Updates in Decompression Illness

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KEYWORDS

• Decompression sickness • Arterial gas embolism • High-fraction oxygen
• Hyperbaric

KEY POINTS

- Decompression sickness (DCS) is a disease resulting from an ascent profile not allowing the orderly elimination of excess gas that was accumulated in tissues during exposure to elevated pressure.
- Decompression sickness (DCS) can present idiosyncratically, affecting a wide range of systems with a variable degree of insult. Masking of important symptoms by chief complaint is possible.
- Arterial gas embolism (AGE) is a disease of frank gas in the arterial systemic circulation following a reduction of ambient pressure so rapid that expanding gases cause pulmonary tissue rupture.
- The first aid for decompression illness (collectively, DCS and AGE) is high fraction oxygen; the definitive treatment is hyperbaric oxygen therapy.
- There are currently no diagnostic tests to confirm decompression sickness.

INTRODUCTION

Diving is a popular recreational pastime, as well as an activity with numerous applications in the scientific, commercial, military, and exploration realms. Although diving can be done safely, the underwater environment is unforgiving. Problems may arise during a dive due to insufficient medical or physical fitness, improper use of equipment, or inadequate management of the high-pressure environment.

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Decompression illness (DCI) is a term used to encompass injuries due to arterial gas embolism (AGE) and decompression sickness (DCS). AGE typically results from pulmonary barotrauma-induced damage to the alveolar wall and introduction of gas into the systemic arterial circulation. DCS, colloquially known as the bends, results from the uncontrolled release of gas from tissues during or after surfacing with inadequate time for equilibration (decompression).

DIVING PHYSICS

The concentration, or tension, of dissolved inert gas within body tissues is a function of ambient pressure. Inert gases normally exist in equilibrium with the ambient environment, effectively a saturated state. When pressure differences (gradients) are created, molecules flow from the area of higher to lower concentration until equilibrium is re-established.

The pressure range of the diving environment is much greater than the pressure range of the air environment. The pressure exerted at sea level by the entire 100 km (62 mi) atmospheric column of gas is 1 atm absolute (ATA), equal to 101.3 kPa or 14.7 psi. In contrast, water pressure increases by 1 atm for every 10 m (33 ft) of seawater and for every 10.4 m (34 ft) of freshwater (Fig. 1).

The lungs play a primary role in gas uptake and elimination and, ultimately, decompression stress. When exposed to increased pressure underwater, the gas in the lungs is compressed. This creates an inflow gradient from concentrated lung gas to the bloodstream and, subsequently, from the bloodstream into tissues as they are perfused. Tissues take up inert gas until saturation is achieved. At the point of saturation, staying longer does not further increase the subsequent decompression obligation.

PREDICTING GAS UPTAKE AND ELIMINATION

Gas uptake and elimination generally follows roughly exponential patterns. The technology is not yet available to measure tissue status directly, so the norm is to rely on

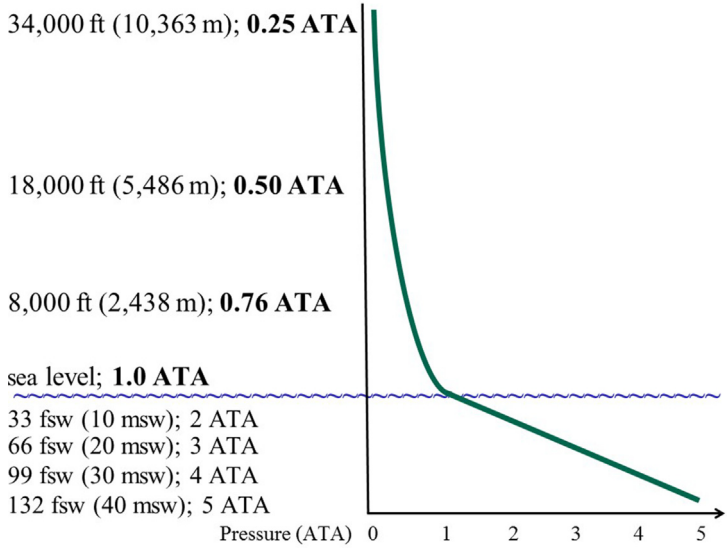

Fig. 1. Air pressure increases slowly from zero at the boundary of space to 1 atm (14.7 psi) at sea level. Water pressure increases much more dramatically, adding 1 atm of pressure for every 10 m of seawater. fsw, feet of seawater; msw, meters of seawater.

mathematical algorithms to predict gas exchange. A range of half-times are used to represent different tissue characteristics; not as exact referents but as mathematical constructs to collectively estimate what happens throughout the body.

The fastest tissues are the lungs, which achieve equilibrium almost instantly. Blood is another extremely fast tissue, followed by the brain. The slowest tissues are those that are relatively poorly perfused, such as ligaments and cartilage, or those that have a high capacity for inert gas uptake, such as fat in poorly perfused areas.

Consider a diver instantly displaced from the surface to a fixed depth as an example. A 5-minute half-time would represent a fast tissue, computed to take up sufficient inert gas uptake to eliminate half of the difference produced by the pressure gradient (50%) in the first 5 minutes. This would be the steepest portion of the uptake curve. The second 5-minute period would eliminate half of the remaining difference (25%). The third 5-minute period would eliminate half of the remaining difference (12.5%) and so on. At the other extreme, a 500-minute half-time could be computed to represent an extremely slow tissue. With no additional influences on the process, equilibration is expected to be nearly achieved after a period equal to about 6 half-times for any given tissue (**Fig. 2**).

Most dives do not last long enough for the diver to reach saturation; these are known as bounce dives. During such exposures, the inflow gradient exists at least through the descent and bottom phase of the dive, which causes continued uptake of inert gases, certainly in slow compartments and probably in intermediate compartments. When the diver ascends, the ambient pressure starts to drop and the gradient begins to reverse, first in fast compartments and then in progressively slower compartments.

When the tissue tension of a gas exceeds the ambient partial pressure of that gas, the tissue is supersaturated. This will be the case of most tissues during and after surfacing. If the degree of supersaturation is modest, equilibration from peripheral tissues into the blood, then lungs, and then atmosphere, will be orderly. If the degree of supersaturation is too great, the elimination of inert gases becomes disorderly, gas phase (bubbles) can form, and DCS may develop.

Bubble formation does not always cause problems but the greater the supersaturation the greater the likelihood that symptomatic DCS will occur. It is a misconception

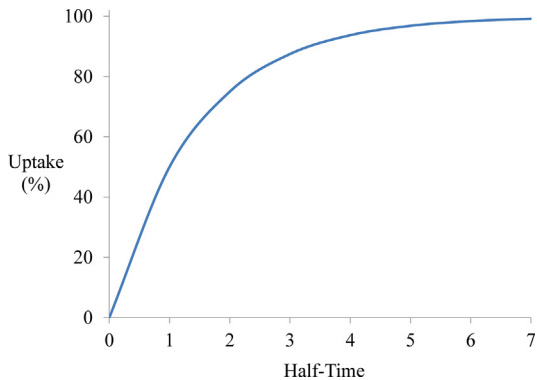

Fig. 2. The half-time concept describes how an undersaturated tissue takes up inert gas. When held at a fixed pressure, enough gas is taken up to eliminate half of the difference in gas pressures in each half-time period. For a hypothetical tissue with a half-time of 10 minutes (and no other influences), 50% of the difference is eliminated in the first 10 minutes, then 25% in the next 10 minutes (half of the remaining 50%), then 12.5%, 6.25%, 3.125%, and 1.56%, and so forth.

that bubbles forming after a dive are of no importance; however, it is also a misconception that bubbles equate to DCS. The formation of gas bubbles during decompression represents a stress greater than is optimal and may lead to DCS.

CONTROLLING DIVE EXPOSURES

Dive computers have supplanted printed dive tables as the primary means of regulating dive profiles over the last 20 years. Dive computers provide more flexible guidance because they continuously assess the pressure-time profile and compute the status of a variety of hypothetical tissue compartments. Exposure limits or decompression obligations are adjusted based on whatever compartment is deemed most critical (effectively the controlling half-time) at any point in the ascent. This is useful because modern divers frequently follow complex descent-ascent profiles, relying on the dive computer to keep track of their state.

Dive computers provide guidance based on the primary determinants of decompression risk: the pressure-time profile and breathing gas. However, gas exchange is also influenced by the timing and intensity of exercise and thermal stressors during a dive, as well as by individual predispositions, none of which are assessed by dive computers in a meaningful way.

Symptoms of DCS may develop after dives conducted within decompression algorithm limits. Decompression algorithms predict outcomes but they do not guarantee them. That a dive is conducted within the limits does not make a DCS hit undeserved.

DEVELOPING DECOMPRESSION SICKNESS

Although DCS is commonly thought of as a bubble disease, bubbles are probably only the gateway to a complex array of consequences. Bubbles can form in or reach a wide variety of tissues, and initiate biochemical cascades to produce secondary insults and effects. Vascular obstruction or interactions may stimulate platelet aggregation, leukocytes activation and aggregation, and fibrin deposition. Intravascular bubbles may also damage the capillary endothelium, possibly leading to increased permeability and tissue edema exacerbating the ischemia process. An increased release of cytokines and/or complement activation has also been demonstrated as possible contributing agents. The inflammatory cascade could play an important role in the pathophysiology of DCI. The variability in activation threshold of this inflammatory cascade might explain the differences between individuals in susceptibility to DCS. The inflammatory and coagulation cascades might also explain the failure of recompression treatment in some cases because, once activated, the resolution of bubbles will not immediately stop the response.

THE MECHANICS OF ARTERIAL GAS EMBOLISM

Unlike DCS, which requires a period of gas uptake, AGE results from an acute decrease in ambient pressure in a tissue unable to accommodate. For example, a diver ascending rapidly from 10 m (2 ATA) to the surface (1 ATA) is faced with a 50% reduction from the bottom pressure. In accordance with Boyle's law ($P_1V_1 = P_2V_2$; assuming constant temperature), an unventilated volume of gas would double during this excursion. Normally, the diver would breathe freely during a slow ascent and constantly re-equilibrate lung volume. If ventilation is not adequate, either due to breath-holding or a localized bronchial obstruction (eg, bronchospasm or mucus plug), an overexpansion injury (pulmonary barotrauma) can result. The maximum sustainable tissue elastic pressure in the alveoli is around 100 to 150 mm Hg (0.13–0.2 atm) over ambient. A pressure

of 0.13 atm is found at a depth of 1.3 m (4 ft) in seawater. A rapid ascent with full lungs and an obstructed airway from this very shallow depth could result in pulmonary barotrauma. Tissue rupture will allow gas from the alveolar space to enter the pleural space (pneumothorax), the pulmonary interstitium, the mediastinum (pneumomediastinum), and/or the pulmonary capillaries. Bubbles entering the pulmonary capillaries can be transported into the systemic arterial circulation and may reach critical tissues, such as the brain or the spinal cord, to produce serious symptoms. The symptoms of AGE can be very similar to those of DCS, sometimes making them difficult to separate.

INCIDENCE OF DECOMPRESSION ILLNESS

The lowest DCI incidence rates have been reported in the scientific diving community at 0.324 per 10,000 person-dives.[1] Divers Alert Network (DAN) estimates of DCI incidence rates in the recreational community range from 2.0 to 4.0 per 10,000 person-dives.[2–4] The DCS incidence rate in commercial decompression diving has been reported to be as high as 35.3 per 10,000 person-dives.[5] A more recent study of commercial diving DCS described incidence rates ranging from 1.4 to 10.3 per 10,000 person-dives, depending largely on the depth of dive operations.[6]

SIGNS AND SYMPTOMS

A variety of classification systems have been established for DCS. One common approach is to describe cases as type 1 or type 2.

Type 1 DCS is usually characterized by musculoskeletal pain and mild cutaneous symptoms. Common type 1 skin manifestations include itching and mild rashes (as distinct from a clear, mottled or marbled, and sometimes raised discoloration of the skin known as cutis marmorata [**Fig. 3**] that may presage the development of the more serious type 2 symptoms). Less common but still associated with type 1 DCS is obstruction of the lymphatic system, which can result in swelling and localized pain in the tissues surrounding the lymph nodes.

Musculoskeletal manifestations of type 1 DCS are articular or periarticular pain. The joint pain usually has a gradual onset and presents as a deep dull ache. Pain intensity

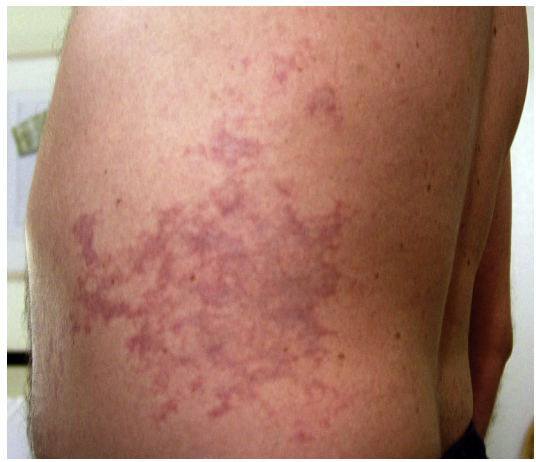

Fig. 3. Skin mottling like this is characteristic of cutis marmorata, a condition that can warn of likely development of more serious type 2 symptoms. (*Courtesy of* N.W. Pollock, PhD, Durham.)

may range from mild to severe. The pain will typically be present at rest and may or may not be exacerbated by movement. Common locations include shoulders, elbows, wrists, hips, knees, and ankles.

Type 2 symptoms are considered more serious. They typically fall into 3 categories: neurologic, inner ear, and cardiopulmonary. Neurologic symptoms include numbness; paresthesia or tingling; muscle weakness; impaired gait, physical coordination or bladder control; paralysis; or change in mental status. Some neurologic symptoms are commonly described as constitutional, such as headache, lightheadedness, unexplained fatigue, malaise, nausea and/or vomiting, or anorexia. Inner ear symptoms include tinnitus, hearing loss, vertigo or dizziness, nausea, vomiting, and impaired balance. Cardiopulmonary symptoms, known as the chokes, include a dry cough, retrosternal pain, dyspnea, and sometimes pink-stained, frothy sputum. Cardiopulmonary involvement occurs when massive bubble loads obstruct a substantial proportion of the pulmonary vascular bed. Cardiopulmonary DCS usually follows highly provocative dive profiles with a significant omitted decompression. It is important to rule out immersion pulmonary edema, which can present like cardiopulmonary DCS.

Type 2 symptoms can develop slowly or quickly. A slow build can obscure the seriousness of the situation, allowing denial to persist. For example, fatigue and weakness may initially be easy to ignore. Less common symptoms, such as difficulty walking, urinating, hearing, or seeing (especially with quick onset) are more difficult to deny.

Other types of classification exist for DCS. For the first-line physician, differentiation between type 1 or type 2 DCS is somewhat unnecessary because DCS is a systemic disease that often involves multiple organs and, in any case, the final treatment approach will not differ that much.

The presentation of DCS is frequently idiosyncratic; that is, its typical pattern can be atypical. In some cases, an affected diver's chief complaint may draw attention away from subtler but potentially more important symptoms. The following list ranks the initial manifestations of DCS, with frequency estimates for each[7]:

- Pain, particularly near the joints (68%)
- Numbness or paresthesia (63%)
- Constitutional symptoms (41%)
- Dizziness or vertigo (19%)
- Motor weakness (19%)
- Cutaneous changes (10%)
- Impaired mental status (8%)
- Impaired coordination (8%)
- Muscle discomfort (7%)
- Pulmonary symptoms (6%)
- Bladder or bowel dysfunction (3%)
- Auditory symptoms (2%)
- Impaired consciousness (2%)
- Lymphatic involvement (2%)
- Compromised cardiovascular function (<1%).

Clinical manifestations are described by system in **Table 1**.

Symptoms might present with some delay. Severe neurologic DCS symptoms usually appear within 10 minutes of surfacing and in 90% of cases symptoms will be present within the first 3 hours.[8] In some cases, it can take up to 24 hours for symptoms to be noticed by the diver.

Table 1
Clinical manifestations of decompression sickness

System involved	Symptoms	Signs
Skin	Itching	Nonspecific skin rash, urticarial rash, well-organized mottling (cutis marmorata)
Lymphatics	Localized pain in the region of lymph nodes	Localized skin and soft tissue swelling (lymphedema)
Musculoskeletal	Articular or periarticular pain, muscular pain	Usually no joint swelling, no redness
Venous blood (pulmonary circulation)	Dyspnea, cough, respiratory distress, retrosternal pain worsened on inspiration	Tachypnea, tachycardia, hypotension, frothy bloody sputum, low oxygen saturation
Central nervous system	Headache, unexplained fatigue, malaise, dizziness, impaired cognitive processes, paresthesias, limb weakness, speech difficulty, visual loss, ataxia, nausea, vomiting, convulsions	Altered state of consciousness, confusion, visual field deficit, unusual distribution of sensory deficits, motor deficits, coordination deficit, gait and walking disturbance, ataxia, positive Romberg sign
Spinal cord	Back pain, girdling abdominal pain, numbness, paresthesias, limb weakness, urinary or fecal dysfunction (with urinary issues being more common)	Motor and/or sensory deficits, anal sphincter weakness, urinary retention, loss of bulbocavernosus reflex, loss of deep tendon reflexes
Peripheral nervous system	Numbness or paresthesias in a peripheral nerve distribution	Patchy sensory deficits
Inner ear	Deafness, vertigo, nausea, vomiting, ataxia, tinnitus	Acute sensorineural hearing loss, nystagmus

MEDICAL ASSESSMENT OF THE INJURED DIVER

The history and physical examination remain the essential tools for the management of suspected DCS. In addition to the usual medical questionnaire, the emergency physician should seek the following information:

- Time of onset and evolution of symptoms and signs: Onset of symptoms less than 10 minutes after surfacing could indicate AGE. Onset of symptoms more than 10 minutes after surfacing are more likely associated with DCS. An exception may appear with the extreme decompression violation possible with technical diving, potentially resulting in symptoms during or immediately after surfacing.
- Profiles of the recent dives: Depth-time exposure, ascent rate, ascent stops (obligatory decompression stops or nonobligatory safety stops), and patterns of repetitive dives. Greater depth and duration imply a larger inert gas burden but using maximum depth alone to assess severity can be misleading given the common use of multilevel dive profiles.
- Breathing gas: The use of oxygen-enriched mixtures (nitrox) is increasingly mainstream, and the use of helium-oxygen and helium-oxygen-nitrogen mixtures

(trimix) is increasingly common. Using nitrox within air exposure limits can substantially reduce decompression stress. Using nitrox to the limits of equivalent air depth tables extends bottom time with a similar risk as using air to the limits of air tables. The other mixed gases are generally used for deeper dives with an intent to limit narcotic effects and optimize decompression.

- Thermal stress: Being warm during the descent and/or bottom phase of a dive will increase inert gas uptake, increasing the subsequent decompression stress. Being cold during the ascent and/or stop phase inhibits inert gas elimination, increasing the decompression stress.[9] Excessive heating during the ascent and/or stop phase can reduce solubility in the peripheral tissues, promoting bubble formation, increasing decompression stress. (Note: physical thermal status is more important than water or air temperatures.)
- Exercise stress: Exercise during the descent and/or bottom phase of a dive will increase inert gas uptake, increasing the subsequent decompression stress. Light exercise during the ascent and/or stop phase promotes inert gas elimination, reducing the decompression stress. Higher levels of exercise during the ascent and/or stop phase can promote bubble formation and increase risk. After-dive exercise, particularly with high joint forces, can promote bubble formation and increase risk.
- Altitude exposure and diving: Flying to a destination near sea level before diving creates no risk (outside the possibility of mild dehydration or impairment due to long periods of relative immobility). Because flights end with compression, the tissues of plane passengers will be undersaturated on landing and subsequently accumulate inert gases to re-establish equilibrium with the ambient pressure. Flying after diving, however, increases decompression stress because the pressure in an aircraft cabin is below that of ground-level atmospheric pressure. Commercial pressurized aircraft must have the capability of maintaining cabin pressure at an equivalent 2438 m (8000 ft), approximately 0.76 ATA. To illustrate, a dive to 20 m (66 ft) at sea level produces an exposure pressure of 3.0 ATA. Returning to the surface at 1.0 ATA produces a 3-fold reduction in pressure (3.0:1.0). Immediately getting on a plane or driving to an altitude of 2438 m would produce a 4-fold reduction (3.0:0.76), a much greater decompression stress.
- State of hydration: Dehydration is often overstated as a risk factor by divers looking for something to blame but a relative state of dehydration can elevate the decompression stress of a given exposure.[10]
- Contributing factors: These include physical and medical fitness (chronic or current), health history (including DCS), and medication use (generally with no research data relative to diving).

A practical likelihood assessment for DCS is summarized in **Table 2**.
The physical examination should focus on the following:

- Vital signs: Shock could be present and may originate from a cardiopulmonary DCS, tension pneumothorax, hypovolemia due to physical trauma, or from a neurogenic shock due to spinal cord DCS. Any abnormality in vital signs should be rapidly identified and managed properly. Vital signs should include rectal temperature to detect hypothermia or heat exhaustion. As with any patient presenting with altered state of consciousness or neurologic symptoms, a blood glucose level should be measured at the bedside to exclude hypoglycemia.
- Cardiac and lung auscultation: Evaluate for diminished sounds on one side, subcutaneous emphysema, displaced trachea, and Hamman's sign (potentially indicating pneumothorax or mediastinal emphysema from a pulmonary barotrauma).

Table 2
Practical likelihood assessment for decompression sickness

	High Likelihood	Low Likelihood[a]
Symptom onset after dive	>10 min–6 h[b]	>12 h
Quality of pain	Unusual[c]	Not unusual
Response to subsequent dive	Symptoms improve at depth	Symptoms unchanged at depth
Dive profile	Difficult without critical examination[d]	—

[a] Symptoms developing at depth, before decompression, will exclude DCS.
[b] Less than 10-minute onset could indicate AGE or an extreme decompression violation possible with technical diving.
[c] Symptoms associated with DCS are commonly described as different from normal pains.
[d] Consultation with subject matter expert is aided by the availability of downloadable dive computers.

- Ear examination: Look for signs of middle-ear barotrauma that could be associated with inner-ear damage. Round window rupture is not easy to differentiate from inner ear DCS.
- Neurologic examination: Access mental status, cognitive function, assessment of gait and walking, cranial nerves, sensory function, limbs strength, cerebellar function, and osteotendinous reflexes. Anal sphincter tone and bulbocavernosus reflex are essential to verify in patients with suspected spinal cord DCS and possible spinal shock. Sensory deficits are often patchy and may not follow the cortical distributions usually seen in patients with acute thrombotic cerebral stroke, or will not conform to dermatomes as seen in patients with traumatic spinal cord injury. Bubbles can cause vascular occlusions at many different locations in the nervous system. The neurologic examination can show very unusual distribution of deficits and can be confusing for the physician. Strange neurologic complaints or findings should not be dismissed as imaginary.
- Skin examination: Look for any rash or swelling.
- Articular examination: Joint examination is often unrevealing in pain-only DCS because there are usually no signs of joint inflammation and joint movement rarely alters the pain. It is commonly recounted that a relief of pain produced by the inflation of a sphygmomanometer cuff over a painful joint could support a diagnosis of musculoskeletal DCS over other strain conditions. This test has poor sensitivity and unknown specificity.[11]
- Abdominal examination: Evaluate for bladder distension that can be present with spinal cord DCS. If present, urinary catheterization will be mandatory.

DIAGNOSTIC WORK-UP

There are no specific diagnostic investigations that can establish the diagnosis of DCS. Research to identify useful diagnostic indicators is ongoing. The following considers some of the available avenues.

Hemoglobin-hematocrit might be useful. In some severe DCS, hemoconcentration can be seen, resulting from increased vascular permeability mediated by endothelial damage and kinin release.[12]

Chest radiographs are essential in the evaluation of the injured divers. Most importantly to exclude pneumothorax and pneumomediastinum in cases in which pulmonary barotrauma is suspected.[13] An untreated pneumothorax is a contraindication for recompression in a hyperbaric chamber. It may also be crucial to recognize and treat

a pneumothorax before any medical evacuation to the hyperbaric center. DCS might not kill the patient but a tension pneumothorax could easily produce a life threat during air transportation. Secondarily, chest radiographs may be diagnostic in cases in which the differential diagnosis includes drowning and immersion pulmonary edema.

Computerized tomography (CT) and MRI have been used to delineate cerebral and spinal cord lesions in patients suffering from neurologic DCS. Although these imaging techniques may detect such lesions, and despite MRI seeming to be more useful than CT,[14] both modalities are surprisingly insensitive and often fail to detect lesions in divers with obvious neurologic deficits.[15–17] MRI seems to have a much better sensitivity to show anomalies in spinal cord DCS than in central nervous system DCS.[18] MRI may also be helpful in predicting clinical outcome in divers with spinal cord DCS.[19] CT scanning might prove useful in excluding other causes of neurologic symptoms, such as subarachnoid hemorrhage or cerebrovascular accident. In obvious cases of neurologic DCS, it should be clearly stated that these imaging techniques are not the priority and they should not cause a delay to recompression therapy.

Limb radiographs have occasionally revealed evidence of gas in the soft tissue and periarticular spaces but the absence of such a finding does not exclude DCS. Limb radiographs have very little diagnostic value and, therefore, are not recommended as part of the routine evaluation for DCS.

Research is ongoing to identify diagnostic tests to confirm DCI. It has been demonstrated that coagulation activation can be present in DCI. A relationship has been found between the plasma D-dimers level and the presence of sequelae in neurologic DCS.[20] The accumulation of microparticles in the blood has also received attention as a possible indicator of decompression stress.[21,22] Although all such work is valid, there is no current clinical relevance.

DIFFERENTIAL DIAGNOSIS OF DECOMPRESSION ILLNESS

Some of the other conditions with similar symptom spectra include inner ear barotrauma; middle ear or maxillary sinus barotrauma; contaminated breathing gas (the effects of which can be concentrated when breathing under pressure); oxygen toxicity; musculoskeletal strains or trauma sustained before, during, or after a dive; seafood toxin ingestion; immersion pulmonary edema; water aspiration; and coincidental neurologic disorders, such as stroke.[7] Additional conditions to consider are hypoglycemia, thermal stress, and age-related conditions. Medical or event history can provide important insights. For example, symptoms of immersion pulmonary edema often develop at depth. This occurrence would rule out DCS, which can only develop during or following ascent.

TREATING DECOMPRESSION ILLNESS

There are several elements in DCI management: on-the-scene evaluation and first aid, transport, and definitive medical evaluation and treatment.

FIRST AID

High partial pressure oxygen is the primary first aid measure for DCI.[23,24] High oxygen concentration in the lungs will accelerate inert gas elimination. High oxygen partial pressure in the bloodstream can also alleviate ischemic insults produced by bubble blockages. Sustained oxygen delivery can reduce or even eliminate symptoms.

Continuous-flow oxygen systems, using nonrebreather or pocket masks, are frequently available in diving environments; however, such equipment delivers modest

oxygen fractions. Much higher fractions can be achieved for spontaneously breathing patients with demand masks.

First aid oxygen rebreather systems can provide the highest fractions with minimal gas use, especially helpful in remote settings with limited oxygen supplies.[25]

Chemical oxygen generating systems may be the only option available in some remote locations. Problematically, such devices typically provide extremely limited flow rates and volumes.[26]

Medical evaluation is advised even if a diver's symptoms improve or disappear with the administration of oxygen because subtle issues can be missed or signs and symptoms can return once oxygen delivery is stopped.

SPECIALIZED RESOURCES

Following patient stabilization, consultation with subject matter experts may be helpful for diagnosis and management plans. DAN may be able to provide consult support. DAN is a not-for-profit organization created to promote diving safety and support divers in need. Services include and emergency hotline (1-919-684-9111), evacuation logistics, nonemergency information, insurance, and education or training, including continuing medical education programs in diving and hyperbaric medicine.

HYPERBARIC OXYGEN THERAPY

The definitive treatment of DCI is hyperbaric oxygen therapy (HBOT), the delivery of oxygen at a partial pressure substantially higher than that achievable at normal atmospheric pressure.

Recompression in a hyperbaric chamber reduces the volume of bubbles. HBOT promotes the elimination of both bubbles and dissolved gas, increases delivery of oxygen to ischemic tissues, reduces tissue edema, reduces blood vessel permeability, counteracts the adherence of leukocytes to brain vessels, and partially blocks lipid peroxidation in reperfused tissues.

The HBOT regimen most commonly used to treat DCS is the US Navy Treatment Table (USN TT) 6.[27] The initial pressurization is to 2.8 ATA, equivalent to the pressure found at 18 m (60 ft) of seawater (**Fig. 4**). Patients breathe pure oxygen, with scheduled air breathing breaks to reduce the risk of central nervous system oxygen toxicity. The usual duration of the USN TT6 treatment is just under 5 hours but extensions can be added at both step pressures if warranted by the patient's response. The table is almost identical to the Royal Navy Treatment Table 62.

HBOT can be conducted in monoplace chambers, often acrylic tubes that hold just 1 patient (**Fig. 5**), or in multiplace chambers that accommodate 1 or more patients plus 1 or more tenders (**Fig. 6**). Multilock chambers allow patients, tenders, or equipment to be transferred into and out of or between chambers while treatment is ongoing.

TREATMENT EFFICACY

Although definitive data are limited, it seems that the best prognosis is achieved with rapid HBOT. This is logical for an acute ischemic event. Although there is an inverse relationship between delay to treatment and complete resolution of symptoms, the current data available have not established a maximum time after which recompression is ineffective. Many anecdotal reports describe clinical improvement in DCS cases treated many days after the onset, even with neurologic involvement. It could be reasonable to offer this therapy even up to several days after the first symptoms.

Treatment Table 6

1. Descent rate - 20 ft/min.
2. Ascent rate - Not to exceed 1 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
3. Time on oxygen begins on arrival at 60 feet.
4. If oxygen breathing must be interrupted because of CNS Oxygen Toxicity, allow 15 min after the reaction has entirely subsided and resume schedule at point of interruption (see paragraph 20-7.11.1.1).
5. Table 6 can be lengthened up to 2 additional 25-min periods at 60 feet (20 min on oxygen and 5 min on air), or up to 2 additional 75-min periods at 30 feet (15 min on air and 60 min on oxygen), or both.
6. Tender breathes 100 percent O_2 during the last 30 min. at 30 fsw and during ascent to the surface for an unmodified table or where there has been only a single extension at 30 or 60 feet. If there has been more than one extension, the O_2 breathing at 30 feet is increased to 60 min. If the tender had a hyperbaric exposure within the past 18 h an additional 60-min O_2 period is taken at 30 feet.

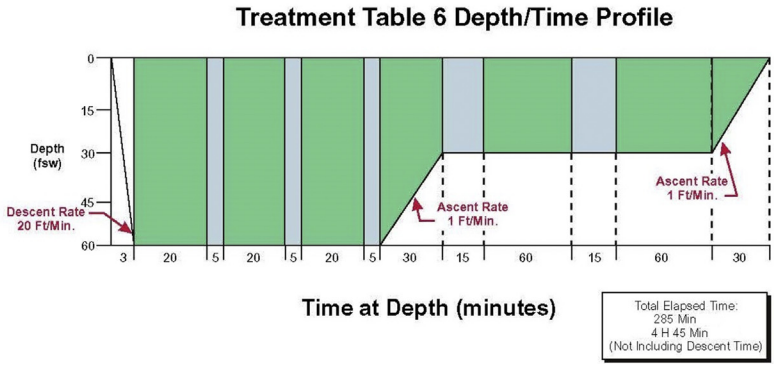

Fig. 4. The USN TT 6 is probably the most widely used protocol to treat DCS. (*From* US Navy Diving Manual, Volume 2, Revision 6. NAVSEA 0910-LP-106-0957. Naval Sea Systems Command: Washington, DC, 2008.)

The course of HBOT will vary according to the particulars of each case. Full resolution of DCS symptoms can often be achieved with 1 or sometimes multiple HBOT treatments. In some cases, resolution will be incomplete, even after many treatments. The normal clinical approach is to continue treatments until no further improvement is

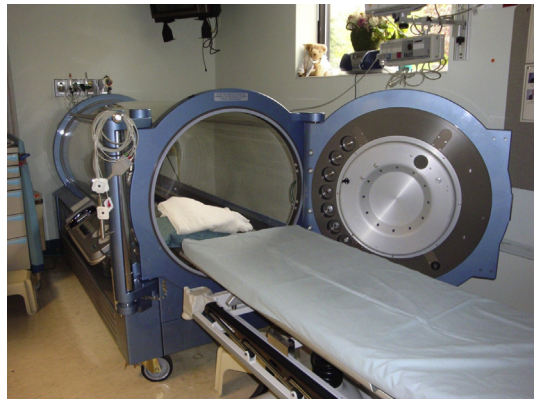

Fig. 5. A monoplace hyperbaric chamber holds a single patient, without any inside support personnel. (*Courtesy of* N.W. Pollock, PhD, Durham.)

Fig. 6. Critical care can be provided within properly equipped, multiplace, multilock hyperbaric chambers. (*Courtesy of* D. Buteau, MD, Quebec City.)

seen in the patient's symptoms. Modest residual symptoms will then often resolve slowly, after the treatment series is ended. Full resolution of symptoms can sometimes take months to achieve and in some instances may never be realized.

UNUSUAL CIRCUMSTANCES

Two of the rapidly growing subdisciplines in diving are freediving and technical diving. Breath-hold diving that was once used for casual snorkeling is now used for a wide range of activities from spearfishing to competitive freediving. Although the equipment requirement is minimal, the range of activity has advanced massively, with current record resting breath-hold times of 11 minutes and 35 seconds, and maximum excursion depths of 214 m (702 ft). The conventional wisdom that DCS cannot develop from breath-hold exposures is being challenged by a substantial number of anecdotal reports in which primarily transient neurologic symptoms have developed. It is important to determine the scope of activity of an individual rather than assuming that all breath-hold diving is benign.

Technical diving represents the other end of the equipment continuum, often using multiple different gas mixtures in different cylinders or, increasingly, closed-circuit rebreathers that dynamically maintain breathing gases at selected setpoints.[28,29] Rebreathers have dramatically expanded the range of recreational and scientific diving. Technical diving relies on decompression algorithms, many that have not been tested beyond the traditional recreational zone of less than 40 m. It is increasingly common to see recreational use in the 100 m range, with many pushing much deeper. The long runtimes associated with technical diving can produce prolonged exposure to high P_{O_2} and a complex patterns of gas breathing. The relatively extreme exposures can also produce decompression insults far beyond what is normally seen in traditional scuba diving. Despite the complexity of exposures and insult, standard HBOT typically produces good therapeutic outcomes and is not contraindicated.

ADJUNCTIVE THERAPIES

Fluid Administration

Fluid resuscitation is advocated for the treatment of divers with DCS. Isotonic fluids are preferable to hypotonic fluids to prevent an osmolar gradient that may contribute

to tissue edema and to prevent electrolyte imbalance. The goal is to maintain a good urinary output (1–2 mL/kg/h) for the patient with a urinary catheter, or clear urine for the patient who is voiding naturally.

Anticoagulants and Antiplatelet Agents

Inert gas bubbles induce platelet accumulation, adherence, and thrombus formation.[12,30] Consequently, a variety of antiplatelet agents, especially aspirin, have been extensively tried but without success. This failure is probably because bubble-induced platelet accumulation is not as rheologically important as the concurrent accumulation of leukocytes.[31] Aspirin is commonly administered to divers suffering from DCI in France.[32]

Animal models of DCS failed to demonstrate any beneficial therapeutic effect of heparin.[33] Heparin may also pose a significant risk as an anticoagulant, potentially inducing tissue hemorrhage in both spinal cord DCS and inner ear DCS. The opposing concern is the thromboembolic risk in bedridden patients with severe neurologic DCS. Administration of subcutaneous low-molecular-weight heparin for thromboembolic prophylaxis is recommended for those cases.

Corticosteroids

Corticosteroids have been used in the past for the treatment of neurologic DCS. The evidence was based mainly on case reports or retrospective studies in which numerous variables were simultaneously evaluated. It is unclear if corticosteroid administration has meaningful impact on the outcome. Evidence from studies on acute traumatic spinal cord injuries showing that high-dose corticosteroids may have limited benefit and can result in invasive infections and avascular necrosis must also be considered.[34–36] Finally, it should be noted that corticosteroids may predispose to central nervous system oxygen toxicity. Currently, the efficacy of corticosteroids in DCI treatment remains unproven.

Nonsteroidal Anti-inflammatory Drugs

A randomized trial examining adjunctive administration of the nonsteroidal anti-inflammatory drug (NSAID), tenoxicam, to divers suffering with DCI showed no difference on the residual symptoms at the completion of HBOT; however, there was a significant reduction in the number of treatments required and no evidence of complications with this adjunctive treatment.[37] One other NSAID for which there is some evidence is indomethacin but only in combination with prostaglandin PgI_2 and heparin.[38,39] NSAIDs are not recommended in the initial first-line treatment of DCS because they can confound the clinical evaluation by masking the pain. They could have some utility for residual pain after the decision has been made to stop HBOT.

Lidocaine

Lidocaine at therapeutic levels has been shown in animal models of cerebral AGE to have the following effects: reduction of intracranial pressure, preservation of cerebral blood flow, reduction of brain edema, preservation of neuroelectrical function, and reduction in infarct size. Possible mechanisms for this cerebral protection include modulation of leukocyte activity, reduction of ischemic excitotoxin release (eg, glutamate), reduction in cerebral metabolic rate and deceleration of ischemic transmembrane ion shifts. A review of the literature[40] identified 1 randomized, double-blind study that demonstrated improved neuropsychological outcomes in cardiac surgery patients receiving lidocaine. Clinical evidence of efficacy in DCI is limited to anecdotal reports. Some case studies of patients suffering from neurologic DCS have shown

recovery even when the patient was recompressed with significant delay or had been refractory to recompression therapy.[41–43] A first retrospective cohort study has been published and found no effect of lidocaine on outcome.[44] Lidocaine is not considered part of the routine therapy for DCS but may be considered as an adjunctive agent for severe cerebral DCS or cerebral AGE.

MEDICAL EVACUATION TO THE HYPERBARIC CHAMBER

If aircraft medical evacuation is used to transfer the patient to the hyperbaric chamber, care should be taken to expose the patient to the least possible reduction in atmospheric pressure. Depressurization would cause further inert gas bubbles expansion and can worsen the condition. Recommendations include flying as low as safely possible for unpressurized aircraft or helicopter (preferably lower than 300 m [1000 ft]) or pressurizing the aircraft to sea level pressure, if possible.

Portable hyperbaric chambers (or hyperbaric stretchers) are sometimes available for recompression treatment in remote locations or for transport (**Figs. 7** and **8**). These devices are generally made of Kevlar and can be pressurized up to 3 ATA. Pressurized air and oxygen are provided via portable gas cylinders. The use of these devices is most appropriate for stable patients with mild to moderate symptoms. They do introduce serious limitations in monitoring and access of patients.

ALTERNATIVE TREATMENT TECHNIQUE: IN-WATER RECOMPRESSION

In-water recompression may be an alternative to chamber recompression in remote locations, if there is neither a nearby chamber nor the means to quickly transport the patient to a chamber elsewhere. The technique involves bringing the diver underwater again, to drive gas bubbles back into solution to reduce symptoms and then slowly decompress in a way that maintains an orderly elimination of the excess gas.

Although in-water recompression is simple in concept, it is practical only with a substantial amount of planning, support, equipment, and personnel; appropriate water conditions; and suitable patient status. Critical challenges can arise due to changes in the patient's consciousness, oxygen toxicity, gas supply, and even thermal stress. An unsuccessful in-water recompression may leave the patient in worse shape than had the attempt not been made. The medical and research communities are divided

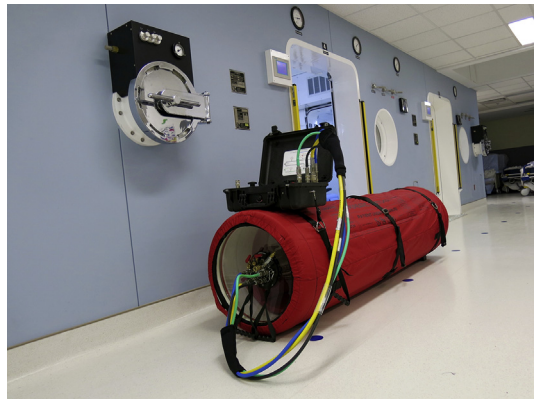

Fig. 7. A hyperbaric stretcher set up in front of a multiplace, multilock hyperbaric chamber. (*Courtesy of* D. Buteau, MD, Quebec City.)

Fig. 8. A hyperbaric stretcher being loaded onto a helicopter. (*Courtesy of* D. Buteau, MD, Quebec City.)

on the utility of in-water recompression. It is beyond the scope of this article to consider all of the relevant factors but it is fair to say that there are probably more situations when in-water recompression should not be undertaken than situations when it would be a reasonable choice.

As a general rule, a diver who develops symptoms consistent with DCS should be removed from the water and receive oxygen first aid on the surface, even if there is likely to be a delay before definitive medical care can be reached.

SUMMARY

DCI represents a relatively rare affliction but a major concern for divers because of the potential severity of the insult and risk of sequelae. Decompression risk is primarily determined by the pressure-time profile and breathing gas but it is also influenced by the timing, intensity, and nature of thermal stressors during a dive, and by individual predispositions.

Signs and symptoms can vary substantially, causing insult to many systems. They frequently involve much more than the classic description of joint pain, with neurologic symptoms increasingly recognized. Neurologic examination findings can be confusing. History and physical examination are critical to evaluation. Symptoms will not develop during the bottom phase of a dive, before decompression. Symptoms of AGE typically present soon after surfacing, normally within 10 minutes. Symptoms of DCS normally appear within 6 hours after surfacing but they may not become apparent for more than 24 hours. Many other medical conditions need to be considered in the differential diagnosis.

High-concentration oxygen is the best first aid for both DCS and AGE. HBOT is the normal definitive medical treatment. The most commonly used and highly effective hyperbaric treatment protocol is the USN TT 6. The search for adjunctive therapies continues but none have been convincingly supported. Early consultation with diving medicine specialists is recommended.

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