

# How Can Hyperbaric Oxygen Contribute to Treatment?

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**In Brief:** Hyperbaric oxygen (HBO<sub>2</sub>) is used in a sports medicine setting to reduce hypoxia and edema and appears to be particularly effective for treating crush injuries and acute traumatic peripheral ischemias. When used clinically, HBO<sub>2</sub> should be considered as an adjunctive therapy as soon as possible after injury diagnosis. Treatment pressures for acute traumatic peripheral ischemia range from 2.0 to 2.5 atmospheres absolute (ATA), with a minimum of 90 minutes for each treatment. Some professional and amateur athletes use HBO<sub>2</sub> to aid endurance performance or to speed recovery from exercise-related fatigue; however, research does not yet support these uses. Clinicians and athletes should keep in mind that HBO<sub>2</sub> is a medical treatment with associated risks.

**I**n recent years, professional and college teams have started using hyperbaric oxygen therapy (HBO<sub>2</sub>) to treat sports injuries. From muscle contusions and ankle sprains to delayed-onset muscle soreness, HBO<sub>2</sub> has been used to facilitate soft-tissue healing (1-7). To minimize the time between injury and HBO<sub>2</sub> treatment, some professional sports teams have on-site centers. Because of the importance of oxygen in the aerobic energy system, many athletes and researchers have also investigated the possible ergogenic effects of HBO<sub>2</sub>.

During HBO<sub>2</sub> treatment, a patient breathes 95% to 100% oxygen at pressures above 1.0 atmosphere absolute (ATA). In America, HBO<sub>2</sub> chambers were first used as a treatment for deep-sea divers who experienced decompression sickness. Modern HBO<sub>2</sub> treatment evolved from these medical activities (8,9). The Undersea and Hyperbaric Medical Society (UHMS) has evaluated the use and effectiveness of HBO<sub>2</sub> for different medical conditions. The UHMS regularly reviews the applications of HBO<sub>2</sub> and lists the diseases and conditions that are supported by scientific information. Currently, the UHMS approves HBO<sub>2</sub> for 13 medical conditions (table 1) (8).

## **TABLE 1. Approved Indications for Hyperbaric Oxygen Therapy**

Air or gas embolism  
Carbon monoxide poisoning  
Clostridial myositis and myonecrosis (gas gangrene)  
Crush injury, compartment syndrome, and acute traumatic ischemia  
Decompression sickness  
Enhancement of healing in selected problem wounds  
Exceptional blood loss (anemia)  
Intracranial abscess  
Necrotizing soft-tissue infections  
Osteomyelitis (refractory)  
Radiation injury (delayed)  
Skin grafts and flaps (compromised)  
Thermal burns

### **Hyperbaric Oxygen Chambers**

There are two general oxygen chamber designs. Multiplace chambers allow two or more people to be treated simultaneously. The large space allows the patients to be accompanied by a healthcare professional or technician. Multiplace chambers are usually metallic and are always pressurized with air. Patients receive oxygen via an oronasal mask, head tent, or endotracheal tube. Chambers require significant monetary and staff resources to operate. Monoplace chambers, on the other hand, are designed to treat a single patient. They are smaller, pressurized with either oxygen or air, and are less expensive to operate than a multiplace chamber. Most monoplace chambers are constructed from clear acrylic plastic that allows easy visualization of the patient (figure 1).

### **Principles of HBO<sub>2</sub>**

Normally, 97% of the oxygen delivered to body tissues is bound to hemoglobin, while only 3% is dissolved in the plasma. At sea level, barometric pressure is 1 ATA, or 760 mm Hg, and the partial pressure of oxygen in arterial blood (PaO<sub>2</sub>) is approximately 100 mm Hg. At rest, the tissues of the body consume about 5 mL of O<sub>2</sub> per 100 mL of blood.

During HBO<sub>2</sub> treatments, barometric pressures are usually limited to 3 ATA or lower. The oxygen content of inspired air in the chamber is typically 95% to 100%. The combination of increased pressure (3 ATA) and increased oxygen concentration (100%) dissolves enough oxygen in the plasma alone to sustain life in a resting state. Under hyperbaric

conditions, oxygen content in the plasma is increased from 0.3 to 6.6 mL per 100 mL of blood with no change in oxygen transport via hemoglobin. HBO<sub>2</sub> at 3.0 ATA increases oxygen delivery to the tissues from 20.0 to 26.7 mL of O<sub>2</sub> per 100 mL of blood.

### **Proposed Healing Mechanisms**

Increased oxygen delivery to the tissues is believed to facilitate healing through a number of mechanisms.

**Vasoconstriction.** High tissue oxygen concentrations cause blood vessels to constrict, which can lead to a 20% decrease in regional blood flow (10). In normoxic environments, tissue hypoxia may develop; however, this is not the case with HBO<sub>2</sub>. The decrease in regional blood flow is more than compensated for by the increased plasma oxygen that reaches the tissue. The net effect is decreased tissue inflammation without hypoxia—a mechanism by which hyperbaric oxygen therapy is believed to improve crush injuries, thermal burns, and compartment syndrome (11,12).

**Neovascularization and epithelialization.** High tissue oxygen concentrations accelerate the development of new blood vessels (12). This can be induced in both acute and chronic injuries. Regenerating epithelial cells also function more effectively in a high-oxygen environment (13). These effects have proven effective in treating tissue ulcers and skin grafts (14).

**Stimulation of fibroblasts and osteoclasts.** In a hypoxic milieu, fibroblasts are unable to synthesize collagen, and osteoclasts are unable to lay down new bone (7,14,15). Collagen deposition, wound strength, and the rate of wound healing are affected by the amount of available oxygen. Ischemic areas of wounds benefit most from the increased delivery of oxygen (16). HBO<sub>2</sub> increases tissue levels of oxygen, allowing for fibroblasts and osteoclasts to function appropriately (13,17). This mechanism may play a role in the treatment of osteomyelitis and slowly healing fractures.

**Immune response.** When tissue oxygen tensions fall below 30 mm Hg, host responses to infection and ischemia are compromised (18). Studies have shown that the local tissue resistance to infection is directly related to the level of oxygen found in the tissue (19,20). High oxygen concentrations may prevent the production of certain bacterial toxins and may kill certain anaerobic organisms such as *Clostridium perfringens*. More important, however, oxygen aids polymorphonuclear leukocytes (PMN). Oxygen is believed to aid the migration and phagocytic function of the PMN (21). Oxygen is converted within the PMN into toxic substrates (superoxides, peroxides, and hydroxyl radicals) that are lethal to bacteria (16,22). These effects on the immune system allow HBO<sub>2</sub> to aid the healing of soft-tissue infections and osteomyelitis (21).

HBO2 has also been found to inhibit PMN adherence on postcapillary venules (23). Although this may seem paradoxical, this effect is beneficial because it helps limit reperfusion injury after crush injury and compartment syndrome.

**Maintaining high-energy phosphate bonds.** When circulation to a wound is compromised, resultant ischemia lowers the concentration of adenosine triphosphate (ATP) and increases lactic acid levels. ATP is necessary for ion and molecular transport across cell membranes and maintenance of cellular viability (24,25). Increased oxygen delivery to the tissue with HBO2 may prevent tissue damage by decreasing the tissue lactic acid level and helping maintain the ATP level. This may help prevent tissue damage in ischemic wounds and reperfusion injuries.

### **Which Sports Injuries Respond to HBO2?**

HBO2 is an effective treatment for crush injuries and other acute traumatic peripheral ischemias because it alleviates hypoxia and reduces edema; however, clinical experience with HBO2 for sports injuries is limited. Also, the criteria for using HBO2 in acute traumatic peripheral ischemias are not clearly established. HBO2 should be considered as an adjunctive therapy as soon as possible after injury diagnosis. Treatment pressures for acute traumatic peripheral ischemia range from 2.0 to 2.5 ATA, with a minimum of 90 minutes for each treatment (26).

HBO2 has been used to treat joint, muscle, ligament, and tendon injuries in soccer players in Scotland. When HBO2 was used in conjunction with physiotherapy, the time to recovery was reduced by 70% (27). The results compared a physiotherapist's estimation of the time course for the injury and the actual number of training days missed. The absence of a control group and objective measures to assess the injury weaken the encouraging findings in this study. HBO2 has been used to treat acute ankle injuries. Borromeo et al (1) conducted a randomized double-blind study of 32 patients who had acute ankle sprains to compare HBO2 treatment at 2.0 ATA with a placebo treatment. Each group received three treatments: one for 90 minutes and two for 60 minutes. The improvement in joint function was greater in the HBO2 group compared with the placebo group. There were no statistically significant differences between the groups when assessed for subjective pain, edema, passive or active range of motion, or time to recovery. Study limitations included an average delay of 34 hours from the time of injury to diagnosis, administration of only three treatments within 7 days, treatment pressure of only 2.0 ATA, and short treatment duration.

### **HBO2 as an Ergogenic Aid**

Professional athletes have reportedly received HBO<sub>2</sub> before sports participation, believing that performance would improve (28). Contradictory findings have been reported regarding the effect of a single HBO<sub>2</sub> treatment on aerobic performance. A Yugoslavian study (29) demonstrated that HBO<sub>2</sub> prior to treadmill running to volitional exhaustion increased peak running velocity and VO<sub>2</sub>max when measured 30 minutes and 3 hours posttreatment. HBO<sub>2</sub> was administered for 60 minutes at 2.8 ATA. Enhanced performance and VO<sub>2</sub>max were attributed to additional oxygen storage in skeletal muscle. However, to our knowledge, this link has yet to be definitively established.

In contrast, two recent studies (30,31) reported no change in submaximal and maximal exercise performance following HBO<sub>2</sub>. Webster et al (30) administered a single 60-minute HBO<sub>2</sub> treatment at 2.0 ATA followed by a cycling test performed on average 22.5 minutes posttreatment. McGavock et al (31) examined aerobic performance following a 90-minute HBO<sub>2</sub> treatment at 2.5 ATA with a running test performed about 40 minutes posttreatment. In both studies, time to exhaustion and VO<sub>2</sub>max were unchanged.

It is difficult to rationalize how prior HBO<sub>2</sub> could enhance performance. Tissue retention of oxygen following treatment is unlikely since tissue autoregulation reduces O<sub>2</sub> levels upon return to a normobaric, normoxic environment (32). Webster et al (30) used near-infrared spectroscopy to examine tissue oxygenation at rest, throughout exercise, and during recovery. Following an HBO<sub>2</sub> treatment, muscle tissue oxygenation during rest and recovery was similar to control.

### **Can HBO<sub>2</sub> Alleviate Fatigue?**

Only two human studies (7,31) have examined using HBO<sub>2</sub> to alleviate exercise-induced fatigue. Following exercise-induced tissue damage produced by eccentric leg extension, HBO<sub>2</sub> treatments had a negligible effect on quadriceps power for up to 96 hours postinjury (7). The acute effects of an HBO<sub>2</sub> treatment were examined on recovery following prolonged running (31). Subjects performed in four exercise-HBO<sub>2</sub> conditions. Exercise was a 90-minute run at 75% to 80% of VO<sub>2</sub>max to produce fatigue. HBO<sub>2</sub> treatments consisted of breathing 95% O<sub>2</sub> at 2.5 ATA for 90 minutes. Submaximal and maximal performances were assessed using oxygen uptake measures during treadmill running. Recovery from exercise-induced fatigue was not enhanced following a single HBO<sub>2</sub> treatment.

### **Risks of HBO<sub>2</sub>**

HBO<sub>2</sub> is not without risk. Its side effects can be divided into two categories: pressure effects and oxygen toxicity (8).

**Pressure effects.** Normally, gas-filled structures in the body include the lungs, middle ear, paranasal sinuses, and intestines. Boyle's law

states that, at a constant temperature, the volume of a gas is inversely proportional to its pressure. Therefore, any bubble or flexible gas-filled space in the body changes volume inversely proportional to the changes in pressure exerted.

Complications may develop when gas-filled structures change volume in response to HBO<sub>2</sub> pressure changes. The consequences of gases expanding and contracting in flexible spaces such as the intestines are usually insignificant (abdominal cramps, flatulence). Gases contained in rigid spaces such as the middle ear and sinuses, however, usually account for more of the troublesome side effects of hyperbaric therapy.

Middle ear barotrauma is the most common complication of HBO<sub>2</sub> treatments (8). It usually produces ear pain and can result in serous otitis media or rupture of the tympanic membrane. Patients with eustachian tube dysfunction are at higher risk for these conditions (33). Sinus pain or "sinus squeeze" is the second most common complication. Both "sinus squeeze" and middle ear barotrauma occur more commonly in patients suffering from an upper respiratory infection and allergic rhinitis (8).

To prevent some of the pressure complications of HBO<sub>2</sub>, autoinflation or equalization techniques are performed including the Valsalva's maneuver, chewing movements, biting down on a closed jaw, or building up the pressure in the pharynx while pinching the nose (34). Patients should be instructed not to perform too vigorous a Valsalva's maneuver or they may suffer a rupture of the round and oval windows. This is usually heralded by the sudden onset of deafness, tinnitus, nystagmus, and vertigo (37).

Another serious but infrequent complication of HBO<sub>2</sub> is pneumothorax, which can occur when a patient holds his or her breath during compression or decompression. Pneumothorax should be considered when a patient reports shortness of breath, cough, or chest pain. Because of the pressure, a simple pneumothorax may quickly develop into a life-threatening tension pneumothorax during HBO<sub>2</sub>, which may require expedient decompression.

**Oxygen toxicity.** Oxygen is a medication, and problems can arise with prolonged or repetitive exposures. Most problems are seen in treatments lasting several hours that are repeated for many days. Oxygen toxicity may cause seizures in some patients. These generalized tonic-clonic seizures are usually self-limited. They may occur spontaneously but are usually preceded by symptoms such as nausea, twitching, auditory changes, or dizziness. Oxygen toxicity seizures are rare; the risk has been estimated to be around 1 in 10,000 exposures at routine treatment pressures (8,35). Periodic air breaks lower the risk during treatment. Five-minute air breaks are

typically administered every 30 minutes when treatments exceed 2.4 ATA.

**Ocular changes.** Progressive myopia can develop in patients undergoing daily treatments for several months. This condition results from refractory changes in the ocular lens but is usually reversible within a few weeks after cessation of HBO<sub>2</sub> (36,37). Retinal changes may occur but are usually limited to premature infants and therefore are typically not an issue in sports medicine (38). Cataracts, once believed to develop after prolonged exposure to oxygen under pressure, are now *not* believed to occur from HBO<sub>2</sub> (36).

### **Final Recommendations**

Soft-tissue injuries are a common problem in sports medicine, and HBO<sub>2</sub> has been used as an adjunctive therapy to enhance recovery. Several different mechanisms are believed to be responsible for the beneficial effects of hyperoxygenation. HBO<sub>2</sub> treatments should be initiated within 24 hours after injury with treatments at 2.5 ATA for 90 minutes. The number of treatments will vary with the type and severity of the injury.

### **References**

1. Borromeo CN, Ryan JL, Marchetto PA, et al: Hyperbaric oxygen therapy for acute ankle sprains. *Am J Sports Med* 1997;25(5):619-625
2. James PB: New horizons in hyperbaric oxygenation. *Adv Exp Med Biol* 1997;428:129-133
3. Kaijser L: Physical exercise under hyperbaric oxygen pressure. *Life Sci* 1969;8(pt 1):929-934
4. Nylander G, Lewis D, Nordstrom H, et al: Reduction of postischemic edema with hyperbaric oxygen. *Plast Reconstr Surg* 1985;76(4):596-603
5. Nylander G, Nordstrom H, Franzen L, et al: Effects of hyperbaric oxygen treatment in post-ischemic muscle: a quantitative morphological study. *Scand J Plast Reconstr Surg* 1988;22(1):31-39
6. Sirsjo A, Lehr HA, Nolte D, et al: Hyperbaric oxygen treatment enhances the recovery of blood flow and functional capillary density in postischemic striated muscle. *Circ Shock* 1993;40(1):9-13
7. Staples JR, Clement DB, Taunton JE, et al: Effects of hyperbaric oxygen on a human model of injury. *Am J Sports Med* 1999;27(5):600-605
8. Undersea and Hyperbaric Medical Society: in Hampson NB (ed): *Hyperbaric Oxygen Therapy: Committee Report 1999*. Kensington, MD, 1999, pp 1-82
9. Arntzenius AKW: *De Pneumatische Therapie*. Scheltema & Holkemas, Boekhandel, Amsterdam, 1887

10. Marino PL: Oxygen transport, in Marino PL (ed): The ICU Book, ed 1. Philadelphia, Lea & Febiger, 1991, pp 14-24
11. Clark JM, Lambertsen CJ: Alveolar-arterial O<sub>2</sub> differences in man at 0.2, 1.0, 2.0, and 3.5 Ata inspired PO<sub>2</sub>. J Appl Physiol 1971;30(5):753-763
12. Boerema I, Meyne NG, Brummelkamp WK, et al: Life without blood: a study of the influence of high atmospheric pressure and hypothermia on dilution of the blood. J Cardiovasc Surg 1960;1:161-164
13. Bird AD, Telfer AM: Effect of hyperbaric oxygen on limb circulation. Lancet 1965;13(1):355-356
14. Bouachour G, Cronier P, Gouello JP, et al: Hyperbaric oxygen therapy in the management of crush injuries: a randomized double-blind placebo-controlled clinical trial. J Trauma 1996;41(2):333-339
15. Knighton DR, Halliday B, Hunt TK: Oxygen as an antibiotic: the effect of inspired oxygen on infection. Arch Surg 1984;119(2):199-204
16. LaVan FB, Hunt TK: Oxygen and wound healing. Clin Plast Surg 1990;17(3):463-472
17. Davis JC, Hunt TK (eds): Problem Wounds: The Role of Oxygen. New York City, Elsevier, 1988, pp 5-30
18. Weiss EL: Connective tissue in wound healing, in McCulloch JM, Kloth LC, Feedar JA (eds): Wound Healing: Alternatives in Management, ed 2. Philadelphia, FA Davis Co, 1994, pp 16-31
19. Hammarlund C: The physiologic effects of hyperbaric oxygenation, in Whelan HT, Kindwall EP (eds): Hyperbaric Medicine Practice, ed 2. Flagstaff, Arizona, Best Pub Co, 1995, pp 37-68
20. Jonsson K, Jensen JA, Goodson WH III, et al: Tissue oxygenation, anemia, and perfusion in relation to wound healing in surgical patients. Ann Surg 1991;214(5):605-613
21. Hunt TK, Zederfeldt B, Goldstick TK: Oxygen and healing. Am J Surg 1969;118(4):521-525
22. Chang N, Mathes SJ: Comparison of the effect of bacterial inoculation in musculocutaneous and random-pattern flaps. Plast Reconstr Surg 1982;70(1):1-10
23. Gottrup F, Firmin R, Hunt TK, et al: The dynamic properties of tissue oxygen in healing flaps. Surgery 1984;95(5):527-536
24. Knighton DR, Halliday B, Hunt TK: Oxygen as an antibiotic: a comparison of the effects of inspired oxygen concentration and antibiotic administration on in vivo bacterial clearance. Arch Surg 1986;121(2):191-195



25. Badwey JA, Karnovsky ML: Active oxygen species and the functions of phagocytic leukocytes. *Annu Rev Biochem* 1980;49:695-726
26. Zamboni WA, Roth AC, Russell RC, et al: The effect of acute hyperbaric oxygen therapy on axial pattern skin flap survival when administered during and after total ischemia. *J Reconstr Microsurg* 1989;5(4):343-347
27. Nylander G, Nordstrom H, Lewis D, et al: Metabolic effects of hyperbaric oxygen in postischemic muscle. *Plast Reconstr Surg* 1987;79(1):91-97
28. Stewart RJ, Yamaguchi KT, Mason SW, et al: Tissue ATP levels in burn injured skin treated with hyperbaric oxygen, abstracted. *Undersea Biomed Res* 1989;16(suppl):53
29. Staples J, Clement D: Hyperbaric oxygen chambers and the treatment of sports injuries. *Sports Med* 1996;22(4):219-227
30. James PB, Scott B, Allen MW: Hyperbaric oxygen therapy in sports injuries. *Physiotherapy* 1993;79(8):571-572
31. Potera C: Healing under pressure. *Phys Sportsmed* 1995;23(11):46-47
32. Cabric M, Medved R, Denoble P, et al: Effect of hyperbaric oxygenation on maximal aerobic performance in a normobaric environment. *J Sports Med Phys Fitness* 1991;31(3):362-366
33. Webster AL, Syrotuik DG, Bell GJ, et al: Exercise after acute hyperbaric oxygenation: is there an ergogenic effect? *Undersea Hyperb Med* 1998;25(3):153-159
34. McGavock JM, Lecomte JL, Delaney JS, et al: Effects of hyperbaric oxygen on aerobic performance in a normobaric environment. *Undersea Hyperb Med* 1999;26(4):219-224
35. Kelly DL Jr, Lassiter KR, Vongsvivut A, et al: Effects of hyperbaric oxygenation and tissue oxygen studies in experimental paraplegia. *J Neurosurg* 1972;36(4):425-429
36. Fernau JL, Hirsch BE, Derkay C, et al: Hyperbaric oxygen therapy: effect on middle ear and eustachian tube function. *Laryngoscope* 1992;102(1):48-52
37. Kindwall E: Contraindications and side effects to hyperbaric oxygen treatment, in Whelan HT, Kindwall EP (eds): *Hyperbaric Medicine Practice*, ed 2. Flagstaff, AZ, Best Pub Co, 1995 pp 83-97
38. Stone JA, Loar H, Rudge FW: An eleven year review of hyperbaric oxygenation in a military clinical setting, abstracted. *Undersea Biomed Res* 1991;18(suppl):80
39. Lyne AJ: Ocular effects of hyperbaric oxygen. *Trans Ophthalmol Soc UK* 1978;98(1):66-68

40. Palmquist BM, Philipson B, Barr PO: Nuclear cataract and myopia during hyperbaric oxygen therapy. Br J Ophthalmol 1984;68(2):113-117

41. Patz A: The effect of oxygen on immature retinal vessels. Invest Ophthalmol 1965;4(6):988-989

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