Adjuvant Hyperbaric Oxygenation Therapy in Hand Edema and Ischemia

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HISTORICAL PERSPECTIVE

The first documented use of hyperbaric (air) therapy dates back to 1662, although the era of modern hyperbaric oxygenation (HBO) began in 1937, when HBO was used in the treatment of decompression sickness (1). The purpose of HBO is to increase the partial pressure of oxygen in the circulation, with a resultant increase in oxygen delivery to the tissues. HBO has become an accepted treatment for a number of acute and chronic conditions, including air embolism, carbon monoxide poisoning, and decompression sickness (2). Under HBO at 2.5 atmospheres absolute (ATA), the partial pressure of oxygen in the blood may reach 1.500 mm Hg. At such pressures, the amount of oxygen dissolved in the plasma becomes highly significant (approximately 4.5 vol%), and is in fact almost sufficient to meet tissue oxygen demands without the need for hemoglobin. Of special interest to the hand surgeon is the use of HBO in crush injury, compartment syndrome, and acute traumatic ischemia. The primary rationale for the use of HBO in these conditions is to increase oxygen delivery to the compromised tissue to a level that can prevent further tissue death and support the natural healing process. In addition, HBO seems to decrease tissue edema. This property may be advantageous when traumatic or surgical hand edema severely reduces tissue perfusion to levels that may result in permanent damage. No specific therapy is currently available for these cases. An increasing body of evidence suggests that HBO minimizes the ischemic insult and effectively decreases tissue edema. Animal studies using a rat model have convincingly demonstrated the ability of HBO to decrease the concentration of arterial leukocytes and neutrophils in compromised gracilis muscle flaps (3). This model of ischemia–reperfusion may be of relevance in microvascular surgery, where neutrophils play a major role in initiating the cascade of tissue-destroying processes (4). Neutrophil-induced tissue injury is caused at least in part by their attachment to vascular endothelial cells. The cell adherence process is mediated by $\beta_2$-integrins, leukocyte membrane glycoproteins whose function is inhibited by HBO (5). In a comparable model of skeletal muscle injury, the effectiveness of repeated HBO treatments for elevated levels of high-energy phosphate compounds (6) and attenuated glutathione depletion (7) have been documented. Both reflect improved recovery of metabolic function. Similar results have been described when muscle phosphorylase activity is measured after an ischemic insult in rats (8). HBO reduced edema and necrosis in a model compartment syndrome in dogs (9). HBO also has been associated with arterial hyperoxygenation and peripheral vasoconstriction (10,11). HBO facilitates neovascularization and the proliferation of fibroblasts and thus may accelerate the repair of damaged tissues (12). HBO is also bacteriostatic to anaerobic microorganisms and at high pressures may even be bactericidal. Thus, HBO may lower the risk of infection, particularly by anaerobic bacteria (13). Taken together, the experimental studies briefly described suggest that HBO may be an attractive clinical therapeutic intervention in cases where routine measures to alleviate edema and ischemia have failed or recovery has been delayed. Admittedly, there are no formal, large-scale, randomized controlled studies to support the use of HBO in these clinical situations. However, theoretical pathophysiological considerations, animal research, and anecdotal reports do favor such a use.

We used HBO in 20 patients for the indications listed in Table 1. All patients tolerated the treatment well, without complications. In eight patients, the need for amputation was avoided by the combination of surgery and HBO. In another eight patients, HBO halted the progression of ischemic changes and promoted the growth of granulation tissue. Reversal of tissue hypoxia and delineation of the demarcation line between necrotic and viable tissue facil-
TABLE 1. List of clinical conditions treated by hyperbaric oxygenation

<table>
<thead>
<tr>
<th>Condition treated</th>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crush injury of the hand</td>
<td>10</td>
</tr>
<tr>
<td>Crush injury of the fingers</td>
<td>6</td>
</tr>
<tr>
<td>Tip amputation</td>
<td>3</td>
</tr>
<tr>
<td>Compartment syndrome</td>
<td>1</td>
</tr>
</tbody>
</table>

inated limited amputation and reconstructive procedures. In four patients, no improvement was observed following HBO therapy. It is emphasized that this experience is anecdotal in nature. However, treatment was considered for patients in whom other measures were of no avail.

■ CASE REPORTS

Case 1

A 26-year-old man sustained a crush injury to the left thumb. The skin was intact, and a fracture of the proximal phalanx was diagnosed. The patient underwent closed reduction and cast immobilization. Twelve hours later, the thumb was blue and cold, and the patient was referred to our unit for investigation and further treatment. Examination demonstrated a superficial laceration in the thenar area, intense cyanosis, marked edema, no sensation at the tip of the thumb, and no refill after blanching. Sticking the tip of the thumb resulted in very sluggish blood drops. Finger plethysmography with a pulse oximeter detected no pulse. The finger was cold, with a temperature of 28°C. The patient was treated in a multiplace hyperbaric chamber and inhaled 100% oxygen through a demand mask at an ambient pressure of 2.5 ATA. Each treatment session lasted 90 min. The patient was treated three times daily during the first 48 h, twice daily for the next 2 days, and once a day for another 2 days, for a total of 12 oxygen sessions. After four treatments, the skin color began to diminish, there was a rise in temperature, and a slow refill after blanching was evident in the nail bed. On completion of the treatment, the color of the thumb was pink, the skin temperature was identical to that of the other thumb, and the two-point discrimination test was 9 mm. On examination a year after therapy, the thumb was found to have recovered fully, with normal sensation.

Case 2

A 38-year-old man suffering from idiopathic dilated cardiomyopathy and insulin-dependent diabetes mellitus was admitted to a local hospital after a hypoglycemic episode. An intravenous glucose 50% solution was administered through the peripheral vein on the back of the hand, resulting in complete arousal of the patient. Extravasation of the solution was noted at that stage. A few hours later, the patient complained of pain, and the hand was seen to be slightly swollen, tender, and red. Cefazolin, 1 g three times a day, and gentamicin, 80 mg twice a day, were started. The condition of the patient’s hand gradually deteriorated, and the following day it was found to be severely swollen and cyanotic. The pain had increased and was intensified by passive muscle stretching.

A diagnosis of compartment syndrome was made, and immediate surgical decompression of the volar and dorsal compartments was performed, including the carpal tunnel and the intersosseous compartment. During surgery, severe edema was found, with thrombosis of small veins. After surgical decompression, improvement was noticed in the muscle belly, and edema was reduced. Two days later, the condition of the hand and forearm had gradually deteriorated. The forearm was less swollen but had become hard, tense, and tender, with swelling of the hand, coldness and cyanosis of the fingers, and signs of median and ulnar nerve compression. The radial and ulnar pulses were palpable. A second surgical decompression was performed. Propagation of the small vein thrombosis was found proximally: the flexor muscle belly appeared pale and tense. Epiyndyotomy was performed. Only at this late stage was the hyperbaric medicine institute consulted, and the patient was referred to us.

On examination, the patient was afebrile. The hand was grossly swollen, cyanotic, cool, and almost anesthetic. Both radial and ulnar pulses were palpable. The patient was treated in a multiplace hyperbaric chamber and inhaled 100% oxygen through a demand mask at an ambient pressure of 2.5 ATA (equivalent to a depth of 15 m seawater). Each treatment session lasted 90 min. During the first 48 h, the patient was treated three times daily, after which he was treated twice daily for 3 days and once a day for another 4 days. A total of 16 oxygen sessions was administered.

During the first 2 days, there was only slight improvement. On day 3, however, marked improvement was noted in the degree of swelling, and the hand gradually became warmer and less painful. On pulse oximetry, a pulse signal was obtained, and saturation above 95% was measured in all fingers except the third and fifth. With continued treatment, the color of the hand gradually improved, and the demarcation line between ischemic and well-perfused tissue progressed distally. Sensation was restored. On completion of the series of treatments, the appearance of the hand and forearm had greatly improved, except for the fifth digit and the distal two phalanges of the third and fourth digits. Two weeks later, there was evidence of dry gangrene in these parts of the fingers, with no infection.

■ INDICATIONS/CONTRAINDICATIONS

We believe that HBO should be considered in cases in which, despite exhaustive surgical efforts, the patient is left with an edematous, ischemic limb. HBO is a safe and simple treatment. Untreated tension pneumothorax is the
only absolute contraindication. Relative contraindica-
tions include upper respiratory tract infection, emphy-
sema with CO₂ retention, an asymptomatic pulmonary le-
sion seen on chest X-ray, a history of thoracic or ear 
surgery, uncontrolled high fever, and pregnancy.

The Undersea and Hyperbaric Medical Society Com-
mittee (2) classified compartment syndrome, crush in-
jury, acute traumatic ischemia, and compromised skin 
grafts and flaps as "approved uses" for HBO. Therefore, 
the hand surgeon must decide which cases may benefit 
from HBO.

**TECHNIQUE**

HBO is based on the delivery of high-pressure oxygen. 
Delivery is achieved by placing the patient in a hyper-
baric chamber, where the pressure is increased by com-
pressed air. Oxygen is delivered by face mask (Fig. 1). In 
certain "monoplace" chambers, the chamber itself is 
pressurized with oxygen; hence, no breathing mask is 
required. The first step in HBO is adequate preparation 
of the patient. A routine chest X-ray and assessment by an 
atolaryngologist of the patient's ability to equilibrate air 
pressure in the middle ear are generally all that are 
required. Age is not a limiting factor, and elderly patients 
can tolerate treatment very well. Treatment itself begins 
by increasing the air pressure in the chamber. In multi-
place chambers, patients are accompanied by an attend-
ant (e.g., nurse, medic) who also breathes compressed 
air during the treatment (Fig. 2). A commonly employed 
treatment protocol is presented in Table 2.

Special attention should be paid to patient complica-
tions during the periods of elevation and reduction of pres-
sure within the chamber. The rate of compression is limited 
by the patient's ability to equilibrate pressure in the middle 
ear. If difficulty is encountered, compression is halted, the 
pressure is slightly reduced to allow easy clearing of the 
blocked Eustachian tube, and treatment is resumed. If the 
patient has a viral upper airway infection, nose drops with 
ephedrine can be of help. In most cases, the treatment pres-
sure (2.5 ATA) is reached within 5–10 min.

Most chambers also have a smaller transfer lock, 
through which additional personnel may enter the main 
treatment chamber, if necessary, to attend a medical 
emergency. Treatment is provided once or twice daily for 
several days until the clinical condition has stabilized and 
is discontinued at the discretion of the surgeon and the 
hyperbaric physician on the basis of daily clinical assess-
ment of the injured hand.

**COMPLICATIONS**

Complications of HBO are related either to pressure or 
to oxygen toxicity. Pressure-related complications in-
clude middle ear, sinus, or pulmonary barotrauma. These 
situations can be avoided by pretreatment assessment. 
Oxygen-related complications are primarily central ner-
vous system (CNS) oxygen toxicity, leading to seizures. 
Oxygen toxicity during treatment is rare because the 
threshold oxygen partial pressure for CNS toxicity is 3.0 
ATA, which is well above the pressure used in routine 
HBO treatments (2.5 ATA). The incidence of seizures 
during such treatments is 0.01% (14). If convulsions oc-
cur, the oxygen mask should be removed immediately. 
In most cases, removing the mask will stop the seizure. 
Pressure changes should not be attempted during con-
vulsions because pulmonary barotrauma may result. 
Other rare complications include reversible myopia and 
the possibility of claustrophobia. Lastly, because the 
personnel attending patients breathe compressed air like
TABLE 2. Typical treatment profile for hyperbaric oxygenation

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Chamber pressure (ATA)</th>
<th>Patient’s breathing gas</th>
<th>Attendant’s breathing gas</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–5</td>
<td>1.0–2.5</td>
<td>Air</td>
<td>Air</td>
</tr>
<tr>
<td>6–50</td>
<td>2.5</td>
<td>Oxygen</td>
<td>Air</td>
</tr>
<tr>
<td>51–55</td>
<td>2.5</td>
<td>Air</td>
<td>Air</td>
</tr>
<tr>
<td>56–100</td>
<td>2.5</td>
<td>Oxygen</td>
<td>Air</td>
</tr>
<tr>
<td>100–104</td>
<td>2.5–1.3</td>
<td>Air</td>
<td>Air</td>
</tr>
<tr>
<td>104–109</td>
<td>1.3</td>
<td>Air</td>
<td>Oxygen</td>
</tr>
<tr>
<td>109–110</td>
<td>1.3–1.0</td>
<td>Air</td>
<td>Air</td>
</tr>
</tbody>
</table>

ATA, atmospheres absolute.

a scuba diver, their decompression requirements must also be considered. Attendants usually breathe oxygen for 5 min at a 3-m stop (1.3 ATA) toward the end of the treatment.

■ CONCLUDING REMARKS

HBO is a valuable adjuvant therapy in cases of the ischemic edematous hand. This treatment combines arterial hyperoxygenation (oxygen partial pressures as high as 1,500 mm Hg) and peripheral vasoconstriction, leading to the reduction of edema (3–8). At oxygen pressures as high as this, the amount of oxygen dissolved in the plasma is almost sufficient to meet tissue oxygen demands without the need for hemoglobin. Because in microvascular occlusion there may be partial obstruction through which plasma flows but which red blood cells are unable to penetrate, oxygen in solution may prove to be the difference between critical anoxia and tissue survival. Thus, the vicious circle of edema, vascular compression, ischemia, edema, and so on may be broken by HBO (15). Our experience with HBO is favorable, although conclusions regarding its efficacy will have to be based on prospective randomized clinical studies.

■ REFERENCES