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Review Article

Applications of hyperbaric oxygen therapy in dentistry: A mini review

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ABSTRACT

Hyperbaric oxygen (HBO) therapy is increasingly used in number of areas of medical practice. It is a unique intervention whose method of action is not well-understood. Clinicians may request its use for their patients, but often its mechanisms are not completely understood. Here, we present a mini review and discussion of HBO and uses of this exciting intervention.

CLINICAL RELEVANCE TO INTERDISCIPLINARY DENTISTRY

Relevance of hyperbaric oxygen therapy to periodontists, prosthodontists, and restorative dentists

- The osseointegrated implants are successfully applied in the dental clinic including oral and facial rehabilitations mainly for patients submitted to mutilating surgeries. However, patients submitted to radiation therapy present risks to treatment with implants owing to adverse effects on bone tissue. Nowadays, literature suggests therapies to improve the success of osseointegration such as HBO therapy that aims to prepare bone and adjacent tissues to receive the implant.
- Therefore, thorough understanding of HBO therapy and its applications is very essential for prosthodontists, periodontists, and other dental specialists to avail the benefits of this useful technique and improve the patient care.

Key words: Hyperbaric oxygen therapy, hypoxia, osteoradio necrosis, periodontal diseases

INTRODUCTION

"HYPER" means increased and "BARIC" means pressure. Hyperbaric oxygen (HBO) therapy is defined as inhalation of oxygen at increased pressure, for potential therapeutic benefit in a variety of clinical situations. The Committee on Hyperbaric Medicine defines HBO therapy as "A mode of medical treatment in which the patient is entirely enclosed in a pressure chamber and breathes 100% oxygen at a pressure >1 atmosphere absolute (ATA)." ATA is the unit of pressure and 1 ATA is equal to 760 mm of mercury or pressure at sea level.

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HISTORICAL BACKGROUND

- In 1900's: Hyperbaric spas flourished in the North American continent and Europe. Lack of a firm physiological basis and poor choice of indications caused scientific stasis in this field for many subsequent years
- In 1930's: HBO was first used in decompression sickness suffered by deep sea divers to recompress by Behnke
- In 1950's: The modern clinical application of HBO began, in parallel with an increased understanding of blood gas analysis and gas exchange physiology
- In 1960's two institutions preeminently pursued the clinical aspects of high pressure oxygenation. Dr. Bakers from the University of Amsterdam developed the use of intermittent HBO, for the treatment of gas-gangrene. Second major focus of interest in this area was Royal Infirmary of Glassgow, where various anesthetic and surgical aspects of HBO were applied and discussed.

Among these were treatment of necrotizing infections and anesthesia under hyperbaric conditions. In 1965: It was first used to assist wound healing when it was noted that burns of the victims of coalmine explosions treated with HBO_2 healed faster. In 1968: Duke University in North Carolina expanded a long-standing program of environmental physiology with the construction of inter-connected multiplace hyperbaric chambers
- Since 1970: Most of the instructional courses, research work and guidance have been provided by Under seas and Hyperbaric Medical Society (Headquarters in Kensington, Maryland). This medical organization publishes guidelines for hyperbaric oxygenation every 2-3 years.

PHYSIOLOGICAL BASIS

When we normally breathe air (with 21% O_2) at sea level pressure, most tissues need of oxygen are met from the oxygen combined to hemoglobin, which is 95% saturated. 100 ml blood carries 19 ml O_2 combined with hemoglobin and 0.32 ml dissolved in plasma. At the same pressure if 100% O_2 (oxygen) is inspired, O_2 combined with hemoglobin increases to a maximum of 20 ml and that dissolved in plasma to 2.09 ml. The higher pressure during HBO treatment pushes more oxygen into solution. The amount of O_2 dissolved in plasma increases to 4.4 ml/dL at a pressure of 2 ATA and to 6.8 ml/dL at 3 ATA. This additional O_2 in solution is almost sufficient to meet tissue needs without contribution from O_2 bound to hemoglobin and is responsible for most of the beneficial effects of this therapy. The principal rationale of HBO therapy is to decrease tissue O_2 tension. Hence, it is reasonable that primary indications are conditions that include either regional or global hypoxia. Another group of indications take advantage of the fact that specific micro-organisms are oxygen intolerant. The increase in hydrostatic pressure inherent in HBO therapy provides an important part of the rationale for use in gas lesion diseases such as gas embolism, etc.[1]

HYPOXIA

Hypoxia or hypoxiation is a pathological condition in which the body as a whole (generalized hypoxia) or a region of body (tissue hypoxia) is deprived of adequate oxygen supply. Variations in arterial oxygen concentrations can be part of the normal physiology, for example, during strenuous physical exercise. A mismatch between oxygen supply and its demand at the cellular level may result in a hypoxic condition. Hypoxia in which there is complete deprivation of oxygen supply is referred to as anoxia.

Hypoxia differs from hypoxemia in that, in the latter, the oxygen concentration within the arterial blood is abnormally low. It is possible to experience hypoxia and have a low oxygen content (e.g. due to anemia) but maintain high oxygen partial pressure (pO_2). Incorrect use of these terms can lead to confusion, especially as hypoxemia is among the causes of hypoxia (in hypoxemic hypoxia).

Generalized hypoxia occurs in healthy people when they ascend to high altitude, where it causes altitude sickness leading to potentially fatal complications: High altitude pulmonary edema and high altitude cerebral edema. Hypoxia also occurs in healthy individuals when breathing mixtures of gases with low oxygen content, e.g. while diving underwater especially when using closed-circuit re-breather systems that control the amount of oxygen in the supplied air. A mild and nondamaging intermittent hypoxia is used intentionally during altitude trainings to develop an athletic performance adaptation at both the systemic and cellular level.

Hypoxia is also a serious consequence of preterm birth in the neonate. The main cause for this is that the lungs of the human fetus are among the last to develop during pregnancy. To assist the lungs to distribute oxygenated blood throughout the body infants at risk of hypoxia are often placed inside an incubator capable of providing continuous positive airway pressure (also known as a humidicrib).

MECHANISM

Hypoxia extends beyond the local wound environment. Reactive oxygen species are produced, including oxygen free radicals. Initially, these cause vasoconstriction followed by vasodilatation. Endothelial cell damage and release of prostaglandins, pro-inflammatory cytokines (tumor necrosis factor-α and interleukin-6) and nitric oxide from vascular endothelium occurs. Subsequent membrane peroxidation further increases the cellular damage. As capillaries become leaky, interstitial edema occurs. Circulation is further compromised with compounded injury. The surgical or medical reestablishment of interrupted circulation sends blood to the ischemic area, providing new oxygen substrate for the formation of more free radicals.

In massive injury the release of inflammatory cytokines and free radicals escape the normal regulatory mechanisms and can lead to multiple organ failure. Hence, a long and catastrophic chain of events can be initiated by O_2 deprivation.

PROCEDURES AND EQUIPMENT

HBO therapy is administered in a hyperbaric chamber. They are of two basic types:[3]

Monoplace chambers: It is transparent, made up of acrylic, can accommodate a single patient and the patient does not require a mask. Primary advantage: Cost and space requirements. Multiplace chambers: Usually of steel (some may be made up of aluminum), can accommodate more than two people and is pressurized with air, while the patients breathe O_2 from a tight fitting mask/circuit. Advantage: Is suitable for critically ill patients requiring ventilation, monitoring and constant attendance. Pressure and duration: It depends upon the indication. It ranges from 2 to 6 ATA for 2-6 h. Decompression sickness/gas embolism may require prolonged, continuous saturation protocols. PIO_2 (partial pressure of inspired) very rarely exceeds 2.8 ATA. Emergency indications for HBO therapy generally require only 2-3 separate chamber treatments.

PRACTICAL ASPECTS OF CARE IN A CHAMBER

- Fire safety: Maintaining electrical components outside the chamber. Passing cables through insulated pass throughs
- Electrical defibrillation: In a hyperbaric chamber defibrillation is controversial, because of the possibility of poor skin contact, arcing and risk of fire. Large metal environment may predispose attendants to shock. Chambers needs to be decompressed prior to use of a defibrillator. Moreover, the latency of bubble formation and onset of decompression sickness symptoms is sufficient to allow a brief excursion to 1 ATA for defibrillation, with subsequent return to previous pressures.

Miscellaneous

Flexible bags are preferred over glass bottles for intravenous infusions so that pressure gradient between the chamber atmosphere and the fluid reservoir does not occur.

If glass bottles at all are to be used (nitro glycerine drip) it must be ensured that the gas space above the liquid is in constant communication with chamber atmosphere. Battery driven syringe infusion pumps are best. Other closed gas filled devices, which must be carefully monitored, are tracheal tube cuffs/face mask seals both of which may be filled with an incompressible medium such as water or saline, instead of air, hence that over or under-expansion does not occur and result in injury or leakage. An exception is flow-directed pulmonary artery catheters; here, the balloon is left deflated with filling port open to the atmosphere during HBO therapy. Positive pressure ventilation may be performed with self-inflating bags and volume cycled ventilators.

CELLULAR AND BIOCHEMICAL BENEFITS OF HYPERBARIC OXYGEN

- Promotes angiogenesis and wound healing
- Kills certain anaerobes
- Prevents growth of species such as *Pseudomonas*
- Prevents production of clostridial alpha-toxin
- Restores neutrophil mediated bacterial killing in previously hypoxic tissues
- Reduces leucocyte adhesion in reperfusion injury preventing the release of proteases and free radicals which cause vasoconstriction and cellular damage.

MEDICAL USES OF HYPERBARIC OXYGEN

Regional hypoxia (compromised graft flap, osteoradionecrosis (ORN), wounds and ulcers), crush injuries, thermal burns, global hypoxia (CO, CN intoxication, and severe anemia), infections (clostridial myonecrosis, necrotizing fasciitis, refractory osteomyelitis, and rhinocerebral mucormycosis), gas lesion conditions (gas embolism and decompression sickness).

THERAPEUTIC EFFECTS OF HYPERBARIC OXYGEN

Hyperoxygenation causes (i) immune stimulation by restoring white blood cell (WBC) function and enhancing their phagocytic capabilities and (ii) neovascularization in hypoxic areas by augmenting fibroblastic activity and capillary growth. This is useful in radiation tissue damage and other problem wounds.[4]

Vasoconstriction reduces edema and tissue swelling, while ensuring adequate oxygen delivery and is thus useful in acute trauma wounds and burns.

Bactericidal for anaerobic organisms and inhibits the growth of aerobic bacteria at pressures >1.3 ATA. It inhibits the production of alpha-toxin by *Clostridium welchii* and is synergistic with aminoglycosides and quinolones. Thus, it is lifesaving in gas-gangrene and severe necrotizing infections.

Reduces half-life of carboxy hemoglobin from 4 to 5 h to 20 min or less and is the treatment of choice for carbon monoxide poisoning in fire victims.

Mechanical effects: Direct benefit of increased pressure helps reduces bubble size in air embolism and decompression illnesses.

Reactivates "sleeping cells" in the penumbra region around central dead neuronal tissue. This is the basis of its use in neurological conditions.

Reduces adherence of WBC's to capillary walls and maybe useful in acute brain and spinal cord injury.

BENEFICIAL EFFECTS OF HYPERBARIC OXYGEN

- Safe therapy with very few and minor side-effects
- Addition of HBO obviates the need for frequent surgical procedures, promotes healing and early mobilization of the patient
- Reduces length of hospitalization and thereby overall treatment and rehabilitation costs
- Emerging role in indications which have lifetime disabilities.

CONTRAINDICATIONS OF HYPERBARIC OXYGEN

Absolute contraindication in patients with untreated pneumothorax and relative contraindications are in patients with optic neuritis, acute viral infection, congenital spherocytosis, uncontrolled acute seizures disorders, uncontrolled high fever, upper respiratory tract infections, pregnancy (if required), psychiatric problems, history of prior thoracic or ear surgery, which would make it impossible to equalize middle ear pressure or pulmonary pressure.

EFFECT OF HYPERBARIC OXYGEN THERAPY ON OSTEORADIONECROSIS

Osteoradionecrosis of the mandible is a significant complication of radiation therapy for head and neck cancer. In this condition, bone within the radiation field becomes devitalized and exposed through the overlying skin or mucosa, persisting as a nonhealing wound for 3 months or more. In 1926, Ewing first recognized the bone changes associated with radiation therapy and described them as "radiation osteitis." In 1983, Marx proposed the first staging system for ORN that also served as a treatment protocol. This protocol advocated that patients whose disease progressed following conservative therapy (HBO, local wound care, and debridement) were advanced to a radical resection with a staged reconstruction utilizing a nonvascularized bone graft.[2]

The basis for applying HBO to ORN is an extension of Marx's theory that ORN is the result of tissue hypoxia, hypocellularity and hypovascularity. The purpose of HBO is to increase the blood-tissue oxygen gradient, which enhances the diffusion of oxygen into hypoxic tissues. The increased oxygen supply stimulates fibroblast proliferation, angiogenesis, and collagen formation. In addition, the increased oxygen tension is bactericidal and bacteriostatic.[5]

After evaluating the literature, it appears clear that advanced ORN requires aggressive surgical therapy, and it has become increasingly evident that HBO alone has minimal if any benefit in the treatment of advanced ORN. In addition, as some recent publications have suggested, HBO may not have a clear role in the treatment of advanced ORN when a vascularized reconstruction is used. The use of HBO in early and intermediate ORN remains important because the benefit seems clear based on numerous retrospective studies. The morbidity of HBO is minimal including transient myopia, middle ear barotrauma and seizures. Absolute contraindications for HBO include optic neuritis, history of chronic obstructive pulmonary disease or congenital pulmonary blebs.[5]

EFFECTS OF HYPERBARIC OXYGEN THERAPY ON PERIODONTAL DISEASE

Periodontitis is an inflammatory disease caused by bacterial biofilms, which adhere on the teeth. The inflammatory and immune responses in periodontitis are a continuum of the normal host response to infection that eventually becomes the pathology when homeostasis is lost. Though there is ample of data to support the beneficial effects of HBO therapy for various medical conditions, very few studies have documented for its use in periodontal diseases. An increase in tissue oxygen tension generally speeds up the healing process in problem wounds in all parts of the body.[6] An oxygen-rich milieu always inhibits the growth of anaerobic microorganisms, effectively supporting antibiotic and surgical therapy. In addition, an oxygen-rich milieu enhances the function of leukocytes, activating or supporting the body's local defense mechanisms in areas that are already frequently poorly perfused, which in turn speeds up the healing process. Subsequent regenerative processes are also influenced by the increase in oxygen tension. Tissue capillarity clearly increases, and fibroblast replication is enhanced.[7]

In addition, the healing process in damaged bone can be accelerated by oxygen therapy or even made possible in the first place. Frequently, oxygen is applied centrally by increasing the oxygen tension in the air breathed in during HBO therapy. This leads to an increase in the amount of oxygen physically dissolved in the blood and thus, to higher oxygen levels in peripheral tissue. The increased concentration of oxygen brings about the change in bacterial milieu described above and improves the healing process in the wound. As serious infections are frequently deep seated, and intact skin constitutes too great of a barrier to diffusion, applying oxygen centrally is generally the only possibility to increase oxygen tension in the affected region. In cases of poorly healing superficial wounds and impaired skin integrity, local external applications can be used instead of central oxygen therapy. The principle behind this type of therapy is occlusion relative to the surrounding area with localized oxygenation.[8]

Shannon *et al.*[9] tested oxygen effects on healing gingival wedge excisions using Sprague-Dawley rats operated controls were maintained at normal pressure in room air experimental groups of 40 rats each were exposed for 90 min daily to one of the following: (1) 20.8% oxygen at 2.4 atmospheres pressure, (2) 100% oxygen at 1 atmosphere pressure, and (3) 100% oxygen at 2.4 atmosphere pressure. Histometric analysis was performed using light microscopy. The controls failed to show healing comparable to experimental animals until the end of 2 weeks. Enhanced connective tissue healing was most significant in the 2.4 atmospheres pressure groups at 3 and 6 weeks when compared with controls. However, by 12 weeks, no significant differences could be detected. Early connective tissue adaptation does not imply eventual attachment as epithelial down growth progressively displaced the connective adjacent to the root in both experimental and control groups.

Oxygen can also be applied locally when the oral mucous membrane is diseased, such that the resorption barrier is reduced, and there is only a short distance of diffusion. In addition, gingival and periodontal infections can also be caused or even dominated by anaerobic microorganisms, making it desirable to impair the milieu to reduce the growth of anaerobic microorganisms.

DISCUSSION

Several studies have described the beneficial role of HBO in the treatment of various human pathologies either alone or in combination with other therapies. Very few studies have been conducted to analyze the effects of HBO therapy on periodontal disease. Chen *et al.* 2002[10] showed that HBO increases local oxygen distribution, especially at the base of the periodontal pocket. This could inhibit the growth of anaerobe bacteria, and also on the other hand, would allow the ischemic tissues to receive an adequate intake of oxygen sufficient for a rapid recovery of cell metabolism.

A combination of both HBO and SRP substantially reduces (by up to 99.9%) the Gram-negative anaerobe loads of the subgingival microflora. HBO or SRP alone produces a temporarily more limited effect on the periodontal anaerobe load, which later reverts to the pretreatment values. HBO exerts its killing action specifically against those micro-organisms which, via the production of sophisticated virulence determinants, are responsible for direct tissue damage. The presence of high counts of periodontopathogenic bacteria enhances the inflammation response which, in turn, is mainly responsible for the clinical signs of periodontal diseases such as gingival edema, bleeding and migration of the gingival crevice with eventual formation of a periodontal pocket. These pathological effects can be measured by means of specific parameters. One of these is the gingival index, which is a reliable parameter for evaluating the degree of inflammation at the gingival level.

Chen *et al.* in 2003,[11] have reported the effects of HBO in a controlled study of periodontitis in 24 patients. The study teeth were divided into four groups based on treatment: (1) HBO therapy, (2) HBO therapy + scaling, (3) scaling, and (4) control. Highly significant differences in gingival indices (GI), sulcus bleeding indices (SBI), probing depth (PD), attachment loss (AL), plaque index (PLI), and gingival blood flow (GBF) were seen in the HBO, the HBO + scaling and the scaling groups compared to the control group. The numbers of subgingival anaerobes as well as the numbers of rods, *fusi* and *spiro* were reduced markedly in these three treatment groups. Statistically greater differences in clinical indices, GBF, subgingival anaerobes number and number of rods were found. Fusi and spiro were found by comparison of HBO + scaling and HBO groups, as well as between the HBO + scaling and scaling groups, but no significant differences were observed in GI, SBI, PD, or AL between the HBO and scaling groups. Results of this study confirmed that HBO therapy combined with scaling and root planning was the most beneficial in the treatment of periodontitis and treatment effect would last >1-year.

Chen *et al.* in 2003[11] studied the therapeutic effects and holding time of HBO on severe periodontitis patients. 30 cases with periodontitis were selected and randomly divided into two groups, that is, the HBO group was exposed to a pressure of 0.25 MPa and control group were rinsed. GI, SBI, PLI, PD, AL and gingival crevicular fluid (GCF) were measured during the first and last clinical visits and 1-year after HBO therapy. The GBF was measured by laser Doppler flow meter. HBO can decrease GI of patients with periodontitis by 1.1, SBI by 1.2, PD and AL by 0.7 mm, volume of GCF by 2.0, and significant differences could be seen in the above indices between pre- and post-HBO therapy. The GBF had a 1.8-fold increase after HBO exposure. GI and SBI 1-year after HBO therapy were larger than that of time after HBO therapy. There were no significant differences in the PLI, PD, AL, GCF, and GBF between post HBO therapy and 1-year after HBO therapy. It was concluded that HBO had good therapeutic effect on severe periodontitis, and this effect lasted for >1-year.

Coulthard *et al.* in 2003[12] investigated the effectiveness of HBO therapy for irradiated patients who required dental implants using data from randomized control clinical trials (RCT's). There was no RCT's comparing with and without HBO for implants treatment in irradiated patients.

Investigation conducted by Chen *et al.*[13] provided good evidence that HBO_2 inhibits the growth of subgingival obligate anaerobes and facultative anaerobes and *Bacteroides melaninogenicus* thus promoting healing of periodontium, which will be of help in the treatment of aggressive periodontitis (AgP). HBO_2 therapy combined with scaling and root planning appears to be even better for synergistic treatment of AgP. The effects can last >2 years. The mechanism of HBO_2 therapy on AgP is not clear. However, following might be involved in the mechanism of action of HBO_2: An inhibition of growth and reproduction of subgingival plaque and anaerobes, and in particular the growth of obligate and facultative anaerobes and *B. melaninogenicus* at the base of the pockets. Collectively, these results indicated a therapeutic benefit of HBO_2 for the treatment of patients with AgP.

Microorganisms can secrete different enzymes that can destroy collagen and growth factors. When the oxygen concentration in gingival tissue is low, amount of bacteria in the periodontal pockets increases. HBO_2 seems to effectively decrease the amount of bacteria and simultaneously inhibit collagenase secretion. A study by Rabkin and Hunt showed that oxygen at 2.0 ATA could inhibit the growth of certain pathogens related to periodontitis. HBO_2 has also shown bactericidal/bacteriostatic effects on *Actinomyces, Bacteroides,* and *Streptococcus.*[14]

CONCLUSION

Hyperbaric oxygen has been successfully used in several medical applications. The therapeutic effect is related to elevated partial oxygen pressure in the tissues. Dental patients too could be benefited with this treatment approach along with the advancement in the medicines and technical equipment's used in the patient care.

KEY POINTS

- A mode of medical treatment in which the patient is entirely enclosed in a pressure chamber and breathes 100% oxygen at a pressure >1 ATA
- Addition of HBO obviates the need for frequent surgical procedures, promotes healing and early mobilization of the patient
- Reduces length of hospitalization and thereby overall treatment and rehabilitation costs
- In periodontics, HBO reduces leucocyte adhesion in reperfusion injury, preventing the release of proteases and free radicals which cause vasoconstriction and cellular damage.

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