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Aviation Medicine

Problems of altitude

I: Hypoxia and hyperventilation

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"Even modern aircraft still expose their passengers to some risks from lowered atmospheric pressure."[1]

Concorde commonly cruises at altitudes of 50 000-60 000 ft (15 240-18 288 m) where, if unprotected, its occupants would be unconscious within 15 seconds and dead four to six minutes later from lack of oxygen.[2] Even at the more usual cruising altitudes of commercial aircraft—30 000-40 000 ft (9144-12 192 m)—unprotected passengers and crew would rapidly succumb. Of course this does not happen, thanks to the protection of the aircraft pressure cabin. The well being of the air traveller within this artificial environment is, however, always threatened by the problems of altitude extending beyond lack of oxygen to decompression sickness, cold, and simple pressure effects.

Physics of the atmosphere

Ascent to altitude is associated with a fall in air pressure parallel by decreases in density and temperature. Thus at 18 000 ft (5486 m) atmospheric pressure has fallen to half its value at sea level and the ambient temperature to about −20°C (fig 1). The fall in total atmospheric pressure and the consequent reduction in the partial pressure of oxygen (Po_2) poses the greatest single threat to anyone who flies, hypoxia. Fortunately, the relationship between oxygen saturation of haemoglobin and oxygen tension, reflected in the shape of the oxygen dissociation curve, minimises the effect. The plateau represents an inbuilt reserve, which is exploited by aircraft designers, and provides protection against hypoxia up to an altitude of 10 000 ft (3048 m) (fig 2). Ascent to this altitude produces a fall in alveolar Po_2 from the normal 13·7 kPa (103 mm Hg) to 8·0 kPa (60 mm Hg) but only a slight fall in percentage saturation of haemoglobin with oxygen. As the altitude progressively rises above 10 000 ft (3048 m) the percentage saturation of haemoglobin falls precipitously and results in hypoxia.

Cabin pressurisation and decompression

Commercial aircraft cabins are pressurised to below 10 000 ft (3048 m)—usually between 5000 and 7000 ft (1524-2134 m)—both as a safety margin for passengers (see previous article, 16 April, p 1229) and because psychomotor performance at novel tasks, which is of relevance to aircrew, deteriorates at altitudes of 8000 ft (2438 m).[3] Pressurisation to sea level, though ideal, is not cost effective.

Aircraft maintain a positive ambient pressure within their cabins by drawing in external air and delivering it compressed to the cabin. The outflow of cabin air is then controlled so as to maintain the required pressure differential. The through flow also ventilates the cabin and provides a means by which the ambient temperature may be controlled. As long as the pressurisation system and the aircraft remain intact, protection is provi-

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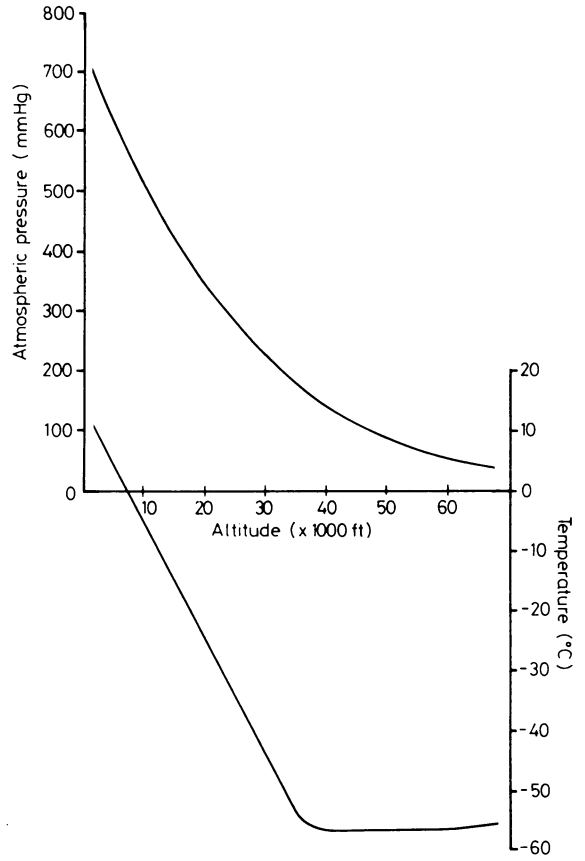

FIG 1—Relation between altitude and pressure and between altitude and temperature.
Conversion: traditional to SI units—atmospheric pressure: 100 mm Hg ≈ 13·3 kPa. (Altitude: 5000 ft ≈ 1500 m.)

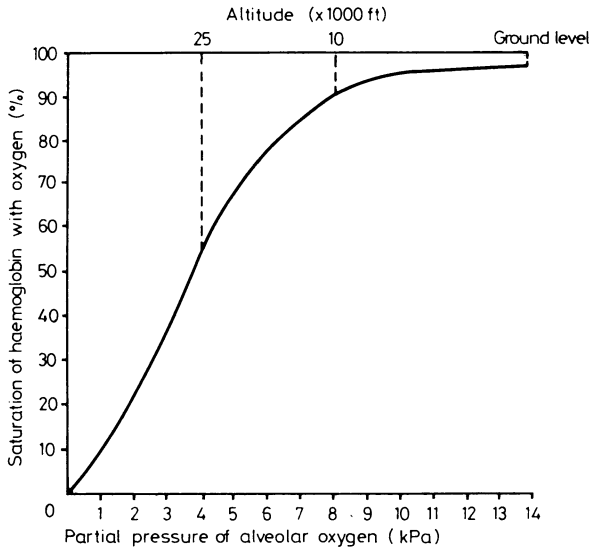

FIG 2—Relation between partial pressure of alveolar oxygen, percentage saturation of haemoglobin with oxygen, and altitude.
Conversion: SI to traditional units—partial pressure of alveolar oxygen: 1 kPa ≈ 8 mm Hg. (Altitude: 5000 ft ≈ 1500 m.)

ded at normal operating altitudes. In practice the degree of pressurisation increases linearly with, but at a slower rate than, actual altitude, from ground level to a high maximum differential pressure (fig 3). Thereafter, cabin altitude increases at the same rate as aircraft altitude along a line determined by the pressure characteristics of the hull, the maximum differential pressure line.

The problems of altitude become manifest if and when cabin

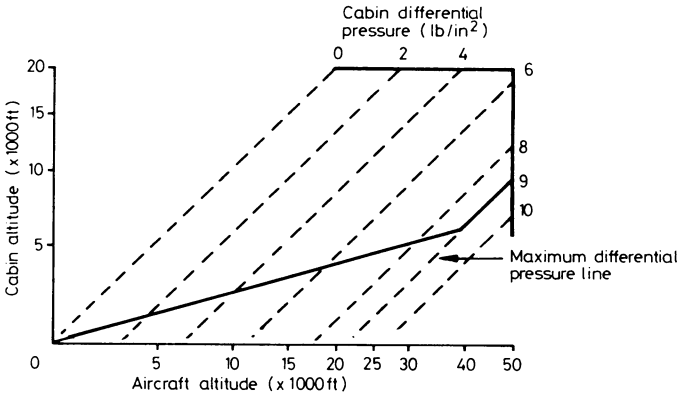

FIG 3—Typical subsonic passenger aircraft pressurisation profile. In this example the maximum differential pressure line is reached when aircraft altitude is 39 500 feet (12 040 m) and cabin altitude is 6000 ft (1929 m).
Conversion: traditional to SI units—cabin differential pressure: 1 lb/in² ≈ 0·07 kg/cm². (Altitude: 5000 ft ≈ 1500 m.)

pressurisation fails. Such failures still occur in both civil and military aircraft, large and small.[4-6] In the case of a slow loss of pressurisation—for example, as a result of malfunction of a control system—the cabin altitude will increase slowly; this is usually rapidly recognised and dealt with by the crew. Hypoxia in susceptible subjects is the main danger. Effects are more dramatic when a rapid decompression occurs—for example, when a window is lost or the pressure hull is ruptured by an explosion. In these cases there is a massive movement of air out through the defect, which will carry with it any loose articles and even passengers close to the breach who are not wearing their seat belts.[4] The air movement creates considerable noise and the sudden cooling causes condensation and misting, thus making both communication and vision difficult. These initial events are rapidly succeeded by the problems of hypoxia, perhaps complicated by cold injury (frostbite and frostnip), and decompression sickness. The rate, duration, and effects of rapid decompression depend on the external and cabin altitudes at the moment of decompression, the volume of the cabin, and the size of the defect.

Combat aircraft are pressurised to a lesser degree so as to achieve a lower structural weight and improve the power to weight ratio. In addition, the risk of sudden loss of cabin pressurisation in these aircraft is increased as a result of enemy action. The degree of pressurisation depends on actual altitude, as in commercial aircraft. The oxygen deficit when cabin altitudes exceed 10 000 ft (3125 m) in military aircraft is compensated for by oxygen enrichment of the air as altitude increases, until at about 30 000 feet (9144 m) 100% oxygen is being delivered. The supply is fed individually to each crew member through a regulator and mask.

Hypoxia

PHYSIOLOGICAL FACTORS

The earliest feature of hypobaric hypoxia is often a subtle personality change perhaps coupled with euphoria, lack of judgment, loss of short term memory, and mental incoordination. This combination is not unpleasant and resembles the early stages of alcoholic intoxication, but its insidiousness is of the greatest danger to the victim. In a crew member it may be disastrous since he is unaware of his failing performance. Subsequent features reflect the stimulation of cardiovascular and respiratory compensatory mechanisms. In moderate hypoxia—for example, air breathing at 25 000 ft (7620 m)[6]—cardiac output and heart rate are increased but overall peripheral resistance is reduced, so that mean arterial blood pressure is unchanged. Cerebral blood flow is increased, though the

degree of increase is modified by the magnitude of coexisting hypocapnia that results from increased respiratory minute volume. Thus cerebral and cardiac perfusion is increased at the expense of less vital organs. Muscular incoordination develops—including slurred speech—and this, together with loss of touch sensation, may prevent the victim from taking effective remedial action. The special senses are also affected; classically, tunnelling of vision occurs, though non-specific visual symptoms are more common. Loss of auditory acuity is a late feature, however, and even then is not pronounced.

Respiration increases under the hypoxic drive to help alleviate cerebral hypoxia but is ineffective, and the symptoms and signs of hyperventilation develop with those of hypoxia. Hyperventilation is a normal response to a fall in alveolar P_{O_2} to below 7·3-8·0 kPa (55-60 mm Hg) and indeed may be the dominant clinical feature. Lightheadedness, feelings of unreality and anxiety, paraesthesiae, visual disturbances—for example, blurring and scotomata—and palpitations are common in people exposed suddenly to altitudes of 25 000 ft (7620 m).

The features of hypoxia vary between subjects, and the rate at which they develop depends on the severity of the hypoxic insult. Thus most of the changes described occur within three to five minutes of sudden exposure to an altitude of 25 000 ft (7620 m) (by which time alveolar $P_{O_2} = 4{\cdot}0$ kPa (30 mm Hg) and alveolar $P_{CO_2} = 2{\cdot}9$ kPa (22 mm Hg),[6] but above 45 000 ft (13 716 m) unconsciousness, often preceded by convulsions, supervenes within 15-20 seconds.

The treatment for hypobaric hypoxia is with oxygen. In passenger aircraft oxygen is delivered through automatic drop down sets whenever the cabin altitude exceeds a certain level, usually 12 000-14 000 ft (3658-4267 m). Each set has a mask attached which should be immediately placed over the face. In many systems the action of pulling the mask with its delivery tube attached on to the face initiates the flow of oxygen. Even this simple manoeuvre may be beyond the ability of passengers when they are exposed to rapid decompression at high altitude.[4 7] Manipulating the equipment may be further complicated if the stricken aircraft subsequently descends rapidly and the passengers are flexed forward in their seats. Seats facing backwards would prevent such flexing and so make the oxygen mask easier to use.[8] The crew on the flight deck are equipped with more elaborate oxygen systems—similar to those in military aircraft—to enable them to accomplish, unimpaired, a rapid descent to a safe altitude of less than 10 000 ft (3048 m). The cabin staff have portable oxygen sets for their own use while helping passengers.

A degree of hypoxia will develop in all occupants of an aircraft when breathing air at altitudes above 10 000 ft (3048 m). Some subjects may, however, become hypoxic below this altitude and will require therapeutic oxygen in the aircraft, while their fellow passengers remain fit and well. Oxygen may be given through the passenger's own automatic set or from a portable emergency supply (see previous article, 9 April, p 1204).

MEDICAL FACTORS

Many factors may cause one person to be more susceptible to lack of oxygen than another. Ill health, especially cardiovascular and respiratory disorders (see previous article, 16 April, p 1269) may obviously lead to problems. Alcohol and other drugs, particularly proprietary cold cures (especially those containing antihistamines), may potentiate the lack of oxygen. Carbon monoxide preferentially displaces oxygen from the haemoglobin molecule and so smoking may raise carboxyhaemoglobin concentrations sufficiently to reduce the reserve, despite enzymatic and haematological compensatory mechanisms. In addition, the interaction of carbon monoxide with haemoglobin shifts the oxygen dissociation curve to the left so further reducing the amount of oxygen available for release to the tissues.[9] Thus an established heavy smoker may already be at an equivalent altitude of several thousand feet while walking to his aircraft. Finally, any condition that increases oxygen requirements, such as exercise or cold, will reduce tolerance to hypoxia.

Occasionally symptoms and signs worsen transiently when oxygen is restored to a hypoxic patient. This oxygen paradox probably results from a combination of a reflex peripheral arteriolar vasodilatation with consequent hypotension and a persistent cerebral vasoconstriction due to hypocapnia associated with hypoxia.[6] The phenomenon is usually mild and lasts for 15-60 seconds, after which recovery is rapid provided that oxygen administration is maintained. Occasionally, however, the paradox may produce convulsions and loss of consciousness.

Hyperventilation

Hypoxia is not the only cause of hyperventilation in flight, but it is the most important and difficult aspect of the differential diagnosis of hyperventilation. Other causes include other environmental stresses, such as whole body vibration in air turbulence, motion sickness, and high ambient temperatures. The most common cause, however, is anxiety or emotional stress. A large proportion of aircrew under training hyperventilate,[10] as do experienced aircrew, especially when confronted with an unusual or severe mental stress such as an inflight emergency. Passengers who are inexperienced air travellers are often affected. The symptoms and signs of hyperventilation (as described above) are all attributable to the resulting hypocapnia.[11] Very rarely does unconsciousness occur, or even tetany. The usual clinical picture is an increasingly anxious passenger with early symptoms requiring help. The simple but effective rebreathing of expired air is the treatment of choice together with a logical explanation but firm instructions to breathe more slowly. In passengers who habitually hyperventilate sedation before the flight may be indicated. Experience and training will reduce the likelihood of hyperventilation among crew members.

Despite the fact that hyperventilation is probably more common during flight than hypoxia, aircrew must assume that any suspicious symptoms or signs are due to hypoxia whenever the cabin altitude exceeds 10 000 ft (3048 m) and not to hyperventilation from another cause. They must also take appropriate remedial action. The importance of the similarities between hyperventilation due to hypoxia and hyperventilation alone, and the possibly fatal implications of misdiagnosis, should be emphasised during training.

Next week's article covers further medical problems due to changes in atmospheric pressure while flying.

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