



Hyperbaric Oxygen Therapy

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Article initiated by: Mário Lima Fontes

All contributors: Mariana Leuzinger Dias, João Tavares Ferreira, Fernando Falcão Reis, Mário Lima Fontes, Neelakshi Bhagat, MD, FACS

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Hyperbaric Oxygen Therapy

Hyperbaric oxygen therapy (HBOT) is characterized by the administration of 100% oxygen at pressures exceeding 1.0 atmosphere absolute (ATA).^[1] According to the Undersea and Hyperbaric Medical Society, pressurization should be at least 1.4 ATA or higher for the therapy to be considered HBOT. The effect of HBOT is dose dependent.^[2]

Description

HBOT has been used as a treatment modality for various diseases, such as retinal artery occlusion, retinal vein occlusion, diabetic macular edema, and acute optic neuropathy or as an adjunct to surgical or pharmacologic interventions; however, to be very clear, this mode of therapy it is not approved for use in these conditions by regulatory authorities. Many cases reports and case series of successful treatment have been described in the literature with this modality.

HBOT can be carried out in either a monoplace or a multiplace chamber. In a monoplace chamber, a single patient is accommodated, the entire chamber is pressurized with 100% oxygen, and the patient breathes the ambient chamber oxygen directly. A multiplace chamber holds 2 or more people and is pressurized with compressed air while patients breathe 100% oxygen via masks, head hoods, or endotracheal tubes.^[3]

Indications

The Undersea and Hyperbaric Medicine Society (UHMS) approves the use of hyperbaric oxygen for 14 conditions for which there is thought to be reasonable scientific evidence or well-validated clinical experience:^[4]

1. Air or Gas Embolism;
2. Arterial Insufficiencies:
 1. **Central Retinal Artery Occlusion (CRAO)**
 2. Selected Problem Wounds
3. Carbon Monoxide Poisoning;
4. Clostridial Myonecrosis (Gas Gangrene);
5. Compromised Grafts and Flaps;
6. Acute Traumatic Ischemias;
7. Decompression Sickness;
8. Delayed Radiation Injuries (Soft Tissue and Bony Necrosis);
9. Sudden Sensorineural Hearing Loss;
10. Intracranial Abscess;
11. Necrotizing Soft Tissue Infections;
12. Refractory Osteomyelitis;
13. Severe Anemia;
14. Acute thermal burn injury.

However, other eye diseases are among the off label uses of HBOT and several reports support its use in ophthalmology.

Historical landmarks

- In 1662, Henshaw, a British clergyman, used organ bellows to change the atmospheric pressure in a sealed chamber called the Domiciliumin, to promote good health and for treating diseases.^[5]
- Almost two centuries later, hyperbaric therapy was revived in France Junod (1834) and later by Pravaz (1837) to treat pulmonary and a variety of other diseases.^[6]
- In 1879, Fontaine performed surgeries in a pressurized mobile operating room.^[6]
- In 1921, in Kansas City, Dr. Orville J. Cunningham built a multiplace hyperbaric compressed air chamber to treat patients with a wide variety of ailments. Later, Cunningham built the biggest hyperbaric chamber in the world (a five-story, 20-meter diameter, hyperbaric building), which had the potential to reach up to 3 atm pressure. This facility was used to treat a diverse collection of ailments but was dismantled in 1937 lack due to a lack of scientific evidence for the use of hyperbaric oxygen therapy.^[7]
- In the 1930s, the US navy began using hyperbaric oxygen therapy for treating decompression sickness.^[6]
- The biggest boost to using hyperbaric oxygen therapy came from the Netherlands with Boerema's work on animals which was so successful that a large operating hyperbaric chamber was built in Amsterdam to conduct complicated heart and lung surgeries. Boerema and his team successfully treated clostridial myonecrosis and severe anemia in pigs.^[8]
- Another luminary who contributed immensely to the use of hyperbaric oxygen therapy is Richard A. Neubauer, who opened the Ocean Hyperbaric Neurologic Center in Lauderdale by the Sea, for treating patients with central nervous system disorders.^[6]
- To address concerns of lack of scientific knowledge and regulation, a nonprofit organization, now known as Under sea and Hyperbaric Medical Society was formed in 1967.^[6]

Physiologic Basis

Under normal conditions at sea level (where ambient air is composed of approximately 21% oxygen), plasma hemoglobin is almost entirely saturated, and there is minimal dissolved plasma oxygen. Under hyperbaric conditions breathing 100% oxygen, there is a marked increase in oxygen dissolved in plasma and, consequently, in tissue oxygenation.^[1]

The value of HBOT is also explained by the physical properties of the gases. At higher pressures, gas bubble size decreases, so oxygen can move into smaller vessels and reduce extravascular tissue damage.^[1]

Another main physiological effect of oxygen relates to vasoconstriction. Increased levels of oxygen cause a decrease in local nitric oxide (NO) production by endothelial cells, thereby leading to vasoconstriction. However, even with reduced blood flow, more oxygen is delivered to the tissues as a result of the hyperoxic state. Further, rapid vasodilation occurs after the ending of hyperbaric oxidation.^[9]

Other effects of HBOT are fibroblast proliferation, leukocyte oxidative killing, toxin (clostridial) inhibition, and antibiotic (fluoroquinolones, amphotericin B, and aminoglycosides) synergy.^[10] Moreover, oxidants, which are increased under HBOT, may serve as cellular messengers to promote healing.^[11]

Reported uses in Ophthalmology

Vascular diseases of the retina

Even though CRAO is the only vascular disease of the retina where HBOT is recommended by the UHMS, there are many reported uses in other conditions.

Central retinal artery occlusion

Effective treatment of CRAO is of particular interest due to the acute onset of vision loss, significant functional morbidity, and relative intractability. By enhancing oxygen delivery to ischemic retinal tissue, HBOT theoretically, can serve as an acute therapy for CRAO aimed at minimizing vision loss before recanalization.^[12] During HBOT, the increased partial pressure of oxygen has marked effects on ophthalmic vasculature, causing reversible vasoconstriction in both the arterioles and venules of the retina.^[9] Despite this vascular response, the retina can remain adequately oxygenated due to the tremendous elevation of plasma oxygen within the choriocapillaris during treatment.^[13] As a result, the hyperoxygenated choroidal vasculature can satisfy the entire retinal oxygen demand, with animal studies showing adequate oxygenation despite retinal artery occlusion.^[14]^[15] Therefore, the presence of an intact choroidal circulation is essential for HBOT to successfully reverse vision loss.^[6]

The time between symptom onset and initiation of HBOT and fundus findings, including the presence of a cherry-red spot, change in macular thickness, and degree of disorganization of retinal inner layers, are emerging as clinical markers that predict HBOT response.^[15]

Widespread recognition of CRAO as an emergent condition, often requiring prompt administration of multiple lines of treatment, has led to the reduction in HBOT referral time. Multiple recent case reports have attributed their success to the prompt delivery of HBOT from CRAO onset.^[16]^[17]^[18] Given that irreversible damage begins after 1.5 hours of retinal ischemia, the best outcomes have been documented when applying HBOT within a couple of hours of symptom onset.

Hadanny et al. proposed that using a visible cherry-red spot as a physiologic indicator for reduced HBOT efficacy may be more predictive than the time lag from CRAO onset.^[19] Other negative predictors of HBOT success are pathologic retinal architecture and macular thickening, which are both direct signs of ischemic severity and progression.^[20]

A positive physiologic indicator of HBOT success is the presence of cilioretinal collaterals.^[21]

In 2001, a retrospective study of 35 HBOT-treated patients and 37 matched controls demonstrated improvement of visual acuity in 82% of patients in the hyperbaric group compared to only 29.7% in the untreated group. The mean improvements as measured by the Snellen chart were 0.1957 and 0.0457 respectively.^[22] This is consistent with the findings of previous case studies showing enhancement of visual acuity in patients with CRAO post-HBOT.^[23]^[24] However, a clinical trial in 2000 investigating HBOT in CRAO patients 1 day after symptom onset reported no significant difference in vision between the HBOT group and the control group receiving standard therapy of ocular massage, paracentesis, and intravenous acetazolamide.^[25] Recent studies have reported promising clinical outcomes, with many indicating near or complete restoration of visual acuity post-HBOT.^[12]^[16]^[17]^[18]^[26]

As per the 14th edition of the UHMS hyperbaric oxygen indications, the protocol for hyperbaric oxygen treatment for CRAO is as follows:^[4]

1. Compress to two ATA;
2. If vision improves significantly at 2 ATA, then remain at this depth for 90 minutes;
3. If vision fails to improve after 30 minutes at 2 ATA, then compress to 2.4 ATA, and if vision improves at this depth, perform according to the US Navy recompression treatment table no 6;
4. In case of no improvement:
 1. Discontinue treatment;
 2. Continue with normobaric oxygen therapy.

HBOT is a non-invasive and may be an option for CRAO patients, however, ^[27] further studies are needed to prove its efficacy. A small retrospective study in Australia in 2023 reported no improvement in patients with BRVO with HBOT.^[28]

Diabetic retinopathy

HBOT was reported to be possibly useful in the treatment of diabetic retinopathy.^[29]

Experimental studies showed that hyperoxia decreases VEGF expression in the adult retina and that HBOT ameliorated the blood-retinal barrier breakdown (BRB). Hence, it can prevent and treat persistent macular edema due to BRB breakdown in patients with diabetes.^[30]

Treatment with HBOT in a case of diabetic macular edema resulted in bilateral improvement in vision from 20/125 to 20/63 in the right eye and 20/320 to 20/160 in the left eye after 14 sessions over the course of 1 month. Although macular edema reoccurred in the succeeding months, HBOT controlled visual decline each time.^[31] Similarly, two clinical trials of patients with macular edema both reported improvements in visual acuity after chronic HBOT. One showed improvement by 2 lines or more in 68% of eyes whereas the other reported a mean increase of 3.5 lines.^[32]^[33] Recently, one prospective cohort study found that HBOT has a thinning effect on the macula in diabetic eyes, further supporting the hypothesized mechanism of hyperbaric oxygen on BRB breakdown.^[34] These early results are encouraging and more studies are needed to elucidate the efficacy of HBOT on diabetic retinopathy and retinal artery occlusions.

Other vascular conditions

Isolated cilioretinal artery occlusion treated with twenty sessions of HBOT (120 minutes, 2ATA) and in another case combined with nonischemic central retinal vein occlusion secondary to high altitude (daily 2 hours of HBOT at 2.5 ATA for 11 days) have also shown good vision improvement.^[35]^[36]

HBOT in patients with retinal vein obstruction improved visual prognosis and in patients who had retinal vein obstruction associated with cystoid macular edema, HBOT improved macular edema and visual acuity.^[33]^[37]

Cystoid macular edema resistant to other therapy modalities was shown to respond to HBOT and to improve visual acuity.^[38]

Multiple sessions of HBOT have been associated with an improvement in visual function and retinal appearance in patients with Purtscher retinopathy.^[39]

Early HBOT has been used for bilateral blindness during hemodialysis.^[40]

Scleral melting and necrosis

Scleral thinning, melting or necrosis may result from the use of beta-irradiation or mitomycin C to prevent recurrence in patients with pterygium.^[1] HBOT showed promising results in these patients, increasing episcleral blood flow and scleral thickness.^[41] Hyperoxia and induction of angiogenesis and fibroblast proliferation seem to be responsible for the benefit from HBOT in these scenarios.^[1]

Ocular infections

Mucormycosis is a rare but serious condition in immune-compromised patients that may involve the orbit. Treatment of this disorder consists of intravenous amphotericin B administration and debridement of the necrotic tissues. Nevertheless, the mortality rate is high. HBOT might be a complementary treatment in these patients. HBOT has direct anti-fungal activity by increasing the production of oxygen-based free radicals. Besides, it has several indirect anti-microbial properties, like a reversal of growth-promoting lactic acidosis, restoration of phagocytosis and augmentation of oxidative burst by polymorphonuclear leucocytes. Lastly, it enhances the antifungal action of Amphotericin B.^[6]

Price and Steves were the first to employ HBOT in a patient with rhinocerebral mucormycosis who had not responded to medical treatment and had refused surgical intervention.^[42] Later, others reported that amphotericin B and surgical intervention along with HBOT had better results.^{[43][44][45]} In a detailed review of cases with rhino-orbital-cerebral mucormycosis, Yohai et al found that HBOT had a favorable effect on prognosis.^[46]

Corneal edema and anterior segment ischemia

There are good physiologic reasons that may account for why HBOT may have beneficial effects on certain ocular diseases. Corneal edema with various etiologies and anterior segment ischemia, especially when associated with sickle-cell anemia, seem to be a proper indication for HBOT.^{[47][48]} Although it is not clinically proven, transcorneal delivery of oxygen was suggested for the treatment of anterior segment necrosis and rubeosis iridis.^[49]

Proliferative vitreoretinopathy due to sickle-cell disease

It was shown that proliferative vitreoretinopathy due to sickle-cell disease may also benefit from HBOT.^[50]

Glaucoma

The vascular theory in glaucoma seems to be a scientific rationale for the application of HBOT. In this sense, some studies reported an improvement in the visual field without significant changes in intraocular pressure, in patients with glaucoma.^{[51][52]} Conversely, another study linked HBOT to a decrease in intraocular pressure.^[53] Further studies are required to determine the role of HBOT in glaucoma.

Optic neuropathy

HBOT showed contradictory results in radiation optic neuropathy. In one study, treatment administered in the first 72 hours of visual loss improved visual acuity to baseline level. On the other hand, HBOT administered at weeks 2 to 6 of visual loss did not improve visual acuity.^[54] Conversely, visual acuity improved for a patient who received HBOT for radiation-induced optic neuropathy for 17 weeks after the onset of optic neuropathy.^[55] However, in a review by Levy and Miller, no beneficial effect was shown for cases with radiation-induced optic neuropathy.^[56]

HBOT provided significant improvement of visual acuity and field in two patients with non-arteritic anterior ischemic optic neuropathy.^[57] However, Arnold et al showed in a controlled clinical trial that HBOT was not effective for the treatment of non-arteritic anterior ischemic optic neuropathy, which was later confirmed by Mathews in a review.^{[58][59]}

Retinitis pigmentosa

Poor blood circulation is a well-known component of retinitis pigmentosa.^[1] HBOT in patients with retinitis pigmentosa improved macular edema and visual acuity.^[60] It also improved electroretinogram responses and the metabolism of retinal photoreceptor cells.^[61]

Other reported uses

HBOT has been used in other conditions as macular detachment, macular hole surgery, toxic amblyopia (eg Quinine toxicity), uveitis, keratoendotheliosis, macular degeneration, refractory pseudomonas keratitis, pyoderma gangrenosum of the orbit, etc.^[6] One case report notes improvement in vision of a patient with acute onset PAMM with HBOT after 14 sessions of hyperbaric oxygen therapy.^[62]

Systemic complications of HBOT

General side effects of HBOT include oxygen convulsions, middle ear abnormalities, and lung rupture, along with claustrophobia in cabins for a single patient. Moreover, squeezing sensation in the chest, retrosternal burning sensation, and temporary lung dysfunctions such as dry cough may occur.^[1]

The only absolute contraindication to HBOT is an untreated tension pneumothorax. Relative contraindications include impaired pressure equalization and cardiac disease.^[1]

Clinical and experimental evidence show that HBOT may be safe when used in pregnancy.^[63]

Ophthalmologic complications of HBOT

HBOT has been reported to be safe, as long as each session does not exceed 20 minutes and oxygen pressure is below 3 atmospheres. Longer exposures may result in a transient narrowing of the visual field. Eyelid twitching is the most common manifestation of oxygen toxicity and may precede a full-blown seizure.^[1]

HBOT is contraindicated in patients with therapeutic intraocular gas, to avoid severe intraocular pressure rise.^[64] Induction of fibroblastic proliferation and unstable refraction may lead to unsatisfactory results in Keratorefractive surgery procedures, especially for photorefractive keratectomy. Therefore, all keratorefractive surgeries should be postponed unless otherwise indicated for those patients undergoing HBOT.^[1]

The rise in arterial oxygen tension leads to the production of reactive oxygen species (ROS), which can incur oxidative stress on the crystalline proteins of the lens. Consequently, ocular complications such as myopia or cataract formation have been observed.^[15]

Hyperoxic myopia

Progressive myopia is a well-known and commonly reported complication of HBOT that occurs at a rate of approximately 0.25 diopters per week.^[1] It is estimated that 60% of patients undergoing HBOT will develop at least a 1-line change on the Snellen eye chart.^[65] In 1978, Lyne documented a myopic shift ranging from 0.5 to 5.5 diopters in a series of patients receiving HBOT at 2.5 ATA. Myopia progressed in severity throughout treatment and gradually reversed upon completion.^[39] A recent longitudinal study reported similar findings, with an average myopic shift of 0.95 diopters after 30 sessions at 2.4 ATA.^[66] The myopic shift generally reverses within 3 to 6 weeks after discontinuing HBOT but can persist for as long as 6 to 12 months.^[22]

Hyperoxic myopia is thought to be related to oxidative changes that alter the refractive index of the crystalline lens.^[66] Lenticular involvement is further supported by the absence of myopic shifts in pseudophakic eyes,^[67] and previous studies have excluded other factors such as changes in retinal thickness due to vasoconstriction, axial length and anterior chamber depth, corneal shape and lens thickness.^[15] It is thought that HBOT affects the refractive index of the nucleus and the deeper layers of the lens cortex.^[66] Since the refractive index gradient of the lens is determined by its composition of structural proteins and water, hyperoxia may modify protein concentrations and local water distribution, leading to a reduction in backscattered light and optical density.^{[68][69]} It may be possible to reduce the risk of refractive change by decreasing the pressure of oxygen in direct contact with the eyes.^{[70][71]} In studies of patients given HBOT either by a hood or oronasal mask, the myopic shift was markedly lower in the latter group where the eyes were kept from direct contact with hyperbaric oxygen. Patients receiving oxygen by mask also demonstrated a lower incidence of myopia and shorter recovery times for visual acuity. This suggests that lenticular oxygen toxicity may stem from both local and systemic effects of hyperoxia.^[71] It has also been reported that the risk of oxygen toxicity can be reduced by providing 5 minutes of room air for every 20 to 25 minutes of HBOT,^[72] and there is evidence to suggest that decreasing treatment pressure reduces the likelihood of myopic change.^{[73][74]} Taking these steps to minimize lenticular oxygen toxicity may prevent the development or limit the severity of hyperoxic myopia, preserving the quality of life for patients undergoing treatment.^[15]

Cataracts

Another potential, but lesser seen complication of HBOT is the formation and progression of nuclear and cortical cataracts.^[15] Palmquist et al. reported the occurrence of cataracts in patients undergoing extensive HBOT regimens involving at least 150 exposures at 2.0 to 2.5 ATA.^[75] Current HBOT regimens seldom exceed 75 sessions, and there is evidence to suggest that many indicated chronic conditions can be appropriately treated with as few as 30 sessions.^[76] Although cataracts are less likely to form under these durations, there have been reported cases in patients receiving as few as 46 treatments at 2.5 ATA.^{[77][78]} A prospective study conducted by Riedl et al. also found a significant increase in nuclear color and opalescence, two characteristics of nuclear cataract, following 40 sessions of HBOT. The change in lens transparency appeared to be linearly related to exposure and persisted on examination 12 weeks after treatment.^[79]

Although the mechanism is not fully understood, it is believed that cataracts form due to oxidative stress to the crystalline lens.^[77] It has been suggested that the nucleus of the lens is particularly susceptible to oxygen toxicity as opposed to the cortex or the epithelium, due to the presence of lower amounts of important antioxidants such as glutathione, which is essential in maintaining the transparency of crystalline proteins in the lens.^[80]
[81]

Given that cataract formation is an irreversible complication of HBOT, it is important to adequately counsel patients on this possibility and its long-term implications prior to initiating treatment.^[15]

Further studies are needed to elucidate the role of HBOT in various ocular disorders.

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