Chapter 32

PRESSURE CHANGES AND HYPOXIA IN AVIATION

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INTRODUCTION

The physiological consequences of rapid ascent to high altitude are a core problem in the field of aerospace medicine. Those who live and work in mountain terrain experience a limited range of altitudes and have time to adapt to the hypoxia experienced at high terrestrial elevations. In contrast, flyers may be exposed to abrupt changes in barometric pressure and to acute, life-threatening hypoxia (see also Chapter 28, Introduction to Special Environments).

In 1875, a landmark balloon flight to 28,820 ft ended in tragedy when the three young French aeronauts on board failed to use their supplemental oxygen effectively; Tissandier survived, but his colleagues, Sivel and Crocé-Spineli, became the first known fatalities due to in-flight hypoxia. In early aviation, too, the lack of oxygen took a regular toll of both lives and aircraft; many military crewmembers were killed by hypoxia, and the performance of many more was significantly impaired in flight. Historic ascents over many years demonstrated that even with inhalation of 100% oxygen, unpressurized flight above 42,000 ft was impractical because of the effects of hypoxia and extreme cold. It was found that humans cannot adapt to hypoxia in flight but must instead be provided with life-support systems that (*a*) maintain physiological normoxia under routine operating conditions and (*b*) protect from significant hypoxia in emergencies.

Pioneering research by physicians, physiologists, and life-support engineers has established reliable techniques for safe flight at high altitudes, as demonstrated by current atmospheric flight in all its forms, military and civilian, from balloon flights to sail planes to supersonic aircraft and spacecraft. Although reliable cabin pressurization and oxygen delivery systems have greatly reduced incidents and accidents due to hypoxia in flight, constant vigilance is required for their prevention.

The aims of this chapter are, therefore, to present a distillation of our current comprehension of the physics and physiology of rapid ascent to high altitude and to describe the technology required to support human existence in that most hostile of environments. To this end, the chapter briefly characterizes the atmosphere within which flight takes place, then describes normal physiological responses to acute hypobaria and hypoxia, and finally describes the life-support systems that enable flyers to operate safely at high altitudes.

Military requirements drove much of the early research in aviation physiology as well as the development of robust life-support systems. Prominent centers for aviation altitude research included the US Air Force School of Aerospace Medicine, San Antonio, Texas; the now-defunct Royal Air Force Institute of Aviation Medicine, Farnborough, Hampshire, England; and various universities whose hypobaric chambers were supported with military research funds.

THE ATMOSPHERE

Earth's atmosphere is vital to our existence: it provides a moderate temperature environment at the surface, a protective barrier against the effects of radiation, and the oxygen needed for the release of biological energy. Flyers depart from the safety of this surface cocoon at their peril.

The physical characteristics of the atmosphere are a complex product of solar heating, ionizing radiation, and ozone formation (Figure 32-1). The upper atmosphere reflects some solar radiation and absorbs the rest, re-radiating infrared energy into the lower atmosphere and thence to Earth's surface. This "greenhouse" effect ensures that Earth's surface is warmer than it would be if it received only direct solar heating.

High-energy particulate material (cosmic radiation) continuously bombards the atmosphere. This primary ionizing radiation collides with atoms in the upper atmosphere at high velocity to create secondary radiation. Fortunately, the atmosphere further reduces the level of this radiation so that it has little effect on life at the surface, but high-altitude flight can produce significant cumulative radiation exposure for flyers.

Structure

The outer limit of the atmosphere is determined by two opposing factors: solar heating tends to expand gases from the outer atmosphere into the surrounding vacuum of space, while gravity tends to pull the gases toward Earth's surface. The structure of the atmosphere is conventionally described in terms of several concentric shells with differing thermal profiles and other characteristics:

- The *troposphere* (0–40,000 ft) is characterized by a steady decrease in temperature with altitude, the presence of varying amounts of water vapor, and the occurrence of large-scale turbulence (weather). This is the realm of most conventional aviation, including

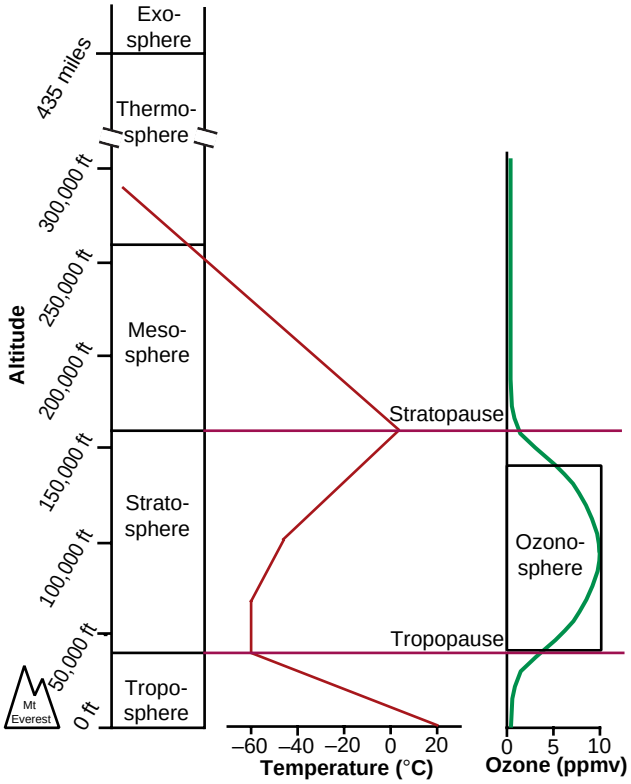

Fig. 32-1. The structure of the atmosphere and the relation of altitude to temperature and ozone concentration. Note that at the top of Mount Everest, the highest point on Earth (29,028 ft), the temperature is about –40°C and ozone concentration is about 1 ppmv. For the next 125,000 ft the temperature becomes more moderate while ozone becomes more, then again less, concentrated. The temperature at the top of Mount Everest, –40°C, is not reached again until about 100,000 ft. Adapted with permission from Harding RM. The Earth's atmosphere. In: Ernsting J, King P. *Aviation Medicine.* 2nd ed. London, England: Butterworths; 1988: 5.

balloon flights.

- The *stratosphere* (40,000–160,000 ft) includes a lower *isothermal* layer, above which temperature increases with altitude. Ozone is formed in the stratosphere, and flight in this region is generally limited to military aircraft.
- The *mesosphere* (160,000–260,000 ft) exhibits a rapid decline in temperature with altitude.
- The *thermosphere*, also called the *ionosphere* (260,000 ft to ~ 435 mi [700 km, the outer limit of the atmosphere]), exhibits extreme temperatures, which vary with solar activity.
- The *exosphere* (≥ 435 mi), where the temperature approaches absolute zero, extends beyond the atmosphere to the vacuum of space.

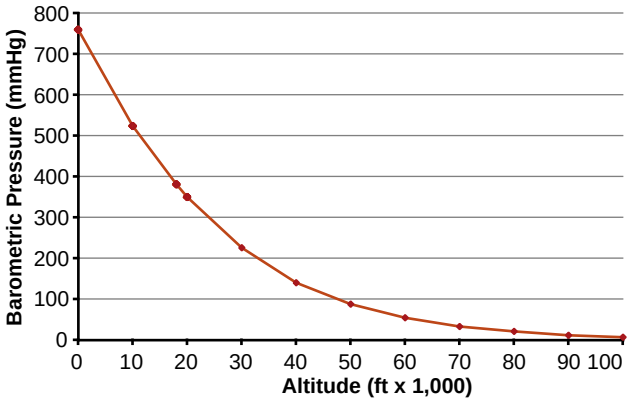

Fig. 32-2. The exponential relation of barometric pressure to altitude. Note that at 18,000 ft (380 mm Hg), barometric pressure is half that at sea level.

The pressure exerted by the atmosphere, termed *barometric pressure* (P_B), falls exponentially with altitude (Figure 32-2), producing a proportional decrease in the partial pressures of oxygen and other constituent gases. The International Civil Aviation Organization's (ICAO's) Standard Atmosphere is an agreed description of the relation among P_B, temperature, and altitude at a latitude of 45° north (Table 32-1).[1] This description forms the basis for calibration of pressure-measuring flight instruments and allows precise comparisons between the performances of different aircraft and aircraft systems. The measurement of physiological variables such as volume and mass of gas, gas flow, and metabolic rate, as well as the related specifications for life-support systems, are profoundly affected by changes in body temperature, P_B, and saturation of water vapor in the lungs. The conditions of measuring physiological variables at altitude are summarized in Exhibit 32-1. (Related information on the larger changes in P_B associated with diving is found in Chapter 30, Physics, Physiology, and Medicine of Diving.)

Composition

The atmosphere is made up of a remarkably constant mixture of nitrogen and oxygen, with traces of other gases (Table 32-2). In addition, the lower troposphere may contain significant amounts of carbon dioxide and toxic gases, reflecting human activity and natural phenomena such as volcanic eruptions. It may also contain increased quantities of water vapor, de-

TABLE 32–1

INTERNATIONAL CIVIL AVIATION ORGANIZATION STANDARD ATMOSPHERE

Altitude (ft)	Altitude (m)	Pressure (mm Hg)	Temperature (°C)	Altitude (ft)	Altitude (m)	Pressure (mm Hg)	Temperature (°C)
0	0	760	+15.0	25,000	7,620	282	–34.5
1,000	305	733	+13.0	30,000	9,144	226	–44.4
2,000	610	706	+11.0	35,000	10,668	179	–54.2
3,000	914	681	+9.1	40,000	12,192	141	–56.5
4,000	1,219	656	+7.1	45,000	13,716	111	–56.5
5,000	1,525	632	+5.1	50,000	15,240	87.3	–56.5
6,000	1,829	609	+3.1	55,000	16,764	68.8	–56.5
7,000	2,134	586	+1.1	60,000	18,288	54.1	–56.5
8,000	2,438	565	–0.9	65,000	19,812	42.3	–56.5
9,000	2,743	543	–2.8	70,000	21,336	33.3	–55.2
10,000	3,048	523	–4.8	80,000	24,384	20.7	–52.1
15,000	4,572	429	–14.7	90,000	27,432	13.0	–49.1
20,000	6,096	349	–24.6	100,000	30,480	8.2	–46.0

Adapted with permission from International Civil Aviation Organization. *Manual of the ICAO Standard Atmosphere*. 2nd ed. Montreal, Quebec, Canada: ICAO; 1964.

pending on the temperature of the air mass and whether it has recently passed over water.

Ozone is the highly reactive, triatomic form of oxygen that may adversely affect the respiratory tract[2]; its formation and destruction in the atmosphere are therefore of great physical and biological importance. Ozone forms in the stratosphere when molecular oxygen absorbs ultraviolet radiation, a process that greatly reduces harmful ultraviolet radiation at lower altitudes. Ozone concentration reaches approximately 10.0 parts per million by volume (ppmv) at 100,000 ft but falls to less than 1.0 ppmv at altitudes below 40,000 ft and to about 0.03 ppmv at sea level (see Figure 32-1).

THE PHYSIOLOGICAL CONSEQUENCES OF RAPID ASCENT TO ALTITUDE

The physiological consequences associated with the physical changes in the atmosphere seen on rapid ascent to altitude include hypoxia and hyperventilation, as a result of reduction in the partial pressure of oxygen (P_{O_2}); barotrauma and the decompression illnesses, as a result of reduction in total pressure; and thermal injury, as a result of decreased temperature (Figure 32-3). Cold (thermal) injury is discussed in *Medical Consequences of Harsh Environments, Volume 1*[3]; the remaining potential problems are addressed below. To a large extent, our understanding of these problems has only been made possible with the help of experimental hypobaric chambers in military and university laboratories worldwide.

Hypoxia

Oxygen is one of the most important requirements for the maintenance of normal function by living systems, as energy for biological processes is generated by the oxidation of complex chemical foodstuffs into simpler compounds, usually with the eventual formation of carbon dioxide, water, and other waste products. Human beings are extremely vulnerable and sensitive to the effects of oxygen lack, and severe deprivation leads to a rapid deterioration of most bodily functions. If the situation persists, death is inevitable. Not without reason is hypoxia generally held to be the most serious single physiological hazard encoun-

EXHIBIT 32-1

VOLUME, TEMPERATURE, PRESSURE, AND WATER VAPOR IN RESPIRATORY MEASUREMENT

BTPS. Gas in the lungs is said to be at *b*ody *t*emperature and *p*ressure, *s*aturated (BTPS). Body temperature is usually regarded as constant at 37°C; water vapor at that temperature reaches a pressure of 47 mm Hg at saturation.

ATPS. Ambient air is usually cooler and dryer than gas in the lungs and is designated as *a*mbient *t*emperature and *p*ressure (ATP). If respiratory volumes are measured using a water spirometer, calculations are made from ambient temperature and pressure, saturated (ATPS), where water vapor pressure is calculated as the saturation value at the temperature of the spirometer.

Conversion from ATPS to BTPS can be expressed mathematically:

$$V_{BTPS} = V_{ATPS} \bullet \frac{273 + 37}{273 + T_{db}} \bullet \frac{P_B - P_{H_2O}}{P_B - 47}.$$

where V represents volume; 273 = melting point of ice, expressed in °K; 37 = body temperature, expressed in °C; T_{db} represents ambient dry bulb temperature, expressed in °C; P_B represents barometric pressure, expressed in mm Hg; P_{H_2O} represents saturated water vapor pressure at T_{db}; and $47 = P_{H_2O}$ at body temperature.

STPD. Metabolic calculations require knowledge of the number of molecules (ie, the mass) of oxygen used and carbon dioxide produced. For this purpose, the gas volumes are expressed as standard temperature and pressure, dry (STPD), where the standard temperature is 273°K (O°C) and the standard pressure is 760 mm Hg (1 atm). Under these conditions, gases comply with Avogadro's law (ie, 1 g-mol of a gas has a volume of 22.4 L_{STPD}), so that the number of molecules contained within the STPD volume can readily be calculated.

Likewise, conversion from ATPS to STPD can be expressed mathematically:

$$V_{STDP} = V_{ATPS} \bullet \frac{273}{273 + T_{db}} \bullet \frac{P_B - P_{H_2O}}{760}.$$

Other Conditions. Specifications for life-support systems may quote gas volumes as atmospheric temperature and pressure, dry (ATPD); and gas consumption figures may be quoted as normal temperature and pressure (NTP). The need to express gas quantities under NTP conditions arises because gases expand on exposure to low barometric pressure, thereby altering the relationship between volume flow and mass flow of a gas. For example, at an altitude of 18,000 ft (0.5 atm), a mass flow of 5.0 L_{NTP}/min will provide a volume flow of about 10.0 L_{ATPD}/min. Because respiration is a volume-flow phenomenon, mass flow versus volume flow has particular relevance for respiratory physiology at altitude.

tered during flight at altitude.

The absence of an adequate supply of oxygen to the tissues, whether in mass or in molecular concentration, is termed *hypoxia* and may be defined in several ways. In aerospace medicine, the concern is with *hypoxic* hypoxia, which is the result of a reduction in oxygen tension in the arterial blood (Pa_{O_2}), and of which acute *hypobaric* hypoxia is one cause.

In military aviation, the principal causes of hypoxia in flight are the following[4(p46)]:

- ascent to altitude without supplementary oxygen (about 10% of casualties);
- failure of personal breathing equipment to deliver oxygen at an adequate concentration or pressure (about 68%); and
- decompression of the pressure cabin at high altitude (about 20%).

The physiological consequences of hypoxia in flight are dominated by the changes seen in three main areas: the respiratory and cardiovascular responses to hypoxia, and the neurological effects of both hypoxia itself and the cardiorespiratory responses to it.[4] The clinical consequences reflect changes seen in all three systems. It is worth remembering that in healthy individuals at sea level, alveolar ventilation is the prime determinant of tissue carbon dioxide level, and local blood flow is the prime determinant of tissue oxygen tension.

TABLE 32-2

ATMOSPHERIC COMPOSITION OF DRY AIR

Gas	Percentage by Volume in Dry Air*
Nitrogen	78.09
Oxygen	20.95
Argon	0.93
Carbon Dioxide	0.03
Neon	$1.82 \cdot 10^{-3}$
Helium	$5.24 \cdot 10^{-4}$
Krypton	$1.14 \cdot 10^{-4}$
Hydrogen	$5.00 \cdot 10^{-5}$
Xenon	$8.70 \cdot 10^{-6}$

*For most practical purposes, however, dry air may be regarded as a mixture consisting of 21% oxygen and 79% nitrogen.
Reproduced with permission from Harding RM. The Earth's atmosphere. In: Ernsting J, King P. *Aviation Medicine.* 2nd ed. London, England: Butterworths; 1988: 5.

Respiratory Responses to Hypoxia

The respiratory responses to hypoxia clearly depend on the manner in which the insult is delivered. Thus, the changes that accompany a slow ascent to altitude when breathing air are different from those seen if the ascent is undertaken when breathing oxygen, and different again if hypoxia is the result of a rapid loss of cabin pressure.

Alveolar Gases When Breathing Air. As described above, ascent to altitude is associated with an exponential fall in P_B (with parallel reductions in both air density and temperature) and also, therefore, in the partial pressures of the component gases of the atmosphere. The fall in the partial pressure of oxygen in the inspired gas (P_{IO_2}) produces a corresponding reduction in the partial pressure of oxygen in the alveoli (P_{AO_2}). But the main determinant of the difference between P_{IO_2} and P_{AO_2} is the partial pressure of carbon dioxide in the alveoli (P_{ACO_2}), as shown in Equation 1:

$$(1) \qquad P_{IO_2} - P_{AO_2} = P_{ACO_2}\left(F_{IO_2} + \frac{1 - F_{IO_2}}{R}\right)$$

where F_{IO_2} represents the fraction of inspired oxygen, and R represents the respiratory exchange ratio.

P_{ACO_2} is itself determined by the ratio of carbon dioxide production to alveolar ventilation, a ratio that is independent of environmental pressure. Provided that this ratio is undisturbed, P_{ACO_2} will remain constant on ascent to altitude. This is indeed what happens during ascent from sea level to about 10,000 ft: P_{ACO_2} remains constant and P_{AO_2} falls linearly with the reduction in environmental pressure.

Above 10,000 ft, however, the partial pressure of oxygen in arterial blood (Pa_{O_2}) falls to a level that stimulates respiration via the arterial chemoreceptors, and so P_{ACO_2} decreases as alveolar ventilation increases. Because there is little if any change in metabolic production of carbon dioxide under these circumstances, the ventilatory response to hypoxia is *hyperventilation* (discussed below). As a consequence of the reduction in P_{ACO_2}, the difference between P_{IO_2} and Pa_{O_2} is less than it would have been had no stimulation of ventilation occurred. So the

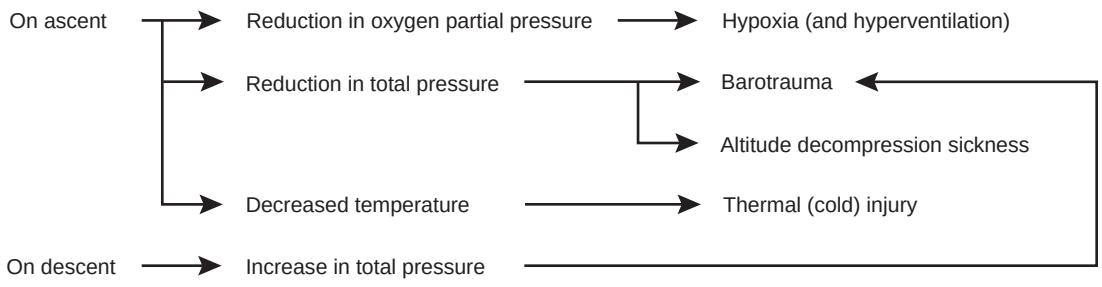

Fig. 32-3. The environmental changes associated with ascent to altitude are dominated by the development of hypoxia as a result of reduced oxygen partial pressure. Those asociated with descent are dominated by the possible development of otic and sinus barotrauma as a result of increased total pressure. Adapted from Harding RM, Mills FJ. Problems of altitude. In: Harding RM, Mills FJ, eds. *Aviation Medicine.* 3rd ed. London, England: British Medical Association; 1993: 71.

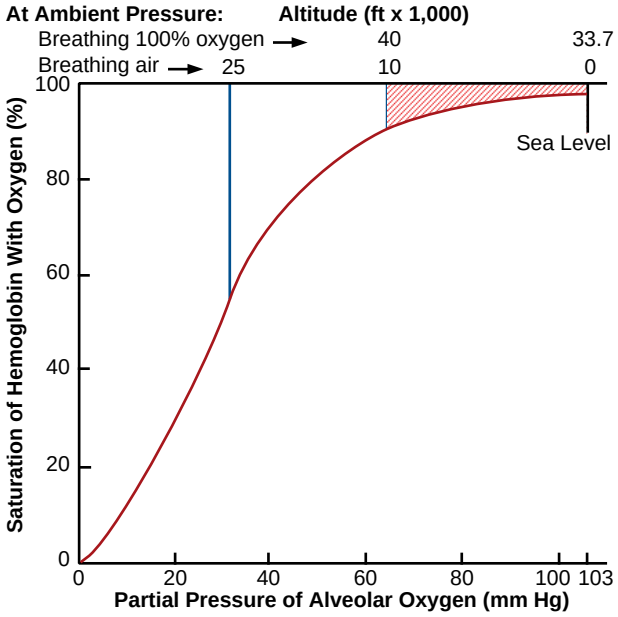

Fig. 32-4. The relation between oxygen saturation of hemoglobin and oxygen partial pressure, as reflected in the sigmoid shape of the dissociation curve, minimizes the physiological effects of a fall in partial pressure. The plateau represents an in-built reserve, or buffer zone, which provides protection for healthy individuals up to an altitude of 10,000 ft, and is exploited by aircraft designers, who maintain commercial aircraft cabins at an altitude below this. Above 10,000 ft, and especially above 25,000 ft, percentage saturation of hemoglobin falls and hypoxia results (unless enriched oxygen is provided). Note that, in this regard, sea level and 33,700 ft, and 10,000 ft and 40,000, can be described as physiologically equivalent altitudes. Adapted from Harding RM, Mills FJ. Problems of altitude. In: Harding RM, Mills FJ, eds. *Aviation Medicine.* 3rd ed. London, England: British Medical Association; 1993: 61.

increase in ventilation is "protective" against hypoxia insofar as it reduces the fall in P_{AO_2} that would otherwise be seen on ascent; however, the magnitude of the effect is itself a compromise between the demand for an adequate oxygen supply (ventilation stimulated via chemoreceptors) and the need to maintain a normal acid–base balance (ventilation inhibited by hypocapnia).

Reaching an altitude of 10,000 ft is consequently a crucial stage in the development of the physiological changes associated with an ascent. A consideration of the oxyhemoglobin dissociation curve helps to show why this should be (Figure 32-4). The relation between the partial pressure of oxygen (P_{AO_2}) and the percentage saturation of hemoglobin with oxygen describes a sigmoid curve. The plateau at the top of the curve represents a physiological reserve, whereby a fall in P_{O_2} (however produced) from the normal 100 mm Hg to about 60 to 70 mm Hg produces very little desaturation. In healthy individuals, a fall in P_{O_2} of this magnitude is seen during an ascent from sea level to 10,000 ft. Above this altitude, the steep part of the dissociation curve takes effect: hemoglobin rapidly desaturates and significant hypoxia develops.

Alveolar Gases When Breathing Oxygen. If ascent is undertaken when breathing 100% oxygen, and provided that P_{ACO_2} remains constant, then P_{AO_2} will fall linearly with environmental pressure until such time as hypoxia stimulates respiration. Thus, when 100% oxygen is breathed, not until an altitude of 33,700 ft does P_{AO_2} fall to 103 mm Hg (ie, to its sea level value [equivalent] when breathing air). And not until an altitude of about 39,000 to 40,000 does P_{AO_2} fall to 60 to 65 mm Hg (ie, to the value seen at 10,000 ft when breathing air). Above 40,000 ft, P_{AO_2}, and therefore Pa_{O_2} too, fall to levels that stimulate respiration even though 100% oxygen is being inspired. Once again, the resulting fall in P_{ACO_2} (ie, hyperventilation) is "protective" against hypoxia, and P_{AO_2} will rise by 1 mm Hg for every 1 mm Hg reduction in P_{ACO_2}. The concept of physiologically equivalent altitudes when breathing air or 100% oxygen is of considerable value in the design of life-support systems (see the discussion below in the Personal Oxygen Equipment section).

If hypoxia is induced by a change from breathing gas with a high oxygen content to air, then P_{AO_2} falls progressively as the nitrogen concentration in the inspired and alveolar gas rises to about 80%. There is frequently a phase during this process when P_{AO_2} is lower than Pa_{O_2}, and so oxygen passes out of the pulmonary circulation into the alveolar gas, thus briefly counteracting the original fall.

Alveolar Gases During Rapid Decompression. The sudden fall in P_B that accompanies a rapid decompression (RD) of an aircraft cabin (that is, a decompression over a period of seconds or less) produces equally severe falls in the partial pressures of alveolar gases. The magnitude of this effect will depend not only on the ratio of the environmental pressure before decompression to that after the event, but also on the composition of the gas being breathed at the moment of decompression.[5]

For example, a decompression from 8,000 ft to 40,000 ft in 1.6 seconds while air is breathed produces a fall in P_{AO_2} from 65 mm Hg to 15 mm Hg, and a fall in P_{ACO_2} to only 10 mm Hg. Although the latter quickly recovers to about 25 to 30 mm Hg, P_{AO_2} remains at about 18 mm Hg for as long as air is breathed

(because at 40,000 ft, P_{IO_2} is only 20 mm Hg).

But a fall in P_{AO_2} to below 30 mm Hg will inevitably be accompanied by profound neurological hypoxia, and such a fall is an equally inevitable consequence of breathing air during an RD to a final altitude greater than 30,000 ft. This is the case even if delivery of 100% oxygen commences at the moment of decompression. Although subsequently, breathing 100% oxygen will cause a rapid rise in P_{AO_2}, the greater the final altitude and the longer the delay in delivering 100% oxygen, the more profound will be the degree of hypoxia. Thus, it is clear that breathing an oxygen-rich mixture before an RD will reduce the severity of hypoxia after it. Indeed, for example, P_{AO_2} will at no time fall below about 60 mm Hg if 100% oxygen is breathed before, during, and after an RD from 8,000 ft to 40,000 ft. These rapid physiological changes have important implications for the design of personal oxygen equipment because an oxygen-rich inspirate must be provided before the event. Furthermore, delivery of 100% oxygen must be initiated within 2 seconds of the start of an RD if significant hypoxia is to be avoided.

Cardiovascular Responses to Hypoxia

Cardiovascular responses to hypoxia involve general and regional changes. In the resting subject, heart rate starts to rise when breathing air at altitudes above 6,000 to 8,000 ft, and is approximately doubled at 25,000 ft. Cardiac output also rises, but stroke volume and mean arterial blood pressure are unchanged. Systolic blood pressure and pulse pressure are usually elevated but peripheral resistance is reduced, overall, with a redistribution of flow by local and vasomotor mechanisms.

Hypoxia causes vasodilation in most vascular beds, but there are some important features in the responses of certain regional circulations. Thus, the redistribution of cardiac output results in an increase in blood flow to the heart and brain at the expense of other, less vital, organs such as the bowel, the skin, and the kidneys. Flow to skeletal muscle is unchanged. Flow in the coronary circulation increases in parallel with cardiac output, but there is a reduction in cardiac reserve such that a profound fall in Pa_{O_2} can cause myocardial depression and, in some cases, a severe compensatory vasoconstriction with cardiac arrest. Electrocardiographic changes are a feature only during profound hypoxia.

Predictably, the cerebral circulation is acutely sensitive to changes in both Pa_{O_2} and P_{ACO_2}. When Pa_{O_2} is greater than 45 to 50 mm Hg, cerebral blood flow is exclusively determined by P_{ACO_2}, to which it bears a directly linear relation over the physiologically tolerable range (20–80 mm Hg). A fall in Pa_{O_2} below 45 mm Hg induces a hypoxic vasodilation, so that a Pa_{O_2} of 35 mm Hg will cause a 50% to 100% increase in cerebral blood flow. A conflict therefore exists between the *vasodilating* effect of hypoxia and the *vasoconstricting* influence of hypocapnia, itself caused by the ventilatory response to hypoxia.

Hypoxic desaturation of the blood by just 20% is sufficient to produce a generalized and rapid vasoconstriction in the pulmonary circulation, which, in the presence of an increased cardiac output, produces a rise in pulmonary artery pressure.

Neurological Effects of Hypoxia

Although the neurological consequences of a gross fall in P_{AO_2} are usually the cause of loss of consciousness in hypoxia, a simple vasovagal syncope occurs in about 20% of cases. In these individuals, loss of peripheral resistance in the systemic circulation is accompanied by a profound bradycardia, a fall in arterial blood pressure, and a failure of cerebral blood flow.

The ability of tissues to function normally will depend critically on tissue oxygen tensions. Once again, the implications of the oxyhemoglobin dissociation curve are highly relevant (see Figure 32-4). In this case, the steep part of the curve reflects the enhanced and protective ability of hemoglobin to unload oxygen at low levels of P_{O_2}. Thus, for example, when breathing air at sea level (where P_{AO_2} = 100 mm Hg), the delivery of 5 mL of oxygen from every 100 mL of blood results in an arteriovenous difference of 60 mm Hg. The extraction of the same quantity of oxygen when breathing air at 18,000 ft (where P_{AO_2} = 32 mm Hg) results in an arteriovenous difference of only 10 mm Hg.

The neurological effects of hypoxia are obviously of great practical significance in aviation. Exhibit 32-2 summarizes the covert features of early cerebral hypoxia, when air is breathed; medical officers should keep in mind, though, the wide variability in individual behavior, which is primarily a consequence of differences in the respiratory responses to hypoxia.

In more-severe hypoxia, provided that vasovagal syncope does not preempt the issue, loss of consciousness occurs when jugular venous oxygen tension falls to 17 to 19 mm Hg, reflecting significant cerebral hypoxia. The cerebral arterial oxygen tension at which this point is reached depends on cerebral blood flow, and so will vary according to the degree of accompanying hypocapnia: it may lie anywhere between 20 and 35 mm Hg.

EXHIBIT 32-2

COVERT FEATURES OF ACUTE HYPOBARIC HYPOXIA

Psychomotor Function
- Choice reaction time is impaired significantly by 12,000 ft.
- Eye-hand coordination is impaired by 10,000 ft, even for well-learned tasks.
- Muscular incoordination increases > 15,000 ft.
- Simple reaction time is affected only > 18,000 ft.

Cognitive Function
- Performance at novel tasks may be impaired at 8,000 ft.
- Memory is increasingly impaired > 10,000 ft.

Visual Function
- Light intensity is perceived as reduced.
- Visual acuity is diminished in poor illumination.
- Light perception threshold is increased.
- Peripheral vision is narrowed (ie, tunneling).

Adapted with permission from Ernsting J, Sharp GR. Harding RM, rev-eds. Hypoxia and hyperventilation. In: Ernsting J, King PF, eds. *Aviation Medicine.* 2nd ed. London, England: Butterworths, 1988: 57.

Again, consciousness will inevitably be lost if P_{AO_2} falls below 30 mm Hg (eg, following an RD to an altitude above 30,000 ft). Even in these instances, however, circulation and brain equilibration times are such that consciousness persists for 12 to 14 seconds after the event.

Clinical Features of Acute Hypobaric Hypoxia

The clinical features of acute hypobaric hypoxia are a combination of the respiratory and cardiovascular responses and the neurological effects described above; consequently, the symptoms and signs are extremely variable. The speed and order of the appearance of signs and of the severity of symptoms produced by a lowering of the P_{IO_2} depend on (1) the rate at which, and the level to which, the oxygen tension falls and (2) the duration of the reduction. Even when these factors are kept constant, however, there is considerable variation among individuals; although for the same individual the pattern of effects does tend to remain similar—a phenomenon that increases the value of routine exposure of aircrew to hypoxia in a training environment. Exhibit 32-3 summarizes the clinically obvious features of hypobaric hypoxia (and of hyperventilation). Several additional factors may influence an individual's susceptibility to hypoxia and so modify the pattern of symptoms and signs produced. These factors include

- physical activity (exercise exacerbates the features of hypoxia),
- ambient temperature (a cold environment reduces tolerance to hypoxia),
- intercurrent illness (the additional metabolic load imposed by ill health increases susceptibility to hypoxia), and
- drugs (many pharmacologically active substances have effects similar to those of hypoxic hypoxia and so mimic or exacerbate the condition; proprietary preparations containing antihistamine constituents are particularly likely to cause problems, as is alcohol).

Although the greater the altitude the more marked will be the features seen, rapid ascent can allow high altitudes to be reached before severe symptoms and signs occur. In such circumstances, however, sudden unconsciousness may precede the classic features. Accordingly, it is useful to consider the clinical picture seen during slower ascents to various altitudes (Table 32-3).

The *Time of Useful Consciousness* (TUC) denotes the interval between the onset of reduced P_{IO_2} and the point at which there is a specified impairment of performance. The latter is most usefully defined as the point beyond which the hypoxic individual can no longer act to correct the situation. The TUC for a resting subject who changes from breathing oxygen to breathing air at 25,000 ft is about 3 to 6 minutes but is reduced to about 1 to 2 minutes if the subject is exercising. The corresponding times at 40,000 ft are 30 seconds (resting) and 15 to 18 seconds (exercising). Quick corrective action is critical during the TUC, and the importance of a rapid, accurate self-diagnosis followed by emergency action cannot be overemphasized. The nature of hypoxia must be taught and demonstrated regularly to aircrew so that they may learn to recognize their individual symptomatology and take appropriate action. For example, a pilot who instantly identifies an RD can switch to 100% oxygen before the inevitable loss of consciousness, thus increasing the likelihood of recovery in time to regain control of the aircraft before it crashes.

Acute hypobaric hypoxia is rapidly and completely reversed if oxygen is administered or if P_{AO_2} is elevated as a consequence of sufficiently in-

EXHIBIT 32-3

SIGNS AND SYMPTOMS OF ACUTE HYPOXIA (AND OF HYPERVENTILATION)

Hypoxia

- Personality change
- Lack of insight*
- Loss of judgment*
- Feelings of unreality
- Loss of self-criticism*
- Euphoria
- Loss of memory
- Mental incoordination
- Muscular incoordination
- Sensory loss
- Cyanosis
- Hyperventilation* — *(Signs and Symptoms of Hyperventilation)*†
  - °*Dizziness*
  - °*Lightheadedness*
  - °*Feelings of apprehension*
  - °*Neuromuscular irritability*
  - °*Paresthesias of the face and extremities*
  - °*Carpopedal spasm*
- Semiconsciousness
- Unconsciousness
- Death

*Because of their sinister covertness, these items have special significance in the early phase of hypobaric hypoxia.
†Although a sign of hypoxia, hyperventilation also has its own signs and symptoms, which are separate from those of hypoxia.
Adapted with permission from Ernsting J, Sharp GR. Harding RM, rev-ed. Hypoxia and hyperventilation. In: Ernsting J, King PF, eds. *Aviation Medicine.* 2nd ed. London, England: Butterworths, 1988: 57.

creased environmental pressure. There are no sequelae other than a persistent generalized headache if the exposure was prolonged. Occasionally, a temporary (15–60 s) worsening of the clinical features may occur as P_{AO_2} is restored, a phenomenon known as the "oxygen paradox," which is probably caused by a combination of arterial hypotension and persistent hypocapnia, and may manifest as clonic spasms and even loss of consciousness. It is, however, usually mild with some decrement in psychomotor performance accompanying a flushing of the face and hands. In the event of a paradox, it is essential that delivery of oxygen is maintained despite the decline in clinical state.

Hyperventilation

Hyperventilation is a condition in which pulmonary ventilation is greater than that required to eliminate the carbon dioxide produced by body tissues. There is a consequent excessive fall in carbon dioxide levels within alveolar gas, the blood (hypocapnia), and the tissues. A reduction in P_{ACO_2} will also lead to a fall in hydrogen ion concentration (that is, to a rise in pH) so that hyperventilation causes a respiratory alkalosis.

The causes of hyperventilation may be summarized thus:

- Hypoxia (hyperventilation is a normal response to a fall in P_{AO_2} below 55–60 mm Hg).
- Emotional stress—particularly anxiety, apprehension, or fear—is the commonest cause of hyperventilation. Anyone can be affected; hyperventilation occurs in student aircrew during training, in experienced air crew (eg, on change of role), and in passengers.
- Pain, motion sickness, environmental stress

TABLE 32-3

SIGNS AND SYMPTOMS OF HYPOXIA RELATED TO ALTITUDE

Breathing Air	Breathing 100% Oxygen	Signs and Symptoms
≤ 10,000 ft	≤ 39,000 ft	No symptoms, but impaired performance of novel tasks.
10,000–15,000 ft	39,000–42,500 ft	Few or no signs and symptoms are present when resting. Any impairment of performance at skilled tasks is unappreciated. Physical work capacity is severely reduced.
15,000–20,000 ft	42,500–45,000 ft	All the overt signs and symptoms in Exhibit 32-3 may appear, and those due to hyperventilation may dominate. Signs and symptoms are severely exacerbated by physical activity.
> 20,000 ft	> 45,000 ft	Marked signs and symptoms are seen, with rapid decline in performance and sudden loss of consciousness. Hypoxic convulsions are likely.

(eg, high ambient temperatures), and whole-body vibration (as produced by clear air turbulence) at 4–8 Hz when flying at low level, can all produce hyperventilation.

- Positive-pressure breathing (see below).

The physiological consequences of hyperventilation comprise cardiovascular responses and neurological effects.[4] Hypocapnia has no effect on either cardiac output or arterial blood pressure, although the former is redistributed. Thus, blood flow through skeletal muscle is increased, while that through the skin and cerebral circulation is reduced. The intense cerebral vasoconstriction acts to reduce local Pa_{O_2}, and the neurological features of profound hyperventilation are probably due to a combination of cerebral hypoxia and alkalosis. A reduction in Pa_{CO_2} to below 25 to 30 mm Hg produces significant decrements in the performance of both psychomotor and complex mental tasks, while the ability to perform manual tasks is compromised by the neuromuscular disturbance associated with a fall in Pa_{CO_2} below 20 mm Hg. There is gross clouding of consciousness and then unconsciousness if Pa_{CO_2} falls below 10 to 15 mm Hg. The increased sensitivity and spontaneous activity in the peripheral nervous system are consequences of the local rise in pH and produce sensory (eg, paraesthesias) and motor (eg, spasms) disturbances.

The clinical features of hyperventilation relate to the extent of the reduction in P_{ACO_2} and are summarized in Exhibit 32-4. In those rare cases where extreme anxiety-induced hyperventilation leads to unconsciousness, recovery naturally occurs as autonomic respiratory control reasserts itself and carbon dioxide levels return to normal. Unfortunately, the hyperventilation associated with hypoxic hypoxia in flight is not subject to such self-correction. Because most of the early symptoms of hypoxia are similar to those of hypocapnia (and indeed the features of hypocapnia frequently dominate the early stages of hypoxia), hypoxia must always be suspected when symptoms or signs of hypocapnia occur at altitudes above about 10,000 ft. Therefore, aircrew have to appreciate that corrective procedures must be based on the assumption that the condition is caused by hypoxia until proven otherwise. In combat aircraft, the appropriate corrective action in cases of suspected hypoxia is the emergency oxygen drill (which selects an alternative supply of gas) as laid down in the aircraft flight procedures. Although a common course of action, it is not appropriate or acceptable to select 100% oxygen from the main aircraft system as a "trial" treatment.

Barotrauma: The Direct Effects of Pressure Change

The human body may be considered to be at a constant temperature, and any gas within closed or semiclosed body cavities will obey Boyle's law on ascent to altitude. So, for example, any such gas will double in volume if it is free to do so on ascent from

EXHIBIT 32-4

SYMPTOMS AND SIGNS OF HYPERVENTILATION

Partial Pressure of Alveolar Carbon Dioxide (mm Hg)	Symptoms and Signs of Hyperventilation
P_{ACO_2} 20–25:	Lightheadedness, dizziness, anxiety (which may produce a vicious circle), and paraesthesias of the extremities and around the lips
P_{ACO_2} 15–20:	Muscle spasms of the limbs (carpopedal) and face (risus sardonicus)
	Augmentation of the tendon reflexes (positive Chvostek's sign)
	General deterioration in mental and physical performance
$P_{ACO_2} < 15$:	General tonic contractions of skeletal muscle (tetany)
$P_{ACO_2} < 10$–15:	Semiconsciousness, then unconsciousness

sea level to 18,000 ft. The lungs, the teeth, and the bowel may be affected by gas expansion during ascent (although aerodontalgia is now rare), while the middle ear cavities and sinuses are particularly affected by compression during descent.[6] A more-complete discussion of barotrauma can be found in Chapter 30, Physics, Physiology, and Medicine of Diving. The problems in lungs, teeth, and bowel are more severe in diving than flight because the pressure changes are greater.

Pulmonary Barotrauma

Expansion of gas within the lungs does not usually present a hazard on ascent because increasing volume can easily vent through the trachea. However, RD with a closed glottis can potentially produce catastrophic aeroembolism. (This topic is also discussed in Chapter 30, Physics, Physiology, and Medicine of Diving).

Gastrointestinal Distension

Expansion of gas within the small intestine can cause pain of sufficient severity to produce vasovagal syncope. Although this is unlikely to occur at normal rates of ascent in both transport and combat aircraft, it is a possibility after rapid loss of cabin pressurization at high altitude in the latter. In military aircrew, gut pain of this nature can also occur during RD undertaken for training purposes; indeed, this is the commonest cause of failure in such training. Gaseous expansion in the small bowel is aggravated by gas-producing foods (eg, beans, curries, brassicas, carbonated beverages, and alcohol). Gas in the stomach and large intestine does not usually cause problems because it can easily be released.

Otic Barotrauma

Expanding gas in the middle ear cavity easily vents through the eustachian tube on ascent and only rarely causes any discomfort. The symptoms of otic barotrauma develop during descent because air cannot pass back up the tube so readily.[7] Pain, which begins as a feeling of increased pressure on the tympanic membrane, quickly becomes increasingly severe unless the eustachian tube is able to open, an event colloquially known as "clearing" the ears. Many experienced aircrew can achieve such opening merely by swallowing, yawning, or moving the lower jaw from side to side. Others perform a deliberate technique to open the tube by raising the pressure within the pharynx. Some people have great difficulty in learning these procedures, and some may be unable to do so even after much coaching and practice.

The most useful of these techniques is the Frenzel maneuver, which is performed with the mouth, nostrils, and epiglottis closed. Air in the nasopharynx is then compressed by the action of the muscles of the mouth and tongue. The Frenzel maneuver not only generates higher nasopharyngeal pressures than the Valsalva maneuver, discussed below, but also achieves opening of the eustachian tube at lower pressures.[7]

In the Toynbee maneuver, pharyngeal pressure is raised by swallowing while the mouth is closed and the nostrils occluded. This is the best technique to use when evaluating eustachian function under

physiological conditions: under direct vision, the observer sees a slight inward movement of the tympanic membrane, followed by a more marked outward movement.

The Valsalva maneuver consists of a forced expiration through an open glottis while the mouth is shut and the nostrils occluded. The increase in intrathoracic pressure is transmitted to the nasopharynx and hence to the eustachian tubes. The rise in intrathoracic pressure is a disadvantage, however, because it impedes venous return to the heart and may even induce syncope.

The acute angle of entry of the eustachian tube into the pharynx predisposes to closure of the tube by increasing P_B during descent. By causing inflammation and edema of the lining of the eustachian tube, upper respiratory tract infections increase the likelihood of developing otic barotrauma and of its ultimate result: rupture of the tympanic membrane. Aircrew are made fully aware of this condition during training and are instructed not to fly if they are unable to clear the ears during an upper respiratory tract infection. In doubtful cases, a nonmoving tympanic membrane can be detected by direct vision. Treatment of otic barotrauma, particularly if blood or fluid is present in the middle ear cavity, should include analgesia, a nasal decongestant, and a broad-spectrum antibiotic.

Sinus Barotrauma

The etiology of sinus barotrauma is the same as that of its otic counterpart. On ascent, expanding air vents easily from the sinuses through their ostia. On descent, however, the ostia are readily occluded, especially if the victim has an upper respiratory tract infection. Characteristically, a sudden, severe, knifelike pain occurs in the affected sinus. The pain continues if descent is not halted and epistaxis may result from submucosal hemorrhage. The development of sinus barotrauma is related to the rate of descent, and its prevention is part of the rationale behind the slow rate of descent employed in transport and civilian aircraft. The possibility of a sinus problem cannot be predicted prior to flight, but flying with a cold will clearly increase the risk. As with otic barotrauma, treatment of sinus barotrauma should include analgesia, nasal decongestants, and a suitable antibiotic.

Altitude Decompression Sickness

Altitude decompression sickness (DCS) is that syndrome produced by exposure to altitude that is not due to low P_{IO_2}, to expansion of trapped or enclosed gas, or to intercurrent illness. Altitude DCS is therefore a diagnosis of exclusion; although it is conventionally regarded as a syndrome similar to the classic diving affliction, there are some fundamental and vitally important differences (Table 32-4).

The etiology of altitude DCS is not fully understood but certainly involves supersaturation of body tissues with nitrogen.[8] Ascent to altitude is associated with a fall in the partial pressure of inspired nitrogen and a corresponding fall in the partial pressure of alveolar nitrogen. Nitrogen consequently starts to leave body stores but, since it is poorly soluble in blood, the partial pressure of nitrogen in tissue falls at a slower rate than does the partial pressure of inspired nitrogen in blood. The tissues and blood may therefore become supersaturated with nitrogen, and bubbles begin to form around pre-existing microscopic nuclei, such as vessel wall irregularities. The bubbles grow as blood gases diffuse into them and can be carried to other parts of the body where they may or may not manifest clinically. Bubble formation is more likely if the partial pressure of nitrogen in tissue is high (notably in fat, which has high nitrogen solubility and low blood flow) or if P_B is low. Bubbles apparently need to reach a critical size before clinical features develop.

Many factors can influence the *occurrence* of clinical DCS; these are summarized in Exhibit 32–5. The clinical *features* of DCS may include any or all of the following[9]:

- Joint and limb pains (the "bends"). Bends pain is the commonest manifestation of altitude DCS and is seen in about 74% of cases. The pain is deep and poorly localized, made worse on movement, and frequently likened to having glass in the joint. Although single, large joints are most frequently affected, more than one joint can be involved and at any site. The pain usually resolves during descent to ground level.
- Respiratory disturbances (the "chokes"). Respiratory involvement is seen in about 5% of cases and has serious implications. Feelings of constriction around the lower chest, with an inspiratory snatch, paroxysmal cough on deep inspiration, and substernal soreness, is followed by malaise and collapse unless descent is initiated.
- Skin manifestations (the "creeps"). Dermal manifestations in the form of an itchy, blotchy rash (perhaps with formication) are seen in

TABLE 32-4

IMPORTANT FACTORS IN COMPARING AVIATORS' AND DIVERS' DECOMPRESSION SICKNESS

Factor	Flying	Diving
Preventive Denitrogenation	Denitrogenation can be used before the mission to reduce the risk of decompression sickness (DCS)	Not applicable to diving
Preexisting Degree of Saturation	Decompression starts from a saturated state	Saturation remains constant during the diving and intervening periods
Inspired Gas	Inspired gas usually contains high oxygen concentration	Diving mixtures must limit oxygen concentration to prevent toxicity
Composition of Gas Bubbles	Bubbles contain nitrogen, oxygen, carbon dioxide, and water	Nitrogen and the noble gases predominate
Pressure in Bubbles (Compared With Sea Level)	Pressure can be very low	Pressure can be very high
Duration of Dysbaria	Time of exposure to altitude is limited	Duration depends on the dive profile
Onset of DCS	DCS occurs during the mission	Risk of DCS on return to surface
Symptoms	Symptoms are usually mild and limited to joint pain	Severe pain and neurological symptoms are frequent
Repeat Exposure	Recompression to ground level is therapeutic	Return to depth is limited and hazardous
Individual Susceptibility	Individual susceptibility varies widely	Individual susceptibility is less varied
Sequelae	There are no documented cumulative effects	Chronic bone necrosis and neurological changes found in divers

about 7% of cases but are of little significance except in association with respiratory symptoms, when urticaria and mottling of the skin of the thorax may be present.

- Visual disturbances. Visual symptomatology is seen in about 2% of cases. Blurring of vision, scotomata, and fortification spectra (zigzag bands of light resembling, at the edges of scintillating scotomata, the walls of a fortified medieval castle, which are most usually seen in migraine attacks) may be reported. There is usually no disturbance of the other special senses (hearing, smell, taste, and touch).
- Neurological disturbances (the "staggers"). Neurological involvement is rare, being seen in only about 1% of cases. Regional paralysis, paraesthesias, anaesthesia, and seizures may all be features.
- Cardiovascular collapse. Occasionally, a pro found cardiovascular collapse may occur either without warning (primary) or subsequent to any of the other manifestations of altitude DCS (secondary). The features of

EXHIBIT 32-5

FACTORS INFLUENCING THE OCCURRENCE OF DECOMPRESSION SICKNESS

Altitude. In healthy individuals who start near sea level, clinical decompression sickness (DCS) is not usually seen at altitudes below 18,000 ft. It is rare between 18,000 and 25,000 ft but becomes increasingly common at altitudes above 25,000 ft.

Duration of Exposure. DCS usually develops after at least five minutes at altitude, with the maximum incidence at 20–60 min after exposure.

Rate of Ascent. The rates of ascent employed in routine military aviation have little if any significant effect on the occurrence of DCS.

Underwater Diving. The increases in pressure sustained during diving (particularly to depths > 15 ft) lead to compression of additional nitrogen in the tissues. Although some of this will evolve into gas during ascent (decompression) to the water's surface, more nitrogen than usual will be present to form more gas bubbles if a further ascent to altitude is undertaken shortly afterwards (also see Exhibit 32-6).[1,2,3]

Reexposure. Repeated exposure over a short time to altitudes at which DCS may occur (eg, paratroop training) will predispose to DCS.

Temperature. The risk of developing DCS increases if environmental temperatures are low.

Exercise. The altitude at which clinical DCS may develop is reduced by exercise.

Hypoxia. The presence of coexisting hypoxia increases both the incidence and the severity of DCS.

Age. The risk of developing DCS increases with age, approximately doubling every decade.

Body Build. Those with much adipose tissue appear to have an increased susceptibility to DCS.

Previous Injury. Physical damage to tissues may predispose to DCS by encouraging formation of nuclei around which bubbles may form.

General Health. Drugs, alcohol, smoking, and intercurrent illness will all increase susceptibility to DCS.

Individual Susceptibility. There appears to be a true individual variation in susceptibility to DCS.

Sources: (1) Furry DE, Reeves E, Beckman E. Relationships of SCUBA diving to the development of aviator's decompression sickness. *Aerospace Med.* 1967;38:825-828. (2) Blumkin D. Flying and diving—A unique health concern. *Flight Safety Foundation's Human Factors and Aviation Medicine.* 1991;Sep/Oct:21-28. (3) Sheffield PJ. Flying after diving guidelines: A review. *Aviat Space Environ Med.* 1990;61:1130-1138.

such a collapse include malaise; anxiety; diminution of consciousness; and pale, clammy, sweaty skin. A bradycardia leads to loss of consciousness. Recovery is usually accompanied by vomiting and a frontal headache.

Although the natural history of altitude DCS is that symptoms and signs resolve on descent—approximately 95%[9] of volunteer subjects affected in hypobaric chamber studies have recovered on reaching ground level—there may very rarely be a persistence or even a worsening of features several hours after return. Such a delayed collapse is usually only seen if severe symptoms and signs of DCS (such as chokes or bends) were present at altitude. In addition to the features of collapse described above, there may be general and focal neurological signs and mottling of the skin. The hematocrit rises, as does the white blood cell count and temperature. Should unconsciousness develop, the outcome is almost invariably fatal.

Arterial Gas Embolism

In addition to the constellation of features colloquially described above as the "staggers," a second neurological syndrome—cerebral arterial gas embolism (CAGE)—may very rarely be associated with RD to high altitude (although it is more common in the hyperbaric environment). In this condition, overinflation of pulmonary tissue results in rupture

of alveoli and escape of gas directly into the arterial circulation. Subsequent embolization to the brain can produce a clinical picture very similar to cerebral decompression sickness, or CNS DCS.[10] (Because of this similarity, the global term decompression *illness* [DCI] has been recommended as a replacement for the more-familiar DCS[10]; this textbook, however, uses the terms DCS and CAGE.)

Diagnosis and Treatment of Decompression Sickness

The differential diagnosis of DCS must include flight stresses such as hypoxia, hyperventilation, abdominal distension, alternobaric vertigo (ie, sudden, powerful vertigo caused by pressure change within the middle ear; the condition usually occurs at low altitude during ascent and may be a potent cause of spatial disorientation), motion sickness, and acceleration atelectasis (ie, the rapid collapse of oxygen-filled basal alveoli due to the ventilation–perfusion abnormalities associated with high +Gz acceleration [ie, positive acceleration along the body's z, or head-to-toe, axis; see Figure 33-1 in Chapter 33, Acceleration Effects on Fighter Pilots]); and intercurrent illness such as ischemic heart disease, spontaneous pneumothorax, and cramp of or injury to the limbs. Although it is usually easy to exclude these conditions, it may be necessary to monitor the hematocrit, white blood cell count, and temperature.

Recovery from altitude DCS is usually complete, but the management of the established condition occurs in several stages. In-flight management involves descent to as low an altitude as circumstances permit (and at least to below an aircraft altitude of 18,000 ft), administering 100% oxygen, keeping still and warm if practicable, and landing as soon as possible where medical aid is available and has been alerted.

The casualty must be seen by a medical officer immediately after landing and be kept under observation for at least 4 hours, even if symptoms improved markedly during descent or have completely disappeared. Failure of symptoms or signs to improve, or any deterioration in condition, or the appearance of new symptoms and signs during the observation period all suggest the possibility of impending collapse, and active treatment should be initiated immediately. Important symptoms suggesting deterioration include headache, nausea, visual disturbances, anxiety, and sweating; signs of significance include hemoconcentration, pyrexia, and peripheral vascular failure (pallor, cyanosis, and weak distal pulses in the presence of near-normal blood pressure). Although the form of active treatment depends on both geographical location and the availability of recompression resources, the order of preference is as follows:

1. Immediate hyperbaric compression with or without intermittent oxygen breathing.
2. Institution of treatment for established or incipient circulatory collapse, followed by early transfer to a hyperbaric facility if possible within reasonable time (< 6 h) or distance. Transport should be by road or low-level flight (< 1,000 ft if possible but not > 3,000 ft).
3. Full supportive treatment for collapse (essential if there is no chance of prompt transfer to a hyperbaric facility). Such treatment should include expansion of plasma volume, administration of 100% oxygen and intravenous steroids, correction of blood electrolytes, and drainage of pleural effusions.

The ideal way in which to prevent altitude DCS is to limit the pressure environment to less than 18,000 ft. This may not always be possible, however, in which case time above 22,000 ft should be kept to a minimum, and the influence of predisposing factors should be minimized. For experimental and training purposes in decompression chambers, DCS can be prevented by denitrogenation (ie, reducing body nitrogen content by breathing 100% oxygen before or during ascent to high altitude, a process also termed "prebreathing"), although such prebreathing is often impracticable for operational purposes. And the possibility of developing DCS as a consequence of operating fixed-wing aircraft out of and into airfields at high terrestrial locations, and of rotary wing operations in mountainous regions, must always be kept in mind (Exhibit 32-6).

LIFE-SUPPORT SYSTEMS FOR FLIGHT AT HIGH ALTITUDE

The means by which protection against the hazards of altitude are provided for the occupants of aircraft include (*a*) the cabin pressurization systems of large transport and civilian aircraft, and of small military combat aircraft, and (*b*) the requirements and design of personal oxygen equipment. The ways in which the requirements for protection in each class of aircraft are achieved and interrelate

EXHIBIT 32-6

FLYING AFTER DIVING

There is a bewildering number of published guidelines from which divers can seek advice about flying after diving: at least 30 published sets of recommendations exist for those who wish to fly after diving within standard air tables; 5 more for saturation divers; and a further 12 to guide those concerned with the in-flight management of decompression sickness or with flying after hyperbaric therapy. It is wise to select a single reference and, in the United Kingdom, the *Royal Navy Diving Manual*, although aimed primarily at service divers, is well-known and authoritative. Article 5122 of the Manual provides simple advice on the minimum intervals between diving and flying, for dives without or with stops (see table). And Article 5121 gives advice for those who intend to fly after diving at altitude (eg, in mountain lakes). In the United States, similar guidelines are recommended by the Federal Aviation Administration (FAA) and the Undersea and Hyperbaric Medicine Society (UHMS). More stringent regulations apply to aircrew who may have participated in sports diving.

Type of Dive	Time Interval Between Diving and Flying (h)	Maximum Altitude*
Without Decompression Stops	≥1	~ 1,000 ft (300 m)†
	1–2	~ 5,000 ft (1,500 m)
	> 2	Unlimited flying in commercial aircraft (usually no more than an effective 8,000 ft [2,400 m])
With Decompression Stops	≤ 4	~ 1,000 ft (300 m)†
	4–8	~ 5,000 ft (1,500 m)
	8–24	~ 16,500 ft (5,000 m)
	> 24	Unlimited

*(or effective altitude in pressurized aircraft)
†eg, flying in helicopters
Adapted from Harding RM, Mills FJ. Problems of altitude. In: Harding RM, Mills FJ, eds. *Aviation Medicine.* 3rd ed. London, England: British Medical Association; 1993: 69.

TABLE 32-5

PROTECTIVE SYSTEMS FOR TRANSPORT AND COMBAT AIRCRAFT

	Protection	
Aircraft	**Primary**	**Secondary**
Transport	Cabin pressurization system (high differential)*	Personal oxygen system for flight deck crew and passengers
Combat	Personal oxygen system	Emergency oxygen supply *plus* Cabin pressurization system (low differential)†

*Large pressure difference between cabin and outside
†Small pressure difference between cabin and outside: cabin pressurized to approximately 22,000 ft because of considerable risk of rapid decompression

are summarized in Table 32-5.

Cabin Pressurization Systems

Cabin pressurization maintains the inside of an aircraft at a higher pressure (and hence lower effective altitude) than that outside the aircraft. The physiological ideal would be to pressurize the cabin to sea level at all times, but this is not cost-effective, so the minimum acceptable level of pressurization is determined by the need to prevent hypoxia, gastrointestinal distension, and altitude DCS, as well as to minimize the possible consequences of sudden loss of pressurization. In addition, the maximum rates of cabin ascent and descent are determined by effects on the middle ear cavities and sinuses.[11] Each of these factors imposes altitude limits (Table 32-6).

The difference between the absolute (or total) pressure within the aircraft and that of the atmosphere outside is termed the *cabin differential pressure,* the magnitude of which depends on the type of aircraft. In large passenger aircraft, where comfort and mobility are important and the risk of RD is small, the cabin is pressurized to about 6,000 ft. Such aircraft are said to have high-differential-pressure cabins because, when the aircraft is flying at high altitudes, a large difference exists between pressure within the cabin and the pressure outside. In combat aircraft, however, where only minimal weight can be devoted to life-support equipment and where there may be a considerable risk of RD, cockpits are pressurized to about 22,000 ft and are called low-differential-pressure cabins.

The relation between the effective cabin altitude and the actual, changing aircraft altitude is termed the *pressurization schedule* (Figure 32-5). For example, US combat aircraft use an isobaric schedule in which cabin altitude is held constant as the aircraft ascends until the maximum differential pressure is reached, after which cabin altitude rises linearly with aircraft altitude. In passenger aircraft, on the other hand, cabin differential pressure initially increases gradually with aircraft altitude until maximum differential pressure is attained. Thereafter, cabin altitude again rises linearly with aircraft altitude.

The cabin pressurization system controls not only the pressure of air within the cabin but also its humidity, mass flow, volume flow (ie, ventilation), and temperature (Figure 32-6). In fact, most of the demand for compressed air provides for cabin ventilation rather than pressurization. Passenger aircraft carry redundant systems and controls, while combat aircraft have a single pressure controller.

Loss of Cabin Pressurization

Loss of cabin pressurization (decompression) is usually the result of a system malfunction that either reduces inflow (as in a compressor failure) or increases outflow (as in leaks through open valves). Such losses are usually slow and are soon recognized and corrected by the crew. RDs are rare events and result from structural faults (eg, a failure of canopy seals, or loss of transparencies, doors, or windows) or enemy or terrorist action. An added complication is the possible Venturi effect of air

TABLE 32-6

CABIN ALTITUDE LIMITS AND PERFORMANCE IMPOSED BY PHYSIOLOGICAL FACTORS

Physiological Factors*	Cabin Altitude Limits and Performance (ft)		
	Transport Aircraft	Both	Combat Aircraft
Hypoxia	8,000		20,000–22,000
Decompression Sickness		< 22,000	
Gastrointestinal Distension	< 6,000		< 25,000
Middle Ears and Sinuses:			
Ascent		5,000–20,000 ft/min	
Descent		< 500 ft/min	

*The predicted consequences of rapid decompression on the lungs must also be taken into account

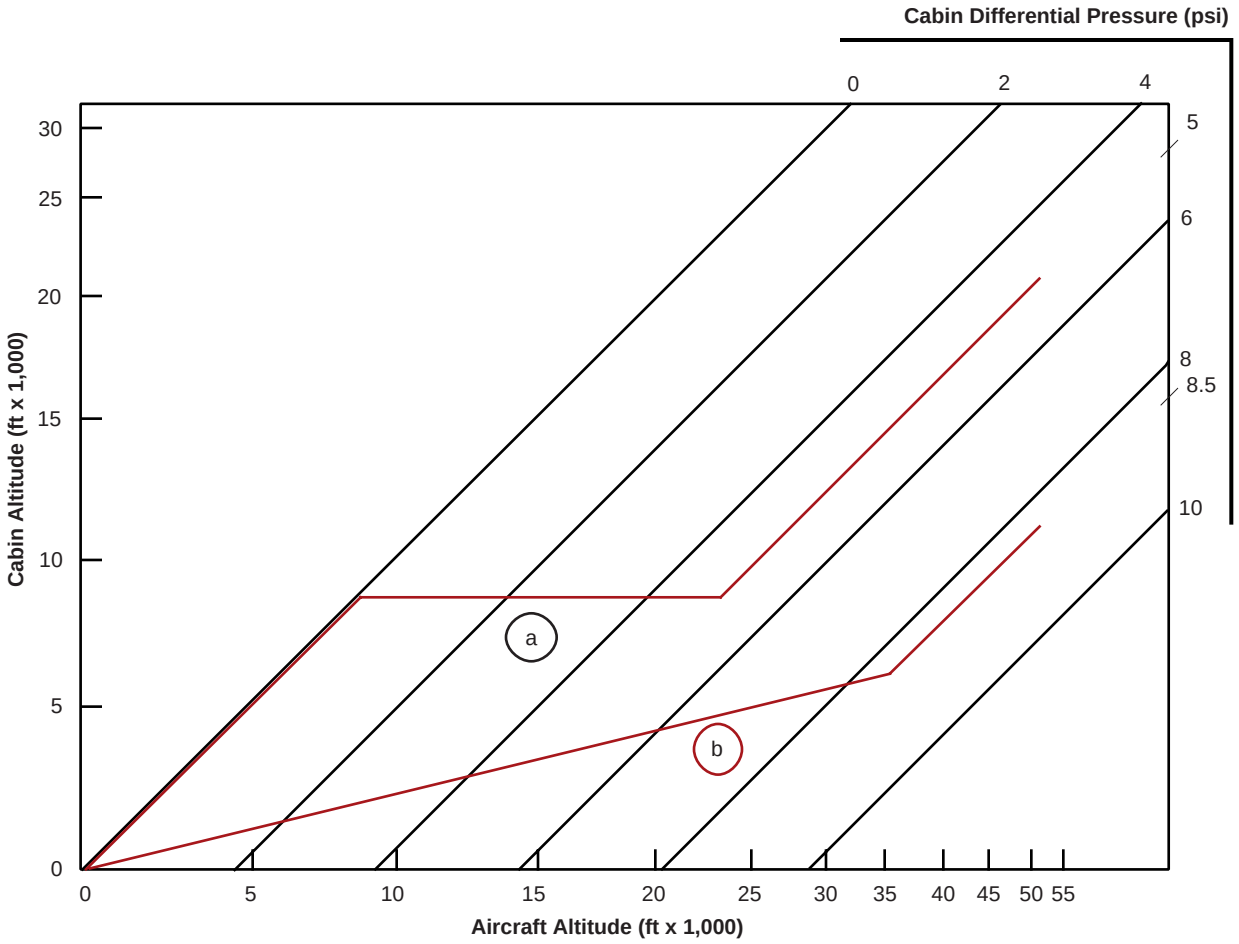

Fig. 32-5. The relation between cabin altitude and aircraft altitude, and the change in differential pressure for two general types of aircraft. Curve (**a**) is an isobaric schedule, of the type used in US combat aircraft. After a period of parallel rise in aircraft and cabin altitude, near Earth's surface, cabin altitude is thereafter kept constant (in this example, at 8,000 ft) until the maximum differential pressure is reached (here, at 5 psi), and cabin altitude again increases with aircraft altitude. Curve (**b**) is an example of the pressurization schedule used for commercial aircraft, in which passenger comfort is an important issue. In this form, cabin altitude is allowed to rise only slowly from ground level with aircraft altitude. Because a high differential pressure can be accommodated, cabin altitude can be maintained at a physiologically acceptable level throughout the aircraft's normal flight profile. Should the aircraft reach its maximum differential pressure altitude (here, 8.5 psi at 36,000 ft), cabin altitude change thereafter is obliged to parallel the change in aircraft altitude. Adapted from Harding RM, Mills FJ. Problems of altitude. In: Harding RM, Mills FJ, eds. *Aviation Medicine.* 3rd ed. London, England: British Medical Association; 1993: 61.

rushing over the defect in the pressure cabin: this "aerodynamic suck" can further reduce cabin pressure and thus increase effective altitude by as much as 10,000 ft.

The biological effects of RD include gas expansion, hypoxia, cold, and decompression sickness. Lung damage can occur if the occupants hold their breaths during the event or if the decompression is so severe that it produces a transthoracic pressure differential of 80 to 100 mm Hg. The profile of the decompression depends on both the differential pressure at the moment of pressure loss and the ratio of cabin volume to the size of the defect. The high-differential systems used in passenger aircraft dictate the need for small windows and fail-safe doors: following the loss of a typical passenger window at 40,000 ft, cabin pressure falls gradually to ambient in about 50 seconds, during which the pilot can accomplish emergency descent. In combat aircraft, on the other hand, decompression to am-

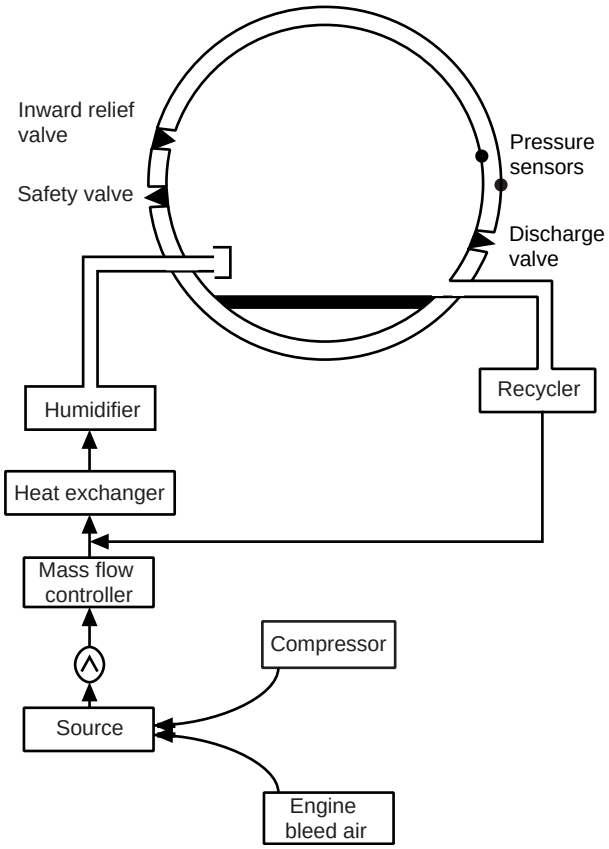

Fig. 32-6. A cabin pressurization system controls not only air pressure within the cabin but also its relative humidity, mass flow, volume flow (ventilation), and temperature. Most of the demands on such a system are based on the requirements for cabin conditioning rather than pressurization. In combat aircraft, the system is simplified by the omission of the humidifier and the recycler.

bient may be almost instantaneous, and the low differential pressure systems of combat aircraft are used in part to mitigate the effect of a large defect in a small cabin volume. In any RD, wind and flying debris within the cabin and through the defect promote confusion and difficulty with hearing and vision.

Personal Oxygen Equipment

The primary purpose of personal oxygen equipment for use in aircraft is to prevent the hypoxia associated with ascent to altitude by maintaining P_{AO_2} at its sea-level value of about 103 mm Hg. In achieving this aim, however, a number of other factors of both a physiological[12,13] and a general[14] nature must be considered. The physiological requirements of oxygen systems, discussed below, include adequate oxygen, nitrogen, and ventilation and flow, all at adequate pressure; positive-pressure breathing; minimum added external resistance; and a means to disperse the expirate. The general requirements of oxygen systems are summarized in Exhibit 32-7.

Physiological Requirements of Oxygen Systems

Adequate Oxygen at Adequate Pressure. Sea-level P_{AO_2} can be maintained during ascent by progressively increasing the percentage of oxygen in the inspired gas (termed *airmix*) until 100% oxygen is provided at 33,700 ft. At this altitude, P_B is 190 mm Hg, P_{AO_2} is 103 mm Hg, the partial pressure of water vapor in alveoli (P_{AH_2O}) is 47 mm Hg, and P_{ACO_2} is 40 mm Hg. Thus, P_{AO_2} at 33,700 ft when breathing 100% oxygen will be the same as P_{AO_2} at sea level when breathing air; this is an example of the concept of equivalent altitudes. Continued ascent will reduce P_{AO_2} even when 100% oxygen is breathed, but healthy individuals will not experience severe hypoxia until 40,000 ft is reached, where P_B is 141 mm Hg and P_{AO_2} is 54 mm Hg (note that the values of P_{AH_2O}, 47 mm Hg, and P_{ACO_2}, 40 mm Hg, are unchanged from 33,700 ft and in fact are unchanged from sea level). This altitude is the upper limit for safe ascent while breathing air at ambient pressure. It would simplify matters if 100% oxygen could be provided at all altitudes, but this would be wasteful as well as producing potential problems with acceleration atelectasis and ear blocks (ie, oxygen ear).

Positive-Pressure Breathing. Above 40,000 ft, hypoxia can be prevented only by providing 100% oxygen under pressure that exceeds P_B by enough to maintain alveolar pressure at 141 mm Hg (called positive-pressure breathing, or PPB). PPB may be applied to the airway using a tightly fastened oronasal mask, but this is uncomfortable and causes distension of the upper respiratory tract, difficulty with speech and swallowing, and spasm of the eyelids due to pressurization of the lachrymal ducts. Whether pressure is applied by mask or by other means (see below), PPB distends the lungs and expands the chest. Overdistension can be prevented by training in the technique of PPB, but even so there is a tendency for inspiratory reserve volume to fall and expiratory reserve volume to rise: pulmonary ventilation may increase by 50% when breathing at a positive pressure of 30 mm Hg. The associated fall in P_{ACO_2} means that hyperventilation is a feature of PPB, although this too can be mini-

EXHIBIT 32-7

GENERAL REQUIREMENTS OF OXYGEN SYSTEMS

Safety Pressure. A slight but continuous overpressure in the system ensures that any leaks will be outboard and prevents the inspiration of hypoxic air.

Optional 100% Oxygen. The ability to select 100% oxygen and positive pressure manually at any altitude provides emergency protection from smoke and fumes. The selection of 100% oxygen is also the first line of treatment should decompression sickness occur.

Simplicity. Insofar as possible, the system should be convenient to use and automatic in operation.

Confirmation of Integrity. The system should allow the user to test its integrity before take-off, confirm normal gas supply in flight, and provide clear warning of any degradation in performance.

Back-up Systems. Military aircraft generally provide a secondary breathing regulator, for use should the primary device fail, as well as a small bottle of reserve oxygen for use if the primary supply fails or is contaminated.

Protection During High-Altitude Escape. A reserve oxygen supply is mounted on the ejection seat of combat aircraft to provide 100% oxygen for use after aircraft abandonment during descent to below 10,000 ft. This reserve, which is usually physically the same as the back-up system described above, is termed the emergency oxygen supply (known colloquially as the EO or Green Apple).

Ruggedness. All items of personal oxygen equipment must function satisfactorily in the extreme environmental conditions of flight (ie, pressure, temperature [especially cold], acceleration, vibration, and windblast). The plight of aircrew whose craft have entered water must also be considered, and antidrowning valves, to prevent water inhalation, are frequently incorporated in components that may be immersed in such circumstances. Antisuffocation valves in the facemask ensure that air breathing remains possible.

mized by training. The cardiovascular effects of PPB include peripheral pooling, impaired venous return, and reduced central blood volume; if there is a loss of peripheral arteriolar tone, then tachycardia and a gradual fall in blood pressure will lead to a collapse resembling a simple vasovagal syncope.[15]

Counterpressure garments provide external support to the chest, abdomen, and limbs to minimize the adverse effects of PPB. An oronasal mask alone can be used only to a pressure of 30 mm Hg, but the addition of an inflated pressure vest and anti-G suit (also called G trousers) raise the level of tolerable PPB to 70 mm Hg. Breathing pressures progressively above this require the use of a pressurized enclosed helmet and then a full-pressure suit. The overall result is that PPB has such severe disadvan-

EXHIBIT 32-8

GET-ME-DOWN AND KEEP-ME-UP

Get-me-down: an emergency life-support system designed to maintain pilot function only long enough for controlled descent of the aircraft to an altitude where ambient barometric pressure and partial pressure of oxygen are sufficient to make cabin pressurization unnecessary. An example is a positive-pressure breathing system for use at very high altitudes, where the required mask pressure can be tolerated for only a limited time.

Keep-me-up: a backup life-support system that allows the pilot to continue flying at normal altitude and perhaps complete the mission before returning to base.

tages that flyers use it for altitude protection only as an emergency "get-me-down" procedure (Exhibit 32-8). The physiology of PPB for enhancement of tolerance to sustained +Gz accelerations (PPB for G [acceleration]: PBG) will clearly be much the same as for altitude protection (PPB for altitude: PBA). But the routine use of the technique for PBG places additional constraints on systems designed for PBA, both in terms of the level of positive pressure delivered and on the design of the counterpressure garments used (see also Chapter 33, Acceleration Effects on Fighter Pilots). The physiological requirements for oxygen during acute ascent to altitude are summarized in Table 32-7.

TABLE 32-7

PHYSIOLOGICAL REQUIREMENTS FOR OXYGEN DURING ASCENT

Altitude (ft)	Source of Oxygen Required to Maintain Physiological Normality During Ascent
0–8,000	Air
8,000–33,700	Air enriched with O_2
33,700–40,000	100% O_2
> 40,000	100% O_2 under pressure

Adequate Nitrogen. To avoid acceleration atelectasis, the inspired gas should contain at least 40% nitrogen, provided that the requirements to protect against hypoxia are not compromised. Figure 32-7 depicts the physiological requirements for the composition of inspired gas (airmix) in relation to cabin and aircraft altitude.[16] The aircraft and cabin altitude areas are directly related to each other (and so create a representative or typical cabin differential profile). They are, therefore, also both related to both of the curves in the figure.

Adequate Ventilation and Flow. The requirements for aircrew breathing systems may surprise

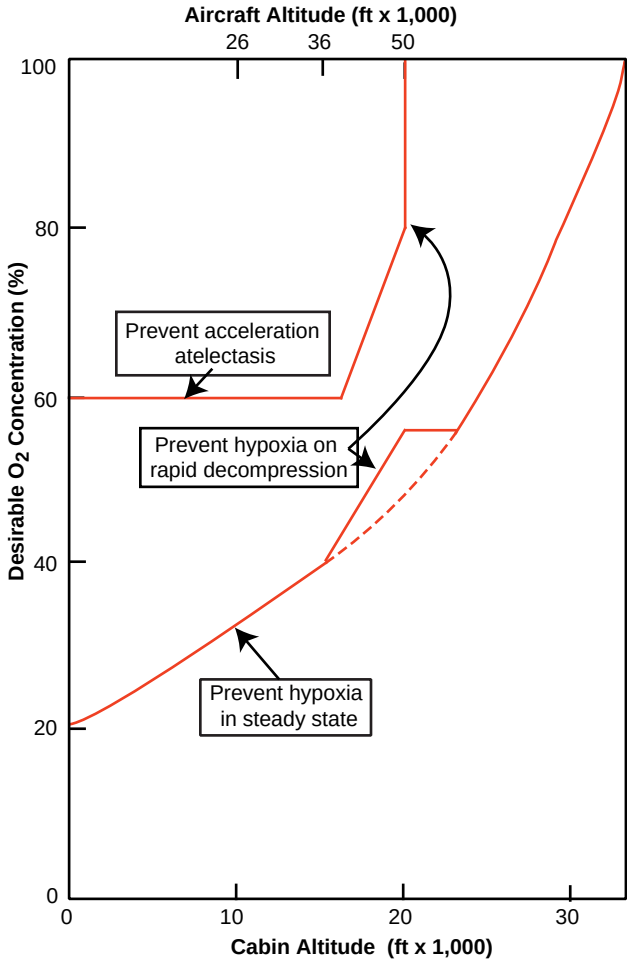

Fig. 32-7. This figure is based on the physiological requirements for inspired gas composition that are summarized in Table 32-7, and relates cabin altitude to the needed oxygen concentration. A representative cabin pressurization profile is generated by the inclusion of aircraft altitude as the upper horizontal axis. It is on this approach that design requirements for oxygen systems are based. The lower curve in the graph represents the *minimum* concentration of oxygen required to prevent hypoxia; the upper curve represents the *maximum* oxygen concentration acceptable if acceleration atelectasis is to be avoided. The kink in the "hypoxia" (lower) curve, the precise position of which will vary with the cabin pressurization profile of the aircraft, reflects the additional oxygen concentration in the inspired gas required to prevent hypoxia should a rapid decompression (RD) occur from within that band of cabin altitude. Similarly, the increase in the "atelectasis" (upper) curve from 60% oxygen at a cabin altitude of about 15,000 ft (in this example) to 80% and then 100% by 20,000 ft reflects the need to breathe 100% oxygen during or immediately after an RD. The slope-to-vertical element is present because of the need to accommodate design and engineering shortfalls in breathing system performance, which would otherwise have to cope with a choke point in this physiologically critical area. Sources: (1) Ernsting J. Prevention of hypoxia—Acceptable compromises. *Aviat Space Environ Med.* 1978;49:495–502. Harry G. Armstrong Lecture. (2) Ernsting J. The ideal relationship between inspired oxygen concentration and cabin altitude. *Aerospace Med.* 1963;34:991–997.

medical officers. Current international requirements,[17] based on in-flight studies, state that an oxygen system for use by military aircrew should be able to deliver a respiratory minute volume of at least 60 L and accommodate peak instantaneous flows of 200 L/min at a maximum rate of change of 20 L/s^2 (all volumes are at ambient temperature and pressure, dry [ATPD]). The greatest demand placed on a breathing system is on the ground prior to takeoff, especially if the pilot ran to the aircraft. Unfortunately, breathing system performance is optimized for function at altitude and so is usually at its worst in this situation.

Minimum Added External Resistance. Added external resistance, whether it affects the inspiratory or the expiratory phase of the respiratory cycle, produces unwanted physiological effects including reduction in minute volume, increase in the work of breathing, and feelings of suffocation. It is therefore most important that the external resistance imposed by a breathing system be kept as low as possible by using (wherever feasible) low-resistance valves and wide-bore hoses and connectors.

Dispersal of Expirate. The breathing system must disperse expired carbon dioxide to ambient (ie, to the cabin), and dead space must be kept as low as possible to avoid significant rebreathing of carbon dioxide.

Personal Oxygen Systems

Personal oxygen systems in military aviation are almost exclusively of the simple, open-circuit type (ie, expired gas is dispersed to the environment). Closed-circuit systems are inherently unsuitable for the robust world of military flying, in that they are complex, their components tend to freeze readily, and nitrogen and carbon dioxide may accumulate.

There are two main types of open-circuit system: continuous-flow systems, which provide gas at a fixed flow throughout the respiratory cycle, and demand-flow systems, which provide gas flow to the user only when an inspiratory demand is made. Demand-flow systems are found in most high-performance military aircraft and as the emergency supply on the flight decks of transport aircraft.

Oxygen is provided either from an onboard store of gaseous or liquid oxygen, which is replenished when the aircraft is on the ground, or from an onboard system (eg, a molecular sieve, discussed below), which produces oxygen in flight.[14] Solid, inert, chemical forms of oxygen storage are used for some emergency systems for passenger use. Whatever the source, however, the oxygen delivered must be of a very high standard; for systems other than molecular sieves, the gas must be at least 99.5% pure with a water content, at standard temperature and pressure, lower than 0.005 mg/L and with defined (low) levels of toxic contaminants.

Gaseous Oxygen Storage. Oxygen gas is stored in steel cylinders of various capacities (400–2,250 L), usually at a pressure of 1,800 psi. The size and number of cylinders depend on the type of aircraft and its role. Oxygen gas storage has the advantages of being simple in construction, easily replenished worldwide, available for use immediately after charging, and secure from loss when not in use. But it also has the great disadvantages of being heavy and bulky. Such storage is therefore used when weight and bulk are of less importance: in some military training aircraft; in transport aircraft, where the supply is intended for use by crew and passengers should cabin pressurization fail, or as the supply for small, portable, and therapeutic oxygen sets; and in combat aircraft as the (small) emergency oxygen supply.

Liquid Oxygen Storage. Liquid oxygen (LOX) is stored in double-walled, insulated, steel containers (rather like vacuum flasks)—called LOX converters—at a pressure of 70 to 115 psi and at a temperature lower than –183°C. Above that temperature, 1 L of LOX vaporizes to produce 840 L of oxygen gas. This expansion ratio makes LOX an attractive source of breathing gas for small combat aircraft, where weight and bulk are at a high premium. The capacity of the LOX converter will depend on the size of the aircraft and its role, but is usually 3.5, 5.0, 10.0, or 25.0 L.

Operation of a LOX system is complex and involves three distinct phases:

1. *filling*, during which liquid oxygen is delivered from an external supply until evaporation and consequent cooling reduces the temperature of the system to –183°C, at which point the converter fills with the liquid;
2. *buildup*, during which LOX is allowed into an uninsulated part of the circuit to evaporate before passing in gaseous form back into the converter, and in so doing raising the operating pressure of the system to the required level; and
3. *delivery*, during which oxygen gas is withdrawn from the system by the user.

LOX storage systems have considerable disadvantages, including

- dangers of handling,
- wastage of oxygen (because much LOX is lost before ever reaching a converter),
- continuous loss after charging because insulation is (necessarily) imperfect (≤ 10% can be lost in 24 h),
- a finite time requirement to reach operating pressure (which means that the storage system is not usable immediately after charging), and
- a potential for the buildup of toxic contaminants with boiling points higher than that of oxygen.

LOX systems are also prone to temperature stratification, a phenomenon whereby layering of LOX at different temperatures occurs within the converter. Subsequent agitation during taxi or flight causes disturbance of these layers, such that colder LOX comes into contact with the gaseous phase, with resulting condensation of the latter and a fall in system pressure. This problem is overcome in some combat aircraft by disturbing the stratification (ie, bubbling a small supply of oxygen gas back through the liquid from which it evolved, so agitating the fluid and eliminating any strata within it) so that the temperature is slightly elevated and uniform throughout the LOX: the system is then said to be stabilized. Despite all of these drawbacks, LOX remained the storage medium of choice for small combat aircraft until recent years, when molecular sieve technology provided a realistic alternative.

Solid Chemical Storage. When ignited with finely divided iron, sodium chlorate burns to produce copious amounts of pure oxygen (Equation 2):

$$(2) \qquad NaClO_3 + Fe \longrightarrow FeO + NaCl + O_2$$

This exothermic reaction is the basis of the solid chemical storage of oxygen used in some passenger-carrying aircraft as a source of emergency supply. Additional advantages include the simplicity, convenience, and long shelf-life of such devices (known as *candles*), although the nature of the reaction makes it unsuitable for use as a supply for a primary oxygen system. (Combustion of oxygen candles in the cargo hold is presumed to be the basis of the loss in the Florida Everglades of the ValuJet DC-9 aircraft in May 1996.)

Onboard Oxygen Production. The onboard production of oxygen overcomes all the logistical and operational penalties of conventional storage systems. Although many physicochemical techniques have been investigated in the search for an effective means of producing oxygen onboard an aircraft, the adsorption of nitrogen by molecular sieve material has proven to be the most successful and has led to the development of molecular sieve oxygen concentrating (MSOC) systems.[14]

Molecular sieves are alkali metal aluminosilicates of the crystalline zeolite family with an extremely regular structure of cages, the size of which determines the size of the molecules that can be held in the sieve. The sieving process is exothermic and depends crucially on pressure, an increase in which enhances adsorption. Pressurization of the sieve (by compressed air from the aircraft engines) causes adsorption of nitrogen molecules and allows oxygen and argon to pass through as the product gas (to a maximum concentration, at present, of 94% oxygen). However, once all the cages are occupied by nitrogen molecules, then nitrogen, too, will appear in the product gas. Depressurization of the sieve allows adsorbed nitrogen to be released from its cages, so that the technique is reversible. An MSOC therefore consists of two or more beds of sieve material used alternately to concentrate oxygen and clear out nitrogen. Because a two-bed MSOC capable of providing the needs of two crewmembers has the same weight and volume as a 10-L LOX converter, requires no coolant, and consumes just 50 W of 28-V direct electrical current, it is an overwhelmingly attractive alternative to conventional storage devices.

The use of an MSOC system does, however, raise some difficulties, including failure of the sieve to produce oxygen if engine power is lost (there is no reserve oxygen in the sieve), or if the aircrew eject from the aircraft at high altitude. Both cases require instantaneous switching to a small gaseous oxygen bottle. Similarly, if the aircraft cabin decompresses at high altitude, a supply of oxygen gas is required during the time needed for the MSOC to respond to the new pressure–altitude condition. In addition, during routine flight, 94% oxygen is too rich to breathe, and a means to dilute it is necessary. This can be accomplished either by prolonging the pressurization cycle time of the MSOC beds, thereby allowing nitrogen to appear in the product gas, or by increasing the flow of gas through the MSOC beds, which has the same effect.

These relatively minor difficulties are outweighed by the clear operational and logistical benefits of MSOC, compared with those of conventional systems. Such benefits include

- the elimination of ground recharging of the oxygen store, and therefore promotion of speedier and safer turnaround of aircraft;
- the elimination of ground manufacture, storage, and transport of oxygen;
- the elimination of the risk of contamination of breathing gas, which exists with LOX; and
- an increase in the overall reliability of the oxygen system, with a consequent reduction in the frequency of routine maintenance.

Oxygen Delivery. The simplest way in which breathing gas from any source can be delivered to the user is by a *continuous-flow* system. Although wasteful, such systems are used to provide oxygen for bail-out and emergency use, and some have been adapted for use by high-altitude parachutists. The interposition of a reservoir between the oxygen supply and the user decreases consumption by 50% to 70%, and although such systems are used for passengers in commercial aircraft, they are not suitable for more-complex applications. Some of these systems, also, have been adapted for use by high-altitude parachutists.

The *demand-flow* system, on the other hand, is more complex. In this system, the flow of gas varies in direct response to inspiration by the user, and it is possible to provide controlled airmix, PPB, safety pressure, and an indication of supply and flow. The key component in the provision of these requirements is the regulator, which is termed a *pressure-demand oxygen regulator* if it is capable of delivering gas under increased pressure.[14] Originally, demand regulators needed to be relatively large to accommodate the size of the control diaphragm and mechanical linkages required to maintain inspiratory resistance at a tolerable level, and so were panel-mounted (ie, they were located in a convenient place on a console). This continues to be the site of choice in most US combat aircraft, as well as in transport and commercial aircraft, where space is less critical. In other countries, the evolution of pneumatic engineering and the ability to miniaturize control surfaces, driven by the increasing demand placed on console space by avionics, led first to man-mounted and then to seat-mounted regulators. Man-mounted regulators are very expensive and complex, prone to damage, and one is required for each crewmember. Seat-mounted regulators overcome these drawbacks and the fault-correction drills are less complicated for the aircrew to follow: the ejection seat is now the site of choice for this component in many non-US combat aircraft.

All demand regulators, wherever located within the cockpit, are designed similarly to enable them to provide various specific automatic and manual functions (Exhibit 32-9). The precise routing of breathing gas to the user from the regulator will depend on the location of the latter and whether an ejection seat is used.

Oxygen Delivery Mask. The final component of a personal oxygen system is the oxygen mask.[14] Masks designed for continuous use by aircrew must satisfy several design requirements, including stability and comfort over long periods, small size to avoid restriction of visual fields, and minimum dead space to avoid rebreathing exhaled carbon dioxide. The mask must be available in an appropriate range of dimensions to fit and seal against all shapes and sizes of face, and the material from which it is made should neither sensitize skin nor itself be adversely affected by human secretions.

All masks designed for use with pressure-demand regulators are of a similar basic design, which includes a flexible molded facepiece (sometimes with a reflected edge) that seals against the face when the mask cavity is pressurized. The facepiece contains openings (ports), in which are mounted the valves appropriate to its role, and the mask microphone; the whole is supported by webbing straps or a rigid exoskeleton, which also provides the means by which the mask is suspended from the protective helmet or headset.

It is clear that the mask used in all open-circuit oxygen systems will require at least a simple expiratory valve so that cabin air cannot be inspired. If, however, the system is capable of delivering safety pressure or PPB, then the expiratory valve must be compensated to prevent its opening under conditions of raised mask cavity pressure. The associated inspiratory valve should be placed high in the mask to minimize the risk of obstruction by debris and to reduce the chance of contact with moist expirate, which can freeze.

Specialized Systems: Pressure Clothing

Extreme altitudes may require additional personal protection by means of pressure clothing.[14,18] In aviation, such clothing is usually worn uninflated and is pressurized only if the cabin altitude exceeds a certain level or if it is necessary to abandon the aircraft at high altitude (> 40,000 ft). Pressure clothing ranges from a full-pressure suit, which applies pressure to the whole person, to partial-pressure

EXHIBIT 32-9

AUTOMATIC AND MANUAL FUNCTIONS OF DEMAND REGULATORS

- Demand regulators provide breathing gas as needed and are essentially boxes divided into two compartments by a flexible control diaphragm: one compartment is open to the environmental pressure of the cockpit, while the other (the demand chamber) communicates at one end with the delivery hose to the user, and at the other with the oxygen supply line. A demand valve in the regulator governs the latter communication. The inspiratory demand of the user creates a negative pressure within the demand chamber, draws the diaphragm inward, and, by a pivot mechanism, opens the demand valve. Gas flows into the regulator and thence to the user until inspiration ceases. Pressure within the demand chamber then rises until the control diaphragm returns to its original position, thereby closing the demand valve.
- Air dilution (airmix) is required to avoid unnecessary wastage of stored gas and to overcome the undesirable effects of breathing 100% oxygen when it is not needed. In systems based on gaseous or liquid storage of oxygen, dilution is achieved by mixing stored gas with cabin air inside the regulator. Oxygen enters the demand chamber through an air inlet port and injector nozzle, which entrains cabin air by a Venturi effect. With altitude, the degree of entrainment must decline so that the valve that allows entry of cabin air is progressively closed by the expansion of an aneroid capsule. The injector dilution mechanism delivers 40% to 50% oxygen during quiet breathing at low altitudes. If toxic fumes are present in the cockpit or if decompression sickness is suspected, then 100% oxygen can be selected manually at any altitude. Many modern oxygen regulators also incorporate a pressure-loaded valve at the air inlet so that air cannot be drawn in unless oxygen pressure is present. This facility removes the risk of unwittingly breathing hypoxic air through the air inlet port should the oxygen supply fail.
  In systems based on molecular sieves, air dilution is accomplished by manipulating oxygen concentration upstream of the regulator. Regulators in such systems therefore have no airmix facility.
- Safety pressure is the slight overpressure (usually ~ 1–2 mm Hg) generated within the mask cavity, which ensures that any leak of breathing gas as a result of an ill-fitting mask will be outboard, thus preventing the risk of hypoxia by inadvertent dilution of breathing gas with cabin air. Safety pressure is achieved by applying an appropriate spring load to the regulator control diaphragm so that the demand valve opens slightly, gas flows downstream, and the required pressure builds up. Once pressure is attained, the diaphragm returns to its resting position and the demand valve shuts. Because the presence of safety pressure within the mask will slightly increase the resistance to expiration, it is usually only initiated (by means of an aneroid control) at cabin altitudes higher than 10,000 ft.
- Pressure breathing is also achieved by spring loading the regulator control diaphragm. The load is once again determined by the expansion of an aneroid, but in this case it progressively increases with altitude so that the magnitude of pressure breathing likewise increases. As a safety margin, pressure breathing usually commences at about 38,000 ft instead of at the theoretical level of 40,000 ft. In regulators that are also capable of providing pressure breathing for G (the unit of acceleration) protection, loading of the pressure breathing module for that purpose is accomplished by a signal from the G valve.
  The presence of a raised pressure within the mask, whether it be safety pressure or pressure breathing, requires that the nonreturn expiratory valve be suitably modified to prevent the continuous loss of pressurized gas from the mask. This is achieved by delivering gas to the back of the expiratory valve at the same (inspiratory) pressure as that passing into the mask, a technique known as compensation. The presence of a connection between the inspiratory pathway and the expiratory valve in turn mandates the need for an inspiratory nonreturn valve if the pressure of expiration is not to be transmitted to the back of the expiratory valve and so hold it shut.
- Function of the demand valve can be confirmed visually by the operation of a flow indicator, which usually takes the form of an electromagnetic circuit completed by the deflection of a small diaphragm in response to flow of gas into the regulator. The presence of flow completes the circuit and causes the magnetic indicator to show white. When flow ceases, the circuit breaks and the indicator shows black. The device therefore operates in time with respiration, and correct function should be confirmed at regular intervals throughout flight. The magnetic indicator is usually an integral part of a panel-mounted regulator but, for obvious reasons, is placed on a visible console in man-mounted or seat-mounted systems.

(**Exhibit 32-9** *continues*)

(**Exhibit 32-9** *continued*)

- In gaseous or liquid oxygen storage systems, an indication of the contents remaining is invariably available to the user, as is an indication of system pressure, a low-pressure warning display, or both.
- Apart from the manual override of the airmix facility, panel-mounted systems incorporate a means to confirm the safety pressure function, while all systems have an ON/OFF switch and a facility to test the pressure breathing function.

garments, which pressurize the respiratory tract simultaneously with a greater or lesser part of the external surface of the body.

In most circumstances, in the event of a failure of cabin pressurization, an immediate descent will be initiated. But operational constraints may require that a military aircraft remain at high altitude until its mission is completed. Thus, pressure clothing may be used either to provide the short-term protection needed until descent can be made to a safe altitude, or to provide long-term protection so that the aircraft and its crew can remain safely at high altitude. The latter can only be attained by using a garment that maintains a pressure equal to or greater than 280 mm Hg around the body, both to prevent hypoxia and altitude DCS and to provide heat to maintain a satisfactory thermal environment. The only form of garment that can fulfil these requirements is a full-pressure suit (for further discussion, please see Chapter 34, Military Space Flight). If the aircraft is able to descend rapidly, however, physiological protection is needed only against hypoxia. Again, a full-pressure suit is the ideal solution because

- it applies the required pressure in an even manner to the respiratory tract and to the entire external surface of the body, and
- pressure gradients between different body parts do not occur; therefore,
- no serious physiological disturbances arise in the cardiovascular system or in the respiratory system.

A full-pressure suit is bulky and all-enveloping, however, and hinders routine flying even when uninflated; for this reason, partial-pressure garments are often a rational alternative. From the physiological standpoint, counterpressure should be applied to as much of the body as possible, but the advantages of the "partial" principle, however (low thermal load, less restriction when unpressurized, and greater mobility when pressurized), make it desirable that counterpressure should be applied to the minimum area of the body. Thus, the proportion of the body covered by partial-pressure garments is a compromise between physiological ideal and operational expediency. Furthermore, because partial-pressure assemblies for high-altitude protection are only used for very short exposures, certain compromises with regard to moderate hypoxia are also acceptable: different considerations apply to the use of such assemblies when used in support of PPB as a means of enhancing tolerance to high, sustained +Gz accelerations.

Using a mask alone, the maximum breathing pressure that can be tolerated is about 30 mm Hg. For a P_{AO_2} of 60 mm Hg to be maintained (ie, an absolute lung pressure [which equals breathing pressure plus environmental pressure] of 141 mm Hg), respiratory protection to an altitude of 45,000 ft is possible. A P_{AO_2} of 45 to 50 mm Hg is acceptable, however, and will provide protection for 1 minute against the effects of loss of cabin pressurization up to 50,000 ft, provided that descent is undertaken to below 40,000 ft within 1 minute. The combination of a PPB mask and a suitable oxygen regulator is widely used to provide this level of get-me-down protection (see Exhibit 32-8).

Trunk counterpressure may be applied by a partial-pressure vest, which comprises a rubber bladder restrained by an outer inextensible cover. The bladder usually extends over the whole of the thorax and abdomen as well as the upper thighs (to avoid the risk of inguinal herniation at high breathing pressures). The bladder is inflated to the same pressure as that delivered to the respiratory tract, but PPB at 70 mm Hg with counterpressure to the trunk alone may induce syncope as a consequence of the large displacement of blood to all four limbs. This circulatory disturbance can be reduced by applying counterpressure to the lower limbs via G trousers. Both the vest and the G trousers may be inflated to the same pressure, or the latter may be inflated to a greater pressure than that being delivered to the vest and mask. If the absolute pressure within the lungs is maintained at 141 mm Hg, the combination of mask, vest, and G trousers will provide protection to a maximum altitude of 54,000 ft. Again, however, a certain degree of hypoxia is acceptable, and a breathing pressure of 68 to 72 mm Hg can be employed at 60,000 ft, where it will provide an absolute pressure in the lungs of 122 to

126 mm Hg, and a P_{AO_2} of 55 to 60 mm Hg. The combination of the discomfort of a high breathing pressure in the mask and a certain degree of hypoxia limits the duration of protection afforded by this ensemble to about 1 minute at 60,000 ft, followed immediately by descent at a rate of at least 10,000 ft/min to 40,000 ft for a total of 3 minutes.

When the required level of PPB exceeds the physiological limits associated with the use of an oronasal mask, use is made of oxygen delivery via a partial-pressure helmet, while a vest provides counterpressure to the trunk, and G trousers to the lower limbs. Partial-pressure helmets give support to the cheeks, the floor of the mouth, the eyes, and most of the head, thereby eliminating the uncomfortable pressure differentials that develop between the air passages and the skin of the head and neck when an oronasal mask is employed. Even when a partial-pressure helmet is used, however, severe neck discomfort may occur during PPB at pressures greater than 110 mm Hg. Finally, it would be physiologically beneficial to include the upper limbs in the areas to which counterpressure is applied (the vest being sleeveless), and a wide variety of garments have been employed to provide this extensive coverage in combination with a pressure helmet.

SUMMARY

The substrate for all aviation is Earth's atmosphere. Although its gas composition is essentially constant, ascent from ground level to the edge of space is accompanied by an exponential decrease in P_B and large, predictable changes in temperature and radiation. As balloons and then airplanes carried humans high above Earth, problems were encountered that were caused by hypoxia, hyperventilation, and altitude DCS; the advent of pressurized aircraft, with their potential for RD, added barotrauma to this list.

Although altitude DCS involves the same mechanisms as diving DCS, there are differences in symptom patterns and treatment: descent to ground level while breathing 100% oxygen constitutes the first and often sufficient line of therapy for victims of altitude DCS, whereas divers must be returned to a hyperbaric environment for treatment. The physiological problems of atmospheric flight are prevented by means of pressurized aircraft cabins, inhalation of oxygen-enriched gas through a mask or pressurized garments, or both. Oxygen supplies may be carried on board as a compressed gas or in liquid form, generated by chemical reactions or scavenged from outside air by onboard molecular sieves.

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