Acute Exposure to Altitude

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Abstract

Acute exposure to altitude principally encompasses aviation and space activities. These environments can be associated with very acute changes in pressure, oxygenation and temperature due to rates and magnitude of ascent that are not experienced in more chronic exposure such as mountaineering. The four key physiological challenges during acute exposure to altitude are: hypoxia (and hyperventilation), gas volume changes, decompression sickness and cold. The brief nature of aviation exposure to altitude provides little opportunity for acclimatisation, leading to markedly different effects when an individual is exposed to the same altitude acutely compared with an acclimatised individual climbing an 8000m (26 347ft) peak. Challenges such as hypobaric decompression sickness are not considered a hazard for chronic altitude exposure but are routine considerations for those flying to high altitude. Protective systems are essential for aircrew and passengers to survive and function during acute exposure to altitude.

Introduction

A modern fast-jet can ascend at a rate of 330m (1000ft) per second and, from brakes-off on the runway, can reach 10 688m (35 000ft) at Mach 1.5 in 2.5 minutes [1]. The sudden loss of aircraft cabin pressurisation (rapid decompression, RD) can ascend occupants from a cabin altitude of 2438m (8000ft) to the outside altitude of 12 192m (40 000ft) or more in a matter of seconds. It is the rate and magnitude of acute exposure to altitude in the aviation environment that differentiate the challenges from those of chronic exposure to altitude. This article presents the effects of acute exposure to altitude and provides some contrast to chronic altitude exposure. The effects described relate to healthy aircrew; the implications for passenger fitness to fly with co-morbidities are beyond the scope of this review.

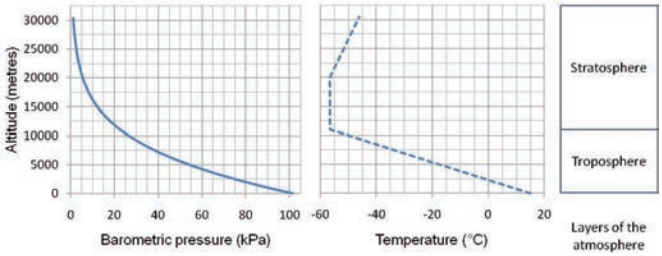

Figure 1. The relationship between pressure, temperature and altitude as described by the ICAO standard atmosphere up to an altitude of 30 480m (100 000ft). The lower two layers of the atmosphere are the troposphere and stratosphere. Temperature continues to rise to the top of the stratosphere (around 48 158m/158 000ft) peaking at -3°C.

The atmosphere and environmental challenges

The chemical composition of the atmosphere is broadly constant from sea level up to an altitude of about 100 000m (330 000ft): 78.09% nitrogen, 20.95% oxygen, 0.93% argon, 0.03% carbon dioxide and traces of rare gases such as neon and helium [2]. The atmosphere is composed of different layers that are defined by their physical characteristics with the lowest two layers, the troposphere and stratosphere, of most relevance to aviation (Figure 1). In addition to the gases described, air in the troposphere contains water vapour and turbulent weather systems.

As one ascends from sea level the principal challenges to aircrew arise from an exponential fall in atmospheric pressure and a drop in temperature (Figure 1). The International Civil Aviation Organisation (ICAO) established a model standard atmosphere to facilitate the identification of standardised international aircraft pressure altitudes, which are elemental to establishing safe altitude separation between aircraft in busy commercial airways [2]. The model assumes a pressure of 760mmHg (101.3kPa) and temperature of +15°C at sea-level with a linear decrease in temperature of 1.98°C per 305m (1000ft) up to 11 000m (36 089ft), the top of the troposphere (Figure 1). Where pressure altitudes are used in this article they refer to the ICAO standard atmosphere. In reality there is significant variation with latitude and season of the year which is of relevance to high altitude parachutists and mountaineers.

The fall in pressure with ascent causes a reduction in oxygen partial pressure (hypobaric hypoxia), expansion of gas trapped in the body, a risk of decompression sickness (DCS) above 5486m (18 000ft), and a risk of ebullism (vapourisation of body tissue water) above 19 202m (63 000ft). In addition to the challenges of acute altitude aircrew may be exposed to heat stress, ozone, radiation, G-forces, noise, communication, vibration, human factors, ejection, or crashes.

Hypoxia and hyperventilation

The effects and risks of acute exposure to altitude were well described many years ago by the early hot air balloonists, including the tragic 1875 ascent of Tissandier, Crocé-Spinelli and Sivel aboard their Zenith balloon: *"Towards 7500m (24 606ft), the numbness one experiences is extraordinary.... One does not suffer at all; on the contrary. One experiences inner joy, as if it were an effect of the inundating flood of light. One becomes indifferent.... Soon I wanted to seize the oxygen tube, but could not raise my arm… Suddenly I closed my eyes and fell inert, entirely losing consciousness."* [3]. Crocé-Spinelli and Sivel were dead when the balloon reached the ground, a result of the severe hypoxia and Tissandier was lucky to survive to tell the tale. Hypoxia is still often called the single

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most important physiological hazard of high altitude flight [4] and the enduring danger is evident in the incidents and deaths it has caused in aviation over many years [4-6]. To mitigate this risk military aircrew are trained to recognise the symptoms of hypoxia in themselves and others in a controlled training environment (hypobaric chambers) so that they can take the appropriate early corrective action were it to occur in flight.

Physiological effects of acute hypobaric hypoxia

The partial pressure of oxygen in moist inspired air can be expressed: $PIO_2 = 0.2095 \times (PB - 47)$. Therefore, as barometric pressure (PB) reduces it causes hypobaric hypoxia. Inhaled air is warmed to body temperature and becomes saturated with water vapour (47mmHg (6.3kPa) at 37°C) displacing other gases. Body temperature does not change with altitude so the effect of water vapour becomes proportionately greater with increasing altitude; at sea level it comprises 6% of the total inspired gas pressure increasing to 19-20% at the summit of Mount Everest (8848m; 29 029ft) and 33% at 12 192m (40 000ft).

Ventilatory response

At sea level the principal driver to increase ventilation is rising arterial partial pressure of carbon dioxide ($PaCO_2$) sensed by central chemoreceptors (medulla and other brain stem sites) through coupled changes in cerebrospinal fluid PCO_2 and pH. By contrast, the peripheral chemoreceptors (carotid and aortic bodies) principally sense hypoxia [7-9] and to a lesser degree carbon dioxide and pH [10]. On ascent to altitude ventilation increases in a hyperbolic manner (hypoxic ventilatory response) in response to reduced PO_2 although the resultant hyperventilation, hypocapnia and respiratory alkalosis [11] reduce the ventilatory drive from PCO_2, ameliorating the increase in ventilation.

The alveolar-arterial oxygen gradient

Oxygen and carbon dioxide move passively through the blood-gas barrier in the lungs as described by Fick's law of diffusion. The diffusion of oxygen from alveoli to pulmonary capillary blood is limited during exercise at moderate altitude [12] and rest at extreme altitude [13]. This is due to a combination of reduced oxygen pressure difference driving diffusion between alveolar gas and capillary blood, and increased cardiac output reducing pulmonary capillary transit time [14]. Whilst this may cause some exercise limitation in chronic exposure, in acute exposure it can cause or hasten loss of consciousness at lower altitudes than would otherwise be expected. Indeed, the recent reports of hypoxic symptoms in helicopter aircrew at altitudes of less than 3048m (10 000ft) [15] appear to be exercise related.

Cardiovascular effects

Tissue oxygen delivery is the product of arterial oxygen content and cardiac output and the latter is increased by acute hypoxia both at rest and for a given level of exercise [16-18]. This is mainly due to an increase in heart rate of 40-50% and 100% above sea level values with acute exposure breathing air to simulated altitudes of 4000-4600m (13 123-15 092ft) [17-18] and 7620m (25 000ft) [19] respectively, which is thought to be due to increased sympathetic activity [20]. There is no consistent change in stroke volume during either rest or exercise during acute altitude exposure [18] and no change in mean systemic arterial blood pressure in humans during acute exposure up to 4600m (15 092ft) [17-18]. Blood flow to the kidneys [21], viscera and skin [22-23] is reduced while it is increased to the brain [24-25], respiratory muscles, adrenals [26] and heart [27].

Cerebrovascular effects of acute exposure to altitude

Hypoxaemia causes cerebral vasodilation [28] with significant increases in cerebral blood flow when PaO_2 <55-60mmHg (7.3-8.0kPa) [24-25]. In contrast, hypocapnia and the associated increase in pH cause a strong cerebral vasoconstrictive response [29]. Harper and Glass [30] found cerebral blood flow was reduced by 40% when $PaCO_2$ fell to 15mmHg (2.0kPa) with PaO_2 maintained at normal levels in mechanically ventilated anaesthetised dogs. Huang and colleagues [31] used Doppler ultrasound to measure internal carotid and vertebral artery flow velocities and found a slight but non-significant increase above sea level values within 2-4 hours of arrival at 4300m (14 108ft).

It has long been known that exposure to acute hypobaric hypoxia can degrade performance or produce complete incapacitation [5]. van Dorp and colleagues [32], using near infra-red spectroscopy (NIRS) and hypoxic gas mixtures (end tidal PO_2 ~40mmHg (5.3kPa); simulating ~5486m (18 000ft)), demonstrated that hypocapnic cerebral vasoconstriction exaggerated both cerebral tissue hypoxia and the degradation of performance. They also demonstrated that the addition of CO_2 to the inspirate improved performance during hypoxia by preventing the hypocapnia-induced pH-associated vasoconstriction of cerebral blood vessels [32], although this is not a new finding and was recognised as early as the Second World War [33]. Acute hypoxia may also cause vasovagal-like syncope in some individuals [34]. It has been suggested that acute exposure to altitude, by increasing cortical activity on EEG, may decrease the threshold for initiation of epileptiform discharge and hence increase susceptibility to seizures [35] although this is not a widely held view.

During acute hypoxia the maintenance of consciousness is a balance between hypoxia and hypocapnia with loss of consciousness at levels that are well tolerated by mountaineers [36]. The competing effects of hypoxia and hypocapnia are a particular concern in general aviation (private-pilot flying). These light aircraft are capable of flying up to 4572-6096m (15 000-20 000ft) at which altitude supplemental oxygen is required, but the aircraft do not have built-in oxygen delivery systems. Some pilots may use small oxygen cylinders and nasal cannulae in combination with pulse oximetry, to maintain saturation at around 90-92%. Hyperventilation raises arterial oxygen saturation and may falsely reassure pilots that they are adequately oxygenated when in reality hypocapnia is worsening cerebral tissue oxygenation and cognitive performance.

Hyperventilation

Hyperventilation leads to hypocapnia that causes dizziness, light-headedness, feelings of unreality, apprehension, neuromuscular irritability, paraesthesia of the face and extremities, and muscle spasms including carpo-pedal spasm (when $PaCO_2$ is less than 15-20mmHg (2.0-2.7kPa)) [37]. Mental and physical performance is impaired and ultimately loss of consciousness (LOC) may ensue. If the cause is voluntary hyperventilation LOC may relieve the cause, facilitating recovery, in stark contrast to hypoxia. It is important for aircrew to know their personal symptoms of hyperventilation-induced hypocapnia because these may be the only indication that they are hypoxic. Aircrew are, therefore, taught to undertake the same drills when faced with symptoms of hyperventilation as for suspected hypoxia when above 3048m

(10 000ft). Other causes of hyperventilation in aviation include emotional stress (anxiety, fear, workload), pain, environmental stresses (heat, whole-body vibration, motion sickness), anti-G straining manoeuvre, or positive pressure breathing for altitude protection above 12 192m (40 000ft) [38].

Signs and symptoms of acute hypobaric hypoxia

"I have slipped the surly bonds of earth and danced the skies on laughter-silvered wings", I "Put out my hand and touched the face of God". Pilot Officer John Magee wrote these famous lines from his poem 'High Flight' following a high altitude (9144m; 30 000ft) test flight in a Spitfire V in September 1941. One theory for his inspiration is the acute in-flight hypoxia that he had experienced a few weeks prior to this when his aircraft oxygen system failed. This section describes the signs and symptoms that can be expected during acute exposure to altitude. There is considerable inter-individual variation in response to acute hypobaric hypoxia [34, 36] and aircrew may be subjectively unaware of the effects on their performance. The concept of a physiological equivalent altitude when breathing 100% oxygen is useful when considering oxygen delivery devices and is included in the following sections [38]. Breathing 100% oxygen at 10 272m (33 700ft), for example, is considered to be physiologically equivalent to breathing air at sea-level (alveolar PO_2 (PAO_2) maintained around 103mmHg (13.7kPa).

Altitudes up to 3048m (10 000ft) breathing air or up to ~11 887m (39 000ft) breathing 100% oxygen

At altitudes of less than 3048m (10 000ft) one may experience breathlessness on exertion and a rise in heart rate with oxygen saturation in the range of 98-87% but would not normally expect other symptoms [38]. Helicopter aircrew have reported features of hypoxia below 3048 m (10 000 ft) [39] and to investigate this Smith [15] exercised six subjects at 30 Watts and 60 Watts for four minutes at sea level, 610m, 2134m, and 2743m (2000ft, 7000ft, and 9000ft). He found that physical activity as low as 2134m (7000ft) can produce arterial haemoglobin desaturation and symptoms of hypoxia similar to that which would normally be expected in a resting person at approximately 3658-4572 m (12 000-15 000 ft) [15].

Denison and colleagues [40] exposed eight subjects to a pressure altitude of 1524m (5000ft) in a hypobaric chamber and found they were slower to learn complex tasks than a matched group breathing an enriched oxygen mix. Night vision has also been shown to be impaired as low as 1524m (5000ft) [41]. Connolly [42] found acute hypoxia (14.1% oxygen in nitrogen simulating 3048 m; 10 000 ft) degraded low contrast acuity progressively with decreasing mesopic luminance and that supplementary oxygen can extend functionally useful vision to lower light levels. These effects of acute hypoxia on night vision may have direct relevance to flight safety; spatial disorientation is more common during night flying with Night Vision Devices than daytime [43] and spatial disorientation is a commonly cited cause of aircraft accidents, 14-30% of major accidents in two studies [44-45]. It is not clear what effect acute hypoxia has on spatial disorientation or human factors, another commonly cited cause of accidents; 77% in one study [46]. These factors may be of relevance to safe altitude limits for flight without supplemental oxygen, particularly at night. Hypoxia has also been listed as a contributing factor in aircraft accidents during recent military operations in Afghanistan.

Between 3048-4572m (10 000ft and 15 000ft) breathing air or between ~11 887-12 854m (39 000ft and 42 500ft) breathing 100% oxygen

In non-pressurised aircraft supplemental oxygen is required for sustained flight above 3048m (10 000ft) and, therefore, based on current regulations aircrew should not be exposed to these effects during routine flight. In unacclimatised subjects it is typically as one ascends above 3048m (10 000ft) that hypobaric hypoxia (PAO_2 <55-60mmHg (7.3-8.0kPa)) triggers hyperventilation [47]. After 20 minutes at 3658m (12 000ft) PAO_2 and $PACO_2$ fall from sea level values of 103 and 37mmHg (13.7 and 4.9kPa) to 51 and 35 mmHg (6.8 and 4.7kPa) respectively [48]. This is exaggerated with increasing altitude and the equivalent values at 4879m (16 000ft) are 45 and 30mmHg (6.0 and 4.0kPa) respectively [48]. At rest oxygen saturations are in the region of 91-78% for subjects acutely exposed to altitudes of between 3048 - 4572m (10 000 and 15 000ft) [38]. While symptoms may be limited at these altitudes they can include headache, visual disturbance, light-headedness, euphoria, fatigue, dyspnoea and an inability to think clearly [49]. Individuals have demonstrated impairment of skilled tasks, reduced response times, reduced exercise capacity, muscular in-coordination, reduced insight, judgement, and short term memory [50-51].

When considering the effect of acute hypoxia on performance the task being undertaken at altitude is important; the cognitive demands and risk in transit at 3658m (12 000ft) are very different from a helicopter landing at night on an unfamiliar snow-covered mountainside. In rotary-wing flying operations rear crew are more physically active and, therefore, at increased risk of exercise exaggerated hypoxaemia [52], which is of particular relevance in medium and large helicopters whose pilots can be heavily reliant on the advice and decision making of rear crew during landing.

4572-6096m (15 000ft to 20 000ft) breathing air or ~12 854-13 716m (42 500ft to 45 000ft) breathing 100% oxygen

At these altitudes higher mental processes and neuromuscular control are impaired with a dangerous reduction in self-criticism, critical judgement, slowed thinking, personality change, disinhibition and pronounced co-existing symptoms of hypocapnia [36, 53-54]. It is possible to become unconscious from hypoxia when breathing air as low as 4879m (16 000ft) with PAO_2 of 40mmHg, if there is marked hyperventilation and hypocapnia [38]. Conversely it is possible to remain conscious for some minutes as high as 7315m (24 000ft) with PAO_2 as low as 25mmHg if there is no hypocapnia [36].

Above 6096m (20 000ft) breathing air or above ~ 13 716m (45 000ft) breathing 100% oxygen

In contrast with mountaineering experience of similar altitudes, above 7000m (22 967ft) most people will lose consciousness during acute exposure, often with little or no warning. An important concept is the 'time of useful consciousness' (TUC; Figure 2), a window of opportunity to recognise the onset of hypoxia and take corrective action before cognitive performance declines too far [55]. During the TUC individuals will experience a more rapid onset of hypoxic symptoms with apprehension, numbness and unsteadiness in addition to those described at lower altitudes and increased muscular in-coordination [56]. TUC may be followed by myoclonic jerks of the upper limbs or convulsions prior to loss of consciousness [38]. Slowing of electroencephalogram activity and loss of consciousness are closely related to jugular venous oxygen

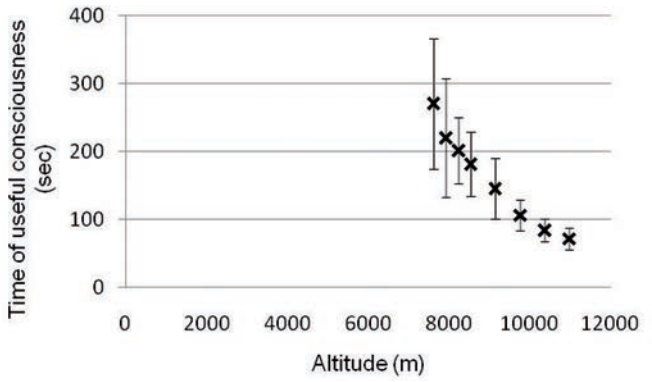

Figure 2. Time of useful consciousness on acute exposure to altitude breathing air [38]. Data presented are mean values with Standard Deviation error bars.

tension and typically occurs at values around 17-19mmHg (2.3-2.5kPa) [57-58]. The corresponding arterial oxygen tension can vary much more widely (20-35mmHg; 2.7-4.7kPa) and is related to cerebral blood flow which is dependent on arterial tensions of oxygen, carbon dioxide and pH [59].

Rapid decompression

The most acute exposure to altitude is that associated with RD, the rate of which is dependent on cabin pressure, atmospheric pressure, and the ratio of the cabin volume to effective cabin wall defect size. Structural failure of a large area of the cabin wall is likely to be catastrophic.

RD is associated with a risk of: i) physical injury from any blast, rushing air or objects; ii) barotrauma of gas containing cavities within the body (lungs, middle ear, gastrointestinal tract or teeth); iii) hypoxia; iv) decompression illness; and v) cold injury. If the initial few seconds are survived hypoxia will reduce TUC, if breathing air, to less than 20 seconds above 10 668m (35 000ft), although this depends on the final altitude, breathing gas prior to RD and time to onset of emergency oxygen (ideally delivered in less than 5 seconds). In addition to the general factors affecting RD the likelihood of lung damage depends on the volume of gas in the lungs at the start of the RD and whether the glottis is open. The aerodynamic suction effect of air rushing over an orifice in a cabin wall can further reduce the cabin pressure and can be of a magnitude equivalent to a further altitude increase of 2134-3048m (7000-10 000ft) in a fast jet with a fully removed canopy at aircraft altitudes up to 10 668m (35 000ft) and speeds of 0.85-0.93 Mach [60], increasing the risks of hypoxia, DCS and cold injury.

Gas volume changes

During ascent ambient pressure drops and gas contained in the body expands; ascent to 5486m (18 000ft) from sea level halves PB and gas contained in the body will double in size in accordance with Boyle's law. On ascent there is passive release of gas from the lungs, sinuses, and middle ear but gastrointestinal tract gas may require some assistance to escape. Gas in the middle ear escapes passively as the pressure builds up opening the Eustachian tube approximately every 152m (500ft) during ascent; this occurs with pressure of 15mmHg (2.0kPa) near sea level decreasing to 3.5mmHg (0.5kPa) at 10 668m (35 000ft) [61]. Poor dentition can lead to severe tooth pain on ascent (orodontalgia). RD may be complicated by trapped intestinal gas, which can cause significant pain with risk of vasovagal syncope, or a closed epiglottis that prevents free escape of lung gas with risk of pneumothorax and arterial gas embolism.

On descent as ambient pressure increases, gas contained in the body reduces in volume. The main risks associated with this change being sinus or otitic barotrauma. Their likelihood is increased by concomitant respiratory tract inflammation, which may occur with infections or hayfever. On descent, the Eustachian tubes become clamped shut by the developing pressure difference and air needs to be actively forced through to equalise the middle ear and prevent otitic barotrauma, which is achieved by swallowing or use of a Valsalva or Frenzel manoeuvre to raise pressure in the nasopharynx. Sinus barotrauma usually presents during descent with sudden onset severe pain that can cause fainting.

Delayed otitic barotrauma is a phenomenon in which aircrew typically wake up with ear pain or deafness the night following a flight breathing 100% oxygen, because oxygen is absorbed from the middle ear twice as quickly as air, causing in-drawing of the tympanic membrane and pain until the pressure is equalised through the Eustachian tube [62-63]. It is prevented by repeatedly clearing the ears while breathing air post-flight to aerate the middle ear.

Alternobaric vertigo is potentially disabling and disorientating and occurs when air pressure in the middle ear equalises at different times on the left and right side [64]. This may occur passively on ascent in the presence of inflammation or old scarring and it may occur on descent due to over-vigorous Valsalva manoeuvres.

Hypobaric (low pressure) decompression illness

Hypobaric decompression illness (DCI) is a rare condition in aviation, encompassing both decompression sickness (DCS; evolved gas) and arterial gas embolism (AGE). DCS may occur in any normal individual under the necessary environmental conditions. AGE in the aviation environment is generally limited to RD and typically presents within minutes of ascent, clinically it is difficult to distinguish from neurological DCS. Hypobaric DCS is considered a risk during acute exposure to altitudes above 5486m (18 000ft) where PB is less than half of sea-level (380mmHg; 50.7kPa), although there are case reports at lower altitudes. DCS can also occur at any altitude when flying after breathing compressed air e.g. SCUBA diving or helicopter dunker Short Term Air Supply training. Therefore, depending on the diving profile and intended cabin altitude 12-48 hours should be spent at ground level before flying after diving.

The risk of hypobaric DCS may be calculated using the Altitude DCS Risk Prediction Computer model and is dependent on magnitude and duration above 5486m (18 000ft), exercise workload and pre-oxygenation [65]. The risk of DCS increases with age [66]. There is significant inter-individual susceptibility to DCS and during World War II aircrew selection tests aimed to identify those less susceptible to DCS for high altitude flying roles [67]. Key to DCS is the evolution of nitrogen micro-bubbles from super-saturated tissues, which occurs in aviation when tissue saturated with nitrogen is exposed to an ambient pressure drop of greater than 50%. The clinical presentation of DCS is wide ranging and one study of 400 cases found joint and limb pains ("bends") in 83% of cases, respiratory symptoms ("chokes") in 2.7%, skin manifestations ("creeps") in 2.2%, and neurological ("staggers") in 0.8% [68]. Cardiovascular collapse may also occur [69]. Hypobaric DCS has a number of distinct differences in contrast with diving related DCS: the risk of hypobaric DCS can be reduced by pre-breathing 100% oxygen (an option not available in diving); symptoms typically occur during flight; descent and return to sea level is therapeutic recompression in aviation and

most cases resolve by sea-level or with the addition of 100% oxygen at ground level [69-70]. In more complicated cases or those that do not resolve on descent definitive treatment for hypobaric DCI, as for diving-related DCI, is recompression in a hyperbaric chamber with hyperbaric oxygen therapy. All UK military cases are discussed with the Royal Navy duty diving medical officer.

The expectation is that most aircrew will return to full unrestricted flying duties and, for example, on recovery from mild DCS without neurological complications the pilot can expect to return to flying within 72 hours provided there are no symptoms and examination is normal [71]. The main aeromedical concern is acute in-flight incapacitation resulting from DCI, but long term neurological impairment is also an important factor.

Military aircrew flying in fast-jets are not routinely exposed to cabin altitudes above 6706m (22 500ft), principally to limit their DCS risk, although a recent paper suggested even this cabin altitude limit should be reviewed and time above 6400m (21 000ft) limited to 30 minutes in the absence of pre-oxygenation and to breathe 100% oxygen during the exposure [72]. Webb and colleagues [72] found a 5% incidence of DCS during six hours breathing 100% oxygen at 6462m (21 200ft) but the incidence increased to 55% at 6858m (22 500ft). In high-altitude parachute operations the multi-engine aircrew and parachutists must pre-breath oxygen for 30 minutes prior to elective cabin depressurisation and exposure to 7620m (25 000ft) ambient pressure altitude. This serves to reduce the body's nitrogen load and reduce the risk of DCS in this population. U-2 pilots can fly with a cabin altitude of 8992m (29 500ft) for more than 15 hours and a survey of U-2 pilot DCS experience found 75.5% had experienced symptoms of DCS in-flight and 12.7% of those had either altered the flight profile or aborted a mission as a result [73]. Since this study U-2 aircrew have adopted a new pre-oxygenation strategy and routinely fly with the pressure suit inflated to reduce their risk of DCS.

Ebullism

Ebullism is the term given to the vapourisation of body water and theoretically occurs when water vapour pressure (47mmHg (6.3kPa) at normal body temperature (37°C)) equals barometric pressure, which occurs at 19 202m (63 000ft). In reality a greater altitude is probably required because the body's normal integument offers some pressure resistance and exposed tissue is normally colder than 37°C.

Ozone and radiation

With higher altitudes one is increasingly exposed to ultraviolet, solar and cosmic radiation, and ozone, the triatomic form of oxygen. Ozone mainly exists in the ozonosphere from around 12 192- 42 672m (40 000 to 140 000ft), created by the irradiation of molecular oxygen in the upper atmosphere by ultraviolet radiation (UV; 200nm wavelength) from the sun. Sea level values of ozone are 0.03 parts per million by volume (ppmv) rising to 1.0 ppmv at 12 192m (40 000ft) with a maximum of 10.0 ppmv at 30 480m (100 000ft) [2]. Ozone is a strong oxident: 0.6-0.6ppmv reduces forced vital capacity and forced expiratory volume in 1 second after 2 hours; 1ppmv causes lung irritation; and 10ppmv may cause fatal pulmonary oedema [2, 74]. It is broken down by longer wavelength UV (210-300nm) in the atmosphere. Ozone is thermally unstable and, therefore, is also broken down when outside air is heated through the engine compressors before it enters the cabin-conditioning system and reaches passengers.

The atmosphere provides protection at sea level from ionising solar and galactic radiation. The primary radiation particles collide with atoms between 18 288m and 36 576m (60 000 and 120 000ft) creating secondary radiation that has less energy but is capable of intense ionization. At 21 336m (70 000ft) the ionizing effect of cosmic radiation is 70 times that at sea-level but the ionizing power diminishes rapidly at altitudes below 15 240m (50 000ft) as further collisions occur with atoms in the atmosphere [2]. The background radiation for sea level inhabitants in the UK is 2.6 millisieverts per year (mSv/yr), and inhaled radon gas contributes the majority of this [75]. For flights in the northern hemisphere mean ambient equivalent radiation dose rates are around 12-15 microsieverts per hour (μSv/hr) for Concorde, 4-5 μSv/hr for long-haul, and 1-3 μSv/hr for short haul [75]. The International Commission on Radiological Protection recommend averaged annual radiation limits of 20 mSv/yr for radiation workers (which includes aircrew and business travellers) and 1 mSv/yr for the general public [75]. This is enshrined in UK legislation in the form of the Ionising Radiation Regulations 1999 which implemented most of the revised European Union Council Directive [76].

Aircrew Protective systems

The aircraft cabin and environmental conditioning systems protect aircraft occupants from weather and temperature extremes. The aircraft cabin is strengthened to maintain a cabin differential pressure such that an aircraft can fly at 10 972-12 192m (36 000-40 000ft), avoiding the turbulent weather at lower altitudes and benefitting from less air resistance, while cabin occupants sit at 1829-2438m (6000-8000ft) pressure altitude. This high cabin differential pressure avoids the need for primary supplemental oxygen systems or risk of decompression sickness but introduces the risk of loss of cabin pressurisation, which may be sudden (RD e.g. failure of a cabin window or the cockpit glass) or slow (e.g. failure of the cabin pressurisation system) and requires an emergency oxygen delivery system. Fast-jet aircraft maintain a lower cabin pressure differential such that the cabin altitude may reach 6706m (22 500ft) while the aircraft cruises at 13 716m (45 000ft) pressure altitude. This reduced cabin differential pressure reduces the hazard from RD, can be achieved with a lighter cabin allowing improved performance and manoeuvrability due to the reduced weight burden, but introduces the need for a primary oxygen delivery system to prevent hypoxia during routine flight, which typically aim to maintain PAO_2 at near sea-level values (103mmHg, 13.7kPa). High performance fast-jets capable of sustained positive (head-to-foot) G-forces (Gz) expose their aircrew to changes in pulmonary physiology that exaggerate sea-level ventilation-perfusion mismatches. During prolonged acceleration exposure, this may cause acute hypoxia at low altitudes.

If aircrew are exposed to a pressure altitude of 11 887m (39 000ft) 100% oxygen can only maintain PAO_2 at 60-65mmHg (8.0-8.7kPa), approximately equivalent to breathing air at 3048m (10 000ft). Above a cabin pressure altitude of 12 192m (40 000ft) aircrew require positive pressure breathing for altitude protection (PBA) to supplement ambient pressure and maintain airway pressure to prevent significant hypoxia. This is analogous to continuous positive airway pressure used in clinical practice albeit with a very different function. PBA increases with altitude to 30mmHg (40.8cmH_2O) at 15 240m (50 000ft) providing absolute airway pressure of 117mmHg (15.6kPa) and PAO_2 45-50mmHg (6.0-6.7kPa) [77]. Above this altitude, chest counter pressure is required to prevent pulmonary barotrauma, and PBA

of 70mmHg (95.2cmH_2O) at 18 288m (60 000ft) can provide an absolute airway pressure of 124mmHg (16.5kPa) and PAO_2 55mmHg (7.3kPa) [77]. This degree of hypoxia allows sufficient time to commence an emergency descent to below 12 192m (40 000ft). PBA systems sometimes employ the concomitant use of inflatable G-trousers to prevent syncope at high breathing pressures.

Summary

The four key challenges of acute exposure to altitude are hypoxia (and hyperventilation), gas volume changes, decompression sickness, and cold. In aviation, the principal means of protection against altitude are a combination of pressurised cabins and oxygen delivery systems, which introduce further challenges of their own. Understanding the challenges of acute altitude exposure is fundamental to the development of aircrew protective systems to maintain performance, prevent accidents and reduce morbidity and mortality.

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