10.2.4 Diving medicine 1664

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SECTION 10 Environmental medicine, occupational medicine, and poisoning 1664 Newly emerging infectious disease SARS is an atypical pneumonia caused by a novel coronavirus which first appeared in the Far East in 2003. Thousands of flights took place to and from what the World Health Organization (WHO) defined as ‘affected areas’ during the outbreak, but transmission occurred only on five flights involving 29 secondary cases (24 cases on one flight). In addition, a further 40 flights were identified, on which one or more probable cases (i.e. symptomatic at the time of travel) travelled, but where no secondary cases developed. Thus, the risk of transmission on board an aircraft is thought to be low. Avian influenza (‘bird flu’) is a highly pathogenic strain A/H5N1 causing an epidemic among birds in Asia, Europe, and Africa. Human infection is very rare, but serious when it occurs. During 2006, WHO reported a total of 109 cases, of which 79 died. None of the reported cases occurred within Europe, and air travel is not thought to be a risk factor. On the other hand, pandemic influenza causes major morbidity and mortality in humans, with serious economic and social conse- quences. It usually affects a large proportion of the global population due to the absence of immunity, and spreads very rapidly throughout the world. Influenza pandemics occurred in 1918 (‘Spanish flu’), 1957 (‘Asian flu’), and in 1968 (‘Hong Kong flu’), all with high mortality. The WHO strategy for rapid containment of an emerging influ- enza pandemic aims to interrupt disease transmission by isolating and treating infectious individuals, treating and quarantining ex- posed people, and minimizing the exposure of uninfected persons. Modelling suggests that restricting air travel will not prevent the global spread of pandemic influenza, but might delay the spread suf- ficiently to allow countries time to prepare. Guidelines can be ac- cessed from http://www.who.org or http://www.cdc.gov. It is important that individuals should not travel on commercial aircraft with a febrile illness. Future issues Aerospace medicine is a subject that is largely understood. There is con- cern among some flight crew about health effects due to oil pyrolysis products in the cabin air. Evidence is conflicting and research is ongoing. The major peer-reviewed journal in the field is Aerospace Medicine and Human Performance (formerly Aviation, Space and Environmental Medicine), published by the Aerospace Medical Association, which is worth reading to keep up to date with aviation medicine’s evolution and innovation. FURTHER READING Bagshaw M (2014). Air contamination health effects. Aerospace Medical Association, Alexandria VA, USA. http://www.asma.org/ asma/media/asma/Travel-Publications/Air-contamination-health- effects-report-v2-7-Apr2014.pdf Campbell RD, Bagshaw M (2002). Human performance and limita- tions in aviation, 3rd edition. Blackwell Science, Oxford. Coker RK (ed) (2004). Managing passengers with respiratory disease planning air travel: British Thoracic Society recommendations. British Thoracic Society Standards of Care Committee, London. DeHart RL, Millet KC, Murphy J (eds) (1996). Fundamentals of aero- space medicine. Williams and Wilkins,

Philadelphia, PA. House of Lords Inquiry (2000). Air travel and health. Her Majesty’s Stationery Office, London. House of Lords Inquiry (2007). Air travel and health: an update. The Stationery Office Ltd, London. Kuipers S, et al. (2007). The absolute risk of venous thrombosis after air travel: a cohort study. PLoS Med, 4, 1508–14. Gradwell DP, Rainford DJ (eds) (2016). Ernsting’s aviation medicine, 5th edition. CRC Press, London. Rosenberg CA, Pak F (1997). Emergencies in the air: problems, man- agement and prevention. J Emerg Med, 15, 159–64. Thibeault C (1997). Special committee report: cabin air quality. Aviation, Space and Environmental Medicine, 68, 80–2. Zuckerman JN (ed) (2013). Principles and practice of travel medicine, 2nd edition. Wiley-Blackwell, Oxford. 10.2.4 Diving medicine David M. Denison and Mark A. Glover ESSENTIALS Diving remains the principal means of exploring and exploiting shal- lower underwater zones. Immersion and rapid change in pressure with depth cause most problems unique to diving. Effects of pressure on gases and ventilation Gas density, partial pressures, and solubility vary proportionately with ambient pressure. At elevated partial pressure, nitrogen be- comes narcotic, as can other inert gases, and contaminants barely detectable at the surface can become toxic. Hyperoxia irritates the lungs and the central nervous system, sometimes causing gener- alized seizures. A safe gas mixture at depth can become hypoxic as the partial pressure of oxygen decreases during the return to surface. Ventilation is compromised at depth and failure of CO_2 elimin- ation increasingly limits activity. Some divers are not distressed by elevated CO_2, but this does not protect them from its toxic effects. Clinical problems associated with diving and fitness to dive Immersion hazards include aquatic flora and fauna, water move- ment, impaired visibility and thermal control, and enhanced sound and blast propagation. Immersion predisposes susceptible individ- uals to pulmonary oedema. Aspiration of seawater can cause pul- monary inflammation and systemic manifestations. Water entering the external auditory meati can induce disabling caloric vertigo. The final common pathway in many diving-related fatalities is drowning. Decompression illness—is caused by ascent from a dive which re- duces ambient pressure. This releases excess dissolved inert gas from tissues, often in the form of bubbles. Alternatively, gas in the airways can expand, rupture the lung, and force its way into the systemic cir- culation via the pulmonary veins. Typical symptoms caused by these bubbles include rash, limb pain and neurological deficit (often motor weakness, numbness and paraesthesiae, also disturbance of higher cerebral function which can impair the diver’s insight). Symptoms de- velop within a few minutes to 24 h of surfacing in most cases and can manifest before reaching the surface in arterial gas embolism arising

 from pulmonary rupture and in decompression from deep, very long duration dives. Management requires exclusion of other diagnoses without delaying first aid treatment of decompression illness with oxygen (as close to 100% as possible) and rehydration, followed by definitive recompression. Intracardiac right–left shunts, such as pa- tent foramen ovale, predispose to the condition. Extracardiac (pul- monary) shunts can also permit a similar paradoxical embolization of bubbles. Barotrauma—gas-filled spaces within, or surrounding, the body will be damaged unless they are flexible enough to accommodate pressure-mediated changes in volume, or they are ventilated to pre- vent distortion. Divers’ ears, sinuses, lungs, carious teeth, or their masks and suits are vulnerable. Long-term consequences of diving—these include aseptic bone in- farcts, impaired higher cerebral function, and hearing loss. Fitness to dive—unrestricted diving demands a high level of phys- ical and medical fitness. Potential disqualifying factors include con- ditions that might incapacitate, impair, injure, or distract a diver; predispose to decompression illness or barotraumas; or mimic de- compression illness. Introduction Divers are exposed to many hazards while remote from medical care. As a result, diving medicine

is largely concerned with preven- tion. It requires a thorough understanding of the diver's environ- ment and work. Some dives are conducted in dry pressurized chambers, but most involve immersion in fluids such as seawater. Immersion and rapid change in pressure with depth are responsible for most diving prob- lems. Ambient pressure in seawater rises by approximately 100 kPa for every 10-m descent. Gas densities and partial pressures are pro- portional to ambient pressure. The amount of a chemically inert gas, such as nitrogen or helium that can dissolve in a diver's body is pro- portional to its partial pressure. A typical shore (Fig. 10.2.4.1) slopes down to between 200 and 300 m at a gradient of about 1 in 50. Diving is largely confined to this continental shelf. Thereafter, the continental slope descends to be- tween 3 and 6 km at a gradient of roughly 1 in 15 to vast flat expanses of soft mud, the abyssal plains, interrupted by occasional mountains and chasms. The deepest point is just over 11 km below the surface. Currents, arising from differences in water temperature and sal- inity, course across the abyssal plains and well up the continental slopes as mineral-rich streams supplying plant life in sunlit upper zones. Animals concentrate here to feed on these plants or on each other. Eighty per cent (80%) of the ocean biomass lies in the top 200 m, mainly close to the shore. Together, these sites form an area equal to Africa, infinitely more fertile and, as yet, virtually unfarmed. Limitations to diving Currents often exceed swimming speed (Fig. 10.2.4.2a) and may re- strict diving to an hour or two each day during slack water. High waves frequently prevent divers from being launched or recovered safely (Fig. 10.2.4.2b). Tidal currents tunnelled along marine Limit for breathing oxygen Salvage from HMS Edinburgh 1981 Probable limit to practical diving at ambient pressure Depth (km) (a) 0 12 100 Percentage of ocean area Continental shelves Continental slopes Abyssal plains Chasms (b) 0 0 Depth (m) 600 0 Percentage of ocean area Limit for air 10 Fig. 10.2.4.1 (a) A cumulative depth versus area plot of the oceans. (b) A similar plot of the top 600 m, including the continental shelves. Tidal current (knots) Usual Some Maximal swim Comfortable swim Tidal currents Borneo Cook Inlet Texas Louisiana North Sea Persian Gulf California Arabian Sea Incidence (%) 50 Texas Arabian Sea North Sea Month of the year Wave heights above 2 m J F M A M J J A D N O S 5 0)b ( )a(Fig. 10.2.4.2 (a) A plot of the usual and the not uncommonly seen tidal currents in eight diving sites around the world. (b) A plot of the percentage incidence of waves exceeding a height of 2 m at different times of the year in three of the diving sites.

 canyons, and springs of fresh water or falls of cold ocean water, can carry divers in unexpected directions without them being aware. Dawn arrives late and dusk comes early to the sea. Light halves in intensity with every 1 or 2 m of descent, and it is effectively 'night' below 80 m. Most recreational diving takes place in clear, shallow, and placid waters. Professional diving occurs throughout the year, alongside or beneath large obstructions, and in turbid waters where finding the task, let alone completing it, can be very demanding. Backscattering often makes artificial illumination ineffective. Underwater, binaural localization of sound is poor. Pressure waves travel almost five times as fast and many times more efficiently through water than air. This increases susceptibility to blast injury. Loss of air conduction raises auditory thresholds by 30–60 dB. Neoprene foam hoods raise thresholds by a further 30 dB or so. Only the surface waters of tropical seas are warm enough for indi- viduals to remain effective without insulation for any length of time (Fig. 10.2.4.3). Body temperature can be maintained at 37°C with minimal effort in air at 18–24°C, the zone of thermal neutrality. In water, this zone is high and narrow (35–35.5°C). Loss of tactile dis- crimination and manual dexterity are major problems when working in cold water. Exercise or excessive insulation in warm water rapidly leads to hyperthermia. Effects of simple immersion Water resists movement,

making most tasks more tiring and less efficient than on land (Fig. 10.2.4.4). A swimmer can sustain about 5 kgf thrust (c. 50 N), enough for propulsion at 1 to 2 knots (1.85–3.7 km/h). Full inspiration makes an adult swimmer about 2.5 kgf (c.25 N) positively buoyant, requiring half of maximum thrust to descend. Breathing out to residual volume results in about 2.5 kgf (25 N) negative buoyancy, requiring half of max- imum thrust to ascend. The neutrally buoyant diver can be poised at will but body weight can no longer be used to apply leverage or torque, or to stay in place against a current. Immersion opposes the effect of gravity and displaces blood from distensible vessels in dependent limbs. About 500 ml enters the chest, distending large veins, and right atrium. A larger volume of blood is displaced if immersion is in cold water, due to the per- ipheral vasoconstriction that results. Local stretch receptors in- terpret this central fluid shift as excess circulating volume and promote diuresis, resulting in hypovolaemia on emersion. Since gas is usually delivered to the mouth at ambient pressure, the pres- sure gradient across mouth, thorax, and upper abdomen can in- crease inspiratory or expiratory effort, depending on the diver's attitude in the water. The displaced blood increases cardiac preload. This predisposes susceptible individuals to 'immersion pulmonary oedema' which can occur despite normal cardiac function, and usually after a dive in cold water or involving strenuous exercise. Prevalence is esti- mated at approximately 1% of the recreational diving population and it can recur. In one study, a history of pulmonary oedema after diving in cold water was associated with elevated peripheral vas- cular resistance, especially after a cold challenge, and an increased risk of developing hypertension. An acute increase in preload and afterload is presumed to cause the oedema. Treatment of persistent mild symptoms is with supplemental inspired oxygen. Diuretics and vasodilators have been used to treat more severe cases. It typically resolves within hours although some fatalities have been reported. Physiological studies and case reports suggest that sildenafil has po- tential for prevention of immersion pulmonary oedema. Divers and swimmers with cardiac compromise are also at increased risk of pulmonary oedema. Aspiration of small amounts of seawater can cause 'saltwater as- piration syndrome', characterized by productive cough, retrosternal discomfort, and haemoptysis during, or within 2 h of, a dive. Fever, aches, malaise, and even impaired consciousness can develop. The casualty is usually normocapnic, often hypoxic, and sometimes has a leucocytosis. Treatment is rest and supplemental inspired oxygen. Warming often helps extrapulmonary symptoms. Most cases resolve spontaneously within 6 to 24 h. Ergometer load (kpm/min) Water Air Constant load 3 2 1 0 Pedal rate (r.p.m.) (b) Water Air Constant speed 3 2 1 0 (a) 0 40 80 120 0 300 600 M O2(l/min) M O2(l/min) Fig. 10.2.4.4 A comparison of oxygen consumption ($\dot{M}O_2$) when pedalling a cycle ergometer in air and under water, (a) at a constant speed (60 rev/min) and (b) at a constant light load. Note the high cost of moving the limbs through water. Most people's aerobic capacity is about 2.5 litres O2/min. Temperature (°C) Depth (m) 100 200 Polar summer Temperate Tropical Polar winter 0 300 −5 5 10 15 20 25 30 0 Fig. 10.2.4.3 Variations in sea temperature with site and depth. Note that water temperatures of less than 20°C are too cold for unclothed individuals to stay in for very long.

 If cool water enters one ear canal before the other, then a transient 'caloric vertigo' can result. Recurrent immersion can cause problems such as otitis externa.

Problems of descent The chest wall can maintain a pressure difference equivalent to 1 or 2 m of water, so gas within the body is virtually at the same pressure as the surrounding sea. The lung of a breath-hold diver is compressed from total lung capacity to residual volume at 30 m (400 kPa), so they will need half of their aerobic capacity to ascend. Variation in gas volume in clothing and equipment further complicates buoyancy control. Barotrauma of descent Barotrauma is the term

used to describe mechanical damage caused by changes in gas volume as pressure varies. Compression will force a diver's face into an unvented mask. The resulting facial oedema and subconjunctival haemorrhages usually resolve spontaneously. If gas is not added to a dry suit on descent, particularly if it is poorly tailored, it can pinch the skin resulting in linear wheals, commonly distributed around the neck, axillae, and groins. These require no active intervention but should not be con- fused with cutaneous signs of decompression illness (DCI). Severe suit squeeze can limit a diver's movements. Blood is drawn into the chest vessels to compensate for reduced lung volume, so lung injury occurs only at very great depths in breath-hold dives. When gas in obstructed sinuses is compressed, sinus walls become oedematous and may bleed. Epistaxis often oc- curs on ascent, as blood or clot is expelled by re-expanding gas. Middle-ear barotrauma is the most common problem in diving. Eustachian tube dysfunction or poor 'ear-clearing' technique pre- vents ventilation of the middle ear. Compression of the trapped gas draws the round and oval windows of the inner ear and the eardrum towards the middle ear space. Eardrum perforations can occur. They normally heal spontaneously, but persistent ruptures require sur- gery. Diving should be avoided until the drums have healed. Strenuous Valsalva-like efforts to ventilate the middle ear raise thoracic pressure. This transmits to the perilymph and can be suf- ficient to rupture the oval or, more typically, the round window. This is known as inner-ear barotrauma. Immediate or delayed ver- tigo, tinnitus, and hearing loss (usually at high frequencies) ensue. Management is bed rest with the head elevated, avoidance of raised intrathoracic pressure, and consultation with an ear, nose, and throat (ENT) surgeon who might elect to explore the middle ear and to re- pair the rupture surgically. If the symptoms appear after a dive, they can mimic vestibular DCI. If there is any doubt, a diving medicine specialist should be consulted. Barotrauma of descent can also affect a blocked external auditory meatus, gas spaces in carious teeth and under fillings or, in the event of loss of breathing gas pressure, the whole body. Problems while at increased pressure For prolonged dives, compressed gas is delivered to the diver at the same pressure as the surrounding water. This can be via a hose from the surface. A continuous flow through the helmet or face mask is easily engineered but is wasteful of gas. Most divers now use valves that provide gas only on demand. Self-contained underwater breathing apparatus (scuba) allows the diver to carry an on-demand supply of gas independent from the surface. Basic configurations of this equipment rarely last for more than 1 h. Rebreather equipment achieves greater endurance by replacing oxygen and removing CO_2 from exhaled gas so that it can be recirculated. Inert gas narcosis At raised partial pressure, nitrogen and several other inert gases with high solubility in lipids act like anaesthetics. Effects develop within minutes and reverse rapidly because they depend on passive solu- tion. Air is often breathed down to depths of 50 m, although sophis- ticated tests of cerebral function show impairment starting at 20 m. When deeper than 50 m, effects become more obvious. Narcosis is completely reversed on ascent. Using a less narcotic gas such as helium allows divers to reach the lowermost parts of the contin- ental shelves without narcosis. Divers can complete routine tasks while narcosed if they have repeatedly rehearsed them at increasing depths. Cognition and problem-solving, however, remain impaired. Hypercapnia Work of breathing and physiological dead space increase as gas be- comes denser at pressure. Hyperventilation is difficult at depth but can still occur. Breathing a dense gas mixture such as air at great depth will cause hypercapnia. Some divers hypoventilate involuntarily and become hypercapnic even in favourable conditions at depths as shallow as 30–40 m. Although these divers enjoy good gas economy, hypercapnia increases risk of inert gas narcosis, cerebral oxygen tox- icity, and DCI. Use of a less dense mixture, such as oxygen-in-helium, reduces this effect. Hypercapnia can also result from equipment mal- function, contaminated gas, or voluntary hypoventilation. Oxygen toxicity Oxygen toxicity is due to complex

biochemical interactions and takes time to develop and to reverse. There is a wide range of inter- and intraindividual sensitivity. Inspired oxygen partial pressure exceeding 50 kPa is toxic to the lungs. Irritation of lung endothelium and epithelium causes a spreading tracheobronchitis and reduction in lung volumes, flows, and gas transfer. Symptoms appear after about 6 h at partial pres- sures around 79–89 kPa and after 3 h at around 200 kPa. Advanced pulmonary changes can be irreversible, but symptoms typically di- minish rapidly in 2–4 h with complete recovery in 1–3 days. Lung function similarly recovers rapidly, although small decrements can persist for more than a week. Although pulmonary damage continues central nervous system toxicity becomes the primary limit to diving (Fig. 10.2.4.5) as in- spired partial pressure of oxygen rises further. This is unlikely to occur if the inspired partial pressure of oxygen does not exceed 200 kPa when at rest in a dry, comfortable environment but exercise, shivering, hypercapnia, anxiety, immersion, and pyrexia potentiate cerebral oxygen toxicity. As a result, inspired oxygen is usually maintained between maxima of 130 and 160 kPa when in water, depending on work levels. Manifestations of oxygen toxicity include visual disturbances, tinnitus, irritability, and dizziness. A general- ized seizure will usually follow if the oxygen partial pressure is not reduced promptly. Toxicity while immersed can be very dangerous

 but, in the safety of a hyperbaric chamber, partial pressures up to 300 kPa are used for maximum therapeutic effect. Multiple therapeutic hyperbaric exposures can cause myopic lens changes. They have also been reported in divers breathing oxygen for many hours. Most are reversible within 12 weeks of last exposure. High-pressure nervous syndrome Breathing oxygen-in-helium at depths in excess of 100 m causes tremor. Impaired higher function and level of consciousness and, in more severe cases, convulsions, occur at greater depths. It is pre- sumed that extreme pressures directly compress nerve components and affect their function. Depth and rate of pressurization influence the nature and incidence of symptoms. Slower compressions reduce both incidence and severity. Some habituation occurs during pro- longed exposure. Adding a small amount of narcotic gas such as ni- trogen to the breathing mixture reduces some of the manifestations but individual variation in side effects and optimum dose makes this technique unsuitable for commercial diving. Problems of ascent On ascent, partial pressures of gases fall as they expand, and less gas can remain in solution. Hypoxia of ascent Hypoxia will occur if breath is held for long enough in any circum- stance. Ordinarily, the rise in CO_2 stimulates the breath-hold diver to take a breath but predive hyperventilation will delay this, some- times to a dangerous extent. A frankly hypoxic mixture can be sup- plied by mistake during a compressed gas dive. In some rebreathers, a hard-working diver can consume oxygen faster than the design delivers it to the mixture (dilution hypoxia). Although the oxygen partial pressure might be sufficient to sustain consciousness at depth in any of these situations, ascent will cause it to fall further. Barotrauma of ascent Middle-ear barotrauma can occur on ascent as well as descent. The mechanism of injury is due to expansion of trapped gas. If one ear ventilates via its Eustachian Tube before the other, uneven vestibular stimulation can cause a transient ‘alternobaric vertigo’. If gas in sinuses cannot escape, it might eventually burst them. Rupture of the ethmoid sinus is rare but feared because of the risk of deep infec- tion. The gut is quite resilient, but in some cases, especially on rapid ascent, ruptures have occurred. Dental barotrauma is also reported and rapid ascents can fracture teeth. In a minority of individuals, the facial nerve or maxillary branch of the trigeminal nerve are exposed to pressure changes in the middle ear and maxillary sinus, respectively. If gas is not vented from the space on ascent, overpressure can impair the nerve’s blood supply, and hence cause a cranial nerve deficit a few

minutes after as- cent. This could be misdiagnosed as DCI. Release of pressure brings about resolution, which is usually within minutes and unlikely to exceed 2 h. Compressed gas diving allows the diver to fill the lungs at depth and, therefore, to burst them on ascent unless they are adequately vented. About 9.3 kPa of sustained overpressure (barely 1 m of sea- water) bursts a lung. Ascent at a controlled rate, breathing nor- mally or exhaling, allows the lungs, which have a time constant of emptying close to 0.3 s, to empty and minimizes the risk of rupture. Ascent at too rapid a rate, or while breath-holding, risks lung rupture. Central tears lead to mediastinal emphysema. Peripheral tears cause pneumothorax. Gas can embolize into the systemic cir- culation, the most significant targets being central nervous system and myocardium. Escaped gas expands as the ascent continues, making matters worse. The victim might lose consciousness or de- velop neurological deficit almost immediately. Otherwise dyspnoea, cough, haemoptysis, voice change, or discomfort in the throat or retrosternal region develop a few minutes later. There might be sur- gical emphysema of the neck and upper chest, increased cardiac dullness or crepitus, and/or evidence of a pneumothorax. Patients with neurological signs should be recompressed as soon as possible. In the meantime, first aid management is oxygen (as close to 100% as possible) and careful rehydration. Recompression will also reduce the volume of escaped gas if severe pneumomediastinum or subcutaneous emphysema threatens the airway. Decompression illness (DCI) More inert gas dissolves in tissues as dive depth or duration increases. On a safe ascent, this gas comes out of solution, often forming bub- bles in the venous circulation, slowly enough for it to diffuse out harmlessly via the lungs. Tissues are said to be ‘supersaturated’ until all of the excess gas is eliminated. If bubbles are too large or too nu- merous, they can block blood vessels, damage vascular endothelium, and induce ‘foreign body’ reactions. More severe decompression can generate extravascular bubbles within solid tissues, causing distor- tion and even rupturing cells. The term decompression sickness de- scribes disease caused by gas coming out of solution. DCI includes both decompression sickness and disease caused by bubbles escaped from a ruptured lung. The lungs can filter out moderate numbers of venous gas emboli before they reach the systemic circulation. It is possible that this ‘filter’ might be circumvented by right–left shunts such as patent for- amen ovale or pulmonary arteriovenous anastomosis. One in four healthy people has a patent foramen ovale, but many are only ‘probe- patent’ with little chance of shunting. Foramina exceeding 10 mm in maximum dimension are found in less than 1%. Over half of the Convulsions Time (h) PIO2 (atm) Reduction in vital capacity 0 10 20 30 5 4 3 2 1 0 Fig. 10.2.4.5 Commonly observed pulmonary and central nervous O2 toxicity versus time curves related to inspired Po2 (PIo2) (constructed from the data of many workers).

 victims of neurological DCI in one study had medium or large patent foramina. Some have questioned whether a patent foramen ovale is a primary cause of decompression illness and have proposed that, for instance, a significant pulmonary bubble load could trigger vasocon- striction, raise right heart pressures, and open a previously ‘closed’ foramen. Increasing intrathoracic pressure by heavy lifting and Valsalva-like manoeuvres to clear ears, could have a similar effect. It has been estimated that the odds ratio of serious decompres- sion sickness in divers with a patent foramen ovale is around 2:5. This estimate did not consider foramen ovale size and larger defects are likely to be associated with higher risk. Absolute risk of decom- pression sickness for the whole diving population is low, however, at a little over 2 per 10 000 dives, and primary screening for foramen ovale is not advocated. ‘Undeserved’ DCI justifies screening with bubble-contrast echocardiography. If a large foramen ovale is found, the usual approach is to advise less provocative diving or percutan- eous closure of the defect. Migraine with

aura is associated with an increased risk of patent foramen ovale and is accepted as an indica- tion for screening. The discovery of a large shunt in a diving candi- date who has not suffered from a diving-related illness would usually be considered to be a significant risk factor for DCI. After breathing an unchanging gas mixture for 24–48 h at a constant pressure, no more gas accumulates in tissues. Decompression from this ‘saturated’ state takes as long as several days, but it does not lengthen if dive duration is extended. This is the basis of saturation diving. A vast amount of experimental work has been done to determine the safe limits to ‘no-stop’ diving (Fig. 10.2.4.6) and the depth–time profiles that must be followed on returning to the surface after longer dives. DCI occurs in about 1% of dives conducted within ‘safe’ schedules, in some 2–3% of dives at the limits of these schedules, and in many badly conducted dives. Signs of arterial gas embolism following pul- monary rupture will usually present within the first 10 min after sur- facing; 50% of all DCI cases will develop symptoms within 1 h of surfacing and 90% within 6 h. The most common presentation in military and commercial diving is limb pain, commonly of the shoulders or elbows in divers, and of the knees and hips in tunnel workers. Pain might present a few minutes or as much as 24 h after a dive, often as a dull and poorly localized ache of gradual onset. It is not usually made worse by moving the joint, although weight bearing might make knee pain worse. Signs of inflammation are uncommon. Left untreated, the pain will regress and disappear over 2 or 3 days. Recompression commonly improves the pain quickly. Although recreational divers also experience limb pain, neuro- logical symptoms are more likely. Sensory disturbance is common, with numbness and paraesthesiae being frequent manifestations. One fulminant form starts with pain distributed along a thoracol- umbar dermatome (girdle pain) followed by loss of sensation and power in the lower limbs. Involvement of the brain is common and can be subtle. This can impair insight and delay a diver’s decision to seek assistance. Denial is also a frequent feature. Any of the higher functions can be in- volved, including loss of short-term memory, altered affect, visual disturbance, and loss of consciousness. Inner-ear DCI can be con- fused with inner-ear barotrauma. Bubbles do not necessarily respect normal anatomical boundaries, and patchy or multisystem presen- tations are common. Cardiopulmonary symptoms and signs are unusual but, if present, usually indicate a severe case. Cutaneous manifestations range from itching, sometimes with a papular rash, through to patches of skin ‘marbling’ characterized by a reticular cyanosis on a pallid back- ground with an erythematous periphery. Blockage of the lymphatic system by bubbles can cause tender nodes and oedema which typic- ally affects face, neck, or breast. It is not unusual to exhibit several manifestations, or for them to appear at different times and to evolve in different ways. Less specific constitutional symptoms, such as fatigue, malaise, headache, and anorexia can be difficult to distinguish from transient self-limiting illnesses, but they are usually of no concern unless other manifest- ations are present or they are severe enough to affect function. Divers developing any manifestation of DCI within 24 h of a dive should be managed as if they have the condition unless an alternative diagnosis is more likely. First aid management is supplemental in- spired oxygen (as close to 100% as possible) and rehydration. All but trivial cases of DCI should be recompressed as soon as possible; it is an effective treatment and reduces the size and promotes resorption of existing bubbles before irreversible infarction and oedema occur while preventing formation of new bubbles. High inspired partial pressures of oxygen facilitate removal of excess inert gas, relieve is- chaemia, and reduce oedema, inflammation, and reperfusion injury. The goal is as complete a resolution of symptoms as possible at depth and to avoid recurrence on surfacing. Relapse or residual symptoms require retreatment, so detailed postrecompression examination is necessary. DCI may fail to resolve completely. Miscellaneous related problems A diver who ascends more rapidly than the planned decompres- sion schedule has ‘omitted decompression’. Risk of DCI

is increased. Treatment is oxygen and, in more extreme cases, recompression. New exposure to an inert gas when saturated with another can increase overall gas burden if there is a mismatch in the rate at which the gases diffuse into and out of a tissue. This can cause a bubble-related disease, for which decompression is not the imme- diate provocation and is known as ‘isobaric counterdiffusion’. The site affected depends on the location of the interface between the Time (h) 10 60 50 40 30 20 Above the line: OK to make an ascent without decompression stops Below the line: must make an ascent with decompression stops Depth (m) 0 0 1 2 3 4 5 6 Fig. 10.2.4.6 The ‘no-stop’ diving curve that determines whether a dive has been shallow and brief enough for the diver to make an ascent to the surface without decompression stops.

 two gases. It can cause inner-ear or skin symptoms in saturation divers. Progression can be halted by altering the gas mixture. It can be treated by recompression and prevented by increasing environ- mental pressure slightly in order to reduce super-saturation of the tissue before changing gas mixtures. Several hours after breathing high fractions of oxygen, a diver can develop an exudate in the middle ear, yet remain able to ventilate the ears. This might be due to consumption of oxygen causing an insidious volume reduction, or due to a direct toxic effect of oxygen upon the middle-ear epithelium. The problem resolves spontan- eously within hours. Differential pressure across a restricted aperture can generate large forces with serious, and often fatal, consequences. Examples include the inflow to a culvert or a sudden breach in a pipe con- taining gas at lower pressure than ambient. Potential mechanisms of injury include entrapment, compromised inspiratory effort, pri- mary trauma, and critical damage to equipment. Problems after the dive Autopsies on some asymptomatic divers with no history of DCI have revealed that their brains and spinal cords contain consid- erably more micro-infarcts than those of nondiving controls. Although the consequences of such damage are considered slight or subclinical, subjective reports of forgetfulness and poor concen- tration have been correlated with diving experience. Subjectively forgetful divers, as a group, performed worse than controls in tests of cognitive function, especially memory. They also had struc- tural differences on brain MRI. A history of welding increases the probability of a diver reporting these problems and the respirable heavy metal particles arising from preparation of surfaces and the welding fumes might be responsible for at least some of these findings. Imaging of long bones of divers and caisson workers show aseptic infarcts (dysbaric osteonecrosis) in a sizeable minority (up to 11%). The incidence is higher in those with a history of overt DCI. Lesions can occur after a single decompression, but their in- cidence rises with age, depth, and diving intensity. Those in the head, neck, or shaft are asymptomatic, but those at juxta-articular surfaces can be disabling. They are more common in caisson workers than divers, but are even seen in professional breath-hold divers, such as the Ama of Japan, in whom the dissolved gas burden must be light. The aetiology is unknown, but gas embolism is the favoured explanation. Commercial diving, especially saturation diving, enlarges total lung capacity and forced vital capacity (FVC). This is attributed to training effects of prolonged breathing of compressed gases. The FEV1/FVC ratio falls, due partly to the rise in FVC, but there are also hints of additional small-airway damage. Pulmonary capillary blood volume, as judged by carbon monoxide transfer, also falls. This appears to be due to transient episodes of hyperoxia during saturation-diving procedures, but might also be associated with venous gas emboli released during decompression. The effects are slight but definite and can be cumulative. There are no obvious clin- ical consequences. Mild high-tone deafness is found in commercial divers and is at- tributed to the noise of gas flows within their helmets. Fitness to dive Fitness assessment balances real and theoretical hazards against em- ployer and physician liability and duty of care, legislation, and the

candidate’s livelihood or desire to dive. Some organizations adopt didactic standards. Others use guidelines, which leave room for judgement by the physician and, sometimes, for informed risk to be carried by the candidate. Military and commercial diving is physically demanding and often remote from medical aid. These divers undergo periodic med- ical examinations. Periodicity and extent of examination depend on local regulations. Many recreational divers simply complete regular health declarations, undergoing examination only if a question is answered ‘positively’. Assessments aim to determine whether candidates: • are sufficiently physically fit to rescue a fellow diver, to swim in swift currents and rough waters, and to undertake any related nondiving tasks • are medically fit and have no problems that might incapacitate, impair, distract, predispose to decompression illness or baro- trauma, or otherwise make them a liability to themselves or others • have an acceptable risk of long-term health consequences from diving • require any restrictions or adjustments We must avoid understatement of the dangers of diving, especially at its most extreme, but must also assess hazard and risk realistically, enabling imaginative solutions for, and greater acceptability of, dis- abled divers who can often dive usefully without jeopardizing health or safety of those involved. An individual who is bodily fit, mentally stable, free of conditions such as epilepsy, obstructive lung disease, ill-controlled diabetes or asthma, alcohol or drug addiction, and has no history of ruptured eardrums or aural surgery is likely to be medically fit to dive. An acute chest, upper airway, or ear infection would be grounds for temporary unfitness. Diving should be avoided while taking medi- cation that could impair exercise capacity, ability to think clearly, or ability to orientate in space. Medical conditions that can mimic DCI deserve careful assessment. Women are advised not to dive during pregnancy as evidence is suggestive, though not yet con- clusive, that the unborn child is at increased risk of developmental defect. Compromised gas flow or gas exchange could predispose to injury or an inability to cope with the respiratory demands of diving. Risk factors for spontaneous lung rupture such as dis- tortion of lung tissue must be carefully assessed. Cross-sectional imaging can identify many more bullae than are visible on plain films but, in the absence of a validated quantification of risk of barotraumatic rupture, interpretation in terms of fitness to dive remains subjective. If a candidate runs several kilometres a day, is a good swimmer, was always good at games at school, and has no history of recent respiratory disease, they are very likely to be fit to dive. FEV1 multi- plied by 35 measures the maximal voluntary ventilation, an indi- cator of respiratory fitness. FEV1 should, therefore, be more than 75% predicted. FVC should also be more than 75% predicted be- cause there is good evidence that subjects with a low FVC and, by

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