Hyperbaric Chamber and Its Ophthalmic Applications

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Hyperbaric oxygen therapy has been used for centuries but it has been accorded more scientific reasoning only recently. Ophthalmic applications of this therapy is an off label use of hyperbaric oxygen but results of its use in Central Retinal Artery Occlusion and many other devastating ocular infections is paving the way for increasing acceptability by ophthalmologists. Recent protocols of therapy are being enunciated and safer hyperbaric chambers are being manufactured to help patients and doctors use this hereto underutilised modality of treatment.

Abstract

Introduction

Hyperbaric Oxygen therapy (HBOT) has been used for centuries by physicians and surgeons but only recently, it has been used to treat ophthalmic disorders. Treatment of ophthalmic disorders is an off label use of hyperbaric oxygen therapy. Having been tried in a large number of animal experiments and clinical trials, the HBOT is now gaining acceptance by the medical fraternity and is being utilised for a variety of systemic and ocular diseases. This form of therapy is given in a device called Hyperbaric chamber. Hyperbaric oxygen therapy has been defined by Committee on hyperbaric medicine, Undersea and Hyperbaric Medicine Society, as breathing 100% oxygen at a pressure greater than one atmosphere absolute (ATA).1

The concept of breathing oxygen under pressure is old, as, in 1662, Henshaw a British clergyman, used organ bellows to change the atmospheric pressure in a sealed chamber called the Domicilium, in which both hyperbaric and hypobaric environments could be created. He used it for promoting good health and for treating diseases.2 Almost two centuries later, hyperbaric therapy was revived in France by a French physicians Junod (1834) and later by Pravaz (1837) to treat pulmonary and a variety of other diseases. In 1879, Fontaine performed surgeries in a pressurised mobile operating room.3 However, HBOT use was not substantiated by any evidence or scientific proof. In 1860, it was introduced in North America by Dr Orville J Cunningham who started treating Spanish Influenza cases in the hyperbaric chamber. In 1928, he built a five story high steel sphere, 68 feet in diameter with 12 bedrooms containing all amenities of a good hotel. A variety of diseases were treated in this “hotel” but was dismantled in 1937 lack due to lack of scientific evidence for the use of hyperbaric oxygen therapy.4 In 1930s, the US navy began using hyperbaric oxygen therapy for treating decompression sickness. The biggest boost to using hyperbaric oxygen therapy came from 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.5 Another luminary who contributed immensely in the uses 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.

To address concerns of lack of scientific knowledge and regulation, a nonprofit organisation, now known as Undersea and Hyperbaric Medical Society was formed in 1967.

Hyperbaric Chamber

The European Council Directive 92/42 (1998) states, that the hyperbaric chamber be treated as a medical device with all its consequences. They directed that its construction should meet the safety standards for human occupancy as specified in ASME PVHO 1 or its International equivalent. Hyperbaric system consists of the following:

Hyperbaric Chamber(s): Multiplace (Figure 1), monoplace, and medical devices Two other hyperbaric chambers which are also used are: mobile chambers and those used for diving purposes. Gamow bag is an inflatable type of hyperbaric chamber generally used in high altitude (Figure 2). Defence Research and Development Organisation (DRDO) has made such a bag for use in high altitude. This is not a true hyperbaric device as its pressure is less than or only 1 ATA.

Air Compressors: One or more for generating pressurised gas to compress the hyperbaric chamber

• Volume tanks for storing compressed air
• Oxygen supply
• Fire suppression system

Hyperbaric Chambers: These are chambers in which oxygen is given under pressure and the manner of their construction enables them withstand pressurization.

Monoplace Chambers and Multiplace Chambers (Table 1)

The hard shell chamber is made from steel and acrylic. Its configuration may be horizontal, vertical, rectangular rib enforced and spherical. The design, fabrication and testing of the chambers is governed by American Society of Mechanical Engineers Pressure vessels for human occupancy (ASME-NHO-1). Most clinical multiplace chambers have two to three compartments, called “locks”. The locks consist of an entry compartment and one to two treatment ones. Mult Place chambers may also have wall mounted passes through which food, medication and equipment is passed into the lock. Hull penetrators also present in the chamber are used for passing piping, wiring and lighting through the pressure boundary. View ports also present in the chambers,
are constructed as per specifications of ASME PVHO1. The number of view ports required in one chamber depend on requirement of external viewing by both the patients and the observers outside the chamber and the light requirement inside the chamber. Pressure relief or “pop – off “valves are inbuilt to release pressure if it exceeds the design limit of the chamber. Soft hyperbaric chambers are inflatable and are not considered true hyperbaric chambers as they can only achieve a maximum depth of 1.3 ATA internally, while the lowest protocol for hyperbaric medicine used by physicians is 1.5 and above. This type of chamber is pressurised with near 100% oxygen and the patient breathes the ambient oxygen directly making it more liable to fire accidents. Duo place chambers are also available which can accommodate one patient and one attendant only. The hyperbaric chambers are classified in to three types depending on the pressure7,8 (Table 2)

| Monoplace Chambers | Multiplace Chambers |
|--------------------|---------------------|
| Designed to treat one patient at a time | Can accommodate 20 or more patients |
| Minimum space necessary for this chamber is 256sq ft. | Occupies at least 2000 to 2500 sq ft |
| Patient lies on a gurney which is pushed into the chamber, no attendant space inside the chamber | Greater space available, patients can lie down and sit inside the chamber; patients can be monitored by an attendant who remains inside the chamber for the period of therapy |
| Lower cost | Higher cost |
| Portable | Static |
| Treatment specific to patient with no risk of decompression sickness to the patient or staff. | Variety of illnesses treated at the same time |
| Problems : Confinement anxiety, feeling of claustrophobia | Potential risk of cross infection |
| Disadvantages: limited access to patients in the chamber, increased risk of fire due to oxygen use. Requires use of muscle relaxants and higher sedation; requirement of special IV pumps located outside the chamber; No physical therapy can be administered on the patient. | Disadvantages: Patients have to breathe oxygen by well fitted head hoods, oronasal masks or endotracheal tubes; requirement of physician and special equipment |
| Advantages: Privacy for patient; isolation in case of infected patient; easy for patient observation; no face mask required for the patient; no special decompression measures required | Advantages: reduction of fire hazard, physical therapy can be performed inside the chamber, psychological testing, computer based mental exercises and research in sports physiology and physical therapy research can be carried out inside the chamber. Exhaled gas from either oxygen delivery system is routed out of the chamber to minimise accumulation inside the chamber to decrease risk of fire |

Oxygen level normally cannot be raised above 3 ATA

Oxygen pressure can be raised to 6 ATA for treating air embolism and decompression sickness

Oxygen under pressure is present in the chamber

Air is present in the chamber

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**Figure 1:** Multi place hyperbaric chamber, Patients inside chamber with oxygen hoods. Indraprastha Apollo Hospital, New Delhi. Courtesy: Dr Tarun Sahni

**Figure 2:** HAPO bag (Courtesy: HAMRC)
layer of the retina with sufficient partial pressure of oxygen until blood flows through the central retinal artery again. Administration of carbogen (95% oxygen and 5% carbon dioxide), intraocular pressure lowering therapy, ocular massage and paracentesis may also be given though the efficacy is debatable but better restoration of visual acuity has been found hemodilution and hyperbaric therapy. Presence of an intact choroidal circulation is essential for HBOT to successfully reverse vision loss. In a large number of studies, overall 65% of cases showed improvement with this therapy. Heather Murphy-Lavoie, Frank Butler and Catherine Hagan have described the titration of oxygen therapy in CRAO if the symptom onset is within 24 hours or less.6

As per the UHMS hyperbaric oxygen Indications, 13th Ed., Weaver, the protocol for hyperbaric oxygen treatment for CRAO is as follows:

a) Compress to two ATA
b) If vision improves significantly at 2 ATA, then remain at this depth for 90 minutes
c) 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 a US Navy treatment Table no 6. In case of no improvement after initial US Navy table No 6, options are:
   aa) Discontinue treatment
   bb) Continue with normobaric oxygen therapy

Not only CRAO, even Cilio retinal artery occlusion treated with twenty sessions of HBOT (120 minutes, 2ATA)20 and in another case, Cilio retinal artery occlusion associated 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 restoration of vision.20

2. Decompression sickness (DCS) with ocular signs and symptoms: This condition occurs in deep sea divers and in persons flying in unpressurised airplanes due to entry of nitrogen and oxygen from the lungs into the blood stream and tissues without sufficient time having been given to allow their slow release. Hyperbaric oxygen is used for recompression. The ocular signs of DCS are nystagmus, pupillary changes, severe accommodative and convergence insufficiency visual field deficits or air bubbles in the retinal vessels.21,22

3. Arterial gas embolism with ocular signs and symptoms- Treatment of gas embolism in both arteries and veins with hyperbaric oxygen is an accepted form of therapy as it reduces the size of the gas bubbles, allowing them to pass through the microcirculation, thus resolving the embolic phenomenon. It also allows increased absorption of nitrogen due to increased partial pressure of oxygen.23,24

4. Ocular gas gangrene: The orbital and ocular tissues are an uncommon site of gas gangrene and good results have been found when treatment has been rendered with surgical debridement, antimicrobials and hyperbaric oxygen. Boerema and subsequently Brummelkamp successfully treated cases of gas gangrene by hyperbaric oxygen therapy which acts by formation of oxygen free radicals.
radicals in relative absence of superoxide dismutases, catalases and peroxidases.\textsuperscript{2,25,26}

5. Nectrotizing soft tissue and fungal (zygomycosis) infections involving the orbit: Rhino- orbito – cerebro mucormycosis has high fatality rate of approximately 35-70% and close to 100% in case of disseminated disease with cerebral involvement. A large number of studies have reported good results with adjunctive hyperbaric oxygen with surgical debridement and Amphotericin B/Posaconazole/Caspofungin in immunocompromised patients. The mechanisms by which HBOT helps to treat such infections are:

(a) Hyperoxic conditions have direct anti fungal activity which has been seen in vitro atmospheres by increased production of oxygen based free radicals

(b) HBOT is able to kill mutant fungi which lack antioxidative enzymes

(c) It has a number of indirect anti microbial properties, like reversal of growth promoting lactic acidosis, restoration of phagocytosis, augmentation of oxidative burst by polymorphonuclear leucocytes

(d) Enhancement of antifungal action of Amphotericin B

(e) Tissue healing is augmented by HBOT by increasing tissue oxygen levels, restoring normal fibroblast function, increasing deposition of collagen and increased angiogenesis and inflammatory cytokines secretion.\textsuperscript{27,28}

6. Carbon monoxide poisoning with visual sequelae: Hyperbaric oxygen can be used successfully to treat carbon monoxide poisoning.\textsuperscript{29}

7. Radiation optic neuropathy/retinopathy: Any source of external radiation used for treating head and neck and intraocular tumors can result in a chronic ocular injury which is progressive due to retinal vascular endothelial damage at the DNA level. Incidence of Radiation optic neuropathy/Retinopathy varies from 3-20%. HBOT has been advocated as a supportive adjunct in its treatment as it improves oxygenation and sensitization of hypoxic cells.\textsuperscript{30}

8. Compromised periorbital skin grafts and flaps: Studies have found Hyperbaric oxygen to help compromised grafts to survive in properly selected patients. 29% improvement in graft survival was found in a prospective blinded clinical trial where HBO2 was administered prior to skin grafting and for three days thereafter.\textsuperscript{31,32}

9. Scleral ischemia or necrosis/ recurrent pterygium: Adjunctive HBOT has been used effectively to treat scleral necrosis following its treatment with beta radiation, Mitomycin – C, by some authors. Assaad and colleagues had no recurrence in patients of recurrent pterygium who had undergone surgical excision, limbal auto graft and treatment with HBOT on the first post operative day and subsequently underwent four treatments.\textsuperscript{33}

Potential Indications

1. Anterior segment ischemia (especially post operative)

2. Ischemic optic neuropathy

3. Ischemic central retinal vein occlusion

4. Branch retinal artery occlusion (especially with visual loss)

5. Cystoid macular oedema with Central retinal vein occlusion

6. Cystoid macular oedema with post surgical inflammation

7. Macular degeneration: Significant visual improvement has been found in a few patients treated with thirty 60 minutes treatment at 1.5 ATA in advanced macular degeneration.\textsuperscript{34}

8. Cystoid macular oedema with intrinsic inflammatory disorders

9. Refractory pseudomonas keratitis

10. Pyoderma gangrenosum of the orbit

Other Reported Uses

1. Toxic Amblyopia (eg Quinine toxicity)

2. Retinitis pigmentosa

3. Macular hole surgery

4. Diabetic retinopathy

5. Uveitis

6. Keratoendotheliopathy

7. Sickle cell hyphema

8. Retinal detachment(including associated sickle cell disease)

9. Glaucoma

Ocular Contraindications For Hyperbaric Oxygen Therapy

1. Hollow orbital prosthesis

2. Presence of intraocular gas in anterior chamber or in vitreous cavity: Gas in the vitreous cavity for repositioning the retina and in the anterior chamber for maintaining the apposition of Descemet’s membrane to stroma is a contraindication to HBOT. Ocular barotrauma can occur during compression and rise of intraocular pressure with resultant CRAO may result during decompression due to HBOT.\textsuperscript{8}

Toxic Effects Of Hbot

1. Barotrauma : blocked Eustachian tube resulting from an inability to equalise pressure on both sides of the tympanic membrane

2. Pneumothorax and air embolism : due to tear in the pulmonary vasculature

3. Pulmonary and neurological oxygen toxicity

4. Retrolental fibroplasias

5. Cataracts\textsuperscript{35,36}

6. Transient reversible myopia (20%)- Myopia is a transient phenomenon due to lenticular nuclear changes, increases during treatment and decreases within a few weeks of termination of HBOT.\textsuperscript{37,38}

7. Hypersensitivity to oxygen: These can be prevented by maintaining pressures below 3ATA and length of therapy, less than 120 minutes.
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