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164 • ENVIRONMENTAL MEDICINE representing 1 J/kg. To take account of different types of radiation and variations in the sensitivity of various tissues, weighting factors are used to produce a unit of effective dose, measured in sieverts (Sv). This value reflects the absorbed dose weighted for the damaging effects of a particular form of radiation and is most valuable in evaluating the long-term effects of exposure. ‘Background radiation’ refers to our exposure to naturally occurring radioactivity (e.g. radon gas and cosmic radiation). This produces an average annual individual dose of approximately 2.6 mSv per year, although this figure varies according to local geology. Effects of radiation exposure Effects on the individual are classified as either deterministic or stochastic. Deterministic effects Deterministic (threshold) effects occur with increasing severity as the dose of radiation rises above a threshold level. Tissues with actively dividing cells, such as bone marrow and gastrointestinal mucosa, are particularly sensitive to ionising radiation. Lymphocyte depletion is the most sensitive marker of bone marrow injury, and after exposure to a fatal dose, marrow aplasia is a common cause of death. However, gastrointestinal mucosal toxicity may cause earlier death due to profound diarrhoea, vomiting, dehydration and sepsis. The gonads are highly radiosensitive and radiation may result in temporary or permanent sterility. Eye exposure can lead to cataracts and the skin is susceptible to radiation burns. Irradiation of the lung may induce acute inflammatory reactions or pulmonary fibrosis, and irradiation of the central nervous system may cause permanent neurological deficit. Bone necrosis and lymphatic fibrosis are characteristic following regional irradiation, particularly for breast cancer. The thyroid gland is not inherently sensitive but its ability to concentrate iodine makes it susceptible to damage after exposure to relatively low doses of radioactive iodine isotopes, such as those released from Chernobyl. Stochastic effects Stochastic (chance) effects occur with increasing probability as the dose of radiation increases. Carcinogenesis represents a stochastic effect. With acute exposures, leukaemias may arise after an interval of around 2–5 years and solid tumours after an Environmental medicine deals with the interactions between the environment and human health. While previously concerned mainly with controlling infectious diseases, the focus at present is predominantly on the multiple physical, chemical, biological and social factors that pose risks to human health. As the environment continually alters, whether through population growth,

economic development or climate change, it plays an important role in disease causation – one that may increase over time. This chapter deals principally with acute effects of environmental hazards on individuals and should be read in conjunction with the chapters on Poisoning (Ch. 7) and Acute Medicine and Critical Illness (Ch. 10). Chapter 5 deals with more general effects of environmental factors on population health. Radiation exposure Radiation includes ionising (Fig. 9.1) and non-ionising radiations (ultraviolet (UV), visible light, laser, infrared and microwave). While global industrialisation and the generation of fluorocarbons have raised concerns about loss of the ozone layer, leading to an increased exposure to UV rays, and disasters such as the Chernobyl and Fukushima nuclear power station explosions have demonstrated the harm of ionising radiation, it is important to remember that it can be harnessed for medical benefit. Ionising radiation is used in X-rays, computed tomography (CT), radionuclide scans and radiotherapy, and non-ionising UV for therapy in skin diseases and laser therapy for diabetic retinopathy. Types of ionising radiation These include charged subatomic alpha and beta particles, uncharged neutrons or high-energy electromagnetic radiations such as X-rays and gamma rays. When they interact with atoms, energy is released and the resulting ionisation can lead to molecular damage. The clinical effects of different forms of radiation depend on their range in air and tissue penetration (Fig. 9.1). Dosage and exposure The dose of radiation is based on the energy absorbed by a unit mass of tissue and is measured in grays (Gy), with 1 Gy Fig. 9.1 Properties of different ionising radiations. Paper Range in air Range in tissue Few centimetres No penetration Few metres Few millimetres Kilometres Passes through Kilometres Passes through Alpha particles e.g. radium, uranium Beta particles e.g. 14carbon, 90strontium X-rays/gamma rays e.g. 131iodine, 99mtechnetium Neutrons Aluminium foil Lead/concrete Protection

In a cold environment, protective mechanisms include cutaneous vasoconstriction and shivering; however, any muscle activity that involves movement may promote heat loss by increasing convective loss from the skin, and respiratory heat loss by stimulating ventilation. In a hot environment, sweating is the main mechanism for increasing heat loss. This usually occurs when the ambient temperature rises above 32.5°C or during exercise. Hypothermia Hypothermia exists when the body’s normal thermal regulatory mechanisms are unable to maintain heat in a cold environment and core temperature falls below 35°C (Fig. 9.2). Moderate hypothermia occurs below 32°C and severe hypothermia below 28°C. Other systems define hypothermia on the basis of symptoms rather than absolute temperature. While infants are susceptible to hypothermia because of their poor thermoregulation and high body surface area to weight ratio, it is the elderly who are at highest risk (Box 9.2). Hypothyroidism is often a contributory factor in old age, while interval of about 10–20 years. Thereafter the incidence rises with time. An individual’s risk of developing cancer depends on the dose received, the time to accumulate the total dose and the interval following exposure. Management of radiation exposure Exposed people should be removed from ongoing exposure (‘get inside, stay inside’), and should take off affected clothing and shower to stop further contamination. If contamination of food and water supplies may have occurred, only bottled water and food in sealed containers should be consumed. The principal problems after large-dose exposures are maintenance of adequate hydration, control of sepsis and management of marrow aplasia. Associated injuries such as thermal burns need specialist management within 48 hours of active resuscitation. Internal exposure to radioisotopes should be treated with chelating agents (such as Prussian blue used to chelate 137caesium after ingestion). White-cell

colony stimulation and haematopoietic stem cell transplantation may need to be considered for marrow aplasia. Extremes of temperature Thermoregulation Body heat is generated by basal metabolic activity and muscle movement, and lost by conduction (which is more effective in water than in air), convection, evaporation and radiation (most important at lower temperatures when other mechanisms conserve heat) (Box 9.1). Body temperature is controlled in the hypothalamus, which is directly sensitive to changes in core temperature and indirectly responds to temperature-sensitive neurons in the skin. The normal 'set-point' of core temperature is tightly regulated within the range 37 ± 0.5°C, which is necessary to preserve the normal function of many enzymes and other metabolic processes. The temperature set-point is increased in response to infection (p. 218). Fig. 9.2 Clinical features of abnormal core temperature. The hypothalamus normally maintains core temperature at 37°C, but this set-point is altered in, for example, fever (pyrexia, p. 218), and may be lost in hypothalamic disease (p. 679). In these circumstances, the clinical picture at a given core temperature may be different.

<37 <35 <32 <28

°C Definitions Clinical features Heat stroke Heat exhaustion Mild hypothermia Severe hypothermia Hot and not sweating Multiple organ failure, confusion, aggression, shock Hot and sweating Headache, weakness, fatigue, irritability, tachycardia, dehydration Tachycardia, vasoconstriction Cold and not shivering Cold, pale skin Depressed consciousness Muscle stiffness Bradycardia and hypotension Coma Dilated, unreactive pupils Cardiac standstill Cold and shivering 'Mumble, stumble, tumble' Lethargy Dehydration Tachypnoea Moderate hypothermia Violent shivering Slurred speech Slow, laboured movements Ataxia, mild confusion Pale with blue lips 9.1 Thermoregulation: responses to hot and cold environments Mechanism Hot environment Cold environment Heat production Basal metabolic rate → ↓ in hypothermia Muscle activity ↓ by lethargy ↑ by shivering ↓ in severe hypothermia Heat loss Conduction* ↑ by vasodilatation ↓ by vasoconstriction ↑↑ in water < 31°C Convection* ↑ by vasodilatation ↓ by lethargy ↓ by vasoconstriction ↑ by wind and movement Evaporation* ↑↑ by sweating ↓ by high humidity ↑ by hyperventilation Radiation ↑ by vasodilatation ↓ by vasoconstriction (but is the major heat loss in dry cold) *These losses are dependent on the relative ambient and skin temperatures.

 aminotransferase and creatine kinase may be elevated secondary to muscle damage and the serum amylase is often high due to subclinical pancreatitis. If the cause of hypothermia is not obvious, additional investigations for thyroid and pituitary–adrenal dysfunction (p. 633), hypoglycaemia (p. 725) and the possibility of drug intoxication (p. 134) should be performed. Management Following resuscitation, the objectives of management are to rewarm the patient in a controlled manner while treating associated hypoxia (by oxygenation and ventilation if necessary), fluid and electrolyte disturbance, and cardiovascular abnormalities, particularly arrhythmias. Careful handling is essential to avoid precipitating the latter. The method of rewarming is dependent not on the absolute core temperature, but on haemodynamic stability and the presence or absence of an effective cardiac output. Mild hypothermia Outdoors, continued heat loss is prevented by sheltering the patient from the cold, replacing wet clothing, covering the head and insulating him or her from the ground. Once in hospital, even in the presence of profound hypothermia, if there is an effective cardiac output then forced-air rewarming, heat packs placed in axillae and groins and around the abdomen, inhaled warmed air and correction of fluid and electrolyte disturbances are usually sufficient. Rewarming rates of 1–2°C per hour are effective in

leading to a gradual and safe return to physiological normality. Underlying conditions should be treated promptly (e.g. hypothyroidism with triiodothyronine 10 µg IV 3 times daily; p. 640). Severe hypothermia In the case of severe hypothermia (< 28°C), patients with cardiopulmonary arrest (non-perfusing rhythm), or those with cardiac instability (systolic blood pressure < 90 mmHg or ventricular arrhythmias), the aim is to restore perfusion, and rapid rewarming at a rate of > 2°C per hour is required. This is best achieved by cardiopulmonary bypass or extracorporeal membrane oxygenation (ECMO). If these are unavailable, then veno–veno haemofiltration, and pleural, peritoneal, thoracic or bladder lavage with warmed fluids are alternatives. Direct heat sources, such as hot water or heat pads, should be used with caution, as these can provoke cardiac arrhythmias or cause burns. Monitoring of cardiac rhythm and arterial blood gases, including H+ (pH), is essential. Significant acidosis may require correction (p. 364). Cold injury Freezing cold injury (frostbite) This represents the direct freezing of body tissues and usually affects the extremities: in particular, the fingers, toes, ears and face. Risk factors include smoking, peripheral vascular disease, dehydration and alcohol consumption. The tissues may become anaesthetised before freezing and, as a result, the injury often goes unrecognised at first. Frostbitten tissue is initially pale and doughy to the touch and insensitive to pain (Fig. 9.4). Once frozen, the tissue is hard. Rewarming should not occur until it can be achieved rapidly in a water bath. Give oxygen and aspirin 300 mg as soon as possible. Frostbitten extremities should be rewarmed in warm water at 37–39°C, with antiseptic added. Adequate analgesia is necessary, as rewarming is very painful. Vasodilators such as alcohol and other drugs (e.g. phenothiazines) commonly impede the thermoregulatory response in younger people. More rarely, hypothermia is secondary to hypothyroidism, glucocorticoid insufficiency, stroke, hepatic failure or hypoglycaemia. Hypothermia also occurs in healthy individuals whose thermoregulatory mechanisms are intact but insufficient to cope with the intensity of the thermal stress. Typical examples include immersion in cold water, when core temperature may fall rapidly (acute hypothermia), exposure to extreme climates such as during hill walking (subacute hypothermia), and slow-onset hypothermia, as develops in an immobilised older individual (subchronic hypothermia). This classification is important, as it determines the method of rewarming. Clinical features Diagnosis is dependent on recognition of the environmental circumstances and measurement of core (rectal) body temperature. Clinical features depend on the degree of hypothermia (Fig. 9.2). In a cold patient, it is very difficult to diagnose death reliably by clinical means. It has been suggested that, in extreme environmental conditions, irreversible hypothermia is probably present if there is asystole (no carotid pulse for 1 min), the chest and abdomen are rigid, the core temperature is < 13°C and serum potassium is > 12 mmol/L. However, in general, resuscitative measures should continue until the core temperature is normal and only then should a diagnosis of brain death be considered (p. 211). Investigations Blood gases, a full blood count, electrolytes, chest X-ray and electrocardiogram (ECG) are all essential investigations. Haemoconcentration and metabolic acidosis are common, and the ECG may show characteristic J waves, which occur at the junction of the QRS complex and the ST segment (Fig. 9.3). Cardiac arrhythmias, including ventricular fibrillation, may occur. Although the arterial oxygen tension may be normal when measured at room temperature, the arterial PO2 in the blood falls by 7% for each 1°C fall in core temperature. Serum aspartate Fig. 9.3 Electrocardiogram showing J waves (arrows) in a hypothermic patient. 9.2 Thermoregulation in old age • Age-associated changes: impairments in vasomotor function, skeletal muscle response and sweating mean that older people react more slowly to changes in temperature. • Increased comorbidity: thermoregulatory problems are more likely in the presence of pathology such as atherosclerosis and hypothyroidism, and medication such as sedatives and hypnotics. •

Hypothermia: this may arise as a primary event, but more commonly complicates other acute illness, e.g. pneumonia, stroke or fracture. • Ambient temperature: financial pressures and older equipment may result in inadequate heating during cold weather.

large extent by adequate replacement of salt and water, although excessive water intake alone should be avoided because of the risk of dilutional hyponatraemia (p. 357). A spectrum of illnesses occurs in the heat (see Fig. 9.2). The cause is usually obvious but the differential diagnosis should be considered (Box 9.3). Heat cramps These painful muscle contractions occur following vigorous exercise and profuse sweating in hot weather. There is no elevation of core temperature. The mechanism is considered to be extracellular sodium depletion as a result of persistent sweating, exacerbated by replacement of water but not salt. Symptoms usually respond rapidly to rehydration with oral rehydration salts or intravenous saline. Heat syncope This is similar to a vasovagal faint (p. 181) and is related to peripheral vasodilatation in hot weather. Heat exhaustion Heat exhaustion occurs with prolonged exertion in hot and humid weather, profuse sweating and inadequate salt and water replacement. There is an elevation in core (rectal) temperature to between 37°C and 40°C, leading to the clinical features shown in Figure 9.2. Blood analyses may show evidence of dehydration with mild elevation of the blood urea, sodium and haematocrit. Treatment involves removal of the patient from the heat, and active evaporative cooling using tepid sprays and fanning (‘strip–spray–fan’). Fluid losses are replaced with either oral rehydration mixtures or intravenous isotonic saline. Up to 5 L positive fluid balance may be required in the first 24 hours. Untreated, heat exhaustion may progress to heat stroke. Heat stroke Heat stroke occurs when the core body temperature rises above 40°C and is a life-threatening condition. The symptoms of heat exhaustion progress to include headache, nausea and vomiting. Neurological manifestations include a coarse muscle tremor and confusion, aggression or loss of consciousness. The patient’s skin feels very hot, and sweating is often absent due to failure of thermoregulatory mechanisms. Complications include hypovolaemic shock, lactic acidosis, disseminated intravascular coagulation, rhabdomyolysis, hepatic and renal failure, and pulmonary and cerebral oedema. Duration of hyperthermia is key to the outcome and immediate cooling should begin at the scene, before transfer to hospital. The aim should be to reduce core temperature by > 0.2°C per minute to approximately 39°C, while avoiding overshooting and hypothermia. The patient should be resuscitated with evaporative or convective cooling. Fluid resuscitation with crystalloid intravenous fluids should be instituted but solutions containing potassium pentoxifylline (a phosphodiesterase inhibitor) have been shown to improve tissue survival. Once it has thawed, the injured part must not be re-exposed to the cold, and should be dressed and rested. While wound débridement may be necessary, amputations should be delayed for 60–90 days, as good recovery may occur over an extended period. As yet, the role of hyperbaric oxygen in the treatment of frostbite requires further research. Non-freezing cold injury (trench or immersion foot) This results from prolonged exposure to cold, damp conditions. The limb (usually the foot) appears cold, ischaemic and numb, but there is no freezing of the tissue. On rewarming, the limb appears mottled and thereafter becomes hyperaemic, swollen and painful. Recovery may take many months, during which period there may be chronic pain and sensitivity to cold. The pathology remains uncertain but probably involves endothelial injury. Gradual rewarming is associated with less pain than rapid rewarming. The pain and associated paraesthesia are difficult to control with conventional analgesia and may require amitriptyline (50 mg nocte), best instituted early. The

patient is at risk of further damage on subsequent exposure to the cold. Chilblains Chilblains are tender, red or purplish skin lesions that occur in the cold and wet. They are often seen in horse riders, cyclists and swimmers, and are more common in women than men. They are short-lived and, although painful, not usually serious. Heat-related illness When generation of heat exceeds the body’s capacity for heat loss, core temperature rises. Non-exertional heat illness (NEHI) occurs with a high environmental temperature in those with attenuated thermoregulatory control mechanisms: the elderly, the young, those with comorbidity or those taking drugs that affect thermoregulation (particularly phenothiazines, diuretics and alcohol). Exertional heat illness (EHI), on the other hand, typically develops in athletes when heat production exceeds the body’s ability to dissipate it. Acclimatisation mechanisms to environmental heat include stimulation of the sweat mechanism with increased sweat volume, reduced sweat sodium content and secondary hyperaldosteronism to maintain body sodium balance. The risk of heat-related illness falls as acclimatisation occurs. Heat illness can be prevented to a Fig. 9.4 Frostbite in a female Everest sherpa. 9.3 Differential diagnosis in patients with elevated core body temperature • Heat illness (heat exhaustion, heat stroke) • Sepsis, including meningitis • Malaria • Drug overdose • Serotonin syndrome (pp. 139 and 1199) • Malignant hyperpyrexia • Thyroid storm (p. 639)

168 • ENVIRONMENTAL MEDICINE on the shape of the sigmoid oxygen–haemoglobin dissociation curve (see Fig. 23.5, p. 917) and the ventilatory response. Acclimatisation to hypoxaemia at high altitude involves a shift in this dissociation curve (dependent on 2,3-diphosphoglycerate (2,3-DPG)), erythropoiesis, haemoconcentration, and hyperventilation resulting from hypoxic drive, which is then sustained despite hypocapnia by restoration of cerebrospinal fluid pH to normal in prolonged hypoxia. This process takes several days, so travellers need to plan accordingly. Illnesses at high altitude Ascent to altitudes up to 2500 m or travel in a pressurised aircraft cabin is harmless to healthy people. Above 2500 m high-altitude illnesses may occur in previously healthy people, and above 3500 m these become common. Sudden ascent to altitudes above 6000 m, as experienced by aviators, balloonists and astronauts, may result in decompression illness with the same clinical features as seen in divers (see below), or even loss of consciousness. However, most altitude illness occurs in travellers and mountaineers. Acute mountain sickness Acute mountain sickness (AMS) is a syndrome comprised principally of headache, together with fatigue, anorexia, nausea and vomiting, difficulty sleeping or dizziness. Ataxia and peripheral oedema may be present. The aetiology of AMS is not fully understood but it is thought that hypoxaemia increases cerebral blood flow and hence intracranial pressure. Symptoms occur within 6–12 hours of an ascent and vary in severity from trivial to completely incapacitating. The incidence in travellers to 3000 m may be 40–50%, depending on the rate of ascent. Treatment of mild cases consists of rest and simple analgesia; symptoms usually resolve after 1–3 days at a stable altitude, but may recur with further ascent. Occasionally, there is progression to cerebral oedema. Persistent symptoms indicate the need to descend but may respond to acetazolamide, a carbonic anhydrase inhibitor that induces a metabolic acidosis and stimulates ventilation; acetazolamide may also be used as prophylaxis if a rapid ascent is planned. High-altitude cerebral oedema The cardinal symptoms of high-altitude cerebral oedema (HACE) are ataxia and altered consciousness. HACE is rare, life-threatening and usually preceded by AMS. In addition to features of AMS, the patient suffers confusion, disorientation, visual disturbance, lethargy and ultimately loss of consciousness. Papilloedema and retinal haemorrhages are common and focal neurological signs may be found. Treatment is directed at improving oxygenation. Descent is essential and dexamethasone (8 mg immediately and 4 mg 4 times daily) should be given. If descent is impossible, oxygen therapy in a portable

pressurised bag may be helpful. High-altitude pulmonary oedema High-altitude pulmonary oedema (HAPE) is a life-threatening condition that usually occurs in the first 4 days after ascent above 2500 m. Unlike HACE, HAPE may occur de novo without the preceding signs of AMS. Presentation is with symptoms of dry cough, exertional dyspnoea and extreme fatigue. Later, the cough becomes wet and sputum may be blood-stained. Tachycardia and tachypnoea occur at rest and crepitations may often be heard in both lung fields. There may be profound hypoxaemia, pulmonary hypertension and radiological evidence should be avoided. Intravenous dextrose may be necessary, as hypoglycaemia can occur. Appropriate monitoring of fluid balance, including central venous pressure, is important, as overaggressive fluid replacement may precipitate pulmonary oedema or further metabolic disturbance. Investigations for complications include routine haematology and biochemistry, coagulation screen, hepatic transaminases (aspartate aminotransferase and alanine aminotransferase), creatine kinase and chest X-ray. Once emergency treatment is established, heat stroke patients are best managed in intensive care. Heat stroke is an emergency with a significant mortality. However, where temperatures can be reduced to < 40°C within 30 minutes of collapse, death rates can approach zero. Patients who have had core temperatures of > 40°C should be monitored carefully for later onset of rhabdomyolysis, renal damage and other complications before discharge from hospital. Clear advice to avoid heat and heavy exercise during recovery is important. High altitude The physiological effects of high altitude are significant. On Everest, the barometric pressure of the atmosphere falls from sea level by approximately 50% at base camp (5400 m) and approximately 70% at the summit (8848 m). The proportions of oxygen, nitrogen and carbon dioxide in air do not change with the fall in pressure but their partial pressure falls in proportion to barometric pressure (Fig. 9.5). Oxygen tension within the pulmonary alveoli is further reduced at altitude because the partial pressure of water vapour is related to body temperature and not barometric pressure, and so is proportionately greater at altitude, accounting for only 6% of barometric pressure at sea level but 19% at 8848 m. Physiological effects of high altitude Reduction in oxygen tension results in a fall in arterial oxygen saturation (Fig. 9.5). This varies widely between individuals, depending Fig. 9.5 Change in inspired oxygen tension and blood oxygen saturation at altitude. The blue curve shows changes in oxygen availability at altitude and the red curve shows the typical resultant changes in arterial oxygen saturation in a healthy person. Oxygen saturation varies between individuals according to the shape of the oxygen–haemoglobin dissociation curve and the ventilatory response to hypoxaemia. (To convert kPa to mmHg, multiply by 7.5.) Summit of Everest (8848 m) Highest permanent habitation (< 5200 m) Arterial oxygen saturation Partial pressure of inspired oxygen

Partial pressure of oxygen (kPa) Pressurised aircraft cabin (< 2400 m)

Arterial oxygen saturation (%)

Altitude above sea level (m)

arrhythmia, sickle-cell disease and ischaemic heart disease. Most airlines decline to carry pregnant women after the 36th week of gestation. In complicated pregnancies it may be advisable to avoid air travel at an earlier stage. Patients who have had recent abdominal surgery, including laparoscopy, should avoid flying until all intraperitoneal gas is reabsorbed. Divers should not fly for

24 hours after a dive requiring decompression stops. Ear and sinus pain due to changes in gas volume are common but usually mild, although patients with chronic sinusitis and otitis media may need specialist assessment. A healthy mobile tympanic membrane visualised during a Valsalva manœuvre usually suggests a patent Eustachian tube. On long-haul flights, patients with diabetes mellitus may need to adjust their insulin or oral hypoglycaemic dosing according to the timing of in-flight and subsequent meals (p. 750). Advice is available from Diabetes UK and other websites. Patients should be able to provide documentary evidence of the need to carry needles and insulin. Deep venous thrombosis Air travellers have an increased risk of venous thrombosis (p. 975), due to a combination of factors, including loss of venous emptying because of prolonged immobilisation (lack of muscular activity) and reduced barometric pressure on the tissues, together with haemoconcentration as a result of oedema and perhaps a degree of hypoxia-induced diuresis. Venous thrombosis can probably be prevented by avoiding dehydration and excess alcohol, and by exercising muscles during the flight. Without a clear cost–benefit analysis, prophylaxis with aspirin or heparin cannot be recommended routinely, but may be considered in high-risk cases. Under water Drowning and near-drowning Drowning is defined as death due to asphyxiation following immersion in a fluid, while near-drowning is defined as survival for longer than 24 hours after suffocation by immersion. Drowning remains a common cause of accidental death throughout the world and is particularly common in young children (Box 9.4). In about 10% of cases, no water enters the lungs and death follows intense laryngospasm (‘dry’ drowning). Prolonged immersion in cold water, with or without water inhalation, results in a rapid fall in core body temperature and hypothermia (p. 165). Following inhalation of water, there is a rapid onset of ventilation– perfusion imbalance with hypoxaemia, and the development of diffuse alveolar oedema. It is not known whether the alveolar oedema is a result of mechanical stress on the pulmonary capillaries associated with the high pulmonary arterial pressure, or an effect of hypoxia on capillary permeability. Reduced arterial oxygen saturation is not diagnostic but is a marker for disease progression. Treatment is directed at reversal of hypoxia with immediate descent and oxygen administration. Nifedipine (20 mg 4 times daily) should be given to reduce pulmonary arterial pressure, and oxygen therapy in a portable pressurised bag should be used if descent is delayed. Chronic mountain sickness (Monge’s disease) This occurs on prolonged exposure to altitude and has been reported in residents of Colorado, South America and Tibet. Patients present with headache, poor concentration and other signs of polycythaemia. The haemoglobin concentration is high (> 200 g/L) and the haematocrit raised (> 65%). Affected individuals are cyanosed and often have finger clubbing. High-altitude retinal haemorrhage This occurs in over 30% of trekkers at 5000 m. The haemorrhages are usually asymptomatic and resolve spontaneously. Visual defects can occur with haemorrhage involving the macula but there is no specific treatment. Venous thrombosis This has been reported at altitudes of > 6000 m. Risk factors include dehydration, inactivity and the cold. The use of the oral contraceptive pill at high altitude should be considered carefully, as this is an additional risk factor. Refractory cough A cough at high altitude is common and usually benign. It may be due to breathing dry, cold air and to increased mouth breathing, with consequent dry oral mucosa. This may be indistinguishable from the early signs of HAPE. Air travel Commercial aircraft usually cruise at 10 000–12 000 m, with the cabin pressurised to an equivalent of around 2400 m. At this altitude, the partial pressure of oxygen is 16 kPa (120 mmHg), leading to a PaO_2 in healthy people of 7.0–8.5 kPa (53–64 mmHg). Oxygen saturation is also reduced but to a lesser degree (see Fig. 9.5). Although well tolerated by healthy people, this degree of hypoxia may be dangerous in patients with respiratory disease. Advice for patients with respiratory disease The British Thoracic Society has published guidance on the management of patients with respiratory

disease who want to fly. Specialist pre-flight assessment is advised for all patients who have hypoxaemia (oxygen saturation < 95%) at sea level, and includes spirometry and a hypoxic challenge test with 15% oxygen (performed in hospital). Air travel may have to be avoided or undertaken only with inspired oxygen therapy during the flight. Asthmatic patients should be advised to carry their inhalers in their hand baggage. Following pneumothorax, flying should be avoided while air remains in the pleural cavity, but can be considered after proven resolution or definitive (surgical) treatment. Advice for other patients Other circumstances in which patients are more susceptible to hypoxia require individual assessment. These include cardiac 9.4 Most common causes of drowning by age Infants/young children • Domestic baths • Garden pools Adolescents • Swimming pools • Rivers, sea, etc. Adults • Water sports, boating, fishing • Occupational Older people • Domestic baths

170 • ENVIRONMENTAL MEDICINE Diving-related illness The underwater environment is extremely hostile. Other than drowning, most diving illness is related to changes in barometric pressure and its effect on gas behaviour. Ambient pressure under water increases by 101 kPa (1 atmosphere, 1 ata) for every 10 metres of seawater (msw) or 33 eet of seawater (fsw) depth. As divers descend, the partial pressures of the gases they are breathing increase (Box 9.5), and the blood and tissue concentrations of dissolved gases rise accordingly. Nitrogen is a weak anaesthetic agent, and if the inspiratory pressure of nitrogen is allowed to increase above 320 kPa (3.2 ata; i.e. a depth of approximately 30 msw), it produces ‘narcosis’, resulting in impairment of cognitive function and manual dexterity, not unlike alcohol intoxication. For this reason, compressed air can be used only for shallow diving. Oxygen is also toxic at inspired pressures above approximately 40 kPa (0.4 ata; inducing apprehension, muscle twitching, euphoria, sweating, tinnitus, nausea and vertigo), so 100% oxygen cannot be used as an alternative. For dives deeper than approximately 30 msw, mixtures of oxygen with nitrogen and/ or helium are used. While drowning remains the most common diving-related cause of death, another important group of disorders usually present once the diver returns to the surface: decompression illness (DCI) and barotrauma. Clinical features Decompression illness This includes decompression sickness (DCS) and arterial gas embolism (AGE). While the vast majority of symptoms of DCI present within 6 hours of a dive, they can also be provoked by flying (further decompression), and thus patients may present to medical services at sites far removed from the dive. Exposure of individuals to increased partial pressures of nitrogen results in additional nitrogen being dissolved in body tissues; the amount dissolved depends on the depth/pressure and on the duration of the dive. On ascent, the tissues become supersaturated with nitrogen, and this places the diver at risk of producing a critical quantity of gas (bubbles) in tissues if the ascent is too fast. The gas so formed may cause symptoms locally, peripherally due to bubbles passing through the pulmonary vascular bed (Box 9.6) or by embolisation elsewhere. Arterial embolisation may occur if the gas load in the venous system exceeds the lungs’ abilities to excrete nitrogen, or when bubbles pass through a patent foramen ovale (present asymptomatically of diffuse pulmonary oedema. Fresh water is hypotonic and, although rapidly absorbed across alveolar membranes, impairs surfactant function, which leads to alveolar collapse and rightto-left shunting of unoxygenated blood. Absorption of large amounts of hypotonic fluid can result in haemolysis. Salt water is hypertonic and inhalation provokes alveolar oedema, but the overall clinical effect is similar to that of freshwater drowning. Clinical features Those rescued alive (near-drowning) are often unconscious and not breathing. Hypoxaemia and metabolic acidosis are inevitable features. Acute lung injury usually resolves rapidly over 48– 72 hours, unless infection occurs (Fig. 9.6). Complications include dehydration, hypotension,

haemoptysis, rhabdomyolysis, renal failure and cardiac arrhythmias. A small number of patients, mainly the more severely ill, progress to develop the acute respiratory distress syndrome (ARDS; p. 198). Survival is possible after immersion for up to 30 minutes in very cold water, as the rapid development of hypothermia after immersion may be protective, particularly in children. Long-term outcome depends on the severity of the cerebral hypoxic injury and is predicted by the duration of immersion, delay in resuscitation, intensity of acidosis and the presence of cardiac arrest. Management Initial management requires cardiopulmonary resuscitation with administration of oxygen and maintenance of the circulation (p. 456). It is important to clear the airway of foreign bodies and protect the cervical spine. Continuous positive airways pressure (CPAP; p. 202) should be considered for spontaneously breathing patients with oxygen saturations of < 94%. Observation is required for a minimum of 24 hours. Prophylactic antibiotics are only required if exposure was to obviously contaminated water. Fig. 9.6 Near-drowning. Chest X-ray of a 39-year-old farmer, 2 weeks after immersion in a polluted freshwater ditch for 5 min before rescue. Airspace consolidation and cavities in the left lower lobe reflect secondary staphylococcal pneumonia and abscess formation. 9.5 Physics of breathing compressed air while diving in sea water Depth Lung volume Barometric pressure PiO2 PiN2 Surface 100% 101 kPa (1 ata) 21 kPa (0.21 ata) 79 kPa (0.78 ata) 10 m 50% 202 kPa (2 ata) 42 kPa (0.42 ata) 159 kPa (1.58 ata) 20 m 33% 303 kPa (3 ata) 63 kPa (0.63 ata) 239 kPa (2.34 ata) 30 m 25% 404 kPa (4 ata) 84 kPa (0.84 ata) 319 kPa (3.12 ata)

The majority of patients make a complete recovery with treatment, although a small but significant proportion are left with neurological disability. Humanitarian crisis Humanitarian crises are common. If the medical profession is to help, it must understand that the emergency treatment of a few sick and injured people is not always the priority and that there is a set of basic needs that must be addressed in order to do the most for the most. A humanitarian crisis can take many forms: an environmental disaster, mass emigration due to drought, conflict or famine, a disease outbreak or any number of natural or man-made events. When this event overwhelms the resources of the affected country’s government, then the international community will often step in to help. Although a full examination of the subject is beyond the scope of this book, there are a few basic principles for managing a humanitarian crisis. The response is broken down into four phases:

1. recognition (first week)
2. emergency response (first month)
3. consolidation phase (to crisis resolution or crisis containment)
4. handover and withdrawal. Recognition Recognition of a disaster may be obvious in a sudden event such as an earthquake or a tidal wave, but less obvious in a disease outbreak or when it stems from internal conflict. The recognition phase is for the host nation rather than the international community, and requests for support will come from the affected country’s government. Emergency response During the emergency phase, responders (whether international, governmental or the charity sector) will undertake an initial assessment of need, set objectives, mobilise resources, coordinate with other agencies and deploy to the crisis in order to deliver an initial response. Consolidation phase The consolidation phase involves matching resources to need, dealing with the crisis, building resilience, supporting infrastructure (human and physical) and instituting

systems to manage the ongoing health needs of the population. Handover and withdrawal Once the crisis is under control and the country is able to manage within resources, a phased withdrawal can occur. Health-care priorities When the normal infrastructure of a country or area fails, then populations are at risk, whether they shelter in place or flee, leading to mass migration. For health-care teams, rather than logisticians, rescue teams or security forces, there are welldefined priorities, which must be in place (Box 9.7). This must all occur during the emergency phase and is followed by the in 25–30% of adults; p. 531). Although DCS and AGE can be indistinguishable, their early treatment is the same. Barotrauma During the ascent phase of a dive, the gas in the diver's lungs expands due to the decreasing pressure. The diver must therefore ascend slowly and breathe regularly; if ascent is rapid or the diver holds his/her breath, the expanding gas may cause lung rupture (pulmonary barotrauma). This can result in pneumomediastinum, pneumothorax or AGE as a result of gas passing directly into the pulmonary venous system. Other air-filled body cavities may be subject to barotrauma, including the ear and sinuses. Management The patient is nursed horizontally and airway, breathing and circulation are assessed. Treatment includes the following: • High-flow oxygen is given by a tight-fitting mask using a rebreathing bag. This assists in the washout of excess inert gas (nitrogen) and may reduce the extent of local tissue hypoxia resulting from focal embolic injury. • Fluid replacement (oral or intravenous) corrects the intravascular fluid loss from endothelial bubble injury and dehydration associated with immersion. Maintenance of an adequate peripheral circulation is important for the excretion of excess dissolved gas. • Recompression is the definitive therapy. Transfer to a recompression facility may be by surface or air, provided that the altitude remains low (< 300 m) and the patient continues to breathe 100% oxygen. Recompression reduces the volume of gas within tissues (Boyle's law), forces nitrogen back into solution and is followed by slow decompression, allowing the nitrogen load to be excreted. *Information required by diving specialists to decide appropriate treatment. See contact details on page 172. 9.6 Assessment of a patient with decompression illness* Evolution • Progressive • Static • Relapsing • Spontaneously improving Manifestations • Pain: often large joints, e.g. shoulder ('the bends') • Neurological: any deficit is possible • Audiovestibular: vertigo, tinnitus, nystagmus; may mimic inner ear barotrauma • Pulmonary: chest pain, cough, haemoptysis, dyspnoea; may be due to arterial gas embolism (AGE) • Cutaneous: itching, erythematous rash • Lymphatic: tender lymph nodes, oedema • Constitutional: headache, fatigue, general malaise Dive profile • Depth • Type of gas used • Duration of dive

 Further information Websites altitude.org A website written by doctors with expertise and experience of expedition and altitude medicine and critical care. diversalertnetwork.org Advice on the clinical management of diving illness and emergency assistance services. emergency.cdc.gov/radiation/ The Centers for Disease Control and Prevention provides information and links on all forms of radiation for patients and professionals. msf.org Information and advice about all aspects of responding to humanitarian crises around the world. Telephone numbers Two organisations can offer national and international advice on diving emergencies and recompression facilities: • Within the UK, the National Diving Accident Helpline on +44 (0)7831 151523 (24 hours). • Outside the UK, contact the Divers Alert Network International Emergency Hotline +1-919-684-9111. implementation of a public health surveillance and reporting system and the mobilisation of local human resources, and their training and deployment during

the consolidation phase in order to prepare for handover to the local competent authority and withdrawal. 9.7 Priorities in a humanitarian crisis

1. Assessment of population need
2. Safe water and sanitation
3. Food and nutrition
4. Shelter and warmth
5. Emergency health care
6. Immunisation of risk groups
7. Control of communicable diseases
8. Ongoing health surveillance and reporting
9. Training and deployment of indigenous health-care workers
10. Handover of responsibility to local authorities

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