hyperbaric oxygen

particular issues for critical care

Preparation for hyperbaric therapy:
- Plans for treatment begin while the patient is still in the ICU, before transport to the hyperbaric chamber is initiated.
- Issues to be addressed include:
(i) informed consent,
(ii) determination that all intravenous/arterial lines and nasogastric tubes/Foley catheters are secured,
(iii) capping all unnecessary intravenous catheters,
(iv) placing chest tubes to one-way Heimlich valves, and
(v) adequately sedating or paralyzing the patient as clinically indicated.

Monitoring & equipment issues
- The patient is attached to equipment at ambient pressure before treatment, and once the treatment pressure is achieved all settings are checked and transducers recalibrated.
- Among the items that must be checked is the cuff pressure of endotracheal tubes. The usual practice is to replace the air in these cuffs with an equivalent volume of sterile saline before treatment to avoid volume changes related to pressurization.
- If glass bottles, pressure bags, or any other gas-filled equipment are used inside a hyperbaric chamber, they must be adequately vented and closely monitored during a treatment.

general

- HBO2 treatment is carried out in either a monoplace (single person) or multiplace (typically 2 to 14 patients) chamber.
- Pressures applied while in the chamber are usually 2 to 3 atmospheres absolute (ATA), the sum of the atmospheric pressure plus additional hydrostatic pressure equivalent to one or two atmospheres.
- Treatments usually are for 2 to 8 hours, depending on the indication, and may be performed from one to three times daily.
- Monoplace chambers are usually compressed with pure oxygen.
- Multiplace chambers are pressurized with air, and patients breathe pure oxygen through a tight-fitting facemask, a hood, or endotracheal tube.
- During treatment, the PaO2 typically exceeds 2000 mm Hg and levels of 200 to 400 mm Hg occur in tissues.

indications

- Air or gas embolism
- Carbon monoxide poisoning
- Clostridial myositis and myonecrosis
- Crush injury, compartment syndrome, acute traumatic ischemia
- Decompression sickness
- Enhancement of healing in selected wounds
- Exceptional blood loss anemia
- Necrotizing fasciitis
- Chronic refractory osteomyelitis
- Radiation necrosis
- Skin flap or graft compromise
- Thermal burns

mechanisms of action

Related to Hyperoxygenation of Tissues

- Angiogenesis in ischemic tissues[4-6] (mechanisms likely include O_2 behaving as intracellular signal transducer leading to augmentation of one or more growth factors[7-9])
- Bacteriostatic/bactericidal actions[10-12]
- Carboxyhemoglobin dissociation hastened[13]
- *Clostridium perfringens* α toxin synthesis inhibited[14,15]
- Phagocytic bacterial killing improved[16]
- Temporary inhibition of neutrophil β_2 integrin adhesion[17-19]
- Vasoconstriction[20,21]

Related to Pressurization

- Reduction of gas bubble volume (Boyle's law)

adverse effects

barotrauma

(i) Middle ear barotrauma
- Middle ear barotrauma is the most common adverse effect of HBO2 treatment
- Standard protocols include instruction of patients on autoinsufflation techniques and adding oral or topical decongestants when needed. When autoinsufflation fails, tympanostomy tubes must be placed.

(ii) Pulmonary barotraumas:
- Pulmonary barotrauma during HBO2 treatment is extremely rare but should be suspected when any significant chest or hemodynamic symptoms occur during, or shortly after, decompression.
- Because the offending gas in virtually all cases will be pure O2, absorption within the body may occur. If symptoms do develop, however, decompression should be stopped and the patient evaluated.
- If pneumothorax is suspected, placement of a chest tube is appropriate. Preexisting pneumothorax should be treated with chest tube drainage before initiating therapy.

oxygen toxicity

(i) pulmonary toxicity
- Pulmonary insults can impair mechanics (elasticity), vital capacity, and gas exchange.
- Most studies have failed to identify any adverse pulmonary effect from standard protocols.

(ii) CNS toxicity:
- CNS O2 toxicity is manifested as a grand mal seizure. This occurs at an incidence of approximately 1 to 4 in 10,000 patient treatments. The risk is higher in hypercapnic patients, and possibly those who are acidotic or with compromise due to sepsis, because an incidence of 7% (23 in 322 patients) was reported in case series of HBO2 treatment of gas gangrene.
- Seizures are relatively easy to manage in most cases: simply reduce the inspired O2 tension while leaving the patient at the same ambient pressure (to avoid pulmonary overexpansion injury when a patient is in tonic convulsion phase).

(iii) ocular toxicity:
- Progressive myopia has been reported in patients who undergo prolonged daily therapy, but this typically reverses within 6 weeks after termination of treatments.
- Development of nuclear cataracts has been reported with excessive treatments that exceed a total of 150 to 200 hours, and the change does not spontaneously reverse.
- Although there is a theoretical risk for retrolental fibroplasia in neonates, there are no reports of this having occurred.
- Currently, experimental and clinical evidence does not indicate that typical HBO2 therapy protocols have detrimental effects on neonates or the unborn fetus.