Extremes of barometric pressure

Jane E Risdall
David P Gradwell

Abstract

Ascent to elevated altitude, commonly achieved through flight, by climbing or by residence in highland regions, exposes the individual to reduced ambient pressure. Although there are physical manifestations of this exposure as a consequence of Boyle's law, the primary physiological challenge is of hypobaric hypoxia. The acute physiological and longer-term adaptive responses of the cardiovascular, respiratory, haematological and neurological systems to altitude are described, together with an outline of the presentation and management of acute mountain sickness, high-altitude pulmonary oedema and high-altitude cerebral oedema. Whilst many millions experience modest exposure to altitude as a result of flight in pressurized aircraft, fewer individuals are exposed to increased ambient pressure. The pressure changes during diving and hyperbaric exposures result in greater changes in gas load and gas toxicity. Physiological effects include the consequences of increased work of breathing and redistribution of circulating volume. Neurological manifestations may be the direct result of pressure or a consequence of gas toxicity at depth. Increased tissue gas loads may result in decompression illness on return to surface or subsequent ascent in flight.

Keywords Acclimatization; acute mountain sickness; decompression illness; hyperbaric exposure; hypobaric hypoxia; inert gas toxicity

Royal College of Anaesthetists CPD Matrix: 1A01

Learning objectives

After reading this article, you should be able to:

- understand the physical effects of changes in ambient pressure and the physiological consequences on the cardiovascular respiratory and neurological systems
- gain an awareness that exposure to reduced ambient pressure produces both acute and more chronic effects, with differing signs, symptoms and time to onset at various altitudes
- develop an awareness of the toxic effects of 'inert' gases at increased ambient pressures and the pathogenesis and management of decompression illness

Most of the world's population lives close to sea-level. Meteorological changes in ambient pressure have little effect on respiratory physiology. Although adaptation to high altitude is seen in those living for prolonged periods at 3000 m or more above sea-level, millions of people are exposed acutely to changes in ambient pressure as a result of flight in aircraft. A few are exposed to more substantial changes in pressure by undertaking extreme ascents or by diving to depth.

High altitude – low pressure

Barometric pressure declines exponentially on ascent. As pressure declines, gas volumes increase and so a fixed volume of gas contains fewer molecules. Thus, although the percentage of oxygen in air remains constant at 20.9%, the partial pressure of oxygen in inspired air falls progressively. This causes a reduction in the pressure gradient for oxygen from the inspired gas to the tissues and hence into mixed venous blood (Figure 1). Therefore ascent to altitude is a specific, hypobaric, cause of hypoxic hypoxia.

Flight within the normal pressurized cabin of a commercial aircraft is equivalent to ascending to 2500 m (8000 ft) and associated with a reduction in barometric pressure of 25%. Ascent to 4000–5000 m (15,000–16,500 ft) will induce hypoxia of such a degree as to stimulate hyperventilation, slow mental processes, impair learning and memory and reduce co-ordination. However, impairment of night vision has been reported at altitudes as low as 1500 m. Ascent to altitudes greater than 5000 m will result in progressively more rapid loss of consciousness. Studies have shown that individuals exposed to an altitude of 7600 m (25,000 ft) for 4–5 minutes will cease to be able to act in a rational manner to correct their predicament. This interval is described as the time of useful consciousness. The time of useful consciousness will be reduced at 9000 m (30,000 ft) to 2.5 minutes and to just 1 minute at 10,500 m (35,000 ft).

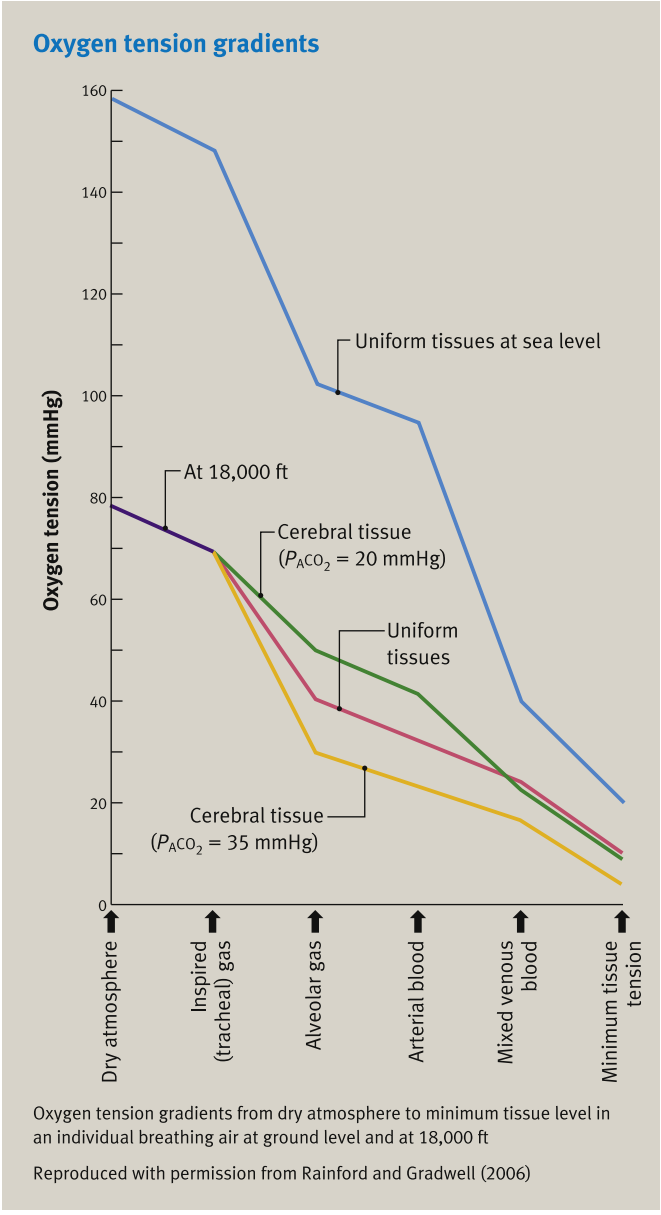
Respiratory system

Oxygen uptake: normal oxygenation is dependent on adequate alveolar ventilation. When alveolar PO_2 is normal there is rapid diffusion of oxygen molecules through the alveolar–capillary membrane into the red blood cells (RBCs) and then into combination with haemoglobin. At rest, the time course for the transit of an RBC through a pulmonary capillary is of the order of 0.75 seconds. At sea-level, breathing air, within 0.25 seconds the oxygen tension in the blood within the capillary approaches that found within the alveoli. However, at altitude the alveolar PO_2 is reduced so the rate of rise of oxygen tension within capillary blood is slower than at sea level. At rest the capillary transit time is still sufficient to allow the oxygen tension to approach that within the alveoli, but when the transit time is reduced by exercise there will be a marked worsening of any hypobaric hypoxia (Figure 2).

Ventilatory response to hypoxia: in most individuals hypoxia induces an increase in ventilation. Although this increases the respiratory work oxygen demand there is an overall fall in

Jane E Risdall *is a Consultant Anaesthetist in the Royal Navy and a Senior Lecturer in Military Anaesthesia and Critical Care. Conflicts of interest: none declared.*

David P Gradwell *is the Senior Consultant in Aviation Medicine in the Royal Air Force, working at the Centre of Aviation Medicine, Henlow. He is the Royal College of Physicians (London) Whittingham Professor of Aviation Medicine and is a Visiting Professor at King's College, London. Conflicts of interest: none declared.*



Oxygen tension gradients from dry atmosphere to minimum tissue level in an individual breathing air at ground level and at 18,000 ft

Reproduced with permission from Rainford and Gradwell (2006)

Figure 1

arterial PCO_2 and a rise in arterial PO_2. This hypoxia-induced ventilatory response is mediated through the aortic and carotid bodies. The associated fall in $PaCO_2$ and the consequent respiratory alkalosis, acts centrally as a respiratory depressant, setting up a conflict in response, the outcome reflecting the balance between the two influences. Respiratory control commonly is more sensitive to changes in $PaCO_2$ and an acute reduction in PaO_2 does not induce a respiratory response until the oxygen tension falls to 6–6.7 kPa (45–50 mmHg). Below this level hypoxia induces a brisk increase in ventilation, exerting a stronger effect than the fall in $PaCO_2$ or hydrogen ion concentration.

The response to acute exposure to altitude is altered in those who acclimatize. The time course of acclimatization varies but the most important changes are in the cardio-respiratory systems and in the composition of the blood. Over a few days at altitude the respiratory alkalosis is corrected by increased excretion of bicarbonate, restoring pH towards normal. There is also an increase in the sensitivity of the respiratory centre to $PaCO_2$, so that despite lowered carbon dioxide tensions in the blood, higher ventilation is maintained. There is also a secondary polycythaemia so that more oxygen is carried by the blood.

Oxyhaemoglobin: when breathing air at sea-level, approximately 98.5% of the oxygen carried in blood is combined with haemoglobin. The shape of the dissociation curve means that moderate variations in PaO_2 have relatively little effect on haemoglobin saturation and hence the oxygen content of the blood. When oxygen tension falls significantly, at altitudes above 3000 m (10,000 ft), oxygen is more readily dissociated from haemoglobin and delivery of oxygen to the tissues is compromised. The presence of a significant proportion of reduced haemoglobin in arterial blood gives rise to cyanosis at altitude.

At altitude, the low $PaCO_2$ moves the oxyhaemoglobin dissociation curve to the left. However, an increase in 2,3-diphosphoglycerate, moves it to the right, but the effect of the alkalosis predominates.

Cardiovascular responses

Heart rate: heart rate is increased on acute exposure to altitudes above 2000–2500 m (6000–8000 ft). At 4500 m (15,000 ft) the rate is about 10–15% above the resting level at sea-level and approximately doubled at rest at 7500 m (25,000 ft).

Stroke volume: stroke volume remains unchanged initially, but with acclimatization to altitude stroke volume is reduced.

Blood pressure: mean arterial pressure remains unchanged on exposure to altitude. Systolic pressure is usually raised and there is an overall reduction in peripheral resistance, with a resulting increase in pulse pressure.

Cardiac output: since heart rate increases and stroke volume is unchanged, cardiac output increases on acute exposure to altitude in proportion to the increase in rate. With a subsequent reduction in stroke volume the cardiac output declines. Well-acclimatized individuals demonstrate a normal relationship between cardiac output and work load, but on initial exposure to altitude an exaggerated response to exercise is observed. The development of a degree of polycythaemia may be the means by which more normal oxygen delivery to the tissues is restored with acclimatization.

Regional cardiovascular effects: hypoxia increases blood flow through the coronary and cerebral circulations, at the expense of renal, splanchnic and cutaneous blood flow. Flow through skeletal muscle may increase by 30–100%. Coronary blood flow increases in parallel with heart rate, matching the metabolic requirements of the myocardium. This prevents the emergence of electrocardiographic signs of hypoxia, even up to the point at which consciousness can be lost but in severe hypoxia myocardial depression will influence the ECG results with T depression and reduced height of the T-wave observed.

The response of the cerebral circulation is determined by the relationship between oxygen and carbon dioxide. At oxygen