Chapter 3

EFFECTS OF DECREASED BAROMETRIC PRESSURE—DYSBARISM

Dysbarism is a general term that includes all the physiologic effects resulting from changes in barometric pressure with the exception of hypoxia. It is worth noting that this broad definition of dysbarism can include all of the disturbances within the body, which result from not only a reduction in the barometric pressure but from an increase in the barometric pressure as well. Thus, *dysbarism* is a broad term that can indicate most of the disorders that are experienced by underwater divers and caisson workers. Many of the disorders have the same etiology as those experienced by the aviator. The barometric pressure changes to which a diver or caisson worker can be subjected may be as great as several atmospheres (one atmosphere is equal to a pressure of 760 mm Hg or 14.7 psi), but the flier can be subjected only to a maximum barometric pressure change that cannot exceed 1 atmosphere. In comparing the diving and flying situations, the most serious disorders experienced by the diver are initiated by return to his normal pressure environment at sea level and can be treated only by returning again to the abnormally high pressure. On the other hand, the flier can experience similar disorders only at the abnormal environment of high altitude.

This chapter is primarily concerned with the disturbances that affect the flier as a result of reduced barometric pressures, either by exposure to high altitudes during actual flight or to simulated altitudes in a low pressure chamber. The principal disturbances that result from *increases* in the ambient pressure, such as the compression effects on the ears and sinuses during descent from altitude, are discussed in detail in chapter 6.

The syndrome resulting from a reduction in the barometric pressure is commonly referred to as *decompression sickness*, a term that includes the entire symptom complex that may develop. Other more limited terms that are sometimes used in a general sense with reference to decompression sickness are *aero-embolism* (gas bubbles in the blood vessels) and *aero-emphysema* (gas bubbles in the tissues). The specific term *ebullism* has recently been suggested to describe the unique medical syndrome with all the symptoms that can occur as a result of the boiling phenomenon at barometric pressures that are less than the vapor tension of body fluids.

The main symptoms of decompression sickness may be classified etiologically as:

a. Effects of evolved gases from body fluids and tissues resulting in such symptoms as bends, chokes, and neurological disorders (Henry's Law); and

b. Effects of expansion of trapped gases within the body resulting in such symptoms as abdominal gas pain and barodontalgia (Boyle's Law).

Etiology

The basic factors that produce this form of dysbarism at reduced barometric pressure are the expansion of trapped gas within the body cavities and the evolvement of gas bubbles in body tissues and fluids, particularly the evolvement of nitrogen that is in solution at normal barometric pressures.

On the reduction of the barometric pressure, gases within the body cavities tend to expand in accordance with Boyle's Law, and if the free escape of this expanding gas is grossly impeded or blocked, abnormally high gas pressures may develop, depending on the

volume of trapped gas, the elastic characteristics of the surrounding organs and tissues, and the extent of the decompression itself. The gastrointestinal tract, the lungs, the middle ear, and cranial sinuses are the chief gas-containing organs and cavities but, ordinarily, the expanding air within the lungs, ears, and sinuses readily escapes during ascent to high altitudes, maintaining a reasonably normal equalization of pressure with the decreasing barometric pressure. On the other hand, the abdominal gas in the gastrointestinal tract often is unable to escape readily, resulting occasionally in a severe dysbaric symptom complex.

The initiation of the disorders resulting from *evolved gases* at high altitudes is best understood by considering first the characteristics and types of gases that are normally in solution in the body fluids and tissues and which are in dynamic equilibrium with the pulmonary alveolar gases, the surrounding barometric pressure, and the metabolic state of the body at sea level. The average adult body normally contains approximately one liter of dissolved "inert" nitrogen, together with a certain amount of metabolically active oxygen and carbon dioxide that are also in solution throughout the body, as well as the ever-present water vapor tension. Moreover, an additional important factor is that body fat and lipids contain about five times as much dissolved nitrogen (and oxygen) as the fat-free tissues and fluids. Thus, an obese person actually contains considerably more dissolved nitrogen per unit of body weight than a lean person. The amount of any gas dissolved in the body is dependent on the particular solubility characteristics of the gas in various types of body fluids (water, plasma, or fat) and is also directly proportional to the partial pressure of the gas with which the liquid is equilibrated in accordance with Henry's Law for dissolved gases. Since carbon dioxide is metabolically generated within the cells and tissues, it is excreted by diffusion in large quantities from the pulmonary blood through the lungs to the essentially carbon dioxide-free atmosphere. Oxygen diffuses continually in the opposite direction from the atmosphere to the blood and tissues. The metabolically inert nitrogen, on the other hand, remains throughout the entire body in a relatively steady state of equilibrium with the nitrogen tension in the lungs and atmosphere. As the barometric pressure decreases with ascent to high altitudes, this equilibrium for nitrogen is drastically upset. The body tissues and fluids become supersaturated, and nitrogen tends to be evolved from solution as gaseous bubbles which initiate the major symptoms of this form of dysbarism. Presumably, the aching and painful symptoms of bends are produced by such bubbles located interstitially in the connective tissue about the bones, joints, and muscles where the limited capillary vascularity is inadequate for quickly carrying away the excessive accumulation of gas. The symptoms of chokes are believed to be caused by the accumulation of such bubbles intravascularly in the pulmonary capillary bed, leading to the typical rapid shallow breathing and, as has been measured in animals, a pulmonary hypertension.

It should be pointed out here that, although nitrogen is the most likely gas for maintaining a bubble, it is not clear as to which gas first initiates the submicroscopic bubble and establishes the conditions for bubble growth to a size capable of producing the symptoms of dysbarism. Water vapor, together with carbon dioxide and oxygen, may first form a relatively unstable gas nucleus into which all gases in solution can further diffuse in proportion to their gas tensions in the liquid. *For example,* carbon dioxide can enter or leave a bubble 50 times more readily than nitrogen and, of course, water vapor will be present the instant that conditions for a bubble are established.

The *evolved bubble theory* is the most convincing and attractive for explaining many of the signs and symptoms of decompression sickness, although it has not as yet been completely proven with absolute certainty. The fact, however, that denitrogenation before exposure to low barometric pressures provides considerable prophylactic

protection against dysbarism, indicates that nitrogen plays a basic role in the etiology of this syndrome. Moreover, after the onset of decompression sickness, recompression usually brings prompt and dramatic relief. On the other hand, the bubble theory fails to explain the delayed occurrence of some of the most serious neurocirculatory manifestations after recompression to ground level. Also, the fact that the incidence of aviator's decompression sickness appears to be higher in the morning than in the afternoon and evening, is difficult to explain on the bubble theory alone. There are strong indications that several other important unknown factors are also involved in the etiology and progress of this disease. Other theories that have been suggested to help explain these apparent discrepancies in the bubble theory include the *vasospasm theory*, based on the observations of Knisely, and the *fat emboli theory*, stemming from Haymaker's postmortem observations and the work of Rait, which indicate a disruption of fatty tissues with involvement of congenital cardiovascular defects and/or liver and nutritional considerations.

It has been shown that the incidence of dysbarism increases with the rate of ascent, the peak altitude, duration of exposure to altitude, exercise and muscular activity, and cold. The incidence decreases with increasing time of denitrogenation.

Individual susceptibility varies widely from person to person and within a given individual from time to time. Age is an important consideration with an increase in susceptibility for older age groups. Whether this is due primarily to age itself or to the fact that older people are often more obese is not clear. There is a good correlation with the incidence of dysbarism and obesity, probably due to the fact that the obese individual has considerably more nitrogen to eliminate than the lean person. Tissue vascularity and blood circulation are also important factors, the efficiency of which often decreases with both age and obesity. On the other hand, physical fitness and previously healed injuries to bones and joints within limits encountered in personnel on flying status, do not appear to influence susceptibility significantly. However, susceptibility is extraordinarily increased by exercise while at altitude. Exercise not only increases the incidence and severity of the symptoms at altitude, but causes the symptoms to occur sooner than when at rest. With vigorous exercise at altitudes of about 38,000 feet, 100% of the population can be expected to develop bends within 30 minutes. In addition, exercise lowers the threshold altitude for the occurrence of bends symptoms. Ordinarily, the evolved gas symptoms of severe dysbarism do not occur below the critical threshold altitude of 30,000 feet, but symptoms may be induced at altitudes as low as 22,000 feet by strenuous exercise. However, even with a person at rest, symptoms have been reported occasionally in this altitude range.

Symptoms

According to a decompression reaction study on 62,160 trainees between 1943 and 1945, the five most prevalent painful reactions that occurred during altitude chamber flights were bends (13%), aerotitis (7.86%), abdominal distresses (4%), sinus pain (1.17%), and barodontalgia. Vasomotor instability, chokes, hypoxia, visual disturbances, and hyperventilation all totaled less than 1%.

Bends are the most frequent manifestation of decompression sickness and, at altitude, are characterized by a deep pain in the bones, joints, and muscles of the extremities, including the hips and shoulders. The pain is often diffuse and poorly localized, and is felt as a boring, gnawing, or aching pain that can progress in intensity to the point of becoming intolerable and incapacitating. The onset of pain can be fulminating, but more often, it is mild and gradual. The pain can be transitory, intermittent or steady, can occur in one joint, or it can progress to several locations in the extremities with different degrees of intensity. No general rule can be stated with regard to onset, duration, intensity, location, or the length of time at

altitude before bends symptoms are first noticed, if at all, when at rest. The knees and shoulders are the locations most often involved. All degrees of bends can lead to general circulatory reactions.

Chokes usually occur later in the course of exposure to altitude than do bends and are characterized by the following: A substernal burning sensation which is referred to the deep respiratory passages; nonproductive cough arising deep within the chest; and aggravation of both of the above manifestations by a deep breath, accompanied by a sense of suffocation and apprehension. The onset is almost inevitably progressive, leading to severe distress within a few minutes. General circulatory reactions are more common with chokes than with bends. Chokes at altitude should be regarded as a dangerous symptom that can lead quickly to the most severe and grave consequences, and prompt descent or recompression should be initiated without delay.

The paresthesias frequently associated with dysbarism are generally of little consequence. Objective skin manifestations, which may occur with or without paresthesias, are seen with some frequency and take the form of either intracutaneous blebs, subcutaneous emphysema, or a mottled skin lesion. The mottled skin lesion, presenting as irregular areas of erythema adjoining areas of cyanotic pallor, is considered a serious sign since it may be associated with chokes and neurocirculatory instability or collapse. Ordinarily, prompt termination of the flight results in rapid disappearance of these symptoms with, however, a residual tenderness over the involved area, becoming maximal several hours postflight and sometimes persisting for 2 or 3 days.

Neurological symptoms occur rather infrequently. The most common type is a transitory visual defect consisting of homonymous scotoma or even hemianopsia, followed by headache, which closely resembles migraine. More rarely, transitory hemiplegia, monoplegia, aphasia, and disorientation occur. The neurological reactions differ from the other symptoms by their tendency to occur shortly after flight as well as during flight.

Abdominal pain is a common symptom resulting from trapped expanding gas. In spite of its different etiology, it occurs at about the same altitude as the other symptoms caused by evolved gas. It makes its appearance typically early in the course of the flight and may progress from a simple feeling of distention to severe, cramp-like pain. When severe, it may lead to circulatory reactions.

Barodontalgia is a painful condition of the jaws and teeth, experienced during or shortly after exposure to lowered barometric pressure in flight or in a low pressure chamber. The causative factors of this painful condition have never been fully explained. In some instances, it has been associated with subclinical pathological conditions that, ordinarily, are not bothersome to nonflying personnel. It is probable that barodontalgia does not occur with a healthy pulp. Over 9% of the fighter pilots of one command reported this type of pain at some time during their flying career.

Predisposing factors in toothache at altitude are large, deep-seated, silver fillings without underlying base materials or insulators, and various stages of inflammation or degeneration of the plup. Toothache at altitude is most likely caused by an underlying lesion in the pulp which, in time, would cause the same symptoms without decompression.

In general, the pain is worse with greater and more rapid decompression and is relieved usually by recompression. The precise "altitude of incidence," severity, and duration of pain will vary with the individual and the type of lesion in the pulp. Occasionally, pain may first appear on descent from altitude or on recompression. Available information indicates that, in some instances, pulpitis of varying degrees may be found together with "spaces" in the pulp.

When several teeth are suspected of causing pain, those with recent amalgam fillings are more probably responsible. Testing with ice may reproduce the pain. A tooth

is to be suspected if it continues to hurt after removal of the ice. A tooth with an open cavity will not be affected by altitude even though the pulp is diseased.

The character of the pain varies in intensity, duration, and location. In many cases, its severity will render the flier militarily ineffective during the periods of painful attacks. The pain may be made to recur by reproducing the same flight conditions in the altitude chamber or when actual flight conditions prevail. Barodontalgia, unlike painful disturbances in the region of the ear and sinus, is more prevalent during ascent and at altitudes. In some cases, however, it has occurred on descent or on the ground following a level flight.

Complications and Sequelae

The most serious complication of dysbarism is a type of neurogenic peripheral circulatory failure or primary shock, consisting of one or all of the following manifestations: intense pallor, profuse sweating, faintness and dizziness, nausea, vomiting, and loss of consciousness. These circulatory reactions are usually initiated at altitudes at which the primary symptoms of bends, chokes or gas pains are most severe, and recede rapidly as the primary symptoms are relieved by descent from altitude. In some instances, the reaction persists after reaching ground level and may develop into the hematogenic form of peripheral circulatory failure or secondary shock.

Delayed circulatory reactions also may occur within several hours after return to ground level. After an apparent asymptomatic interval, these delayed reactions may present the typical picture of secondary shock with weak, thready pulse, hypotension, and intense hemoconcentration. A few fatal cases of this type have been encountered. Neurological symptoms sometimes may accompany such delayed shock. Hemiplegia and coma have resulted and, in some instances, permanent residuals.

Treatment

The only effective prophylactic measure against decompression sickness is denitrogenation before ascent to altitude. By breathing air containing a reduced pressure of nitrogen, the latter is removed from the body. The most rapid denitrogenation is accomplished by breathing pure oxygen. Above 20,000 feet, the effectiveness of denitrogenation is greatly reduced. Breathing pure oxygen at ground level for 15 minutes will reduce the incidence of bends and chokes at 38,000 feet by approximately 50%. Dysbarism is rare in current operational flying as long as the cabin altitude remains below 30,000 feet.

The prophylaxis of gas pains is a more difficult problem. The only reliable guide is to eliminate, as far as possible, conditions or procedures that cause abdominal distress at ground level since symptoms of these conditions are likely to be aggravated by altitude. Figures are given in cases per 100 persons exposed.

DISTRIBUTION OF ALTITUDE CHAMBER REACTIONS

Symptom	*Air Force CY 1964*	*Air Force CY 1965*
Aerotitis	8.73	8.66
Aerosinusitis	1.59	1.86
Barodontalgia	0.23	0.30
Abdominal Gas Pain	1.96	1.85
Bends	0.31	0.30
Chokes	0.003	0.008
Neurological	0.002	0.00
Other (skin, etc.)	0.78	0.67
TOTAL	13.605	13.648

NOTE: 52,113 aircrew members trained in 1964 and 49,603 in 1965.

A failure of a reactor to respond to recompression must be viewed as a serious event. Cerebral, visceral or cardiovascular gaseous embolization at the arteriolar and precapillary levels initiates focal ischemia. Recompression exerts a positive mechanical effect on bubbles to cause a decrease in their size, but recompression cannot be expected to influence directly existing irreversibly damaged areas.

Recent successes in therapy have established the value of chamber compression to

more than one atmosphere in the treatment of severe cases of altitude dysbarism. The few severe cases that have thus far been treated have shown good recovery at 3 to 6 atmospheres absolute pressure. Several therapeutic compression chambers are located at geographically strategic sites within the Air Force. The treatment of severe cases of altitude dysbarism by this means is the current method of choice. If the patient is transported by air, cabin pressure must be maintained at or near sea level.

Involvement of motor and sensory functions in either spinal cord or cortex, visceral involvement, or direct cardiovascular involvement produces a remarkable disorder commonly termed "neurocirculatory collapse." In its mildest form, the disorder is seen as a self-limiting, transitory vasomotor instability, indistinguishable from a syncopal reaction. In its severest form, the disorder presents widespread neurological involvement and acute hypovolemic shock, with progressive deterioration to a fatal termination. Within this framework, neurocirculatory collapse may be seen as an almost uncomplicated neurological disorder, or primarily as a shock-like syndrome. Generally, both neurological and circulatory involvements are seen.

Treatment is largely empirical and expectant. Immediate recompression to ground level is required. Compression to more than one atmosphere has merit but must be considered in the light of the availability of such a specialized treatment facility. Complete bed rest must be emphasized, even in the cases of mild vasomotor instability where the patient may be asymptomatic at rest, but demonstrates postural hypotension on sitting or standing. All cases which demonstrate postural hypotension for more than an hour or two should be hospitalized and observed for at least 24 hours. Depending on the nature and severity of the disorder, supplemental breathing oxygen should be given. The use of an oxygen-carbon dioxide mixture not to exceed 3% carbon dioxide is probably useful.

For cases that are recognized as hypovolemic shock, vigorous supportive treatment is extremely important. The use of plasma expanders is necessary. These should be given in amounts sufficient to maintain an adequate hourly urine output; 30 to 50 ml/hour suggests successful replacement. The hematocrit and the vital signs (blood pressure, pulse) are important indices of successful treatment as well.

Each case is a peculiar individual event and the Flight Surgeon must be prepared to treat acute pulmonary edema, acute congestive failure, or even a cardiac arrhythmia, all of which are accompaniments of this remarkable and serious complication of evolved gas dysbarism.

Rapid Decompression

The development of the pressurized cabin in aircraft has introduced a new potential hazard for the flier in the event that this cabin pressurization is accidentally and suddenly lost, either as the result of enemy action or the spontaneous rupture of the cabin structure. It is thus necessary to consider carefully the range of human tolerance to such sudden decrease in the barometric pressure. First, it is important to distinguish between the possible effects that can occur *during* the rapid decompression itself, such as being physically injured or actually blown out of the aircraft through the opening, and the effects of the low barometric pressure that is encountered *after* decompression, such as hypoxia and dysbarism and, in addition, if above 63,000 feet, the vaporization of body fluids if adequate protective equipment is not available immediately.

The two main factors that influence the severity of a decompression are: a. The rate and time of the decompression; b. the absolute change in the barometric pressure. The faster the decompression and the greater the change in pressure, the more severe can be the effects. In turn, the basic factors that determine the rate and the severity of a decompression are: (1) The volume of the pressurized compartment; (2) the size of the opening in the cabin; (3) the pressure differential ($P_{cabin} - P_{ambient}$); (4) the pres-

sure ratio ($P_{cabin}/P_{ambient}$); and (5) the flight altitude at which the decompression takes place.

The relationship of the volume of the cabin to the size of the opening and the *pressure ratio* determines the time of a decompression (not the pressure differential). The larger the volume and the smaller the opening, the slower will be a decompression and, also, the larger the pressure ratio, the slower the decompression. Furthermore, the higher the flight altitude for any given pressure differential, the longer it will be before a cabin decompresses completely; the higher the altitude the larger the pressure ratio. (A thorough analysis of decompression times and the above relationships are in the *General Theory of Rapid Decompression* by Haber and Clamann, listed in the references.)

Figure 3–1 illustrates the theoretical expansion of body gases during decompressions up to altitudes of 60,000 feet. This relative gas expansion (RGE), however, has certain analytical limitations since it does not take into account the *volume* of the expanding gas.

The physiological effects of explosive decompression are produced by rapid expansion of gases within body cavities; the degree of decompression that can be withstood safely is determined either by the extent or the rate of expansion. When the expansion is slow, the body gases tend to escape readily

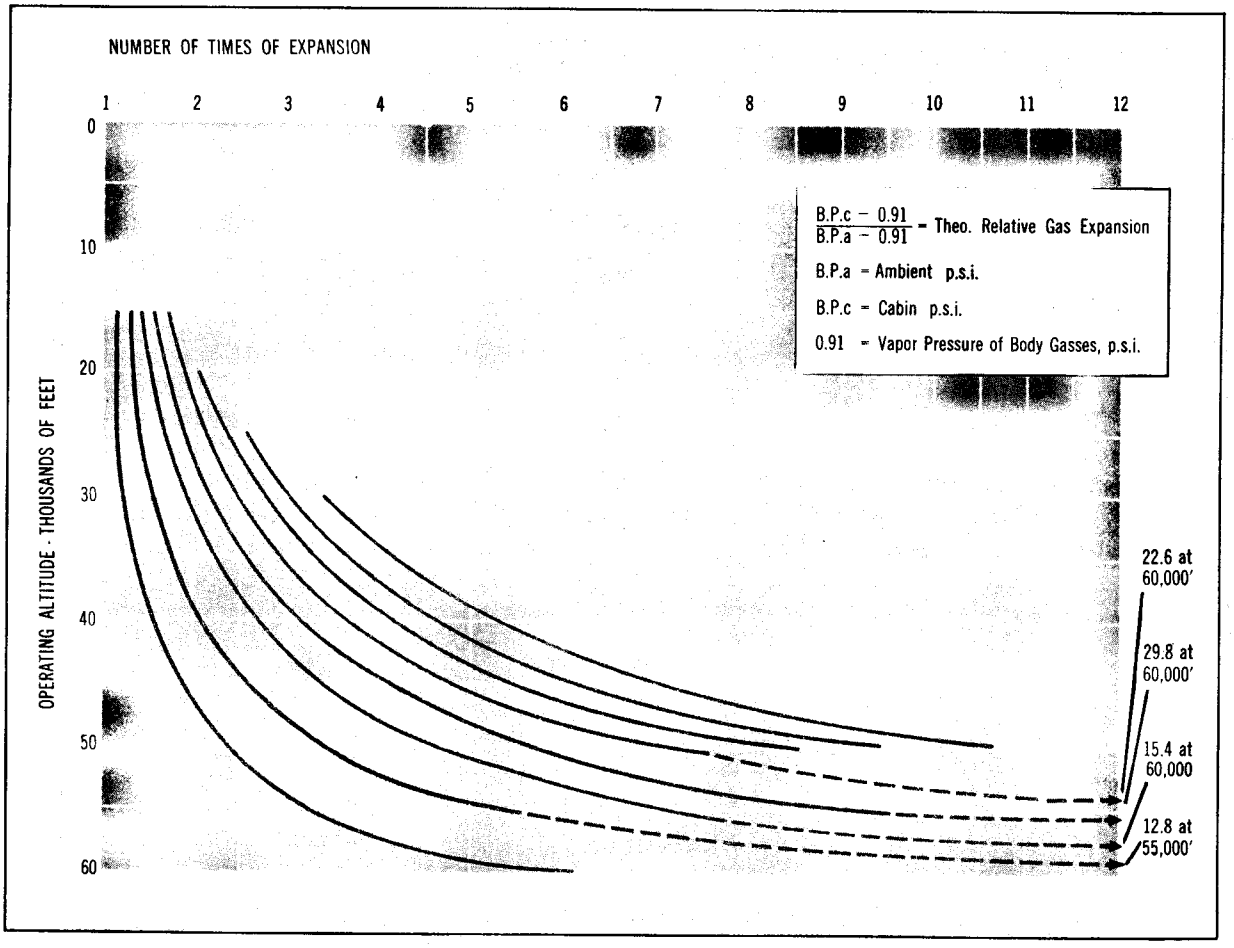

Figure 3–1. Theoretical Expansion of Internal Body Gases Upon Equalization of Cabin Differentials From 1.0 Through 8.0 psi.

or become redistributed before dangerous pressures are built up. When the decompression and the degree of expansion are slight, the lungs and hollow viscera can distend safely to make room for the expanded gas.

Expanding gas is normally expelled from the lungs with little resistance through the open airways and trachea, but if the flow of escaping gas is blocked, physiological stretching of the distended lung tissue commences when the gas volume is double the vital capacity. In decompressions with expansion of gases to several times their original volume at an extremely rapid rate, the response of the lungs is the limiting factor in tolerance of normal subjects to explosive

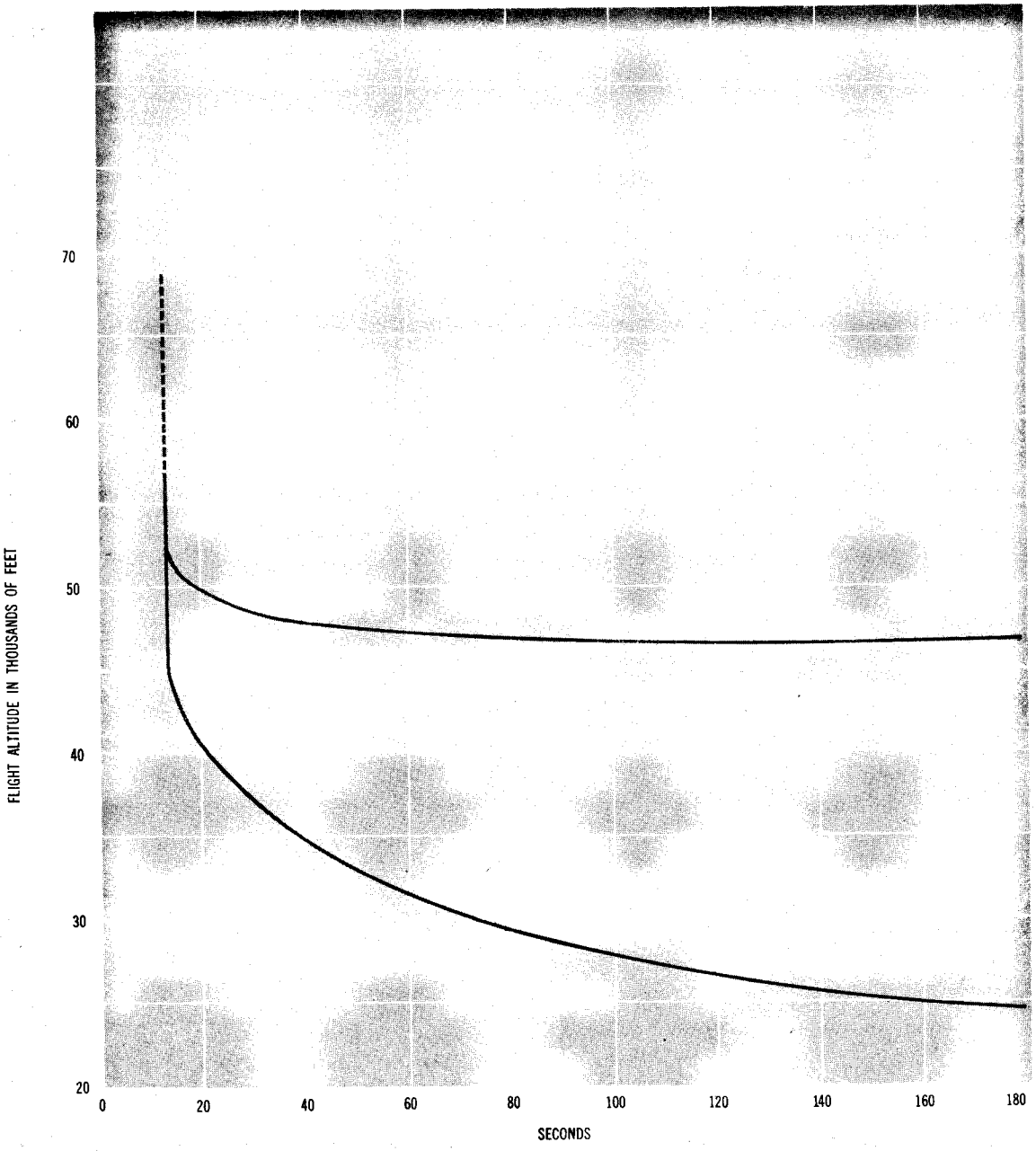

Figure 3–2. Flight Altitude in Thousands of Feet.

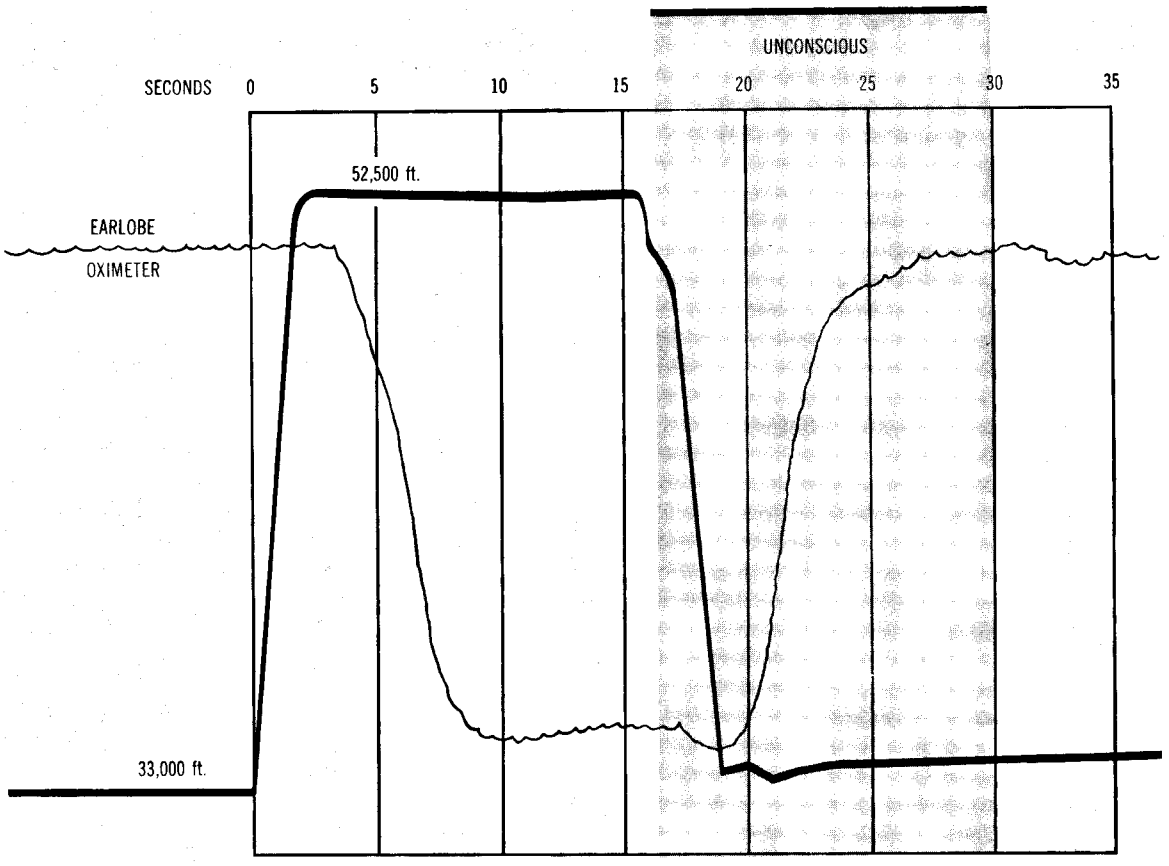

Figure 3–3. Minimum Free Interval in Which Hypoxic Manifestations Are Latent.

decompression. Critically high pressures will occur in the lungs if the glottis happens to be closed at the moment of decompression, either due to voluntary breath-holding, or, inadvertently, as in the act of swallowing. Traumatic aero-mediastinum and aero-embolism into the systemic circulation due to pulmonary lesions have been observed in a few rare instances, with one fatality.

The expansion of gases within the gastro-intestinal tract causes distention which may give rise to an occasional twinge. Persons with excessive abdominal gas, however, may suffer more severe pain. No aural discomfort has been observed during rapid decompression, possibly because the eustachian tubes are blown open immediately and remain open during the change in pressure.

If a flier must remain at high altitude following a sudden decompression, the symptoms of dysbarism may be expected to occur if adequate denitrogenation was not accomplished prior to the decompression. Experimental work with humans has revealed that extraordinarily rapid rates of decompression can be tolerated with little difficulty. Rapid decompressions from 8,000 to 35,000 feet in less than 0.1 second have been tolerated.

While the immediate mechanical effects of rapid decompression on occupants of a pressurized cabin will seldom be incapacitating, the menace of subsequent hypoxia becomes more formidable with increasing operational altitudes. The time of consciousness after loss of cabin pressure (10,000 feet or less) while breathing air is indicated in the lower curve of figure 3–2. The upper curve represents the time available on decompression to altitudes above 45,000 feet, using oxygen from a simulated cabin altitude of 33,000 feet (or less).

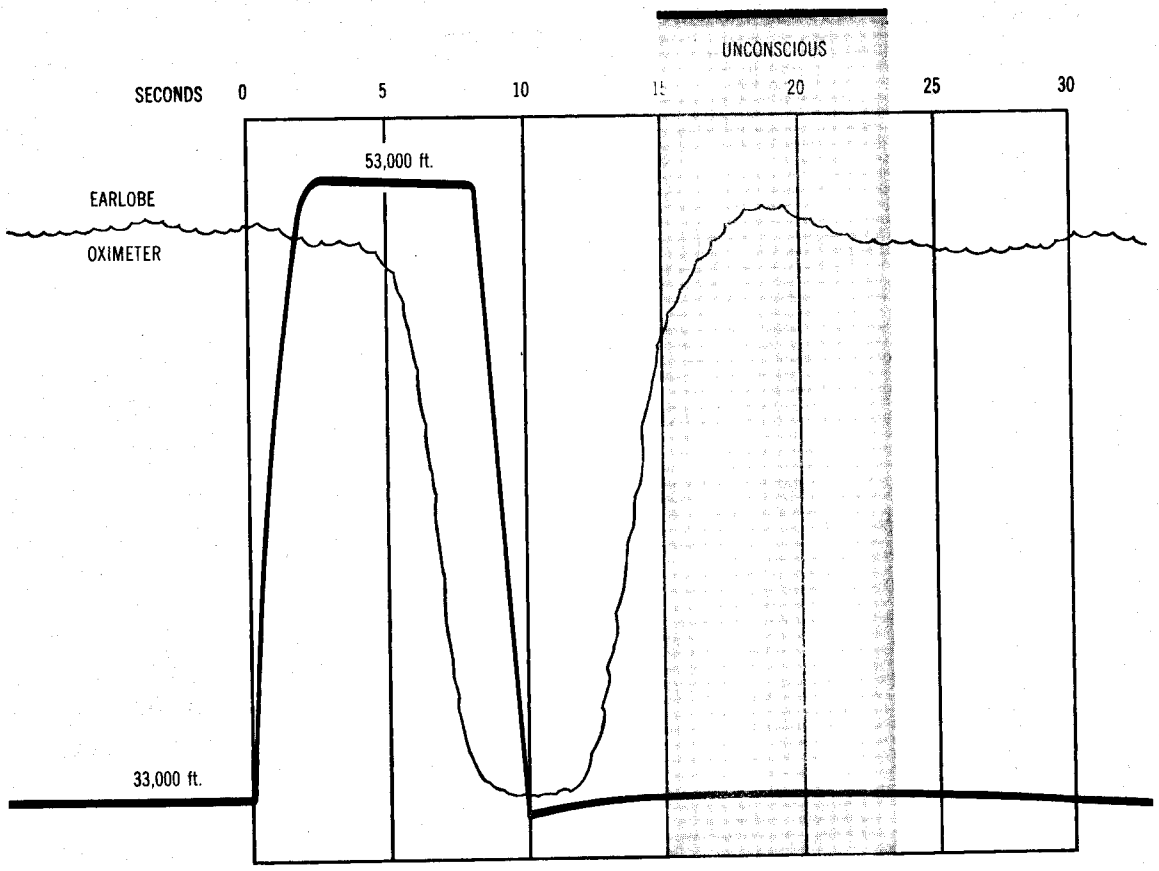

Figure 3–4. Loss of Consciousness After Rapid Decompression Is Terminated Before the Latent Period of Hypoxia Has Elapsed.

The advantage gained by breathing oxygen is most convincing up to 45,000 feet, but becomes less and less significant at higher altitudes. Both curves converge at 52,000 feet where the time of consciousness is the same, regardless of whether air or oxygen is breathed. It is not implied that the use of oxygen equipment is of no value in such an emergency. On the contrary, the chances of survival by recompression, pressure breathing, or free fall will be infinitely better when oxygen is available throughout.

As evident from figure 3–2, the time of consciousness reaches a minimum of approximately 15 seconds at 45,000 feet, breathing *air*. At 52,000 feet, the same minimum exists when *oxygen* is used. Figure 3–3 demonstrates this latent period that can be accounted for partly by the circulation time of blood from the lungs to the brain and by the tissue oxygen reserves.

If the exposure to altitude after rapid decompression is terminated before the latent period of hypoxia has lapsed (figure 3–4), loss of consciousness will ensue, nevertheless, at the time when the blood, which has been deprived of its oxygen, takes effect on the brain. This may take place even after adequate oxygen pressure has been regained in the lungs by recompression. Only when the exposure to a critical altitude (above 45,000 feet with air, above 52,000 feet with oxygen) does not exceed 5 to 6 seconds can temporary loss of consciousness be avoided by protective devices designed to become effective within that time.

For a limited approximation in predicting the possible stress on the human body during

a rapid decompression, the following two formulas have been found to be useful in predicting the probable danger limit of this stress to the human body. These two formulas are:

$$\text{RGE (calculated)} = \frac{Pc - 0.91}{Pa - 0.91}$$

$$\text{RGE (maximum)} = 2.1 + \frac{3.79\ Vc}{A}\sqrt{\frac{Pc - Pa}{Pa}}$$

where: RGE = relative gas expansion; A = total cross-sectional area in square inches of the opening in the pressurized compartment (in predicting A, the largest Plexiglas opening is suggested); Vc = volume of the pressurized compartment in cubic feet; Pa = outside atmospheric pressure in pounds per square inch; and Pc = inside compartment pressure in pounds per square inch.

If RGE (calculated) is greater than RGE (maximum), danger exists; if RGE (maximum) is greater than RGE (calculated), the operating conditions may be considered safe.

REFERENCES

The reader should insure the currency of listed references.

Adler, H. F., *Neurocirculatory Collapse at Altitude,* USAF School of Aviation Medicine, Special Project Report (June 1950).

Armstrong, Harry G., *Aerospace Medicine,* Chapters 12 & 13, The Williams and Wilkins Co., Baltimore (1961).

Benzinger, Th., *Explosive Decompression,* German Aviation Medicine in World War II, pages 395–408, Washington DC (1950).

Comfort, E. C. and Wilson, J. W., *Some Factors Affecting Time Consciousness at High Altitudes,* Air Force Technical Report No. 5970 (November 1949).

Fulton, J. F., *Decompression Sickness, Caisson Sickness, Diver's and Flier's Bends and Related Syndromes,* W. B. Saunders Co., Philadelphia (1957).

Gillies, J. A., *A Textbook of Aviation Physiology,* Chapters 5, 6, 7 & 8, Pergamon Press, Inc., New York (1965).

Haber, F. and Clamann, H. G., *A General Theory of Rapid Decompression,* Project No. 21-1201-0008, Report No. 3, USAF School of Aviation Medicine (1953).

Hall, F. G., *Factors Affecting Consciousness Time at Altitude, Part II,* Air Force Technical Report No. 6009 (September 1950).

Haymaker, W. and Johnston, A. D., *Pathology of Decompression Sickness, a Comparison of the Lesions in Airmen With Those in Caisson Workers and Divers,* Military Medicine 117:285 (1955).

Knisely, M. H. and Block, E. H., *Microscopic Observations of Intravascular Agglutination of Red Cells and Consequent Studying of the Blood in Human Diseases,* Anatomical Record, Volume 82:34 (1942).

Luft, U. C., Clamann, H. G., and Opitz, E., *The Latency of Hypoxia on Exposure to Altitudes Above 50,000 Feet,* Journal of Aviation Medicine 22:117 (April 1951).

Rait, W. L., *The Etiology of Postdecompression Sickness in Aircrewmen,* US Armed Forces Medical Journal 10:790 (1959).

Ward, J. E., *The True Nature of the Boiling of Body Fluids in Space,* Journal of Aviation Medicine 27:429 (1956).