Decompression Illness in Sports Divers: Part II
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Abstract and Introduction

Abstract

Decompression sickness (DCS) results from gas coming out of solution in the bodily fluids and tissues when a diver ascends too quickly. This occurs because decreasing pressure lowers the solubility of gas in liquid. Also, the expansion of gas in the lungs may lead to alveolar rupture, also known as "Pulmonary Overinflation Syndrome," which may, in turn, result in arterial gas embolism (AGE). DCS, AGE, and all of their presentations are grouped together under the heading "decompression illness". Joint pain is the most common complaint in DCS, especially in the elbow, shoulder, hip and knee. Blockage of vessels results in ischemia and infarction of tissues beyond the obstruction, and inflammatory changes can lead to extravasation into the tissues, resulting in edema and further compromising the circulation. Involved skin displays a mottled appearance known as "cutis marmorata." In the lymphatic system, bubbles may result in regional lymphedema. More severe cases may involve the brain, the spinal cord, or the cardiopulmonary system. Neurologic manifestations may include sensory deficits, hemiplegia, paraplegia, paresthesias, and peripheral neuropathies. Possible cardiopulmonary effects include massive pulmonary gas emboli or myocardial infarction. Decompression sickness is treated with recompression in a chamber to 60 FSW or deeper, associated with hyperbaric oxygen breathing. In the US, this therapy is usually guided by a Navy Treatment Table. These tables are very effective, especially when recompression is begun promptly.

Introduction

On the earth's surface, the human body is exposed to an ambient pressure which is the result of the combined partial pressures of all the gases in the earth's atmosphere. At sea level, the force of this pressure is described as 1 atmosphere absolute (ATA). As a diver descends, exposure to increasing pressure forces more gas to dissolve in the bodily fluids and tissues, as described by natural gas laws. Upon ascent through the water column, the solubility decreases again. Rapid ascent may lead to bubble formation and decompression sickness (DCS) or alveolar rupture ("Pulmonary Overinflation Syndrome" [POIS]), with resultant bubbles in the arterial circulation (arterial gas embolism [AGE]).[1]

DCS, AGE, and all of their presentations are grouped together under the heading "decompression illness" (DCI).[2] Treatment consists of recompression in a chamber using air or a combination of helium-oxygen. Bubbles may form in blood vessels, where they may cause ischemia and infarct, and in tissues, where they
may initiate an inflammatory response. Inflammatory changes can lead to extravasation into the tissues, further compromising the circulation and resulting in edema.

Hyperbaric exposures (situations where there are elevated pressures) can occur during construction and tunneling projects, in hyperbaric oxygen treatment facilities and in aviation. (The airman is subject to the same problem as divers, except that the situation is reversed--bubbles form on ascent, due to a decrease in pressure and supersaturation. Returning to the ground increases pressure and is analogous to recompression. However, DCS symptoms may occur after returning to the ground and sometimes require additional recompression.)

Recreational scuba diving is the most common type of hyperbaric exposure, especially since the explosive growth of sports scuba (self-contained underwater breathing apparatus) diving in the past decade. Hyperbaric oxygen (HBO) treatment is gaining popularity as the definitive therapy for a growing number of disorders, including decompression sickness, AGE, CO poisoning, clostridial infections, crush injuries, diabetic leg ulcers, skin graft failures, refractory osteomyelitis, thermal burns, necrotizing soft tissue infections, and osteoradionecrosis.

It is incumbent on physicians to be fully conversant with the diagnosis and treatment of decompression illness, especially because the hyperbaric chamber is now widely recognized as effective in reversing the sometimes-deadly changes that take place with DCI.

Medical Management of DCS and AGE

**Early response at the dive site.** As with other emergency life support situations, the ABCs come first: maintain an airway, assure ventilation and accomplish circulation. The standard left decubitus head down position should be avoided because it may promote cerebral edema; the patient should be placed in a supine position. Other measures include:

- Provide 100% oxygen through a tight-fitting mask. This helps to off-gas inert gases. Resuscitation equipment should be available on all dive boats and in all dive facilities. Divers should refuse to dive if this equipment is not readily available.
- Give copious fluids as needed to maintain good urinary output. Fluids should be administered at a rate greater than 0.5ml/kg/hr--usually 1 L qhr or 1 L q4hr, titrated against the hematocrit, which should be maintained at less than 50%. The hemoconcentration associated with decompression sickness is the result of increased vascular permeability mediated by endothelial damage and kinin release.[3] The fluids can be given orally if the diver is conscious--if not, give fluids by intravenous, if available. Avoid using hypotonic fluids, such as D5W, using 0.9% saline instead. Insert a urinary catheter if there is spinal cord DCS.
- Give steroids if there is neurological DCS; dexamethasone 10 to 20 mg IV initially, followed by 4 mg every 6 hours; diazepam (5 to 10 mg) controls the dizziness, instability and visual disturbances associated with labyrinthine (vestibular) damage to the inner ear.
- Seizure activity is treated with a loading dose of Dilantin. Seizures result from damage incurred...
from cerebral bubbles formed from DCS or air embolism (resulting from pulmonary barotrauma); they can also result from oxygen toxicity associated with the treatment schedule. *Dilantin* (phenytoin) is given IV at 50 mg/min for 10 minutes for the first 500 mg and then 100 mg every 30 minutes thereafter. Blood levels of *Dilantin* should be monitored to maintain a therapeutic concentration of 10 to 20 mcg/mL. Levels over 25 mcg/mL are toxic.[4]

Some people provide aspirin, 600 mg, for its anti-platelet effects; this modality is debatable because of the possibility of associated spinal cord hemorrhage. Lidocaine has been shown to be protective in animal models but has not been studied adequately in humans.[5]

Attempting to treat the diver by returning him/her to the water, (known as in-water recompression), is hazardous not only to the diver, but to the caregivers who have to be re-subjected to pressure. This should not be attempted unless special arrangements have been made to do so. For example, in Australia, because of the great distances and time lags involved in reaching a recompression chamber, dive operators have a system of surplus air and oxygen tanks ready for in-water recompression.

**Transportation.** Ascending to an altitude greater than 1000 feet should be avoided. Sea level aircraft that are acceptable for transportation include the military C9, the Cessna Citation and the Lear Jet. Commercial aircraft fly at 5000 to 8000 feet cabin pressure. The "ABCs" initiated at the dive site should be continued while in transport.

**Treatment in the chamber.**[6] The treatment of choice for decompression illness, whether DCS or AGE, is recompression in a multiplace, hands-on chamber. It should have the capability of locking personnel and equipment in or out with trained attendants available for critical care monitoring.

**Multiplace chambers.** These units (Fig. 1) can accommodate between 2 to 18 patients, depending upon configuration and size. They incorporate a minimum pressure capability of 6 atmospheres absolute. Patients are accompanied by hyperbaric staff members, who may enter and exit the chamber during therapy via an adjacent access lock or compartment. The multiplace chamber is compressed on air and patients are provided with oxygen via an individualized internal delivery system. A dedicated compressor package and high volume receivers provide the chamber's air supply.

Advantages include constant patient attendance and evaluation (particularly useful in treating evolving diseases such as decompression sickness), and multiple patients treated per session; disadvantages include high capitalization and staffing costs, large space requirements and risk of decompression sickness in the attending staff.
Figure 1. Multiplace chambers accommodate between 2-18 patients, depending upon configuration and size. They have a pressure capability of 6 atmospheres absolute. Reprinted from Hyperbaric Medicine, Brooks Airforce Base.

Duoplace chambers include the Reneau (Proteus) and the Sigma II with pressurization capabilities to 6 ATA and 3 ATA respectively. The chambers are compressed with air, and the patient breathes oxygen by an individualized internal delivery system. Advantages include constant patient attendance, with access limited to the head and neck; disadvantages include relatively high capitalization cost for single patient treatments and risk of decompression sickness in the attending staff.

The multiplace chamber is not always possible, however, and the monoplace chamber is sometimes the only alternative. Hart and coworkers,[7] as well as Kindwall and colleagues[8] have developed protocols with the monoplace chamber, utilizing Navy Treatment Table 6 (Table I) which can be used with air breaks.

**Monoplace chambers.** These units (Fig. 2), first introduced in the 1960s are designed for single occupancy. They are constructed of acrylic, have a pressure capability of 3 atmospheres absolute and are compressed with 100% oxygen. Recent technical innovations have allowed critically-ill patients to undergo therapy in the monoplace chamber. The high flow oxygen requirement is supplied via the hospital's existing liquid oxygen system.
Advantages of this chamber include that it provides the most cost efficient delivery of hyperbaric oxygen (capitalization and operating costs), and that it presents essentially no risk of decompression sickness to the attending staff. Disadvantages include relative patient isolation and increased fire hazard.

Treatment goals in all instances are to reduce bubble size and surface area while providing hyperbaric oxygenation (HBO). HBO reduces edema, blocks WBC adherence, protects and preserves the microcirculation, corrects hypoxia (100% oxygen under pressure produces 7 volume % in the plasma), blocks reperfusion injury, and facilitates the removal of dissolved gas from the lungs through perfusion.\[9\]

**Outcome.** The most recent DAN (Divers Alert Network) report (1994 data) suggests that complete resolution of symptoms occurred in only 56% of cases while 28% of divers had neurologic sequelae and 17% continued to experience pain.\[10\] Travel after treatment of DCS should be delayed for at least 48 hours; 72 hours for arterial gas embolism. Recurrence of symptoms has occurred with flying more than one week after the initial event. Diving should not be resumed if there is any residual neurological damage.

**Summary**

Decompression illness is the combination of decompression sickness and arterial gas embolism; DCS is a disorder resulting from the reduction of ambient pressure with the formation of bubbles from supersaturated dissolved gas in the blood and tissues, usually associated with pain and/or neurologic manifestations; AGE is the result of air being forced through ruptured alveoli caused by ascent with a closed glottis and results in air bubbles blocking arteries in the brain and heart. Both entities are treated by recompression with oxygen in a chamber. Before initiating a dive vacation to a remote location, it would be wise to check on the availability of a recompression chamber and surface oxygen. Many dive sites pay
little attention to pre-dive planning[11] and evacuation can often be prolonged, resulting in permanent damage.

**Tables**

**Table I. US Navy Treatment Table 6: Oxygen treatment of Type II Decompression Sickness**

<table>
<thead>
<tr>
<th>Depth (feet)</th>
<th>Time (minutes)</th>
<th>Breathing Media</th>
<th>Total Elapsed Time (hr:min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>60†</td>
<td>20</td>
<td>O2§</td>
<td>0:20‡</td>
</tr>
<tr>
<td>60</td>
<td>5</td>
<td>Air</td>
<td>0:25</td>
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<td>60</td>
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<td>O2</td>
<td>1:10</td>
</tr>
<tr>
<td>60</td>
<td>5</td>
<td>Air</td>
<td>1:15</td>
</tr>
<tr>
<td>60 to 30</td>
<td>30</td>
<td>O2</td>
<td>1:45</td>
</tr>
<tr>
<td>30</td>
<td>15</td>
<td>Air</td>
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<td>O2</td>
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<tr>
<td>30 to 0</td>
<td>30</td>
<td>O2</td>
<td>4:45</td>
</tr>
</tbody>
</table>

* Treatment of Type II or Type I decompression sickness when symptoms are not relieved within 10 minutes at 60 feet.

† Descent rate--25 ft/min. Ascent rate--1 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.

‡ Time at 60 feet begins on arrival at 60 feet.
§ If oxygen must be interrupted because of adverse reaction, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption.

¥ Caregiver breathes air throughout unless he has had a hyperbaric exposure within the past 12 hours, in which case he breathes oxygen at 30 feet.

¶ Extensions to Table 6: Table 6 can be lengthened up to 2 additional 25 minute oxygen breathing periods at 60 feet (20 minutes on oxygen and 5 minutes on air) or up to 2 additional 75 minute oxygen breathing periods at 30 feet (15 minutes on air and 60 minutes on oxygen) or both. If Table 6 is extended only once at either 60 or 30 feet, the tender breathes oxygen during the ascent from 30 feet to the surface. If more than one extension is done, the caregiver begins oxygen breathing for the last hour at 30 feet during ascent to the surface.

Adapted from the US Navy Diving Manual.

References

Suggested Readings


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