A PROVIDER'S REFERENCE FOR CLINICAL HYPERBARIC OXYGEN THERAPY

Hyperbaric oxygen is the intermittent administration of 100% oxygen at pressure greater than that at sea level. At the Davis Hyperbaric Lab, this is accomplished in one of two multiplace or walk-in hyperbaric chambers. These chambers are pressurized with air to reach treatment depth. Once at "depth", the patient breathes 100% oxygen through a hood, mask, or endotracheal tube. Under these conditions, the arterial partial pressure of oxygen (P02) will approach 1,700 mm Hg at a pressure equivalent to 45 feet of sea water (2.4 ATA).

Hyperbaric oxygen therapy is the primary treatment for decompression sickness (the bends), arterial gas embolism and severe carbon monoxide poisoning. It has been shown to be an important and useful adjunct in the treatment of gas gangrene, chronic refractory osteomyelitis, radiation tissue damage, anaerobic infections, crush injury, compartment syndromes, and other acute traumatic ischemias. Enhancement of healing is common during and following hyperbaric therapy in selected problem wounds, including diabetic ulcers, arterial insufficiency ulcers, and mixed soft tissue infections. It is also utilized as an adjunct in necrotizing soft tissue infections of subcutaneous tissue, muscle and/or fascia (particularly in the compromised host). The use of adjunctive hyperbaric oxygen therapy in the treatment of chronic refractory osteomyelitis and radiation necrosis of either bone or soft tissue is well accepted. Early adjunctive hyperbaric oxygen therapy has also been found to be beneficial in preservation of compromised skin grafts or flaps, or in preparation of a site for grafting. Additional considerations include thermal burns and excessive blood loss anemia.

During the late 1930's, oxygen at pressure was proposed as a treatment of decompression sickness. In the early 1960's Dutch investigators showed the efficacy of hyperbaric oxygen in the treatment of gas gangrene and anemic states. Later in that decade, hyperbaric oxygen therapy became the standard for naval diving accidents. Subsequent studies have shown the efficacy of oxygen in the treatment of wounds, enhancement of white cell killing ability, preservation of hypoxic tissue, and angiogenesis. The clinical indications for the use of adjunctive hyperbaric oxygen therapy continue to be defined.

MECHANISMS ACTION

Oxygen inhaled under pressure dissolves in plasma. At 3 atmospheres absolute, an arterial PO2 of nearly 2,200 mmHg may be achieved and up to 6.9 volumes percent of oxygen may be forced into solution, a quantity sufficient to temporarily maintain life in the absence of hemoglobin.

FIBROBLAST PROLIFERATION

Nonhealing tissues are often hypoxic with tissue oxygen tensions frequently in the range of 5 to 15 mmHg. Although low tissue oxygen tensions stimulate fibroblast activity, tissue oxygen tensions of at least 30 to 40 mmHg (optimal tissue oxygen tensions are in the 40 to 90 mmHg range) are necessary for fibroblast proliferation, collagen synthesis, and the development of a collagen matrix to support capillary budding into avascular areas. Raising the tissue oxygen tension in hypoxic wounds can readily be accomplished through the intact peripheral circulation with hyperbaric oxygen therapy.

NEOVASCULARIZATION

Following hyperbaric oxygen sessions, restoration of low PO2 to physiologic levels will stimulate capillary proliferation. The previously stimulted fibroblasts provide a scaffolding and infrastructure to support new capillary ingrowth.

ENHANCEMENT OF WHITE CELL KILLING

Polymorphonuclear leukocytes in low oxygen tensions (e.g., 5 to 15 mmHg) show diminished ability to kill organisms through the peroxidase system (an oxygen-dependent mechanism). The killing ability of white blood cells can be greatly enhanced by increasing tissue oxygen tensions.

VASOCONSTRICTION

Exposure to oxygen at pressure causes a 15 to 25% reduction in blood flow resulting in less diapedesis and bleeding in areas of capillary damage. This effect might seem to be undesirable in ischemic tissue, but the tenfold increase in oxygen content of the plasma and the resultant increase in the capillary diffusion distance of oxygen more than compensates for decreased arterial flow.

ENHANCEMENT OF ANTIBIOTIC ACTIVITY

Evidence shows that certain antibiotics may be more effective in the presence of elevated oxygen tensions. These include aminoglycosides, vancomycin, sulfonamides, and quinolones.

THE HYPERBARIC MEDICINE DIVISION

The Hyperbaric Medicine Division is under the supervision of physicians and physiologists, fellowship trained in hyperbaric medicine and operations. The Division is also staffed with experienced nurses and medical technicians fully trained in the medical and physical aspects of hyperbaric medicine, and cross-trained in critical care. In addition, aerospace physiology technicians trained in hyperbaric operations and medicine support daily patient treatments.

THE CHAMBER COMPLEX

Our large clinical chamber is capable of treating up to 10 patients per treatment. Our smaller chamber has the capacity to treat 2-3 additional patients. All chambers have elevator locks. Treatments are staffed by inside medical attendents and monitored by a physician and a physiologist via direct visualization, video, and audio communication links. For single patient treatments a monoplace chamber also exists as an option for hyperbaric therapy.

WOUND CARE

Our clinical Hyperbaric Medicine program has over 20 years experience in managing complicated non-healing wounds. Patients will receive daily "wound care center" evaluations. Our staff will make recommendations for ongoing wound care in order to maximally enhance the wound healing environment.

OUTPATIENT AND INPATIENT CARE

Hyperbaric oxygen therapy may be given on an inpatient or outpatient basis, depending on the patient's condition. Hyperbaric consultation is available on a 24-hour a day, 7day a week basis.

CONSULTATION PROCEDURE

Patients are seen in consultation upon referral from their attending physician. Hyperbaric oxygen therapy is an adjunct to continuing medical and surgical care. Referred patients will remain under the primary care of their referring physician.

PRECAUTIONS

The therapy is essentially painless. It is common for patients to experience pressure changes within their middle ears during compression. The attending staff will assist the patient in adjusting to these changes. Patients with colds or upper respiratory infections may be predisposed to middle ear or sinus barotrauma and should not undergo therapy until the condition abates, unless emergency or urgent hyperbaric oxygen therapy is inidicated. Patients with severe claustrophobia may not tolerate the daily 2 hour treatments.

Smoking and hyperbaric oxygen therapy are clinically incompatible. All patients are urged to stop smoking during the period of their therapy. If this is not possible, therapy may have to be discontinued.

Treatment protocols have been carefully designed to minimize pulmonary and central nervous system oxygen toxicity. Certain drugs and other medications may produce unwanted side effects and should be avoided. The hyperbaric physician will provide information on drugs and medications in question. Rigorous safeguards are followed to minimize complications. Sorne patients undergoing prolonged periods of daily hyperbaric oxygen treatment may experience visual acuity changes that are usually reversible. The exact mechanism remains obscure, but is likely due to lenticular changes.

Other contraindications to hyperbaric therapy are severe asthma or COPD, or significant cataracts. Your hyperbaric consultant will be able to advise you on these patients.

ACCEPTED INDICATIONS

Approved uses for hyperbaric oxygen therapy by the Undersea and Hyperbaric Medical Society as described in the Hyperbaric Oxygen Therapy Committee Report, 1996.

- 1. Air Gas Embolism
- 2. Carbon Monoxide Poisoning and Smoke Inhalation Carbon Monoxide Complicated
 - by Cyanide Poisoning
 - by Cyanide Poisoning
- 3. Decompression Sickness
- 4. Clostridial Myonecrosis (Gas Gangrene)
- 5. Thermal Burns
- 6. Radiation Tissue Damage (Osteoradionecrosis)
- 7. Necrotizing Soft Tissue Infections (Subcutaneous Tissue, Muscle, Fascia)
- 8. Enhancement of Healing in Selected Problem Wounds
- 9. Crush Injury, Compartment Syndrome, and other Acute Traumatic Ischemias
- 10. Osteomyelitis (Refractory)
- 11. Exceptional Blood Loss (Anemia)
- 12. Skin Grafts and Flaps (Compromised)
- 13. Intracranial Abscess

RESEARCH

Our staff is on the forefront of research in the areas of recompression therapy, crush injuries, NBC combat casualty care, nonhealing wounds and burns. We work to broaden the understanding and acceptance of HBO therapy through both clinical and basic scientific research. We are leaders in the area of hyperbaric chamber design and fabrication. In fact, the initial prototype for the concrete multiplace hyperbaric chamber was constructed here.

CLINICAL TRAINING

Military medical personnel can receive training in hyperbaric operations and therapy. The following programs are instructed by the Davis Hyperbaric Lab staff and managed through the School of Aerospace Medicine at Brooks AFB, TX. Information on course availability and application procedures can be obtained by contacting your unit training office and referring to AFCAT 36-2223.

Hyperbaric Training for Health Technicians BSAZY4XOX1.....1 week

Hyperbaric Medicine Fellowship BSOZY48A4.....52 weeks

Clinical Hyperbaric Physiology Fellowship BSOZY43A4......39 weeks

Clinical Hyperbaric Nursing B30ZY46X0......7 weeks

Advanced Clinical Hyperbaric Medicine Training B3AZY4XOX1-005......2 weeks

Hyperbaric Training for Health Care Officers B30ZY48X0..... 1 week

Hyperbaric Chamber Enlisted Team Training B3AZY4MOS 1 1 week 3 days

D.O.D. CLINICAL HYPERBARIC MEDICINE FACILITIES

- 1. HYPERBARIC MEDICINE DIVISION Brooks AFB, TX 78235-5000 Com: (210)536-3281 DSN: 240-3281
- 2. DAVID GRANT MEDICAL CENTER Department of Hyperbaric Medicine Travis AFB, CA 94535-5300 Com: (707)423-3987 DSN: 799-3987
- USAF MEDICAL CENTER Department of Hyperbaric Medicine Wright Patterson AFB, OH 45433-5300 Com: (573) 257-8603 DSN: 787-8603
- 4. EISENHOWER US ARMY MEDICAL CENTER Department of Hyperbaric Medicine Ft Gordon, Ga. DSN: 773-2264
- NAVY OPERATIONAL MEDICINE INSTITUTE (NOMI) Department of Hyperbaric Medicine Pensacola NAS, FL 32508-1047 Com: (904) 452-3378 DSN: 922-3378

INDEX OF HYPERBARIC INDICATIONS

AIR GAS EMBOLISM

Recompression in a hyperbaric chamber is the treatment of choice for gas embolism of the arterial or venous system. This includes emboli arising from surgery, diagnostic procedures, renal dialysis, gynecologic manipulations, or pulmonary overpressure accidents during scuba diving. Gas embolism is common enough in cardiovascular surgery to earn the statement "air is the bane of cardiac surgery." Treatment must be initiated during the acute period. Results are best if recompression is within minutes after the incidenthowever, evidence of patients who have recovered with delayed treatment makes recompression mandatory, even in late cases.

RATIONALE: Recompression of gas bubbles in a hyperbaric chamber is the only definitive form of treatment known. Bubbles are compressed, ischemic tissues are oxygenated, and nitrogen is off-loaded. Diving medicine physicians now feel that repeated treatments with hyperbaric oxygen after the initial recompression hasten resolution of residual signs in some cases.

SOURCE: UHMS Publication Committee Report CR(HBO) 1996

CARBON MONOXIDE POISONING AND SMOKE INHALATION CARBON MONOXIDE COMPLICATED BY CYANIDE POISONING

Treatment should be initiated if possible within 6 hours of exposure in that efficacy seems to diminish markedly after that time. Treatment with 100% oxygen at sea level is indicated for a diagnosis of carbon monoxide poisoning. Hyperbaric oxygen therapy is mandatory for patients with severe poisoning exhibiting alteration in mental status or neurological signs, cardiovascular dysfunction, pulmonary edema,or severe acidosis, irrespective of carboxyhemoglobin levels.

RATIONALE: Hyperbaric oxygen hastens the dissociation of carbon monoxide from hemoglobin, myoglobin, cytochromes and other hemoproteins (exceeding that possible at 1 atmosphere), as well as providing tissue oxygenation via saturated plasma. Hyperbaric oxygen has been shown to antagonize lipid peroxidation while sea level oxygen has not. Hyperbaric oxygen should be strongly considered for victims of smoke inhalation since they commonly have a combined, synergistic poisoning with carbon monoxide and cyanide. Hyperbaric oxygen may directly reduce toxicity and augment antidote treatment of cyanide. Hundreds of cases reported in the literature have all yielded the same positive results: rapid improvement in symptoms and by a probable reduction of the incidence of late sequelae. Treatment with hyperbaric oxygen must be predicated more on the history and clinical picture than the carboxyhemglobin levels. COHgb levels are useful in confirming carbon monoxide exposure, but unreliable as a prognostic indicator.

Hyperbaric oxygen should begin during the acute episode. Several treatments may be necessary to completely clear symptoms.

RATIONALE: This disease is caused by nitrogen bubble formation in the vascular system and in tissues, in volumes sufficient to interfere with the function of an organ or cause alterations in sensation. Secondary surface active effects of the nitrogen bubbles include activation of the complement system, lyophobic effects, and increased blood viscosity, to name just a few. Protocols for hyperbaric treatment of decompression sickness have been available for nearly 100 years. Recompression of gas bubbles in the hyperbaric chamber is the only definitive form of treatment known. The clinical manifestations range from skin eruptions and joint pain to neurological dysfunction and profound fatigue, and rarely to an end stage of shock and death. Prompt treatment is important to mechanically reduce bubble size and to help reduce activation of the complement system and the clotting cascade that occur at the blood-bubble interface.

SOURCE: UHMS Publication CR(HBO) 1996

CLOSTRIDIAL MYONECROSIS (Gas Gangrene)

Hyperbaric oxygen is used as an adjunct to surgery and antibiotics in the treatment of clostridial myositis, myonecrosis or spreading clostridial cellulitis with systemic toxicity (or a presumptive diagnosis of any of the three).

RATIONALE: Gas gangrene is caused by a number of pathogenic clostridial organisms which multiply, producing necrotizing tissue toxins and a characteristic picture of pain, fever, and tachycardia out of proportion to the clinical presentation. Ninety percent of cases are caused by Clostridium perfringens which grow rapidly in low oxygen tensions. Restricted growth and a halt in alpha toxin production is evident at tissue oxygen tensions above 250 mmHg. Experimentally, hyperbaric oxygen therapy should be started early and continued until progress of the anaerobic infection is completely halted. It is recommended that a patient with gas gangrene receive 3 treatments in the first 24 hours, then twice a day treatments for the next 4 to 5 days. Hyperbaric oxygen must be started on the basis of clinical impression and gram stain results, without delays for bacterial confirmation. Elevation of oxygen tensions in the region of functioning capillaries in the infected wound halts alpha toxin production. Necrotic tissue can be debrided more conservatively, salvaging more viable tissue than would otherwise be possible. Although surgery and antibiotics remain the primary treatment, the need for emergency, life saving ablative surgery is often obviated as hyperbaric oxygen effects a chemical barrier to progression of the disease.

THERMAL BURNS

The burn wound is a complex and dynamic pathophysiologic process characterized by a zone of coagulation, surrounded by a region of stasis, bounded by an area of hyperemia. An intense inflammatory reaction leading to rapid edema formation, increased microvascular permeability, and sluggish blood flow, results in thrombosis, ischemia, and advancing necrosis. The basic problems in repair of burns include susceptibility to infection, prolonged healing, and excessive scarring. These problems are greatly increased due to the loss of the integumentary barrier and compromised or obstructed microvasculature. This prevents humeral and cellular elements from reaching the burned tissue, as well as delayed regeneration and healing.

RATIONALE: A significant body of data clearly supports the efficacy of hyperbaric oxygen in the treatment of thermal injury. A reduction in fluid requirements, less conversion of partial to full thickness injury, preservation of marginally viable tissue, improved microcirculation, reduction in edema, faster epithelialization, less inflammatory response, enhancement of PMN killing, preservation of tissue creatine phosphate, adenosine triphosphate, and decreased wound lactate have all been reported. A significant reduction in hospital stay and cost of treatment, noted when comparing patients treated with adjunctive hyperbaric oxygen therapy with those who did not, has also been reported. Hyperbaric oxygen therapy used as an adjunct to traditional burn care demonstrates greatest effects when initiated within the first 4 hours following the injury, or as quickly as possible.

SOURCE: UHMS Publication CR(HBO) 1996

RADIATION TISSUE DAMAGE (Osteoradionecrosis)

Patients must be treated in close coordination with referring physicians. Hyperbaric oxygen therapy must be part of an overall plan in which debridement, resection of involved bone, bone grafting, myocutaneous flap reconstruction, and specific antibiotic therapy are included. Pre- and postoperative hyperbaric oxygen is indicated to achieve a resolution.

RATIONALE: At some time after irradiation for therapy of malignancies, a small percentage of patients develop disabling, painful, and potentially fatal tissue breakdown. The basic physiology of this process is a progressive obliterative endarteritis with resultant tissue ischemia. In soft tissue radionecrosis, newer reconstructive techniques often allow total excision of the involved area with repair by vessel-bearing myocutaneous flaps. In some cases, however, particularly the mandible and maxilla, total surgical ablation would be disfiguring and reconstruction impossible. Hyperbaric oxygen therapy for radiationdamaged tissue was introduced in the early 1970's. With daily elevation of oxygen tension in hypoxic, hypocellular, and hypovascular bone and soft tissue near regions of functioning capillaries, fibroblast proliferation, collagen synthesis, and angiogenesis proceed. The killing ability of hypoxic leukocytes is enhanced with the elevation of tissue oxygen tensions. Preoperative hyperbaric oxygen prepares a vascular, noninfected wound to enable the surgeon to successfully debride and later reconstruct such entities. Unplanned but required surgery in previously irradiated tissue has an increased incidence of complications. Adjunctive hyperbaric oxygen therapy is indicated postoperatively in this clinical setting.

NECROTIZING SOFT TISSUE INFECTIONS

(Subcutaneous Tissue, Muscle, Fascia)

Hyperbaric oxygen therapy may be used as an adjunct to surgical and antibiotic treatment of soft tissue infections, with tissue necrosis, due to mixed aerobic and anaerobic organisms. These conditions include necrotizing cellulitis, progressive dermal gangrene, and severe cases of anaerobic streptococcal myositis, crepitant anaerobic cellulitis, and necrotizing fasciitis.

RATIONALE: Organisms other than clostridia can cause necrotizing infections. In these synergistic situations one characteristic is the presence of bacterial species with differing oxygen requirements. Favorable clinical reports indicate an adjunctive role for hyperbaric oxygen in these difficult and often life- or limb-threatening infections. Primary management remains adequate surgical debridement and antibiotic coverage. Hyperbaric oxygen is a useful adjunct in difficult cases. The oxygen tension in infected tissues is low. Increasing tissue oxygen tensions enhances white cell killing of bacteria, promotes inhibition of anaerobic organism growth, and increases the oxidation reduction potential. The high mortality and morbidity associated with these conditions warrant the addition of adjunctive hyperbaric oxygen therapy.

SOURCE: UHMS Publication CR(HBO) 1996

ENHANCEMENT OF HEALING IN SELECTED PROBLEM WOUNDS

Problem wound is an often used term to describe a group of hypoxic and/or hypoperfused wounds which have failed standard medical and surgical therapy. These include diabetic wounds, dehiscent amputation sites, non-healing traumatic wounds, and vascular insufficiency ulcers. All of these wounds have the underlying problem of tissue hypoxia, with oxygen tension usually below 20 mmHg, and, therefore, are more prone to infection.

RATIONALE: The elevation of oxygen tension by hyperbaric oxygen therapy has powerful effects on wound dynamics. Hyperbaric oxygen enhances leukocyte bactericidal activity and promotes the fibroblast-collagen support needed for neovascularization. Hyperbaric oxygen therapy for difficult wounds should be undertaken with the clear understanding that it must be part of a vigorous and coordinated surgical team approach to total patient management.

CRUSH INJURY, COMPARTMENT SYNDROME AND OTHER ACUTE TRAUMATIC ISCHEMIAS

Hyperbaric oxygen therapy must be initiated as soon as possible after the injury and must be used as an adjunct to standard surgical treatment, including vascular repair as indicated.

RATIONALE: Crush injury involves severe trauma to bone, soft tissue, nerve, and vascular structures. Viability may be questionable. There is a gradient of injury ranging from viable compromised tissue to that which is irreparably damaged. Compartment syndromes and threatened reimplantations can additionally produce various degrees of ischemia, edema, and tissue damage. Therapy should be initiated within 48 hours after the injury to be most effective, preferably within the first 4 to 6 hours. With 100% oxygen inhalation at increased pressure, plasmadissolved oxygen is delivered to marginally perfused tissues in the wound sustaining viability, enhancing leukocyte killing, and reducing edema.

SOURCE: UHMS Publication CR(HBO) 1996

OSTEOMYELITIS (Refractory)

Cases accepted for adjunctive hyperbaric therapy must be judged to be refractory to adequate surgical debridement, appropriate parenteral (or equivalent) antibiotics, and nutritional support. Hyperbaric oxygen provides periodic elevation of bone and tissue oxygen tensions from hypoxic to normal or hyperoxic levels. This promotes angiogenesis, increased leukocyte killing, aminoglycoside transport across bacterial cell walls, and osteoclast activity in removing necrotic bone. Judgement in declaring a given case refractory or critical must be made jointly by the surgeon and the hyperbaric physician.

RATIONALE: Following anecdotal case reports of successful use of adjunctive hyperbaric oxygen therapy in the 1960's, controlled animal studies have clearly demonstrated the benefit of hyperbaric oxygen. Published clinical series that utilized adjunctive hyperbaric oxygen therapy for chronic refractory osteomyelitis and follow-up data have confirmed the controlled animal data. When used according to guidelines, hyperbaric oxygen is clinically efficacious and cost effective. In a limited review, cost effectiveness was five-fold in favor of using hyperbaric oxygen for refractory osteomyelitis.

SKIN GRAFTS AND FLAPS (Compromised)

Hyperbaric oxygen therapy is not necessary or recommended for the support of normal, uncompromised skin grafts or flaps. However, hyperbaric oxygen is effective in promoting capillary proliferation and granulation to prepare a surgical site. This preparation is essential in patients who require grafts or flaps to cover areas where compromised microcirculation or hypoxia contribute to questionable viability of the transplanted tissue.

RATIONALE: Hyperbaric oxygen therapy can help to maximize the viability of compromised graft or flap tissue following transplantation, thereby reducing the need for repeat surgical procedures.

SOURCE: UHMS Publication CR(HBO) 1996

INTRACRANIAL ABSCESS

Intracranial abscess was added to the list of "approved indications" for HBO by the 1996 UHMS Committee Report. Over the last two decades there has been a gradual drop in mortality to the present 17%. However, certain patients remain problematic at best. These include patients with multiple abscesses and/or deep or dominant abscesses, patients who are compromised hosts, patients in whom surgery is contraindicated, and patients unresponsive to standard surgical and antibiotic regimens. Here HBO plays a role. Although only 20 patients have been reported, there is a 0% mortality. This is a remarkable finding. As a result, patients meeting these criterion should be seriously considered for HBO therapy.

EXCEPTIONAL BLOOD LOSS (Anemia)

Exceptional blood loss anemia occurs when the patient has lost sufficient red cell mass to compromise respiratory requirements and cannot or will not receive transfusions because of medical or religious reasons.

RATIONALE: The intermittent use of hyperbaric oxygen therapy will supply enough oxygen in the severely anemic patient to support the basic metabolic needs of the respective tissues of the body until red blood cells are restored. Most vitally, our brain requires a supply of 6 volumes percent of oxygen to support its basic metabolic requirements. Hyperbaric oxygen at 3 ATA will place 6 volumes percent of dissolved oxygen in an intravascular acellular perfusate. Thus, hyperbaric oxygen is a valuable adjunct when used early in treating acute blood loss in those who cannot receive blood replacement for medical or religious reasons.