Hyperbaric Research Papers

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History of Undersea and Hyperbaric Medicine

Therapeutic hyperbaric medicine, the concept and use of compressed air, was first recorded as early as 1662. Initially, with no scientific or medical basis, the efficacy of hyperbaric medicine has evolved significantly through trialing. Through experimentation and clinical trials, specific scientific criteria has defined both appropriate and experimental uses of hyperbaric oxygen.

In the late 1600’s the first chamber known as a dominicilium was constructed and hyperbaric air compression was exercised to a depth of approximately 2 atmospheres. Currently it is thought that any therapeutic affects on patients was purely psychosomatic. Shortly thereafter, larger dominicilii were constructed to accommodate a larger volume of patients as well as having the ability to descend the patients to depths greater than 2 atmospheres.

Fontaine, a French surgeon, made some significant contributions to the concepts of hyperbaric medicine. In the 1800’s, with surgeries performed within the hyperbaric chamber, he introduced the use of nitrous oxide as a general anesthetic. Post operative patients expressed marked decreases in cyanosis as well as a reduction in postoperative hernias. It was hypothesized and later shown that nitrous oxide was discovered to be significantly safer to use under the increased hyperbaric pressures of oxygen. In 1891, Dr. Corning brought hyperbaric medicine to the United States and was the first to operate the chamber with an electric compressor. He pioneered spinal anesthesia under HBO conditions. By 1956 Dr. Ite Boerema, University of Amsterdam, published his first paper on the performance of complex operations within a hyperbaric surgery suite to include
the correction of transposition of the great vessels, tetralogy of Fallot, and pulmonic stenosis.

In the mid 1900’s scientific data started to support the value of hyperbaric medicine. Along with the observation of the initial decrease in postoperative cyanosis and hernias, nitrogen and oxygen toxicity thresholds were documented. Regular clinical use of HBO technology was finally in place by 1955. Prior to 1955, hospital and clinical operations were skeptical about HBO treatment effectiveness and refused to implement HBO programs. A large factor, which caused the skepticism, could have been the lack of communication between HBO operators world wide. Treatment regimes as well as patient outcomes were not shared and the data required to backup HBO efficacy was not in place nor was there a standardization of processes.

By the end of the 1960’s an International Congress on Hyperbaric Oxygenation was formed and information and techniques was shared throughout the world. And by the end of 1900’s hyperbaric medicine was in full swing. Various organizations had been formed to include Undersea and Hyperbaric Medical Society (UHMS), the Hyperbaric Society (HS) of the UK, the U.S. National Academy of Sciences/National Research Council as well as a renewed interest in HBO by the U.S. Navy and other branches of these major organizations have formed a committee, the Medical Education and Standards Committee. The purpose of the committee was to approve hyperbaric faculty and education.

The future of hyperbaric medicine is wide open. As HBO is presented to medical students in many parts of the United States and around the world, more and more physicians are open to the possibilities of HBO. Now a multi-specialty discipline
approach is used as HBO has been approved for the healing of chronic non-healing wounds and several other conditions. Because of this approach, most new chambers are being added to wound care specialty centers. As HBO technology progresses, its publications, uses and future is dependent on research. HBO has been shown to lower morbidity as well as be financially feasible within the health-care system.
High-Low Pressure Physics

The “Laws” of physics apply to those that believe they exist and even those that don’t. It is the comprehensive understanding of these laws of high and low pressure physics which are integral for successful hyperbaric oxygen treatment. Considerations are from safety under pressure changes, both high and low, to the therapeutic viability of the prescribed treatment.

Globally, the American and Imperial systems are used in the measurement, calibration and governing of HBO control systems. Common between both systems is the designation of 1 absolute atmosphere (ATA) at sea level equivalence. Converted, 1 ATA is equal to 14.7 psig or 1kg/cm$^2$. The ATA unity makes the treatment regimes identical between countries. In European countries, often times, the term “Bar” is substituted for ATA interchangeably.

The for mentioned “Laws” of physics which need to be under consideration for the safety and treatment of patients are as follows: 1) Boyle’s Law, 2) Charles’ Law, 3) Dalton’s Law and 4) Henry’s Law. “Robert Boyle observed that for a body of ideal gas at constant temperature, the volume is inversely proportional to the pressure.” Applied to HBO treatment, volume changes at the initial pressurization will have a maximum effect on the middle ear risking the patient, and tender, for barotraumas. However, the deeper the internal chamber environment becomes, the barotraumas risk becomes less likely. Pressurization should be initiated and implemented slowly to minimize negative affects.

“Charles observed that for a body of ideal gas at constant pressure, the volume is directly proportional to the absolute temperature.” HBO is primarily interested in the inverse of his law: “compression of a gas will make it hotter.” Therefore, with fast
pressurization, the internal chamber environment can become uncomfortably warm. Combined, both laws can be represented as \((P_1V_1/T_1 = P_2V_2/T_2)\). The importance of these laws combined is the comfort and safety during descent and ascent.

John Dalton stated “in a mixture of gases, the sum of the partial pressures of the gases in the mixture equals the total pressure.” Dalton’s law aids in the calculations of the effects of pressure changes. If we observe an oxygen pressure at 160 mmHg and a nitrogen pressure at 600 mmHg, respectfully at sea level, we will mathematically achieve 760 mmHg total pressure at 1 ATM.

Henry’s Law states that “the partial pressure of a gas dissolved in a liquid is equal to the partial pressure of that gas exerted on the surface of the liquid at equilibrium.” In combination with Dalton’s Law, we are able to determine the partial pressures of a gas dissolved in a liquid and we can conclude an equality of diving pressures to HBO pressures at equivalent ATM pressures. With these laws under consideration, various treatment options are available based on requirements, diving, wound care, experimental considerations. The design of HBO treatments can also be augmented with the addition of other gasses such as helium. The patient would still have enough O\(_2\), for instance 3% of O\(_2\) of 5320 mmHg total gas at 7 ATA would still be 160 mmHg of O\(_2\) which is enough to sustain life; compatible treatments for saturation divers. The ability to maintain an O\(_2\) concentration of 160 mmHg or 0.21 ATM under all circumstances, no matter what the pressure is normoxic mixtures.

Bunsen’s Solubility Coefficient (BSC) determines the amount of gas in solution in a liquid, like nitrogen in our bodies during diving exercises. By multiplying the partial pressure of the gas by its BSC, the actual amount of gas dissolved can be determined.
Knowing the nitrogen solubility differences between oil and water helps us to understand the nitrogen solubility within different tissues of the body, i.e. lipids and blood or CSF. We are now able to determine which gases can remain in tissues longer than others, such as nitrogen versus helium, and the estimate their effects on the body.

Combining all the laws and principals provides tools for the HBO physician to design treatment guidelines. Understanding the consequences and benefits of pressure changes provide for safety guidelines as well as occupant comfort. It is well known that the laws of physics apply even in the absence of there awareness.
Oxygen Toxicity and Nitrogen Narcosis

Oxygen and nitrogen are the general gasses in our atmosphere and what we breathe in order to sustain life. Although, on average we breathe about 79% nitrogen and 21% oxygen, we only utilize portions of oxygen in each breath. Nitrogen is expelled as quickly as it is inhaled and mammalian lungs do not permit it’s intake into the body. Oxygen, being essential for life, is also toxic at elevated levels beyond our body’s ability to utilize and counter act via antioxidant mechanisms.

Biochemical concern oxygen poisoning is formation of oxygen free radicals in the body and the ability to neutralize and remove these toxic components. A hyperoxic environment can produce an inactivation of critical body enzymes, cellular membrane damage and possible oxidation of proteins as well as lipids. Antioxidants used for our defense include superoxide dismutase, catalase, glutathione peroxidase, glutathione reductase, and the enzymes of the pentose monophosphate shunt pathway. Also, in our diet we ingest glutathione, selenium, vitamin E and vitamin C which have antioxidant properties.

Oxygen toxicity has been shown to initiate oxygen-induced seizures. Abruptly or preceded by other symptoms, such as facial twitching, treatment of hyperoxic seizure conditions include drugs that inhibit cytochrome P-450 monooxygenases in neuron cells and thus inhibit the sources of neurotransmitter synthesis. Iron chelators such as the 21-amino steroids, can be used to preserve lipid structures from peroxidation. The adrenergic blocking properties of propranolol aid in the case of neurological manifestations and may decrease cerebral blood flow. It is also theorized that low dose
caffeine produces cerebral vasoconstriction and act as an antioxidant which may have antiepileptic properties aiding in the delay of oxygen convulsions.

Grand mal convulsions have been noted in patients under HBO treatment at partial pressures greater than or equal to 2.0 ATA. Progressive myopia and other ocular tribulations have also been noted. Therefore, HBO tenders are assigned to monitor the patient for leading signs of neurological abnormality. Once detected, the oxygen supply can be removed and replace with the ambient air of the chamber which resembles a normal 21% oxygen mixture. Oxygen treatment can continue once the toxic effects have subsided.

HBO treatment tables have been developed in an effort to diminish the side effects of hyperoxia therapy. The partial pressure of oxygen is kept below the 0.5 ATA limits imposed by pulmonary irritation and distress. Treatment is organized into periods of pure oxygen separated by longer periods of “normal” air exposure. The UPTD tables account for repeated exposures to hyperoxic environments. The cumulative effects of pulmonary oxygen toxicity with repeated exposures can be estimated by multiplying the UPTD of the treatment by the number of treatments. These tables are utilized as guides for treatment plans. Predefined tables have been created by the United States Navy and are the current starting place for treatment development; USN Tables 6 and 6a.

Mammalian experimentation with oxygen tolerance modification has been observed. Adrenocortical hormones, CO₂ inhalation, epinephrine, norepinephrine, hyperthermia, insulin and vitamin E deficiency seem to accelerate the onset of oxygen toxicity. Conversely, propranolol, reserpine, succinate, disulfiram, hypothermia, vitamin E, and adrenergic and ganglionic blocking drugs retard the onset of toxicity. While
studies on humans insufficient, this knowledge of retarding and exacerbating agents can help with the implementation of the HBO treatment.

Around depths of 3 ATA, nitrogen buildup in the body can cause a condition known as nitrogen narcosis. Neurological symptoms include confusion, a goofy persona, bad judgment and mental unawareness. Nitrogen narcosis can be easily reversed by depressurization of the patient. Unless specific treatment is mandated for a patient, most treatments are well above the 3 ATA pressure depths.

With the concepts of oxygen and nitrogen toxicity in mind, treatment programs are developed in a conservative manner. Toxicity levels in general are not achieved and patients are afforded the security of a lucrative treatment with minimal toxic risks involved. The assistance the US Navy and their experiences have provided physicians with much of the research required to provide adequate and safe services to patients.
Physiological Effects of Hyperbaric Oxygen

Hyperbaric oxygen has two major effects on the body. A mechanical effect which is useful in reducing bubble size and the effects of an increased partial pressure of oxygen. Under the circumstances of an increase in partial pressure above 1 ATA, oxygen is then utilized as a drug with specific indications and side effects. Understanding how oxygen affects an HBO environment will aid the treating physician in decreasing the side effects of oxygen under pressure and increase the viability of its treatment.

Boyle’s law tells us that volume is inversely proportional to the absolute pressure. Under consideration is the potential barotrauma in the middle ear, sinus squeeze and pulmonary squeeze or rupture upon decompression. However, under recompression, the concept of compressing excess nitrogen gas, then displacing with oxygen, allows the nitrogen bubbles to conform to a spherical shape. Spherical bubbles are then able to be passed through the vasculature to the lungs where they can be expelled. Other conditions are patients suffering from gas gangrene or gas embolism. Under pressure the gas bubble volume will decrease in size and better perfusion will lead to the reduction of pain and or a decrease in clinical symptoms. Under compression nitrogen and helium rapidly diffuse from cells into the blood. Based on Boyle’s law, the decrease in size and spherical shape will cause less vascular damage due to platelet activation.

At 2.8 ATA the blood plasma is capable of becoming saturated with an excess of 6% oxygen above and beyond that of the hemoglobin carrying capacity. Therefore, the blood plasma is capable of carrying enough oxygen to meet the needs of the body’s tissues. Numerous physiologic effects occur within the body under these conditions. They include suppression of alpha-toxin production in gas gangrene, enhancement of
leukocyte-killing activity, decrease in white cell adherence to capillary walls, vasoconstriction in normal vessels, restoration of fibroblast growth and collagen production, stimulation of superoxide dismutase production and many more. Some studies have show a decrease in blood flow to hyperoxic tissues, however, the excess oxygen saturation of blood that does make it to the tissues well compensates for the vasoconstriction.

Other studies have shown an increase in healing of hypoxic wounds, the inhibition of clostridium perfringens, a reduction of carbon monoxide toxicity, improvement of bone repair, and suppression of autoimmune responses. For example, the tensile strength of wounds with relation to collagen deposition and the rate at which they close is directly affected by the amount of available oxygen. Open wounds will heal faster and potentially with less scaring in a hyperoxic environment. It has also been found that endothelial cell proliferation is increased while under an HBO environment just after 15 minutes and fibroblasts after 120 minutes, therefore speeding up the healing of endothelial injury. Also under consideration is the “Robin Hood” effect. Experiments were conducted on diabetic patients who had lost the majority of their blood supply to their foot extremities. Through a series of HBO treatments, it was evident that angiogenesis had taken place and blood flow, although marginally, had returned to ischemic tissues.

With a decreased lipid peroxidation, muscular reperfusion injury and bowel reperfusion syndrome have been decreased by HBO treatments. Improved tissue salvage in ischemia-reperfusion injuries were demonstrated with HBO in combination with pharmacological piracetam.
Oxygen as a prescribed drug under pressure conditions has a many-sided comprehensive effect on the organs and tissues of the body. The primary interest in HBO treatment is in the aspect of metabolism where oxygen is no longer seen as a simple oxidizer. Through following the guidelines and experimentation, HBO treatment is relatively safe and seemingly effective for many potential uses, even ones yet to be discovered.
Decompression Sickness

The presence of bubbles formed from gases that have become dissolved in the tissues of the body during a sojourn at raised environmental pressure is known as decompression sickness (DCS) or decompression illness (DCI). Other similar manifestations and pathogenetic conditions exist which are considered to be parallel to DCS such as arterial gas embolism (AGE), which is a secondary condition to decompression damage of the lungs (pulmonary barotraumas, PB). Historically, the validity of HBO treatment has been questioned, but over time, the validity of DCS has been well documented and the modern day physician has been armed with numerous accounts of positive HBO treatment regimens.

As divers ascend Boyle’s Law allows gasses to revert from a soluble state back into bubbles (a gaseous state). At controlled ascents, these bubbles are transported to the lungs for expulsion. However, in the case of DCS, these bubbles can form and deposit either intravascular or extravascular, most commonly, uncontrolled ascents from depths greater than 10 meters. Under pressure, the hydrophobic cell membranes afford a prime environment for compressed nitrogen to dissolve into solution within our bodies. Nitrogen is the primary gas we are concerned with in DCS conditions.

As nitrogen bubbles form during ascent, the bubbles can take a variety of pathways. If uncontrolled, the bubbles can block the surface activity at the blood-gas interface or cause problems by simple mechanical expansion. Mechanical expansion can disrupt arteriole blood flow of the brain and or pressure on nerve cells causing neurological manifestations, while blockage of the blood-gas interface prevents the gas exchange in the alveoli of the lungs. Also, as bubbles form and pass through the
vasculature, hypoxia is possible due to obstruction. Along with gas emboli and obstruction, endothelial injury activates platelet aggregation and complement C5a. Considering the location of injury, spinal blood flow obstruction is common and detrimental if not treated promptly.

Recognizing DCS can be blatant or challenging depending on the severity of presentation. Cutaneous signs are itching, purpuric rash, usually over the upper trunk, and “Peau d’orange” is possible along lymphatic channels in the limbs along with possible edema. Musculoskeletal pain is the most widely known manifestation of DCS, also known as “The Bends”. Based on type of diving, the most common complaints are knees and shoulders, however any synovial joint is at risk for nitrogen gas accumulation with exception of the temperomandibular joint. Other warning signs include malaise, anorexia and a degree of fatigue disproportionate to the amount of preceding activity. Circulatory collapse can be signified by retrosternal pain accompanied with dry cough and shallow respirations. A feeling of spaciness, dysarthria, visual disturbances or occasionally psychosis represent initial neurological symptoms while more serious conditions such as ipsilateral deafness or ascending paraplegia and even quadriplegia can occur. “Any neurological symptom or deficit after a dive should be considered to be a manifestation of decompression illness, until proven otherwise, and must be managed accordingly.”

If symptoms arise within 24 hours of a dive, DCS should be the initial diagnosis. Treatment needs to be implemented immediately before damage becomes permanent and treatment becomes less effective. In an effort to maximize treatment validity, neurologic and cardiopulmonary illness may be conducted at depth after treatment has begun. Under
emergent conditions, this could make the difference between a successful or detrimental outcome.

In general, USN Tables 5 and 6/6A have been the standard treatments for both DCS type 1 and type 2. Treatment protocols of alternating oxygen and air at a depth of 3 (2.8) to 2 ATA in the hyperbaric chamber are the most common prescriptions. Based on the severity of DCS, Table 5 might be employed for cutaneous symptoms, while “The Bends” symptoms would use a Table 6/6A. The difference between the two is the oxygen/air interval and overall length of treatment. In even more severe cases of DCS (Type 3) a heliox mixture can be employed which take the patient significantly beyond 3 ATA.

As the pressure in the chamber increases and the patient and tender descend to the prescribed depth, it is possible to see worsening symptomatic conditions. Termed “bone bubble”, it is theorized that the presence of small bubbles underneath the periostium of bone shrinks and moves around causing pain in an area which is rich in pain receptors. The process of recompression can be retarded but should not come to a halt until the desired treatment depth is reached. The “bone bubble” will eventually be reabsorbed and the pain will dissipate.

In conclusion, DCS conditions should be treated as soon as possible. Residual neurological symptoms may indicate longer treatments until symptoms subside or until no further improvement is noted in the patient. Divers of all types make up the majority of DCS cases and treatment tables have been designed and implemented successfully if medical help and facilities are available within a reasonable time frame from the first onset of symptoms.
Vascular Gas Embolism

Vascular gas embolism is the introduction of mobile gas forms within the vascular system, either arterial or venous. Arterial emboli is of much greater importance than venous emboli, however gas within the venous system may paradoxically get into the arterial system, known as a paradoxical embolism. As pulmonary arterial pressures increase, the right atrium may temporarily exceed pressures in the left atrium and a normally closed probe-patent may be inadvertently opened casing an intra-atrial septal defect. The severity of vascular gas embolism can range from negligible to fatal. If the embolus follows a path to the coronary artery, immediate death may be the end result.

Mechanical blockage of arterial vessels is the primary mechanism of how vascular gas embolism facilitates its damage. Arterial damage is generally greater than venous damage due to the release of gasses as they pass through the lungs, acting as a natural filter for the balance of partial pressures. If gas embolus obstructs vasculature on the arterial side, blood flow is obstructed, causing coagulative necrosis, platelet aggregation forcing endothelial injury and eventual leukocytosis causing an arterial thrombus. The end results being irreversible damage due to hypoxia and ischemia.

The causes of gas embolism are numerous. Commercial and sport diving, water accidents such as escape from a submerged vehicle, insufflations of air into the vagina during pregnancy and other gynecologic manipulation, and iatrogenic causes are a few of the listed causes. Under diving conditions, if a diver holds their breath during ascent, intrapulmonary pressures become greater than the surrounding water pressure and force a condition known as burst lung syndrome. Complications of burst lung syndrome include mediastinal emphysema, pneumothorax, or arterial embolus. In submerged vehicle
accidents, victims often hold their breath to the last moment and then race to the surface without exhaling. Their actions will usually find them unconscious on the shoreline or face down in the water due to the same expansion of gases as they ascend.

The vagina is also an entry point of gas into the body. During pregnancy examinations or even during sex play, air insufflation has been reported to produce cerebral catastrophic accidents. Air passes through the cervical os → to dissect between the placental membranes and the uterine lining → entering the uterine sinusoids → to the vena cava → to the right heart → to the lungs. Any bolus greater than a liter of gas arriving at the lungs may overwhelm the pulmonary filter and cause leakage of some gas through the intrapulmonary shunts → this will lead to arterial embolus passing through the left heart and through the carotid arteries.

Iatrogenic emboli are the most common cause of cerebral accidents seen in the hospital. Bubbles are introduced into the arterial side from heart lung machines, open central lines, percutaneous lung biopsy, cardiac catheterization, renal dialysis and head and neck surgeries. Patients susceptible to atrial septal defects are especially prone to gas emboli. Physicians need to be able to offer hyperbaric treatment and offer this treatment as a possible consequence of procedures to patients and disclose the possibilities of hyperbaric treatments to the patients prior to their procedures. It is estimated that 30% of adults in the U.S. posses a atrial septum that may become patent. This is especially important to consider with such patients susceptible to slight increases of pressure in the right atrium.

Treatment for a victim of gas emboli is a must. The Trendelenburg position, in which the patient is positioned on his or her left side, is an initial step in management or
placing the patient in a head down position (10 min max in head down position). Both, until hyperbaric treatment can be initiated, under no circumstances is the patient allowed to become vertical, even in the absence of symptoms. In general, once a hyperbaric facility is reached a U.S. Navy Table 5 and/or 6 are commonly used treatment prescriptions. After recompression treatment has been completed, lidocaine has been given in antiarrhythmic doses as a protective effect in acute cerebral ischemia caused by gas embolism.

Hyperbaric facilities are in place for the vascular gas embolism, however, do to the varying causes, it is obvious that hyperbaric practices needs to be employed in more than just a diving arena. Do to iatrogenic causes and even accidental “Near Drowning” in lakes and rivers, it is important to offer recompression therapy. The diagnosis and recognition of gas emboli provide just one more reason for the implementation of hyperbaric medicine.
Problem Wounds

Hyperbaric oxygen has shown a positive outcome in the treatment of problem wounds. From thermal burns to recurrent or persistent diabetic ulcers, hyperbaric oxygen has increased the ability of the body to deliver oxygen to the damaged tissues. It has been shown that patients treated under hyperbaric conditions vs. normobaric conditions have benefited significantly in the duration of the healing time.

Oxygen tension is a major controlling factor in bacterial killing, resistance to infection, collagen synthesis and deposition, angiogenesis, and epithelization. Under hypoxic conditions, some or all of these processes are impeded. The role of hyperbaric oxygen in these cases would be to saturate the potential microvasculature that still remains with oxygen in order to deliver at least a marginal amount of O2 in order to promote the healing process.

Healthy wounds in well-vascularized tissues are extremely resistant to infection. Ischemic wounds, however, are highly susceptible. Inhibitors of oxidative pathways, such as cyanide and hypoxia, do not inhibit phagocytosis, but greatly impair leukocyte migration rates and microbial killing. The ability to produce oxygen radicals and to kill via oxidative mechanisms is greatly proportional to local oxygen tension in the wound environment. It has been shown in microbial killing mechanisms; the oxygen dependent system is required for adequate antibacterial activity in granulocytes. The system requires the consumption of large amounts of molecular oxygen immediately after phagocytosis in a process called the “respiratory” or oxidative burst. Deficiency in this oxygen dependent system results in the dramatic lessening of the antimicrobial properties of polymorphonuclear cells, or PMN’s. Hypoxic conditions from lesser NAD+
concentrations due to the amplified lactate dehydrogenase activity prevent the inhibition of the adenosine diphosphoribose system and collagen synthesis takes place.

In the role of angiogenesis, blood vessel growth cannot proceed without necessary substrates, like oxygen, regardless of the amount of stimulating cytokines present. Hyperbaric transport of oxygen can sometimes deliver just enough oxygen to deprived tissues to initiate vascular epithelium growth factor by macrophages which is a required process to promote angiogenic factors needed for wound-healing. Oxygen is also important for wound epithelization.

Epithelization is a process dependent upon cell movement and coverage. For this reason, wounds heal more effectively in a “moist” environment. Cells migrate more effectively through a moist environment, partially because ambient oxygen diffuses far more readily through wet rather than dry tissues.

Hyperbaric oxygen is primarily used in cases of non-healing wounds. The etiology of a specific wound type might dictate the nature of treatment and the warranting of hyperbaric oxygen to aid in the repair process. Conditions ranging from a diabetic pressure ulcer, venous ulcers, amputation candidates, graft-treated wounds or others resulting from venous diseases and burns are major treatment candidates. Wounds are assessed and ranked based on their severity. Stage 1: non-blanchable erythema of intact skin. Stage 2: Partial thickness skin loss involving epidermis and/or dermis. Stage 3: Full thickness skin loss involving damage or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia. And Stage 4: Full thickness skin loss with extensive destruction, tissue necrosis or damage to muscle, bone, or supporting structures. This is the National Pressure Ulcer Advisory Panel grading system.
Assessment of diabetic foot disorders are ranked based on their severity via the Wagner Grading System which assesses the amount of gangrene within the patient as well.

It is important that the hyperbaric practitioner assures that the wound has enough blood supply to heal prior to initiation of wound management and hyperbaric treatments. Determination of viable oxygen blood supply, oxygen toximetry, is determined by the use of a transcutaneous oxygen-pressure device (T-CpO₂). The successful HBO candidate is able to achieve a value of 100 mmHg within approximately 20 minutes when breathing 100% O₂ at one atmosphere via a tight-fitting mask. If the patient fails the initial “Oxygen Challenge”, the patient should be placed into the chamber and given 100% O₂ for 20 minutes at a depth of 2.5 atmospheres. If the patient does not achieve a T-CpO₂ greater than 200 mmHg then the patient should be rejected for hyperbaric treatment, in the absence of infection. If the periwound T-CpO₂ is less than 30 mmHg, it can be assumed that the wound will not heal without solving the oxygen deficit problem. Studies have shown that volunteers who didn’t attain these values did not benefit from treatment. Reasons for these results may have been due to preexisting vascular diseases, extensive necrotic tissue damage or by inadequate perfusion of these tissues.

Wound healing has a number of factors stacked against successful closure. Along with infections and ischemia, proper medical treatment is faced with health risk factors such as nutritional deficits, vascular insufficiency, diabetes mellitus, soft tissue infections, chronic tobacco use, radiation damage, immunosuppression, renal failure and cancer. Hyperbaric oxygen therapy provides a significant increase in tissue oxygenation in the hypoperfused, infected wound. It provides molecular oxygen at a cellular level; it also significantly increases leukocyte bactericidal activities.
Carbon Monoxide Poisoning

Carbon monoxide is colorless, odorless, tasteless, and nonirritating; it is one of the most common causes of poisoning in the world today. It is a byproduct of fossil fuel combustion and fire which has an affinity for hemoglobin more than 200 fold than that of oxygen and is a primary competitor for the binding sight. Chronic exposure via pollution, cigarette smoke, coal and oil home heating, and industrial workers exposed to CO have shown and increase propensity to cardiovascular and pulmonary diseases. About 1/3 of carbon monoxide poisoning victims remain undiagnosed because of the difficulty of detection. Also, the byproducts of chemical combustion in fires, such as hydrogen cyanide and sulfide along with several other chemicals may cloud the presentation and diagnosis of CO poisoning. Many of these chemicals are considered to be asphyxiants. The sooner a proper diagnosis of CO poisoning is made, the better the odds of prevention of irreversible injury.

Acutely, hyperventilation is seen in many patients. Most patients will present with neurologic deficits. Initially based on exposure, tightness across the forehead and headache were experienced at levels between 10-20% COHb. Levels of 20-30% resulted in a throbbing headache in the temporal regions while 30-40% produced severer headache, generalized weakness, visual changes, dizziness, nausea and vomiting, and ultimate collapse. 40-50% produced syncope, tachycardia, and tachypnea and levels above 50% were associated with coma, convulsiveness and death above 60% due to cardiac depression and respiratory failure. Survivors of serious poisoning were known to develop neurological and psychiatric sequelae such as dementia, Parkinsonism, amnesia, and depression. Patients may not present with symptoms until up to three weeks after
the event of poisoning. Because of the affinity of CO for Hb a patient may be rendered in a persistent hypoxic state if the frontal and parietal lobes of the brain become affected severely enough. However, often times, just flu-like symptoms in a group of family members or even problems with pets might trigger the thought of CO poisoning.

Children often times present with lethargy and syncope as the most important clues in their diagnosis. They may also experience nausea, vomiting and diarrhea. Based on age, children may have a number of complexities within the hyperbaric environment. Sometimes pressure equalization is difficult and the child may experience barotraumas or even develop convulsions due to oxygen toxicity. However, the majority of children benefits from HBO treatment and returned to a “normal” state post treatment.

Cyanide impairs oxidative phosphorylation resulting in the depletion of cellular high energy phosphate stores and lactic acidosis. It does this via binding of the cyanide anion to the ferric iron of mitochondrial cytochrome oxidase. In general acute treatment is a kit which contains amyl nitrite perles for inhalation, as well as vials of 10% sodium nitrite and 25% sodium thiosulfate for intravenous administration. It produces methemoglobin which the ferric iron of the heme moiety will bind to cyanide and free the cytochrome oxidase to function in the respiratory chain.

Simultaneous poisoning of cyanide and CO causes an induction of carboxyhemoglobin, which synergistically has the added functional anemia caused by severe levels of methemoglobin formation. Synergistic poisoning of cyanide and CO is evident in cigarette smokers and this combination may explain the deaths associated with sub-lethal levels of carboxyhemoglobin. Hypoxia caused by methemoglobinemia can be improved with hyperbaric oxygen.
HBO treatments generally accelerate the rate of CO dissociation from hemoglobin. Brown and Piantadosi demonstrated that HBO at 3 ATM markedly accelerated the dissociation of CO from the cytochrome oxidase, and hence would be expected to relieve whatever compromise in oxidative phosphorylation is caused by CO binding to cytochrome oxidase. And finally HBO is related to the cascade of vascular injury triggered by CO poisoning. Treatment was found to be effective for preventing brain oxidative injury in animals because it inhibited leukocyte adherence to the vasculature that was mediated by B2 integrins.

The diagnosis and treatment for CO and/or cyanide poisoning need to be carried out swiftly. Due to fetal risks of intra uterine death due to high CO environments, pregnant women should be treated with HBO even if they appear asymptomatic. General ambulatory patients with symptomology or a possible history of poisoning, to include risk factors, such as smoking, could overt permanent neurological deficits with prompt HBO treatment.
Gas Gangrene

Clostridia are the bacterial genus which causes the myonecrotic infection known as gas gangrene. Accidental trauma is the most common cause with 50% of the cases arising as a consequence of contamination of traumatic wounds. 80-90% of contamination is with C. perfringens while the remainder of the genus composes the difference of infection often times in combination with one another. A single untreated episode of gas gangrene will rapidly advance a patient to the death. Among the treatments available, HBO integration has been shown to be a successful addition to the treatment regime.

Alone, our bodies have virtually no inflammatory response to the presence of the Clostridia organism and wound drainage is nonpurulent. Exotoxins propel the disease process and may be better described as an intoxication rather than an infection. Considering that alpha toxin is the only lethal toxin produced in significant quantity, its action, synergized by the action of other nonlethal toxins elaborated results in a rapidly spreading liquifacitive necrosis. Therefore, the aggressiveness and progressive nature of gas gangrene depends on the continuous production of alpha toxin.

There are two major requirements for the development of gas gangrene: 1) Clostridial contamination, and 2) A decreased oxidation-reduction potential in the wound. A lowered oxidation-reduction potential is a circulatory failure in the local area rendering the wound ischemic and hypoxic. There are four recognized forms of necrotizing clostridial disease, clostridial myonecrosis with toxicity, localized clostridial myonecrosis, clostridial cellulites without toxicity, and clostridial cellulites with toxicity.
Clostridial myonecrosis with toxicity is considered to be “true gas gangrene,” and is characterized by diffuse and rapid spread from the initial site of involvement. The wound is blanched and tense to the touch from severe edema, toxicity rapidly progresses from this point onward. Gas bubbles may be seen in the drainage, felt as crepitations in soft tissue and often there may be some degree of hemolysis as a rule in gas gangrene. With localized clostridial myonecrosis the infection tends to remain localized and the manifestation of toxic symptoms are few to none, this is relatively uncommon and found in nonsterile injection of drugs/IV drug users. Clostridial cellulites without toxicity involve the limitations of the subcutaneous tissue without infestation of muscle. This is also called a “gas abscess.” Clostridial cellulites with toxicity present in a small number of patients the same as toxic clostridial myonecrosis. It is just as deadly as “true gas gangrene,” and requires invasive treatment measures. Patients with precipitating conditions such as DM or atherosclerosis are at greater risk of developing gas gangrene following traumatic wounds and therefore must be treated aggressively and carefully.

HBO treatment is an adjunct treatment in combination with surgical and antibiotic treatments. If diagnosed early enough, invasive surgical procedures may be avoided due to HBO intervention. HBO reduces the toxicity of gas gangrene infections by exposing the clostridial organisms to oxygen pressures of 3 ATM. This in turn, inhibited the production of the detrimental alpha toxins. In conjunction with antibiotics, such as penicillin G, gas gangrenous infections are brought under control by bactericidal killing via oxidative burst performed by PMN’s.
It is recommended to administer at least two hyperbaric oxygen treatments before the patient is taken to surgery for definitive debridement. Usually the disease process has been arrested by this time and operative and anesthetic risks have been decreased as well as well demarcated lines are clearly visible between viable and nonviable tissues. Post surgery, the patient recovery seems to show a favorable wound-healing and decreased hospitalization.
Soft Tissue Infections

Hyperbaric oxygen treatment presents evidence for the bacteriostatic and bactericidal effects of hyperoxia on microbial organisms. Increased levels of O2 tensions in tissues inhibit microbial growth by inhibiting various microbial metabolic reactions. Selected organisms are targeted because of reactive oxygen species or free radicals which are lethal for microorganisms that either lack or possess limited antioxidant defenses. Patients with chronic granulomatous disease lack the enzyme NADPH-oxidase which is necessary for oxygen-dependent killing of Pseudomonas aeruginosa and Staphylococcus aureus. Neutrophil activities increase due to oxidative burst mechanisms and pharmacologic half-lives of various antibiotics are increased and rendered more effective.

Under HBO conditions aerobic and anaerobic bacterial organisms become retarded. Anaerobic bacteria cannot survive in normoxic conditions because they lack the antioxidant enzymes to combat the oxygen free radicals. Hyperoxic conditions provide a synergistic environment for anaerobic destruction. Facultative and aerobic bacteria do have antioxidant enzymes and react differently under various conditions. The growth of some aerobic bacteria is enhanced by hyperoxic conditions but inhibited under HBO conditions which can be atmospheric dependent. For example, prolonged exposure to O2 tensions greater than 1.5 ATM inhibits the growth or is bacteriostatic for E. coli and P. aeruginosa, however, 1 hour intermittent exposure at 2 ATM has no effect on the growth of P. aeruginosa or S. aureus. In general HBO is bactericidal for aerobic and facultative anaerobic bacteria at pressures and/or durations which are greater than clinical uses.
The goal of HBO treatment is to induce the body’s own natural defenses to be able to ward off invading microbes. This can be accomplished by improving tissue oxygen perfusion necessary for oxidative burst-mediated antimicrobial killing by PMNs and reduced localized edema with improved circulation offered by higher oxygen load in the blood. With the increase in antibiotic half-life HBO increases the efficacy of the antibiotic chosen for the specific microorganisms. Under chronic conditions with necrotizing infections, HBO augments tissue survival by sustaining cellular oxidative metabolism. Hyperoxic conditions decrease tissue necrosis by decreasing bacterial numbers, and by enhancing the survival of ischemic infected tissue.

Besides bacteriostatic properties, HBO provides a killing ground for several other organisms. Parasitic infections like protozoa and helminthes are also affected based on the antioxidant properties which they may possess. Hyperoxic conditions work fairly well for most protozoa, but with helminthes it is dependent on the stage within the lifecycle. Adult schistosomes for example have higher levels of antioxidant enzymes than the larval stages and are protected against killing by increase O2 tensions. Basically, parasites have limited antioxidant enzyme defenses, but the differences and susceptibilities of their demise rests within the lifecycle at the time of exposure to greater O2 tensions.

In conclusion, soft tissue infections in general can benefit from HBO treatment. With the improved antimicrobial activity of PMNs by oxidative burst and a reduction in endotoxin production hyperoxic conditions have provided a valuable resource in the arsenal against infections. Even under experimental conditions, no harm has been
brought to a patient under HBO conditions and in times of uncertainty, HBO may be an adjunctive therapy buying time or synergistically adding to current treatments.
Osteomyelitis

Osteomyelitis is literally translated into inflammation of the bone. With the addition of bacterial or fungal infections into its definition, hyperbaric oxygen can be used as part of a treatment regime added to antibiotic and invasive surgical maneuvers. There are 4 recognized stages of osteomyelitis as characterized by the University of Texas Medical Branch in Galveston Texas. Stage 1) Medullary osteomyelitis, Stage 2) Superficial osteomyelitis, Stage 3) Localized osteomyelitis, and Stage 4) Diffuse osteomyelitis. In general, the most receptive patients are those having pre-existing conditions, like DM, peripheral vascular diseases, or extensive soft tissue scarring which prevents adequate wound coverage. The Immunocompromised or malnourished may also benefit from HBO intervention.

Anaerobic and many microaerophilic organisms lack the ability to produce superoxide dismutase and catalase which are the enzymatic mechanisms used by aerobic bacteria to degrade toxic oxygen radicals. Because of this, HBO therapy renders both intracellularly and extracellularly anaerobic organisms extremely sensitive to the oxygen radicals developed during treatment. The end result is that anaerobes are lethally affected by increased oxygen tensions directly induced by HBO, however many aerobes survive.

HBO uniquely assists in fibroblastic activities by facilitating oxygen tensions to greater than 200 mmHg. Fibroblasts are then able to produce collagen which forms a protective matrix and returns the collagen deposition back to normal values. In osteomyelitis, the reconstruction of bone is essential for recovery post surgery. HBO provides a means to increase both bone repair, bone strength and vessel ingrowths.
Studies have shown, however, there is an optimal HBO treatment period where potential hindrances might be experienced if HBO treatment is too much or too little. Bone repair is hindered when HBO treatment goes beyond the optimal ranges: 90-120 minutes at 2-3 ATA of 100% O2, once daily. Decreased treatments will result in slow bone healing due to inhibition of fibroblast, osteoclast, osteoblast, and macrophage activity. Above the accepted treatment, and sustained intervals, fibroblast activity is highly unregulated resulting in a thick collagenous deposition resulting in a repair that is rich in collagen, but structurally weak.

In the right combination of antibiotics, surgery and HBO treatment, healing from osteomyelitis is favorable. Aminoglycoside antibiotics have been shown to be enhanced with HBO treatments, resulting in improved efficacy of the drug. Considering that aerobic organisms may thrive in hyperoxic conditions, it is still beneficial to treat under HBO. As HBO promotes the activation of the bodies own PMNs, HBO is considered a vehicle for the necessary substrate (oxygen), for the PMNs to kill both anaerobic and aerobic organisms. In conjunction with warding off infection, HBO provides adequate oxygen for fibroblast activity, leading to angiogenesis and wound healing.

General protocols call for a once per day treatment with HBO pre and post surgery for osteomyelitis. This is because research has identified multiple treatments actually hinder bone formation and growth. Ideally, HBO should be employed during the treatment of osteomyelitis as it has also been shown to decrease overall treatment costs, but the hyperbaric group, surgeons, and infections-disease consultants all need to agree on the specified protocol which would be best suited for their patient.
Sternal Disease

Healthy individuals rarely experience sternal infections. Infection and disease come with those with depressed immune states, illicit drug usage with unclean needles, or trauma, which usually will be involved to establish direct or blood borne inoculation of the causative organism. Open mediastinal thoracic surgeries employing the techniques of sternotomy can also precipitate an infection within the sternum. Mortality in the first year following an operation complicated by infection is about 25%. Presentation and treatment is similar in management to osteomyelitis with the addition of incision, drainage, and intravenous antibiotics. The addition of HBO therapy has been effective in patient treatment and outcomes.

Treatment for sternal disease usually consists of incision, drainage, debridement, coupled with ingress-egress irrigation using antimicrobial agents. Later the development of the myocutaneous flaps aided dramatically in the survival of patients. Vascularized flaps reduced both the mortality and the morbidity of sternal wound problems; however this process requires multiple operative procedures to continuously repair the flaps in layers including a final skin graft. Successful closure of sternal disease requires the salvage of marginal tissue, reversal of local hypoxia, improvement of host defenses, healing processes, and perfusion along with adequate debridement, muscle or omental coverage, and appropriate antimicrobial therapy.

HBO therapy has served as a valuable tool in the success of these types of wound healing. HBO has provided a means to salvage marginal tissues and preventing of advanced necrosis or infection by reversal of the local hypoxia. The reduction of edema by hemodynamic alterations and prevention by ATP preservation along with endothelial
gap closure also affords the HBO advantage. HBO also prevents or reduces thrombus formation and further decreased perfusion by decreasing leukocyte and platelet adherence to capillary walls. Which in turn, the oxidative burst mechanism aids leukocyte killing of phagocytized bacteria, fibroblastic proliferation and subsequent fibroblast elaboration of collagen precursors, and cross-linkage to for the collagen matrix necessary for neovascularization required for the healing processes of such a serious wound.

There are some special considerations when using HBO therapy for sternal wound infections. The physician must keep in mind the known effects of HBO exposure in health and applicable disease states. In controlled studies, a decreased heart rate, cardiac output and stroke volume has been observed. There is an elevation in systemic vascular resistance and blood pressure. And coronary blood flow is reduced as well, however, the hyperoxia achieved within the HBO environment is sufficient to overcome any potential adverse effects noted. The effects of HBO on a myocardial infarction patient would increase the mean arterial pressure and systemic vascular resistance. There is also an increase in afterload during hyperoxia which is due to vasoconstriction. With this in mind, there are two theoretical points in which marginal hearts could fail. One, if they were incapable of tolerating the increased systemic vascular resistance during treatment, and/or managing the increased load with increased output following the treatment.

The physician should keep in mind pretreatment evaluation of the patient with the sternal wound at risk, or the already dehisced and/or infected sternal wound. They should consider the presence of known risk factors, preoperative evaluations including cardiac catheterization, echo, ECG, rhythm monitoring and pulmonary function studies. An
operative report and post-operative course may also assist the physician in determining the value of HBO adjunctive treatments.

In general, hyperbaric oxygen therapy should be considered for sternal wounds, both infected, failed or even as a prophylactic if a sternotomy must be performed. Some hospitals have mandated HBO adjunct treatments, unless their condition is such that the patients risk of treatment outweighs the potential benefits of the therapy. Economically, the costs of treatment with hyperbaric oxygen have also been shown to be a health system advantage and a decrease in hospitalization has been associated with HBO use.
Intracranial Abscess

Based on the concepts of the management of gas gangrene and necrotizing soft tissue infections, the treatment of intracranial abscess with adjunctive HBO was approved by the UHMS in 1994. Considering that it is hard to find experimental data with this type of wound, the collection of data regarding case reports are a high priority. In 1993 a last ditch effort was made to save a young mother’s life via HBO and with favorable results.

Intracranial abscesses account for only three to five admissions per year at large medical centers, however the mortality from various countries around the world range from as little as even 0% in the US between 1978 and 1986 to as high as 31% in Sweden from 1973 – 1985. Over a period of 20 years, observations have noted that anaerobes account for up to 90% of the bacteria isolated for intracranial foci. Considering the effects of HBO in enhancing the leukocyte-mediated, host-defense mechanisms, the delivery of hyperoxic blood to the sight of abscess or in cases of concomitant osteomyelitis of the skull is of utmost importance. Under edematous conditions, HBO has a direct relationship to the reduction of edema which in turn reduces intracranial pressure, which could be life-threatening to the patient.

HBO also guarantees sufficient oxygen delivery to potentially hypoxic areas of the brain and its vasoconstrictive effects are additive to those of hyperventilation all aiding in the prevention of secondary brain damage. It is also theorized that HBO produces a reversible opening of the blood-brain-barrier, leading to an improved penetration of antibiotics through non-inflamed meninges.
HBO treatment is a mandatory adjuvant to current standards of treatment for ICA. Along with the appropriate antibiotic regimes, FNAC, drainage, resection, and appropriate neurosurgical management, HBO has been approved for a number of conditions.

1) Anaerobic or mixed pathogens
2) Multiple abscesses
3) Abscess in a deep or dominant location
4) Compromised host
5) In situations where surgery is contraindicated or where the patient is a poor surgical risk
6) No response, or further deterioration, in spite of standard surgical care and antibiotic treatment

The therapeutic effect of HBO on anaerobic and miscellaneous flora is well documented. The earlier the administration of HBO, the better the potential for a successive treatment. A delay in treatment would obscure the potential outcomes and patient benefit is diminished severely. Treatment regimes are recommended at 2.5 ATA 2 times per day and the length of treatment depends on an individual basis per client.
Fungal Disease

Invasive, systemic fungal infections generally only occur in patients with other debilitating conditions like diabetes, severe burns or the immunocompromised. Research has shown that unless the fungus is exposed to pressures of 10 – 10.5 ATM of oxygen, there is little to no direct inhibitory effect on the fungus. These pressures are not attainable by the human body and the effects would be lethally toxic.

HBO treatments at attainable pressures are effective via a secondary pathway. The enhancement of the bactericidal action of the PMNs is important to prevent secondary infection. HBO has also been shown to aid in the efficacy of antifungal medicines like Amphotericin B in the treatment of Candida albicans. Another adjunct of HBO treatment is the increase in fibroblastic collagen production which provides a supportive structural matrix for possible neovascularization which is imperative for subsequent healing in poorly perfused and hypoxic areas of radionecrotic wounds.

Clinically, the majority of HBO treatments are focused on mucormycosis fungal infections. Mucormycosis is a fungal invasion of blood vessel walls with branching, non-septated, hyphal forms of the fungus. Initially, the invasion involves the capillaries and small arterioles, with succeeding spread through the ethmoid and sphenoid sinuses and cribriform plate into the orbit and intracranially. Later, the attack of the larger vessels, such as the internal carotid artery, occurs. The vessel incursion results in a blood flow barrier with a successive distal hypoxia, and inhibition of phagocytosis; factors that enhance further fungal growth. It becomes a vicious cycle of infectious spread.

As the larger vessels obstruct, diminishing blood flow leads to a hypoxic condition which in turn leads to further inhibition of PMN oxidative burst and
phagocytosis eventually leading to the ultimate demise of the patient. With HBO, increased oxygen tension provides potential hypoxic tissues with adequate oxygenation which in turn inhibits fungal growth and aids in the prevention of secondary infections. In combination with surgeons and pharmacologic therapies, multiple HBO treatments are often required for a successful outcome. Studies have shown that without HBO treatment, morbidity has been relatively high, as opposed to patients with HBO treatment which cut the morbidity in half. In general, the treatment of rhinocerebral mucormycosis patients has been supported by HBO intervention as long as it is accompanied by aggressive surgical and pharmacological primary treatments.

Other fungal infections have been experimented on. Aspergillosis, Candidiobolus coronato, coccidioidmycosis, and pseudosallescheria boydii to name a few. Invasive Aspergillosis has been treated with success as a last ditch effort, but ongoing treatment for general infections does not have enough data to support HBO as a mandatory treatment option. As with Aspergillosis, the other fungus species, responded similar in nature, however, there is little data to support extensive HBO therapies. The number one use for HBO under fungal conditions is to aid in the efficacy of Amphotericin B and prevention of secondary infections, considering the immunocompromised is the number target for opportunistic fungal infections.
Radiation Injury

Regardless of where or how an accident involving radiation happens, three types of radiation induced injury can occur: External irradiation, contamination with radioactive materials, and incorporation of radioactive material into body cells, tissues, or organs. External irradiation occurs when all or part of the body is exposed to penetrating radiation from an external source, much like a chest x-ray. Contamination refers to radioactive materials in the form of gases, liquids, or solids released into the environment causing external and/or internal exposure. Incorporation of radioactive material means to uptake the radioactivity into body cells, tissues and target organs like, bone, liver, thyroid, or kidney for example. Incorporation is secondary to contamination based on the chemical properties of the radiation and the target structures it is capable of affecting.

Radiation is used in medicine most commonly for treatment of cancer, severe thyroid disorders and imaging; sometimes in doses in excess of 5,000 cGy (centi-Gray) or 5,000 rads (which is considered to be a potentially fatal dose). Essentially, any tumor can be eradicated if the absorbed dose of radiation is high enough.

Hyperbaric oxygen is used to support capillary angiogenesis and fibroplasia. Radiation injury creates a pattern of injury which does not permit the normal revascularization of tissues and HBO is able to augment the process of healing and reinitiate revascularization. Progressive radiation fibrosis and capillary loss with time have shown to be almost linear and are an unavoidable repercussion of therapeutic radiation. Oxygen at normobaric pressures have been shown ineffective of producing angiogenesis or fibroplasia. However, at pressures under HBO of 2.4 ATA, the dosage of oxygen is now therapeutic while still avoiding toxicity.
There are three phases of HBO healing: the “lag phase,” the “rapid rise phase,” and the “plateau phase.” The lag phase refers to a stage of no measurable change in transcutaneous oxygen. Radiated tissue response begins with macrophage activity, fibroblastic collagen production and endothelial proliferation, which occur during the first 6-8 treatments. The rapid phase is caused by proliferation of lumenized and functional capillaries, which is measured by the transcutaneous monitor as increased capillary density. Eventually, the patient will reach the plateau phase where the maximum effects have been accomplished and revascularization has reached an end point; at this point angiogenesis ceases. It has been shown that the new vessels induced by HBO do not involute after cessation of HBO any faster than vessels involute at a normal rate of aging. HBO also does not induce a supervascularization in nonradiated tissues.

Revascularization of radiated tissue is not a spontaneous process because the diffuse wounding pattern is such that only shallow oxygen gradients are created. The physiochemical response which identifies an injured area as a wound does not develop, and the body, in a sense, does not recognize the radiation injury as a wound. The late radiation effect on cells makes the tissue worse with time, the contamination and worsening of the “Three-H” pattern make the nature tissue oxygen gradients even less (Hypovascular, Hypocellular, and Hypoxic tissues). However, through HBO treatment, an underlying shallow oxygen gradient is magnified into a steep oxygen gradient increasing oxygen supply at the cellular level. The steep oxygen gradients then trigger the body to recognize the radiated tissue as a wound and angiogenesis is initiated through a series of biochemical steps in a normal fashion for wound healing.
In the case of osteoradionecrosis, surgical debridement is required together with
the absolute requirement of adjunctive hyperbaric oxygen. This condition is
characterized by 1) radiation; which produces 2) Hypovascular-hypocellular-hypoxic
tissue; which may undergo 3) tissue breakdown; which is an imbalance where cell death
and collagen lysis exceed the homeostatic mechanisms of cell replacement and collagen
synthesis, which leads to 4) a non-healing wound, where the metabolic demands exceed
the oxygen and vascular supply. There are three stages, stage I is an uncomplicated,
exposed, radiated bone which has failed to heal over six months. Treatment consists of a
mandatory 30 sessions of HBO. Stage II – if the patient does not respond to the 30 HBO
treatment sessions, they are considered stage II and treatment consists of surgical removal
of exposed bone, layered closure of the wound and 10 more treatments of HBO therapy.
Stage III is defined by a wound that dehisces, exposing bone once again or if the patient
initially presents with orocutaneous fistula, a pathologic fracture, or radiographically
evident osteolysis to the inferior mandibular boarder. Stage III treatment consists of a
transoral continuity resection of the involved portion of the mandible and an excision of
any necrotic soft tissue.

The major limiting factor in radiation therapy is the damage caused to
surrounding healthy tissues. The damages sustained from radiation injuries as applied to
the entire body produces swelling, degeneration, and necrosis of the vascular
endothelium. Radioactive conditions leads to the same presentation as obliterative
endarteritis which will progress slowly to a “Three-H” tissue bed. HBO as an adjunct to
traditional therapies has minimized the extent of damage and even induced healing in
hypoxic tissues. HBO has also shown not to produce or accelerate cancer or cause more to emerge from dysplastic mucosa.
Exceptional Blood Loss Anemia

The concept of exceptional blood loss anemia stems from the inability of an individual to receive red-cell replacement for medical or religious reasons and/or when the patient has lost sufficient red-cell mass to compromise respiratory requirements. HBO treatments provide a method of saturation of up to 6% unbound oxygen in the remaining blood for direct use by tissue cells. It also exerts a favorable stimulus on erythropoiesis by increasing circulating erythropoietins – causing an early replacement of the lost erythrocytes.

The major indications for HBO are as follows: 1. Post-Hemorrhage for class IV Hemorrhage, when the patient cannot accept blood replacement for medical or religious reasons. HBO is indicated repetitively for shock, disorientation to coma, ischemic changes of the myocardium as demonstrated on the ECG, and Ischemic gut as demonstrated by a sprue-like diarrhea, 2. Increased O2 Debt.

Routine treatment for blood loss anemia usually consists of replacement of blood volume with crystalloids/plasma expanders for hemorrhage, medical support for the severe anemia: hemetemics, antibiotics, and cardiotonics as needed and hyperbaric oxygen to be administered immediately and repetitively. HBO treatment is provided until the hematocrit and hemoglobin rises to levels which alleviate the preceding symptoms and signs. Hematocrit levels around 22.9% and hemoglobin of 7.7 Gm% are considered appropriate levels. Along with the co-administration of erythropoietin, which has no be rejected by religious groups, HBO treatment is the only current possible treatment in the absence of whole blood to aid in the rectification of blood loss.
In conclusion, considering that there are no satisfactory blood substitutes available, HBO treatments are integrated with minimal risks, barotrauma being the only major HBO risk. HBO offers the nontraditional patient a fighting chance to treat exceptional blood loss anemia without whole blood transfusions. Currently, researchers are under several investigative routs to create “non” blood, blood substitutes to keep up with the potential demands as populations grow.
Traumatic Peripheral Ischemias

Any direct trauma is at risk for severe hypoxia of the tissues involved. Traumatic peripheral ischemias (TPIs) which meet the criteria for HBO intervention are crush injuries, compartment syndromes, burns, frostbite, threatened flaps and threatened replantations. The role of HBO in TPIs is to counteract tissue hypoxia and the consequences of hypoxia that arise secondary to the trauma itself. The ischemias of concern arise from direct injury to blood vessels as a result of the traumatic event, indirect injury, compartment syndrome, where decreased flow in the microcirculation occurs because of fluid leakage, with or without vascular collapse, from the external pressure of the tissue fluid, stasis, vasoconstriction, occlusion, or combinations of all these. The consequence is that insufficient oxygen is available for tissues to meet their metabolic needs.

Edema enters the mix because it increases the diffusion distance from the capillary to the cell also contributing to the hypoxia. Microcirculation ceases because the interstitial fluid pressure will exceed the capillary perfusion pressure within an enclosed space which leads to the collapse of capillary beds. Next is the gradient of injury. The range of tissue that is mildly or minimally injured to the amount of tissue that is irreversibly destroyed. Between these two extremes is a gradient of tissue injury which determines the severity of the overall damage. In general, tissue destruction results from either primary or secondary causes. Primary destruction of tissues is a direct effect of the injury, where secondary effects are damaged caused remote to the actual site of injury. Secondary hypoxia is often a consequence of the body’s attempt to manage the primary
injury or a reflection of the inability of host factors to function sufficiently to heal the wound.

Neutrophils generate oxygen radicals during reperfusion which attach to the post-capillary venule endothelium in the reperfused tissue. Do to an overwhelmed system of injury, the “no-reflow” phenomenon occurs. The oxygen radicals cause vasoconstriction of the precapillary arterioles which stop the flow of blood through the capillaries. In a sense this can be a prelude into another cause of secondary injury which is stasis, which in turn, also leads to a reduction or stoppage of blood flow to the hypoxic and ischemic tissues.

With a severe reduction in blood flow, the breeding grounds for bacteria at the sight of injury is created and bacteria can grow almost without restraint. Antibiotics are render ineffective without blood flow and PMN efficacy is severely diminished. This is where HBO treatment shines. With HBO, sufficient oxygen tensions for fibroblast function, at least 30 mmHg, and the production of the collagen matrix is set in motion in order to begin neovascularization and wound repair. If there is no infection, then fibrous tissues will be able to fill in the injury space.

The key effect of HBO treatment is the effect when stasis of cellular elements restricts red blood cell flow through the microcirculation. Plasma continues to stream through the microcirculation and HBO will allow the delivery of the physically dissolved oxygen to the hypoxic and ischemic tissues. In general, HBO treatment is indicated in any condition where tissue hypoxia is extreme and, in the use of transcutaneous oxygen measurements, levels which exceed 200 mmHg can be achieved.
Reperfusion Injury

Ischemia-reperfusion (I-R) is the process of the generation of oxygen-derived free radicals produced during the reperfusion of traumatically injured tissues. In the attempt to restore perfusion to patients with High-energy trauma, extremity compartment syndromes, amputated body parts, or failing flaps, all patients will experience varying degrees of tissue ischemia. Various tissue types have different thresholds of ischemic and I-R injury tolerances. Hyperbaric oxygen therapy has been shown to increase the tissue thresholds and allow for a greater success rate of healing.

Oxygen-derived free radicals are the central core of the I-R biochemistry. These free-radicals are toxic to all biological substances causing cell death by lipid peroxidation and propagation of more free-radicals. The production of these free-radicals is mediated by xanthine oxidase (XO) and neutrophils which in turn create superoxide and hydroxyl free-radicals which are most often implicated in I-R. The pathway of XO is well studied. In the process of ischemia, anaerobic metabolism depletes ATP stores in favor of purine base formation → leading to the accumulation of hypoxanthine. Excess Ca++ influx from membrane pump failure activates an intracellular protease converting xanthine dehydrogenase to XO. Upon reperfusion, XO then converts hypoxanthine to xanthine and the superoxide radical. As the reactions progress hydrogen peroxide is formed along with a highly reactive hydroxyl radical.

The role of the neutrophil is the liberation of large quantities of extracellular superoxide. Superoxide then dismutates to hydrogen peroxide which then reacts with ferritin producing the OH radical once again. H2O2 also can generate hypochlorous acid (HOCl) via myeloperoxidase enzymes released from azurophilic granules. It is
suggested that neutrophil free-radicals can be more potent and dangerous than XO, specifically in skeletal muscle I-R injuries. Neutrophil endothelial adherence and microarteriolar vasoconstriction are important morphological events leading to the microcirculatory failure associated with I-R injury.

HBO therapy has been employed to aid in the decrease of I-R. Initial experiments on skin flaps showed an improved healing and survival of the flap. Another experiment reduced skeletal muscle edema and necrosis in a hind limb tourniquet ischemia and a hind limb compartment syndrome, both excitement with a rat and a dog. One hypothesis is the vasoconstriction caused by HBO. This might account for the decrease in edema by reducing capillary pressures, however, it is assumed not to be the primary cause for reduction in skeletal muscle edema. HBO did however, have a profound effect on leukocyte adherence to the walls of the microvasculature endothelium. The microvasculature remained clear and patent allowing for a continued flow of oxygenated blood to ischemic tissues. However, the mechanism by which neutrophil adherence is diminished is not yet known.

HBO treatments should be employed immediately after perfusion of injured tissue has been restored. In general, anything greater than 6 hours is indicated for HBO; however, HBO seems to be well tolerated by all patients. Once the patient is out of surgery, often times myringotomy tubes are placed prophylactically because the patient is not able to help in clearing their ears while in the chamber due to incomplete recovery from the general anesthetic, this helps to prevent hemotympanum.

Although there are no well-controlled clinical trials, HBO has improved the lives of many I-R victims. The primary hypothesis being the blockage of neutrophil
endothelial adherence and the blockage of arteriolar vasodilatation. This in turn prevents many of the free-radicals from forming and being able to be transported to the site of injury and causing detrimental ischemic damage to the tissues being reperfused.
Skin Grafts and Flaps

In cases of preoperative or postoperative irradiation or in other cases where there is decreased microcirculation or hypoxia, HBO has been shown to be extremely useful in preservation of tissues. HBO minimizes the need for regrafting or repeat flap procedures by maximizing the viability of the compromised tissues. In uncompromised grafts, HBO has minimal effects and is not indicated. The reduction of blood flow due to arteriovenous shunts in the distal portion of a skin flap is the main cause for flap failure. This results in a reduction of the needed nutrients to the capillary network. The vasoconstrictive properties of HBO might act to close the shunts selectively in non-ischemic areas and allow a greater blood flow to ischemic tissue.

Hypoxic tissues with oxygen tensions frequently below 15 mmHg are considered to be compromised tissues. Minimum oxygen tensions of 30 to 40 mmHg are required for the synthesis of fibroblasts and subsequent development of a collagen matrix for capillary budding in avascular areas. HBO treatment allows the delivery of these minimum levels to the hypoxic tissues to allow fibroblastic activity. Fibroblasts are thought to be facultative anaerobic and is stimulated by both intermittent hypoxia and hyperoxia. The restoration of oxygen tension values to physiologic levels by HBO supports the hypoxic tissue until adequate circulation is reestablished and capillary proliferation can be promoted. Therefore, the concept of closing the arteriovenous shunts, stimulation of fibroblasts, enhancement of collagen synthesis and neovascularity as well as hyperoxygenate the tissues is integral for flap and graft success rates.

HBO and grafts have been extensively studied under animal and human controls. Under animal models, several types of clinical flaps were studied to include, free skin
grafts, pedicle flaps, random flaps, irradiated wounds/flaps, composite grafts as well as axial pattern flaps. It was found out that even though all blood supplies to these types of tissues are all different, the key factor to the flap necrosis was the hypoxia. Several experiments showed the viability of HBO treatments to rectify the hypoxic conditions enough to allow the grafts to properly take.

General treatment recommendations are at a pressure of 2.0 – 2.5 ATA and range from 90 – 120 minutes. Initially, two times per day until the graft becomes stable where the treatment would be reduced to once per day thereafter until healing is complete. Peer review is required after 20 treatments when preparing a recipient site for a flap or graft and following 20 treatments after a flap or graft has been placed into its recipient site.
Thermal Burns

Thermal burns are “a complex and dynamic injury characterized by a central zone of coagulation, surrounded by an area of stasis, and bordered by an area of erythema.” Edema develops locally at the sight of injury and also in distant uninjured tissues. Red cell aggregation, white cell adhesion to venular walls, and platelet thrombo-emboli also occur distant to the site of thermal injury. The failure of surrounding tissue to supply borderline cells with oxygen and nutrients necessary to sustain viability promotes an ongoing tissue compromise and expansion of the thermal injury sight. By reducing edema via HBO treatment, the progression of cellular necrosis and expanding and distal injury is halted or minimized and healing time is reduced with less long standing patient after effects. The primary goal, reduce edema, raise oxygen tissue tension, decrease infection, and promote long term healing with decreased disfigurement of the affected tissue.

The reduction of edema in HBO therapy is theoretically accomplished by several mechanisms. HBO directly imposes its hyperoxic properties on the inhibition of neutrophils to adhere to the distant and local microvasculature. In turn, decreasing endothelial injury in these sights and directly preventing platelet aggregation and thrombo-embolic activity preserving the microvasculature and aiding to maintain oxygen tissue tensions around the wounded areas. By preserving the microvasculature the surrounding tissues are able to deliver needed humoral and cellular elements to injured tissues aiding the immune system by increasing the delivery of immunoglobulins and PMNL. HBO is able to also aid in the preservation of the healing process to include
chemotaxis, phagocytosis, and increased killing ability of infective agents, which remain the leading cause of death in burn patients.

HBO is generally utilized in patients with greater than 20% of total body exposure, partial or full thickness injury, or with involvement of the hands, face, feet or perineum. In general, a twice daily regimen of 90 min at 2 ATA in addition to descent and ascent time. Treatment should be rendered as early as possible, even during emergent resuscitation of the patient. Special attention is given to fluid levels as burn victims may require several liters of fluid per hour. And febrile patients should be monitored closely to prevent oxygen toxicity as they are more susceptible per theoretical data. The larger the burn area, the longer and more aggressive the treatment regime. It is not recommended to transport patients from a treatment facility to an off site hyperbaric facility, this would expose the patient to more harmful potential infective agents while in transport causing a plethora of unneeded extra complications. Considering previous studies in this course, HBO treatments will also aid the surgeon in increased efficacy of flaps and grafts as well as decrease the overall hospitalization time and treatment costs.

Studies have shown that with severely burned patients, the reduction of re-hospitalizations, failed surgical procedures, surgical debridement, and overall hospitalization time can sometimes be cut in half with HBO treatment. This was able to be estimated because of patients who, for one reason or another, elected not to undergo HBO treatment. These patients incurred costs in excess of HBO treated patients as well as extended hospitalizations and a multitude of surgical procedures.

In conclusion, HBO treatments are cost-effective adjuncts to any burn victim. The value of depressed edema development leading to a decrease in remote and local
tissue injury as well as increased post operative healing benefits has been demonstrated to be an overall benefit to the patient as well as the treating physicians. In some cases, patients are also able to decrease some of the extreme plastic reconstructive surgeries because of the prevention of the spread of tissue damage beyond the initial exposed tissues.
Experimental Uses of Hyperbaric Oxygen

HBO treatment potentials have just scratched the surface of research. There are several known advantages and approved treatment regimes for non DCS related medical conditions. Considering the vast arena of medical conditions which have not been looked into, there are several experimental uses of HBO treatments which are currently under way. We will touch on treatment of the brown recluse spider, severe head injury, Hansen’s disease, Myocardial infarction, femoral head necrosis and adhesive or incomplete ileus as it is associated with abdominal surgery.

The brown recluse spider venom consists of approximately 9 different cytotoxic and inflammatory complex peptides. Broken down, the venom is 99.8% composed of a spreading factor like hyaluronidase and only 0.02% actually skin-necrotizing factor. In the non-immunodeficient patient, the severity of the spider bite is not based on being bitten, but the amount of venom injected by the spider and the location of the bite, where fatty areas of the body usually express more severe signs and symptoms. Initially, the bite will appear with just redness and then quickly progress to a dusky, mottled appearance creating concentric rings of erythema and ischemia with a sharply demarcated boarder around the bit site within 12 hours. Over a period of 2 to 3 days, the dusky area will show signs of a halo ring around a central core of necrosis as it sinks below the level of the skin. Within two weeks the necrotic center scab falls off leaving an open ulcer with extensive ulceration which takes another 6 to 8 weeks or longer to heal. And in the case of fatty areas, the ulceration can lead to a systemic infection and even death, especially in the immunocompromised patient.
Besides the reduction of the edema and enhanced tissue oxygenation, HBO treatments are thought to exhibit possible direct inactivating effects on the Loxosceles venom. In unpublished works, 35 patients, none required surgical grafting after the spider bite after receiving between 2 and 18 HBO treatments. HBO shows dramatic effects on the change in color of the dermato-necrotic lesion to normal pink skin color while in the chamber. The diffuse morbilliform rash which accompanies the spider bite is also dramatically reduced. And HBO treatments are a staple in the regime if a flap is required do to delayed HBO treatments of the initial bite within the first 48 hours of exposure.

The treatment of severe head injury and HBO is under investigation as well. Although with mixed results, theoretically, HBO inherently increases the available plasma O2 allowing viable brain cells to maintain glucose metabolism and oxygen consumption and minimizing ischemia within the brain. Raised oxygen tensions leads to vasoconstriction therefore decreasing cerebral blood flow by up to 22% with an end result of a decrease in intracranial pressure. Under controlled studies, HBO treatments had a reduction of mortality as compared to non-HBO treated patients from 17% with HBO and 32% with non-HBO treatment. With patients having a GCS score of 4-6, those with mass lesions and those with increased inter cranial pressure, the mortality reduction was as much as 50%. However, the functional recovery of the surviving patients was not satisfactory and the mechanisms of HBO treatment is not fully understood when considering severe head injuries.

Hansen’s disease is a chronic infection caused by mycobacterium leprae with a spectrum ranging form the paucibacillary forms to multibacillary forms. Dapsone in
conjunction with rifampin, clofazimine and ethionamide have been recommended as the mainstay treatment of Hansen’s disease, however; this is just a control process, not a cure. HBO intervention seems to be related to a combination of factors: a mild alteration in immune response, interference in some fashion with the O2 metabolisme of this obligate intracellular parasite, or perhaps some improvement in leukocyte functionality to fight the organism. The studies supplied are mixed worldwide from complete remission or cures to no success at all. Much research is still required to determine the effects of HBO if any.

With consideration for acute myocardial infarct, HBO treatment has its greatest effects on the decrease of reperfusion injury. It was shown that HBO treatment at 3 ATA decreased myocardial oxygen consumption, reduced excess myocardial lactate production, and narrowed the oxygen difference between the coronary artery and coronary sinus blood leading to a better oxygen delivery to ischemic tissues. When t-PA is added into the mix in order to eliminate a thrombus, the effects of HBO in combination was synergistic in animal studies. There was a dramatic reduction in myocardial injury where only 1% of the heart was maximally injured and only 12% with moderate injury as opposed to a control group with 36% maximal and 24% moderate injury. So, along with the significant benefits of HBO in aiding with reperfusion, the research of general MI treatment with HBO as an adjunct therapy in human models is under way.

The death of bone cells (osteocytes) and a subsequent loss of structural integrity of the femoral head are caused by a loss of the blood supply to the femoral head and is known as femoral head necrosis. Bone viability, healing, and remodeling require oxygen. An increase of oxygen through HBO aids in the enhancement of bone resorption
and remodeling through stimulation of osteoclasts. Primarily, HBO treatment in femoral head necrosis is due to improved oxygen tension in tissue fluids at the site of bone necrosis preventing further loss of ischemic bone cells by preventing the perpetuating effects of edema and ischemia at the site of injury. The edema is decreased by HBO induced vasoconstriction which in turn, permits better perfusion to the site of injury following the same mechanisms as in a compartment syndrome. In initial studies, HBO treatments improve the outcome of patients with femoral head necrosis as opposed to those who follow the natural course of the disease. HBO provided a significant increase in both short-term and long-term success by as much as 97% and 81%. Included in these numbers are a small number of patients who underwent HBO treatment as an adjunct to other orthopedic treatments as well, where the majority of the patients were treated non-surgically. In general, HBO treatments are directed at the underlying Pathophysiology of femoral head necrosis. Facilitation of the reduction of edema and oxygenation of hypoxic tissues and later the enhancement of the host’s remodeling processes through stimulation of bone resorption, revascularization and osteogenesis. It’s greatest benefits may be as an adjunct to orthopedic interventions.

In adhesive or incomplete ileus associated with trauma or surgery, the patient experiences an acute intestinal obstruction. As healing progresses, sometimes air can be trapped within the intestinal lumen and HBO can be used to reduce this volume of air, dissolve the gas into the blood allowing for respiratory expulsion, as well as increase O2 tension within the hypoxic tissues associated with the traumatic event. Often times, the prevention of post surgical eradication of ileus can be attributed to HBO therapy.
In conclusion, HBO treatment regimes are under investigation for several potential medical complications. As medicine expands, HBO treatments will be sure to expand and evolve with it. Either as adjunctive therapies or maybe even mainstay therapies for undiscovered or unrealized conditions, HBO treatments will continue to be explored as possible partial or full management of medical situations.
Controversial Uses of Hyperbaric Oxygen

The concept of a controversial use of HBO therapy is one of an endless list. Controversial uses include, but are far from limited to, autism, ALS, MS, HIV/AIDS, steroid enhancement for sports, “General health and well being”, and countless others. Among controversial uses are also medical conditions which have had little or no research presented as well as experimental control studies, which in some cases might be unethical depending on the type of injury presentation. One source claims to use “negative hyperbaric pressures” by decreasing the pressure on the patient as if they were flying in an un-pressurized airplane. For the scope of this paper, we will touch on MS and ALS as neuromuscular diseases positive therapy has been claimed.

MS is characterized by a triad of inflammation, demyelination and gliosis; the course can be relapsing-remitting or progressive. MS affects approximately 350000 Americans and 1.1 million individuals worldwide. There are many “off color” treatment options available, such as megadose vitamins, calcium orotate, bee stings, cow colostrums, procarin, chelation, acupuncture and HBO treatments. However, it is suggested that patients should be weary of costly and potentially hazardous unproven treatments which lack biologic plausibility. HBO has been experimented with safely and the first reports on the efficacy of HBO in MS appeared in European literature and later the results were confirmed by Dr. Neubauer in 1975 when he administered HBO to a patient with osteomyelitis who also had MS: the MS markedly improved during the course of the treatment. The theory behind treatment of MS states that HBO may affect any number of features of the MS process. It is somewhat immunosuppressive and is effective in inhibiting EAE. It affects local tissue oxygenation and thus might diminish
the effects of local inflammation leading to myelin breakdown or inhibiting myelin repair. In animal studies HBO reduced the edema and blood-brain barrier dysfunction, which are well-recognized features of MS. Although controversial, the study of MS and HBO therapy warrants research and controlled studies.

Another neurological disorder, amyotrophic lateral sclerosis, is the most common form of progressive motor neuron disease. It is a prime example of a neurodegenerative disease and is arguably the most devastating of the neurodegenerative disorders. The pathologic hallmark of motor neuron degenerative disorders is death of lower motor neurons consisting of anterior horn cells in the spinal cord and their brainstem homologues innervating bulbar muscles and upper, or corticospinal, motor neurons originating in layer five of the motor cortex and descending via the pyramidal tract to synapse with lower motor neurons either directly or indirectly via interneurons. HBO treatments have been employed by several free standing clinics which make claims of positive ALS results; however there is no evidence of retardation of the disease via HBO treatments or any other treatment for that matter. Some research actually suggests that HBO treatment is harmful to ALS patients who have progressed into the natural respiratory dysfunction stage of the disease. Dr. Edward A. Oppenheimer, MD, FCCP, a pulmonologist in Los Angeles, CA, postulated that HBO treatment can be dangerous for people with ALS who have impaired respiratory muscle function and is therefore not indicated. When a person has significant respiratory muscle weakness due to neuromuscular disease, the treatment needed is assisted ventilation, not oxygen. Oxygen does not improve airflow into the lungs. Oxygen therapy for people with ALS who have
respiratory muscle impairment can result in further reduction in breathing (decreased respiratory drive) leading to complications including unnecessary deaths.

Hyperbaric oxygen therapy is usually offered as a referral and consultation service by a specially trained physician. Each patient should be carefully evaluated to determine the relative risks and benefits of hyperbaric oxygen therapy. Acceptable indications for hyperbaric oxygen therapy have been determined by Medicare, the Undersea and Hyperbaric Medical Society (UHMS), and the American College of Hyperbaric Medicine (ACHM).
References


