Hyperbaric Oxygen Therapy

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HISTORY & INTRODUCTION

The modern clinical application of hyperbaric oxygen therapy began in the late 1950's, in parallel with an increased understanding of blood gas analysis and gas exchange physiology. Use of oxygen at high pressures, for treatment of decompression sickness had previously been documented,[1] but it remained an isolated medical curiosity.

Exposure of patients to hyperbaric pressures for therapeutic purposes had been introduced in several large facilities even decades before, following the principles elucidated by Paul Bert. These early applications, however, suffered from an over optimistic view. Hyperbaric spas flourished in 1900's 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.[2,3] In the early 1960's two institutions pre-eminently 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 Glasgow, where various anaesthetic and surgical aspects of HBO were applied and discussed. Among these were treatment of necrotizing infections and anaesthesia under hyperbaric conditions.

In 1968 Duke University in North Carolina expanded a long-standing programme of environmental physiology with the construction of inter-connected multiplace hyperbaric chambers.

Since 1970, most of the instructional courses research work and guidance has been provided by Underseas and Hyperbaric Medical Society (HQ in Kensington, MD).[4] This medical organization publishes guideliness for hyperbaric oxygenation every 2-3 years.

DEFINITION

Hyperbaric oxygen therapy is defined as inhalation of oxygen at increased pressure, for potential therapeutic benefit in a variety of clinical situations.

INDICATIONS AND RATIONALE

There are two sets of indications – first, those issued by Hyperbaric Medical Society :-

Gas Bubble Disease
- Air embolism
- Decompression sickness

Poisoning
- CO
- CN
- CCL_4
- H_2S

Infections
- Clostridial myonecrosis
- Other soft tissue necrotizing infections
- Refractory chronic osteomyelitis
- Mucormycosis

Acute Ischemia
- Crush injury
- Compromised skin flaps

Chronic Ischemia
- Radiation necrosis (soft tissue, radiation cystitis and osteoradionecrosis)
- Ischemic ulcers, including diabetic ulcers

Acute Hypoxia
- Support of oxygenation during therapeutic lung lavage
- Exceptional blood loss anemia (when transfusion is delayed or not available)

Thermal Injury (Burns)
- Brown recluse spider envenomation
- Second set of indications are those published in Journal of Hyperbaric Medicine, which are updated from time to time (1999 latest).[5]

Regional Hypoxia
- Compromised graft flap
- Osteoradionecrosis
- Problem wounds and ulcers

Crush Injuries

Thermal Burns

Global Hypoxia

CO, CN intoxication
Severe anemia

Infection

Clostridial myonecrosis
Necrotizing fascitis
Refractory osteomyelitis
Rhinocerebral mucormycosis

Gas Lesion Conditions

Gas embolism
Decompression sickness

RATIONALE IN GENERAL

The principal rationale of HBO therapy is to decrease tissue O_2 tension. So 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 rationale for use in gas lesion diseases.

Regional Hypoxia

Rationale for use – two fold.

1. Large O_2 gradient possible with an inspired O_2 partial pressure of 2 – 2.5 ATA allows some degree of O_2 delivery and O_2 tension elevation in the hypoxic zone, unless there is a complete absence of blood flow that may occur due to occlusion of large proximal blood vessels.
2. Second benefit – Stimulation of angiogenesis in previously hypoxic areas.

Examples

Compromised surgical graft / flap : HBO has been shown to improve the survival of those flaps which are clearly compromised after surgery. Numerous trials have demonstrated improved graft survival with intermittent HBO therapy.[6,7]

Osteoradionecrosis : HBO has been shown to be of benefit in more chronic forms of regional hypoxia such as osteoradionecrosis of head and neck. It is due to obliterative endarteritis and has been estimated to occur in upto 10% of patients receiving radiotherapy for carcinoma. It is characterized by necrosis, loss of tissue integrity and occasionally pathologic fractures.

Infection is generally secondary to loss of tissue integrity.

Repeated daily HBO therapy has been shown to promote repair and revascularization / neovascularization of necrotic one.

Enhanced angiogenesis is due to improved macrophage and fibroblast function.

Problems wounds particularly microvascular ulcers; here HBO reduces infection, improves healing and promotes granulation tissue base for subsequent skin grafting.

Crush injuries have also been successfully treated with HBO with the aim:- High arterial O_2 tension, improves oxygenation, causes modest vasoconstriction which may reduce post-traumatic edema and possibility of compartment syndrome. Similar principles may stand for its use in patients of burns. Besides a decrease in cost and length of hospital stay has been reported.[8, 9]

Global Hypoxia

Two major categories of global hypoxia for which hyperbaric O_2 has been shown to be a useful therapeutic tool.

1. **First and most important is CO and CN intoxication.**
   CO intoxication interferes with O_2 delivery and utilization because of its high affinity for hemoglobin, the leftward shift of O_2-Hb dissociation curve and interference with specific catabolic enzymes such as cytochrome oxidase especially under hypoxic conditions.
   Results : Tissue hypoxia; Neurological depression; Homodynamic instability.
   Patients who have had symptomatic CO exposures and who recover are at risk for delayed (2-20 days) neurological symptoms presumably due to ischemia and reperfusion injury in the CNS.
   Principle of therapy : The increased concentration of O_2 molecules achieve with HBO competes with CO for hemoglobin binding sites and speeds up the elimination of CO.
   Patients with a history of unconsciousness, hemodynamic instability or extremes of age, current evidence suggests that early use of HBO therapy significantly reduces incidence of delayed neurological sequelae. [10,11]
   A similar rationale exists for use of HBO in cyanide poisoning, but because pure cyanide exposure is rare and can be effectively treated by antidote therapy, HBO therapy should be reserved for refractory cases, or cases of mixed CN/CO poisoning which may be commonly seen following smoke inhalation.
2. **Severe, acute anemia** is a clear example of global hypoxia. At 2.5–3.0 ATA inspired oxygen sufficient arterial oxygen content is dissolved in plasma to meet metabolic needs making HBO useful to support life until RBC's become available for transfusion. This indication, however, is limited to only several hours of continuous therapy or to cases where intermittent treatment is sufficient to bolster a marginal O_2 delivery.[12]

Infections

Rationale : Anaerobic infections often develop in hypoxic areas because of a lack of adequate host response to the infection, and ischemic hypoxic areas often develop because of infection.

Part of the rationale for HBO therapy remains the relief of ischemia and improvement of host response to infection.

Some micro-organisms are sensitive to high O_2 tension attained through HBO, which is the second rationale for its use.

Clostridia myonecrosis / Gas gangrene : Clostridium perfringens is O_2 sensitive. It is rapidly progressive and life threatening due to a series of toxins produced by it.

While toxins are O_2 stable their production by the bacteria is inhibited by elevation of tissue O_2 tension to 300 mm Hg and above.[13] Rapid diagnosis is essential and HBO should be used early besides giving parenteral antibiotics and surgical debridement.

Necrotizing soft tissue infections : HBO my be adjunctive to conventional to allow a host response. It may have a role in Fournier's disease.

Refractory osteomyelitis : Osteomyelitis that has not responsed to conventional therapy is labelled refractory ostomyelitis and has been successfully treated with HBO. Rationale O_2 tension in area of infection is increased. Leucocytes and osteoclast function is improved. Susceptibility of micro-organisms to antibiotics is also increased.

HBO therapy has also been used to treat life threatening fungal infections like rhinocerebral mucormycosis and published reports have shown encouraging results.

Gas Lesion Disease

Two principal types are :-

Decompression sickness : An increased pressure leads to an increased tissue content of inert gas especially for more soluble gases like nitrogen. On decompression the tissues eliminate gas which is in dissolved state if decompression is gradual. But if decompression is rapid, then the gas is in undissolved state in blood, lymph, tissues resulting in a gas phase and mobile bubbles about 100 microns in size, but which may aggregate if blood flow is sluggish and form bigger gas bubbles leading to symptoms. There is general agreement now, that both stationary and mobile gas bubbles are the aetiology of decompression sickness syndromes.

C/F :
- Vague, non-specific complaints such as unusual fatigue.
- Cutaneous decompression sickness :- Mottled rash and pruritus.
- Pain-only decompression sickness :- Deep-seated pain in an around major joints (particularly knee and shoulders) referred to as "bends".
- Inner-ear syndrome :- Tinnitus, vertigo a hearing loss.
- Pulmonary decompression sickness :- Fulminant and life threatening and fortunately uncommon characterized by dyspnea, substernal pain and pulmonary edema.

Caisson's disease : It is a chronic form of decompression sickness due to repetitive prolonged low pressure 2 – 3 (ATA) exposures as experienced by pressurised Caisson workers. There is :- Aseptic osteonecrosis, usually of long bones (femur, humerus), years after pressure exposures. It may be due to bubble formation in the marrow cavity, elevating medullary pressures and causing ischemia and necrosis.

HBO therapy is the primary mode of therapy in decompression sickness syndromes. Rapidity of administrationi remains an important determinant of successful outcome. But symptom resolution coincident with HBO therapy as long as a week after pressure exposure has been reported.

Gas Embosom : It is an acquired condition, whereby gas is admitted to vasculature and circulation. *Causes :-* Pulmonary barotrauma.

Iatrogenic / traumatic causes (surgery, catheters, trauma, abortions, orogenital sex etc.). Endogenous gas bubbles are seen in decompression sickness.

Small volumes of air / gas admitted in I/V lines (< 50 ml) is efficiently trapped by pulmonary circulation and causes little or no effect. On the contrary even smaller volumes of air / gas admitted to arterial side can be catastrophic. Venous emboli may be arterialized through shunts.

Rationale use of HBO therapy : Two principal components – pressure and hyperoxia. Increased hydrostatic pressure will cause a decrease in volume of emboli. Hyperoxia may improve O_2 delivery downstream of the obstructing emboli. For its success HBO needs to be instituted early. Hyperoxia also maximizes the gradient for elimination of gas (generally nitrogen) in the emboli.

PROCEDURES & EQUIPMENT

HBO therapy is administered in a hyperbaric chamber. They are of two basic types :- Monoplace; Multiplace.

Monoplace chambers : It is transparent, made up of acrylic, can accommodate a single patient and patient does not require a mask. *Primary advantage* :- Cost and space requirements.

Multiplace chambers : Usually of steel (some may be made up of aluminium), 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. Every monoplace chamber can be equippe with full range monitoring and critical care capability.

Pressure and duration :- It depends upon the indication. It ranges from 2-6 ATA for 2-6 hours.

Decompression sickness / Gas embolism may require prolonged, continuous saturation protocols. PIO_2 very rarely exceeds 2.8 ATA.

Emergency indications for HBO therapy generally require only 2-3 separate chamber treatments whereas problem wounds often require 40 or more daily sessions.

PRACTICAL ASPECTS OF CARE IN A CHAMBER

1. **Fire Safety :** Maintaining electrical components outside the chamber. Passing cables through insulated pass throughs.
2. **Electrical Defibrillation :** In a hyperbaric chamber defibrillation is controversial, because of 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 I/V 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 (NTG 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, so that over or under-expansion does

not occur and result in injury or leakage. An exceptional is flow-directed pulmonary artery catheters, here the balloon is left deflated with filling port open to atmosphere during HBO therapy.

Positive pressure ventilation may be performed with self-inflating bags and volume cycled ventilators are preferred.

RISKS & COMPLICATIONS OF HBO THERAPY

Barotrauma

Aural barotraumas is the commonest complication (minimized by topical or systemic vasoconstrictors or in specific cases by myringotomy).

Pulmonary barotrauma (rare but if suspected HBO therapy should be immediately stopped). Pneumothorax should be treated with chest tube insertions (likewise for pre-existing pneumothorax).

Decompression Sickness

Decompression sickness is unlikely if patient breathes 100% O_2 since little nitrogen uptake occurs. Chamber attendants are at higher risk, as they breathe air. If an attendant becomes unavoidably involved then decompression rate can be decreased.

Oxygen Toxicity

CNS toxicity muscular twitchings

Seizures

Narrowing of visual fields

Termination of oxygen inhalation is sufficient.

Changes of chamber pressure should await cessation of seizures as breath holding during seizures and concurrent decompression may increase the risk of pulmonary barotrauma.

Pulmonary toxicity : Can be reduced by intermittent air breathing.

Visual Function

Progressive myopia : Due to prolonged daily therapy as oxygen / pressure affects lens shape and refractory index. It is reversed within days to weeks after completion of therapy.

Cataracts : In unusually long course of therapy such as :- 150 to 200 daily exposures to 2 to 2.5 ATA and does not reverse after cessation of therapy.

Neonates : Prolonged exposure to even 1 ATA may induce retrolental fibroplasia.

Other Risks and Side-effects

Claustrophobia : This is usually seen in isolated patients especially in monoplace chambers. May require sedation for successful therapy. Sedation must be use cautiously as hypoventilation and hypercapina may lower the threshold for O_2 induced seizures.

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