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SECTION 10 Environmental medicine, occupational medicine, and poisoning 1656 18 months imprisonment. Although the Health and Safety at Work Act 1974 has remained unchanged in this time, the new guidelines represent the most significant legal impact in the last 40 years. The changing world of work An ageing working population, equality and diversity, and flex- ible working (sometimes referred to as the “gig economy” in which work is undertaken without a formal contract of employment guar- anteeing a “normal” working week—often resulting in employers treating the relevant workers as if they were self-employed and re- sponsible for making all the choices about the work that they do) are some features of the rapidly changing world of work. Over half of the British workforce is now employed in smaller organizations, and over 90% of businesses employ fewer than 10 people. Part-time working has grown, and women now constitute half the workforce. Globalization has intensified competitive pressures, particularly on manufacturers. Public tolerance of accidents is very low and so there is pressure to make further improvements in occupational safety. In the United Kingdom, there will soon be more people over 65 than under 18 and many older people will continue to work past what was regarded as normal retirement age. Above all, we may be entering a new period of work shaped by Artifical Intelligence, robotics and other technological aspects. A number of studies have recently been undertaken exploring the impact of these issues on health and safety at work. Advice and assistance Those likely to be affected deserve to be properly protected against risks to their health and safety at work. Employers have a duty to protect their employees by sensible risk management. Workers also have a duty to protect their own and others’ health and safety. Sensible risk management requires effective systems to control those risks that arise frequently and have serious consequences. Balancing benefits and risks often requires expert help. The Management of Health and Safety at Work Regulations re- quire employers to appoint ‘ . . . one or more competent persons . . . ’ to assist them in meeting their duty of controlling risks. Employers and managers are in the best position to understand the health and safety issues in their business. Coupled with the knowledge of employees, this is often enough to ensure that risks are properly controlled, especially where the hazards are those commonly en- countered at work and methods for their control are already estab- lished practice. However, if the risks are complex or large numbers of employees are

involved, expert help may be needed. Employers can rely on one or more of their employees to give them competent help, provided the employees have been given enough time, training, and access to information. The employer could: • train or develop the necessary skills in an existing employee • recruit someone with the necessary skills • make use of consultancy support staff Formally qualified health and safety practitioners can work with a team of risk managers, including occupational health advisers. In the United Kingdom, the Institution of Occupational Safety and Health sets standards and awards qualifications. Preventing accidents at work makes an important contribution to the health and well-being of all who may be affected by an en- terprise, but achieving this aim requires a systematic approach and leadership. FURTHER READING Bird FE, Germain GL (1966). Damage control (a new horizon in ac- cident prevention and cost improvement). American Management Associations, New York, NY. British Safety Council and Robertson Cooper (2018). Future risk: The impact of work on health, safety and well-being. London. Eves D, Gummer J (2005). Questioning performance: the director's essential guide to health, safety and environment. UK Institution of Occupational Safety and Health (IOSH), Wigston, Leicestershire. Frick K, et al. (eds) (2000). Systematic occupational health and safety management—perspectives on an international development. Pergamon, Oxford. Health and Safety Executive (2013). Managing for health and safety, 3rd edition. HSG65. http://www.hse.gov.uk/pubns/books/hsg65.htm Health and Safety Executive and Institute of Directors (2013). Leading health and safety at work, INDG417(rev1) 06/13. http://www.hse. gov.uk/pubns/indg417.pdf Reason J (1997). Managing the risk of organizational accidents. Ashgate, Aldershot. Sentencing Council (2015). Health and safety offences, corporate man- slaughter and food safety and hygiene offences: definitive guideline. https://www.sentencingcouncil.org.uk/wp-content/uploads/HS- offences-definitive-guideline-FINAL-web.pdf Woolf AD (1973). Robens Report—the wrong approach? Industrial Law J, 2(1), 88. 10.2.3 Aviation medicine Michael Bagshaw ESSENTIALS Travel by air is a safe means of transport, but puts people at various physiological risks and is a potential means of spreading infectious disease. Physiological risks associated with flying include hypoxia— atmospheric pressure falls with altitude. The minimum cabin pres- sure in commercial passenger aircraft (565 mm Hg, 75.1 kPa) brings a healthy individual's arterial P along the plateau of the oxyhaemo- globin dissociation curve until just at the top of the steep part, but does not cause desaturation. By contrast, people with respiratory dis- ease and a low arterial oxygen pressure may desaturate, which can be overcome by administering 30% oxygen, this being equivalent to breathing air at ground level. Guidance for assessing a passenger's fitness to fly is provided by the websites of the Aerospace Medical Association and the British Thoracic Society. A second physiological risk is increased exposure to cosmic radiation, although there is no evidence that this leads to abnormality or disease. Other medical problems associated with flying include (1) venous thromboembolism—the relative risk is significant, but the absolute risk is very low. Medical practitioners need to be circumspect in advising preventative measures, taking account of the efficacy and risk profile of any intervention, but compression stockings and/or a single prophylactic dose of low molecular weight heparin may be re- commended in high-risk cases. (2) Jet lag—there is no simple solution

 for combating the effects of jet lag: the individual must evolve strat- egies to suit their particular needs. There is no evidence of a causative association between the use of engine bleed air for pressurization and ill health of aircraft occupants. Transmission of disease—there is no evidence that the pressurized aircraft cabin itself encourages transmission of disease, and recircu- lation of cabin air is not a risk factor for contracting symptoms of upper respiratory tract infection. It is important that individuals with a febrile illness should not travel on

commercial aircraft. Restricting air travel will not prevent global spread of pandemic influenza, but might delay the spread sufficiently to allow countries time to prepare. Introduction To answer practical questions about the effects of flight on the body, it is necessary to understand the physics and physiology of flight, the discipline of aviation medicine. Aerospace medicine is very much a specialized discipline, with a history traced back to the descriptions of altered physiology during balloon ascent by Glaisher and Coxwell in 1862. Aviation medicine concerns the well-being of humans in flight within the Earth's atmosphere, whereas space medicine concerns the welfare of humans flying beyond the atmosphere and the Earth's gravitational pull. Space medicine addresses the problems asso- ciated with very prolonged flight times and life support within a self-contained environment, as well as weightlessness, exposure to high doses of cosmic radiation, and the psychological aspects of pro- longed spaceflight. Those seeking information on the specific effects of space flight are referred to the specialized texts in the 'Further reading' section at the end of this chapter. Physics of the flight environment The Earth's atmosphere is an oxygen-rich gas shielding the ground below from solar radiation above. Subjected to gravity, compressed under its own weight, the atmosphere is denser close to the ground than further away. Long waves of infrared light penetrate it easily but heat the ground below. Heated ground reradiates some of this heat at shorter wavelengths which are absorbed by carbon dioxide and water vapour, making the air close to the surface much warmer than that higher up. Short waves of ultraviolet sunlight, absorbed by oxygen molecules early in their journey, create a belt of ozone at high altitudes. Some rays intercepted in the same region generate secondary rays that extend lower down. Very few reach the ground. At sea level, the atmosphere exerts a pressure of about 760 mm Hg (101 kPa); it is variably moist, has a temperature that ranges from – 60°C to +60 °C, and moves at wind speeds from 0 to 160 km/h. With increasing altitude, the temperature, pressure, and water content of the atmosphere fall and wind speeds increase (Fig. 10.2.3.1). Atmospheric pressure Total gas pressure falls with altitude in a regular manner, halving every 18 000 ft (5500 m) (Fig. 10.2.3.2). The oxygen content of the atmosphere (20.93%) is constant to very high altitudes, so the same curve can be used to obtain the ambient oxygen pressure by rescaling the ordinate (Fig. 10.2.3.2). The oxygen pressure of physiological importance is that which exists in ambient air when it is warmed and wetted on entering the bronchial tree. This raises water vapour pres- sure to about 47 mm Hg (6.3 kPa) regardless of the total gas pressure outside. The oxygen pressure in moist inspired gas (PiO_2) fully satur- ated with water vapour at 37 °C is given by the relationship: $PiO = FiO\ Patm\ 47\ 2\ 2 - (\ )$ O_3 concentration (p.p.m.) 0 10 Temperature (°C) −60 0 40 Pressure (atm) 0 1 150 100 50 0 Heat lets molecules escape Some rays are absorbed Infrared UV Cosmic Gravity pulls atmosphere down Cosmic Altitude (ft × 103) Fig. 10.2.3.1 Some physical features of the Earth's atmosphere, showing the variations in barometric pressure, air temperature, and ozone concentration with altitude. (NB: There is an international aviation safety convention that all altitudes are given in feet.) The shaded diagram on the left illustrates how the Earth's atmosphere is compressed under its own weight. The atmosphere absorbs much solar radiation.

 where FiO_2, the fractional concentration of oxygen in the inspirate, is 0.2093 and Patm = atmospheric pressure. Atmospheric temperature The atmospheric temperature reduces at 1.98 °C/1000 ft (300 m) from the standard sea level temperature of 15°C, to the tropo- pause (40 000 ft (12 200 m)). It remains stable at –56 °C up to about 80 000 ft (24 400 m) and then rises to almost body temperature at about 150 000 ft (46 000 m), but by then air density is so low that its temperature is unimportant. Atmospheric ozone Atmospheric ozone is formed by ultraviolet irradiation of diatomic oxygen

molecules which dissociate into atoms. At very high alti- tudes, all oxygen exists in the monatomic form. Lower down, some of this monatomic oxygen combines with oxygen molecules to form the triatomic gas ozone, with concentrations up to 10 parts per mil- lion (ppm). The ozonosphere normally exists between 40 000 and 140 000 ft (12 200 and 42 700 m). Below 40 000 ft (12 200 m), the irradiation is normally too weak for significant amounts of ozone to form. Concentrations of 1 ppm at sea level can cause lung irritation. Modern passenger jet aircraft are fitted with catalytic converters in the environmental control system, which break down the ozone before it enters the pressurized cabin. Cosmic radiation Aircraft occupants are exposed to elevated levels of cosmic radiation of galactic and solar origin. The sun has a varying magnetic field, which reverses direction approximately every 11 years. Near the reversal, at 'solar min- imum', there are few sunspots and the sun's magnetic field extending throughout the solar system is relatively weak. At solar maximum, there are many sunspots and other manifestations of magnetic turbulence. The Earth's magnetic field has a larger effect than the sun's mag- netic field on cosmic radiation approaching the atmosphere. The protective effect is greatest at the equator and least at the magnetic poles. At jet aircraft operating altitudes, galactic cosmic radiation is 2.5–5 times more intense in polar regions than near the equator. The Earth's surface is shielded from cosmic radiation by the at- mosphere, the ambient radiation decreasing with altitude by ap- proximately 15% for each increase of around 2000 ft (dependent on latitude). Protection against effects of cosmic radiation The International Commission on Radiological Protection (ICRP) recommended in 1991 that exposure of flight crew members to cosmic radiation in jet aircraft should be considered part of occupa- tional exposure to ionizing radiation. The ICRP limits for occupational exposure are a 5-year average effective dose of 20 millisieverts (mSv) per year, with no more than 50 mSv in a single year. The annual limit for the general public is 1 mSv. Cosmic radiation doses The effect of ionizing radiation depends not only on the dose ab- sorbed, but also on the type and energy of the radiation and the tis- sues involved. These factors are taken into account in deriving the dose equivalent measured in Sieverts (Sv). However, doses of cosmic radiation are so low that figures are usually quoted in microsieverts (μSv) or millisieverts (mSv). Calculated and measured doses are well within the ICRP recommended limits. Health risks of cosmic radiation While it is known that there is no level of ionizing radiation ex- posure below which effects do not occur, current epidemiological evidence indicates that the probability of airline crew members or passengers suffering any abnormality or disease as a result of ex- posure to cosmic radiation is very low. Physiology of flight The physiological effects of flight are distinguished from those of terrestrial high altitude because exposures are relatively rapid, brief, and not cumulative. Flyers do not adapt to the hypoxic environment, unlike inhabitants of terrestrial high altitudes. However, the aircraft can be a means of transporting an individual to a high-altitude destination. Hypoxia Oxygen has a dual role in most animal cells, being simultaneously life-giving and extremely poisonous. In air, or dissolved in simple solution, it is benign and only ionized with difficulty. However, once an electron is successfully attached to an oxygen molecule it becomes a highly corrosive superoxide ion, forming a cascade of other very destructive oxygen radicals. This is an essential feature of oxygen toxicity, which is discussed in Chapter 10.2.4. Superoxide dismutase and various peroxidases have evolved to protect most cells from the effects of spontaneous formation of oxygen radicals by quenching the ions as rapidly as they appear. Other enzymes have evolved which harness this property in a controlled way. There are three types: oxidases, oxygenases, and hydroxylases. Quantitatively, cytochrome a3 oxidase is the most important because, using oxygen as the ultimate electron sink, it allows many metabolic processes to proceed at the same time un- locking and trapping most of the energy the body needs (oxidative phosphorylation). Oxygenases

introduce an oxygen molecule into organic molecules creating new compounds. Although these enzymes consume only a small fraction of the body’s total oxygen requirement, they are particularly important for production and dismemberment of many critical compounds such as the amine transmitters of the brain. PB Pressure (atm) 1.0 0.8 0.6 0.4 0.2 0.00 20 40 60 80 0 20 40 60 80 PO2 0.20 0.15 0.10 0.05 0.0 Altitude (ft × 103) Fig. 10.2.3.2 The variations of barometric pressure (PB) and ambient oxygen pressure (PO2) with altitude.

10.2.3 Aviation medicine 1659 Hydroxylases insert one atom of oxygen and another of hydrogen into organic molecules. They too are responsible for many critical metabolic processes and for the denaturation of many drugs in the liver, kidney, and elsewhere. These enzymes differ in their affinity for oxygen, described by the Michaelis constant (for oxygen). This constant (KmO2) is that partial pressure of oxygen which, when all other factors are equal, allows an oxygen-consuming reaction to proceed at half its maximum vel- ocity. The major oxidase (cytochrome a3), which is the cocatalyst of oxidative phosphorylation, has a very high oxygen affinity and thus a very low KmO2, of 1 mm Hg or less. Thus, this particular type of oxygen consumption, representing 80–90% of the whole, can proceed at high rate down to very low levels of oxygen supply. By contrast (Fig. 10.2.3.3), the other enzymes, which are quantitatively less important but qualitatively critical, have Michaelis constants for oxygen that vary from 5 to 250 mm Hg (0.7–33.3 kPa). A fall in oxygen supply will influence these processes long before oxidative phosphorylation is affected and at times when overall oxygen con- sumption is diminished little if at all. When humans are exposed to hypoxia, systemic and intracel- lular changes operate together to minimize hypoxic injury and restore adequate oxygenation. Emerging evidence indicates that the hypoxia-inducible factor (HIF) family of transcription factors plays a central regulatory role in these homeostatic changes at both the systemic and cellular levels. HIF was discovered through its action as the transcriptional activator of erythropoietin, and has sub- sequently been found to control intracellular hypoxic responses throughout the body. HIF is primarily regulated by specific prolyl hydroxylase-domain enzymes (PHDs) that initiate its degradation via the von Hippel-Lindau tumour suppressor protein (VHL). The oxygen and iron dependency of PHD activity accounts for regula- tion of the pathway by both cellular oxygen and iron status. Recent studies conducted in patients with rare genetic diseases have begun to uncover the wider importance of the PHD-VHL-HIF axis in systems-level human biology. These studies indicate that, in addition to regulating erythropoiesis, the system plays an important role in cardiopulmonary regulation. Although Fig. 10.2.3.2 describes how ambient oxygen pressure is related to altitude, it does not convey the pressure of oxygen to be found in the lungs. That pressure is determined by two equations (Fig. 10.2.3.4). The alveolar ventilation equation states that alveolar CO2 pressure (PaCO2) depends only on CO2 excretion (CO2) and alveolar ventilation (Va), so: PaCO = k CO /Va . 2 2 ( ) The alveolar air equation states that since at any one time there is a fixed trading ratio between oxygen uptake and CO2 excretion (R = CO2/O2), alveolar oxygen pressure (PaO2) can be calcu- lated from the moist inspired oxygen pressure (PiO2*) and alveolar PCO2, so: PaO = PiO * PaCO /R 2 2 2 −( ) Progressive hypoxia leads to a mild hyperventilation (i.e. a rise in Va and fall in PaCO2). Thus, it is possible to plot alveolar oxygen pressure against altitude (Fig. 10.2.3.5a). When arterialized blood leaves a healthy lung the oxygen pres- sure is some 10 mm Hg less than that in the alveoli, due to uneven The Michaelis–Menten equation when the substrate is oxygen: MO2/MO2 max = PO2/(PO2 + KmO2) Cytochrome a3 oxidase Other oxidases and oxygenases 1 5 25 100 250 KmO2 PO2 (mmHg) 1.0 0.8 0.6 0.4 0.2 0.0 1.2 MO2/MO2 max 0 40 80 120 160 Fig. 10.2.3.3 Curves of oxygen uptake (O2) as a fraction of the theoretical maximum (O2max) against the partial pressure of oxygen (PO2) for a

family of oxygen-handling enzymes with Michaelis constants for oxygen (KmO2) from 1 to 250 mm Hg. PACO2 ∝ MCO2/VA Time PAO2=PIO2 – PaCO2/R Partial pressure The Alveolar air equation pictures VA trapped in a bag, and notes there must be a link between the rise in PCO2 and the fall in PO2, so that, for most practical purposes: The Alveolar ventilation equation ignores dead-space, and supposes there is a stream of oxygen-rich CO2 free gas, VA and says, for practical purposes: VA Plus the CO2 output, MCO2 Minus the almost equal O2 uptake, MO2 VA Fall ∝ MO2 Rise ∝ MCO2 MO2 MCO2 Fig. 10.2.3.4 Graphical representations of the alveolar ventilation and alveolar air equations. PO2 (mmHg) (a) Inspired air Alveolar gas Arterial blood (b) Blood PO2 (mmHg) 0 0 1 0 Oxygen saturation (%) Whole-blood O2-Hb dissociation a. v 0 150 100 50 0 150 100 50 0 Altitude (ft × 103) a-vΔ MO2/Q 30 20 10 Fig. 10.2.3.5 (a) Variations in moist inspired, alveolar, and arterial oxygen pressure (PO2) with altitude in normal men. (b) The conventional oxygen–haemoglobin dissociation curve of whole blood plotted to the same pressure scale as the left-hand graph, so that arterial O2 content can be read directly (at the same horizontal level as the PO2 curve). It also emphasizes that the arteriovenous oxygen content difference (a–vΔ) is proportional to the ratio of oxygen uptake (MO2) to local blood flow (Q).

SECTION 10 Environmental medicine, occupational medicine, and poisoning 1660 matching of ventilation to perfusion, some anatomical shunting, and an almost nominal obstacle to diffusion. In resting people, the alveolar–arterial oxygen gradient does not change much with alti- tude, although the relative importance of the factors contributing to it alter considerably; so subtracting a further 10–15 mm Hg de- scribes the relation between arterial oxygen pressure and altitude (Fig. 10.2.3.5). The most important change is the loss of pressure driving oxygen from the alveoli to blood, as the fall in alveolar PO2 is much greater than that in mixed venous PO2 (because of the shape of the oxygen dissociation curve). As a result, the alveolar–venous gradient for oxygen diffusion is smaller and equilibration slower than at ground level. People ascend to altitude in a matter of minutes, rather than over several days, and adapt to hypoxia by an increase in blood flow and a modest hyperventilation, limiting the effects of hypoxia. The effects are shown in Fig. 10.2.3.6. Individuals abruptly exposed to altitudes of 10 000 ft (3000 m) and above suffer mental and physical effects, and is the ceiling above which aviators are provided with oxygen. To allow a margin of safety, the maximum certified cabin altitude in civilian pas- senger aircraft is 8000 ft (2440 m), at which barometric pressure is 565 mm Hg and arterial oxygen pressure is around 55 mm Hg (see Fig 10.2.3.5b, the oxyhaemoglobin dissociation curve), and venous oxygen pressures have only fallen by 1–2 mm Hg. Even at this altitude, there is a decrease in performance. The latest gener- ation of passenger aircraft are manufactured from newer materials which provide greater strength from a given mass, thus allowing a higher differential cabin pressure with a lower cabin altitude. Two physiological features of altitude hypoxia are important in aviation. The first is the total lack of awareness of cerebral impair- ment. The second is the time of useful consciousness, describing how rapidly consciousness is lost thus dictating how quickly the condition must be recognized and corrective action taken. The time of useful consciousness is the interval after the onset of hypoxia during which an individual can carry out some purposeful activity. The general relation between this time interval and the alti- tude of sudden exposure is shown in Fig. 10.2.3.7a. It diminishes from about 4 min at 25 000 ft (7620 m) to a minimum of roughly 15 s, which is reached at 35 000 (10 700 m) to 40 000 ft (12 200 m). This asymptote represents the sum of the 7 s or so required for blood to travel from the lungs to the brain and the time needed for the brain to utilize the oxygen already dissolved in its substance. In trained and healthy individuals breathing normally (i.e. with an alveolar PCO2 of 35–40 mm Hg (4.7–5.3 kPa)),

the dose of hypoxia acceptable before loss of useful consciousness is equivalent on a curve of alveolar PO2 against time, to an area of 150 mm Hg/s, where PO2 is less than 38 mm Hg (5.1 kPa) (Fig. 10.2.3.7b). However, this is sensitive to many other factors, such as the degree of hyperventilation and the acceleration to which the individual is exposed at the time. Hyperventilation causes cerebral vasoconstriction, and positive headwards accel- eration (+Gz) opposes the upward flow of blood to the brain. Exertion quickens loss of consciousness, because blood transits quickly through the lungs leaving insufficient time for oxygen equilibration. The minimum cabin pressure of 565 mm Hg (75.1 kPA) (8000 ft (2440 m)) in commercial passenger aircraft, will bring a healthy individual's arterial PO2 along the plateau of the oxyhaemoglobin dissociation curve until just at the top of the steep part (Fig. 10.2.3.5), still saturated. At ground level, people with respiratory disease may have arterial oxygen pressures as low as 55–60 mm Hg (7.3–8 kPa). As they ascend to 8000 ft (2440 m) their arterial PO2 will fall further. If their hypoxaemia at ground level is due to a mismatch of ventilation to perfusion, as is usually the case, the drop in arterial PO2 will not be as extensive as in healthy people (about 40 mm Hg or 5.3 kPa), but if it is due to diffusion defect associated with desaturation on exertion, as in some fibrotic con- ditions, it may be greater. However, in either event, it can be re- versed completely by the administration of oxygen, 30% oxygen at 8000 ft (2440 m) being equivalent to breathing air at ground level. Given prior notice, most airlines can provide a personal oxygen supply for any passenger, although there may be a charge. (The altitudes of the patient's destination and transit points en route should also be considered.) Oxygen equipment and pressure cabins Aircraft operating below 10 000 ft (3000 m) do not require oxygen equipment. Many sophisticated light aircraft which can cruise above 10 000 ft do not have pressurized cabins, so oxygen equipment must be provided. Other aircraft that fly higher usually have reinforced cabins capable of holding a high-differential pressure between inside and Detectable losses in learning and night vision 0.20 0.15 0.10 0.05 0.00 Altitude (ft × 103) Must breathe pure oxygen at positive pressure to survive beyond here PO2 (atm) Degeneration in already-learnt tasks Physical weakness Coma on exertion Coma within minutes at rest Death 0 80 60 40 20 Fig. 10.2.3.6 A summary of the functional consequences of altitude hypoxia. Time (min) (a) Average Normal range Time of useful consciousness (b) Alveolar PO2 (mmHg) Time (s) Consciousness is lost once this area exceeds about 150 mm Hg/s 24 8 6 4 2 0 0 120 80 60 40 20 0 Altitude (ft × 103) 30 28 26 Fig. 10.2.3.7 (a) Variations in the time of useful consciousness with altitude. (b) One way of expressing the dose of hypoxia needed to bring about loss of consciousness.

 out. These are the high-differential type, seen in passenger and transport aircraft generally, and the low-differential variety found in military high-performance aircraft. The former, holding a high transmural pressure, maintain cabin pressure above 565 mm Hg (8000 ft (2440 m)). They provide an environment in which the occupants breathe cabin air. However, it is possible that the pres- surization system can fail, allowing the cabin pressure to fall to the external ambient value. This can be limited by descent below 10 000 ft (3000 m), subject to air traffic control and terrain con- straints. An emergency oxygen supply is available for passengers and crew. The aircraft's environmental control system automatically man- ages the internal cabin environment, providing healthy and com- fortable surroundings for all occupants. There are regulatory requirements for minimum cabin air pressure, maximum levels of carbon monoxide, carbon dioxide and ozone, and minimum ven- tilation flow rates. The cabin air must also be free from harmful or hazardous concentrations of gases or vapours. The cabin air supply is bled from the outside air entering the air- craft engine, or may be supplied from the outside air via

electrically driven compressors. It is then passed through the air-conditioning packs and mixed with filtered recirculated air before distribution to the cabin. The system provides approximately 20 cubic feet (566 litres) of air per minute per passenger, of which about 50% is re- circulated air (compared with up to 80% recirculated in buildings and other forms of public transport), giving a complete cabin air exchange every 2–3 minutes. These high ventilatory flow rates maintain normal pressurization, as well as temperature control and the removal of odours and carbon dioxide. The high flow rates also ensure that the volume of oxygen far exceeds the requirements of the aircraft occupants (0.34 litre/min at rest and 0.85 litre/min when walking). The air is distributed to the cabin via overhead ducts and grills running the length of the cabin. The airflow circulates around the cabin rather than along the cabin and is continuously extracted through vents at floor level as shown in Fig. 10.2.3.8. The recirculated air is passed through high efficiency particu- late air filters of the same specification used in hospital operating theatres, giving 99.99% efficiency in the removal of physical contam- inants such as microbial particles. Aircraft cabin air has been dem- onstrated to be bacteriologically cleaner than the air in buildings, trains, or buses. Although clean, the aircraft cabin air remains dry. During the flight, moisture is derived from the metabolism and activities of the cabin occupants as well as from the galleys and washrooms, giving a maximum relative humidity in the order of 10–20%. These levels are associated with surface drying of skin, mucous membranes, and cornea which may cause discomfort. Normal homeostatic mechanisms prevent dehydration and no harm to health has been demonstrated. A high-differential cabin limits the aircraft’s range and manoeuv- rability and increases the risk of catastrophic damage if the fuselage is punctured. So, military high-performance aircraft are fitted with low-differential cabins, which prevent cabin pressure falling below 280 mm Hg (37.2 kPA) (equivalent to a pressure altitude of 25 000 ft (7620 m)). At this level decompression illness becomes a poten- tial hazard (see next). In such aircraft, oxygen equipment is used routinely. Mechanical effects of pressure change In civilian passenger and transport aircraft the climb to cruise alti- tude takes about 30 min and involves a maximum fall of about 200 mm Hg (26.6 kPA) in cabin pressure (to the equivalent of 8000 ft (2440 m)). Descent to land takes much the same time. Body fluids and tissues generally are virtually incompressible and do not alter shape to any important extent when such pressures changes are applied. The same is true of cavities such as the lungs, gut, middle ear, and facial sinuses that contain air, provided that they can vent easily. Gas-containing spaces that cannot vent easily be- have differently. The thoraco-abdominal wall can develop transmural pressures of +100 mm Hg or so briefly, but is normally flaccid and has a trans- mural pressure of a few millimetres of mercury. Gas within will usually be at a pressure very close to that outside, and must follow Boyle’s law. Ascent from ground level (760 mm Hg) to 8000 ft (2440 m) (565 mm Hg or 75.3 kPa) will expand a given volume of trapped gas in a completely pliable container by about 35%. This may cause slightly uncomfortable gut distension in healthy people, but it is not an important problem. Even very diseased lungs can vent themselves over a minute or so. In consequence, the risk of lung rupture in normal flight is extremely rare (Fig. 10.2.3.9). Cabin air flow Fig. 10.2.3.8 Cabin air circulation and distribution. Each tube has an expiratory resistance, R Each balloon has a capacity, C The chest wall is very floppy The time-constant of emptying of any balloon is proportional to the product, RC. Maximum balloon volume Zero Transmural pressure (mm Hg) 0 0 1 0 Normal breathing Approximate range of bursting pressures C Fig. 10.2.3.9 A graphical summary of the factors determining lung rupture.

 The cavity of the middle ear vents easily, but sometimes fails to fill because the lower part of the Eustachian tube

behaves as a non- return valve, especially when it is inflamed. As a result, the cavity equilibrates quite easily on ascent but does not refill on descent, and the eardrum bows inwards, causing pain that can be severe (otic barotrauma). Altitude-induced decompression illness If ambient pressure falls quickly to less than half its original value, the gas dissolved in blood and tissue fluids may come out of solu- tion precipitously, forming bubbles and obstructing flow in small blood vessels. The time symptoms take to develop varies widely be- tween individuals and shortens markedly as the altitude of exposure rises. A guide to these times and variability is given in Fig. 10.2.3.10. Symptoms usually resolve quickly after a descent of a few thou- sand feet and rarely persist after descent to ground level, breathing oxygen. Should they persist, treatment should be along the lines de- tailed in Chapter 10.2.4. Atmospheric pressure halves at 18 000 ft and decompression illness occurs rarely, if at all, below this altitude. It is very rare below 25 000 ft (7600 m) and therefore is normally of no concern at normal passenger aircraft cabin altitudes, although the risk continues to be significant in some military flights. However, it does occasionally occur in those passengers who have been exposed to a hyperbaric environment prior to flight, such as divers and tunnel workers. Sub- aqua divers (q.v.) are advised to allow a minimum of 12 hours to elapse between diving and flight, or 24 hours if the dive required decompression stops. Clinical aspects of aviation medicine Travel by air is a safe means of transport. However, from the physio- logical point of view, flying is a means of putting people at risk as well as being a potential means of spreading infectious disease. Modern technology, coupled with stringent training requirements for flight crew, minimizes these risks but clinicians need to be aware of the ap- plications of physics and physiology to the flight environment. It can be difficult to apply epidemiological principles when con- sidering incidence and outcomes of medical conditions acquired during flight or the spread of infectious disease, because the passen- gers disperse after the flight before clinical symptoms or signs have become manifest. However, organizations such as the Aerospace Medical Association, the European Civil Aviation Conference and the World Health Organization have supported or undertaken epidemiological studies to establish the prevalence of conditions such as flight-related deep venous thrombosis (DVT) and venous thromboembolism (VTE), spread of tuberculosis (TB), and spread of newly emerging infectious diseases such as severe acute respira- tory syndrome (SARS) and avian flu. Jet lag Besides sleep, the major influence on waking performance and alert- ness is the internal circadian clock. Circadian rhythms fluctuate on a regular cycle, which lasts something over 24 hours. The circadian rhythms are controlled by the suprachiasmatic nucleus of the hypo- thalamus. Many body functions have their own circadian rhythm and they are synchronized to a 24-hour pattern by ‘zeitgebers’ (time givers), light being among the most powerful. Moving to a new light/dark schedule (as in time zone changes) leads to a discrepancy between internal suprachiasmatic nucleus timing and external environmental cues. The internal clock can take days or weeks to readjust, depending on the number of time zones crossed (desynchronosis). Fatigue is defined as the likelihood of falling asleep. Therefore, in practical terms, there is little difference between chronic fatigue and acute tiredness. Fatigue can be caused by sleep loss and circadian desynchronosis, but it can also result from low motivation and low levels of external stimulation. Preventative measures Sleep scheduling: • At home the best possible sleep should be obtained before a trip; • On a trip, as much sleep per 24 hours should be obtained as would be at home; • Feelings should be trusted—if the individual feels sleepy and cir- cumstances permit, then they should sleep. Good sleep habits: • A regular presleep routine should be developed; • Sleep time should not be reduced; • The individual should avoid going to bed hungry, but should not eat or drink heavily before going to bed; • Alcohol or caffeine should be avoided before bedtime. Caffeine consumption may be used to increase alertness. A cup of coffee usually

takes between about 15 and 30 minutes to become ef- fective, and the effect lasts for between 3 and 4 hours. However this is less effective for individuals who regularly drink large amounts of caffeine-containing beverages. Bright light (more than 2500 lux), used at the appropriate time in the circadian cycle, can help to reset the circadian clock. After flying east, the traveller should be exposed to evening light, but morning light avoided. Conversely, when travelling west, morning light should be sought, and evening light avoided. This makes the best use of the natural zeitgebers in resetting the body clock. Incidence (%) Frequency of decompression sickness at the end of 2 h exposure Rest Exertion Probable threshold 20 100 50 0 Altitude (ft × 103) 40 30
Fig. 10.2.3.10 The incidence of decompression sickness (percentage) at the end of 2 hours of exposure to various altitudes in men at rest, or exerting themselves.

10.2.3 Aviation medicine 1663 Temazepam is a short-acting benzodiazepine with a short half- life. Many people find this drug helpful in promoting sleep and if used for two or three days after travel, can assist in resetting the sleep cycle. Melatonin is secreted by the pineal gland with a rhythm linked to the light/dark cycle through the suprachiasmatic nucleus. It is ef- fective in inducing sleep when taken at the appropriate stage in the circadian cycle. However, if taken at the wrong stage, it can disrupt the sleep/wake cycle and destabilize sleep patterns. This limits its usefulness in treating jet lag. There is no simple or single solution for combating the effects of jet lag. The individual has to evolve the strategies to suit his or her particular needs. Traveller’s thrombosis (DVT/VTE) Long haul travel is associated with prolonged periods of immobility, a recognized risk factor for DVT first described by Virchow in 1856. However, there have been concerns as to whether there are other factors specific to air travel which further increase the risk. In the general population DVT occurs in 1–3 per 1000 people per year, of which 20% give rise to pulmonary embolism. Increasing age is known to be a strong risk factor, possibly due to decreased mo- bility and reduced muscular tone. The pathogenesis of thrombosis still relies on the basic premise of Virchow who identified circulatory stasis, hypocoagulability, and endothelial injury as the risk factors. Several clinical studies have shown an association between air travel and the risk of DVT, with the risk of VTE in travellers increasing with the distance travelled. A recent case–control study showed that all modes of travel increased the risk of venous throm- bosis about twofold, with an absolute risk of one thrombosis per 6000 journeys. It has been found that combinations of risk factors synergistically increase the risk of thrombosis. In people with factor V Leiden, the risk of thrombosis after flying was about 14 times increased and in women using oral contraceptives, it was around 20-fold increased. It has also been shown that the risk rises with the number of flights taken in a short time-frame, as well as with the duration of the flight. Most of these clots are asymptomatic and disperse naturally. Thus, even though the overall risk of venous thrombosis after air travel is only moderately increased, clear subgroups can be identi- fied in whom the risk is higher. The low humidity of the aircraft cabin does not in itself lead to de- hydration. Excessive alcohol consumption may cause dehydration, but there is no evidence that this is a significant risk factor leading to DVT. Two studies of reduced oxygen partial pressure with nonhypoxic control groups found no evidence of coagulation. There is no evi- dence that hypoxia or the hypobaric environment of an aircraft cabin is a significant risk factor for the development of DVT. Although there is good evidence for the value of aspirin in preventing arterial thromboembolic disease, its role in the preven- tion of venous thromboembolic disease is much less clear. The side effect profile is significant. There is no evidence to support the use of aspirin in preventing the development of DVT during flight. For those travellers at medium to high risk of DVT, there is evi- dence that the use of compression stockings appears to substantially lower the risk of asymptomatic DVT, but it remains unclear as to whether this reduction is clinically significant. One study has shown that for

20–40% of travellers, the com- mercially available stockings do not fit adequately. It is essential for stockings to be correctly fitted so as to provide adequate compres- sion to stimulate venous return. Although the use of low molecular weight heparin for the preven- tion of DVT in the aviation setting is not supported by direct evi- dence, in a high-risk traveller consideration may be given to a single prophylactic dose prior to flying. While the relative risk of developing venous thrombosis when flying is significant, the absolute risk of developing symptomatic DVT is very low. The absolute risk of developing a pulmonary em- bolus during or after a flight between the United Kingdom and the east coast of the United States has been calculated as less than one in a million. Medical practitioners need to be circumspect in advising any pre- ventative measures, taking careful account of efficacy and risk pro- file of the preventative method. Passenger fitness to fly Medical clearance is required when: • fitness to travel is in doubt as a result of recent illness, hospitaliza- tion, injury, surgery or instability of an acute or chronic medical condition; • special services are required (e.g. oxygen, stretcher, or authority to carry or use accompanying medical equipment, such as a venti- lator or a nebulizer). Medical clearance is not required for carriage of an invalid passenger outside these categories, although special needs (such as a wheel- chair) must be reported to the airline at the time of booking. It is vital that passengers remember to carry with them any essen- tial medication, and not pack it in their checked baggage. Deterioration on holiday or on a business trip of a previously stable condition, or an accident, can often give rise to the need for medical clearance for the return journey. A stretcher may be re- quired, together with medical support, and this can incur consid- erable cost. It is important for all travellers to have adequate travel insurance. Assessment criteria The passenger’s exercise tolerance can provide a useful guide on fitness to fly; if unable to walk a distance greater than about 50 m without developing dyspnoea, there is a risk that the passenger will be unable to tolerate the relative hypoxia of the pressurized cabin. A good source of guidance is provided by the web sites of the Aerospace Medical Association and the British Thoracic Society. Spread of infectious disease There is no evidence that the pressurized cabin itself makes trans- mission of disease any more likely, and it has been shown that re- circulation of cabin air is not a risk factor for contracting symptoms of upper respiratory tract infection. Data suggest that risk of dis- ease transmission to susceptible passengers, by person-to-person droplet spread within the aircraft cabin, is associated with sitting within two rows of a contagious passenger for a flight time of more than 8 hours.

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