

# 04 - 476 Hyperbaric and Diving Medicine

## 476 Hyperbaric and Diving Medicine

**Diabetes Mellitus** Well-controlled diabetes is not a contraindication for travel to high altitude. Most of the high-altitude diabetes advice is based on patients with type 1 diabetes and not type 2 diabetic patients with comorbidities. Altitude is known to both increase and decrease insulin sensitivity. An eye examination before travel may be useful. Insulin pumps are increasingly used, but bubble formation in the system may need to be closely monitored. For most diabetic patients, a continuous glucose monitor (CGM) is likely the best choice for glucose monitoring in the high-altitude environment, but a reliable glucometer should be carried by all diabetic patients. Regardless of the glucose monitoring modality selected, patients should be comfortable with the plan and transition appropriately. It is important for companions of diabetic trekkers to be fully aware of potential problems like hypoglycemia and have ready access to sweets.

**Chronic Lung Disease** Depending on disease severity and access to medical care, preexisting lung disease may not always preclude high-altitude travel. A proper pretravel evaluation must be conducted. Supplemental oxygen may be required if the predicted PaO<sub>2</sub> for the altitude is <50–55 mmHg. Preexisting pulmonary hypertension may also need to be assessed in these patients. If the result is positive, patients should be discouraged from ascending to high altitudes; if such travel is necessary, treatment with sustained-release nifedipine (30 mg twice a day) should be considered. Small-scale studies have revealed that when patients with bullous disease reach ~5000 m, bullous expansion and pneumothorax are not noted. Compared with information on chronic obstructive pulmonary disease, fewer data exist about the safety of travel to high altitude for people with pulmonary fibrosis, but acute exacerbation of pulmonary fibrosis has been seen at high altitude. A handheld pulse oximeter can be useful to check for oxygen saturation.

**PART 15 Disorders Associated with Environmental Exposures**

**Chronic Kidney Disease** Patients with chronic kidney disease can tolerate short-term stays at high altitudes, but theoretical concern persists about progression to end-stage renal disease. Acetazolamide, the drug most commonly used for altitude sickness, should be avoided by anyone with preexisting metabolic acidosis, which can be exacerbated by this drug. In addition, the acetazolamide dosage should be adjusted when the glomerular filtration rate falls to <50 mL/min, and the drug should not be used at all if this value falls to <10 mL/min.

**Cirrhosis** Of patients with cirrhosis, 16% may have portopulmonary arterial hypertension,

and 32% may have hepatopulmonary syndrome; these conditions may be detrimental at high altitude as they may cause exaggerated hypoxemia. Thus, screening for these problems is important in cirrhotic patients planning a high-altitude trip. In addition, acetazolamide may be inadvisable in these patients as the drug may increase the risk of hepatic encephalopathy. COVID-19 Evaluation is warranted before planned high-altitude travel in individuals who required care in an intensive care unit or suffered from myocarditis or arterial or venous thromboembolism. Preparticipation evaluation is also warranted in patients with persistent symptoms at least 2 weeks after a positive COVID-19 test or hospital discharge. Depending on the results of this evaluation, planned high-altitude travel may need to be modified or even deferred pending resolution of the identified abnormalities. Dental Problems Air resulting from decay in the root system could expand on ascent and lead to increasing pain. A good dental checkup before a trekking or climbing trip may be prudent. Malignancy Patients with current or previous malignancy may be affected in a variety of ways by altitude. Both radiation of the neck or paraganglioma/chemodectomas (which are seen at higher rates in residents of high-altitude areas) may result in carotid body dysfunction and reduced acclimatization. Pulmonary toxicities from chemotherapies (e.g., bleomycin) and radiation of the chest increase pulmonary artery pressures and impair lung function, theoretically increasing the risk of HAPE and decreasing exercise tolerance. Some chemotherapies such as bleomycin have traditionally been thought to increase susceptibility of lung tissue to oxygen toxicity and may complicate the use

of supplemental oxygen, if needed. Cardiotoxic therapies for cancer decrease exercise tolerance and increase cardiovascular disease. Such patients will be at increased cardiovascular risk with strenuous activity or hypoxic stress that is expected at altitude. Patients with known metastasis to bone or those at high risk should be evaluated prior to expedition travel. ■ ■CHRONIC MOUNTAIN SICKNESS AND HIGH-ALTITUDE PULMONARY HYPERTENSION IN HIGHLANDERS The largest populations of highlanders live in the South American Andes, the Tibetan Plateau, and parts of Ethiopia. Chronic mountain sickness (Monge's disease) is a disease in highlanders that is characterized by excessive erythrocytosis with moderate to severe pulmonary hypertension leading to cor pulmonale. This condition was originally described in South America and has also been documented in Colorado and in the Han Chinese population in Tibet; it is much less common in Tibetans or in Ethiopian highlanders. Migration to a low altitude results in the resolution of chronic mountain illness. Venesection and acetazolamide are helpful. High-altitude pulmonary hypertension is also a subacute disease of long-term high-altitude residents. Unlike Monge's disease, this syndrome is characterized primarily by pulmonary hypertension (not erythrocytosis) leading to heart failure. Indian soldiers living at extreme altitudes for prolonged periods and Han Chinese infants born in Tibet have presented with the adult and infantile forms, respectively. High-altitude pulmonary hypertension bears a striking pathophysiologic resemblance to brisket disease in cattle. Descent to a lower altitude is curative. ■ ■FURTHER READING Basnyat B: High altitude pilgrimage medicine. *High Alt Med Biol* 15:434, 2014. Basnyat B, Murdoch D: High altitude illness. *Lancet* 361:1967, 2003. Hillebrandt D et al: UIAA medical commission recommendations for mountaineers, hillwalkers, trekkers, and rock and ice climbers with diabetes. *High Alt Med Biol* 24:110, 2023. Keyes LE et al: Blood pressure and altitude: An observational cohort study of hypertensive and nonhypertensive Himalayan trekkers in Nepal. *High Alt Med Biol* 18:267, 2017. Luks AM et al: Return to high altitude after recovery from coronavirus disease 2019. *High Alt Med Biol* 22:119, 2021. Luks AM et al: Wilderness Medical Society clinical practice guidelines for the prevention, diagnosis, and treatment of acute altitude illness: 2024 update. *Wilderness Environ*

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## Hyperbaric and Diving

Medicine Hyperbaric medicine is the treatment of certain health disorders using whole-body exposure to pressures  $>101.3$  kPa (1 atmosphere or 760 mmHg). In practice, this invariably includes breathing oxygen during the exposure, thus the administration of hyperbaric oxygen therapy (HBO2T). The Undersea and Hyperbaric Medical Society (UHMS) defines HBO2T as “a medical procedure requiring a physician’s prescription and oversight” in which “patients must have their entire body placed within a hard sided hyperbaric chamber that meets

FIGURE 476-1 A monoplace chamber. The chamber is compressed with 100% oxygen. Although direct patient access is not possible, intravenous administration of fluids and drugs, ventilation, and invasive monitoring can be performed. (Prince of Wales Hospital, Sydney.) the American Society of Mechanical Engineers and Pressure Vessels for Human Occupancy (ASME-PVHO-1) code, and the National Fire Protection Agency (NFPA 99) code and standards for hyperbaric chambers, at a pressure of not less than 2.0 atmospheres absolute (ATA) (202.65 kPa) while breathing physician prescribed medical grade oxygen for an amount of time that is typically between 90-120 minutes per treatment.” Historically, compression chambers were first used for the treatment of divers and compressed air workers suffering decompression sickness (DCS; “the bends”). Now they are used for treating a variety of problems, and relevant chambers are variously called a hyperbaric chamber, recompression chamber, or decompression chamber, depending on the clinical and historical context. They may be capable of compressing a single patient (a monoplace chamber) or multiple patients and attendants as required (a multiplace chamber) (Figs. 476-1 and 476-2). In multiplace chambers, the chamber atmosphere is pressurized with air and the patient breathes oxygen through a special delivery system. The same may be true of monoplace chambers, although in some, the chamber environment is pressurized with oxygen and the patient simply breathes from that environment. ■ ■MECHANISMS OF HYPERBARIC

OXYGEN THERAPY In the late 1800s, the earliest systematic use of pressure as a therapeutic modality was in treating decompression sickness, a disorder caused by bubbles in blood or tissues. The use of pressure (recompression) to instantly reduce bubble size made mechanistic sense FIGURE 476-2 A multiplace chamber. Multiplace chambers are compressed with air; 100% oxygen is administered via head tent, mask, or endotracheal tube. These chambers can treat several patients simultaneously and can easily accommodate critically ill and intubated patients. (Karolinska University Hospital.)

and met with obvious clinical success when instituted. In the 1960s, oxygen breathing during hyperbaric exposure was incorporated into recompression protocols to increase the diffusion gradient for inert gas from bubble to tissue to blood and alveoli. This too appeared to improve outcomes. Recompression of divers is further discussed under “Diving Medicine.”

Use of HBO2T in nondiving disease focused initially on indications such as carbon monoxide poisoning, nonhealing wounds, and radiation tissue injury, where it was believed that hypoxia was a fundamental contributory problem and that hyperbaric oxygen could ameliorate this. Indeed,

breathing oxygen at hyperbaric pressures not only ensures complete saturation of hemoglobin in blood but also results in markedly elevated quantities of dissolved oxygen in arterial plasma at a very high  $P_{O_2}$ . Most HBO2T regimens involve oxygen breathing at between 203 and 284 kPa (2 and 2.8 ATA). Breathing oxygen at 284 kPa (2.8 ATA) can theoretically increase the arterial  $P_{O_2}$  to more than ~270 kPa (~2025 mmHg) and the volume of oxygen dissolved in plasma to ~60 mL/L, with the latter being sufficient to sustain life without hemoglobin as proven in animal studies. The very high arterial  $P_{O_2}$  markedly improves oxygen diffusion distances through tissues. As with pressure and oxygen breathing for bubble disorders in diving, the use of markedly elevated plasma oxygen levels in treating hypoxic wounds and irradiated tissue also met with apparent success, initially reinforcing the belief that intermittent support of oxygen-dependent healing processes such as fibroblast replication, collagen deposition and cross-linking, and angiogenesis was the principal mechanism by which HBO2T could be helpful. As important as this mechanism may be, more recent research has revealed that intermittent hyperbaric hyperoxia has physical and biochemical/cell signaling effects that extend well beyond simple support of oxygen-dependent healing processes and the duration of the hyperbaric exposure. Indeed, there are pharmacologic effects that are profound and relatively long-lasting. Some of these are summarized in Fig. 476-3.

CHAPTER 476 Hyperbaric and Diving Medicine Thus, although removal from the hyperbaric chamber results in a rapid return of poorly vascularized tissues to their hypoxic state, even a single dose of HBO2T produces changes in fibroblast, leukocyte and angiogenic functions, and antioxidant defenses that persist many hours after oxygen tensions are returned to pretreatment levels. Explanations for this effect focus on production of reactive oxygen species (ROS) such as superoxide ( $O_2^-$ ), hydrogen peroxide ( $H_2O_2$ ), and reactive nitrogen species (RNS) such as nitric oxide (NO) during exposure to hyperbaric hyperoxia. It appears that these species, often considered harmful in biological tissues, participate in diverse cell signaling pathways involved in production of a range of cytokines, angiogenic growth factors, and other modulators of inflammation and tissue repair whose net effect is to accelerate healing. Such mechanisms are complex and at times apparently paradoxical. For example, it is well established by *in vitro* and *in vivo* studies that notionally proinflammatory pulses of oxidative stress induced by hyperbaric hyperoxia actually result in subsequent anti-inflammatory effects such as reduced leukocyte  $\beta_2$  integrin adhesion molecule expression, an effect that may be protective when flaps and grafts are threatened or if endothelium is damaged by circulating bubbles. Most of the important indications for HBO2T are now recognized as benefiting in some way from activity in these cell signaling pathways. For example, when used to treat chronic hypoxic wounds, HBO2T has been shown to enhance bacterial killing and phagocytosis by providing the oxygen substrate for macrophage activity; stimulate the synthesis of multiple angiogenic growth factors; inhibit leukocyte activation and adherence to damaged endothelium; and mobilize pluripotent vasculogenic progenitor cells from the bone marrow. The interactions between these mechanisms remain a very active field of investigation. Figure 476-3 also depicts several other mechanisms of action that may be relevant in certain situations. Hyperoxia tends to induce vasoconstriction and that, along with the exaggerated standing osmotic gradient between an ultra-high  $P_{O_2}$  in arteriolar blood and the much lower  $P_{O_2}$  in tissues, may reduce tissue edema. Hyperoxia is also directly toxic or bacteriostatic to anaerobic bacteria.

Hyperbaric oxygen Enhanced inert gas diffusion gradients between bubble, tissue, and lungs High arterial  $PO_2$  Hydrostatic compression Bubble volume reduction Enhanced  $O_2$  diffusion Generation of ROS and RNS Osmotic effect Restoration of tissue normoxia Enhanced phagocytosis,

angiogenesis, and fibroblast activity DCS CAGE Wound healing, radiation tissue injury PART 15 Disorders Associated with Environmental Exposures FIGURE 476-3 Mechanisms of action of hyperbaric oxygen. There are many consequences of compression and oxygen breathing. The cell-signaling effects are potentially most important. Examples of indications are shown in the shaded boxes. CAGE, cerebral arterial gas embolism; DCS, decompression sickness; RNS, reactive nitrogen species; ROS, reactive oxygen species. ■ ■ INDICATIONS FOR HYPERBARIC

OXYGEN THERAPY The “accepted” indications for HBO2T are evolving and sometimes controversial. In 1977, the UHMS systematically examined claims for the use of HBO2T in >100 disorders and found sufficient evidence to support routine use in only 12. The Hyperbaric Oxygen Therapy Committee of that organization has continued to update this list periodically (to the current list of 15) (Table 476-1) with an increasingly formalized system of appraisal for new indications and emerging evidence. Around the world, other relevant medical organizations have generally taken a similar approach. However, accepted indications vary considerably—particularly those recommended by hyperbaric medical societies in Russia and China where HBO2T has gained much wider support than in the United States, Europe, and Australasia. Nevertheless, there are now >30 Cochrane reviews summarizing the randomized trial evidence for 27 putative indications, and numerous other systematic reviews, including attempts to examine the cost-effectiveness of HBO2T. Following are short reviews of three important indications currently accepted by the UHMS, and a brief summary of several exploratory indications. Late Radiation Tissue Injury Radiotherapy is a well-established treatment for suitable malignancies. In the United States alone, ~300,000 individuals annually will become long-term survivors of cancer treated by irradiation. Developments in radiotherapy techniques such as intensity-modulated radiation therapy and hypofractionated stereotactic radiation therapy have improved the precision of radiation delivery. This has allowed the use of higher doses of radiation with better cure rates, but the incidence of radiation-related complications has not changed much. Serious radiation-related complications developing months or years after treatment (late radiation tissue injury [LRTI]) will significantly affect between 5 and 10% of long-term survivors, although incidence varies widely with dose, age, and site. LRTI may involve bone or soft tissue and is most common in the head and neck, chest wall, breast, and pelvis (where it may involve the bowel or bladder).

Hyperoxic vasoconstriction Edema reduction Anti-inflammatory effects e.g. ↓β2 integrins ↑Wound growth factors Stem cell mobilization Threatened grafts/flaps DCS CAGE Crush injury PATHOLOGY AND CLINICAL COURSE One explanation for long-term injury to irradiated tissue is that initial radiation exposure produces fibrosis and an obliterative endarteritis from which the tissue may be incapable of recovering because of its hypovascular hypoxic nature. Indeed, its microvasculature may continue to deteriorate slowly over months to years with breakdown occasionally occurring spontaneously or when accelerated by an event like trauma or surgery. An alternative model of pathogenesis suggests that rather than a primary hypoxic state, the principal trigger is an overexpression of inflammatory cytokines that promote fibrosis, probably through oxidative stress and mitochondrial dysfunction, with tissue hypoxia as a secondary provocation. Ultimately, and once again often triggered by a further physical insult such as surgery or infection, there may be insufficient TABLE 476-1 Current List of Indications for Hyperbaric Oxygen Therapy

1. Air or gas embolism (includes diving-related, iatrogenic, and accidental causes)
2. Carbon monoxide poisoning (including poisoning complicated by cyanide poisoning)

3. Clostridial myositis and myonecrosis (gas gangrene)
4. Crush injury, compartment syndrome, and acute traumatic ischemias
5. Decompression sickness
6. Arterial insufficiency including central retinal arterial occlusion and problem wounds
7. Severe anemia
8. Intracranial abscess
9. Necrotizing soft tissue infections (e.g., Fournier's gangrene)
10. Osteomyelitis (refractory to other therapy)
11. Delayed radiation injury (soft tissue injury and bony necrosis)
12. Skin grafts and flaps (compromised)
13. Acute thermal burn injury
14. Sudden sensorineural hearing loss
15. Avascular necrosis (aseptic osteonecrosis) Source: The Undersea and Hyperbaric Medical Society (2024).

oxygen to sustain normal function, and the tissue becomes necrotic (radiation necrosis). LRTI may be life-threatening and significantly reduce quality of life. Historically, the management of these injuries has been unsatisfactory. Conservative treatment is usually restricted to symptom management, whereas definitive treatment traditionally entails surgery to remove the affected part and extensive repair. Surgical intervention in an irradiated field is often disfiguring and associated with an increased incidence of delayed healing, breakdown of a surgical wound, or infection. HBO2T may act by several mechanisms, including edema reduction, vasculogenesis, and enhancement of macrophage activity (Fig. 476-3). However, the ability of HBO2T to promote angiogenesis in irradiated tissue represents the most important effect. The intermittent application of HBO2T is the only intervention shown in multiple studies to increase the microvascular density in irradiated tissue in both animals and humans.

**CLINICAL EVIDENCE** The typical course of HBO2T consists of 30–60 once-daily compressions to 202.6–243.1 kPa (2–2.4 ATA) for 1.5–2 h each session, often bracketed around surgical intervention if required. Although HBO2T has been used for LRTI since at least 1975, most clinical studies have been limited to small case series or individual case reports. A 2023 Cochrane systematic review included 18 randomized controlled trials (RCTs) published since 1985 and was able to draw the following conclusions based on meta-analysis. Pooled analysis of studies across all LRTI types suggested HBO2T improves tissue healing (risk ratio [RR] of healing with HBO2T, 1.39; 95% confidence interval [CI], 1.02–1.89). In patients with osteoradionecrosis of the jaw or at risk of osteoradionecrosis of the jaw following planned surgery, HBO2T reduced the risk of wound dehiscence (RR, 0.24; 95% CI, 0.06–0.94). The report also detailed individual randomized studies with notable positive results, particularly in regard to radiation proctitis and radiation cystitis. There were also negative randomized studies in both these indications. Current UHMS guidelines (see Huang in “Further Reading”) support use of HBO2T for most forms of LRTI, a problem that is notoriously difficult to treat by other means. The American Society of Colon and Rectal Surgeons designated HBO2T as a class 1B intervention (strongly indicated based on moderate-quality evidence) for radiation proctitis. A previous strongly positive RCT in treating bleeding radiation cystitis (RR for complete or significant improvement after HBO2T, 3.63; 95% CI, 1.69–7.79) was recently corroborated by the Dartmouth Multicenter International Registry Initiative. In 370 bleeding patients One schema for using transcutaneous oximetry to assist in patient selection for HBO2T. If the wound area is hypoxic and responds to the administration of oxygen at 1 ATA or 2.4 ATA, treatment may be justified. Problem wound referred

for assessment Transcutaneous wound mapping on air PtcO<sub>2</sub> <40 mmHg\* PtcO<sub>2</sub> >100 mmHg  
Transcutaneous mapping on 100% oxygen 1 ATA PtcO<sub>2</sub> 35–100 mmHg Transcutaneous mapping  
on 100% oxygen 2.4 ATA HBO<sub>2</sub>T indicated PtcO<sub>2</sub> >200 mmHg FIGURE 476-4 Determining  
suitability for hyperbaric oxygen therapy guided by transcutaneous oximetry around the wound  
bed. \*In diabetic patients, <50 mmHg may be more appropriate. PtcO<sub>2</sub>, transcutaneous oxygen  
pressure.

treated with HBO<sub>2</sub>T, the Radiation Therapy Oncology Group hematoma score fell from a median of  
2 (interquartile range [IQR], 2) to 0 (IQR, 2) (see Moses et al. in “Further Reading”). Despite  
occasional positive case reports, results for radiation-induced neurologic injuries (brain and spinal  
cord) are less encouraging.

**Selected Problem Wounds** A problem wound is any cutaneous ulceration that requires a prolonged  
time to heal, does not heal, or recurs. In general, wounds referred to hyperbaric facilities are those  
for which sustained attempts to heal by other means have failed. Problem wounds are common  
and constitute a significant health problem. It has been estimated that 1% of the population of  
industrialized countries will experience a leg ulcer at some time. The global cost of chronic wound  
care may be as high as U.S. \$25 billion per year. **PATHOLOGY AND CLINICAL COURSE** By definition,  
chronic wounds are indolent or deteriorating and resistant to the wide array of treatments applied.  
Although there are many contributing factors, these wounds most commonly arise in association  
with one or more comorbidities such as diabetes, peripheral venous or arterial disease, or pro-  
longed pressure (decubitus ulcers). A common denominator among these various contributors is  
hypoxia. First-line treatments are aimed at correction of the underlying pathologies (e.g., vascular  
reconstruction, compression bandaging, normalization of nutritional deficiencies or normalization of  
blood glucose level). HBO<sub>2</sub>T should be regarded as an adjunctive therapy to be applied  
simultaneously with amelioration of all modifiable risk factors for nonhealing wounds and good  
general wound care practice to maximize the chance of healing. Not all problem wounds will  
respond to HBO<sub>2</sub>T. A selection process based on responses to an oxygen challenge is often  
recommended. Wounds in hypoxic tissues often display poor or absent healing and have potential  
to benefit from HBO<sub>2</sub>T depending on the cause, magnitude, and reversibility of hypoxia. Some  
causes of tissue hypoxia will be reversible with HBO<sub>2</sub>T (e.g., microvascular disease and tissue  
edema), whereas some will respond better to surgical revascularization (e.g., critical arterial  
stenosis). When tissue hypoxia can be overcome by a high driving pressure of oxygen in the  
arterial blood, this can be demonstrated by measuring transcutaneous Po<sub>2</sub> using a modified  
transcutaneous Clarke electrode. It follows that many guidelines for patient selection for HBO<sub>2</sub>T  
include the interpretation of transcutaneous oxygen tensions around the wound while breathing  
air and oxygen at pressure (Fig. 476-4). Other methods include simple ankle-brachial index and  
skin perfusion pressure. **CHAPTER 476 Hyperbaric and Diving Medicine** Contraindication, critical  
major vessel disease, or surgical option available No Suitable for compression? Yes Not hypoxic  
(PtcO<sub>2</sub> >40 mmHg) PtcO<sub>2</sub> <35 mmHg unresponsive HBO<sub>2</sub>T unlikely to be effective PtcO<sub>2</sub> <100  
mmHg HBO<sub>2</sub>T indicated on a case-by-case basis. Consider alternatives. PtcO<sub>2</sub> >100 but <200  
mmHg

**CLINICAL EVIDENCE** The typical course of HBO<sub>2</sub>T consists of 20–40 once-daily compressions to  
2–2.4 ATA for 1.5–2 h each session but is highly dependent on the clinical response. Both  
retrospective and prospective cohort studies suggest that 6 months after a course of therapy,

~70% of indolent ulcers will be substantially improved or healed. Often these ulcers have been present for many months or years, suggesting the application of HBO<sub>2</sub>T significantly improves the healing trajectory, either primarily or in conjunction with other strategies. A 2015 Cochrane review included 12 RCTs and concluded that the chance of a diabetic ulcer healing improved with HBO<sub>2</sub>T (10 trials; RR, 2.35; 95% CI, 1.19–4.62). Although there was a trend toward reduced risk of major amputations with HBO<sub>2</sub>T, it did not reach statistical significance (RR, 0.36; 95% CI, 0.11–1.18). A 2024 meta-analysis of 29 RCTs (see Chen et al. in “Further Reading”) reported that in patients with diabetic foot ulcers treated with HBO<sub>2</sub>T there was increased complete healing (odds ratio [OR], 2.8; 95% CI, 2.3–3.5) and reduced amputations (OR, 0.41; 95% CI, 0.18–0.95). Using data from a recent randomized trial, the cost per limb saved in treating severe (Wagner stage III/IV) diabetic foot ulcers with HBO<sub>2</sub>T in the Netherlands was €19,005 (95% CI, –€18,487 to €264,334) (see Brouwer et al. in “Further Reading”).

**Carbon Monoxide Poisoning** Carbon monoxide (CO) is a colorless, odorless gas formed during incomplete hydrocarbon combustion. Although CO is an essential endogenous neurotransmitter linked to NO metabolism and activity, it is also a leading cause of poisoning deaths and, in the United States alone, results in >50,000 emergency department visits per year and ~2000 deaths. Although there are large variations from country to country, about half of nonlethal exposures are due to self-harm. Accidental poisoning is commonly associated with defective or improperly installed heaters, house fires, and industrial exposures. The motor vehicle is by far the most common source of intentional poisoning.

**PART 15 Disorders Associated with Environmental Exposures**

**PATHOLOGY AND CLINICAL COURSE** The pathophysiology of CO exposure is incompletely understood. CO binds to hemoglobin with an affinity >200 times that of oxygen, directly reducing the oxygen-carrying capacity of blood and further promoting tissue hypoxia by shifting the oxyhemoglobin dissociation curve to the left. CO is also an anesthetic agent that inhibits evoked responses and narcotizes experimental animals in a dose-dependent manner. The associated loss of airway patency together with reduced oxygen carriage in blood may cause death from acute arterial hypoxia. CO also causes harm by other mechanisms including direct disruption of cellular oxidative processes, binding to heme proteins including cytochrome a/a<sub>3</sub>, and peroxidation of brain lipids. The brain and heart are the most sensitive target organs, in part due to their high blood flow, poor tolerance of hypoxia, and high oxygen requirements. Minor exposures may be asymptomatic or present with vague constitutional symptoms such as headache, lethargy, and nausea, whereas higher doses may present with poor concentration and cognition, short-term memory loss, confusion, seizures, and loss of consciousness. Carboxyhemoglobin (COHb) levels on admission may confirm exposure but do not reliably reflect the severity or the prognosis of CO poisoning. Over the longer term, surviving patients commonly have neuropsychological sequelae that, for uncertain reasons, are sometimes delayed after an interval of days, weeks, or even months of apparent recovery. Motor disturbances, peripheral neuropathy, hearing loss, vestibular abnormalities, dementia, and psychosis have all been reported. Risk factors for poor outcome are age >35 years, exposure for >24 h, acidosis, and loss of consciousness. CO poisoning is one of the longest-standing indications for HBO<sub>2</sub>T, based traditionally on the obvious connection between exposure and tissue hypoxia and the ability of HBO<sub>2</sub>T to rapidly overcome this hypoxia. Moreover, CO is eliminated rapidly via the lungs on application of HBO<sub>2</sub>T, with a half-life of ~21 min breathing oxygen at 2.0 ATA versus 5.5 h or 71 min breathing air or oxygen (respectively) at sea level. In practice, it seems unlikely that HBO<sub>2</sub>T could be delivered in time to prevent either acute hypoxic death or irreversible global cerebral hypoxic injury in severe cases. Thus, if HBO<sub>2</sub>T is

beneficial in CO

poisoning, it must be by reducing the likelihood of persisting and/or delayed neurocognitive deficits through a mechanism(s) other than the simple reversal of arterial hypoxia due to high levels of COHb. There is evidence that relevant mechanisms include reversal of CO binding to cytochromes, upregulation of oxidative stress defenses, reduction in leukocyte adherence, impairment of lipid peroxidation, and others.

**CLINICAL EVIDENCE** The typical course of HBO2T consists of two to three compressions to 2–2.8 ATA for 1.5–2 h each session. It is common for the first two compressions to be delivered within 24 h of the exposure. To date, there have been six RCTs of HBO2T for CO poisoning, although only four have been reported in full. While a Cochrane review suggested there is insufficient evidence to confirm a beneficial effect of HBO2T on the chance of persisting neurocognitive deficit following poisoning (34% of patients treated with oxygen at 1 atmosphere vs 29% of those treated with HBO2T; OR, 0.78; 95% CI, 0.54–1.1), this may have more to do with poor reporting and inadequate follow-up than with evidence that HBO2T is not effective. In the most methodologically rigorous of the analyzed studies (see Weaver et al. in “Further Reading”), at 6 weeks after poisoning, 46% of patients treated with normobaric oxygen alone had cognitive sequelae compared to 25% of those who received HBO2T ( $p = .007$ ; number needed to treat [NNT] = 5; 95% CI, 3–16). At 12 months, the difference remained significant (32 vs 18%;  $p = .04$ ; NNT = 7; 95% CI, 4–124) despite considerable loss to follow-up. On this basis, HBO2T remains widely advocated for the routine treatment of patients with moderate to severe CO poisoning, in particular in those older than 35 years, presenting with a metabolic acidosis on arterial blood-gas analysis, exposed for lengthy periods, or with a history of unconsciousness. ■ ■

**EXPLORATORY INDICATIONS**

**Inflammatory Bowel Disease** There is mounting evidence that HBO2T enhances healing in flairs of fistulizing Crohn’s disease or severe ulcerative colitis. Indeed, there are already two small positive RCTs along with numerous observational studies and case reports of apparent benefit. A multicenter RCT is underway. Benefit in these disorders may arise from enhancement of healing processes described earlier and/or anti-inflammatory activity.

**Mild Traumatic Brain Injury** After anecdotal reports of success, HBO2T became a focus of interest for the treatment of veterans returning from Afghanistan with postconcussive syndromes. This led to the execution of three sham-controlled RCTs by the different branches of the U.S. military, which demonstrated improvements in patients receiving both HBO2T and the sham. These improvements were considered due to placebo effects. However, a fourth study appeared to show benefit for HBO2T independent of any placebo effect, and collective re-evaluation of all studies suggests that placebo effects account for some but not all of the measured improvements. It follows that there is ongoing interest in HBO2T for mild traumatic brain injury, but a large definitive study is needed.

**SARS-CoV-2 (COVID-19) Infection** Although currently of dwindling relevance, it is notable that during the COVID-19 pandemic there were two RCTs of relatively small size and low quality that evaluated the benefit from HBO2T in hypoxic patients at risk of requiring intubation and ventilation. The studies were underpowered for outcomes such as death or need for mechanical ventilation but did show a faster trajectory of recovery in patients treated with HBO2T. Those studies along with several observational studies and case reports were recently summarized (see Boet et al. in “Further Reading”). To date, cases of pulmonary barotrauma precipitated by gas trapping in COVID pneumonia have not emerged, but little can be concluded about safety based on reporting of relatively low numbers of cases. ■ ■

**ADVERSE EFFECTS** HBO2T is generally well tolerated and safe in clinical practice. Patients often experience an adverse event at some time during their treatment

course, but most are mild and self-limiting. Adverse effects are associated with both alterations in pressure (barotrauma) and the administration of oxygen. Barotrauma Barotrauma may occur when any noncompliant gasfilled space within the body does not equalize with environmental pressure during compression or decompression. Middle ear pain during compression (analogous to descent in an airplane) is the most common complication of HBO<sub>2</sub>T, reported in ~30% of patients. Most result in either no or only trivial injury as a result, and prevention by slower compression and training in insufflation of the middle ear via the Eustachian tube is usually successful. Patients unable to insufflate the middle ear and unconscious patients typically require myringotomies or formal grommets across the tympanic membrane. Other less common sites for barotrauma of compression include the respiratory sinuses and dental caries. The lungs are potentially vulnerable to barotrauma of decompression (see “Diving Medicine” below), but the decompression following HBO<sub>2</sub>T is so slow that pulmonary barotrauma is extremely rare and has only been seen when a patient has slowly communicating gas trapping lesions such as bullae.

**Oxygen Toxicity** The practical limit to the oxygen dose (whose components are inspired pressure and duration) in a single treatment session is cerebral oxygen toxicity. The most common acute manifestation is a tonic-clonic seizure, often preceded by facial twitching, anxiety, and agitation. The cause is thought to be an effect of ROSs on excitable neurons. Although clearly dose-dependent, onset is very variable both between individuals and within the same individual on different days. In large series reporting routine clinical hyperbaric practice (compressions to 2–2.4 ATA), the incidence of seizures is ~1–2/10,000 compressions. Chronic oxygen poisoning most commonly manifests as myopic shift. This is due to alterations in the refractive index of the lens following oxidative damage that reduces the solubility of lenticular proteins, a process similar to that associated with senescent cataract formation. Up to 75% of patients show alteration in visual acuity after a course of 30 treatments at 202.6 kPa (2 ATA). Although most return to pretreatment values 6–12 weeks after cessation of treatment, a small proportion do not revert and may require a change in corrective lenses. A more rapid maturation of preexisting cataracts has occasionally been associated with HBO<sub>2</sub>T. Although a theoretical problem, the development of pulmonary oxygen toxicity over time does not seem to be problematic in practice—probably due to the short and intermittent nature of the exposures. ■ ■

**CONTRAINDICATIONS** There are few absolute contraindications to HBO<sub>2</sub>T. Untreated pneumothorax is considered a contraindication because intrapleural gas may expand on decompression and come under tension. Prior to any compression, patients with a pneumothorax should have a patent chest drain in place. The presence of other obvious risk factors for pulmonary gas trapping such as bullae should trigger a careful analysis of the risks of treatment versus benefit. Recent systemic bleomycin or mitomycin C treatment deserves mention because of its association with a partially dose-dependent pneumonitis in ~20% of people. Patients treated with bleomycin, particularly those suffering pneumonitis, are often considered at risk of deterioration in ventilatory function following exposure to high oxygen tensions, even some years after treatment. Published experience suggests the relationship between distant bleomycin exposure and subsequent risk of pulmonary oxygen toxicity may have been overstated, and such patients have undergone uncomplicated HBO<sub>2</sub>T. Nevertheless, any patient with a history of receiving these drugs (particularly recently and particularly if there was a consequent pneumonitis) should be carefully evaluated and counseled prior to exposure to HBO<sub>2</sub>T. ■ ■

**CHALLENGES AND CONTROVERSIES IN HYPERBARIC MEDICINE** Despite an increased understanding of mechanisms and an improving evidence base, hyperbaric medicine has struggled to achieve

widespread adoption in treating relevant disorders. There are several contributing factors, but high among them are a lack of relevant teaching at medical schools and a paucity of large well-conducted RCTs that clearly demonstrate efficacy in the targeted indications.

In turn, it should be appreciated that there are multiple factors that combine as an impediment to large RCTs in hyperbaric medicine. First, funding for clinical research has been difficult in an environment where the pharmacologic agent under study is abundant, cheap, and unpatentable. Second, some of the indications (such as necrotizing fasciitis and central retinal artery occlusion) are rare, sporadic, and acute. Conducting RCTs of treatment in such problems is notoriously difficult, especially where the putative treatment is available in only a small minority of hospitals. Not surprisingly, many of the accepted treatments applied in such problems are not supported by large RCTs. Third, the natural history of an evolving indication in HBO<sub>2</sub>T is that treatment might first be tried sporadically in a small number of patients on the basis that its use makes biological sense. Unlike a new drug under development, there are no significant regulatory barriers to this. If apparently successful, then there is a risk that application of HBO<sub>2</sub>T may become “accepted” before an RCT is proposed or organized, and clinicians then become reluctant to have patients randomized into a study. Fourth, a course of HBO<sub>2</sub>T typically involves substantial commitment in time and logistical effort. This is a disincentive to recruitment of patients into a study with a high likelihood of being allocated to a placebo group. Finally, the potentially powerful placebo effect of HBO<sub>2</sub>T is well recognized and highlights the importance of blinded sham controls. Despite these impediments to RCTs, many have been successfully executed across the various indications for HBO<sub>2</sub>T. Some have emerged long after the indication was accepted onto the UHMS list on the basis of lower-level evidence. A good example is crush injury and traumatic ischemia, for which the best supporting RCT has only recently emerged (see Miller et al. in “Further Reading”). Recognizing these challenges, however, an international clinical registry has also been created and is now beginning to produce useful outcome data that, at the very least, can be compared to the known natural history during standard management of the various monitored indications (see “Late Radiation Tissue Injury,” above, and Moses et al. in “Further Reading”).

**CHAPTER 476 Hyperbaric and Diving Medicine** Another challenge to the widespread acceptance of HBO<sub>2</sub>T for established uses is the continuing misguided advocacy for hyperbaric therapy (sometimes using air breathing) as a panacea for treating numerous diseases, improving general well-being, and slowing aging. The prescription and delivery of HBO<sub>2</sub>T requires no medical license in many jurisdictions. Some claims of efficacy refer to small supportive studies with a high risk of bias or placebo effect. Physicians should focus their practice on accepted or approved indications that are periodically updated (see Huang in “Further Reading”).

**DIVING MEDICINE** Underwater diving is both a popular recreational activity and a means of employment in a range of tasks from underwater construction to military operations. It is a complex activity with unique hazards and medical complications arising mainly as a consequence of the dramatic changes in pressure associated with both descent and ascent through the water column. For every 10.1-m increase in depth of seawater, the ambient pressure (Pamb) increases by 101.3 kPa (1 atmosphere) so that, for example, a diver at 20 m depth is exposed to a Pamb of 303.9 kPa (3 ATA), made up of 1 ATA due to atmospheric pressure and 2 ATA generated by the water column. ■ ■

**BREATHING EQUIPMENT** Most diving is undertaken using a self-contained underwater breathing apparatus (scuba) consisting of one or more cylinders of compressed gas connected to a pressure-reducing regulator and a demand valve activated by inspiratory effort. Some divers use rebreathers, which comprise a closed or semi-closed breathing circuit with a carbon dioxide scrubber and an oxygen addition system designed to maintain a safe

inspired  $P_{O_2}$ . Exhaled gas is recycled, and gas consumption is limited to little more than the oxygen metabolized by the diver.

Rebreathers are therefore popular for deep dives where expensive helium is included in the respired mix (see below). Occupational divers frequently use “surface supply” equipment where gas, along with other utilities such as communications and power, is supplied via an “umbilical” cable from the surface.

All these systems must supply gas to the diver at the ambient pressure of the surrounding water or inspiration would be impossible against the water pressure. For most recreational diving, the respired gas is air. Pure oxygen is rarely used because there is a dose-dependent risk (where “dose” is a function of exposure time and inspired  $P_{O_2}$ ) that oxygen may provoke seizures above an inspired  $P_{O_2}$  of 130 kPa (1.3 ATA). The maximum acceptable inspired  $P_{O_2}$  in diving is often considered to be 161 kPa (1.6 ATA), which would be achieved when breathing pure oxygen at 6 m or air at 66 m. This is a conspicuously lower  $P_{O_2}$  than routinely used for hyperbaric therapy (see earlier), reflecting a higher risk of oxygen toxic seizures during immersion and exercise. In order to maintain a safe  $P_{O_2}$  and avoid dangerous oxygen exposures, very deep diving requires the use of inspired oxygen fractions lower than in air ( $F_{O_2}$  0.21), and divers tailor the oxygen content of their gases to remain within recommended exposure guidelines. Deep-diving gases include helium as a substitute for some or all of the nitrogen to reduce both the narcotic effect and high gas density that result from breathing nitrogen at high pressures.

### ■ ■ SUITABILITY FOR DIVING

The most common reason for physician consultation in relation to diving is for the evaluation of suitability for diver training or continuation of diving after a health event. Occupational diver candidates are usually compelled to see doctors with specialist training in the field, both at entry to the industry and periodically thereafter, and their medical evaluations are usually conducted according to legally mandated standards. In contrast, in most jurisdictions, prospective recreational diver candidates simply complete a self-assessment medical questionnaire prior to diver training. If there are no positive responses, the candidate proceeds directly to training, but positive responses mandate the candidate see a clinician, often a primary care physician, for evaluation of the identified medical issue. In the modern era, such consultations have evolved from a simple proscriptive exercise of excluding those with potential contraindications to an approach in which each case is considered on its own merits and an individualized evaluation of risk is made. Such evaluations require integration of diving physiology, the impact of associated medical problems, and knowledge of the specific medical condition(s) of the candidate. A detailed discussion is beyond the scope of this chapter, but several important principles are outlined below.

### PART 15 Disorders Associated with Environmental Exposures

There are three primary questions that should be answered in relation to any medical condition reported by a prospective diver: (1) Could the condition be exacerbated by diving? (2) Could the condition make a diving medical problem more likely? (3) Could the condition prevent the diver from meeting the functional requirements of diving? As examples of positive answers to these questions (respectively): epilepsy is usually considered to imply high risk because there are epileptogenic stimuli such as high inspired oxygen pressures encountered in diving that could make a seizure (and drowning) more likely; active asthma is considered to increase risk because it could predispose to air trapping and pulmonary barotrauma (see below); and ischemic heart disease increases risk because it could prevent a diver from exercising sufficiently to get out of a difficult situation such as being caught in a current. It can be a complex matter to recognize the relevant interactions between diving and medical conditions and to determine their impact on

suitability for diving. There may follow an equally complex discussion about whether such interactions impart a disqualifying risk, and this may be influenced by the individual candidate's level of risk acceptance and the extent to which others involved (such as dive partners) might be affected. Guidelines are occasionally published on assessment of diving candidates with risk factors for important comorbidities like cardiovascular disease or who have suffered topical problems such as COVID-19 infection (see Sadler in "Further Reading"). Physicians interested in regularly conducting such evaluations

should obtain relevant training, for which short courses are offered by specialist groups in most countries. ■ ■BAROTRAUMA Barotrauma is essentially tissue injury arising as a result of ambient pressure changes. Middle-ear barotrauma (MEBT) in diving is similar to the problem that may occur during descent from altitude in an airplane, but difficulties with equalizing pressure in the middle ear are exaggerated underwater by both the rapidity and magnitude of pressure change as a diver descends or ascends. Failure to periodically insufflate the middle-ear spaces via the eustachian tubes during descent results in increasing pain. As the Pamb increases, the tympanic membrane may be bruised or even ruptured as it is pushed inward. Relative negative pressure in the middle ear results in engorgement of blood vessels in the surrounding mucous membranes and leads to effusion or bleeding, which can be associated with a conductive hearing loss (Chap. 36). MEBT is much less common during ascent because expanding gas in the middle-ear space tends to open the eustachian tube automatically. Barotrauma may also affect the respiratory sinuses, although the sinus ostia are usually widely patent and allow automatic pressure equalization without the need for specific maneuvers. If equalization fails, pain usually results in termination of the dive. Difficulty with equalizing ears or sinuses may respond to oral or nasal decongestants. Much less commonly, divers may suffer inner ear barotrauma (IEBT). Several explanations have been proposed, of which the most favored holds that forceful attempts to insufflate the middle-ear space by Valsalva maneuvers during descent result in transmission of pressure to the perilymph via the cochlear aqueduct and outward rupture of the round window, which is already under tension because of relative negative middle-ear pressure. The clinician should be alerted to possible IEBT after diving by a sensorineural hearing loss or true vertigo (which is often accompanied by nausea, vomiting, nystagmus, and ataxia). These manifestations can also occur in vestibulocochlear decompression sickness (DCS; see below) but should never be attributed to MEBT. Immediate review by an expert diving physician is recommended, and urgent referral to an otologist will often follow. The lungs are also vulnerable to barotrauma but are at most risk during ascent. If expanding gas becomes trapped in the lungs as Pamb falls, this may rupture alveoli and associated vascular tissue. Gas trapping may occur if divers intentionally or involuntarily hold their breath during ascent or if there are bullae. The extent to which asthma predisposes to pulmonary barotrauma is debated, but the presence of active bronchoconstriction must increase risk. For this reason, asthmatics who regularly require bronchodilator medications or whose airways are sensitive to exercise or cold air are usually discouraged from diving. While possible consequences of pulmonary barotrauma include pneumothorax and mediastinal emphysema, the most feared is the introduction of gas into the pulmonary veins leading to cerebral arterial gas embolism (CAGE). Manifestations of CAGE include loss of consciousness, confusion, hemiplegia, visual disturbances, and speech difficulties appearing immediately or within minutes after surfacing. The management is the same as for DCS described below. The natural history of CAGE often includes substantial or complete resolution of symptoms early after the event. This is probably the clinical correlate of bubble involution and redistribution with consequent restoration of blood flow. Patients exhibiting

such remissions should still be reviewed at specialist diving medical centers because secondary deterioration or re-embolization can occur. Unsurprisingly, these events can be misdiagnosed as typical strokes or transient ischemic attacks (TIAs) (Chap. 438) when patients are seen by clinicians unfamiliar with diving medicine. All patients presenting with neurologic symptoms after diving should have their symptoms discussed with a specialist in diving medicine and be considered for recompression therapy. ■ ■ DECOMPRESSION SICKNESS DCS is caused by the formation of bubbles from dissolved inert gas (usually nitrogen) during or after ascent (decompression) from a compressed gas dive. Bubble formation is also possible following decompression for extravehicular activity in space and with ascent to altitude

in unpressurized aircraft. DCS in the latter scenarios is probably rare in comparison with diving, where the incidence is ~1 in 5000–10,000 recreational dives. Breathing at elevated Pamb results in increased uptake of inert gas into blood and then into tissues. The rate at which tissue inert gas equilibrates with the inspired inert gas pressure is proportional to tissue blood flow and the blood-tissue partition coefficient for the gas. Similar factors dictate the kinetics of inert gas washout during ascent. If the rate of gas washout from tissues does not match the rate of decline in Pamb, then the sum of dissolved gas pressures in the tissue will exceed Pamb, a condition referred to as supersaturation. This is the prerequisite for bubbles to form during decompression, although other less well-understood factors are also involved. Deeper and longer dives result in greater inert gas absorption and greater likelihood of tissue supersaturation during ascent. Divers control their ascent for a given depth and time exposure using algorithms that often include periods where ascent is halted for a prescribed period at different depths to allow time for gas washout (decompression stops). Although a breach of these protocols increases the risk of DCS, adherence does not guarantee that it will be prevented. DCS should be considered in any diver manifesting postdive symptoms not readily explained by an alternative mechanism. Bubbles may form within tissues themselves, where they cause symptoms by mechanical distraction of pain-sensitive or functionally important structures. They also appear in the venous circulation, almost certainly after forming in capillary beds as blood passes through supersaturated tissues. Some venous bubbles are tolerated without symptoms and are filtered from the circulation in the pulmonary capillaries. However, in sufficiently large numbers, these bubbles are capable of inciting inflammatory and coagulation cascades, damaging endothelium, activating formed elements of blood such as platelets, and causing symptomatic pulmonary vascular obstruction. Circulating bubbles can induce endothelial leak, intravascular hypovolemia, and high hematocrit. Moreover, if there is a right-to-left shunt through an atrial septal defect, patent foramen ovale (PFO), or an intrapulmonary shunt, then venous bubbles may enter the arterial circulation (25% of adults have a PFO). The risk of cerebral, spinal cord, inner ear, and skin manifestations appears higher in the presence of significant shunts, suggesting that these “arterialized” venous bubbles can cause harm, perhaps by disrupting flow in the microcirculation of target organs. Circulating microparticles, which are elevated in number and size after diving, are currently under investigation as indicators of decompression stress and as injurious agents in their own right. How they arise and their exact role in DCS remain unclear. Table 476-2 lists manifestations of DCS grouped according to organ system. The majority of cases present with mild symptoms, including musculoskeletal pain, fatigue, and minor neurologic manifestations such as patchy paresthesias. Serious presentations are much less common. Pulmonary and cardiovascular manifestations can be life-threatening, and spinal cord involvement frequently results in permanent disability. Latency is variable. Serious DCS usually manifests within 60 min of surfacing, but mild symptoms may not appear for several hours.

Symptoms arising >24 h after diving are very unlikely to be DCS, unless the diver is exposed to reduced ambient pressure such as during commercial air travel or surface travel at higher altitude. The presentation may be confusing and nonspecific, and there are no useful diagnostic investigations. Diagnosis is based on integration of findings from examination of the dive profile, the nature and temporal relationship of symptoms to diving, and the clinical examination. Some DCS presentations may be difficult to separate from CAGE following pulmonary barotrauma, but from a clinical perspective, the distinction is unimportant because the first aid and definitive management of both conditions are the same. **TREATMENT Decompression Sickness** First aid for either DCS or CAGE includes horizontal positioning, in the lateral decubitus position if consciousness is impaired;

**TABLE 476-2 Manifestations of Decompression Sickness ORGAN SYSTEM MANIFESTATIONS**

Musculoskeletal	Limb pain	Neurologic	Cerebral Confusion	Visual disturbances	Speech disturbances
Spinal	Muscular weakness	Paralysis	Upper motor neuron signs	Bladder and sphincter dysfunction	Dermatomal sensory disturbances
Abdominal pain	Girdle pain	Vestibulocochlear	Hearing loss	Vertigo and ataxia	Nausea and vomiting
Peripheral	Patchy nondermatomal sensory disturbance	Pulmonary	Cough	Dyspnea	Cardiovascular
Hemoconcentration	Coagulopathy	Hypotension	Cutaneous Rash, itch	CHAPTER 476 Lymphatic	

Soft tissue edema, often relatively localized Constitutional Fatigue and malaise intravenous fluids (preferably glucose-free, isotonic intravenous, but if unavailable and the patient is sufficiently conscious, oral fluids); and sustained 100% oxygen administration. The latter accelerates inert gas washout from tissues and promotes resolution of bubbles. Definitive treatment of DCS or CAGE with recompression and hyperbaric oxygen is justified in most instances, although some mild or marginal DCS cases may be managed with first aid measures alone—an option that may be invoked by experienced diving physicians under various circumstances, but especially if evacuation for recompression is hazardous or extremely difficult. Long-distance evacuations are preferably undertaken using a helicopter flying at low altitude or a fixed-wing air ambulance pressurized to 1 ATA, although for serious cases, evacuation should not be delayed only because normobaric evacuation is not possible. Breathing oxygen en route is recommended. If oxygen toxicity is a concern during prolonged evacuation, expert opinion can provide guidance as to a schedule (see below). **Hyperbaric and Diving Medicine** Recompression reduces bubble volume in accordance with Boyle's law and increases the inert gas partial pressure difference between a bubble and surrounding tissue. At the same time, oxygen administration markedly increases the inert gas partial pressure difference between alveoli and tissue. The net effect is to significantly increase the rate of inert gas diffusion from bubble to tissue and tissue to blood, thus accelerating bubble resolution. Hyperbaric oxygen also helps oxygenate compromised tissues and may ameliorate some of the proinflammatory effects of bubbles. Various recompression protocols have been advocated, but there are no data that define the optimum approach. Recompression typically begins with oxygen administered at 2.8 ATA, the maximum pressure at which the risk of oxygen toxicity remains acceptable in a hyperbaric chamber. There follows a stepwise decompression over variable periods adjusted to symptom response. The most widely used algorithm is the U.S. Navy Table 6, whose shortest format lasts 4 h and 45 min. Typically, shorter follow-up recompressions are repeated daily while symptoms persist and appear responsive to treatment. Adjuncts to recompression include intravenous fluids and other supportive care

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