

How Can Hyperbaric Oxygen Contribute to Treatment?

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In Brief: Hyperbaric oxygen (HBO₂) 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, 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 atmospheres absolute (ATA), with a minimum of 90 minutes for each treatment. Some professional and amateur athletes use HBO₂ 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 HBO₂ is a medical treatment with associated risks.

In recent years, professional and college teams have started using hyperbaric oxygen therapy (HBO₂) to treat sports injuries. From muscle contusions and ankle sprains to delayed-onset muscle soreness, HBO₂ has been used to facilitate soft-tissue healing (1-7). To minimize the time between injury and HBO₂ 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 HBO₂.

During HBO₂ treatment, a patient breathes 95% to 100% oxygen at pressures above 1.0 atmosphere absolute (ATA). In America, HBO₂ chambers were first used as a treatment for deep-sea divers who experienced decompression sickness. Modern HBO₂ treatment evolved from these medical activities (8,9). The Undersea and Hyperbaric Medical Society (UHMS) has evaluated the use and effectiveness of HBO₂ for different medical conditions. The UHMS regularly reviews the applications of HBO₂ and lists the diseases and conditions that are supported by scientific information. Currently, the UHMS approves HBO₂ 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).

Figure 1: Courtesy of J. Scott Delaney, MD

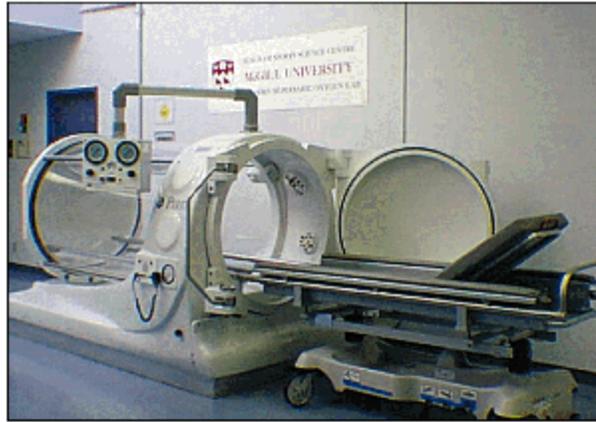


FIGURE 1. The monoplace hyperbaric oxygen chamber at McGill University is used for research and clinical applications.

Principles of HBO₂

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 (P_aO_2) is approximately 100 mm Hg. At rest, the tissues of the body consume about 5 mL of O₂ per 100 mL of blood.

During HBO₂ 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. HBO₂ at 3.0 ATA increases oxygen delivery to the tissues from 20.0 to 26.7 mL of O₂ 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 HBO₂. 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). HBO₂ 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 HBO₂ 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 HBO₂ before sports participation, believing that performance would improve (28). Contradictory findings have been reported regarding the effect of a single HBO₂ treatment on aerobic performance. A Yugoslavian study (29) demonstrated that HBO₂ prior to treadmill running to volitional exhaustion increased peak running velocity and VO₂max when measured 30 minutes and 3 hours posttreatment. HBO₂ was administered for 60 minutes at 2.8 ATA. Enhanced performance and VO₂max 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 HBO₂. Webster et al (30) administered a single 60-minute HBO₂ 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 HBO₂ treatment at 2.5 ATA with a running test performed about 40 minutes posttreatment. In both studies, time to exhaustion and VO₂max were unchanged.

It is difficult to rationalize how prior HBO₂ could enhance performance. Tissue retention of oxygen following treatment is unlikely since tissue autoregulation reduces O₂ 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 HBO₂ treatment, muscle tissue oxygenation during rest and recovery was similar to control.

Can HBO₂ Alleviate Fatigue?

Only two human studies (7,31) have examined using HBO₂ to alleviate exercise-induced fatigue. Following exercise-induced tissue damage produced by eccentric leg extension, HBO₂ treatments had a negligible effect on quadriceps power for up to 96 hours postinjury (7). The acute effects of an HBO₂ treatment were examined on recovery following prolonged running (31). Subjects performed in four exercise-HBO₂ conditions. Exercise was a 90-minute run at 75% to 80% of VO₂max to produce fatigue. HBO₂ treatments consisted of breathing 95% O₂ 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 HBO₂ treatment.

Risks of HBO₂

HBO₂ 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 HBO₂ 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 HBO₂ 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 HBO₂, 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 HBO₂ 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 HBO₂, 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 HBO₂ (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 HBO₂ (36).

Final Recommendations

Soft-tissue injuries are a common problem in sports medicine, and HBO₂ has been used as an adjunctive therapy to enhance recovery. Several different mechanisms are believed to be responsible for the beneficial effects of hyperoxygenation. HBO₂ 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.

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