude illness. Lancet 361:1967, 2003.

Convertino VA: Mechanisms of microgravity induced orthostatic intolerance: implications for effective countermeasures. J Gravit Physiol 9:1, 2002.

Eckberg DL: Bursting into space: alterations of sympathetic control by space travel. Acta Physiol Scand 177:299, 2003.

Hackett PH, Roach RC: High-altitude illness. N Engl J Med 345:107, 2001.

Harm DL, Jennings RT, Meck JV, et al: Gender issues related to spaceflight: a NASA perspective. J Appl Physiol 91:2374, 2001.

Hochachka PW, Beatty CL, Burelle Y, et al: The lactate paradox in human high-altitude physiological performance. News Physiol Sci 17:122, 2002.

Hoschele S, Mairbaurl H: Alveolar flooding at high altitude: failure of reabsorption? News Physiol Sci 18:55, 2003.

Hultgren HN: High-altitude pulmonary edema: current concepts. Annu Rev Med 47:267, 1996.

Rupert JL, Hochachka PW: Genetic approaches to understanding human adaptation to altitude in the Andes. J Exp Biol 204(Pt 18):3151, 2001.

Smith SM, Heer M: Calcium and bone metabolism during space flight. Nutrition 18:849, 2002.

West JB: Climbing Mount Everest without oxygen. News Physiol Sci 1:25, 1986.

West JB: Man in space. News Physiol Sci 1:198, 1986.

West JB: High Life—History of High-Altitude Physiology and Medicine. Bethesda, MD: American Physiological Society, 1998.

Zhang LF: Vascular adaptation to microgravity: what have we learned? J Appl Physiol 91:2415, 2001.

Physiology of Deep-Sea Diving and Other Hyperbaric Conditions

When human beings descend beneath the sea, the pressure around them increases tremendously. To keep the lungs from collapsing, air must be supplied at very high pressure to keep them inflated. This exposes the blood in the lungs also to extremely high alveolar gas pressure, a condition called *hyperbarism*. Beyond certain limits, these high pressures can cause tremendous alterations in body physiology and can be lethal.

Relationship of Pressure to Sea Depth. A column of seawater 33 feet deep exerts the same pressure at its bottom as the pressure of the atmosphere above the sea. Therefore, a person 33 feet beneath the ocean surface is exposed to 2 atmospheres pressure, 1 atmosphere of pressure caused by the weight of the air above the water and the second atmosphere by the weight of the water itself. At 66 feet the pressure is 3 atmospheres, and so forth, in accord with the table in Figure 44–1.

Effect of Sea Depth on the Volume of Gases-Boyle's Law. Another important effect of depth is compression of gases to smaller and smaller volumes. The lower part of Figure 44–1 shows a bell jar at sea level containing 1 liter of air. At 33 feet beneath the sea, where the pressure is 2 atmospheres, the volume has been compressed to only one-half liter, and at 8 atmospheres (233 feet) to one-eighth liter. Thus, the volume to which a given quantity of gas is compressed is inversely proportional to the pressure. This is a principle of physics called *Boyle's law*, which is extremely important in diving physiology because increased pressure can collapse the air chambers of the diver's body, especially the lungs, and often causes serious damage.

Many times in this chapter it is necessary to refer to *actual volume* versus *sea-level volume*. For instance, we might speak of an actual volume of 1 liter at a depth of 300 feet; this is the same *quantity* of air as a sea-level volume of 10 liters.

Effect of High Partial Pressures of Individual Gases on the Body

The individual gases to which a diver is exposed when breathing air are *nitrogen*, *oxygen*, and *carbon dioxide;* each of these at times can cause significant physiologic effects at high pressures.

Nitrogen Narcosis at High Nitrogen Pressures

About four fifths of the air is nitrogen. At sea-level pressure, the nitrogen has no significant effect on bodily function, but at high pressures it can cause varying degrees of narcosis. When the diver remains beneath the sea for an hour or more and is breathing compressed air, the depth at which the first symptoms of mild narcosis appear is about 120 feet. At this level the diver begins to exhibit

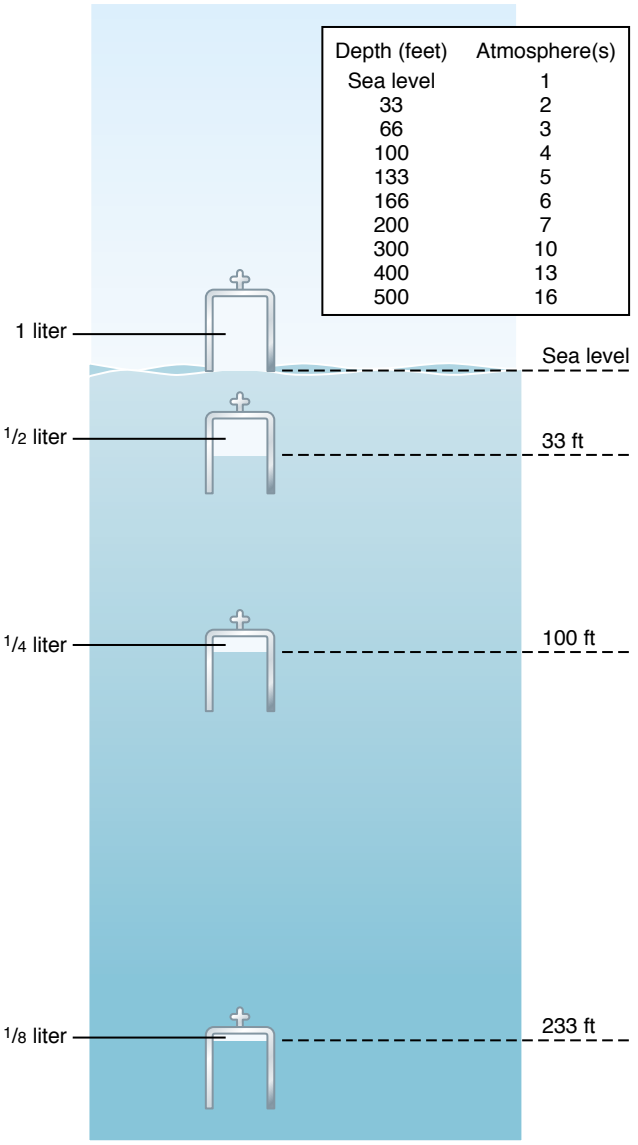

Figure 44–1

Effect of sea depth on pressure (*top table*) and on gas volume (*bottom*).

joviality and to lose many of his or her cares. At 150 to 200 feet, the diver becomes drowsy. At 200 to 250 feet, his or her strength wanes considerably, and the diver often becomes too clumsy to perform the work required. Beyond 250 feet (8.5 atmospheres pressure), the diver usually becomes almost useless as a result of nitrogen narcosis if he or she remains at these depths too long.

Nitrogen narcosis has characteristics similar to those of alcohol intoxication, and for this reason it has frequently been called "raptures of the depths." The mechanism of the narcotic effect is believed to be the same as that of most other gas anesthetics. That is, it dissolves in the fatty substances in neuronal membranes and, because of its *physical* effect on altering ionic conductance through the membranes, reduces neuronal excitability.

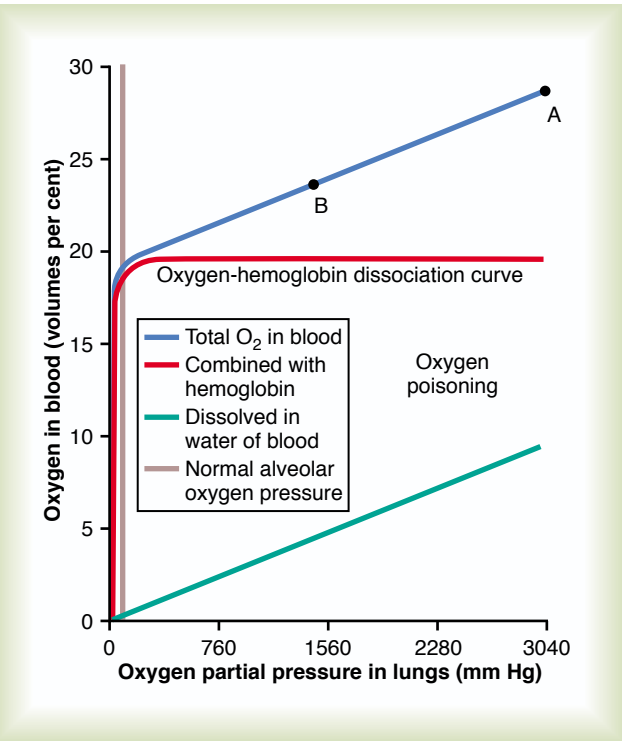

Figure 44–2

Quantity of oxygen dissolved in the fluid of the blood and in combination with hemoglobin at very high P_{O_2}s.

Oxygen Toxicity at High Pressures

Effect of Very High P_{O_2} on Blood Oxygen Transport. When the P_{O_2} in the blood rises above 100 mm Hg, the amount of oxygen dissolved in the water of the blood increases markedly. This is shown in Figure 44–2, which depicts the same oxygen-hemoglobin dissociation curve as that shown in Chapter 40 but with the alveolar P_{O_2} extended to more than 3000 mm Hg. Also depicted by the lowest curve in the figure is the *volume of oxygen dissolved in the fluid of the blood* at each P_{O_2} level. Note that in the normal range of alveolar P_{O_2} (below 120 mm Hg), almost none of the total oxygen in the blood is accounted for by dissolved oxygen, but as the oxygen pressure rises into the thousands of millimeters of mercury, a large portion of the total oxygen is then dissolved in the water of the blood, in addition to that bound with hemoglobin.

Effect of High Alveolar P_{O_2} on Tissue P_{O_2}. Let us assume that the P_{O_2} in the lungs is about 3000 mm Hg (4 atmospheres pressure). Referring to Figure 44–2, one finds that this represents a total oxygen content in each 100 milliliters of blood of about 29 volumes per cent, as demonstrated by point A in the figure—this means 20 volumes per cent bound with hemoglobin and 9 volumes per cent dissolved in the blood water. As this blood passes through the tissue capillaries and the tissues use their normal amount of oxygen, about 5 milliliters from each 100 milliliters of blood, the

oxygen content on leaving the tissue capillaries is still 24 volumes per cent (point B in the figure). At this point, the P_{O_2} is approximately 1200 mm Hg, which means that oxygen is delivered to the tissues at this extremely high pressure instead of at the normal value of 40 mm Hg. Thus, once the alveolar P_{O_2} rises above a critical level, the hemoglobin-oxygen buffer mechanism (discussed in Chapter 40) is no longer capable of keeping the tissue P_{O_2} in the normal, safe range between 20 and 60 mm Hg.

Acute Oxygen Poisoning. The extremely high tissue P_{O_2} that occurs when oxygen is breathed at very high alveolar oxygen pressure can be detrimental to many of the body's tissues. For instance, breathing oxygen at 4 atmospheres pressure of oxygen ($P_{O_2} = 3040$ mm Hg) will cause brain *seizures followed by coma* in most people within 30 to 60 minutes. The seizures often occur without warning and, for obvious reasons, are likely to be lethal to divers submerged beneath the sea.

Other symptoms encountered in acute oxygen poisoning include nausea, muscle twitchings, dizziness, disturbances of vision, irritability, and disorientation. Exercise greatly increases the diver's susceptibility to oxygen toxicity, causing symptoms to appear much earlier and with far greater severity than in the resting person.

Excessive Intracellular Oxidation as a Cause of Nervous System Oxygen Toxicity—"Oxidizing Free Radicals." Molecular oxygen (O_2) has little capability of oxidizing other chemical compounds. Instead, it must first be converted into an "active" form of oxygen. There are several forms of active oxygen called *oxygen free radicals*. One of the most important of these is the superoxide free radical O_2^-, and another is the peroxide radical in the form of hydrogen peroxide. Even when the tissue P_{O_2} is normal at the level of 40 mm Hg, small amounts of free radicals are continually being formed from the dissolved molecular oxygen. Fortunately, the tissues also contain multiple enzymes that rapidly remove these free radicals, including *peroxidases*, *catalases*, and *superoxide dismutases*. Therefore, so long as the hemoglobin-oxygen buffering mechanism maintains a normal tissue P_{O_2}, the oxidizing free radicals are removed rapidly enough that they have little or no effect in the tissues.

Above a critical alveolar P_{O_2} (above about 2 atmospheres P_{O_2}), the hemoglobin-oxygen buffering mechanism fails, and the tissue P_{O_2} can then rise to hundreds or thousands of millimeters of mercury. At these high levels, the amounts of oxidizing free radicals literally swamp the enzyme systems designed to remove them, and now they can have serious destructive and even lethal effects on the cells. One of the principal effects is to oxidize the polyunsaturated fatty acids that are essential components of many of the cell membranes. Another effect is to oxidize some of the cellular enzymes, thus damaging severely the cellular metabolic systems. The nervous tissues are especially susceptible because of their high lipid content. Therefore, most of the acute lethal effects of acute oxygen toxicity are caused by brain dysfunction.

Chronic Oxygen Poisoning Causes Pulmonary Disability. A person can be exposed to only 1 atmosphere pressure of oxygen almost indefinitely without developing the *acute* oxygen toxicity of the nervous system just described. However, after only about 12 hours of 1 atmosphere oxygen exposure, *lung passageway congestion*, *pulmonary edema*, and *atelectasis* caused by damage to the linings of the bronchi and alveoli begin to develop. The reason for this effect in the lungs but not in other tissues is that the air spaces of the lungs are directly exposed to the high oxygen pressure, but oxygen is delivered to the other body tissues at almost normal P_{O_2} because of the hemoglobin-oxygen buffer system.

Carbon Dioxide Toxicity at Great Depths in the Sea

If the diving gear is properly designed and functions properly, the diver has no problem due to carbon dioxide toxicity because depth alone does not increase the carbon dioxide partial pressure in the alveoli. This is true because depth does not increase the rate of carbon dioxide production in the body, and as long as the diver continues to breathe a normal tidal volume and expires the carbon dioxide as it is formed, alveolar carbon dioxide pressure will be maintained at a normal value.

In certain types of diving gear, however, such as the diving helmet and some types of rebreathing apparatuses, carbon dioxide can build up in the dead space air of the apparatus and be rebreathed by the diver. Up to an alveolar carbon dioxide pressure (P_{CO_2}) of about 80 mm Hg, twice that in normal alveoli, the diver usually tolerates this buildup by increasing the minute respiratory volume a maximum of 8- to 11-fold to compensate for the increased carbon dioxide. Beyond 80-mm Hg alveolar P_{CO_2}, the situation becomes intolerable, and eventually the respiratory center begins to be depressed, rather than excited, because of the negative tissue metabolic effects of high P_{CO_2}. The diver's respiration then begins to fail rather than to compensate. In addition, the diver develops severe respiratory acidosis, and varying degrees of lethargy, narcosis, and finally even anesthesia, as discussed in Chapter 42.

Decompression of the Diver After Excess Exposure to High Pressure

When a person breathes air under high pressure for a long time, the amount of nitrogen dissolved in the body fluids increases. The reason for this is the following: Blood flowing through the pulmonary capillaries becomes saturated with nitrogen to the same high pressure as that in the alveolar breathing mixture. And over several more hours, enough nitrogen is

carried to all the tissues of the body to raise their tissue P_{N_2} also to equal the P_{N_2} in the breathing air.

Because nitrogen is not metabolized by the body, it remains dissolved in all the body tissues until the nitrogen pressure in the lungs is decreased back to some lower level, at which time the nitrogen can be removed by the reverse respiratory process; however, this removal often takes hours to occur and is the source of multiple problems collectively called *decompression sickness*.

Volume of Nitrogen Dissolved in the Body Fluids at Different Depths. At sea level, almost exactly 1 liter of nitrogen is dissolved in the entire body. Slightly less than one half of this is dissolved in the water of the body and a little more than one half in the fat of the body. This is true because nitrogen is five times as soluble in fat as in water.

After the diver has become saturated with nitrogen, the *sea-level volume of nitrogen* dissolved in the body at different depths is as follows:

Feet	Liters
0	1
33	2
100	4
200	7
300	10

Several hours are required for the gas pressures of nitrogen in all the body tissues to come nearly to equilibrium with the gas pressure of nitrogen in the alveoli. The reason for this is that the blood does not flow rapidly enough and the nitrogen does not diffuse rapidly enough to cause instantaneous equilibrium. The nitrogen dissolved in the water of the body comes to almost complete equilibrium in less than 1 hour, but the fat tissue, requiring five times as much transport of nitrogen and having a relatively poor blood supply, reaches equilibrium only after several hours. For this reason, if a person remains at deep levels for only a few minutes, not much nitrogen dissolves in the body fluids and tissues, whereas if the person remains at a deep level for several hours, both the body water and body fat become saturated with nitrogen.

Decompression Sickness (Synonyms: Bends, Compressed Air Sickness, Caisson Disease, Diver's Paralysis, Dysbarism). If a diver has been beneath the sea long enough that large amounts of nitrogen have dissolved in his or her body and the diver then suddenly comes back to the surface of the sea, significant quantities of nitrogen bubbles can develop in the body fluids either intracellularly or extracellularly and can cause minor or serious damage in almost any area of the body, depending on the number and sizes of bubbles formed; this is called *decompression sickness*.

The principles underlying bubble formation are shown in Figure 44–3. In Figure 44–3*A*, the diver's tissues have become equilibrated to a high *dissolved nitrogen pressure* (P_{N_2} = 3918 mm Hg), about 6.5 times the normal amount of nitrogen in the tissues. As long as the diver remains deep beneath the sea, the pressure against the outside of his or her body (5000 mm Hg) compresses all the body tissues sufficiently to keep the excess nitrogen gas dissolved. But when the diver suddenly rises to sea level (Figure 44–3*B*), the pressure on the outside of the body becomes only 1 atmosphere (760 mm Hg), while the gas pressure inside the body fluids is the sum of the pressures of water vapor, carbon dioxide, oxygen, and nitrogen, or a total of 4065 mm Hg, 97 per cent of which is caused by the nitrogen. Obviously, this total value of 4065 mm Hg is far greater than the 760 mm Hg pressure on the outside of the body. Therefore, the gases can escape from the dissolved state and form actual bubbles, composed almost entirely of nitrogen, both in the tissues and in the blood where they plug many small blood vessels. The bubbles may not appear for many minutes to hours, because sometimes the gases can remain dissolved in the "supersaturated" state for hours before bubbling.

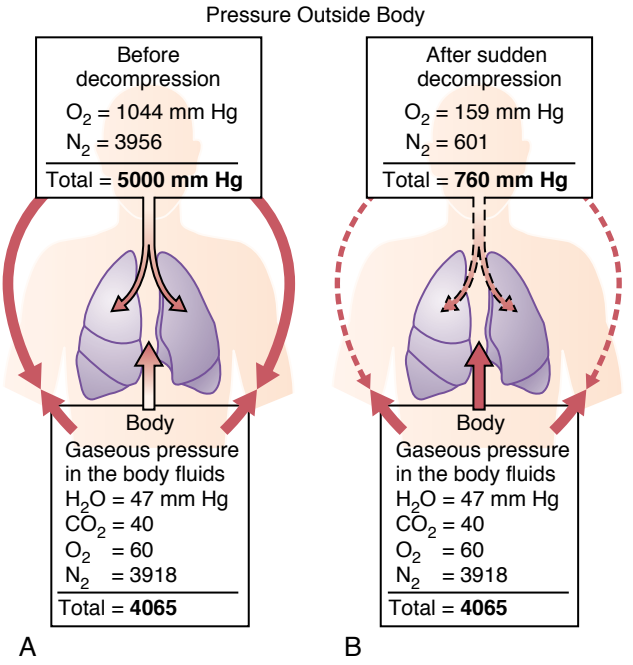

Figure 44–3

Gaseous pressures both inside and outside the body, showing (*A*) saturation of the body to high gas pressures when breathing air at a total pressure of 5000 mm Hg, and (*B*) the great excesses of intra-body pressures that are responsible for bubble formation in the tissues when the lung intra-alveolar pressure body is suddenly returned from 5000 mm Hg to normal pressure of 760 mm Hg.

Symptoms of Decompression Sickness ("Bends"). The symptoms of decompression sickness are caused by gas bubbles blocking many blood vessels in different tissues. At first, only the smallest vessels are blocked by minute bubbles, but as the bubbles coalesce, progressively larger vessels are affected.

Tissue ischemia and sometimes tissue death are the result.

In most people with decompression sickness, the symptoms are pain in the joints and muscles of the legs and arms, affecting 85 to 90 per cent of those persons who develop decompression sickness. The joint pain accounts for the term "bends" that is often applied to this condition.

In 5 to 10 per cent of people with decompression sickness, nervous system symptoms occur, ranging from dizziness in about 5 per cent to paralysis or collapse and unconsciousness in as many as 3 per cent. The paralysis may be temporary, but in some instances, damage is permanent.

Finally, about 2 per cent of people with decompression sickness develop "the chokes," caused by massive numbers of microbubbles plugging the capillaries of the lungs; this is characterized by serious shortness of breath, often followed by severe pulmonary edema and, occasionally, death.

Nitrogen Elimination from the Body; Decompression Tables. If a diver is brought to the surface slowly, enough of the dissolved nitrogen can usually be eliminated by expiration through the lungs to prevent decompression sickness. About two thirds of the total nitrogen is liberated in 1 hour and about 90 per cent in 6 hours.

Decompression tables have been prepared by the U.S. Navy that detail procedures for safe decompression. To give the student an idea of the decompression process, a diver who has been breathing air and has been on the sea bottom for 60 minutes at a depth of 190 feet is decompressed according to the following schedule:

10 minutes at 50 feet depth
17 minutes at 40 feet depth
19 minutes at 30 feet depth
50 minutes at 20 feet depth
84 minutes at 10 feet depth

Thus, for a work period on the bottom of only 1 hour, the total time for decompression is about 3 hours.

Tank Decompression and Treatment of Decompression Sickness. Another procedure widely used for decompression of professional divers is to put the diver into a pressurized tank and then to lower the pressure gradually back to normal atmospheric pressure, using essentially the same time schedule as noted above.

Tank decompression is even more important for treating people in whom symptoms of decompression sickness develop minutes or even hours after they have returned to the surface. In this case, the diver is recompressed immediately to a deep level. Then decompression is carried out over a period several times as long as the usual decompression period.

"Saturation Diving" and Use of Helium-Oxygen Mixtures in Deep Dives. When divers must work at very deep levels—between 250 feet and nearly 1000 feet—they frequently live in a large compression tank for days or weeks at a time, remaining compressed at a pressure level near that at which they will be working. This keeps the tissues and fluids of the body saturated with the gases to which they will be exposed while diving. Then, when they return to the same tank after working, there are no significant changes in pressure, so that decompression bubbles do not occur.

In very deep dives, especially during saturation diving, helium is usually used in the gas mixture instead of nitrogen for three reasons: (1) it has only about one fifth the narcotic effect of nitrogen; (2) only about one half as much volume of helium dissolves in the body tissues as nitrogen, and the volume that does dissolve diffuses out of the tissues during decompression several times as rapidly as does nitrogen, thus reducing the problem of decompression sickness; and (3) the low density of helium (one seventh the density of nitrogen) keeps the airway resistance for breathing at a minimum, which is very important because highly compressed nitrogen is so dense that airway resistance can become extreme, sometimes making the work of breathing beyond endurance.

Finally, in very deep dives it is important to reduce the oxygen concentration in the gaseous mixture because otherwise oxygen toxicity would result. For instance, at a depth of 700 feet (22 atmospheres of pressure), a 1 per cent oxygen mixture will provide all the oxygen required by the diver, whereas a 21 per cent mixture of oxygen (the percentage in air) delivers a Po_2 to the lungs of more than 4 atmospheres, a level very likely to cause seizures in as little as 30 minutes.

Scuba (Self-Contained Underwater Breathing Apparatus) Diving

Before the 1940s, almost all diving was done using a diving helmet connected to a hose through which air was pumped to the diver from the surface. Then, in 1943, Jacques Cousteau popularized a *self-contained underwater breathing apparatus*, known as the SCUBA apparatus. The type of SCUBA apparatus used in more than 99 per cent of all sports and commercial diving is the *open-circuit demand system* shown in Figure 44–4. This system consists of the following components: (1) one or more tanks of compressed air or some other breathing mixture, (2) a first-stage "reducing" valve for reducing the very high pressure from the tanks to a low pressure level, (3) a combination inhalation "demand" valve and exhalation valve that allows air to be pulled into the lungs with slight negative pressure of breathing and then to be exhaled into the sea at a pressure level slightly positive to the surrounding water pressure, and (4) a mask and tube system with small "dead space."

The demand system operates as follows: The first-stage reducing valve reduces the pressure from the tanks so that the air delivered to the mask has a pressure only a few mm Hg greater than the surrounding water pressure. The breathing mixture does not flow continually into the mask. Instead, with each

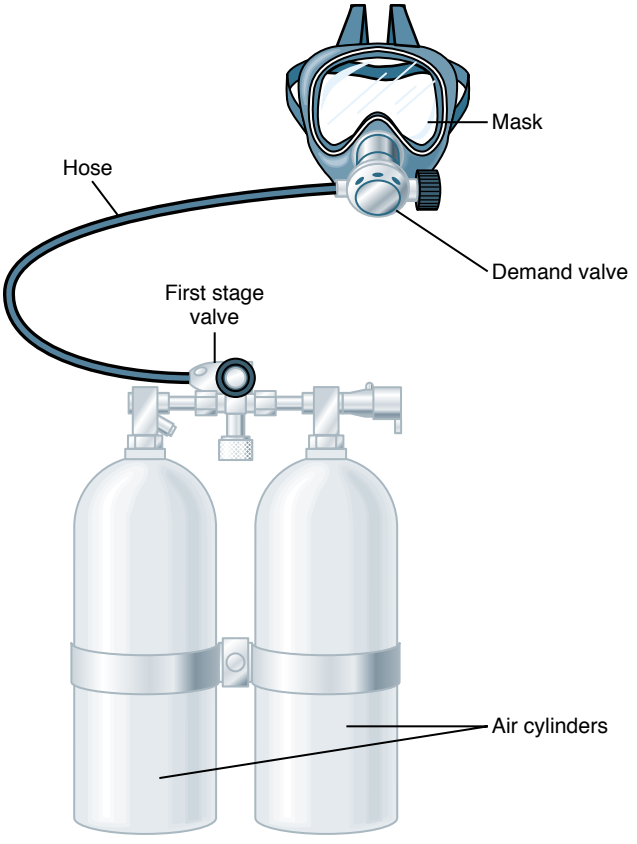

Figure 44–4

Open-circuit demand type of SCUBA apparatus.

inspiration, slight extra negative pressure in the demand valve of the mask pulls the diaphragm of the valve open, and this automatically releases air from the tank into the mask and lungs. In this way, only the amount of air needed for inhalation enters the mask. Then, on expiration, the air cannot go back into the tank but instead is expired into the sea.

The most important problem in use of the self-contained underwater breathing apparatus is the limited amount of time one can remain beneath the sea surface; for instance, only a few minutes are possible at a 200-foot depth. The reason for this is that tremendous airflow from the tanks is required to wash carbon dioxide out of the lungs—the greater the depth, the greater the airflow in terms of *quantity* of air per minute that is required, because the *volumes* have been compressed to small sizes.

Special Physiologic Problems in Submarines

Escape from Submarines. Essentially the same problems encountered in deep-sea diving are often met in relation to submarines, especially when it is necessary to escape from a submerged submarine. Escape is possible from as deep as 300 feet without using any apparatus. However, proper use of rebreathing devices, especially when using helium, theoretically can allow escape from as deep as 600 feet or perhaps more.

One of the major problems of escape is prevention of air embolism. As the person ascends, the gases in the lungs expand and sometimes rupture a pulmonary blood vessel, forcing the gases to enter the vessel and cause air embolism of the circulation. Therefore, as the person ascends, he or she must make a special effort to exhale continually.

Health Problems in the Submarine Internal Environment. Except for escape, submarine medicine generally centers around several engineering problems to keep hazards out of the internal environment. First, in atomic submarines, there exists the problem of radiation hazards, but with appropriate shielding, the amount of radiation received by the crew submerged beneath the sea has been less than normal radiation received above the surface of the sea from cosmic rays.

Second, poisonous gases on occasion escape into the atmosphere of the submarine and must be controlled rapidly. For instance, during several weeks' submergence, cigarette smoking by the crew can liberate enough carbon monoxide, if not removed rapidly, to cause carbon monoxide poisoning. And, on occasion, even freon gas has been found to diffuse out of refrigeration systems in sufficient quantity to cause toxicity.

Hyperbaric Oxygen Therapy

The intense oxidizing properties of high-pressure oxygen (*hyperbaric oxygen*) can have valuable therapeutic effects in several important clinical conditions. Therefore, large pressure tanks are now available in many medical centers into which patients can be placed and treated with hyperbaric oxygen. The oxygen is usually administered at Po_2s of 2 to 3 atmospheres of pressure through a mask or intratracheal tube, whereas the gas around the body is normal air compressed to the same high-pressure level.

It is believed that the same oxidizing free radicals responsible for oxygen toxicity are also responsible for at least some of the therapeutic benefits. Some of the conditions in which hyperbaric oxygen therapy has been especially beneficial follow.

Probably the most successful use of hyperbaric oxygen has been for treatment of *gas gangrene*. The bacteria that cause this condition, *clostridial organisms*, grow best under anaerobic conditions and stop growing at oxygen pressures greater than about 70 mm Hg. Therefore, hyperbaric oxygenation of the tissues can frequently stop the infectious process entirely and thus convert a condition that formerly was almost 100 per cent fatal into one that is cured in most instances by early treatment with hyperbaric therapy.

Other conditions in which hyperbaric oxygen therapy has been either valuable or possibly valuable include decompression sickness, arterial gas embolism,

carbon monoxide poisoning, osteomyelitis, and myocardial infarction.

References

Butler PJ: Diving beyond the limits. News Physiol Sci 16:222, 2001.

Kooyman GL, Ponganis PJ: The physiological basis of diving to depth: birds and mammals. Annu Rev Physiol 60:19, 1998.

Leach RM, Rees PJ, Wilmshurst P: Hyperbaric oxygen therapy. BMJ 317:1140, 1998.

Neuman TS: Arterial gas embolism and decompression sickness. News Physiol Sci 17:77, 2002.

Nilsson GE: Surviving anoxia with the brain turned on. News Physiol Sci 16:217, 2001.

Russi EW: Diving and the risk of barotrauma. Thorax 53(Suppl 2):S20, 1998.

Wang C, Schwaitzberg S, Berliner E, et al: Hyperbaric oxygen for treating wounds: a systematic review of the literature. Arch Surg 138:272, 2003.

Wang J, Li F, Calhoun JH, Mader JT: The role and effectiveness of adjunctive hyperbaric oxygen therapy in the management of musculoskeletal disorders. J Postgrad Med 48:226, 2002

West JB, Fu Z, Gaeth AP, Short RV: Fetal lung development in the elephant reflects the adaptations required for snorkeling in adult life. Respir Physiol Neurobiol 138:325, 2003.