CHAPTER 2

PHYSIOLOGICAL EFFECTS OF ALTITUDE

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Introduction

1. Flight at high altitude exposes flying personnel to environmental conditions in which the unprotected human body may not be able to function. It is important, therefore, that the physical limitations of the body and method of extending these limitations are thoroughly understood by all aircrew and particularly by captains of aircraft who may be responsible for the safety and well-being of untrained passengers.

2. In order to understand the effects of altitude on man, it is essential to know something about the characteristics of the atmosphere and also have a basic understanding of man's respiratory requirements.

Physics of the Atmosphere

3. This subject is dealt with fully in AP 3456A, Part 1, Sect 1, Chap 1 and it is necessary here to emphasize only a few factors which are of particular significance in a study of the effects of altitude on the aviator.

a. *Composition of the Atmosphere.* The composition of the atmosphere for all practical purposes is constant from ground level to 300,000 feet. The composition, by volume, of dry air is:

(1) Oxygen – 21%

(2) Nitrogen, including the rare gases of which argon is the main one – 79%

(3) Carbon dioxide – a trace

Ozone which is formed by the action of ultraviolet radiation upon oxygen is also present at trace concentrations. The concentration of water vapour in the atmosphere varies with the degree of saturation (relative humidity) and the temperature. Typically water vapour forms 1–2% by volume of atmospheric air.

b. *Atmospheric Pressure and Altitude.* With ascent from the surface of the earth the atmosphere becomes progressively less dense. Thus the pressure exerted by the atmosphere falls in an approximately exponential manner with vertical distance from the ground, the pressure at an altitude of 18,000 feet (380 mm Hg) being half that at sea-level (760 mm Hg). The relationship between the pressure exerted by the atmosphere and altitude (ICAO international standard atmosphere) is given in an abbreviated form in Table 1.

Altitude (feet)	Pressure	
	(mm Hg)	(psi)
0	760	14.70
8.000	565	10.92
18,000	380	7.34
25,000	282	5.45
34,000	188	3.63
40,000	141	2.72
50,000	87	1.68
60,000	54	1.04
100,000	8	0.16

Table 1. Relationship between Atmospheric Pressure and Altitude

Since the relationship between atmospheric pressure and altitude is exponential the change of pressure for a given change of altitude falls with ascent to altitude. The change of pressure per 1,000 feet change of altitude is illustrated in Table 2.

Altitude (feet)	Change of Pressure (mm Hg) per 1,000 feet Altitude
500	27.1
10,000	20.2
20,000	14.6
30,000	10.3
40,000	6.8

Table 2. Change of Pressure and Altitude

c. *Partial Pressure of Gases.* The physiological effects of a given gas is related to its molecular concentration. This quantity is expressed by the partial pressure of the gas. The partial pressure exerted by a gas in a mixture of gases is the pressure which would exist if it only occupied the whole mixture. Thus the partial pressure of a gas x, (P_x) which constitutes y% by volume of a gas mixture, which has a total pressure of P_T is given by:

$$P_x = \frac{y}{100} \times P_T$$

For example the partial pressure of oxygen (P_{O_2}) in dry air at a pressure of 760 mm Hg is:

$$P_{O_2} = \frac{21}{100} \times 760 = 160 \text{ mm Hg}$$

Similarly, the partial pressure of nitrogen (P_{N_2}) in dry air at a pressure of 760 mm Hg is

$$P_{N_2} = \frac{79}{100} \times 760 = 600 \text{ mm Hg}$$

The sum of the partial pressures of the constituents of a gas mixture equals the total pressure (P_T) exerted by the mixture. Thus for dry air

$$P_{O_2} + P_{N_2} = P_T$$

Since the total pressure exerted by the atmosphere falls exponentially with altitude (sub para 3.b refers) it follows that the partial pressure of oxygen in dry air falls with altitude in a similar manner, as illustrated in Table 3.

Altitude (feet)	Partial Pressure of Oxygen in Dry Air (mm Hg)
0	160.0
8,000	118.7
18,000	79.8
25,000	59.2
40,000	29.6

Table 3 Partial Pressure of Oxygen in the Atmosphere at Altitude

d. *Temperature and Altitude.* Solar radiation heats the surface of the earth and this warms the lowest layer of the atmosphere. Above the surface of the earth the temperature falls steadily with altitude throughout the troposphere at the adiabatic rate of approximately 2°C per 1,000 ft. This fall in temperature ceases at the tropopause which is approximately 35,000 ft; however, as the tropopause is higher over the equator, the temperature at that level is much lower than over the poles. In the stratosphere the temperature is fairly constant at about – 55°C.

e. *International Standard Atmosphere.* The international standard atmosphere is derived from average conditions and is used as a basis for comparison. At sea level it has a pressure of 760 mm Hg, a temperature of 15°C and a mean lapse rate of 1.98°C per 1,000 ft.

1 standard atmosphere = 760 mm Hg
= 29.9 in Hg
= 14.7 psi
= 1,013 mb

The relationship between pressure and altitude defined by the international standard atmosphere (ICAO, 1964) is illustrated in Table 1.

Anatomy and Physiology of Respiration

4. The energy essential for living processes is obtained by the oxidation by oxygen of complex food stuffs. Thus oxygen is one of the most important materials required for the maintenance of normal function by living cells. The cells of the brain are particularly sensitive to oxygen lack. The body is only able to store very small quantities of oxygen. Thus cessation of the oxygen supply to the brain results in unconsciousness in 6–8 seconds and irreversible damage ensues if the oxygen supply is cut off completely for longer than about 4 minutes. The maintenance of normal function requires that oxygen be delivered to the cells of all tissues of the body and that the supply is matched to the rate of consumption of oxygen so that the partial pressure of oxygen (P_{O_2}) is maintained above a certain critical value. Oxidation of complex food stuffs produces, amongst other substances, carbon dioxide. The carbon dioxide so formed must be removed from the tissues and voided to the atmosphere since accumulation of this gas in the tissues interferes with normal function. The processes whereby the oxygen in the atmosphere is transported to the tissues and the carbon dioxide in the tissues is transported to the atmosphere is termed respiration. Several steps are involved in these transport systems:

a. Exchange between the atmosphere and the gas within the lungs – by ventilation of the lungs (breathing).

b. Carriage of oxygen and carbon dioxide between the lung gas and the tissues by the circulating blood.

c. Exchange between the circulating blood and the tissues where oxygen is consumed and carbon dioxide is produced.

5. Gas exchange between the external atmosphere and the blood which transports

oxygen and carbon dioxide around the body takes place within the lungs. The structure of the latter is well suited to promoting the rapid transfer of oxygen and carbon dioxide between the lung gas and the blood. Within the lung the air passages divide repeatedly ending eventually in very small air sacs (alveoli) of which the adult lung contains some 300 million giving an effective area for gas exchange of 50–100 square metres. The walls of the alveoli are very thin and the blood flowing through the lungs is thus brought into very close proximity to the gas in the air sacs (alveolar gas). In a young adult the volume of the alveolar gas at the end of a normal expiration is approximately 3.0 litre, whereas the maximum amount of gas which can be held in the lungs is of the order of 6.5 litre. The passage of a gas across the walls of the alveoli is controlled essentially by the differences of the partial pressures of the gas in the blood and alveolar gas. Thus oxygen is taken up by the blood flowing through the lungs as long as the partial pressure of oxygen (P_{O_2}) in the alveolar gas is greater than the P_{O_2}) in the blood flowing into the lungs. As oxygen enters the blood, increasing the concentration of oxygen in it, the P_{O_2} of the blood also rises. The area of the alveolar wall is so great and the wall separating the alveolar gas and the blood is so thin that the P_{O_2} of the blood leaving the lungs virtually always equals the P_{O_2} in the alveolar gas. Similarly the exchange of carbon dioxide is driven by the difference between the partial pressure of carbon dioxide (P_{CO_2}) in the blood flowing into the lungs and the lower P_{CO_2} in the alveolar gas. Also the P_{CO_2} of the blood leaving the lungs equals P_{CO_2} in the alveolar gas. Thus the P_{O_2} and P_{CO_2} in the alveolar gas reflect closely the partial pressures of these gases in the blood flowing from the lungs to the tissues of the body. The oxygen removed from the alveolar gas by the blood is replenished by the ventilation of the lungs with air. This process, external respiration, also removes the carbon dioxide added to the alveolar gas by the blood flowing through the lungs.

6. Air enters the nose and mouth during inspiration and is carried down through the larynx (voice box) and the trachea (windpipe) to the lungs. During its passage, the air is warmed to body temperature (37°C), humidified so that it becomes saturated with water vapour at body temperature (partial pressure of water at 37°C is 47 mm Hg) and filtered. Within the lungs the inspired air mixes with the alveolar gas thereby adding oxygen to it. The portion of the alveolar gas expelled from the lungs during expiration carries carbon dioxide to the atmosphere. The ventilation of the lungs with air is normally regulated so that the P_{CO_2} of the alveolar gas is held constant over a wide range of rates of production of carbon dioxide by the tissues of the body. Thus at rest the average volume of each breath is approximately 0.5 litre and the average rate of breathing is approximately 16 breaths per minute so that the lung ventilation is $0.5 \times 16 = 8.0$ litre per minute. When the rate of production of carbon dioxide is increased as in physical exercise both the depth and rate of breathing are increased. The volume of a single breath typically increases during heavy exercise to 2.5 litre and the rate of breathing to 40–50 breaths per minute giving a total ventilation of 100–125 litre per minute. Trained athletes can achieve lung ventilations of the order of 150–200 litre per minute.

7. The composition of the alveolar gas depends upon the composition of the inspired gas and the balance between ventilation of the lungs on the one hand and the rates of consumption of oxygen and production of carbon dioxide on the other. It has already been seen (para 6) that the ventilation of the lungs is normally regulated in relation to the latter so that the P_{CO_2} of the alveolar gas is held constant. The 'normal' average value of the alveolar P_{CO_2} is 40 mm Hg (range 38–42 mm Hg). The composition of the alveolar gas when breathing air at sea level is given in Table 4. The table also shows the concentration of each gas by volume of the dry gas.

Gas	Partial Pressure (mm Hg)	Concentration of Dry Gas by Volume %
Oxygen	100	14.0
Carbon Dioxide	40	5.6
Nitrogen	573	80.4
Water Vapour	47	–
Total	760	100.0

Table 4. Composition of Alveolar Gas, Breathing Air at Ground Level

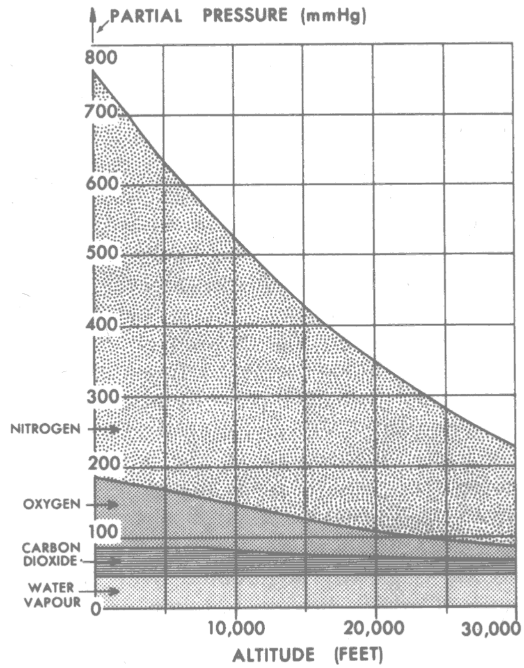

Fig 1. Composition of Alveolar Gas - Breathing Air at Altitude

8. With ascent to altitude, breathing air, the fall of the P_{O_2} in the atmosphere (para 3.c) produces a fall in the P_{O_2} in the alveolar gas. Reduction of the alveolar oxygen tension to below 55–60 mm Hg produces a reflex increase in the ventilation of the lungs, so that the ventilation increases relative to the rate of production of carbon dioxide by the body and the alveolar P_{CO_2} is reduced below normal. The lower the alveolar P_{O_2} is below 55–60 mm Hg, the greater is the increase in ventilation and the larger is the reduction of alveolar P_{CO_2}. The partial pressure exerted by the water vapour in the alveolar gas is unaffected by ascent to altitude as it depends solely on the temperature of the gas in the lungs which remains constant at 37°C. Typical values of the partial pressures of the constituents of the alveolar gas when breathing air at various altitudes are illustrated in Table 5 and Figure 1.

Altitude (feet)	Partial pressure in Alveolar Gas of:			
	Water Vapour (mm Hg)	Oxygen (mm Hg)	Carbon Dioxide (mm Hg)	Nitrogen (mm Hg)
0	47	100	40	573
8,000	47	65	40	423
18,000	47	40	28	265
25,000	47	30	22	183
35,000 (1)	47	18	12	103
(1) Immediately after rapid decompression to 35,000 feet.				

Table 5. Typical Partial Pressures of Alveolar Gases when Breathing Air at Various Altitudes

Carriage of Oxygen in the Body

9. Oxygen is transported from the lungs to the tissues and the carbon dioxide produced by the tissues is transported to the lungs by the circulating blood. Although both oxygen and carbon dioxide are soluble in water the amount that is carried in this manner in the blood is much too small to meet the demands of the tissues. The blood red cells contain a red pigment, haemoglobin, with which oxygen forms a loose compound, oxyhaemoglobin. The amount of oxygen held in the blood as oxyhaemoglobin is a function of the partial pressure of oxygen in the blood (P_{O_2}). Oxygen is taken up where the P_{O_2} is higher, as in the lungs, and released where the P_{O_2} is lower, as in the tissues. A special mechanism also exists in the blood whereby its capacity for carbon dioxide is greatly augmented over that of water. Carbon dioxide is taken up where the P_{CO_2} is higher, as in the tissues, and is released where the P_{CO_2} is lower, as in the lungs. As has been described earlier (para 5), the P_{O_2} and P_{CO_2} of the blood leaving the lungs equal the partial pressures of these gases in the alveolar gas. The blood pumped to the tissues by the heart through the systemic arteries also has the same P_{O_2} and P_{CO_2} as the alveolar gas. As the blood flows through the extensive network of thin walled, small vessels (capillaries) which permeate all the tissues of the body, oxygen is released and carbon dioxide is taken up. The blood flow to an organ is normally regulated so that it matches the demands for oxygen delivery and carbon dioxide removal of its tissues. When these increase, as in muscle tissue during physical exercise, the muscle blood flow and indeed the amount of blood pumped by the heart are greatly increased. Thus heavy physical exercise such as running increases the output of the heart by about five fold over the resting value. The matching of blood flow to tissue demands for oxygen is normally such that between 25% and 75% of the oxygen contained in the arterial blood is given up by the blood as it flows through the tissues. The blood flowing from the tissues to the lungs has therefore a lower P_{O_2}, and a higher P_{CO_2} than the arterial blood and the alveolar gas. These differences of partial pressure result in oxygen being taken up and carbon dioxide unloaded as the blood flows through the lungs and comes into intimate contact with the alveolar gas.

10. The fall of the P_{O_2} in the alveolar gas which occurs with ascent to altitude whilst breathing air (para 8) reduces the P_{O_2} of, and the amount of, oxygen contained in the blood leaving the lungs and arriving at the tissue capillaries. This reduction, if moderate, will not decrease the rate at which oxygen is delivered to the tissues, but will reduce the partial pressure of oxygen in the tissue. Several mechanisms, including an increase in blood flow, come into operation to minimise the fall of P_{O_2} in the tissues. A more severe reduction of the P_{O_2} and oxygen content of the blood flowing to the tissues results in P_{O_2} of parts of the tissues falling to 0 in spite of the compensatory mechanisms coming into play. The critical level of alveolar P_{O_2} at which this situation arises in the brain, causing unconsciousness, is of the order of 30–35 mm Hg.

Hypoxia

11. It has been seen that oxygen is one of the most iimportant materials required for the maintenance of normal function by living material. The absence of a supply of oxygen adequate in quantity or partial pressure, a condition termed hypoxia, almost always results in a rapid deterioration of most functions and may cause death. Man is extremely sensitive and vulnerable to the effects of deprivation of oxygen. Thus the 25% reduction of the partial pressure of oxygen (P_{O_2}) in the atmosphere associated with ascent to an altitude of 8,000 feet produces a detectable impairment of mental performance; whilst sudden decompression to 50,000 feet, which reduces the alveolar P_{O_2} to 10 mm Hg, causes unconsciousness in 10 seconds and death in 4–6 minutes.

12. It is generally recognised that the most serious single hazard to man during flight is the reduction of the P_{O_2} produced by ascent to altitude. Failure of oxygen-equipment and/or of cabin-pressurisation so that the individual has to breathe air at high altitude quickly leads to incapacitation and perhaps death. The risks are greater in aviation in that a degree of hypoxia which, from the physiological view-point might not be fatal in itself, may have fatal results because of deterioration of performance in an individual leading to loss of control of an aircraft. In the past, oxygen-lack has taken a regular toll of both lives and aircraft. In World War II many aircrew were killed by hypoxia in flight: the ability of many more aircrew to perform their tasks was impaired by the condition. Although improvements in the performance and reliability of cabin-pressurisation and oxygen-delivery systems have greatly reduced incidents and accidents due to hypoxia, constant vigilance remains essential.

13. The causes of hypoxia in flight are:

a. Ascent to altitude without supplemental oxygen.

b. Failure of personal breathing equipment to supply oxygen at an adequate concentration and/or pressure.

c. Decompression of pressure cabin at high altitude.

The time course of the changes produced by breathing air at altitude is a function of the manner in which the condition is induced. Typically the changes are produced slowly by ascent at the usual rate for an aircraft of 2,000 – 3,000 feet per minute; more rapidly by the reversion to breathing air after failure of oxygen-delivery equipment; and fastest by a rapid decompression. Although breathing air during a steady ascent at 2,000–3,000 feet per minute is an uncommon cause of hypoxia in these days, it is convenient to describe the changes induced by hypoxia in this manner since the relatively slow rate of climb allows a semi-steady state to be maintained during the ascent. The manner in which these changes are modified by other causes of hypoxia will then be described.

Symptoms and Signs of Hypoxia

14. The symptoms and signs of hypoxia are extremely variable. The speed and order of appearance of signs and of the severity of symptoms produced by breathing air at altitude depend upon the altitude, the duration of the exposure and rate of either ascent or failure of the oxygen supply at altitude. The other major factor affecting the intensity of hypoxia at altitude is the degree of physical exercise; exercise markedly intensifies the effects of a given degree of hypoxia. Fatigue, exposure to cold, ingestion of alcohol or certain stimulant drugs such as benzedrine also increase the severity of the disturbances induced by a given intensity of hypoxia. Finally there is considerable individual variability in the symptoms and effects of hypoxia. Generally the higher the altitude, the more marked the symptoms. Rapid rates of ascent, however, allow higher altitudes to be reached before severe symptoms occur. In these circumstances, unconsciousness may supervene before any or many of the symptoms of hypoxia appear.

15. The effects of slow ascent (less than 4,000 feet per min) to altitude whilst breathing air are as follows:

a. *Altitudes up to 10,000 feet.* The seated individual (unless he is carrying out heavy exercise) has no symptoms. His ability to perform most complex tasks is unimpaired. The speed with which he can react to novel conditions is however significantly impaired at altitudes above 6,000–8,000 feet. It is possible to show in the laboratory that the ability to detect targets at low levels of illumination is impaired at altitudes above 5,000–6,000 feet. This degree of impairment however has no practical implications – night vision is only impaired significantly when the altitude exceeds 12,000–14,000 feet.

b. *Altitudes between 10,000 and 15,000 feet.* The resting individual has little or nothing in the way of symptoms but his ability to perform skilled tasks such as aircraft control and navigation is impaired, the impairment increasing with altitude above 10,000 feet. The individual is frequently unaware of the hypoxia or of the impairment of performance which it produces. Indeed he may well believe that he is performing better than usual! Physical exercise, particularly at altitudes above 12,000 feet, frequently produces mild symptoms, especially breathlessness. Exposure to these altitudes for longer than 10–20 minutes often induces a severe headache.

c. *Altitudes between 15,000 and 20,000 feet.* Above about 15,000 feet, symptoms of hypoxia occur even in individuals at rest. There is marked impairment of performance, even of simple tasks, together with a loss of critical judgement and will power. Thinking is slowed, there is muscular inco-ordination with trembling and clumsiness and marked changes in the emotional state. Thus the individual may become hilarious, pugnacious or morose. He may become physically violent. Again he usually has no insight into his condition; an effect which makes hypoxia such a potentially dangerous hazard in aviation. The individual frequently feels light-headed and tingling in the lips and limbs. Darkening of vision is a common symptom although generally the subject is unaware of the change until oxygen is breathed, when there is a marked apparent brightening of level of illumination. Hearing is not usually markedly impaired until the hypoxia becomes severe. Physical exertion greatly increases the severity of all the effects. It often causes unconsciousness.

d. *Altitudes above 20,000 feet.* Breathing air at altitudes above 20,000 feet results in severe symptoms even in individuals at rest. Mental performance and comprehension decline rapidly and unconsciousness supervenes with little warning. Jerking of the upper limbs occurs quite often before consciousness is lost and convulsions may occur after unconsciousness has supervened. Exertion at altitudes above about 20,000 feet rapidly leads to unconsciousness.

16. In moderate and severe hypoxia the depth and rate of breathing are increased and this effect can usually be seen on exposure to breathing air at altitudes above 15,000–18,000 feet. Above 18,000 feet the high concentration of haemoglobin which has given up its oxygen in the capillaries of the skin gives rise to blueness of the lips, tongue and face as well as the skin of the limbs (seen best in the finger nails).

17. Interruption of the supply of supplemental oxygen at altitudes above 10,000 feet with reversion to breathing air is a more frequent cause of hypoxia in flight than ascent without added oxygen. As the altitude is increased the times between the reversion to breathing air and the consequent impairment of performance, followed at the higher altitudes by loss of consciousness, rapidly decrease. The time which elapses between sudden reversion to breathing air and loss of useful consciousness, ie the point at which an individual is no longer able to carry out his task, is very variable, especially at altitudes below 28,000–30,000 feet. The ranges of times of useful consciousness found at various altitudes are presented in Table 6 and Figure 2.

Altitude (feet)	Time of Useful Consciousness (range – seconds)
25,000	150 – 360
27,000	130 – 250
30,000	100 – 180
34,000	60 – 100
36,000	55 – 85

Table 6. Times of Useful Consciousness Following Sudden Reversion to Breathing Air

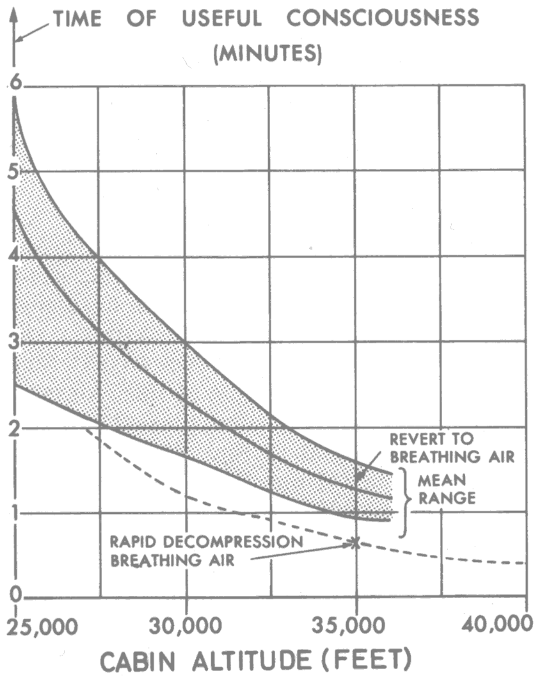

Fig 2. Time of Useful Consciousness on:
a. Sudden Reversion from Breathing Oxygen to Breathing Air at Given Altitudes.
b. Rapid Decompression (when Breathing Air) from 8,000 ft to Given Final Altitude.

18. The hypoxia induced by either slow ascent whilst breathing air or cessation of supplemental oxygen at a given altitude are described in the previous paragraphs. The severity and rate of onset of hypoxia when it is induced by a sudden failure of the pressure cabin of an aircraft (ie time for decompression to above an altitude of 20,000 feet less than 1½ minutes) are considerably greater than when the hypoxia is induced by cessation of supplemental oxygen at the same altitude. Thus serious impairment of performance will occur within 1½ minutes on rapid decompression whilst breathing air to 25,000 feet. It may be seen (Figure 2) that the higher the final altitude the shorter the time between the decompression and the consequent impairment of performance. Oxygen breathing must be commenced within a few seconds of the beginning of a rapid decompression to altitude between 15,000 to 30,000 feet if no impairment of performance due to hypoxia is to occur. Rapid decompression to altitudes above 30,000 feet will result in transient impairment of performance even if 100% oxygen is breathed as the decompression commences. These facts emphasise the importance of the correct use of oxygen equipment in the event of the decompression of an aircraft which is pressurised to provide a cabin altitude below 8,000 feet when the occupants will probably be breathing air whilst the aircraft is at high altitude. This lesson is even more important in aircraft with small, highly pressurised cabins when loss of a windscreen or entrance door will result in a rapid decompression of the cabin and hence the very rapid development of hypoxia.

Prevention of Hypoxia at Altitude

19. It has been seen (para 15) that the hypoxia associated with breathing air at altitudes greater than 8,000 feet (an alveolar partial pressure of oxygen of 65 mm Hg) produces a significant impairment of skills involved in flying. The maximum cabin altitude at which aircrew may operate without supplemental oxygen is therefore 8,000 feet. In a low differential pressure cabin aircraft (in which the cabin altitude reaches 16,000–25,000 feet at the ceiling of the aircraft) it is normal practice to use supplemental oxygen from ground level, as with high rates of ascent it is possible to exceed a cabin altitude of 8,000 feet rapidly. The reduction of the partial pressure of oxygen (P_{O_2}) in the air which occurs with ascent to altitude and which gives rise to hypoxia can be prevented by increasing the concentration of oxygen in the inspired gas. In all

RAF oxygen delivery systems designed for use by aircrew the concentration of oxygen is increased with ascent to altitude so that the P_{O_2} of the alveolar gas does not fall below that associated with breathing air at ground level ie an alveolar P_{O_2} of 100 mm Hg (Table 4). The oxygen concentration required at an altitude of 34,000 feet in order to maintain an alveolar P_{O_2} of 100 mm Hg is 100% (Table 7).

Partial Pressure (mm Hg) of:	Altitude (feet)		
	34,000	40,000	45,000
Oxygen	100	60	36
Carbon Dioxide	40	34	28
Water Vapour	47	47	47
Total Pressure (mm Hg)	187	141	111

Table 7. Partial Pressures of Alveolar Gases, Breathing 100% Oxygen at Altitudes of 34,000, 40,000 and 45,000 feet

20. Ascent to altitudes above 34,000 feet, even whilst breathing 100% oxygen, results in the alveolar P_{O_2} falling below that produced by breathing air at ground level, ie P_{O_2} of 100 mm Hg. Breathing 100% oxygen at an altitude of 40,000 feet produces an alveolar P_{O_2} of about 60 mm Hg (Table 3) ie an intensity of hypoxia equivalent to that produced by breathing air at an altitude of 8,000–10,000 feet. Ascent to altitudes higher than 40,000 feet breathing 100% oxygen gives rise to significant hypoxia. As indicated by the corresponding alveolar P_{O_2}s, the intensity of the hypoxia produced by breathing 100% oxygen at 45,000 feet (Table 7) is slightly more severe than the hypoxia produced by breathing air at 18,000 feet (Table 5). The maximum altitude at which it is acceptable to fly an unpressurised aircraft, considering hypoxia alone, when oxygen is breathed at ambient pressure, is 40,000 feet. In the event of decompression of a pressurised aircraft when rapid descent is initiated immediately the pressure cabin falls, breathing 100% oxygen at ambient pressure will provide adequate protection against severe hypoxia at cabin altitudes up to 43,000 feet. Severe hypoxia can only be avoided on exposure to altitudes above 40,000 feet by increasing the total pressure of the gases in the lungs above the pressure of the environment, a technique termed (positive) pressure breathing.

Pressure Breathing

21. It has been seen (para 20) that exposure to altitudes above 40,000 feet, even breathing 100% oxygen, gives rise to significant hypoxia. Prevention of hypoxia on exposure to altitudes above 40,000 feet involves the administration of 100% oxygen and maintaining the total pressure of the alveolar gas equal to that which exists at 40,000 feet ie 141 mm Hg. This is achieved by delivering 100% oxygen to the respiratory tract at a pressure greater than that of the environment. This technique is termed (positive) pressure breathing. When the altitude to which protection is required is greater than 60,000 feet or if protection above 40,000 feet is required for longer than a few minutes the pressure at which oxygen is delivered to the respiratory tract is chosen so that it maintains the total pressure within the oxygen mask, and hence in the alveoli, equal to 141 mm Hg. The positive pressures required at various altitudes to maintain this standard are presented in Table 8. Other standards which are discussed in later paragraphs are also shown in Table 8.

22. Pressure breathing, which creates pressure differentials between the respiratory tract and other parts of the body, produces a number of disturbances, some of which limit the magnitude of the pressure which can be applied. These disturbances also determine the counter-measures which must be taken in order to allow the use of higher pressures.

a. *Effect of Pressure Breathing on Head and Neck.*

(1) The most striking feature of breathing at high pressure using an oxygen

Altitude (feet)	Atmospheric Pressure (mm Hg)	Positive Pressure Required		
		To Maintain 141 mm Hg abs (mm Hg)	Mask Alone (mm Hg)	Mask, Pressure Jerkin, G Trousers (mm Hg)
40,000	141	0	0	0
45,000	111	30	17	28
50,000	87	54	30	50
56,000	66	75	–	70
70,000	33	108	–	–
100,000	8	133	–	–

Table 8. Schedules of Pressure Breathing above 40,000 feet

mask is the distension of the mouth and throat that occurs when the pressure exceeds about 10–15 mm Hg. At higher pressures the floor of the mouth and the whole of the throat are widely distended, and above about 60–70 mm Hg, this distension can give rise to severe discomfort.

(2) In certain individuals, oxygen under pressure may force its way up the tear ducts which connect the inner corners of the eyes to the nose and blow onto the surface of the eyes causing spasm of the eyelids.

(3) In order to sustain breathing pressures in excess of 70 mm Hg, a pressurised helmet which applies the same pressure to the eyes and neck as is being transmitted to the lungs is used. This support to the neck and throat avoids the effects described above and also permits speech at high breathing pressures.

b. *Effect of Pressure Breathing on Respiration.*

(1) Pressure breathing inflates the lungs, causing the lungs and chest to expand. In the relaxed subject, a breathing presssure of only 20 mm Hg distends the lungs completely. During the normal breathing cycle, inspiration is achieved by active muscular contraction, whereas breathing out simply requires the relaxation of the muscles. In pressure breathing this process is reversed. Breathing-in consists of a controlled relaxation of the muscles as the gas under pressure inflates the lungs. Breathing out consists of controlled contraction of the same muscles. Thus the pattern of muscular contraction required during pressure breathing differs markedly from that of normal breathing. The unusual pattern is associated with a tendency to overbreathe. Pressure breathing is a technique which has to be learnt.

(2) The maximum pressure which can be breathed without counterpressure to the chest and abdomen is 30 mm Hg. Breathing pressures much above 50–60 mm Hg without trunk counterpressure cause marked inflation of the lungs which may result in serious damage to the latter.

(3) The respiratory disturbances produced by pressure breathing can be minimised by applying counterpressure to the external surface of the trunk. At high levels of pressure breathing it is not sufficient to apply counterpressure to the chest alone, as the diaphragm and the abdominal muscles play an important part in breathing.

The standard Royal Air Force garment, the pressure jerkin, applies counterpressure to the chest and abdomen by means of a bladder which is inflated to the same pressure as that which is delivered to the mask or pressure helmet. The counterpressure applied by the pressure jerkin allows pressures as high a 140 mm Hg to be breathed with comparative ease.

c. *Effects of Pressure Breathing on the Circulation.* The rise of pressure within the chest produced by pressure breathing has very significant effects upon the heart and circulation. The rise of pressure in the lungs is transmitted to the blood in the heart and great vessels within the chest and abdomen. The increase of pressure in these areas results in blood being displaced from within the trunk into the limbs and to the loss of the fluid part of blood out of the vessels into the tissues of the limbs. The amount of blood displaced out of the chest and abdomen increases as the breathing pressure increases. The amount of fluid lost into the tissues of the limb is greater the higher the breathing pressure and the longer the time for which it is operative. Both the displacement of blood into the periphery and the loss of fluid into the tissues reduce the amount of blood available for the maintenance of the circulation. When this reduction exceeds a critical value the blood pressure falls and a faint occurs. There are limits therefore to the magnitude and duration of pressure breathing which can be tolerated with safety. This tolerance can be increased by applying counterpressure to the limbs so reducing the displacement of blood and the loss of circulating fluid into the tissues.

23. In practice, pressure breathing with or without counterpressure to parts of the body (by means of a partial pressure suit) is used to provide short duration protection against hypoxia during emergency exposures to altitudes above 40,000 feet produced by either failure of cabin pressurisation or ejection at high altitude. The other effects produced by decompression to high altitude, eg decompression sickness (para 29), in addition to the disturbances produced by pressure breathing limit the duration of the exposure. Descent is initiated immediately the decompression occurs and provided that there is no serious structural damage to the aircraft it is carried out at the maximum possible rate. Compromises related to the maximum absolute pressure in the lungs and the maximum breathing pressure have been accepted and proved experimentally, thereby providing a number of high altitude protective assemblies.

a. *Pressure Breathing Mask Alone.* It has been seen that the maximum pressure which can be breathed using a mask alone is 30 mm Hg. The compromise set in this assembly is to provide this breathing pressure at an altitude of 50,000 feet (Table 8). As indicated by the total pressure in the mask and alveolar gas employed in this assembly at 50,000 feet, ie 30 + 87 = 117 mm Hg, it allows considerable hypoxia at the maximum altitude at which it is used. A pressure sealing mask used with an oxygen regulator which provides a pressure of 30 mm Hg at 50,000 feet will provide protection to an altitude of 50,000 feet provided that descent is initiated within one minute of the start of this decompression at a rate exceeding 10,000 feet min^{-1}.

b. *Pressure Breathing Mask with Pressure Jerkin.* With counterpressure applied to the chest and abdomen by means of the pressure jerkin, pressures of up to 70 mm Hg can be breathed using a mask. The displacement of blood and fluid into the limbs, however, limits the altitude to which the combination of a mask and pressure jerkin can be used safely to 52,000 feet.

c. *Pressure Breathing Mask, with Pressure Jerkin and G-Trousers.* The displacement of blood and fluid into the lower limbs produced by pressure breathing may be greatly reduced by inflating the standard G-trousers to the same pressure as is supplied to the mask and pressure jerkin. With this degree of counterpressure it is acceptable to breathe at a pressure of 70 mm Hg at an altitude of 56,000 feet. The relationship between altitude and breathing pressure employed in this assembly is given in the fifth column of Table 8. It provides protection to an altitude of 56,000 feet proviided that descent is initiated within ½ minute of the start of this decompression at a rate exceeding 10,000 feet min^{-1}.

d. *Pressure Helmet and Pressure Jerking and G–Trousers.* Much higher breathing pressures can be tolerated when wearing a pressure helmet. When counterpressure is applied to the trunk by the pressure jerkin and to the lower limbs by the G-trousers a pressure of the order of 110 mm Hg can be breathed for several minutes without serious disturbances. This assembly, when used with an oxygen regulator which maintains a pressure of 141 mm Hg absolute in the mask and counter pressure garments (Table 8, column 3), will provide protection to an altitude of 70,000 feet provided that descent is initiated within one minute of the start of the decompression at a rate exceeding 10,000 feet min^{-1}.

Hyperventilation

24. The ventilation of the lungs is controlled by the respiratory centre in the brain, which in turn is controlled by the partial pressure of carbon dioxide (P_{CO_2}) in the blood. A rise of P_{CO_2} in the blood stimulates the respiratory centre and increases ventilation of the lungs. A decrease in blood P_{CO_2} has the opposite effect. The respiratory centre is extremely sensitive to small changes in P_{CO_2} and continuously adjusts the ventilation of the lungs to maintain the partial pressure of this gas at the normal level. During exercise the rate and depth of respiration increase to keep pace with the increased rate of production of carbon dioxide by the tissues. Thus, over a wide range of physical activity, the P_{CO_2} of the alveolar gas remains constant at the resting value of about 40 mm Hg (Table 4) in spite of the rate of production of carbon dioxide varying 8–10 fold.

25. The ventilation of the lungs may be increased out of proportion to the rate of production of carbon dioxide, and then the P_{CO_2} in the alveolar gas and in the blood and tissues will be reduced below their normal values. This condition is termed hyperventilation. Hyperventilation may be produced voluntarily. It can also be produced by anxiety, apprehension or fear. The condition occurs not uncommonly in student aircrew during flying training. Aircraft passengers who are afraid or anxious frequently hyperventilate. Hyperventilation is one of the normal responses to hypoxia (para 8). It is also produced by a rise of body temperature. Whole body vibration at frequencies of the order of 4–8 Hz induces over-breathing. Another procedure which produces hyperventilation is pressure breathing (para 24a). Whatever the cause of the hyperventilation, the individual who is over-breathing is unlikely to be aware that he is doing so until such time as it produces ill-effects.

26. The excessive removal of carbon dioxide from the blood and tissues which results from hyperventilation gives rise to the following symptoms:

a. Tingling in the hands, the feet and the lips.

b. Spasm of the muscles of the hands and feet.

c. Vague feeling of unreality.

d. Lightheadedness and dizziness.

e. Faintness.

f. If prolonged, unconsciousness.

The lowering of the P_{CO_2} in the tissues of the brain also causes an impairment of performance.

27. Hyperventilation is a condition to be avoided. In order to reduce the likelihood of

hyperventilation occurring in flight the following points should be observed:

a. Learn to breathe in a normal manner particularly when carrying out tasks which are known to predispose to hyperventilation.

b. Beware of the tendency to over-breathe during periods of intense concentration or tension.

c. Do not attempt to overcome suspected hypoxia by voluntary over-breathing.

28. It is possible for individuals to confuse the symptoms of hypoxia and hyperventilation. When symptoms are experienced at cabin altitudes at which hypoxia could occur it should always be assumed that the cause is hypoxia. A thorough check and recheck of oxygen equipment should be made immediately whilst every effort is made to breathe in a normal and controlled manner.

Decompression Sickness

29. Decompression sickness is the name given to a group of symptoms which may occur as a result of exposure to reduced atmospheric pressure, excluding those due to hypoxia or the expansion of pre-existing gas contained in the hollow cavities of the body. It can, therefore, occur either in an aircraft at altitude or in a decompression chamber. It is sometimes referred to as the "bends", a term which is used to describe the commonest symptoms of decompression sickness, namely, pain in the muscles or joints.

30. Decompression sickness can occur in normal individuals who have no predisposing disease and there is a very wide individual variation in susceptibility. It does not occur below 18,000 feet and is rare below 25,000 feet. The incidence of the condition increases rapidly with increasing height above that altitude. The duration of exposure to low pressure is also a very significant factor in the development of the condition. The effects of the altitude and the duration of the exposure upon the incidence of symptoms of decompression sickness are illustrated in Figure 3.

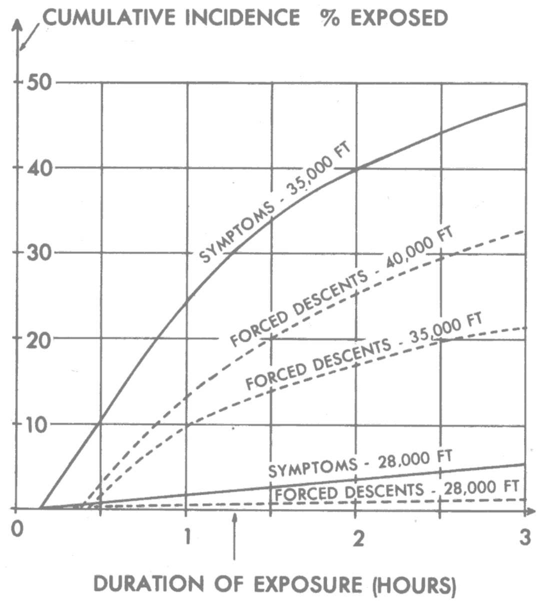

Fig 3. Cumulative Incidence of:
a. Symptoms of Decompression Sickness.
b. Forced Descents caused by Decompression Sickness.
(Fit young men - seated and at rest)

The symptoms of decompression sickness are:

a. *Bends*. The commonest severe symptom of decompression sickness is pain in a joint or limb, the so-called "bends". The pain may be mild or severe. A mild pain will often develop into severe or agonizing pain if altitude is maintained. If the pain is accompanied by pallor, sweating and nausea or vomiting, the subject is very likely to collapse. Less frequently, the pain may disappear without becoming severe. The pain is most likely to occur in the upper part of the arm near the shoulder, the knee, wrist and ankle; more than one of these areas may be affected at the same time. It usually starts as a mild ache, rather like the after-effect of unaccustomed exercise, and if allowed to progress, may become a deep pain spreading up and down the limb causing clumsiness and weakness and eventually complete disablement of the limb. The early mild pain often encourages the subject to move or rub the affected part, which only makes matters worse. On descent symptoms pass off around 18,000 ft to 22,000 ft, although residual stiffness and a mild ache may persist for some time. Immediate re-ascent causes immediate recurrence.

b. *Effects on the Skin.* Itching and tingling of the skin frequently occur and are usually transient effects and of little significance. Localized skin rashes are sometimes observed.

c. *Chokes.* This is the name given to a respiratory disturbance which may occur, but is a misnomer as the subject does not choke. It takes the form of a sore, burning feeling in the centre of the chest, with pains on breathing in and paroxysms of coughing. The symptoms of chokes could be described as similar to those caused by the inhalation of an irritant gas. This is not a very common condition but it should be taken very seriously and an immediate descent to below 18,000 ft should be started, otherwise collapse may follow. Chokes may or may not be preceded by the bends. Although the condition is relieved by descent there may be a residual soreness in the chest.

d. *Neurological Symptoms.* The effects on the nervous system are very varied and usually short lasting. Commonly the eyes are affected in the form of a temporary defect in the field of vision. Infrequently there may be weakness or even paralysis of one or both limbs of one side of the body. There is often a feeling of uneasiness or an inability to concentrate. After recompression a severe headache may develop.

c. *Collapse.* This can occur with or without other symptoms being present. The collapse is a typical faint and is characterized by pallor, sweating, nausea, giddiness and then unconsciousness. Post-decompression collapse may occur after return to ground level and up to five hours, or even longer, after landing. This type of collapse is usually preceded by some form of decompression sickness at altitude, but not always. Decompression collapse is not common but, should it occur, it must be treated as a medical emergency.

31. Decompression sickness is caused by the liberation of nitrogen bubbles in the body due to exposure to a lowered atmospheric pressure. The body is normally saturated with nitrogen so that there is sufficient nitrogen in solution in each tissue and fluid of the body to produce a partial pressure of gas equal to the P_{N_2} in the alveolar gas. When the pressure of the environment is lowered by ascent to altitude the nitrogen in solution in the tissues, saturated at sea level pressure, will now be in a state of supersaturation and under certain conditions will come out of solution. Bubble formation is influenced by many factors, such as movement of the tissues (hence the need to restrict movement of the affected part), alterations in the circulation of body fluids and rapid change in gas pressure. The bubbles tend to be released in tissues with the least blood supply and greatest amount of dissolved nitrogen. This combination of circumstances occurs principally in fatty tissues. The bubbles which are released cause pain by pressing on nerve endings. They also pass into the circulation and can cause disturbances in the lungs and the brain.

32. The factors influencing the incidence of decompression sickness are:

a. *General Factors.*

(1) *Altitude.* The condition very rarely occurs below 18,000 ft and the frequency increases with altitude, particularly above 25,000 ft.

(2) *Rate of Ascent.* The range of rates of ascent which occurs in aircraft does not affect the incidence.

(3) *Duration of Exposure.* The longer the duration of exposure, the greater the proportion of individuals affected.

(4) *Exercise.* Exercise, whilst at altitude, markedly increases the incidence and severity of symptoms.

(5) *Re-exposure.* Re-exposure to altitude within about 48 hours, increases an individual's susceptibility.

(6) *Hyperbaric exposure.* Exposure to breathing air at pressures above one atmosphere such as occurs in sea diving, by increasing the amount of nitrogen dissolved in the tissues, greatly increases susceptibility to the condition. Thus after a recent dive, breathing air, decompression sickness may occur on ascent to as low an altitude as 6,000 feet (*See* para 35).

b. *Personal Factors*

(1) *Age.* The incidence increases with age; each decade approximately doubles the susceptibility.

(2) *Body Weight.* As has already been mentioned, fat has a higher nitrogen content than other body tissues so that obesity predisposes to symptoms of decompression sickness.

(3) *Recent Injury.* There is some evidence to suggest that joint lesions and recent limb injuries increase susceptibility.

33. The treatment of decompression sickness is immediate recompression, as fast as is tolerable, to as low an altitude as possible. Except where operational considerations make maintenance of altitude essential descent should be made to an aircraft height at which the cabin altitude is less than 10,000 feet. In severe cases, or if symptoms persist, a landing should be made as soon as possible. If practical, the affected individual, if he is suffering from severe bends, chokes, neurological disturbances or collapse, should be laid flat and given 100% oxygen to breathe. Medical advice should be sought immediately by radio-telephony. Whenever decompression sickness occurs in flight the affected individual should receive medical attention as soon as possible after landing.

34. The incidence of decompression sickness can be markedly reduced by pre-oxygenation, ie by washing out the nitrogen in the body with oxygen. This is done by breathing 100% oxygen at ground level for some time before take-off. This procedure is time-consuming since it has to be carried out for at least an hour and possibly several hours, depending upon the height and duration of exposure which is expected. For example, breathing oxygen at ground level for three hours will protect a high percentage of subjects when exposed to 40,000 ft for three hours. Individuals who pre-oxygenate on the ground must proceed to their aircraft and transfer to 100% oxygen on the aircraft system without taking a breath of atmospheric air.

35. Decompression sickness is a condition which is best avoided. The most satisfactory method of prevention is limiting the maximum altitude to which aircrew are exposed to below 25,000 feet, by means of pressurisation of the cabin or, in unpressurised aircraft, limiting the maximum cabin altitude to 25,000 feet. Since a small but significant proportion of individuals may develop decompression sickness at altitudes below 25,000 feet the tendency over the last 20 years has been to reduce the maximum cabin altitudes of combat aircraft towards or below 20,000 feet. The marked increase in susceptibility to decompression sickness which follows exposure to breathing air at environmental pressures greater than 1 atmosphere requires that following such an exposure individuals must not ascend to altitude either in an aircraft or a decompression chamber until sufficient time has elapsed for the excess nitrogen to be eliminated from the body. The period spent at ground level before flight is to exceed 12 hours after swimming using compressed-air breathing apparatus and to be greater than 24 hours if a depth of 30 feet has been exceeded.

Vapourisation of Tissue Fluids

36. A further effect of exposure to a reduced pressure is the vapourisation of tissue fluids, resulting in a quite rapid, painless swelling of the affected part. Above 63,000 ft the total atmospheric pressure is less than the vapour pressure of the body fluids at deep body temperature. In regions of the body where the hydrostatic pressure of the body fluids is low, collections of water vapour could be formed. In practice, this condition is not likely to occur until the pressure is considerably lower than the equivalent of 63,000 ft. This condition has been observed in the hands of subjects wearing partial pressure suits at very high altitudes (above 65,000 ft). It disappears again on descent below that height. There is no residual disturbance of function due to this phenomenon and it can be prevented by applying pressure to the area concerned. In the case of the hands, for example, it can be avoided by wearing close-fitting leather gloves.

Effect of Change of Altitude on the Ears and Sinuses

37. The head contains a number of gas-filled cavities which communicate with the nose; these are the middle ear cavities and the nasal sinuses. The gas contained in these spaces expands and contracts on ascent and descent and so long as communication with the nose remains open to permit gas to flow out of and into these cavities no disturbances will occur. However, if free exchanges of gas in and out of these cavities does not occur with change of altitude, a very high pressure difference can soon arise, with painful and serious consequences. As already noted in para 3, the change of pressure for a 1,000 feet change of height is much greater at low than at high altitude, and thus the disturbances caused in the ears and sinuses by change of altitude occur predominantly at the lower altitudes.

38. The cavity of the middle ear is separated from the exterior by a thin diaphragm, the ear drum, and communicates with the nose via the Eustachian tube whose walls are soft and normally collapsed together. (Fig 4).

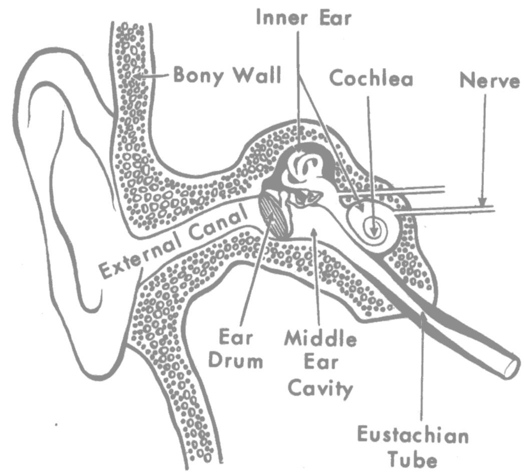

Fig 4. The Human Ear

39. During ascent, as the ambient pressure decreases, the expanding gas in the middle ear cavity readily escapes along the Eustachian tube, so that pressure is equalized on either side of the ear drum. Since the anatomical structure of the tube is such that this gas can escape easily (*see* Fig 4), disturbances are very rare during ascent. This passive ventilation of the middle ear may be heard as a popping sensation in the ear.

40. During descent, the collapsed wall of the Eustachian tube tends to act as a valve, preventing gas from flowing back into the middle ear cavity. The increase in pressure on the outside of the ear drum progressively distorts the drum inwards as the descent continues. Gas must flow into the middle ear cavity via the Eustachian tube during descent if the drum head is to be restored to its normal resting position. Several actions may be employed to open the Eustachian tube and allow gas to flow into the middle ear, such as yawning, swallowing or

pushing the jaw forward. If such actions fail, pinching the nose and blowing into it (as in blowing the nose) is very effective. This method must be used with some care lest the ears become over-inflated, resulting in discomfort, which can be confused with a failure to clear the ears. Another widely used method is to pinch the nose, close the glottis (the gap between the vocal cords) and raise the floor of the mouth. Each individual soon finds, by trial and error, the method which suits him best.

41. During a descent, the ears must be cleared constantly as difficulty is likely to occur when the pressure difference across the ear-drum is allowed to build up. This pressure build-up pushes in the ear drum causing pain and deafness which can become very severe as the pressure differential increases. The condition is known as otitic barotrauma, that is to say, damage to the ear by pressure. As the differential across the drum reaches about 50 mm Hg the pain is very severe and, when it reaches approximately 90 mm Hg, it is not possible to equate this pressure or "clear the ears" by voluntary effort. Further descent at this stage would cause rupture of the drum. In cases where voluntary actions such as those described fail to relieve the condition, it is best (if fuel permits) to climb again until the ears are clear and let down again at a reduced rate, being careful to keep the middle ears inflated.

42. A head cold is likely to cause congestion and swelling of the Eustachian tubes, just as the lining of the nose is affected. Thus it may become difficult or impossible to clear the ears. Aircrew with head colds should not fly unless they can clear their ears satisfactorily on the ground.

43. The nasal sinuses are cavities in the bones of the face and skull having a lining similar to that of the nose, with which they communicate along narrow tunnels. During ascent and descent gas flows freely out of and into the sinuses. In the presence of inflammation of the lining of these sinuses, as in sinusitis or with a severe head cold, swelling may obstruct the outlets. This will cause pain, which can be severe, during a descent. The condition is known as sinus barotrauma and may be felt in the cheek, forehead or deep in the head. In severe cases, the pain can be quite blinding and also accompanied by watering of the eyes. If sinus barotrauma occurs during flight, the rate of descent should be slowed and attempts made to force gas into the sinuses by raising the pressure in the nose by pinching the nostrils, closing the mouth, and breathing out hard. Any infection or inflamation in the sinuses is a further reason for seeking medical advice as to fitness to fly.

Abdominal Distension

44. In healthy individuals, the stomach and intestines contain a variable quantity of gas (0–300 millilitres). On ascent, this abdominal gas expands and normally will escape either upwards or downwards through the mouth or anus as the case may be. A few individuals have particular difficulty in venting this gas even at modest rates of ascent and this is most common amongst inexperienced aviators. The higher the rate of ascent, the greater is the problem of expelling the gas quickly as it is expanding. Healthy experienced aircrew may on occasions experience difficulty during particularly rapid and large increases in altitude. The symptoms caused by an inability to expel this gas during ascent vary from mild discomfort to severe pain in the abdomen and vomiting. The incidence of symptoms from the expansion of abdominal gas is, however, insignificant amongst experienced aircrew, except at cabin altitudes in excess of 30,000 ft. This problem can be aggravated by intestinal infection or the consumption of too many gas-forming foods.

Effects of Changes of Pressure on the Lungs

45. The lungs, being air-containing cavities, are also affected by rapid change of

environmental pressure. Only extremely high rates of decrease in the environmental pressure could, however, cause damage to the lungs by over-expanding them to the point of rupture, because of the relatively wide bore air passages along which the gas can escape from the lungs. In practice, very rapid decompressions over a wide range of pressure, which could possibly give rise to lung damage, will occur in the event of a serious structural failure of an aircraft. It is possibly, however, for lung damage to occur if the breath is held during a wide range decompression. It is clearly important, therefore, to ensure that intentional breath holding is avoided during practice decompression. Such an action, particularly with inflated lungs, would carry a grave risk of lung rupture.

46. Lung damage due to rapid or explosive decompression is extremely rare even when the decompression occurs over a wide pressure differential.

Effects of Low Temperature

47. The effects of low temperature on the body depend upon four factors:

a. The absolute temperature.

b. The speed of air movement.

c. The duration of exposure.

d. The amount of protection.

48. As already stated at the beginning of this chapter, the temperature falls steadily with altitude throughout the troposphere at the adiabatic lapse rate of approx 2° per 1,000 ft. In the stratosphere, the temperature is fairly constant at about – 55°C. The following table 9 gives some typical temperatures at various altitudes; based on the ICAO International Standard Atmosphere.

Altitude (feet)	Temperature (°C)
Sea Level	15
5,000	5
10,000	–5
15,000	–15
20,000	–25
25,000	–35
30,000	–45
35,000	–55
40,000	–55

Table 9. Atmospheric Temperatures at Various Altitudes

49. Exposure to a temperature of –40°C when wearing normal flying clothing leads to gross impairment of function after only a few minutes. Parts of the body which are bare, or only lightly clad, very soon become cold, numb, stiff and functionless; this is particularly noticeable in the fingers. There is an associated dulling of the senses and general incapacity. If exposure to this temperature is continued, the deep body temperature drops to a critically low level, producing a state of coma, and in time, death.

50. Exposure to a low environmental temperatures in flight due, for example, to the loss of the canopy, can therefore become a limiting factor in deciding the altitude at which the flight can be continued. In many cases it may be necessary to initiate immediate descent and even then frostbite of the exposed areas of the body may occur, particularly if the aggravating factor of wind-chill is present. The chances of frostbite occuring will be greater if hypoxia is present.

51. In the event of high altitude escape, there is a marked possibility of frostbite, but even a light covering, such as afforded to the hands by cape leather gloves, is sufficient to delay and even prevent serious damage.

Cabin Pressurisation

52. Aircrew operating aircraft at moderate and high altitudes are normally protected against the effects of exposure to the environment in which the aircraft is flying by pressurisation of the crew compartment.

Conditioned air is fed into the cabin and allowed to escape through discharge valves. The opening of the discharge valves is controlled so that the desired pressure difference is created between the interior of the cabin and the external environment of the aircraft.

53. Man is accustomed to sea level conditions so it would be ideal to maintain sea level pressure in the aircraft cabin at all times. For military aircraft, however, this is impracticable and not always desirable from the point of view of weight, complexity and the hazards arising from loss of pressure due to enemy action. In practice, the pressure differential and thus the cabin altitude, is chosen for a particular aircraft as a compromise between the physiological ideal and the proposed performance and role of the aircraft.

54. Two major types of cabin pressure schedules are employed in military aircraft, namely high differential and low differential. In aircraft with high differential pressure cabins the maximum cabin altitude is generally 8,000 feet. A differential pressure of 9.2 psi is required at an aircraft altitude of 50,000 feet to produce a cabin altitude of 8,000 feet. High differential pressure cabins are typically used in large aircraft such as medium bombers, maritime reconnaissance and transports. The crew and passengers flying in this type of pressure cabin normally breathe cabin air throughout flight. Oxygen equipment is fitted in order to provide protection against hypoxia in the event of a decompression. In combat situations, when the risk of decompression is increased, some or all of the crew may use their oxygen equipment at a cabin altitude of 6,000–8,000 feet in order to ensure full protection against hypoxia should cabin pressurisation be lost. The degree of pressurisation employed in the low differential pressure schedule is such that at the altitude ceiling of the aircraft the cabin altitude is in the range 20,000 to 25,000 feet, the exact value varying from one aircraft type to another. A maximum differential pressure of 4–5 psi is typically employed in this type of pressurisation schedule. The low pressure differential schedule is used in fighter type aircraft, where the risk of failure of the pressure cabin due to battle damage or loss of a canopy is higher and the large weight penalty of a high differential pressure cabin in unacceptable. Crew operating low differential pressure cabin aircraft use their oxygen equipment throughout flight. Most bombers with high differential pressure cabins are also fitted with a cabin pressure control system whereby a low differential pressure schedule can be selected, as desired, in flight.

Loss of Cabin Pressure

55. The pressurisation of the cabin of an aircraft may fail because air is no longer pumped into the cabin, there is a failure in the cabin pressure control system or a defect develops in the wall of the cabin. In military aircraft the jettisoning of a canopy prior to ejection is an example of the latter type of failure. The rate at which the cabin altitude increases varies with the type of failure, the aircraft and cabin altitudes, and the size of the orifice or defect in the cabin wall. When a defect in the wall of the pressure cabin is the cause, the final cabin altitude after loss of pressure may exceed considerably the actual altitude of the aircraft. This additional reduction of the pressure within the cabin is due to the external flow of air over the defect. The effect is termed aerodynamic suction. Its magnitude varies from aircraft to aircraft, with the position of the defect, and with the aircraft speed. It can result for example in a cabin altitude of 50,000 feet at an aircraft altitude of 40,000 feet. Loss of cabin pressurisation does not necessarily imply loss of cabin heating since, if the failure is in the integrity of the cabin wall, hot air will continue to enter the cabin from the engines. Large aircraft also have a considerable heat capacity so that a period of time may elapse before the cabin air temperature approaches that of the external environment.

56. Failure of a pressure cabin has two distinct groups of effects upon the cabin occupants. The first group are caused by the change in pressure itself and include lung damage and abdominal distension. The second group of effects are due to the exposure of the occupants to increased altitude.

a. The severity of the first group of effects is related to the magnitude of the pressure change and the rate at which it occurs. Even when the loss of cabin pressure is very rapid the incidence of lung damage will be infinitesimally low. Following rapid decompression a small proportion of aircrew may suffer from abdominal distension.

b. The incidence and severity of the effects which arise due to the exposure to increased altitude are closely related to the final cabin altitude. The most important effect is hypoxia, and its magnitude is influenced by whether the crew are breathing air or oxygen (para 18). Decompression sickness is rare if the duration of the exposure to high altitude is short (few minutes only). If, however, oxygen lack is prevented and the occupants of the cabin are exposed to altitudes in excess of 25,000 feet for any length of time, some of them will develop decompression sickness (para 29). A reduction in cabin temperature may be associated with loss of cabin pressure. If the duration of exposure to low temperature is short little reduction in efficiency will occur. Directly the exposure is extended beyond a few minutes, however, serious impairment of performance and injury will occur.

57. Thus, in summary, the principle physiological hazard associated with failure of the pressure cabin of an aircraft at high altitude is hypoxia. If descent to low altitude is delayed for operational or structural reasons then decompression sickness or the effects of low temperature or both together will be added to the risk of hypoxia. The immediate action to be taken in the event of a failure of cabin pressurisation at altitude is to ensure that oxygen is being delivered to the oxygen mask and that the latter is adequately sealed to the face. Whenever structural and operational considerations allow, immediate descent to as low an altitude as possible should be carried out at the maximum practical rate. Rapid descent is essential when a decompression results in a cabin altitude greater than 40,000 feet since none of the pressure breathing systems available in the Royal Air Force provides long duration protection against hypoxia or decompression sickness.

58. Whenever a decompression results in a cabin altitude greater than 22,000–25,000 feet descent to a cabin altitude below this level should be carried out as soon and as quickly as operational considerations allow. When passengers are being carried in transport aircraft immediate emergency descent so that the cabin altitude is reduced to less than 15,000 feet (ideally 8,000 feet) is essential, even if passenger oxygen equipment is available, since it is unlikely that more than half the passengers will use the latter correctly during and immediately after the decompression. Should fuel and operational considerations make maintenance of a higher cabin altitude essential then the re-ascent should only be performed after the appropriate checks that the passengers are receiving oxygen have been made.


GROSVENOR HOUSE
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