

HYPERBARIC OXYGEN FOR ATHLETES

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ABSTRACT

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

Hyperbaric oxygen (HBO) 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 should be considered as an adjunctive therapy as soon as possible after injury diagnosis. Hyperbaric oxigenation (HBO) could accelerate the removal of blood lactic acid after exercise and training.

Key word: Hyperbaric oxygen (HBO), sport, lactic acid

Introduction

The continuing desire to improve performance, particularly at the national and international levels, has led to the use of ergogenic aids. Ergogenic aids are defined as “a procedure or agent that provides the athlete with a competitive edge beyond that obtained via normal training methods”. Random drug testing has been implemented in an effort to minimize an athlete's ability to gain an unfair advantage. However, other means of improving performance have been tried. Blood doping has been used to enhance endurance performance by improving oxygen delivery to working muscles. As oxygen is carried in combination with the hemoglobin, it seems logical that increasing the number of red blood cells (RBC's) in the body would increase the oxygen carrying capacity to the tissues and result in improved performance. The first experiments of removing and then reinfusing blood showed a significant improvement in performance time on the treadmill. This process is now known as blood doping (Wilson Judy R. 2004).

During the last century, competitive sport has progressively become more professional; march of technological progress and improvement in exercise physiology knowledge have contributed to improve efficiency of training. Current training sessions (often performed twice daily) are quantitatively and qualitatively optimized to induce a maximal or supra-maximal training load (Barnett, 2006). Consequently, it is crucial for competitive athletes to make the most of rest periods and to consider that the loading-recovery cycle constitutes the key point of the training process (Marc Vanderthommen , Souleyma Makrof and Christophe Demoulin, 2010).

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. 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. 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 (Scott Delaney, MD; D. L. Montgomery, PhD, 2001).

Hyperbaric Oxygen

Hyperbaric oxygenation (HBO) involves the use of oxygen under pressure greater than that found on earth's surface at sea level (Jain, KK., 1996). Hyperbaric oxygenation involves providing a person with 100% inspired oxygen while in an environment where the pressure is greater than that at sea level (760 mmHg; 14.7 pounds per square inch-psi; or one atmosphere absolute-ATA). At a pressure of 2.4 ATA (45 feet of sea water) and breathing 100% oxygen there is an increase in the arterial partial pressure of oxygen (PaO₂) from 100 mmHg to over 2000 mmHg. The increased pressure causes the oxygen to dissolve in the plasma leaving the hemoglobin carrying capacity of the RBC unaltered. Under normal conditions, almost all

oxygen is transported by hemoglobin and very little is dissolved in the plasma. This sets up a very large gradient at the tissue level that can raise the tissue oxygen levels to over 300 mmHg (Irvine D. Prather and Judy R. Wilson, 2004).

Oxygen is a key component for the production of energy in the body. It operates as part of the aerobic metabolism. This form of energy production is similar to that of a fire. Wood may provide the combustible material, which is combined with oxygen to produce carbon dioxide (CO₂), heat, and water. The body uses a similar process though without the heat. The main transport agent of this cold energy production is ATP (Adenosine Triphosphate) which is an adenosine-derived nucleotide that supplies large amounts of energy to cells for various biochemical processes, including muscle contraction and sugar metabolism, through its hydrolysis to ADP. Without the activity of ATP and ADP, the cells would die (Roque R. Wicker, 2011).

Hyperbaric oxygen therapy (HBOT) is a method of administering a higher dose of oxygen at an increased atmospheric pressure. The Undersea and Hyperbaric Medical Society describes HBOT as “the inhalation of 100 per cent oxygen while the entire patient is enclosed within a chamber at pressures of at least 1.4 atmosphere absolute or greater.”² HBOT provides a pharmacological dose of oxygen needed to stimulate and support wound healing for recalcitrant hypoxic wounds (Martine Albert, 2008). Hyperbaric oxygen therapy (HBO) is the inspiration of 100 percent oxygen while the body is subjected to increased atmospheric pressure, which has the effect of hyper oxygenating the tissues. HBO is used as a treatment for a number of medical conditions including carbon monoxide poisoning, “the bends,” and soft tissue injuries. HBO acts primarily to reduce localized tissue hypoxia while reducing edema through vasoconstriction. A growing number of professional sports teams including the National Hockey League, the National Football League, and the National Basketball League have also began to utilize HBO as an injury recovery technique (Babul and Rhodes 2000).

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 (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

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 (J. Scott Delaney, MD; D. L. Montgomery, PhD, 2001).

Oxygen is usually taken into the blood from alveoli by breathing, where it combines with the Alhemoglobin in blood and is carried to peripheral tissues. However, since the oxygen absorption of hemoglobin is low at 100mm Hg, the oxygen saturation is limited by the hemoglobin concentration in the blood. By performing HBO treatments, according to Henry's law (in the conditions of temperature regularity, the quantity of the gas dissolved in contact with a liquid is proportional to partial pressure of the gas), more oxygen is dissolved in the plasma of the pulmonary vein via the alveolar, which increases oxygen that reaches the peripheral tissues. Consequently, using pure oxygen with twice the atmospheric pressure, it becomes possible to raise the partial pressure of oxygen to near the 1000mm Hg. This is essentially the conditions of application of HBO for medical treatment. In HBO treatment, the capacity of the gas stored inside the body is reduced by high pressure, and then the partial oxygen pressure (pO₂) of arterial blood is raised in the treatment of diseases such as hypoxia disease, cell damage from infection, or a malignant tumour. Although these effects are the same for each disease, this treatment is currently used only in the field of emergency medicine (Yoshimasa Ishii, Masataka Deie, Nobuo Adachi, Yuji Yasunaga, Patrick Sharman, Yutaka Miyanaga, and Mitsuo Ochi, 2006).

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.

Physiologic Effects of HBO on the Healthy Human Body

The important general effects of hyperoxia on a healthy human body are listed in Table 1. The effects vary according to the pressures used, the duration of exposure, and health of the subject. Unless otherwise stated, HBO refers to the use of 100% oxygen. The effects of HBO on each system; both in health and in disease, will be discussed in chapters dealing with disorders of those systems. As an introduction, some important effects are described here briefly (Jain, KK.,1996).

Vasoconstriction.

High tissue oxygen concentrations cause blood vessels to constrict, which can lead to a 20% decrease in regional blood flow. 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

Neovascularization and epithelialization.

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

Stimulation of fibroblasts and osteoclasts.

In a hypoxic milieu, fibroblasts are unable to synthesize collagen, and osteoclasts are unable to lay down new bone. 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. 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. Studies have shown that the local tissue resistance to infection is directly related to the level of oxygen found in the tissue. 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. Oxygen is converted within the PMN into toxic substrates (superoxides, peroxides, and hydroxyl radicals) that are lethal to bacteria. These effects on the immune system allow HBOT to aid the healing of soft-tissue infections and osteomyelitis. HBOT has also been found to inhibit PMN adherence on postcapillary venules. 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. 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. 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%. 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 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.

Healing occurs in a predictable sequence of events that rests on a successful cellular and biochemical chain of events. Any disruption to these events can have a detrimental effect on the healing process. The key to successful management of any type of ulceration lies not only with the accurate identification of the underlying cause of the ulcer but also with the appropriate measures to remove or modify the causative factors interfering with healing.

Any interruption of normal healing will result in a chronic wound. In many cases the pathological environment can be remedied with best-practice interventions aimed at correcting the offending agents, and with the judicious consideration of the local wound-care needs: i.e., debridement, moisture balance and bacterial balance. However, some chronic wounds still do not respond and become recalcitrant to any type of intervention. Tissue hypoxia is a key element of healing failure.

Adequate oxygenation fuels the cellular function essential to the tissue-repair process. As the wound enters the inflammatory phase initiating the healing process, the requirements

for oxygen increase and are accelerated due to oxidative cellular mechanisms. During normal wound healing, the fibroblasts are stimulated to make collagen. This supports the development of a collagen matrix, which forms a scaffold that supports new blood vessels (angiogenesis). Angiogenesis enables the delivery of oxygen and nutrients required to promote the healing process. The probability of wound healing is extremely high if the tissue oxygen tension (pO₂) at the wound site is 40 mmHg, where fibroblastic activity and cellular interactions are supported.

Oxygen moves from areas of high concentration (pressure) to areas of low concentration (pressure). Wounds naturally have a hypoxic central zone that is surrounded by normal perfusion in the adjacent tissue. This produces an oxygen gradient from the peripheral tissues to the centre of the wound. The central hypoxic zone therefore creates a demand for oxygen and engages the adjacent tissue to provide oxygen to support the healing process through angiogenesis. Subsequently, hypoxic peri-wound tissues cannot provide adequate levels of oxygen to support cellular proliferation and the creation of new vessels to the central zone. A lack of oxygen, or poor oxygenation, prevents a normal healing pattern.

Oxygen is key to the phagocytosis and killing of bacteria by neutrophils or polymorphonuclear cells (PMNs). Neutrophil phagocytosis creates a 25-fold increase in oxygen consumption. As the oxygen tension falls below 30 mmHg the efficiency of bacteriocidal action of PMNs begins to drop off dramatically. Therefore, hypoxia weakens the tissues' resistance to bacteria. Oxygen also stimulates macrophages to produce angiogenic substances— as vascular endothelial growth factor (VEGF)—that attract and stimulate endothelial cells. Therefore, hypoxia weakens the neovascularization process.

When wound hypoxia is the systemic cause of the healing failure, providing oxygen at the wound site is, essentially, treating the cause. Hyperbaric oxygen therapy is an adjunctive therapeutic modality in which the patient is given a high volume of pure oxygen to breathe in an environment of elevated atmospheric pressure, which leads to an increase in tissue oxygen pressures at the wound site.

HBOT stimulates fibroblast activity by providing an environment rich in oxygen, which supports the formation and deposition of collagen. The tissue's tensile strength is then improved. HBOT reduces wound infection and potentiates the effect of antibiotics through its direct impact on anaerobic bacteria so they cannot proliferate and will eventually die, as well

as through its indirect impact on aerobic bacteria by enhancing the microbicidal function of the PMNs (Albert, Martine, 2008).

Hiperbaric Oxygen for Athletes

Only two human studies 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 post injuries. The acute effects of an HBO2 treatment were examined on recovery following prolonged running. 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. Sub maximal 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 (Scott Delaney, MD; D. L. Montgomery, PhD, 2001).

The purpose of this research was to determine the effects of hyperbaric oxigenation (HBO) on blood lactic acid and pCO₂ after sub maximal exercise. This study was held at The Naval Health Institute (Lembaga Kesehatan Kelautan/LAKESLA) by using the hyperbaric chamber. Statistical analysis showed that there was no significant difference ($p > 0,05$) in lactic acid and pCO₂ level between NBO and HBO groups immediately after exercise. The significant difference ($p < 0,05$) was shown in lactic acid and pCO₂ level between NBO and HBO groups 30" after exercise. It should thus be concluded that passive rest under hyperbaric condition could accelerate the removal of lactic acid through aerobic metabolism, with an increased of pCO₂. It should be noted too that under hyperbaric condition we imposed in this study (100% oxygen, 2,4 ATA), the lactic acid level has reached the prescribed resting (pre exercise) lactic acid value (2 mmol/l) (Untari, Ni Komang Sri Dewi, 2003).

The research was aimed to study the effects of a 30 minute exposure to 2.5 ATA with 100% O₂-inhalation on lactate concentration after muscular fatigue from incremental exercise on a cycle ergometer. The subjects were 60 male naval cadets aged 20-23 years whose physical fitness was equivalent to the average athletes. All volunteers participated in the first VO₂ max exercise test to obtain their baseline data and randomly assigned into 3 groups of 20. The 3 groups were: Rest recovery group (RR), Oxygen recovery group (OR), and the Hyperbaric

oxygenation (HBO₂) recovery group (HR). The volunteers took the incremental exercise test (Ordinary Lamp Protocol) on a cycle ergometer to exhaustion, then rested according to the above assigned groups. Blood samples were taken from each volunteer before the experiment, at exhaustion and every 5-minute intervals after the exhaustion for 30 minutes and immediately assayed for lactate concentration. The results showed significant decrease of blood lactate concentration at 15, 20 and 25 minutes intervals after the exhaustion in the HR group compared to the others. It might be initially concluded that HBO₂ enhanced the rate of lactate removal from peripheral blood vessels, therefore it shortened the recovery time (Sueblinvong T, Egtasaeng N, Sanguangrangsirikul S. 2004).

The best experience of HBO use as a recovery method from muscular fatigue occurred during the Nagano Winter Olympics with seven participating athletes. After physical activity, the athletes received HBO treatment for 30–40 minutes at 1.3 ATA, with a maximum of six times per athlete and an average of two times per athlete. An athlete who experienced tension and sharp pain of the abdominal muscles was able to play a game after HBO treatment with almost no pain. Another player who was experiencing anterior knee pain was treated within 2 days of injury and was able to perform afterwards with no difficulty. These test cases show that HBO treatments demonstrate high performance without adverse effects such as muscular pain and nerve paralysis. Players were also able to benefit from the conditioning effects of HBO treatment (Yoshimasa Ishii, Masataka Deie, Nobuo Adachi, Yuji Yasunaga, Patrick Sharman, Yutaka Miyanaga, and Mitsuo Ochi, 2006).

One suggested application of HBO is to increase the rate at which lactate is removed from exercised muscles because this has been proposed to decrease muscular fatigue. In a study testing lactate clearance times, Babul and Rhodes (2000) found significant differences between passive recovery with 100 percent oxygen and passive recovery with ambient breathing. A study involving the recovery from injury for professional soccer players suggested a 55 percent decrease in recovery time for those athletes utilizing HBO. However, this study was highly subjective in nature relying on a physiotherapist's estimation of injury recovery time versus the reality of recovery time (Babul and Rhodes 2000). Other studies have produced conflicting results. Two such studies looked at the recovery from ankle sprains. The first suggested a 30 percent faster return to normal function while the second showed no significant difference between the HBO group and the control group.

From this discussion, it should be clear that the accumulation of lactate in wounds is not due to hypoxia. Lactate is produced largely in the oxidative burst (above) and in the “Warburg phenomenon”, in which rapidly dividing cells rely heavily on aerobic glycolysis the metabolism of glucose to lactate in the presence of oxygen as their prime energy source (Hunt TK, R. S. Aslam, 2004).

It would seem logical then that such an elevation of blood and tissue oxygen tensions could provide a competitive advantage to athletes competing in aerobic events. In order to test this hypothesis we had experienced runners predict their times for a 5 km course and exposed them to hyperbaric oxygenation. Following exposure, they ran the course and compared actual with predicted times. Nine volunteers were solicited from the Fort Worth Runners Club and were between the ages of 18 and 65 years with sufficient running experience to be able to predict their time on a local 5 km course with which they were familiar. They reported to the Hyperbaric Medicine Facility where they received ninety minutes of 100% oxygen in three thirty minute periods interspersed with two ten minute breaks for air and liquids. They were pressurized in a multiplace hyperbaric chamber to 2.2 ATA.

Conclusions

Hyperbaric oxygenation (HBO) involves the use of oxygen under pressure greater than that found on earth's surface at sea level. Hyperbaric oxygen therapy (HBO) is the inspiration of 100 percent oxygen while the body is subjected to increased atmospheric pressure, which has the effect of hyper oxygenating the tissues. Only two human studies have examined using HBO₂ to alleviate exercise-induced fatigue. The best experience of HBO use as a recovery method from muscular fatigue. One suggested application of HBO is to increase the rate at which lactate is removed from exercised muscles because this has been proposed to decrease muscular fatigue. Some research was to determine the effects of hyperbaric oxygenation (HBO) on blood lactic acid. Hyperbaric oxygen (HBO) 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. Hyperbaric oxygenation (HBO) could accelerate the removal of blood lactic acid after exercise and training.

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