

Hyperbaric Oxygen Therapy – Untangling The Truth

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Introduction

Hyperbaric oxygen therapy... Is it myth and mystery? Does it work? It is undeniable for most of us that this subject was never part of the syllabus in medical school. Among surgeons, they vaguely remember seeing the term somewhere, in one of the many chapters in the surgical textbooks.

A term more familiar among divers and doctors working in the naval base, hyperbaric oxygen therapy (HBOT) is the treatment modality for decompression sickness and air embolism. Over the past 50 years, HBOT has been used for a variety of other medical conditions. Unfortunately, it has also built up great skepticism as the treatment was more often used without acceptable scientific evidence of efficacy and safety. However, in the past 20 years, several trials have demonstrated that HBOT is beneficial to certain medical conditions. Many recognised organizations have also been formed to make sure that the treatment is used adequately and efficiently.

In an attempt to separate ‘the sheep from the goats’ official from Medicare, a health insurance programme administered by the United States government, appointed an Ad Hoc committee in 1972 to identify medical conditions for which hyperbaric oxygen had been shown to have efficacy and merited insurance coverage. This Hyperbaric Oxygen Therapy Committee soon became a standing committee of the Undersea and Hyperbaric Medical Society (UHMS). Their first committee report was published in 1977. This report has since been updated every 3-4 years.

The following indications are based on the 2003 report:

1. Air or Gas Embolism

2. Carbon Monoxide Poisoning and Carbon Monoxide Poisoning Complicated by Cyanide
3. Clostridal Myositis and Myonecrosis (Gas Gangrene)
4. Crush Injury, Compartment Syndrome, and other Acute Traumatic Ischaemias
5. Decompression Sickness
6. Enhancement of Healing in Selected Problem Wounds
7. Exceptional Blood Loss (Anemia)
8. Intracranial Abscess
9. Necrotizing Soft Tissue Infections
10. Osteomyelitis (Refractory)
11. Delayed Radiation Injury (Soft Tissue and Bony Necrosis)
12. Skin Grafts & Flaps (Compromised)
13. Thermal Burns

What about ‘Topical Hyperbaric Oxygen’?

Hyperbaric oxygen therapy should not be confused with topical oxygen therapy via oxygen extremity chambers, oxygen boots, oxygen body bags or even oxygen bars. **The large body of literature supporting the use of systemic hyperbaric oxygen cannot appropriately be used as support for topical oxygen therapy.** HBOT – **sometimes termed ‘systemic hyperbaric oxygen therapy,’** delivers enhanced oxygen levels to diseased or injured tissues **through inhalation of oxygen in a pressurized chamber.** The inhaled oxygen is transported through the circulatory system to the site of pathology. **Topical oxygen on the other hand, even if administered through open wounds, only penetrates superficially and can only impact on that 30-50 micron depth through which it will diffuse under optimal conditions.**

How does HOB2 work?

Hyperbaric oxygen therapy has two resulting effect: An increased in pressure and a raised PaO₂. These two effects are the basis of the mechanism of action in the application of HBOT.

The beneficial effect of an increased in pressure relates to gas volumes being reduced- a universal principle that is known as Boyle’s Law. In the treatment of gas bubble injuries (like Air Embolism from diving or caused iatrogenically) circulating bubbles are ‘crushed’ by the

increased pressure thus facilitation reperfusion.

How does the effect of a raised PaO₂ help in the medical conditions such as problem wounds, delayed radiation injury, infections and compromised skin grafts and flaps?

The oxygen carrying capacity of haemoglobin in a healthy adult breathing air is normally already 97% saturated, with only 0.3ml/L oxygen being dissolved in the plasma. A slight increase in FiO₂ would have fully saturated all the haemoglobin, and that is the limit of the amount of oxygen that is transportable via the haemoglobin system.

The oxygen that is dissolved in the plasma is a function of the PaO₂, i.e. pressure dependent. Again this relates to a nostalgic principle which we all knew as Henry's Law: *the volume of a gas (e.g. oxygen) that is dissolved in a fluid (e.g. plasma) being proportional to the partial pressure of that gas in equilibrium with the fluid.* By increasing the FiO₂ and the atmospheric pressure it is possible to dissolve a large amount of oxygen in the plasma. Breathing 100% oxygen at 3 atmospheres absolute will result in approximately 6.9 vol% oxygen in the plasma, raising the PaO₂ to about 2,000 mmHg. This amount of dissolved oxygen is sufficient to maintain tissue oxygenation without the help of hemoglobin.

What are the physiological effects of HBO₂?

Hyperbaric oxygen causes vasoconstriction, but because of the large amount of oxygen that is dissolved in the plasma tissue oxygen is not compromised. Obviously this has important clinical applications for reducing oedema secondary to injury/trauma.

In severe infections tissue oxygen is usually compromised. Superoxide generation by polymorphonuclear leukocytes, which is essential for bacterial killing, is critically dependent on tissue oxygen levels. Hyperbaric oxygen restores general oxygen levels enabling PM leucocytes to effectively kill micro-organisms. Infections such as Necrotizing fasciitis, Gas gangrene and other severe infections responds well when HBO₂ is added on in the treatment protocol. Often a correction of tissue hypoxia will does result in a remarkable recovery from infection.

In the context of wound healing, apart from the restoration of leucocytic function to fight infection, hyperbaric oxygen administration results in improved cellular metabolism, tissue oxygenation, collagen formation and angiogenesis. HBO2 has also been found to up-regulate platelet-derived growth factors receptors and possibly some other growth-factors that are responsible for angiogenesis.

The area of reperfusion injury has been studied. HBO2 has been shown to reduce leucocyte adherence to venular endothelium in ischaemia-reperfusion of skeletal muscle, suggesting a reduction in the inflammation of the post-ischaemic tissue. Recently a group in the UK reported improved myocardial function and clinical outcome in CABG patients when HBO2 was administered per-operatively to prevent ischaemia-reperfusion injury.

Another area of fascinating outcome is in post-radiation tissue injury. The hypocellular, hypoxia and hypovascular tissue that results as a sequelae to radiotherapy often leads to tissue breakdown and post-radiation necrosis (e.g. osteoradionecrosis, radiation proctitis, radiation cystitis). Typically this condition is similar to problem wounds and therefore it is not surprising that patients with post-radiation tissue injuries are significantly improved by the administration of HBO2.

Steve Thom and his group in Pennsylvania demonstrated that stem cells production is increased 2-fold in mice after just one HBO2 session, and up to 8-fold after a series of 20 HBO2. This has important implications for future clinical applications of stem cells research.

Complications

Complications that occur secondary to HBOT are uncommon if standard of hyperbaric care is maintained. The most common complications (which is less than 1% of treated patients) are middle ear barotraumas. This is similar to the ear pain experienced by some passengers in an aircraft upon descent. Claustrophobia, an often cited 'complication' of treatment in a hyperbaric chamber, is even more uncommon. Often in a multiplace chamber (a chamber accommodating more than 2-persons) the new patient's fear will be allayed by seeing other patients inside together with him/her. The presence of a medical personnel accompanying him inside the treatment chamber is also a reassuring factor. In most modern monoplace chambers, the hull is often made clear acrylic plastics. That has helped reduce the claustrophobic feeling

in many patients undergoing treatment in these monoplaces.

Oxygen toxicity is rare. It manifests in two major forms: central nervous system and pulmonary. Within the standard treatment pressures of 2.0 to 2.4 ATA used for most clinical conditions the incidence of oxygen convulsion is approximately 1 per 10,000 patient-treatment or 0.01%. Pulmonary oxygen toxicity occurs only after prolonged exposures and manifests itself as chest-pain, cough with symptoms resembling those of COPD. This is reversible once treatment is temporarily discontinued.

Patient Selection

From a clinical standpoint, not all patients will benefit from hyperbaric oxygen treatment. Patient selection is important and ought to be based on evidence of benefit and risk. In many areas the clinical benefits of treatment with hyperbaric oxygen is well demonstrated (e.g. selected problem wounds, decompression illness, radiation induced injury to tissues, necrotizing fasciitis and gas gangrene), with expected outcome.

Summary

In summary, clinical hyperbaric oxygen is useful for a number of conditions. The mechanisms on how hyperbaric oxygen exerts its effect is complex and a number of its mechanisms are still being elucidated. The development in this field is exciting. Several randomized studies are being completed and will be made reported in the next 1-2 years. Many new areas of applications, both old and new, are being reported. However more sturdy research will be needed to translate this into clinical practice.

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