Hyperbaric oxygen therapy

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Literature review current through: **Jan 2024.**
This topic last updated: **Dec 11, 2023.**

INTRODUCTION

Hyperbaric oxygen (HBO) serves as primary or adjunctive therapy for a diverse range of medical conditions (table 1) [1-5]. In the United States, there are several thousand hospital-based and freestanding hyperbaric facilities that offer either single-occupant ("monoplace") or multiple-occupant ("multiplace") chambers. Information regarding the location of hyperbaric facilities can be accessed through the Undersea & Hyperbaric Medical Society website (www.uhms.org) or via the Divers Alert Network Emergency Hotline (United States phone number: 1 (919)-684-9111).

The mechanisms of action, administration, risks, and outcomes of HBO therapy for its currently accepted indications will be reviewed here. Smoke inhalation, carbon monoxide poisoning, and diving complications are discussed separately in greater detail. (See "Carbon monoxide poisoning" and "Complications of SCUBA diving" and "Inhalation injury from heat, smoke, or chemical irritants".)

MECHANISMS OF ACTION

Most of the benefits of hyperbaric oxygen (HBO) are explained by the simple physical relationships determining gas concentration, volume, and pressure [6]. HBO is most commonly

used under conditions of tissue hypoxia or to treat decompression sickness or gas embolism, in which gas bubbles obstruct blood flow.

Increased oxygen delivery — Henry's Law states that the amount of an ideal gas dissolved in solution is directly proportional to its partial pressure. Thus, the dissolved plasma oxygen concentration of 0.3 mL/dL at sea level (1.0 atm) increases to 1.5 mL/dL upon administration of 100 percent oxygen, while hyperbaric oxygen delivered at 3.0 atm yields a dissolved oxygen content of 6 mL/dL. The latter figure is sufficient to meet resting tissue oxygen extraction requirements irrespective of the adequacy of the hemoglobin-bound oxygen pool. The ability of HBO to augment oxygen content and independently meet resting tissue oxygen requirements has led to its use in conditions of compromised oxygen delivery, such as profound anemia, carbon monoxide (CO) poisoning, and both acute and chronic ischemia [2,7-10].

Reduction of gas bubble size — The use of hyperbaric oxygen therapy for decompression illness is based upon Boyle's Law, since the volume of nitrogen bubbles is inversely related to the pressure exerted upon it. At 3.0 atm, bubble volume decreases by approximately two-thirds. Further bubble dissolution is accomplished by the replacement of inert nitrogen within the bubbles with oxygen, which is then rapidly metabolized by tissues [2].

Antagonism of carbon monoxide — Carbon monoxide (CO) binds to hemoglobin with an affinity 200 to 250 times as great as that of oxygen (see "Carbon monoxide poisoning"). The presence of carboxyhemoglobin (COHb) results in a leftward shift and hyperbolic shape of the oxyhemoglobin dissociation curve, corresponding to a marked decrease in oxygen-carrying capacity and in tissue oxygen release (figure 1). The four to six hour half-life of COHb at ambient air decreases to 40 to 80 minutes with 100 percent normobaric oxygen (NBO). With HBO therapy, the half-life of COHb decreases further to 15 to 30 minutes [11,12].

Improved wound healing — In vitro, HBO modulates local and systemic effects found in both acute and chronic injury, ischemia, and inflammation [13-15]. Many of its effects are mediated by reactive oxygen species (eg, oxygen-derived free radicals, hydrogen peroxide, hypochlorous acid) and reactive nitrogen species (eg, nitric oxide), which are generated in tissue [16]. Local hyperoxia induces vasoconstriction and reduces vasogenic edema following acute trauma [13]. HBO ameliorates ischemia-reperfusion-induced leukocyte influx [14]. By altering conditions of local hypoxia, HBO facilitates fibroblast proliferation, angiogenesis, and wound healing [7,17,18]. HBO augments neutrophil bactericidal activity, limits clostridial exotoxin and spore production, kills anaerobes such as Clostridium perfringens, and inhibits the growth of several other bacterial pathogens [18-21].

TECHNIQUE

Multiplace chambers allow closer monitoring of critically ill patients, while single occupancy chambers are most appropriate for the treatment of chronic medical conditions in stable patients [2]. Chamber pressure is usually maintained between 2.5 and 3.0 atm, with treatment lasting 45 to 300 minutes depending upon the indication. Acute therapy may require only one or two treatments, while chronic medical conditions may warrant up to 30 or more sessions. Typically, hyperbaric therapy is administered with pressurized oxygen or air. Pressures exceeding 2.8 to 3.0 atm, particularly over prolonged exposure hyperbaric periods, dramatically increase the risk of both neurologic and pulmonary oxygen toxicity. Helium/oxygen (heliox) or nitrogen/oxygen (nitrox) mixtures are indicated only in certain instances of decompression illness [22,23]. (See "Adverse effects of supplemental oxygen".)

CONTRAINDICATIONS

The only absolute contraindication to HBO therapy is untreated pneumothorax. Relative contraindications include obstructive lung disease, asymptomatic pulmonary blebs or bullae on chest radiograph, upper respiratory or sinus infections, recent ear or thoracic surgery, uncontrolled fever, and claustrophobia [24]. However, these relative contraindications, including severe obstructive lung disease, should not deter clinicians from using HBO to treat patients with severe neurological injuries or other life or limb-threatening conditions. Pregnancy was once believed to represent a contraindication to hyperbaric oxygen (HBO), but now is considered an impetus to pursue HBO therapy among patients with carbon monoxide (CO) intoxication [12]. (See "Carbon monoxide poisoning".)

Patients with a history of a seizure disorder are at risk for complications related to central nervous system (CNS) toxicity from high oxygen concentrations [6,18]. The incidence of CNS complications in normal risk patients is small and should not dissuade clinicians from using HBO therapy for appropriate indications. However, the risk in patients who are at increased baseline risk (eg, history of seizures or recent brain surgery) is unknown. The decision to use HBO in these patients must be made on a case by case basis after careful consideration of the potential (but manageable) risks and benefits, and the patient's values and preferences. (See 'Complications' below.)

The adverse effects of several medications are thought to be exacerbated by HBO therapy, but evidence of such effects is scant. As an example, the administration of supplemental oxygen to patients who have been treated with bleomycin has been associated with pulmonary toxicity.

However, a review of 15 bleomycin-exposed patients who underwent HBO therapy reported that while bleomycin and HBO may independently cause pulmonary toxicity, a synergistic effect was not found [25]. Concurrent treatment with doxorubicin has also been considered a contraindication to HBO therapy because of the concern that oxidative stress may worsen doxorubicin-induced cardiotoxicity. However, in a rat model, hyperbaric oxygen exposure was found to reduce cardiac systolic dysfunction and the histopathologic changes associated with doxorubicin [26]. Given the dearth of data in humans, the decision to use HBO in patients being treated with bleomycin or doxorubicin must be made on a case by case basis after careful consideration of the potential risks and benefits, and the patient's values and preferences.

COMPLICATIONS

Hyperbaric therapy is generally safe and well tolerated. Most side effects are mild and reversible, although severe consequences can occur in rare cases [6].

- Middle ear barotrauma is the most common side effect of hyperbaric oxygen, with an incidence of approximately 2 percent [6]. It is more common in patients undergoing multiple treatments [27]. (See "Ear barotrauma".) Middle ear effusions (which may be hemorrhagic) and tympanic membrane rupture occur infrequently. Middle ear symptoms preclude repeat therapy in less than 1 percent of patients; they may be alleviated by teaching the patient autoinflation techniques or by the placement of tympanostomy tubes [6]. (See "Acute otitis media in adults".)

- Sinus barotrauma (sinus squeeze) is the second most common complication of hyperbaric oxygen and is usually seen in patients with upper respiratory tract infections or allergic rhinitis. It may be alleviated by pretreatment with nasal decongestants, steroids, or antihistamines [6].

- Reversible myopia due to direct oxygen toxicity to the lens is seen in some patients. While its etiology is unclear, it usually resolves within days to weeks after the last treatment [6].

- Pulmonary barotrauma is unusual, provided any pneumothoraces have been identified and decompressed before initiating HBO [2]. Pulmonary edema has been reported rarely in patients undergoing HBO therapy for CO poisoning [28].

- Pulmonary oxygen toxicity, manifested by chest tightness, cough, and a reversible decline of pulmonary function, occurs most commonly in patients receiving multiple treatments or previously exposed to high oxygen levels [2]. (See "Adverse effects of supplemental oxygen".)

- Seizures due to central nervous system oxygen toxicity are a rare but dramatic consequence of HBO treatment. An analysis of 62,614 HBO sessions involving 2334 patients identified only one patient with a seizure that was clearly related to oxygen toxicity [29]. The risk is increased by HBO exposure greater than 90 to 120 minutes and by pressures greater than 2.8 to 3.0 atm. Patients receiving glucocorticoids, insulin, thyroid replacement, and sympathomimetic medications may be at higher risk of central nervous system oxygen toxicity. HBO has been associated with hypoglycemia in some patients with diabetes, and hypoglycemia should therefore be considered in the differential diagnosis of HBO-associated seizures [18].

  Seizures due to oxygen toxicity do not typically result in permanent structural brain damage [6]. However, a case report describes a patient undergoing HBO therapy who experienced a seizure followed by an ischemic stroke. The authors speculated that seizure-induced demand ischemia may have caused the stroke [30].

  Seizures should be managed acutely by reducing the inspired oxygen concentration to that of air (FIO2 = 0.21), administering anticonvulsant therapy, and if necessary, terminating hyperbaric treatment. Oxygen toxicity may be prevented by alternating between short (five minute) intervals of air and longer intervals (30 minutes) of 100 percent oxygen, to limit oxygen free radical formation [2]. Patients in monoplace chambers should have therapy terminated, as airway management and monitoring are compromised. The need for further treatment with lower FIO2 should be addressed. (See "Convulsive status epilepticus in adults: Management", section on 'Emergency antiseizure treatment'.)

- Decompression sickness may occur in patients breathing compressed air that contains nitrogen. The likelihood of decompression sickness is reduced by administration of 100 percent oxygen toward the end of the treatment period and by adherence to standard United States Navy guidelines governing gradual decompression [18]. Decompression sickness does **not** occur in patients breathing 100 percent oxygen.

INDICATIONS FOR CLINICAL USE

Hyperbaric oxygen (HBO) serves as primary or adjunctive therapy for a diverse range of medical conditions (table 1) [2-4,31].

Carbon monoxide or cyanide poisoning — Carbon monoxide (CO) poisoning is a leading cause of death from poisoning. HBO reduces the half-life of carboxyhemoglobin, an observation

that has led to the study of HBO for preventing the late neurocognitive deficits associated with severe CO poisoning. The role of HBO in the treatment of CO and cyanide poisoning is discussed separately. (See "Carbon monoxide poisoning", section on 'Hyperbaric oxygen therapy' and "Cyanide poisoning", section on 'Adjunct treatment with hyperbaric oxygen'.)

Air embolism

Decompression sickness — Self-contained underwater breathing apparatus (SCUBA) divers breathing compressed air who return to the surface too rapidly, and aviators ascending over 5500 meters are at risk for decompression sickness and arterial gas embolism. Those with a patent foramen ovale are at increased risk [32]. Bubble formation in tissues or in blood occurs as the partial pressure of inert gas (mostly nitrogen) exceeds that of ambient air. The obstruction of vessels and lymphatics by bubbles is accompanied by the activation of leukocytes, endothelial damage, and resultant alterations in capillary permeability [22]. Uncontrolled ascent while diving may cause arterial and venous gas embolism from pulmonary overinflation with subsequent alveolar rupture.

Decompression sickness manifests a range of severity from self-limited rash or joint pain to focal neurologic deficits, paralysis, seizures, hypovolemic shock, and death (table 2). Patients typically develop symptoms within several hours of ascent, with rapid onset and severe symptoms implying a more ominous course. (See "Complications of SCUBA diving", section on 'Decompression sickness'.)

HBO is the primary treatment for decompression sickness and arterial gas embolism from SCUBA diving [33,34]. It is unclear whether the efficacy of HBO is due primarily to decreased bubble size and relief of local hypoxia, or to modulation of the pathologic effects mediated by bubbles in tissue and in vessels [2]. Patients should initiate HBO treatment as soon as possible, because a sharp decrease in the successful treatment of cerebral air emboli has been noted after a four to five hour delay [35,36]. (See "Complications of SCUBA diving", section on 'Hyperbaric oxygen therapy'.)

Treatment consists of one or more sessions at 2.5 to 3.0 atm. Most patients respond to one treatment. If symptoms persist, additional sessions are indicated until clinical resolution. Some patients with neurologic symptoms suffer deterioration after seemingly successful recompression treatment. This phenomenon may be due to the slow re-expansion of residual gas bubbles upon termination of hyperbaric therapy, post-ischemic reperfusion injury, or re-embolization from an underlying pulmonary abnormality. In such patients with severe deficits, as many as 20 sessions may be required [34].

Resources to help clinicians with diagnosis and management are available. (See "Complications of SCUBA diving", section on 'Additional resources'.)

Adjunctive treatment with glucocorticoids, lidocaine, or a combination of prostacyclin, indomethacin, and heparin has been reported, but the efficacy of these interventions is unclear [37,38]. (See "Complications of SCUBA diving", section on 'Adjunctive treatments'.)

Hydrogen peroxide exposure — Hydrogen peroxide poisoning can cause air-gas embolism with potentially life-threatening toxicity; HBO may benefit selected patients, which is discussed in detail separately. (See "Household nonpharmaceutical product ingestions", section on 'Hydrogen peroxide'.)

Others — HBO is not routinely administered in patients with air embolism but is a useful adjunct in severe cases. Venous and arterial emboli may develop from air directly introduced through a variety of means, including central venous catheter placement; cardiac, neurologic, and otolaryngological surgery; or from pulmonary overinflation with subsequent alveolar rupture from blast injury or mechanical ventilation [39]. (See "Air embolism".)

Acute traumatic or thermal injury — HBO has been recommended as adjunctive therapy for a range of acute traumatic and ischemic syndromes, including crush injuries, compartment syndromes, and situations of vascular compromise [7]. Animal models of ischemia and compartment syndromes suggest that the benefit of HBO may be due to a combination of increased tissue oxygenation, reduced edema through hyperoxia-induced vasospasm, protection from reperfusion injury and secondary ischemia, and antimicrobial effects [7,13,40-42].

The potential efficacy of this approach was evaluated in a trial in which 36 patients with crush injuries were randomly assigned to either a 90 minute twice daily HBO treatment or a 90 minute twice daily sham treatment consisting of NBO administered within a hyperbaric chamber for a total of six days and begun within 24 hours of surgery [43]. The group treated with HBO had significantly more complete healing (17 versus 10 patients) and required fewer skin flaps, grafts, vascular surgery, or amputation (1 versus 6 patients).

In contrast, a randomized prospective trial evaluating adjunctive HBO in 125 burn victims failed to demonstrate any benefit in mortality, length of stay, or the extent of autografting [44]. A subsequent systematic review found only two high quality trials designed to assess this question, including the one described above, and concluded that there was insufficient evidence to support the use of HBO following thermal injury [45].

Radiation injury — Previously irradiated tissue is characterized by fibro-atrophic changes, with decreased vascularity, impaired cellular proliferation, and local hypoxia that can persist long after radiation therapy. Subsequent injury (eg, dental extraction) or surgical manipulation may lead to soft tissue radionecrosis and osteoradionecrosis, manifested by edema, ulceration, poor wound healing, and infection [17,18,46]. The value of HBO has been studied in patients with laryngeal, oropharyngeal, and other head and neck cancers who develop osteoradionecrosis following radiation therapy. In theory, HBO has the potential to improve this condition because of its impact on collagen synthesis and vascular density. (See "Management of late complications of head and neck cancer and its treatment", section on 'Osteoradionecrosis and soft tissue necrosis'.)

Unfortunately, the available data are conflicting, and the benefit of HBO to prevent or treat established osteoradionecrosis of the jaw in irradiated patients with head and neck cancer is uncertain [47]. (See "Management of late complications of head and neck cancer and its treatment", section on 'Osteoradionecrosis and soft tissue necrosis'.)

Several studies suggest that HBO may reduce soft tissue radionecrosis and improve reconstructive outcome in patients who have received chest, pelvic, perineal, or extremity irradiation [48-56]. Furthermore, a Cochrane Collaboration review suggested that HBO may be of benefit for patients with late radiation tissue injury to the head, neck, bladder, anus, and rectum [57]. There was no benefit for neural tissue; however, randomized trials have not been conducted, and questions persist regarding the ultimate benefit of this approach.

Protocols for prevention and treatment of osteoradionecrosis and soft tissue radionecrosis generally have included 20 to 30 preoperative HBO sessions at 2.4 atm delivered over 90 minutes, followed by 10 postoperative treatments [18].

Infection — The primary treatment regimen for aggressive soft tissue infections such as clostridial myonecrosis (gas gangrene), necrotizing fasciitis, and Fournier's gangrene consists of antibiotic therapy and aggressive debridement. A number of retrospective and observational studies have evaluated the role of HBO as an adjunctive therapy for these and other types of infections [58-63].

Several mechanisms of action have been proposed, which in part parallel those explaining HBO's benefit in wound healing. The first involves the generation of reactive oxygen species (ROS). The resultant oxygen stress can have both bacteriostatic or bactericidal effects, particularly on anaerobic bacteria. A second mechanism involves HBO's suppression of the production of cytokines and other inflammatory mediators. While the details have not been fully elucidated, this appears to have an antimicrobial effect and to promote wound healing.

Finally, hyperoxia produced by HBO increases the bactericidal activity of certain antibiotics, including beta-lactams, aminoglycosides, and quinolones [64].

Although no randomized, controlled trials in humans have been conducted, a rigorous study evaluating combinations of antibiotics, surgery, and HBO for clostridial infection in a dog model has demonstrated a 35 percent survival increase with adjunctive HBO [65]. In patients with clostridial myonecrosis, HBO may reduce mortality and better define necrotic tissue, facilitating more precise amputation and debridement [18]. (See "Clostridial myonecrosis".)

Adjunctive HBO may decrease mortality and limit the extent of debridement in Fournier's gangrene [59,60] and necrotizing fasciitis [61-63,66]. However, high-quality data are limited due to a lack of randomized trials [67]. A retrospective review using a nationwide database (United States Nationwide Inpatient Sample) identified 45,913 cases of necrotizing soft tissue infection and reported a statistically significant reduction in mortality among those patients (n = 405) treated with HBO (4.5 versus 9.4 percent) [66]. This advantage persisted after controlling for other predictors of mortality and possible confounders (odds ratio [OR] 0.49; 95% CI 0.29-0.83).

Smaller studies, also retrospective, have reported conflicting results. One observational study of 26 patients with Fournier's gangrene found a significantly lower mortality rate among HBO-treated patients (7 versus 42 percent) [60]. A second observational study of 29 patients with necrotizing fasciitis noted significantly fewer debridements and a lower mortality rate (23 versus 66 percent) when HBO was employed [63]. In contrast, a subsequent study of 42 patients with Fournier's gangrene suggested increased mortality, morbidity, and cost of therapy among patients treated with HBO [68].

HBO has been advocated for use in other severe invasive infections such as cutaneous soft-tissue and rhinocerebral mucormycosis (or zygomycosis) and actinomycotic brain abscesses, although data in support of these indications are less robust [21,69]. HBO also has been advocated for therapy of refractory osteomyelitis, although most supportive evidence comes from cohort and case studies [70]. (See "Necrotizing soft tissue infections".)

When used to treat acute infections, HBO should be implemented early, with two to three daily 90 minute HBO sessions at 3 atm. These high pressures maintain tissue oxygen tension above 300 mmHg (sufficient to inhibit clostridial spore and exotoxin production) [18].

Nonhealing ulcers, skin grafts, and wound healing — HBO has been used as an adjunct therapy in the treatment of nonhealing wounds, compromised skin grafts, and other wounds. The use of HBO for these indications is reviewed separately. (See "Basic principles of wound management", section on 'Hyperbaric oxygen therapy'.)

Sensorineural hearing loss — Small studies suggest that HBO therapy improves hearing in patients with idiopathic sensorineural hearing loss, including those who have failed a trial of steroids [71,72]. (See "Sudden sensorineural hearing loss in adults: Evaluation and management", section on 'Patients refractory to initial therapy'.)

Frostbite — Some experts use HBO as adjunctive therapy for treating frostbite injuries, but we do not recommend routine use. Evidence supporting benefit is based on observational studies, case reports, and physiologic studies [73-80]. HBO can mitigate ischemia-reperfusion insult (ie, inflammatory cascades, production of chemotactic compounds) and induce micro-angiogenesis, although these benefits have not been demonstrated specifically for frostbite [6,13,81]. (See "Frostbite: Emergency care and prevention".)

Future directions — A number of potential HBO uses remain poorly validated and require more rigorous evaluation. Future indications for HBO may be derived from its apparent modulation of ischemia-reperfusion injury and inflammation. Preliminary animal and human studies evaluating uses in syndromes as disparate as acute coronary syndrome, including myocardial infarction, the systemic inflammatory response syndrome, traumatic brain or spinal cord injury, sickle cell crisis, fibromyalgia, and acute stroke have been conducted, with variable results [1,15,82-89]. Further investigation will need to be conducted before HBO can be endorsed for these potential indications.

HBO is being investigated for reversal of acute hypoxemia from coronavirus disease 2019 (COVID-19) infection and for treatment of persistent neurocognitive and cardiac symptoms ("long COVID") [90-96]. A proposed mechanism of action relates to HBO's anti-inflammatory effect, which may lessen the organ damage caused by the excessive immune response to COVID.

SUMMARY AND RECOMMENDATIONS

- **Indications for clinical use** – Hyperbaric oxygen (HBO) is a reasonable therapy in the following circumstances:
  - Arterial gas embolism and/or decompression sickness from SCUBA diving. The evidence and use are presented above and separately. (See 'Decompression sickness' above and "Complications of SCUBA diving", section on 'Hyperbaric oxygen therapy'.)
  - Significant cases of carbon monoxide poisoning or hydrogen peroxide exposure. The indications and evidence are presented separately. (See "Carbon monoxide poisoning",

section on 'Hyperbaric oxygen therapy' and "Household nonpharmaceutical product ingestions", section on 'Hydrogen peroxide'.)

- **Possible additional indications for clinical use** – HBO may be useful in the care of patients with severe anemia, actinomycotic brain abscesses, acute crush injuries, prior radiation therapy, aggressive soft tissue infections, nonhealing ulcers, sensorineural hearing loss, cyanide poisoning, or compromised skin grafts and flaps (table 1). Further research is required in these situations to confirm the benefits of HBO and justify its significant costs and potential risks. (See 'Indications for clinical use' above.)

- **Contraindications** – Untreated pneumothorax is the sole absolute contraindication to HBO therapy. Relative contraindications include obstructive lung disease, asymptomatic pulmonary blebs or bullae on chest radiograph, upper respiratory or sinus infections, recent ear or thoracic surgery, and uncontrolled fever. (See 'Contraindications' above.)

- **Complications** – Possible complications of HBO therapy include middle ear barotrauma (most common), sinus barotrauma, reversible myopia, pulmonary barotrauma, pulmonary oxygen toxicity, seizures (rare), and decompression sickness. (See 'Complications' above.)

- **Mechanism of action** – A basic description of the mechanisms of HBO is provided in the text. Fundamentally, HBO works by providing oxygen at high atmospheres of pressure, thereby increasing the concentrations of oxygen delivered to tissues. (See 'Mechanisms of action' above.)

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Topic 326 Version 31.0

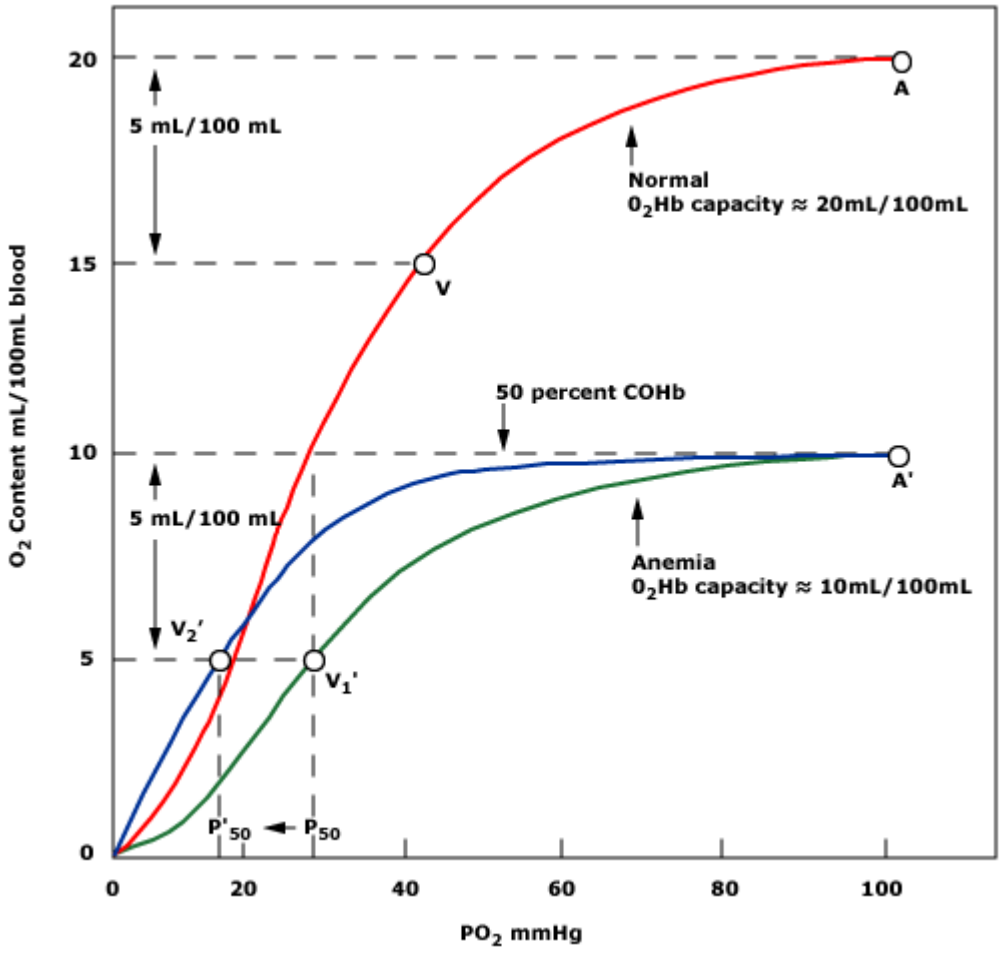
GRAPHICS

Therapeutic roles for hyperbaric oxygen

Air or gas embolism
Carbon monoxide poisoning
Decompression sickness
Hydrogen peroxide poisoning
Clostridial myonecrosis
Crush injury and other forms of traumatic ischemia
Enhanced healing of problematic wounds, including diabetic wounds
Severe anemia
Actinomycotic brain abscess
Necrotizing soft tissue infections
Refractory osteomyelitis
Radiation necrosis of soft tissue and bone
Compromised skin grafts and flaps

Graphic 75304 Version 3.0

Effect of carboxyhemoglobinemia on oxygen content and delivery



Red curve demonstrates the normal relationship between arterial (A) and venous (V) oxygen content. In general, a difference in arterial and venous oxygen content of 5 mL per 100 mL blood is expected at rest. The green line depicts the effect of a 50 percent decrease in hemoglobin concentration, which decreases arterial (A') and venous (V'1) oxygen content but does not change the partial pressure at which hemoglobin is 50 percent saturated (P_{50}). The blue curve shows the effect of a 50 percent carboxyhemoglobinemia (COHb), which both decreases oxygen carrying capacity (A') and impairs peripheral unloading of oxygen from hemoglobin under conditions of low oxygen tension (V'2), shifting P_{50} to the left (P'_{50}). These changes result in a profound reduction in both oxyhemoglobin saturation and delivery of oxygen to peripheral tissues.

Graphic 50058 Version 2.0

Clinical manifestations of decompression sickness (DCS) according to organ system*

Affected organ system and manifestations	% of DCS cases	Description
Musculoskeletal: pain	50-65	Often described as a deep, boring ache in shoulder, elbow, hip, or knee area, unaffected by joint movement, usually without local tenderness; may be multifocal and poorly localized
Cutaneous		
Rash and itch	5-10	An erythematous, poorly demarcated patch, often itchy, or a more clearly circumscribed, reticular rash with cyanotic discoloration (cutis marmorata or livedo racemosa); usually a truncal or proximal distribution
Patchy paresthesia	40-50	Nondermatomal distribution; often described as tingling
Lymphatic: subcutaneous swelling	1-5	Truncal distribution, similar to that of rash, particularly involving upper chest and shoulders
Spinal cord		
Motor weakness	20-25	Typically, paraplegia or quadriplegia witl upper motor neuron signs; severity ranges from subtle to dense
Numbness or dense paresthesia	20-30	Dorsal columns appear to be vulnerable proprioception may be affected
Bladder and sphincter dystonia	1	Bladder also becomes insensate; urinary retention
Girdle, chest, back, or abdominal pain	1-5	Typically, a premonitory symptom that precedes other spinal symptoms
Inner ear		
Vestibular: vertigo, ataxia	10-20	Usually accompanied by nausea and nystagmus; 75% of inner ear DCS cases have no other symptoms
Cochlear: hearing loss, tinnitus	1-5	Cochlear manifestations are less common than vestibular manifestations in DCS

Brain (cerebral): cognitive impairment, scotomata, visual field changes, focal weakness, ataxia	5-10	Typically, mild executive dysfunction (eg, impaired concentration or memory); gross focal lesions considered to be less common in DCS than in AGE
Cardiopulmonary: dyspnea, cough, chest pain ("the chokes")	1-5	Typically associated with provocative events such as rapid ascent or omitted decompression after deep dives
Cardiovascular: hemoconcentration, shock, coagulopathy	<1	Rare; typically associated with provocative events such as rapid ascent or omitted decompression after deep dives
Constitutional, including fatigue, malaise, headache	20-40	Often described as similar to a viral infection

AGE: arterial gas embolism.

* Approximate proportions of divers affected are based on data from various sources.[1-3]

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Graphic 139053 Version 1.0