Hyperbaric Oxygen Therapy and its role in Sports Medicine

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

Hyperbaric oxygen (HBOT) 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, HBOT should be considered as an adjunctive therapy as soon as possible after injury diagnosis.

During HBOT treatment, a patient breathes 95% to 100% oxygen at pressures above 1.0 atmosphere absolute (ATA). 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 (PaO2) is approximately 100 mm Hg. At rest, the tissues of the body consume about 5 mL of O2 per 100 mL of blood. During HBOT 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. HBOT 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 HBOT. 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). HBOT 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 HBOT to aid the healing of soft-tissue infections and osteomyelitis (21). HBOT has also been found to inhibit PMN adherence on postcapillary venules (23). Although this may seem paradoxic, 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 maintainance of cellular viability (24,25). Increased oxygen delivery to the tissue with HBOT 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. HBOT is an effective treatment for crush injuries and other acute traumatic peripheral ischemias because it alleviates hypoxia and reduces edema; however, clinical experience with HBOT for sports injuries is limited. Also, the criteria for using HBO2 in acute traumatic peripheral ischemias are not clearly established. HBOT 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). HBOT has been used to treat joint, muscle, ligament, and tendon injuries in soccer players in Scotland. When HBOT 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. HBOT 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 HBOT 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 HBOT 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.

Source – www.hyperbaric-care.com