The Role Of Hyperbaric Oxygen Therapy In Emergency Medicine

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Introduction

Hyperbaric oxygen therapy (HBOT) has been alternately called “highly effective”[1] and “a therapy in search of diseases”.[2] Modern use of hyperbaric oxygen in clinical medicine began in 1965 with the work of Churchill-Davidson[3] and Borema.[4] Following its initial successful use in cardiac surgery, carbon monoxide poisoning, and gas gangrene, researchers were eager to treat a variety of other conditions in hyperbaric chambers, often without much scientific rationale. Because of this, hyperbaric oxygen therapy fell into disfavor until the 1970’s when several significant events took place. Medicare convened a panel of members in the Undersea Medical Society (UMS) to help establish guidelines in 1972. The same society convened a workshop in 1975 after which Davis and Hunt edited the first clinical textbook in hyperbaric medicine.[5] An *ad hoc* committee of the UMS was convened in 1976 which led to the formation of the Hyperbaric Oxygen Committee that now publishes a report of accepted medical conditions every two years. Potential indications for HBOT are rigorously screened for data “at least as convincing as that for any other treatment modality for that disorder”.[6]

Studies into the physiologic effects of oxygen under pressure have elucidated much more information over the past twenty years concerning the mechanism of action of HBOT. Research in the effects of HBOT at the cellular level have provided enough data that the majority of rational physicians no longer consider HBOT as magic, voodoo, or merely a waste of time and money. In emergency medicine, there are two main indications for HBOT: Carbon Monoxide, Cyanide or Hydrogen Sulfide Poisoning, and a constellation of symptoms categorized as Decompression Illness.

Carbon Monoxide Poisoning

Carbon monoxide is a leading cause of death by poisoning in the United States.[7,8] Most common sources are automobile exhaust (accidental or purposeful), faulty heaters, and building fires. The pathophysiology of CO poisoning can be found in any textbook of toxicology or emergency medicine and is beyond the scope of this article. A few significant points bear mentioning. Many factors affect the actual clinical presentation, such as the inhaled CO concentration, duration of exposure, rate and depth of breathing, heart rate, co-morbid illnesses and most importantly, the time between discovery of the patient after exposure and arrival at a hyperbaric chamber. Until recently, many textbooks listed severity of symptoms as they related to arterial carboxyhemoglobin levels. Recent data suggest that COHgb levels are merely an indication of exposure, and that arterial pH is a much more sensitive indication of severity of exposure.[9, 10] Hyperbaric oxygen causes CO dissociation from hemoglobin to occur at a rate greater than achievable by breathing pure oxygen at sea level (Table 1).[11-13]

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Table 1. Half-Life Of Carbon Monoxide In Humans

Breathing Media	Half Life
Room Air at sea level	5.3 hours
100% O_2 at sea level	1.3 hours
100% O_2 at 3.0 ATA	23 minutes

There is evidence that CO is directly toxic to brain via a mechanism that is not related to hypoxia.[14] The initial effect of CO on cerebral blood flow (CBF) is to induce a very large increase in flow, rather than to cause a hypotensive hypoperfusion.[15] This increase in CBF is sufficient to maintain oxygen delivery to the brain.[16] The difference between CO exposure and dilutionally hypoxic rabbits was that brain function (measured as a cortical somatosensory response) was greatly inhibited in the former, but preserved in the latter.

Thom[17,18] has demonstrated a marked benefit with HBOT in CO poisoning that stands independent of the rhetoric in clinical literature regarding how to treat CO poisoned patients.[19,20] His studies show that CO causes a cascade of microvascular endothelial injury and that binding and subsequent activation of leukocytes is a central component of brain injury triggered by CO. The specific process by which hyperbaric oxygen inhibits this leukocyte-endothelial adhesion is related to a class of glycoproteins on the leukocyte surface called b_2-integrins. This oxygen related effect is quite discreet. Another interesting finding which supports this mechanism is that animals pre-treated before with HBOT prior to CO exposure received the same benefit as those treated after exposure to CO.

Clinical presentation varies greatly in patients poisoned with CO. The most common physical findings include tachycardia and tachypnea. There may be associated mild hypotension.[21] Headache and nausea or vomiting are common. Be suspicious of an entire family who goes to bed feeling well and all wake up sick at the same time. Inquire if there are pets in the house behaving strangely. The cherry-red color described in textbooks is rarely seen.

COHgb levels above 15% increase the risk for myocardial infarction and a 9% COHgb level lowers the ventricular fibrillation threshold.[7] The reported incidence of delayed neuropsychiatric syndrome (cognitive dysfunction, personality changes, aphasia, apraxia, apathy, disorientation, hallucinations, gait disturbances, mood changes, violence, verbal aggressiveness and impulsiveness) ranges from 3 to 40%.[22-24] Of equal importance and interest is the possibility of long-term cardiac sequelae and myocardial dysfunction as a result of CO poisoning.[25]

Methylene chloride is a component of paint thinner. It is converted to CO by the cytochrome P-450 after it enters the liver. Endogenously created CO derived from methylene chloride has a much longer half life than exogenously inhaled CO, but the symptoms are similar. CO poisoning should be suspected in persons who exhibit toxicity after using paint thinning products in a closed environment. Another increasingly more common source of CO is propane-powered motors such as those found in warehouse fork lifts and industrial floor buffers. Ideally, these propane engines should burn cleanly if properly maintained. However, a poorly maintained propane engine will emit nearly as much CO as an internal combustion (gasoline) engine.

Performing double-blind studies in a hyperbaric chamber is difficult. A sham treatment must include pressurization to avoid patients knowing that they were not under pressure. This requires sophisticated gas mixing systems beyond the capability of most clinical hyperbaric facilities. In addition, most hyperbaric physicians have enough respect for the clinical data available that they consider it unethical not to treat appropriate patients with HBOT. The advantages of HBOT over surface oxygen in treating acute CO poisoning can be seen by comparing clinical reports from Copenhagen and Seattle.[26,27] The results of these studies are summarized in Table 2. Except for the HBOT, treatment of patients in each study was similar.

The decision for the emergency physician is: When should I refer a patient for hyperbaric oxygen therapy? There is no concrete consensus even among hyperbaric physicians. Epidemiologic studies suggest that prognosis is poorer for patients who have underlying cardiovascular disease, are more than 60 years old, or have suffered any interval of unconsciousness due to CO.[21, 27]

We use the following criteria to determine a patient's candidacy for HBOT:

- history of an interval of unconsciousness;
- an objective neurologic deficit or altered mental status;
- ischemic EKG changes or chest pain in a patient exposed to CO;
- pregnant patient with COHgb $\geq$ 15% (fetal hemoglobin binds CO more 'tightly' than maternal hemoglobin);
- recurrent symptoms within three weeks of original treatment;
- patient with COHgb $\geq$ 25-30%;
- symptoms in less severe poisoning that do not resolve after four hours of 100% O_2 via non-rebreather mask in the emergency department.

Table 2. Comparison of Surface Oxygen and HBOT in CO Poisoning

Site	# of patients	Loss of consciousness	Deaths	Neurologic Sequelae
Copenhagen (100%O_2 at sea level)	79	76	23 (30%)	11 (14.5%)
Seattle (HBOT)	115	74	11 (14.9%)	2 (2.7%)

Cyanide Poisoning

Carbon monoxide and cyanide poisoning frequently occur simultaneously in victims of smoke inhalation. [28-33] In combination, these two agents exhibit synergistic toxicity.[34, 35] The difficulty in cyanide detection is that blood levels are more difficult to obtain in the emergency department, and levels are ordered with much less frequency than are COHgb levels. While CO binds reversibly to the ferrous (Fe^{2+}) iron in the cytochrome oxidase a3 system, cyanide irreversibly binds to the ferric (Fe^{3+}) iron at the cytochrome a oxidase level. Therefore, while HBOT is a primary therapy utilized in the dissociation of CO from hemoglobin, it is supportive to the use of a cyanide antidote kit in situations of cyanide poisoning. The rationale for utilizing HBOT in cyanide poisoning is several fold: it mitigates the hypoxia induced by cyanide by supersaturating the plasma with oxygen until the sodium nitrite in the antidote kit converts cyanhemoglobin to methemoglobin and it lessens the hypoxic effect of methemoglobin itself.[36-43] One must be cautious in administering HBOT simultaneously with a cyanide antidote kit because the methemoglobin level may be directly lowered by hyperoxia (at least 4 ATA), possibly reducing the efficacy of antidotal therapy.[44] In reality, 100% oxygen is never administered at pressures greater than 3 ATA because of the significant increase in CNS oxygen toxicity. HBOT is recommended as an adjunct to the treatment of combined CO poisoning complicated by cyanide poisoning.[6]

Hydrogen Sulfide Poisoning

Hydrogen sulfide (H_2S) is a highly toxic, inflammable, colorless gas, readily recognized by its characteristic "rotten eggs" odor. The mechanism of toxicity is similar to that of cyanide and CO poisoning. H_2S is commonly found as a contaminant ambient gas in mining. Nitrites aid the conversion of sulfhemoglobin to methemoglobin. Although not very common, individual case reports have shown that HBOT is successful in treating H_2S poisoning, especially when combined with the cyanide antidote kit.[45]

Treatment

There is no universally accepted treatment protocol for the treatment of CO, cyanide or hydrogen sulfide poisoning with HBOT. Because the $T_{1/2}$ of CO at 3 ATA is 23 minutes, a common treatment protocol is to subject the patient to two or three half-lives of CO at 3 ATA on 100% oxygen with five minute air breaks in between the oxygen-breathing periods. These “air breaks” have been found to lessen the likelihood of oxygen toxicity. Patients are instructed to follow-up within 24 hours for reassessment. Retreatment is based on recurrence of significant symptoms (neurologic deterioration, headaches, confusion, nausea, irritability or personality change) if any. A syndrome of chronic carbon monoxide poisoning has been recognized in which patients are exposed to long-term low levels of CO. These patients are symptomatic with relatively low (normal) levels of COHgb because CO is lipophilic and tissue levels will be elevated. On occasion, repeat ABG’s six hours after initial HBOT will reveal COHgb elevations again without additional exposure, suggesting long term exposure. These patients will require repeated HBOT.

Decompression Illness

Decompression illness (or DCI) is a general term used to describe a broad spectrum of signs and symptoms of inert gas (N_2) problems or dysbaric injuries related to SCUBA diving. *An arterial gas embolism* (AGE) is characterized by gas bubbles in the arterial system generally caused by air passing through the walls of the alveoli into the bloodstream. AGE can result after breathing compressed gas followed by voluntary breath-holding (such as during a rapid ascent); or it can result from a pathologic condition which traps air in the lungs while ascending to the surface. Only a 4-ft ascent during breath holding is sufficient to bring about enough of a pressure increase to cause AGE. Symptoms of AGE are usually immediate in onset and generally involve changes in level of consciousness, paralysis or other cerebral symptoms. *Decompression sickness* (DCS) is a syndrome caused by bubbles of inert gas (N_2) formed in the tissues and blood stream after SCUBA diving. DCS usually results from a deep dive or prolonged exposure to breathing compressed gas at depths greater than 20 feet/6.1 meters. Symptoms may be confined to the musculoskeletal system and consist of joint or muscle pain, or may involve the central nervous system with symptoms of numbness, tingling and other complaints. *Type I DCS* refers to pain involving the joints or muscles, or skin bends, or fatigue without other symptoms. *Type II DCS* includes neurological and cardiorespiratory symptoms. It bears mentioning that breath-holding diving (where no compressed gas is involved) does not expose a diver to DCI risk.

Florida has the dubious honor of having both the highest annual number of cases of DCI (42.9%) and the highest number of fatalities from diving accidents in the United States (21.2%).[46] For this reason, it behooves emergency physicians in Florida to become familiar with the symptoms, signs and treatment of divers with DCI. Divers are generally divided into two groups: those who have very little knowledge of recognition of DCI, and those who think they have a great deal of knowledge (sometimes inappropriate) about the risk profiles and likelihood of DCI. A common fallacy includes not being able to get DCI if diving within the tables (there is a 1-5% inherent risk of DCI when following tables, and many other factors contribute to the likelihood of a DCI incident than merely the dive profile).

Factors that contribute to the likelihood of DCI are listed in Table 3. A good rule of thumb is that a diver who exhibits symptoms after an exposure to compressed air breathing has DCI until proven otherwise. Certainly, based on the medical history of the diver, and other factors, every under-water occurrence may not be DCI (See Tables 4 and 5). The differentiation of Type I, Type II DCS, or AGE is not so important in the emergency department as is the recognition of a DCI that requires recompression therapy.

Table 3. Factors Influencing The Risk Of DCI

- Fatigue
- Drugs/Alcohol
- Caffeine
- Water Temperature
- Visibility
- Exertion
- Physical Conditioning
- Compressed Air Consumption
- Equipment Function
- Diver Experience

Table 4. Diseases That May Mimic DCI

- Seafood toxin ingestion
  - Ciguatera
  - Puffer fish poisoning
  - Paralytic shellfish poisoning
- Guillain-Barré Syndrome
- Porphyria
- Transverse myelitis
- Seizures
- Spinal cord compression
- Cerebral thromboembolic disease
- Migraine
- Pulmonary edema of immersion

DCI cannot occur unless there is sufficient volume of inert gas dissolved in the tissues, so that when the ambient pressure maintaining it in solution is sufficiently reduced, the gas leaves solution and

Table 5. Difference In Presentation Of Type II DCS And AGE

	Type II DCS	AGE
Onset	20 minutes to several hours minutes of surfacing	during ascent or within
Cause	overextension of tables	rapid ascent
Symptoms	transverse or patchy (mimic spinal cord trauma)	symptoms vertical (mimic CVA)
Loss of consciousness	possible	common

forms bubbles. There are both mechanical effects and physiologic effects of bubble formation. Among the mechanical (bubble-related) effects is assumed to be the occlusion of arterioles. Most of the evidence for the presence of *in vivo* gas bubbles in the blood stream following DCI is based on their detection by Doppler flowmeters. Many limitations have been attributed to this technique.[47] However, the overwhelming evidence is that during decompression, gas bubbles are first detected on the venous side of the circulation and that arterial bubbles are rarely observed except in cases of severe DCI.[48-50]

The ability of bubbles to distort tissue and obstruct blood flow would be injurious even if the fluid surrounding the gas phase were inert. The blood, however, is a highly reactive fluid, and the effects of a bubble are amplified by the activation of systems usually quiescent during normal vascular flow. The interface between the gas phase and blood is a physical-chemical discontinuity, and its maintenance is associated with enormous electrochemical forces. These forces cause the denaturation of proteins, accumulation of globules of free fat, and expose active sites on enzymes in the blood that activate coagulation and complement systems. All these mechanisms extend any mechanical blockage of the circulation with progressive clotting and further damage tissue by a reduction of blood flow, the formation of edema, toxic oxygen species (free radicals) and by the attraction of leukocytes to the area. The physiologic role of HBOT in the amelioration of these effects of DCS are similar to the effects shown in the prevention of neutrophil-endothilial adhesions by the inhibition of b_2-integrins in CO poisoning.

Type I DCS is characterized by aching pain in a limb that occurs 20 minutes to several hours after surfacing from a compressed air dive. There are usually no physical signs associated with Type I DCS. Initially the deep, aching pain is characterized as dull and vaguely localized. When the pain is well localized, it is often described as being adjacent to rather than within the joint. In short dives on compressed air, the upper limbs are affected two to three times more often than the lower limbs, with the shoulder being the most common site.[51] The sternoclavicular joint has never been reported to be involved in a DCI. When more than one site is involved, they are not usually symmetrically distributed. Fatigue is a frequent sequel to exposure to pressure, even if the workload is light. It is usually transient, therefore often ignored. Occasionally, the exhaustion is sufficient to provoke comment, and then is often a harbinger or accompaniment of more serious signs of DCS.[52]

DCS Type II may occur alone or in combination with musculoskeletal pain. One series estimated that about 30% of Type II DCS was accompanied by pain.[53] There are three manifestations of Type II DCS. *Pulmonary DCS (chokes)* is relatively rare and generally occurs with rapid emergency ascents. The onset is usually heralded by a sensation of substernal discomfort that commences within minutes after reaching the surface. Pneumothorax, pneumomediastinum or subcutaneous emphysema may be found. It may be accompanied by a cough and deep inspiration may provoke paroxysms of coughing. The breathing pattern becomes shallow and rapid, cyanosis develops, as well as signs of right heart failure. At this stage the patient is in cardiovascular shock and immediate recompression is required as well as fluid resuscitation and pressors. The incidence of *Neurological Type II DCS* is common in sport divers. It is actually more common (62.7%) than the milder DCS I (25.3%) or the more severe AGE (12.0%).[46] The central nervous system is the particular target organ of DCS. Brain dysfunction is often manifested by confusion, drowsiness, fatigue or indifference. The pain and paraplegia of spinal cord decompression occurs earlier and more often attracts the most attention. The anatomy of the spinal cord, which protects it so well from minor trauma, renders it uniquely susceptible to DCS. The mechanical structure of the vertebral column shields the cord from most injury. The redundant collateral arterial supply assures that the cord will be nourished directly from the aorta even when less favored tissues are allowed to become ischemic. The venous drainage of the cord, slowed and made pendular by respiratory pressure changes, makes it uniquely vulnerable to venous infarction. This vulnerability causes a unique spinal cord DCS different from any other neurological syndrome. Spinal DCS presents a clinical picture of diffuse multilevel cord disease. *Vestibular DCS* is relatively common and presents as dizziness, nausea, nystagmus vomiting and, occasionally, hearing loss and tinnitus. The incidence of these symptoms varies from 13 to 72 percent of Type II DCS. Differentiating this syndrome from otic barotrauma is important. The symptoms of vestibular DCS occur during ascent, those of otic barotrauma occur during descent.

Rapid ascent (without breath holding) may result in inert gas coming out of solution (plasma) in a rapid fashion and traverse the arterial supply into the cerebral circulation causing an *arterial gas embolism* (AGE). A sudden change in sensorium (during the ascent or shortly after surfacing) is the most common symptom and ranges from disorientation to coma. Focal neurological deficits such as hemiplegia or monoplegia may occur depending on the location of the lesions. Liebermaster's sign (presence of pallor or mottling of the tongue) may be present.

Skin bends is a benign condition which commences with itching, which may be intense. The pruritic areas, usually limited to the trunk, are first reddened by vasodilation in the dermis, then vascular stasis results in a characteristic mottling of the skin. Although recompression will promptly relieve the itching, it will resolve over a period of days if left alone. Treatment is more for comfort than to avoid possible complications. The main risk of non-treatment is the possibility of missing more severe symptoms of DCI that would require HBOT.

There are other "dysbaric injuries" that, although they occur while under pressure, do not require HBOT. These include pneumomediastinum, barosinusitis or barotitis and caloric-induced vertigo (which must be differentiated from vestibular DCS).

Treatment

The emergency department treatment in most cases of DCS or AGE is fairly straightforward. One hundred percent oxygen should be administered by mask or endotracheal tube. The reasoning is that one is not only trying to deliver oxygen to relatively ischemic, hypoxic tissues, but also trying to eliminate as much inert gas as possible. Patients with DCI can become severely dehydrated. Immersion in water that is colder than body temperature inhibits antidiuretic hormone and results in a diuresis that can often be significant. Intravenous fluid hydration is preferred even if the patient is totally conscious because providing enough oral hydration at the same time as maintaining adequate oxygenation is difficult. Patients may need up to 8 liters of IV fluids before urine output is obtained. It warrants mentioning that the symptoms of DCI may resolve on supplemental oxygen during transport or during emergency department treatment. This does not eliminate the need for hyperbaric therapy. There is almost always a recurrence of symptoms, and the delay to HBO increases the likelihood of residual symptoms after treatment. Diagnosis of DCI is made by history and physical examination (Table 6). No laboratory findings will assist in the diagnosis. Chest x-rays are helpful to diagnose a pneumothorax that will need decompression prior to HBOT. Waiting for laboratory results, CT scans, or other diagnostic studies will serve to delay HBOT without adding any additional value to the diagnosis.

Despite the basic requirements of oxygen and IV fluids being considered elementary and essential in the treatment of DCI, statistics show that only 33% of DCI cases receive oxygen before transport to HBOT, and only 25% receive IV fluids.[46] In cases of neurologic involvement, IV corticosteroids have been suggested.[54]

Folklore states that a Trendelenberg, left lateral decubitus position should be utilized for patients with AGE. The theory for lateral decubitus is to trap any bubbles in the right atrium thereby lessening circulating air, and to protect the airway in case of vomiting. This may have some merit. The Trendelenberg rationale takes advantage of gravity and bubble buoyancy to minimize cerebral embolization. Buoyancy of bubbles has no effect on arterial distribution[55], and prolonged head-down position can potentiate cerebral edema in the injured brain.[56] For these reasons, Trendelenberg is not recommended. If the nearest hyperbaric facility is too far for surface transportation, air evacuation must be used. It is important that the patient not be exposed to decreased barometric pressure at altitude, so helicopter transportation is frequently utilized, and flight altitude must be kept to no greater than 800-1000 feet above ground level (AGL).

Table 6. History And Physical Examination Of A Diver Suspected Of Having A Decompression Injury

HISTORY

a) Dive profile:
    depth of last dive
    bottom time
    time from surfacing to onset of symptoms
    lengths, bottom times, surface intervals of previous dives on same day
    diving done and profiles for past 7 days
    water conditions (temperature, currents, visibility)
    amount of exertion done while diving/type of activity (fishing, recreation, construction, etc.)
b) drugs/EtOH used
c) past history of DCS
d) type of equipment used
e) past medical history, medications, allergies
f) symptoms in order of onset and severity
g) what symptoms have resolved/lessened/worsened since surfacing
h) time from symptoms onset to arrival in health care facility

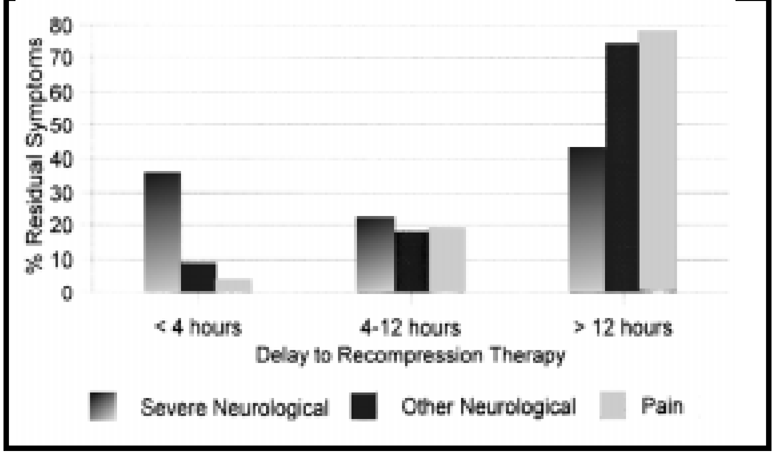
PHYSICAL EXAMINATION

a) Scalp: signs of trauma
b) Eyes: pupillary size, reaction to light, nystagmus or dysconjugate gaze, fundi normal or pale
c) Ears: TM's clear, no hemorrhage, hemotympanum, perforation, vertigo, hearing loss, tinnitus
d) Nose & throat: epistaxis, congestion
e) Neck: JVD, trachea midline, no stridor or adventitious sounds
f) Chest: trauma? Lungs clear, = breath sounds, Heart regular, no Hammond's crunch
g) Abdomen: BS normal, no tenderness
h) Rectal: sphincter tone
I) Extremities: pulses, DTR's, sensation, motor strength, pain in joints (worse with movement?)
j) *Neuro: Cranial Nerves, dermatomes, level of consciousness, bladder/bowel control, Romberg, heel-toe, rapid alternating movements
k) Skin: ecchymosis, mottling, petechiae, erythema

** A detailed neurological exam is critical. It is the only tool which can differentiate Type I from Type II and measure success of treatment!*

There are several standard treatment tables utilized in the treatment of DCI. The most common are the United States Navy Treatment Tables. The table chosen depends on the severity of symptoms, and the length of treatment also depends on the rapidity with which symptoms resolve

Figure 1. Percentage Of Divers With Post-Compression Residual Symptoms Related To Delay To Recompression Therapy



under pressure. If there are residual symptoms after the initial recompression treatment, the patient is treated daily until symptoms are totally resolved, or there are three consecutive treatments without objective improvement. Likelihood of total resolution is multifactorial, but depends on symptom severity and length of time from symptom onset until commencement of recompression therapy. Despite appropriate recompression therapy, many divers will exhibit residual symptoms. Prompt treatment with HBOT is directly correlated with success of treatment as measured by its ability to reduce or totally resolve symptoms (see Figure 1). Approximately 18% of divers treated for DCI will have some permanent sequelae after treatment is completed. Divers who are treated with HBOT require observation for the following 24 hours to watch for recurrence of symptoms. They can be discharged from the hyperbaric department after 30 minutes if there is a reliable adult at home to observe them. We recommend that they remain within 30 minutes travel distance of the chamber in case there is a need for repeat treatment. Patients are to return for follow-up and repeat examination 24 hours after their initial treatment. Recommendations for returning to work or flying after diving depend on the dive profile, the degree of resolution of symptoms after treatment, and the treatment table used. The Divers Alert Network maintains a 24-hour emergency hotline to assist medical personnel in diagnosis and treatment options and referral to the nearest hyperbaric facility. Their number is 919-684-8111.

Side-Effects

Side-effects during HBOT are rare. The most common is barotitis, which occurs more frequently in patients being treated for conditions other than those related to diving. Barosinusitis may also occur. These side effects are easily treated with decongestants and analgesics. Oxygen toxicity seizures are the most serious consequences of HBOT. There are many factors which influence a patient's individual susceptibility to these seizures such as fever, medications which may lower seizure threshold (NSAID's, phenothiazines, high doses of IV penicillin), existing seizure disorder and pressure at which the HBOT is being administered. The incidence is 1.3:10,000 treatments. These seizures are easily treated by removing the oxygen mask and allowing the patient to breathe ambient chamber air. Pressure in the chamber should not be decreased while the patient is actively seizing in order to avoid pulmonary overpressurization which could cause pneumothorax or air embolism. Oxygen toxicity seizures to not predispose a patient to a seizure disorder.

Current research into other uses of HBOT include a multi-center study on HBOT and thrombolytics in the treatment of acute myocardial infarction (HOT Study), and another on the utility of HBOT in early stroke, traumatic brain injury, and spinal cord trauma.

There are approximately 28 hyperbaric chambers in Florida. Baptist Medical Center maintains the only treatment facility in Northeast Florida, with other nearby chambers being Tallahassee, Gainesville and Orlando.

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