Bubble Related Diseases

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Decompression Sickness

Decompression sickness (DCS) is defined as an illness that follows a reduction in environmental pressures sufficient to cause formation of bubbles from gases dissolved in body tissues. Decompression sickness is a true occupational disease first described in relation to syndromes which developed in caisson or tunnel workers working in closed, pressurized spaces during construction of tunnels. DCS was first described as the "bends" because of the development of lower extremity or abdominal pain causing the patient to bend over. It was also designated the "chokes" when associated with dyspnea and a choking sensation, the "staggers" when accompanied by vertigo related to inner ear disruption, and the "niggles" or “creeps”, which refers to unusual skin sensations. Decompression sickness in naval operations is related to high altitude or underwater activities using compressed gas mixtures. Aviation decompression sickness can occur during low pressure chamber (altitude chamber) activities, flight in depressurized or unpressurized aircraft, and in high altitude high opening (standoff) parachute operations. Altitude decompression sickness is induced by exposure to ambient pressures less than sea level. It is related to altitudes usually above 18,000 feet. Aviators are protected from decompression sickness by maintaining cabin altitude via pressurization and by denitrogention by prebreathing oxygen to reduce body nitrogen stores. Prior to flight, aviation personnel can reduce their tissue nitrogen by breathing 100 percent oxygen. Currently the highest rate of altitude-related decompression sickness in naval aviation operations involves low pressure chamber activities.

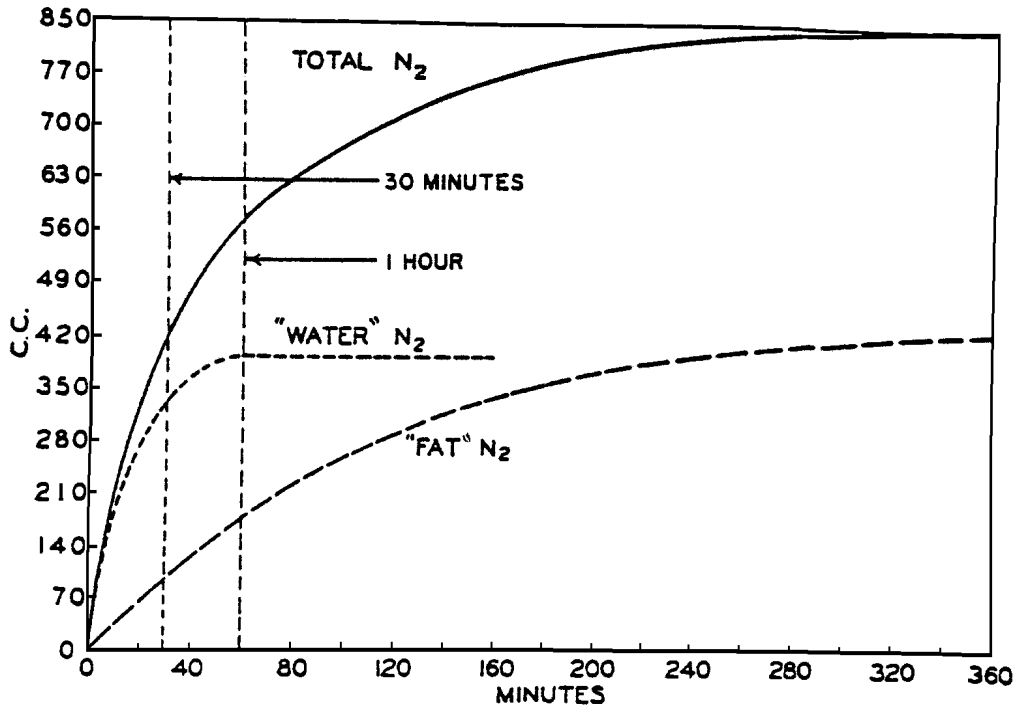
DCS related to diving is much more common than in aviation, and symptoms tend to be more severe. The reason is simply that relatively small excursions in depth produce very large excursions in pressures. Underwater Construction Teams are Seabee units who are at risk for such illnesses, and knowledge of diagnosis and treatment of these conditions is necessary. The Diving Medical Officer (DMO) should always be consulted in cases of DCS for advice on transport to the nearest treatment capable facility and for final disposition of the case.

Bubble Formation Theory

Decompression sickness results from bubbles formed as dissolved gases come out of solution in tissues due to a drop in ambient pressure. The principal gas involved is nitrogen, and to a lesser extent, carbon dioxide.

As nitrogen in air is inhaled, it dissolves in the body and reaches equilibrium with the liquid phase (tissue and blood). The concentration of nitrogen dissolved is proportional to the partial pressure of nitrogen in the inhaled gas (Henry's Law). As one descends below the surface, these partial pressures increase with depth. As one ascends from depth or climbs in altitude, the partial pressures of the gases in the breathing mixture decrease. If the nitrogen partial pressure in the breathing gas is reduced or eliminated, a gradient is established across the alveoli. Nitrogen is

off-gassed from the various tissue compartments, and may require 12 hours or more to reach equilibrium. The rate of inert gas uptake and elimination depends on : (1) gas concentration gradient between blood and tissue, (2) tissue blood flow, and (3) the ratio of blood and tissue gas solubilities. For example, nitrogen is five times more soluble in fat than in water. Gas uptake and elimination are expressed as tissue half times.



The rate at which nirogen is eliminated from the body at sea level when pure oxygen is breathed (Clamann, 1961)

The formation of bubbles is influenced by: (1) supersaturation of tissues with gaseous nitrogen, and (2) the presence of gas micronuclei. Supersaturation results when tissue inert gas tension (PN_2) exceeds ambient barometric pressure (PB). Critical supersaturation occurs when inert gas comes out of solution and forms bubbles. Early research suggested that once a critical supersaturation (constant allowable) ratio was attained, bubbles would form. Current theory suggests that supersaturation is related to a variable allowable ratio. This is influenced by time, pressure differential, and tissue nitrogen half time. Gas micronuclei may form in areas of negative hydrostatic pressure, such as in turbulent blood flow or areas of shearing action in joints. Gas micronuclei may arise de novo and are called autochthonous bubbles. Bubbles may form in blood, lymphatics, or tissue. Inert gas tension is higher in capillary or venous blood than in arterial blood.

To successfully tolerate an ambient pressure reduction, a time-pressure profile must be selected which does not allow this critical ratio to be exceeded. Tabulated safe time-pressure profiles are

called decompression tables.

Bubbles have two pathophysiological effects. First, the direct mechanical effects of the bubbles may result in vessel obstruction or tissue distortion, causing pain, ischemia, infarction, or dysfunction. The second effect, tissue-bubble interface surface activity, results in denaturation of proteins and aggregation of platelets, causing endothelial damage and the release of pain mediating substances. Because the bubbles may form in different places in the body, they may give rise to multifocal lesions which do not necessarily follow dermatomal or anatomical distributions.
Once the bubbles are formed, they tend to expand as dissolved gases and continue to come out of solution. Carbon dioxide, a highly diffusable gas, contributes to bubble enlargement, especially if formed in excess by vigorous exercise. For this reason, DCS patients should be kept at rest. Decompression sickness is a progressive systemic disease. Although the initial manifestation of DCS may be of a relatively trivial nature, further expansion or formation of bubbles elsewhere may result in a life threatening situation if treatment is not initiated promptly. The various clinical syndromes may occur in any combination.

Clinical Syndromes of Decompression Sickness
Decompression sickness is classified as either Type I or Type II. This clinical classification is useful because it helps establish treatment, prognosis, and medical disposition.

Type I Decompression Sickness
Type I decompression includes: (1) limb pain (musculoskeletal symptoms), (2) skin bends (cutaneous symptoms), and (3) lymphatic bends (lymph node swelling and pain).

1. Limb Pain. The most common presenting symptom of DCS is pain, accounting for 60 to 70 percent of Altitude DCS, and 80 to 90 percent of dive related DCS. Joint pain is by far the most common type. But other types of pain may occur. The shoulder is the most common site of joint pain. The elbow, wrist, hand, hip, knee and ankle may also be involved. Upper extremity pain is more common than lower extremity pain (lower extremity pain is ususally seen in saturation divers). The characteristic pain of Type I DCS usually begins gradually. Called the "niggles" by divers, it is slight when first noticed, and may be difficult to localize. It may be located in a joint or may be only a muscle ache. The pain tends to increase in intensity over time and is usually described as a deep, dull ache. The limb may be held preferentially in certain positions to reduce the pain intensity (guarding). The hallmark of Type I pain is its dull, aching quality and its confinement to particular areas. It is present at rest and may or may not be made worse with movement. The pain may be relieved by an inflated blood pressure cuff over the site. The most difficult differentiation is that Type I DCS and pain resulting from a muscle sprain or bruise.
A sharp, knife-like pain that shoots down an extremity or encircles the body trunk (radicular or dermatomal pain), thoracic or abdominal pain, tingling or burning pain (paresthesias), or pain that moves from one area to another or arises from the nervous system is treated as Type II DCS (see

below). If there is any doubt as to the cause of the pain, assume that the diver or aviator is suffering from DCS and treat him accordingly. Frequently, pain may mask other more significant symptoms and a thorough neurological exam is indicated. Pain should not be treated with analgesic medication. Bilateral pain, truncal pain, or hip pain is treated as Type II DCS (see below).

2. Cutaneous (Skin) Bends. The most common cutaneous manifestation of DCS is itching. Itching (pruritus) or crawling sensation (formication), usually occurs in dry hyperbaric (chamber) dives and does not require recompression. Mottling or marbling of the skin, known as Cutis Marmorata, is caused by venous obstruction by intravascular bubbles, and precedes the more serious forms of DCS. It usually starts as intense itching, progresses to redness, then to patches or linear areas of dark purple-blue discoloration of the skin. The skin may feel thickened and the rash may be raised. Visible skin bends (marbling) should be treated with recompression.

3. Lymphatic Bends. Lymphatic obstruction by bubbles may cause localized pain in the lymph nodes and swelling of the area. Recompression will usually provide prompt relief of pain. However, the swelling may take somewhat longer to resolve completely and may still be present at the completion of treatment. Lymphatic bends are rare.

Type II Decompression Sickness

Type II DCS is the most severe form of DCS. Patients may present with neurological, cardiorespiratory, or inner ear symptoms, pain, or shock. Type I symptoms may be present at the same time. Thirty to 40 percent of Type II Altitude DCS cases have associated limb pain. In Altitude DCS cases 85 to 90 percent are Type I, and 10-15 percent will be Type II DCS.
In the early stages, the symptoms of Type II DCS may not be obvious; and the patient may consider them inconsequential. The patient may feel fatigued or weak and attribute this to overwork. Even as the weakness becomes more severe, the individual may not seek treatment until walking, hearing, or urinating becomes difficult. For this reason, symptoms must be looked for during the postdive or postflight period and treated before they evolve further. Fifty percent of DCS cases present within 30 minutes, 85 percent by one hour, and only one percent after six hours.
Many of the symptoms of Type II DCS are the same as those of arterial gas embolism (AGE), although AGE usually presents within 10 minutes. The treatment for arterial gas embolism is also an appropriate treatment for DCS.

1. Neurological Symptoms. These symptoms may be the result of involvement at any level of the nervous system. Peripheral nervous system involvement may present with patchy peripheral paresthesias (burning or tingling) or numbness or weakness (usually mild and confined to one extremity). Spinal cord DCS may present with numbness, weakness, paralysis, or urinary dysfunction. Spinal cord DCS is more commonly the result of diving activities and accounts for less than 10 percent of Type II Altitude DCS cases.

Brain DCS is the most common form of Type II Altitude DCS. Disturbances of higher cortical function may result in personality changes, confusion, or inappropriate behavior. Hemiplegia, hemisensory loss, incoordination, or tremor may occur. Symptoms of classic migraine, with unilateral headache and scotoma, may be a presentation of Type II DCS. Headache and visual disturbances occur in 30 to 40 percent of Type II Altitude DCS. Brain DCS signs may be subtle and may be overlooked or passed off as being inconsequential. Loss of consciousness, which may be due to neurological or cardiorespiratory collapse, is a sign of fulminant DCS or AGE.
Inner ear DCS may result in vertigo, dizziness, tinnitus, and hearing loss. It may be difficult to distinguish from a round or oval window rupture. Inner ear DCS usually occurs in deep helium-oxygen dives of long duration.

Pain that is bilateral or involves the trunk or hip is considered Type II DCS. Divers who develop pain while under pressure should be treated for Type II DCS.

The occurrence of any neurological symptom following a dive or flight should be considered a symptom of Type II DCS or arterial gas embolism. Fatigue is not uncommon after long dives or flights. Fatigue that is unusually severe may be a sign of CNS involvement.

2. Cardiopulmonary Symptoms. If profuse intravascular bubbling occurs, symptoms of "chokes" may develop due to congestion of the pulmonary vasculature. Pulmonary DCS or "chokes" is manifested by: (1) burning substernal chest pain, often aggravated by breathing, (2) cough, and (3) shortness of breath (dyspnea). Pulmonary DCS makes up 5 to 10 percent of Type II Altitude DCS. Symptoms of increasing lung congestion may progress to complete circulatory collapse, loss of consciousness, and death if recompression is not instituted.

Factors Associated with Decompression Sickness

Altitude or Depth Attained. DCS occurring from altitude exposures below 18,000 feet is rare and usually results from other predisposing factors. In an Air Force series of Altitude DCS cases, only 13 percent occurred below 25,000 feet, and 79 percent occurred above 30,000 feet. In diving operations, DCS should not occur in water shallower than the no decompression limit. Deeper and longer dives result in DCS upon return to the surface unless a slow staged decompression back to the surface is followed. Rate of ascent (change in pressure differential) will also effect DCS incidence.

Duration of Exposure. Altitude DCS is rare in exposures of less than five minutes at altitude. Exposures of 20 to 60 minutes show an increased occurrence of DCS.

Surface Interval Prior to Reexposure. Reexposure to altitudes over 18,000 feet within three hours increases the risk of DCS. Sea level intervals of 24 to 48 hours may be required between

altitude exposures to reduce the risk of DCS to baseline. U.S. Navy guidelines for low pressure chamber flights above 18,000 feet include 48 hour surface intervals, and no more than three chamber flights in a seven day period. For altitudes from 10,000 to 18,000 feet, a 24-hour surface interval is required. Surface intervals for dive operations are dependent on residual nitrogen times calculated from the dive tables.

Flying after Diving. Following hyperbaric exposure to compressed gas, a person has an excess of dissolved gas (residual nitrogen) which continues to off-gas at a predictable rate. Exposure to a hypobaric environment may accelerate this off-gassing leading to bubble formation and DCS. DCS following diving has occurred as low as 7000 feet. Following a 1600 foot deep saturation dive, team members developed DCS four days later on a commercial air flight. OPNAVINST 3710.7 states *"Under normal circumstances, flight personnel shall not fly or perform low pressure chamber runs within 24 hours following scuba diving, compressed air dives, or high pressure chamber runs. Under circumstances where an urgent operational requirement dictates, flight personnel may fly within 12 hours of scuba diving, providing no symptoms of aeroembolism develop following surfacing and the subject is examined and cleared by a flight surgeon."*

Diving at Altitude. Diving at altitude refers to diving at elevations higher than sea level, such as mountain lakes. Current U.S. Navy dive tables are based on sea level surface. Diving at altitude may increase the DCS risk. U.S. Navy diving above 2300 feet requires CNO approval.

Prior DCS. In the diving community, prior DCS or subclinical DCS might increase the risk of DCS. Approximately five to 10 percent of Altitude DCS cases had prior DCS. Other factors such as age and injury may be confounding variables.

Occupation. Earlier studies revealed higher Altitude DCS incidence in insider observers of low pressure chambers. A recent study found identical rates in students and inside observers. Again confounding variables may be a factor.

Age. Incidence rates of DCS in those age 40 to 45 years is three times that of 19 through 25 year olds. U.S. Navy divers over 45 years old must be waived to dive and are restricted to supervisory type dives.

Gender. A recent study showed an association of DCS with female sex. The relative risk of Altitude DCS was twice that of men. Other studies have shown that DCS in women is temporarily related with the perimenstrual portion of the menstrual cycle. These results show an association, not necessarily causal, and are being further studied.

Exercise. Exercise appears to increase the incidence of DCS. Exercise leads to increased muscle perfusion, an increase in inert gas uptake, shear forces in joints causing gas micronuclei, and

increased carbon dioxide which may accelerate bubble growth. Decompression time is extended for divers engaged in strenous activity. The altitude equivalent with exercise is an additional 3000 to 5000 feet. Individuals undergoing altitude exposures over 18,000 feet should refrain from vigorous exercise for 12 hours prior to exposure and three to six hours following exposure. This will avoid predisposing factors and confusion regarding musculoskeletal pain and limb bends.

Injury. Recent injury may predispose to DCS. Although the exact mechanism is unclear, local inflammatory reaction, changes in perfusion, and gas micronuclei may be involved.

Temperature. Very cold ambient temperature increases the risk of DCS perhaps by changes in nitrogen washout from peripheral vasoconstriction.

Body Morphology. Although body weight does not affect DCS incidence, body fat does appear to be a predisposing factor, probably related to increase in tissue nitrogen stores.

Inspired carbon dioxide concentration. Increased carbon dioxide in inspired gas predisposes to DCS because of its high solubility in gas micronuclei.

Hypoxia. Hypoxia has been antecdotally related to DCS.

Personal Factors. Alcohol ingestion, dehydration, and fatigue have anecdotally been associated with DCS.

Arterial Gas Embolus. Dive profiles conducive to arterial gas embolus (bouyant ascent) may produce a nidus of bubble nuclei into which dissolved gases could diffuse where they would have otherwise remained in solution.

Venous Gas Embolus and Atrial Septal Defects. Venous bubbles are detected by precordial doppler ultrasound following reduction in ambient pressures in otherwise asymptomatic people. These venous gas bubbles are normally filtered from the pulmonary circulation by the lung. Several recent articles have implicated atrial septal defects such as a patent foramen ovale as predisposing to DCS by allowing these otherwise silent venous bubbles to pass into the arterial circulation where they are spread throughout the body. Patent foramen ovale, detected with bubble contrast ultrasound techniques, has been detected in significantly higher numbers of Type II DCS cases where the dive profile was not likely to cause DCS (undeserved DCS). Patent foramen ovale has also been implicated in Altitude DCS cases.

Altitude Decompression Sickness Versus Diving Decompression Sickness

The cause, clinical effects, and treatment of these two syndromes are idential. However, altitude DCS tends to result in cerebral lesions, whereas DCS occurring during diving is more likely to involve lesions of the spinal cord. The reason for this difference is unknown. It is important to

note that the entire spectrum of clinical manifestations is possible in either type.

Differential Diagnosis of Altitude Decompression Sickness

1. Musculoskeletal (non-DCS) limb pain
2. Hypoxia
3. Hyperventilation
4. Carbon monoxide poisoning
5. Spatial disorientation
6. Air sickness
7. Trapped gas abdominal distention
8. Alternobaric vertigo
9. Perilymph fistula
10. Acceleration atelectasis
11. Spontaneous pneumothorax
12. Migraine syndrome
13. Entrapment neuropathy
14. Cervical radiculopathy

Pulmonary Overinflation Syndromes

The pulmonary overinflation syndromes are barotrauma disorders caused by gas expanding within the lung, resulting in alveolar rupture. The syndromes encountered include arterial gas embolism, pneumothorax, mediastinal emphysema, subcutaneous emphysema, and rarely pneumopericardium.

Alveolar rupture may result from excessive positive pressure (failed regulator) or failure of gas to escape from the lung during ascent. This may occur from voluntary breath holding during a panic ascent or from localized pulmonary obstruction (asthma, secretions, and calcification). Pulmonary bullae are particularly susceptible to alveolar rupture.

Arterial Gas Embolism

Arterial gas embolism is caused by entry of gas emboli into the arterial circulation where they are dispersed throughout the body. The organs susceptible to arterial gas embolism, the CNS and heart, are responsible for life threatening symptoms. In all cases of arterial gas embolism, pneumothorax is a possibility.

Symptoms of arterial gas embolism are likely to show up within a minute or two after surfacing. Any CNS symptom other than unconsciousness which occurs much later than 10 minutes after surfacing is rarely the result of arterial gas embolism. Anyone who has obtained a breath of compressed gas from any source at depth, whether from diving apparatus, Helicopter Emergency Escape Device (HEEDS) bottle, or a diving bell, and who is unconscious or loses consciousness within 10 minutes of reaching the surface, must be assumed to be suffering from arterial gas

embolism. Recompression therapy must be started immediately.

Characteristics of Arterial Gas Embolism

Sudden Onset. The onset is usually sudden and dramatic, often occurring within seconds after arrival on the surface or even before reaching the surface. The signs and symptoms may include dizziness, paralysis, weakness in the extremities, large areas of abnormal sensation, blurring of vision, or convulsions. During ascent, the diver may have noticed a sensation similar to that of a blow to the chest. The victim may become unconscious without warning and may even stop breathing.

Similarity to DCS. Some of these symptoms may also be experienced by a diver suffering from DCS. If the dive has been to a depth of less than 33 feet, DCS is unlikely and arterial gas embolism must be assumed. If the only symptom described is pain, arterial gas embolism is unlikely. DCS or one of the other pulmonary overinflation syndromes, which are not usually acute emergencies, should be considered.

Masking of Symptoms. Some symptoms may be masked by environmental factors or by other less significant symptoms. A diver who is chilled may not be concerned with numbness in an arm which may actually be a sign of nervous system involvement. Pain from any source may divert attention from other symptoms. The natural anxiety that accompanies a "close call," such as the failure of the diver's air supply, or egress from a helicopter lost at sea, might mask a state of confusion caused by an arterial gas embolism to the brain. A diver coughing up blood or bloody froth may be showing signs of ruptured lung tissue, or he may merely have bitten his tongue or experienced a case of sinus squeeze.

Spontaneous Improvement. Symptoms of arterial gas embolism may improve spontaneously without treatment. If left untreated, these symptoms may recur with increased severity. Even if the symptoms resolve, treat the diver as if symptoms were still present.

Arterial Gas Embolus Versus Decompression Sickness

At times it may be difficult to distinguish arterial gas embolism from DCS. The treatment for arterial gas embolism is usually longer and deeper than that for DCS because the danger from brain damage is so much greater. Recompression treatment for arterial gas embolism will also be adequate treatment for DCS. If there is any doubt as to the correct diagnosis, assume arterial gas embolism. Although both DCS and AGE may present within minutes of reaching the surface, symptoms presenting after 10 minutes are not consistent with AGE. AGE usually presents with substantial neurological symptoms localized to brain or higher cortical centers. If spinal cord symptoms are present, it is more likely DCS. Certain dive profiles (short, shallow dives) are not likely to cause decompression sickness and would be more consistent with AGE (i.e., HEEDS training). Ascents from depth that are uncontrolled are more consistent with AGE. A patient with other signs of Pulmonary Overinflation Syndromes (POIS) is more likely to have AGE.

Other Pulmonary Overinflation Syndromes

Expanding gas trapped in the lung may enter tissue spaces causing mediastinal emphysema, subcutaneous emphysema, pneumothorax, and pneumopericardium. Tension pneumothorax may be life threatening requiring thoracostomy. Mild pneumothorax may respond to 100 percent 0_2. Pneumopericardium is rare. It is generally reported only on radiographs. Recompression therapy is not necessary for emphysema or pneumothorax, and may convert a simple pneumothorax into a tension pneumothorax.

Treatment of Bubble Related Disorders

Recompression Therapy. The only satisfactory treatment for DCS or AGE is recompression therapy. Medical therapy and observation only have an adjunctive role in the management of DCS or AGE once the diagnosis is made. Once the diagnosis is made, the patient should be transported as quickly as possible to a recompression chamber where appropriate therapy can be administered according to current protocals (NAVSEA 0994-LP-001-9010). Altitude chamber personnel are well trained in applying these theraputic methods to patients with DCS and AGE.

A brief synopsis of these methods is included here. Actual recompression therapy must be administered by trained chamber personnel in accordance with Navy diving procedures.

Air Treatment Tables. A treatment table is a time-pressure profile applied in a recompression chamber to treat patients with DCS and other dysbaric illnesses. The pressure is measured in Feet Sea Water (FSW). There are two basic types of treatment tables, those using air only, and those where 100 percent oxygen is available in the chamber. The first treatment tables introduced were air tables. Patients treated with air tables are pressurized in an air atmosphere while breathing the air in the chamber. Although these patients receive the benefits of pressure, they also take up additional nitrogen during the treatment which must be removed by slow decompression. Therefore, air tables are quite lengthy.

Oxygen Treatment Tables. The more recently developed oxygen treatment tables pressurize the patient with air, but oxygen is available for breathing by mask (Built in Breathing System or BIBS). Oxygen breathing provides several advantages. The increased oxygen partial pressure provides life-sustaining oxygen to tissues compromised by bubbles. No nitrogen is inhaled by the patient so an increased alveolar nitrogen gradient is created to remove nitrogen from the body. Also, no additional nitrogen is dissolved in the patient's tissues. During the treatment this permits a more rapid reduction of pressure, or ascent, from treatment depth to the surface. As oxygen tables are considerably shorter, there is less risk of DCS to the inside tenders. Oxygen Tables are superior to the older air tables, and should be used whenever possible. A disadvantage of oxygen tables is that oxygen toxicity may occur. The oxygen treatment tables include air breaks (five minute interruptions when air is breathed instead of oxygen) to reduce the likelihood of oxygen toxicity. Acute oxygen toxicity causes increased irritability of the CNS. Symptoms of CNS

oxygen toxicity include visual abnormalities (such as tunnel vision), tinnitis, nausea, twitching, irritability, dizziness, and seizures. When oxygen tables are used, the inside tenders (the medical observers inside the chamber) breathe oxygen during part of the treatment to reduce their tissue nitrogen tension and minimize their risk of bends.

Indications for Hyperbaric Oxygen Therapy

The oxygen treatment tables are useful in treating a variety of nondiving illnesses, such as carbon monoxide toxicity, cyanide poisoning, gas gangrene, and smoke inhalation. The increased oxygen tension will help displace these toxins by mass action. Additionally, enough oxygen will dissolve in serum that significant anemic states can be overcome (serum pressurized to 60 FSW can support life without red cells or hemoglobin). NAVMEDCOMINST 6320.38A limits the use of the U.S. Navy hyperbaric chambers for nondiving illness to carbon monoxide toxicity, cyanide poisoning, gas gangrene, iatrogenic gas embolism, and smoke inhalation. Other uses require prior approval from the Chief, Bureau of Medicine and Surgery. In addition, the Undersea and Hyperbaric Medical Society has approved recompression therapy for radiation necrosis, refractory osteomyelitis, selected burns, nonhealing wounds, failing skin flaps and grafts, necrotizing soft tissue infection, acute anemia, and crush injuries. A number of disorders, such as Multiple Sclerosis and stroke, have been treated with recompression therapy in experimental settings.

Indications for Specific Treatment Tables

The treatment tables (Table 13) were given arbitrary numerical names as they were historically developed. The treatment tables a medical officer should be familiar with are Treatment Tables 4, 5, 6, 6A, and 7.

Treatment Table 5 - Type I DCS Only. Treatment Table 5 (TT 5) in Figure 20 is an oxygen table used to treat Type I DCS. At two hours and 15 minutes, it is the shortest table. The patient is pressurized to 60 FSW for two oxygen periods, brought to 30 FSW for one additional oxygen period, and slowly brought to the surface. The patient also breathes oxygen while changing depths. Five minute air breaks between oxygen periods prevent CNS oxygen toxicity.

Treatment Table 6 - Type II DCS (Except Inner Ear DCS), Type I DCS with Pain Over 10 Minutes at Depth on TT 5. If the Type I symptoms do not resolve within 10 minutes at 60 FSW or if the patient has Type II DCS, treatment is completed using Treatment Table 6 (Figure 21), (the patient is "brought out" on a Treatment Table 6). This oxygen table lasts four hours and 45 minutes. It is similar to Treatment Table 5 except the times at 60 FSW and 30 FSW are increased. Additionally, if clinically indicated (i.e., if symptoms are not resolved), Treatment Table 6 may be lengthened. A total of four additional time periods, called extensions: two at 60 and two at 30 feet may be administered as needed.

Treatment Table 6A - AGE, Inner Ear DCS. Treatment Table 6A is used to treat arterial gas

embolism. Treatment Table 6A is just like Treatment Table 6, except the patient is first brought to 165 FSW for 30 minutes on air to compress intra-arterial bubbles as much as possible. Oxygen cannot be used at this depth because of oxygen toxicity. After the initial period of deep recompression, the patient is brought to 60 FSW. The rest of the treatment is like Treatment Table 6.

Treatment Tables 4 and 7. For very sick patients two additional tables are available, Treatment Tables 4 and 7. Treatment Table 4 is used to treat symptoms refractory to treatment at 60 feet by increasing the depth to 165 feet. Treatment Table 4 is also used to allow gas embolism patients more time at 165 feet than permitted by Treatment Table 6. Oxygen cannot be used until the patient reaches 60 feet. Because the tissues become nitrogen-saturated due to the extended time at depth, the patient must be brought to the surface very slowly. Treatment Table 4 takes 38 hours and 11 minutes to complete, and is basically an air saturation decompression table.

For the patient with life-threatening DCS unresponsive to treatment, the option of Treatment Table 7 is available. This table provides for maximal treatment time at 60 feet. The treatment includes a stay at 60 feet of at least 12 hours, with an extremely gradual saturation-type ascent lasting 36 hours. There is no upper limit on the time the patient may be kept at 60 feet. Treatment Table 7 should be used only by a Diving Medical Officer who has support personnel and other assets readily available to properly execute treatment.

Treatment Tables 4 and 7 are not used to treat minor neurological deficits which persist during treatment with Treatment Table 6 or 6A. Instead, these patients are retreated daily until symptoms no longer improve.

Twenty-four hour consultation is available with the Experimental Diving Unit at Panama City (NEDU) (AUTOVON 436-4351, Commercial (904) 234-4351), or the Naval Medical Research Institute (NMRI) at Bethesda, MD (AUTOVON 295-1839, Commercial (202) 295-1839) for questions regarding hyperbaric treatment or triage. Questions on Hyperbaric Oxygen therapy (HBO) for nonbubble related diseases may be referred to Wright Patterson AFB Medical Center at (513) 257-8603.

Triage and Referral of Altitude DCS Patients

All patients with Type II DCS must be recompressed urgently or evacuated promptly for hyperbaric treatment.

Patients with Type I DCS should be closely questioned about the onset of their symptoms. Patients whose symptoms appear at altitude, then resolve spontaneously on descent, should be placed in 100 percent oxygen and observed for two hours for evidence of presentation or recurrence of DCS. After two hours of observation, they are grounded for one week and returned to light duty. They must be warned to seek treatment promptly if any symptoms

reoccur. Any recurrence must be treated with hyperbaric therapy.

Patients who first develop Type I symptoms at ground level after flight, or whose symptoms start at altitude and persist at ground level, must be placed on 100 percent oxygen while recompression or evacuation is arranged. If symptoms resolve while awaiting transportation, evacuation is postponed; and, these patients are observed on 100 percent oxygen for 24 hours. Any recurrence must be treated with hyperbaric therapy. Patients who remain symptom-free for the 24 hour observation period are grounded for one week and placed on limited duty with no physical training for at least 72 hours. They are advised to return promptly for reevaluation if symptoms recur. Current U.S. Navy diving medicine protocols are to treat all patients referred for altitude DCS regardless of whether or not their symptoms have resolved. Therefore, once patients are evacuated, they will be treated.

Aeromedical Evacuation

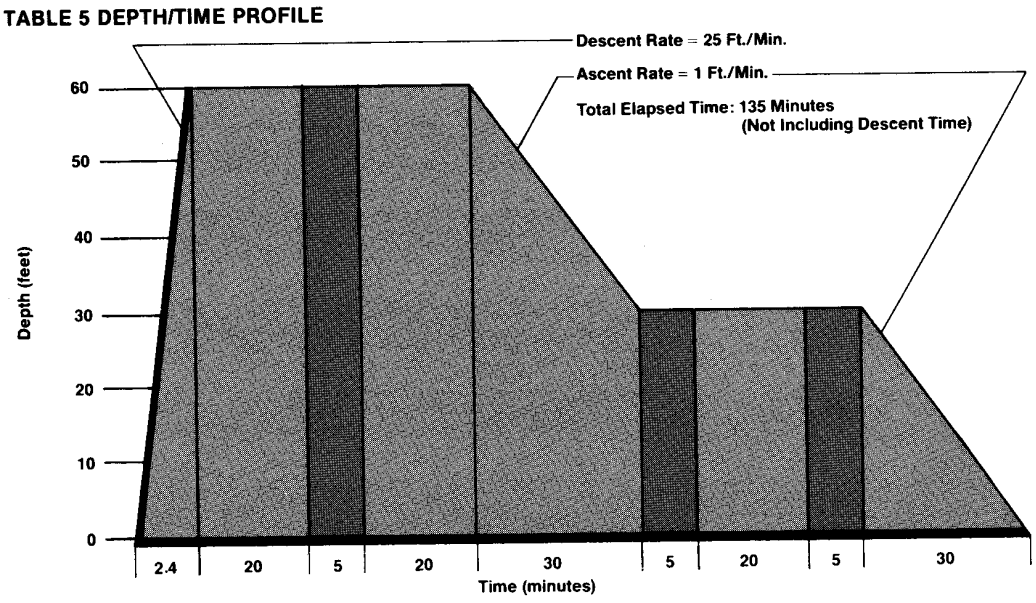
Some points specific to evacuation of hyperbaric patients bear mentioning. First, the medical department should know the location of the nearest recompression chambers, and how to contact

List of U.S. Navy Standard recompression treatment tables

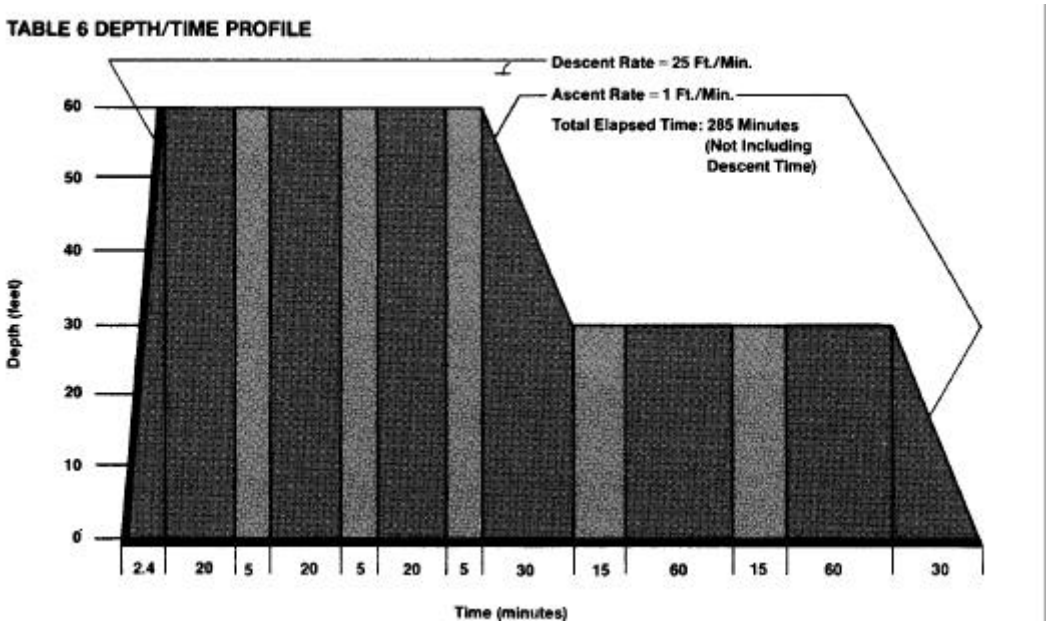
TABLES USED WHEN OXYGEN AVAILABLE	
4 Air/Oxygen treatment of type II decompression sickness or Gas Embolism	Treatment of worsening symptoms during the first 20-min oxygen breathing period at 60 feet on Table 6 or unresolved arterial gas embolism symptoms after 30 min at 165 feet.
5 Oxygen treatment of Type I Decompression Sickness	Treatment of type I decompression sickness when symptoms are relieved within 10 minutes at 60 feet and a complete neurological exam was done and is normal.
6 Oxygen treatment of Type II Decompression Sickness	Treatment of Type II decompression sickness or Type I decompression sickness when symptoms are not relieved within 10 minutes at 60 feet.
6A Air and oxygen treatment of Gas Embolism	Treatment of gas embolism symptoms relieved within 30 min at 165 feet. Use also when unable to determine whether symptoms are caused by gas embolism or severe decompression sickness.
7 Air and Oxygen Treatment of life threatening or extremely serious symptoms	Treatment of unresolved severe symptoms at 60 feet after initial treatment on Table 6, 6A or 4. Used only in consultation with a Diving Medical Officer.
TABLES USED WHEN OXYGEN NOT AVAILABLE	
1A Air treatment of Type I decompression sickness – 100 foot treatment	Treatment of Type I decompression sickness when oxygen unavailable and pain relieved at a depth less than 66 feet.
2A Air treatment of Type I decompression	Treatment of Type I decompression sickness when oxygen unavailable and pain is relieved at a depth

sickness – 165 foot treatment	greater than 65 feet.
3 Air treatment of Type II decompression sickness or gas embolism	Treatment of Type II symptoms or gas embolism when oxygen unavailable and symptoms are relived within 30 minutes at 165 feet.
4 Air treatment of Type II decompression sickness or gas embolism	Treatment of symptoms which are not relieved within 30 minutes at 165 feet using Air Treatment Table 3.

Standard United States Navy treatment table 5 – pain only bends



Standard United States Navy treatment table 6 – decompression sickness



personnel there. Contact should be made and the case discussed prior to transport or concurrently with transport. The aircraft should be pressurized to an altitude of 500 feet or less to prevent further bubble formation and expansion. The patient should be placed on 100 percent oxygen if available. Earlier guidelines recommended placing the patient with AGE in the left lateral decubitus position with the head down during transport, apparently to keep the bubbles from the head and heart. This may have the effect increasing intracranial pressure and reducing ventilation. The supine position is appropriate in an alert person. However, an unconscious person may be placed in the lateral decubitus position to prevent aspiration. The patient should be supine, neck in the neutral head position, and uncramped with the extremities uncrossed. The patient should also be placed so that the face is visible to the tender. The patient should not be permitted to sleep, so that changes in neurological status will be readily detected. Intravenous fluids, such as Ringers Lactate or normal saline, should be used. Free water solutions such as D5W should be avoided as they may contribute to cerebral swelling. A plastic IV bag may be used as a pillow. This will also serve to maintain the IV. Dexamethasone, while controversial, can be given 10 mg IV stat followed by 4 mg IV or IM q6hr. Inflatable cuffs, such as endotrachial cuffs, should be filled with water, not air.

Flying After Diving

Required intervals between diving and flying are given in Table 14.

Table 14

Category	Surface Interval Before Flight
Flight Crew, Divers	24 hours
No-Decompression Dive	12 hours
Decompression Dive (nonsaturation)	24 hours
Saturation Dive	72 hours

Aeromedical Disposition

Once the patient has been diagnosed, evacuated, and treated, the question arises as to their flight status. The flight surgeon must be consulted for these cases to conduct a complete fitness to continue physical examination. The aeromedical disposition is made based on diagnosis,

classification, treatment course, and duty status.

Any documented history of gas embolism should be worked up for pulmonary bullae and other causes of Pulmonary Overinflation Syndrome as well as for atrial septal defects. Any persistent neurological sequelae of DCS or AGE are considered disqualifying.

Disposition following Hyperbaric Recompression

The required intervals between the completion of recompression treatment and flying as a passenger are provided in the following table.

TIME FOLLOWING COMPLETION OF RECOMPRESSION TREATMENT

CONDITION	TME ON STATION (5 Min away)	IN LOCAL AREA (30 Min away)	MAY FLY (As passenger)
Type I DCS	2 hrs	24 hrs	24 hrs
Type II DCS	6 hrs	48 hrs	48 hrs
DCS/AGE treated On TT 6A/4/7	12-24 hrs	72 hrs	72 hrs
DCS/AGE with Residual symptoms	12-24 hrs	72 hrs, (Cleared by DMO)	72 hrs, (Cleared by DMO)
Inside Tender on TT 5/6/6A		12 hrs	12 hrs
Inside Tender on TT 4/7		48 hrs	48 hrs