Hyperbaric Oxygenation Therapy in the Treatment of Cerebral Palsy: A Review and Comparison to Currently Accepted Therapies

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ABSTRACT

Hyperbaric oxygenation therapy (HBOT) has shown promise in clinical trials and is sought by many parents of children with cerebral palsy (CP). There is unusual resistance to expanding the indications for this modality, which is the only treatment available for certain conditions, such as decompression sickness and air embolism, and which is effective in a number of others related to wound healing. A recent study that showed notable improvements in children with CP treated with slightly pressurized air, as well as those treated with a standard protocol for HBOT, is invoked to deny effectiveness of HBOT. Political and economic considerations, rather than purely scientific ones, play an important role in this controversy. Further systematic research is needed, but in the meantime children should not be denied access to HBOT.

Hyperbaric oxygenation therapy (HBOT) in the treatment of certain conditions (for example: decompression accidents, gas gangrene, burns) is supported by substantial clinical literature. Some other conditions (for example: skin or tissue grafts, specific cases of anemia) are also on the accepted indication list but with scant support from formal clinical trials. Its use in some conditions has proved ineffective, and in others, especially neurologic, it has been very controversial.

In 1994 Harch reported the first North American case of HBOT in a child with cerebral palsy (CP).[1] Around the year 2000, some researchers[2] affirmed that, following HBOT, some patients with CP experienced improvement in motor function ,and decreased muscle spasms. Controversy soon developed in the newspapers. Political factors have impeded further research and the adoption of new clinical applications.[3-8]

What is HBOT?

In 1999 the drug definition of HBOT was refined and restated as the use of oxygen at greater than atmospheric pressure as a drug to treat basic pathophysiologic processes and the associated diseases.[9] Under normal atmospheric pressure at sea level—760 mm Hg, 1 atmosphere absolute or 1 ATA—hemoglobin in the blood is already 97% saturated with oxygen, with very little capacity for increasing oxygen transport.[10] Oxygen is also dissolved directly in the plasma in a more bioavailable form. According to Henry's Law, the absorption of a gas is directly related to the partial pressure of the gas. About 17 times as much oxygen can be carried in the plasma when the patient breathes 100% oxygen at a pressure of 3 ATA, compared with breathing room air at sea level (see Table 1). The added pressure can also reduce blood flow to the damaged areas, and hence reducing edema, without compromising oxygenation.[11]

The pressure must be applied to the entire body. This is accomplished either in a single-person chamber, usually pressurized with 100% oxygen, or a multi-place chamber, which is pressurized with air while patients breathe oxygen through a mask or hood. Multi-place chambers have a reduced fire hazard, but there is some variability in the concentration of oxygen actually inhaled because of leakage around the mask. This is less true with the use of a hood.

In Canada, in the public system, there are fewer than a dozen hyperbaric chambers available to treat various medical conditions.

Risks of HBOT

HBOT, particularly at pressures lower than 1.75 ATA, involves little risk of any major complications. In fact, the risks of HBOT are minimal when technicians obey safety regulations and follow a specific protocol. They include rare decompression accidents.

Adverse effects of HBOT on the human body include barotrauma most commonly involving the middle ear, sinuses, or dental restorations. This is reported to occur in 2% of patients.[12] Most patients are able to prevent ear barotrauma by using simple self-inflation techniques. Reversible myopia may occur during high-pressure treatments. Some patients do not tolerate confinement in a small enclosed space.

There are only few absolute contraindications to HBOT (pneumothorax, and treatment with adriamycin, vincristine, and similar drugs). Conditions such as respiratory infection, chronic sinusitis, epilepsy, optic neuritis, certain lung diseases, and claustrophobia must be carefully evaluated before treatment is authorized.[13] Significant adverse effects are very uncommon: see Table 2.

Rationale for HBOT in CP

CP is most often caused by an ischemic/hypoxic injury during the perinatal period. While hypoxia may cause cell death, there may sometimes be a zone called the "ischemic penumbra," in which brain cells receive just enough oxygen to survive, but not enough to function normally. Since that discovery, many have asked the question: to what extent can HBOT reactivate damaged neurons?

It is generally admitted that the cells to which the blood flow is dramatically reduced for 10 minutes or so (less than 10 ml of blood per 100 g of brain tissue per minute)[15] undergo necrosis and form the core of a lesion. With less severe hypoxia, some researchers believe that cells can survive for a long time in an "idling" state, and might be reactivated if blood flow is restored. Those who observed a decrease in spasticity and functional improvements with HBOT hypothesized that neurons might be viable but inactive much longer than previously believed.[16,17]

Table 1. Quantity of Oxygen Dissolved in the Blood Owing to Pressure[37]

Pressure	Percentage of O_2 inhaled	Quantity of O_2 (in ml) dissolved in 100 ml of blood
1 ATA	21% (normal air)	0.32
1 ATA	100%	1.7
2 ATA	100%	3.7
3 ATA	100%	5.6