10 Decompression Sickness

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Early recompression with HBO is a recommended treatment for decompression sickness. This chapter looks at:

Introduction

Decompression sickness (DCS) is one form of dysbarism, which is a general term applied to all pathological changes secondary to altered environmental pressure. Other forms are pulmonary barotrauma and also aseptic bone necrosis, which is likely to be a form of decompression sickness. DCS is caused by gas phase formed by a sufficiently rapid reduction of environmental pressure to cause supersaturation of the gases dissolved in the tissues. The principle component is most usually nitrogen, but when helium and oxygen mixtures are used it is helium. DCS is also described by the terms, caisson disease, "the bends" (joint pains), "the chokes" (pulmonary symptoms), the "staggers" (vestibular symptoms) and "hits" (spinal cord symptoms). DCS occurs in divers and also in those who work in compressed-air as in caissons and tunnels. It can also result from a reduction of normal barometric pressure, such as in a hypobaric chamber, and in aircraft at altitudes in excess of 5000 meters even when oxygen is breathed. It complicates flight in some high altitude military aircraft and may occur when astronauts don suits for undertaking extravehicular activity. Bubble formation may also be a component of altitude sickness in climbers making a rapid ascent. At sea level almost 1 liter of nitrogen is dissolved in the body. A little less than one-half of this is dissolved in water and a little more than one-half in the fat, which constitutes only 15% of the normal male body – nitrogen is five times more soluble in fat than in water. In diving the additional amount of nitrogen that dissolves in the body depends upon the depth and the duration of a dive. For steady state conditions the volume of nitrogen that is liberated returning from 10 m to normal barometric pressure is 2 liters. A helium and oxygen exposure to the same conditions would result in only 1 liter of gas dissolved with the difference being mainly due to the lower solubility of the gas in fat. However on resumption of air breathing at 1 ATA the elimination of helium is very rapid. To achieve steady state at a particular pressure – often known as saturation – requires many hours and the time required in greater for nitrogen than for helium. The formulation of decompression tables is generally based on methods introduced by Haldane from his observation that decompression sickness is rare when the absolute pressure is halved. However he warned against the extrapolation of this principle to pressures above 6 ATA. Figure 10.1 shows the approximate half-lives of nitrogen in various tissues. However the demonstration of gas formation in tissues after a decompression halving the absolute pressure indicates that this method is empirical and can only be used as a guide in decompression table formulation. After achieving steady state conditions in air diving the central nervous system has a high concentration of nitrogen because of the high solubility of the gas in lipids. Using helium the amount dissolved under equivalent steady state conditions

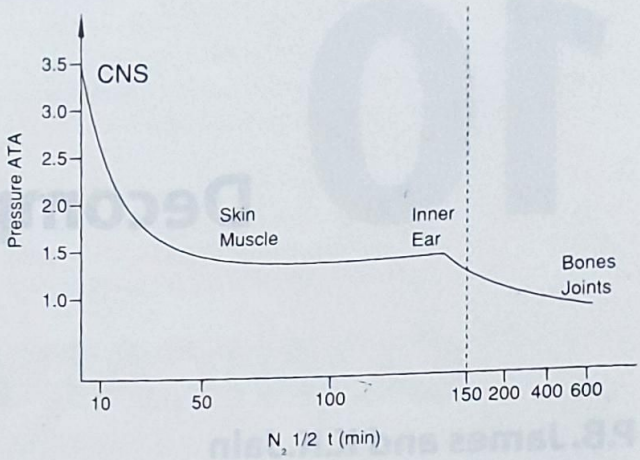

Figure 10.1
Half-life of nitrogen in various tissues.

is much less because of the lower solubility of the gas in fat. Commercial diving expanded dramatically with the exploration for offshore oil and gas and the experience gained has influenced practice in both military and amateur diving. For depths in excess of 50 msw "saturation" techniques have been developed where divers live at constant pressure in a helium and oxygen environment (heliox), using a bell to transfer to the water. Attempts to use nitrox for saturation dives have not been commercially successful. Operational dives have been undertaken to 450 msw using heliox and experimental dives to 523 msw, using mixtures of hydrogen, helium and oxygen. The inclusion of hydrogen reduces gas density and the work of breathing and also ameliorates the effects of the high pressure on nervous system function. These symptoms are known as the high pressure nervous syndrome (see Chapter 3).

Recently several physicians have proposed a new term for describing disorders resulting from decompression: decompression illness (DCI) which is proposed to encompass disorders previously known as DCS and AGE (arterial gas embolism). The reason given for the new proposal is that the etiology of decompression disorders is difficult to define and that DCS and AGE are difficult to separate from each other. This proposal is not widely accepted and we will be using the traditional terms: DCS and arterial gas embolism (see Chapter 11).

Pathophysiology

Bubble Formation

The elimination of the excess "inert" gas taken up during a dive ultimately depends upon the transfer of gas from blood to the respired gas in the lungs. All decompression tables assume that blood passing through the lungs is

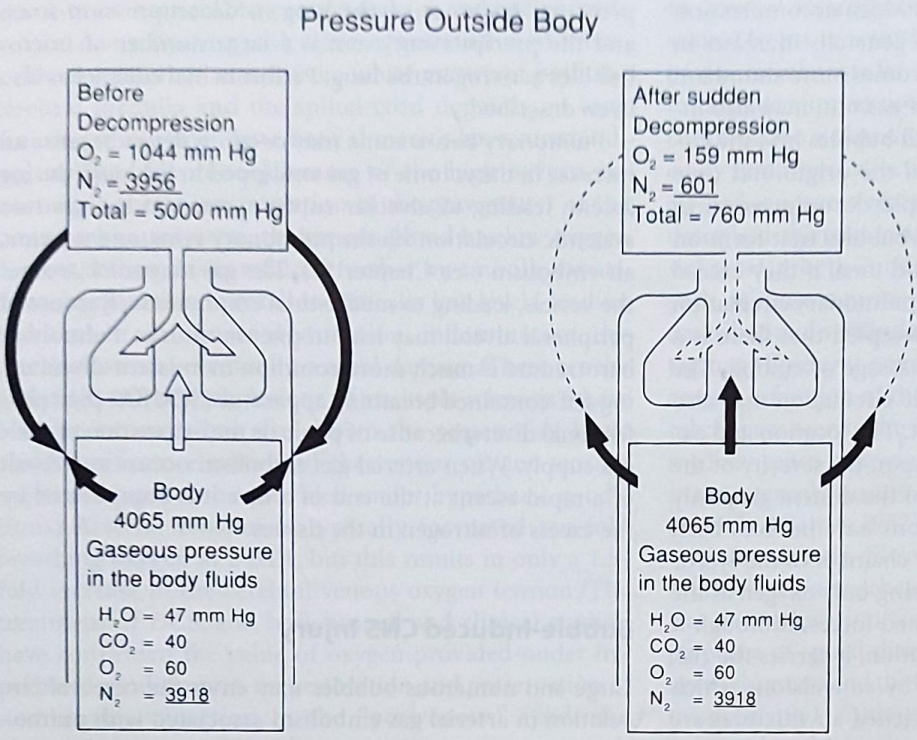

Figure 10.2
Bubble formation after decompression due to great excess of intrabody pressure shown at right.

equilibrated with the partial pressure of the gas being respired, but because of ventilation/perfusion mismatch in the lung this cannot be the case and some supersaturated blood must achieve the systemic circulation on decompression. Decompression sickness can follow repeated breathhold dives because the elevation of the partial pressure of the nitrogen in the air compressed in the lungs is reflected in an increase in the plasma and tissue nitrogen content. Cerebral symptoms have been associated with multiple breathhold dives. Decompression beyond the unsaturation associated with the metabolic use of oxygen (the oxygen window) produces supersaturation and risks the emergence of dissolved gas from solution. The formation of gas phase can be imaged in tissue planes in, for example, muscle, using ultrasound. The principle component is the diluent gas present, but oxygen, carbon dioxide and water vapor also contribute to the gas volume. Gas formation is believed to depend on the presence of gas micronuclei, which are very small quantities of undissolved gas and the formation of gas bubbles may occur very rapidly. It is universally agreed that the formation of gas is the initial event in the etiology of DCS. The principles underlying bubble formation are shown in Figure 10.2. There is considerable evidence that gas formation in the tight connective tissue of tendon is responsible for the classical joint pain of the "bends". Investigations using radiographs in aviators decompressed to altitude has demonstrated gas phase in the ligaments and tendons of the knee and these observations are relevant to those seen in decompression after exposure to hyperbaric environments. Sequential perfusion has been observed in connective tissue and this intermittent perfusion is probably a major factor in gas formation in this and other tissues, because a tissue will take up gas when it is perfused, but release of gas will be limited if the microcirculation closes during decompression. The gas exchange will then be diffusion, not perfusion. Also, because the oxygen contained in the area of tissue will be metabolized, there may be a reduction in the inherent unsaturation as more nitrogen is absorbed.

Intravascular bubbles are often detectable in the pulmonary artery during or after decompression. The timing is dependent on the nature of the dive. For example, circulating bubbles may be detected during the decompression from heliox saturation dives, but in air diving they are generally only detectable after decompression has been completed. The principles underlying bubble formation are shown in Figure 10.2.

Electron microscopy studies of human tissues from fatal cases of DCS have shown that each bubble is covered with an osmiophilic, nonhomogeneous coat of a flocculent material that is associated with an electrokinetic zonal activity. This surface coat reduces the rate of nitrogen elimination via the blood-lung barrier when bubbles are trapped in the pulmonary capillaries. Bubbles induce changes in vascular permeability and in severe decompression sickness this may precipitate hypovolemic shock and a reversible blood sludging. In addition to formation of bubbles in tissues, humoral agents may be released from tissues secondary to trauma caused by expanding gas. Intra-arterial bubble for-

mation occurs only if there is a very sudden decompression from a high pressure exposure. DCS generally increases in severity as the free gas in the body becomes more abundant. Exercise increases the elimination of gas but, may also increase the release of gas in tissues and bubbles into the circulation. Lynch *et al* (1985) studied the origin and time course of gas bubbles following rapid decompression in hamsters. Their data indicated that bubbles first form on the venous side of the circulation and then, if they exceed a certain number, move through the pulmonary circulation into the systemic circulation. It is accepted that there is a threshold for the transpulmonary passage of emboli. The transfer of bubbles into the systemic circulation may also occur through an atrial septal defect. The location and extent of bubble formation depends upon the severity of the supersaturation and the solubility of the diluent gas. Only in very severe experimental situations have bubbles been found intracellularly, in the anterior chamber of the eye, or in the cerebrospinal fluid. Diving using only oxygen as the respired gas is employed in the armed forces. Although it is not associated with bubble formation, it carries the risk of acute oxygen toxicity manifest by convulsions which may lead to drowning. Oxygen enriched air mixtures are used to reduce the risk of decompression sickness or extend bottom time and are becoming popular in amateur diving. They do not eliminate the risk of DCS and, again, because there is a very real risk of convulsions from oxygen toxicity, a full face mask or helmet should be used.

Pulmonary Changes

The first attempt to detect bubbles using ultrasound found them present in the inferior vena cava in a pig during decompression at 4 ATA after an exposure to compressed air at a pressure of 6 ATA for an hour (Gillis *et al* 1968). Human studies have used transcutaneous ultrasonic detectors which are much less sensitive than implanted devices. Bubbles can be detected in the pulmonary artery in the majority of divers after significant dives, but they generally do not produce symptoms. Experimental studies have shown that venous bubbles can cross the lungs of anesthetized dogs when driving pressures are high enough to overcome the normal filtering function of the lungs. Bubbles trapped in the lung may also cross the pulmonary circulation as a result of the reduction in their size on compression. Before decompression tables were formulated, pulmonary decompression sickness – the "chokes" – often proved fatal and the events have been followed in experimental animals. The pulmonary changes are accompanied by hypoxemia, pulmonary hypertension, and respiratory distress. These features are shared by other microembolic syndromes as, for example, fat embolism, and are examples of the (adult) respiratory distress syndrome. Noncardiac pulmonary edema has been found to be the principal response of the lung to decompression stress and the precipitating event is a large number of microbubbles arriving in the lung. Peribronchial edema has also been described.

Pulmonary barotrauma may occur in divers because an increase in the volume of gas entrapped in the lungs during ascent, leading to alveolar rupture, entry of the gas into systemic circulation via the pulmonary veins, and systemic air embolism (see Chapter 11). The gas may track around the vessels, leading to mediastinal emphysema. Rupture of peripheral alveoli may lead to pneumothorax. Pulmonary barotrauma is much more common in amateur divers using self-contained breathing apparatus (SCUBA) than professional divers, because of panic or the exhaustion of their gas supply. When arterial gas embolism occurs as a result of a rapid ascent at the end of a dive it is complicated by the excess of nitrogen in the tissues.

Bubble-Induced CNS Injury

Large and numerous bubbles may enter the cerebral circulation in arterial gas embolism associated with pulmonary barotrauma, and cause gross obstruction to flow and ischemia. However, in contrast, bubbles formed on decompression are small, generally measuring about 25 microns on the surface. As with solid micro-emboli, the diameter of microbubbles is a critical factor in their behavior in the circulation, but the size of gaseous emboli depends on the absolute pressure. Ischemia is a major factor in bubble-induced CNS injury in decompression sickness. In experiments using labeled granulocytes, ischemia activates the chemotaxic process at an early stage and that granulocytes may be involved in CNS injury. Ischemia is conventionally associated with vascular occlusion, but gas embolism also causes endothelial damage and in the CNS this involves opening of the blood-brain barrier and edema. This has also been produced by the transit of microbubbles without initial being associated with ischemia (Hills & James 1991). Opening the blood-brain barrier causes the extravasation of plasma proteins which triggers the complement cascade. This induces the inflammatory cascade and ischemia only develops when focal edema causes compression of the microcirculation. Pearson *et al* (1992) observed bubbles in the cerebral circulation which preceded changes in evoked potentials in an experimental model. Blood complement also induces granulocyte clumping in DCS. The result of these interactions between blood and the damaged tissues may well be a major determinant of the extent of neuronal recovery following focal blood brain barrier disturbance and ischemia in the CNS. Fat embolism may also occur on decompression sickness and even cause death from an acute disseminated encephalitis. Fat emboli being fluid also cross the lung filter and cause cerebral edema because of damage to the

blood-brain barrier. The protein leakage and edema leads to focal demyelination with relative preservation of axons.

The nutrition of areas of the white matter of both the cerebral medulla and the spinal cord depends on long draining veins which have been shown to have surrounding capillary free zones. Because of the high oxygen extraction in the microcirculation of the gray matter of the central nervous system, the venous blood has low oxygen content. When this is reduced further by embolic events, tissue oxygenation may fall to critically low levels, leading to blood-brain barrier dysfunction, inflammation, demyelination and eventually, axonal damage. These are the hallmarks of the early lesions of multiple sclerosis where MR spectroscopy has also shown the presence of lactic acid. Significant elevation of the venous oxygen tension requires oxygen to be provided under hyperbaric conditions. Arterial tension is typically increased ten-fold breathing oxygen at 2 ATA, but this results in only a 1.5-fold increase in the cerebral venous oxygen tension. The treatment of DCS, and both animal and clinical studies, have confirmed the value of oxygen provided under hyperbaric conditions in the restoration and preservation of neurological function in the "perivenous" syndrome (James 2007).

An alternative mechanism by which bubbles may generate CNS injury is their nucleation within the white matter. Bubbles are seen in the myelin sheaths on the rapid decompression of experimental animals from high pressures. Autochthonous (formed in situ) bubbles have been shown to traumatize neurons at the site of nucleation and compress adjacent ones and this is one mechanism which can explain the sudden onset of symptoms. However the conditions used in these experiments are much more extreme than in most cases of human decompression sickness. Because of the need for a reliable animal model and to avoid the unpredictability of decompression sickness which characterizes human dives very extreme conditions and often double exposures have been used.

The most obvious disability from neurological (Type 2) DCS in diving is paraplegia due to the involvement of the spinal cord, but this is a rare complication of hypobaric decompression, indicating the importance of bubble size and tissue gas loading. Most divers with spinal cord symptoms when questioned actually admit to symptoms which indicate disturbed brain function. Three mechanisms have been postulated to explain the pathophysiology of spinal cord lesion:

- Arterial bubble embolism
- Epidural venous obstruction leading to infarction
- Autochthonous bubbles

The paramount difficulty is the attribution of spinal cord decompression sickness to arterial embolism has been the failure to recognize a natural disease of the spinal cord due to arterial microembolism. However this has been answered by the recognition that multiple sclerosis (MS) may be due to subacute fat embolism and retinal changes occur in both decompression sickness and MS. In MS as in decompression sickness the clinical presentation is dominated by symptoms affecting the spinal cord which can be described as a transverse myelitis. Late deterioration has been described thirteen years after spinal cord decompression sickness. In both conditions focal cranial nerve problems, such as optic neuritis and oculomotor palsies have been described and vestibular damage may leave permanent nystagmus. Epidural venous obstruction generally leads to central infarction of the cord and not the characteristic focal changes seen in the decompression sickness. Gas or edema may cause ischemia in the cord because of an increase in the internal pressure due to non-elasticity of the pia mater (Hills & James 1982).

Unfortunately, fibrocartlilaginous embolism has been overlooked in the debate about the mechanism of neurological decompression sickness. Material from the nucleus pulposus of spinal disks can cause embolic damage to the nervous system and the first case was described in 1961 (Naiman *et al* 1961). Although retrograde venous flow has been suggested as the mechanism, the post-mortem finding of a 200 micron fibrocartilage embolus in the middle cerebral artery of a 17-year-old girl (Toro-Gonzalez *et al* 1993) has demonstrated beyond doubt that system embolization does occur. As in decompression sickness material may gain access to the systemic circulation by transpulmonary passage, or through an atrial septal defect. The girl, who had collapsed while playing basketball, died of myocardial infarction, and emboli were found in the coronary arteries. The size range of emboli, from 20 to 200 microns, indicates that the microcirculation of the lung may be sizing the material. The mechanism has been described in other mammals, where it is now regarded as a relatively common cause of neurological symptoms. The cases described include an 11-day-old lamb (Jeffrey & Wells 1986).

Changes visible with both light and electron microscopes are seen in the spinal cords of animals subjected to severe experimental DCS. The finding of widened myelin sheaths showing a banded pattern of myelin disruption may be compatible with autochonous gas, but similar patterns are seen in experimental allergic encephalomyelitis due to edema.

Divers may be at risk of long-term CNS damage from non-symptomatic hyperbaric exposure. The effect of severe, controlled hyperbaric exposure was investigated on goats exposed to various dive profiles over a period of 5 years, with some experiencing DCS (Blogg *et al* 2004). MRI was done and the animals were then sacrificed for neuropathological examination the brain and spinal cord. No significant correlation was found between age, years diving, DCS or exposure to pressure with MRI-detectable lesions in the brain, or with neuropathological lesions in

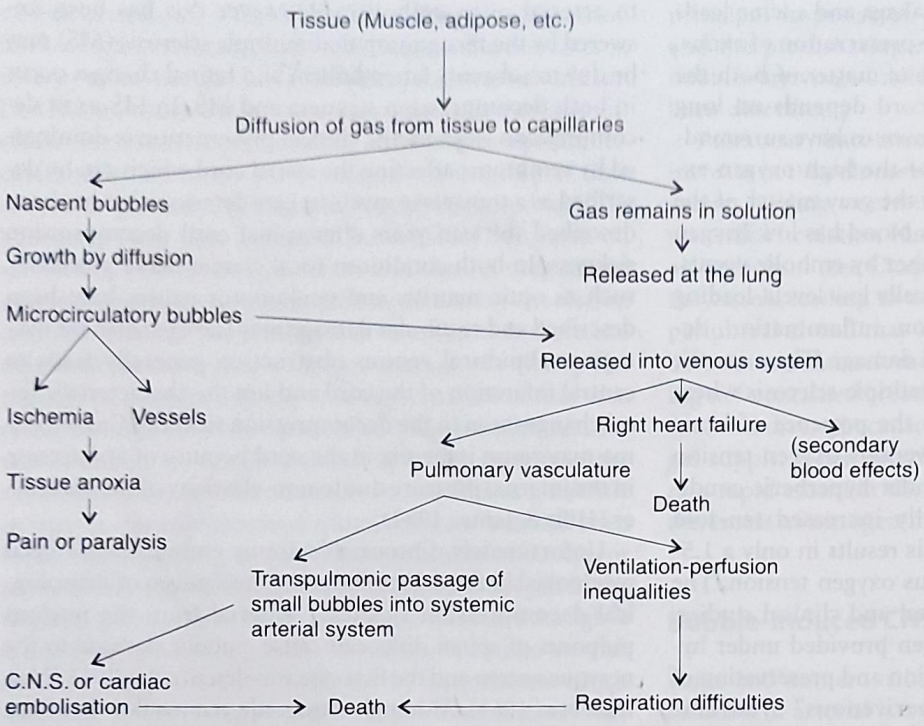

Figure 10.3
Physiological sequelae in decompression sickness.

the brain or spinal cord. However, spinal scarring was noted in animals that had suffered from spinal DCS.

Changes in Blood

Even asymptomatic decompression of sufficient severity can be associated with a reduction of the number of circulating platelets by one-third during a 24-h period following a severe dive. A smooth muscle-activating factor released during decompression may potentiate other bioactive amines, such as bradykinin, serotonin, and histamine, which are known to be involved in shock caused by rapid decompression. Hyperagglutinability of the platelets is important in the pathogenesis of DCS. This phenomenon may be based on production of metabolites of arichodonic acid and prostaglandin-like compounds.

Adhesion of platelets to the surfaces of bubbles and formation of platelet aggregates have been shown by scanning electron microscopy. Other platelet agonists like ADP, epinephrine, and serotonin, which may be present in vivo, accelerate this interaction, and the platelet antagonists have been shown to depress platelet aggregation. These factors may delay gas resolution on recompression.

Dysbaric Osteonecrosis

Dysbaric osteonecrosis (DON) has been reported in humans and experimental animals after a single hyperbaric air exposure with inadequate decompression. It is usually considered to be the result of gas bubbles entering the end arteries in the bone, and is seen most commonly in compressed-air workers.

Jones *et al* (1993) hypothesize that DON does not result from primary embolic or compressive effects of the nitrogen bubbles on the osseous vasculature. These authors report the presence of gas bubbles in the fatty marrow of the femoral and humeral heads and lipid and platelet aggregates were found on the surface of marrow bubbles. Fibrin platelet thrombi were found in systemic vessels, suggesting that injured marrow adipocytes can release liquid fat, and this fat embolism causes the release of thromboplastin, and other vasoactive substances that can trigger systemic intravascular coagulation and DON.

Role of Free Radicals

There is increasing awareness of the role of oxygen-derived free-radicals in reperfusion injury. However occlusion of flow is more a feature of air embolism rather than decompression sickness, the latter being associated with increased vascular permeability and inflammation. After the first phase of DCS caused by the mechanical action of bubbles, the symptoms in the second phase may result from oxygen-free radicals associated with ischemia and hypoxia. It has been recognized since the introduction of the minimal recompression tables using 100% oxygen at 2.8 ATA (US Navy Manual 1970) that these procedures could be associated

with worsening and oxygen toxicity with free radical formation may be a component in this deterioration. However, it is universally recognized that recompression treatment should be carried out as quickly as possible and that recompression with the additional use of heparin, superoxide dismutase and catalase does not improve the outcome of severe DCS in experimental animals.

A schematic of the pathogenesis of the forms of DCS is shown in Figure 10.3.

Clinical Features

Decompression Sickness in Diving

Haldane (1907) classified DCS into three categories: Type I, joint pain; Type II, systemic symptoms or signs, caused by the involvement of the CNS or the cardiopulmonary systems; and Type III, characterized by convulsions and death. The first two parts of the classification are still the accepted standard internationally.

The clinical features of DCS are shown in Table 10.1. DCS is a disease that manifests itself in a variety of organ systems. However studies of both compressed air workers and divers have shown that Type I DCS is the most common presentation, but the nature of the dive is important. For example, mild joint pains are not unusual during heliox saturation decompressions but Type II symptoms are extremely rare. In surface decompression procedures Type II DCS is more common and on deep dives presents more frequently than Type 1 symptoms. It is obvious that joint pain is easier for a diver to recognize than symptoms affecting the nervous system.

The majority of divers who have undertaken surface-orientated dives experience symptoms within 3 h of surfacing, although the onset of symptoms may be delayed for as long as 35 h. In air diving nearly one-half of the cerebral cases become apparent within 3 min of surfacing, and a similar proportion of spinal cases also become apparent within 3 min of surfacing.

The most serious sequelae are those involving the CNS: neurologic manifestations of DCS comprise symptoms from the cerebral hemispheres, the spinal cord, as well as vestibular disturbances (Jain 2009i). The most common area involved is the lower thoracic segments spinal cord, but the level can vary from C4 to L1. In air diving with in-water decompression CNS involvement occurs in 25% of DCS cases. Late deterioration of spinal cord function has been described thirteen years after an episode of decompression sickness (Dyer & Millac 1996). Other neurologic syndromes may also occur. Some divers develop evidence of acute cerebral hemisphere dysfunction, such as hemiparesis, aphasia, or hemianopsia. Memory loss, convulsions, and even coma can occur.

Table 10.1
Signs and Symptoms of Decompression Sickness (DCS)

Type 1 DCS: Limb and joint pains (bends), skin rash
Type II DCS
Neurological
1. Cerebral
   - visual disturbances
   - aphasia
   - hemiplegia
   - memory loss
   - convulsions
   - coma
2. Spinal ("hit")
   - sensory disturbances of extremities: paresthesias, numbness
   - weakness, difficulty in walking
   - bladder dysfunction
   - paraplegia or quadriplegia
3. Vestibular disturbances ("staggers")
   - nystagmus
   - vertigo

Pulmonary ("chokes")
1. Dyspnea
2. Hyperventilation
3. Chest pain
4. Acute respiratory distress syndrome (ARDS)

Cardiac
1. Tachycardia
2. Cardiac arrhythmias

Repetitive breath-hold diving can lead to accumulation of nitrogen in blood and tissues, which may give rise to DCS. MRI in four professional Japanese breath-hold divers (Ama) with histories of diving accidents showed cerebral infarcts localized in the watershed areas of the brain (Kohshi *et al* 2005). A survey conducted on their island revealed that many Ama divers had experienced stroke-like events. A clinical feature of DCS in breath-hold diving is that the damage is limited to the brain. Although the mechanisms of brain damage in breath-hold diving are unclear, nitrogen bubbles passing through the lungs or the heart so as to become arterialized are most likely to be the causative factor.

Cardiac arrhythmias (premature ventricular contractions) have been reported in DCS. Pulmonary symptoms occur in about 2% of cases. Noncardiogenic pulmonary edema is an uncommon manifestation of Type II DCS. It usually occurs within 6 h of a dive and is believed to be caused by microbubbles in the pulmonary circulation. Shock is rare in DCS but may follow severe dives.

Altitude Decompression Sickness

Altitude DCS is usually seen in aviators at an altitude of 6098 m (20,000 ft), but it may occur at lower altitudes in

those with risk factors for DCS. A case of DCS with rapid decompression at 2439 m (8000 ft) and a good response to recompression therapy has been reported by Rudge (1990a). A review of 133 cases from the United States Air Force by Wirjosemito *et al* (1989) showed that the most common manifestations were joint pain (43.6%), headaches (42.1%), visual disturbances (30.1%), limb paresthesias (27.8%), and mental confusion (24.8%). Spinal cord involvement, chokes, and unconsciousness were rare. HBO treatment was successful in 97.7% of the cases, and residual deficits were noted in only 2.3% of the cases. Altitude-related DCS can present with a wide variety of symptoms in the same patients, such as nausea, headache, fatigue, and respiratory difficulty, which can be misdiagnosed as viral illness. Rudge (1991) reported two such patients in whom the symptoms resolved following recompression with HBO, thus confirming the diagnosis of DCS.

Cerebral hypoxia is usually not a feature of diving-related DCS, but explains the greater severity of the cerebral presentations at altitude DCS. Sheffield and Davis (1976) used HBO in the treatment of a pilot who underwent rapid decompression from 753 hPa (2348 m altitude) to 148 hPa. The pilot lost consciousness in 5 to 8 s. Supplemental oxygen was given after a delay of 6 to 8 min. On the ground the pilot was blind and disoriented and remained so for the next 6.5 h until HBO therapy was started. The pilot eventually recovered with no neurological deficits. Davis *et al* (1977) reviewed 145 cases of altitude DCS and recommended immediate compression to 2.8 ATA and a series of intermittent oxygen-breathing and air-breathing periods during the subsequent slow decompression. The US Air Force has modified the US Navy procedure Table 6.

Optic atrophy has been reported in a parachutist after repeated hypobaric exposures (Butler 1991) and vision improved with recompression and HBO therapy.

After a review of 233 cases treated at the USAF School of Aerospace Medicine, Rudge and Shafer (1991) concluded that, as in diving, the treatment of altitude DCS with compression therapy is most useful when it is begun as early as possible. The greater the delay in treatment, the longer the symptoms of DCS persist and the greater the rate of residual symptoms.

Another manifestation at altitude is acute mountain sickness (AMS), which usually occurs in individuals ascending above 3000 m without adequate acclimatization. The clinical signs and symptoms of AMS include headache, nausea, irritability, insomnia, dizziness, and vomiting. In some individuals AMS may proceed to cerebral edema and/or pulmonary edema. Cerebral edema is considered to be secondary to hypoxic cerebral vasodilation and elevated capillary hydrostatic pressure, but it cannot be ruled out that bubbles may be a contributing factor. These events elevate peripheral sympathetic activity that may act in concert with pulmonary capillary stress failure to cause pulmonary edema but again pulmonary entrapment of bubbles offers an alternative explanation. Oxygen breathing and descent from altitude are proven effective measures for AMS and a portable hyperbaric chamber has been found to be useful. During acute ascent in the Alps, an early 3-hr pressurization of unacclimatized subjects using air was shown to slightly delay the onset of AMS, but did not prevent it or attenuate its severity on presentation (Kayser *et al* 1993).

Extravehicular activity during missions on space stations and to establish a permanent presence on the Moon carry a risk of DCS because of the reduction of pressure required to use space suits. Loss of pressure from a space suit would be rapidly fatal unless immediate recompression is carried out and it has been suggested that space stations and lunar missions should include a hyperbaric treatment capability.

Ultrasonic Detection of Bubbles

It is of course not possible in conventional diving to use ultrasonic monitoring, but it has been used to develop and monitor decompression procedures. Gillis *et al* (1968) were the first to describe bubbles on decompression experimentally. Spencer (1976) used ultrasound to detect venous gas emboli in divers and stated that in these experiments no bends developed prior to detection of bubbles over the precordium. However Powell *et al* (1983) noted that during decompression with elevated oxygen, precordially determined bubbles at depth were predictive of limb pain in only 50% of cases. Seventy percent of the divers encountered bends without detectable bubbles. The amplitude of the Doppler-detected pulmonary artery flow sound, however, increased, and it was suggested that this may have indicated the presence of numerous microbubbles.

Pulse-echo ultrasound imaging techniques have been used to study the formation of bubbles. These can monitor the extent of bubble formation during decompression with a view to predicting symptoms. The results of such studies confirm that:

- A threshold of supersaturation for bubble formation exists
- The earliest bubbles are intravascular
- There is usually an accumulation of stationary bubbles before precordial bubbles are detected and symptoms of Type 2 DCS develop.

Diagnosis

DCS is rare unless the patient has been exposed to pressures greater than 2 ATA although cases have been described from long exposures to lesser pressures. The diagnosis can be made on the basis of history and clinical features and it is essential to stress that if a significant dive has been under-

taken then the presumption must be made that the symptoms are due to decompression sickness not natural disease.

The differential diagnosis of neurological DCS, particularly in atypical cases, should include multiple sclerosis. A case is reported of clinically definite multiple sclerosis presenting as neurological decompression sickness 3 weeks following SCUBA dive (Jan & Jankosky 2003). There was no improvement with HBO treatment, and further evaluation led to the diagnosis of multiple sclerosis.

Recompression has been advocated as a definitive test for DCS, and providing it is undertaken immediately it is a valuable guide. However immediate recompression is usually only possible in commercial diving operations where in most cases a chamber is on site. In Type 1 DCS a small increase in pressure may resolve the pain and on decompression it may recur in the same site suggesting a mechanical origin. In general, the higher the pressure of the onset of pain the greater the pressure increase that is required for its resolution indicating a relationship to gas volume. With CNS symptoms, as bubbles induce tissue hypoxia by interfering with blood flow it may be difficult to determine if improvement is due to a reduction in gas volume with pressure, or the resolution of edema and hypoxia from the use of a high partial pressure of oxygen.

In general laboratory tests are not helpful in decompression sickness, but several have been used in experimental animal studies and some human laboratory dives.

Fibrinogen Degradation Products Test

The fibrinogen degradation products test reflects disseminated intravascular coagulation or agglutination. The diagnostic value of this test in the absence of clinical information is questionable.

Bone Scanning

A Tc 99 bone scan has been shown to be positive as early as 72 h after the traumatic insult in a patient with joint pain Type 1 DCS. This test, however, like epidemiological studies, has not shown a relationship between symptoms of DCS and the sites of bone lesions.

X-rays

Bone necrosis due to decompression may be detectable using X-rays, but it may take 6 months or longer for the radiological changes to appear. More than 10% of men who have been diving for 12 or more years have some bone necrosis and the proportion is much higher in compressed-air workers. The most frequent sites are the head of the humerus, the lower part of the shaft of the femur, and the upper end of the tibia.

Imaging

Abnormalities detected in CT scans of patients who have symptoms of neurological involvement after decompression and are treated by recompression frequently cannot be correlated with clinical manifestations. Therefore, CT scan is not a cost-effective method for post-treatment evaluation in DCS. As CT is a method of densitometry is not surprising that it has failed to reveal useful information and it has been superseded by magnetic resonance imaging (MRI) which has much greater soft tissue resolution. Medullar lesions after scuba diving have also been demonstrated using MRI (Sparacia *et al* 1997). MRI can be useful in follow-up studies and in early diagnosis of DCS when symptoms do not fit the classic picture or loss of consciousness occurs during surfacing (Aksoy 2003).

Electrophysiological Studies

EEG has been a useful technique to monitor the effect of recompression in cerebral disturbance in experimental animals and somatosensory evoked potentials have also shown abnormalities when the spinal cord is involved in DCS.

Neuropsychological Assessment

Neuropsychological assessment cannot be used as a test in acute decompression sickness because it requires considerable time and expertise. However, cognitive impairment can be detected by neuropsychological testing, even in the absence of neurological signs. Monitoring of the recovery of neurological deficits following HBO therapy can be demonstrated by using this method.

Treatment

Emergency Management and Evaluation

In amateur divers there may be problems in the differentiation of air embolism and decompression sickness on the history because they may run out of gas and make a rapid ascent. Barotrauma is exceptionally rare in altitude excursions and also in commercial divers who usually have an unlimited supply of gas provided from the surface. It is also less likely to occur at the greater depths achieved in bell diving, because the volumetric change is less for a given pressure change with increasing depth. It

is important to recognize that barotrauma can occur on rapid ascent from a depth of a few meters. However, the essentials of therapy are the same for both air embolism and decompression sickness, that is, after ensuring a clear airway and using cardiopulmonary resuscitation when necessary, a diver must be given 100% oxygen as soon as possible and transferred to a chamber. The treatment of air embolism is described in Chapter 11. The Diver Alert Network (DAN) provides a 24 hr hotline in the USA and internationally for advice regarding the management of diving accidents.

Recompression and HBO Treatment

The objectives of recompression are:

- To reduce bubble volume
- To redistribute and redissolve gas
- To reduce tissue edema and hypoxia

The joint pain of Type I DCS resulting from in surface orientated diving generally resolves rapidly with prompt rapid recompression. In very rare cases there may be an initial increase in the pain which is thought to be due to a "squeeze." Many cases will improve breathing oxygen at the surface and it is important to note that no sequelae have been described from this presentation of DCS. It is important to ensure that the patient does not have neurological symptoms and as a neurological examination is difficult and unreliable in the presence of pain and reliance must be placed on the patient's account of their symptoms. Even 100% oxygen at normal atmospheric pressure is also of value in Type II DCS but it must be emphasized that a tight fitting mask must be used to ensure that there is minimal dilution of the oxygen by air. Fluids can be given by mouth if the patient is not nauseated, otherwise intravenous fluid therapy should be used.

Recompression has been established as the definitive treatment for DCS and was first introduced in compressed-air working where traditionally air was used. However recompression breathing air is accompanied by the respiration of additional nitrogen and problems with air recompression tables led to the development of recompression to 2.8 ATA breathing oxygen. This became standard practice following the development of Tables 5 and 6 by the US Navy in 1970. This pressure reduces the volume of any gas present by almost a third and has the advantage of counteracting the hypoxic/ischemic effects of DCS, particularly those on the CNS. The limitation of oxygen breathing is that it cannot be undertaken at pressures higher than 2.8 ATA because of oxygen toxicity. Although rare, convulsions do indeed occur at 2.8 ATA using US Navy procedures. The more complex procedures necessary in commercial diving are beyond the scope of this text, but recompression and increased partial pressures remain the cornerstones of therapy.

For type I DCS (joint pain only), USN Table 5 may be used, as shown in Figure 10.4. The schedule is 135 min in length and has 5-min breaks before beginning the ascent from 60 fsw (2.8 ATA). However it must be emphasized that if pain does not resolve or Type II symptoms are present USN Table 6 must be used and many centers have ceased using Table 5 because of the relapse rate and failure to recognize underlying neurological problems. If symptoms of joint pain do not respond within 10 min of oxygen breathing at 2.8 ATA, USN Table 6, that is the schedule shown in Figure 10.5, should be used. Three sessions of 20 minutes oxygen breathing are undertaken at 2.8 ATA interspersed by 5 minute air breaks. The 150-min period at 30 fsw (1.9 ATA) is divided into alternating periods of 60-

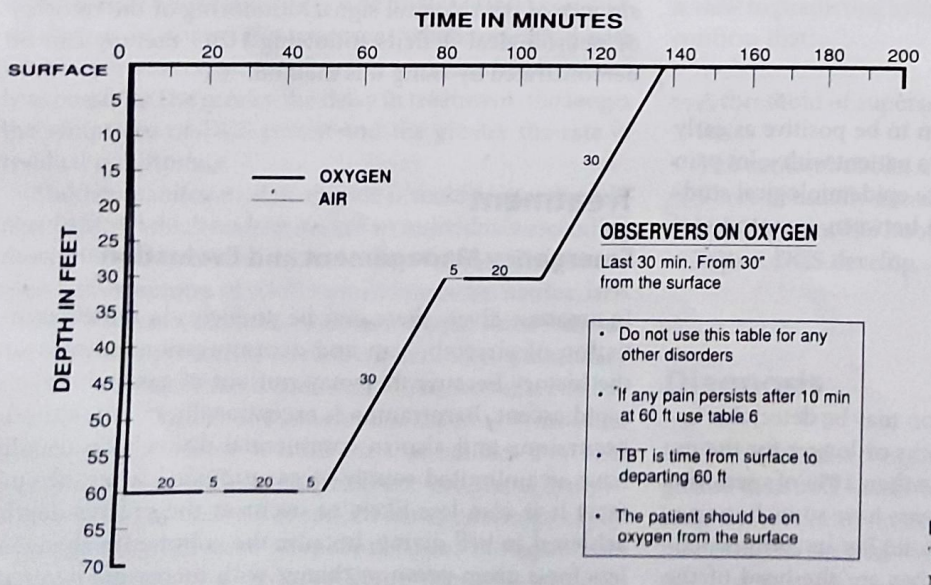

Figure 10.4
The US Air Force modification of US Navy treatment Table 5.

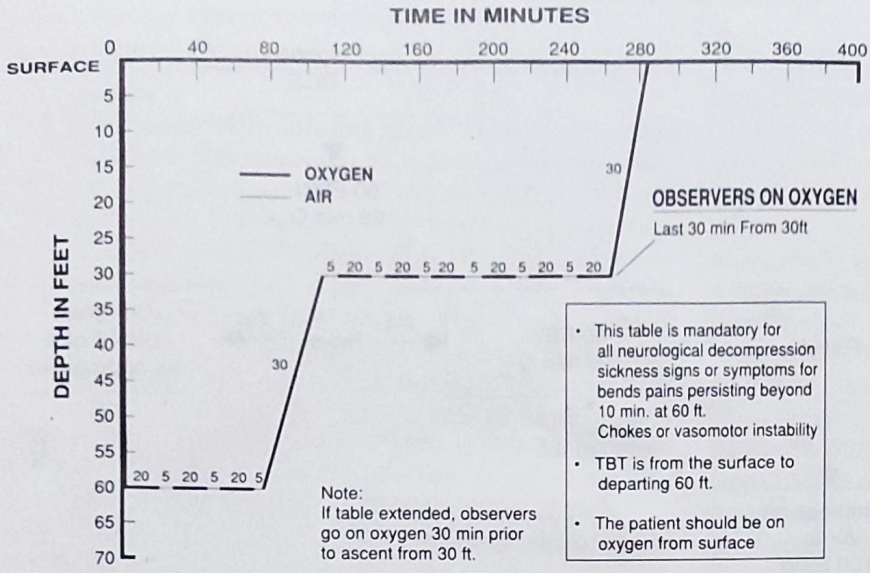

Figure 10.5
The US Air Force modification of the US Navy treatment Table 6.

min of oxygen breathing with two 15 minute periods of air breathing. The total length of this schedule is 285 min, and it may be extended by 100 min if necessary. The US Air Force has produced modifications of these tables for use in altitude decompression sickness.

As an alternative, the flow chart (Figure 10.6) should be consulted. If a COMEX 30 schedule is to be followed, the information is given in Tables 10.2 and 10.3. The gas of preference for this schedule is 50/50 helium and oxygen as much less satisfactory results have been obtained using a 50/50 nitrogen/oxygen mixture. USN Table 6A (1979) is intended for use when it is unclear whether the symptoms are due to air embolism or decompression sickness. Lee *et al* (1988) have modified this table by adding three or more stops from 165 feet to 60 feet. With this approach the total cure rate has been 72%, a substantial improvement over their first recompression response of 37.9%.

Management of Altitude Decompression Sickness

Altitude decompression sickness is treated with hyperbaric therapy has usually been treated the same manner as diving DCS. Expanding space operations and higher flying, more remotely placed military aircraft have stimulated a re-examination of this paradigm. Butler *et al* (2002) prospective treated 12 patients with a new treatment table. USAF Treatment Table 8 (TT8) consists of 100% oxygen delivered at 2 ATA for four 30-min periods with intervening 10-min air breaks (a total oxygen dose of 2 h). Treatment was successful in 9 of 10 cases with Type I altitude DCS. One failure with a recurrence of elbow pain required further therapy. Two patients were treated for Type II altitude DCS with one failure (incomplete clearance of sensory deficits and weakness in the shoulder) requiring further therapy. Although TT8 had two failures, its successes suggest that a new protocol using TT8 for the treatment of altitude decompression sickness is viable but requires further clinical trials.

Management of Neurological Manifestations of Decompression Sickness

Spinal Cord DCS

The response of spinal cord DCS to HBO therapy depends upon the pathophysiology of the lesions. Magnetic resonance imaging (MRI) of the spinal cord should be performed, which may reveal demyelination and also show dorsal white matter lesions typical of venous infarction (Kei *et al* 2007). Cases with short latency may have direct neuronal damage and hemorrhage present. In general such cases require more HBO treatments, and fare less well than those with DCS of late onset. In the latter case, ischemia contributes to neurological deficits and is responsive to HBO treatments. Improvement in MRI findings is not associated with improved clinical status, suggesting that delayed damage subsequent to initial spinal cord lesions may affect the clinical course (Yoshiyama *et al* 2007). HBO therapy was shown by the US Navy to be more effective than the air recompression tables and this has been confirmed in spinal cord DCS.

Helium and oxygen mixtures have been extensively used in recompression therapy in commercial diving (James 1981). The treatment of spinal cord DCS with helium saturation has significant advantages when there has been no clear improvement in neurological status after three 20-min sessions of 100% oxygen at 2.8 ATA. Helium has the

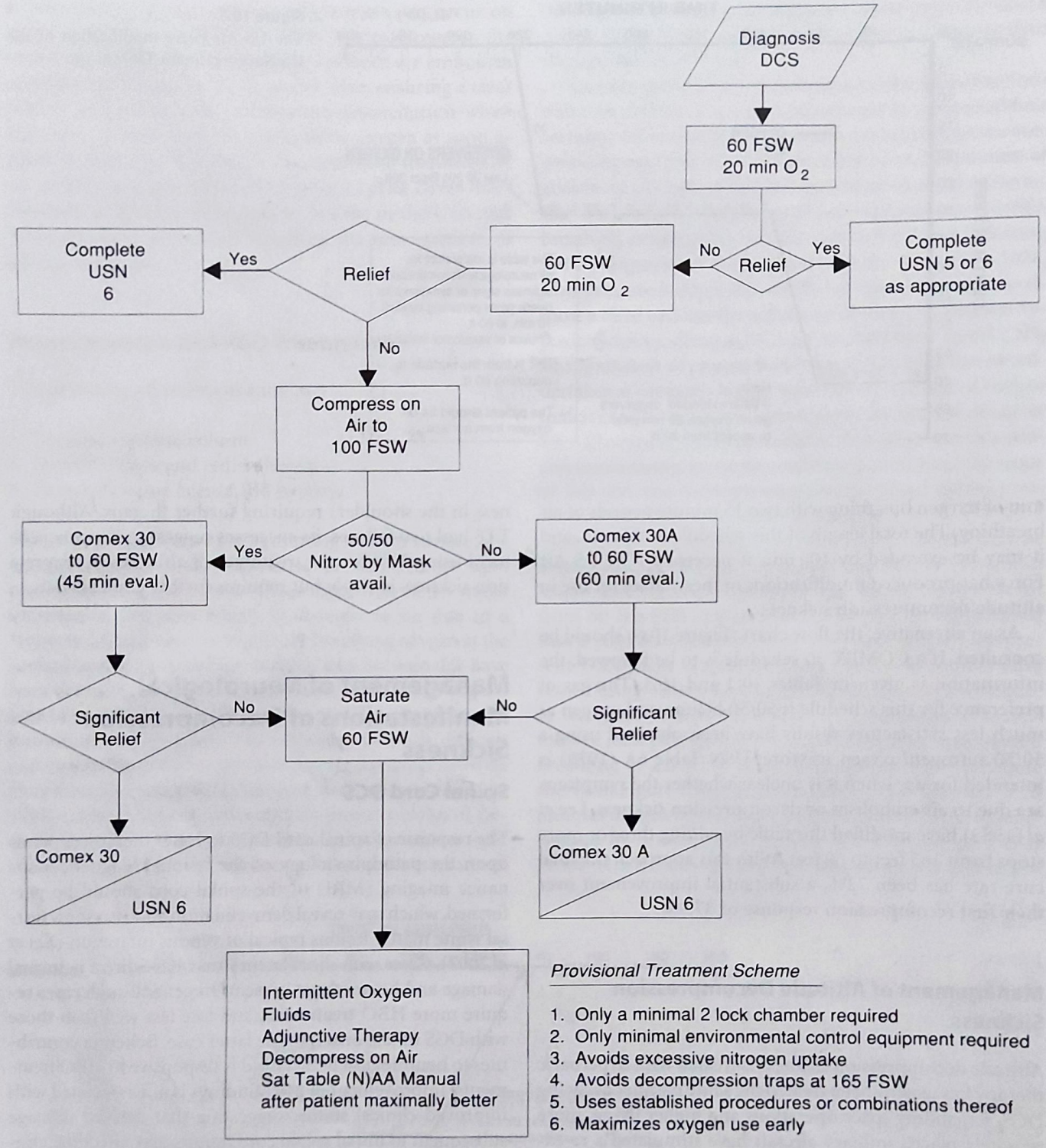

Figure 10.6
Flow chart for decision-making in decompression sickness.

advantage of increasing the rate of nitrogen elimination from the tissues. Kol *et al* (1993) have used helium in cases of spinal cord injury in DCS following air diving. They treated six cases using Comex-30 oxy-helium tables initially and some had additional HBO therapy. Five of these patients made full recoveries and 2 had mild residual neurological deficits. In one case a US Navy Table 6 had failed, but the patient recovered dramatically on recompression to 30 msw breathing a 50/50 helium and oxygen mixture 24 hours later on Comex 30.

HBO at lower pressure (2 ATA) can be a useful tool in the treatment of acute and subacute phases of CNS ische-

mia and continued treatment is now recommended by the US Navy.

Aharon-Peretz *et al* (1993) reviewed their experience in treating 68 sports divers with spinal cord DCS at the Israeli Naval Medical Institute over a period of 16 years. Hydration and 100% oxygen breathing were used until the patients reached the hyperbaric chamber. All patients received recompression therapy based on US Navy treatment tables using oxygen, except for six who were treated on Comex Treatment Table CX-30, which uses a 50/50 helium and oxygen mixture in addition to oxygen. Full recovery was achieved in 79% of these patients. Ball (1993) reviewed 49 cases of spinal DCS from a US naval station and classified them according to severity and time to recompression with oxygen. Delay in treatment was found to worsen the outcome for severely injured divers. Residual severity after all treatments was correlated with the severity after first treatment. Retreatment did not alter the outcome in these patients.

Inner Ear Disturbances

Inner ear disturbances are unusual in air diving but may follow a switch from heliox to air in mixed gas diving. In this situation the problem may reside in the inner ear, the brain stem or the cerebellum. Hearing loss is also an unusual manifestation of decompression sickness and in the absence of other manifestations of DCS, it is difficult to distinguish it from middle and inner ear barotrauma. Once the diagnosis is established, immediate recompression with HBO may result in complete recovery of hearing (Talmi *et al* 1991). These disturbances can be treated with vasodilators, anti-inflammatory agents, and HBO. The last is a useful adjunct even if applied after a delay.

Facial Baroparesis

Ischemic neuropraxia of the facial nerve occurs during decompression if impaired Eustachian tube function causes the overpressure to persist in the middle ear in a person with a deficient facial canal; it is not a common event. The importance of recognition of this complication lies in differentiating it from DCS of the CNS and avoiding prolonged recompression therapy. There is no definite treatment, but HBO may be used, as this approach has been found useful in cases of Bell's palsy (see Chapter 19).

Retinal and Optic Nerve Sequelae

This is a rare complication of DCS. A case has been reported in a fisher-diver who presented with loss of vision, but recovered after HBO treatments even though the therapy was started after a delay of two weeks (Hsu *et al* 1992).

Late Sequelae of DCS

Divers who have had episodes of DCS are more liable to be hospitalized in the following years. These admissions may be due to late sequelae of DCS some of which are:

1. Persistent joint and limb pains.
2. Aseptic necrosis of bone. The cause of aseptic necrosis of bone, which is a late manifestation of DCS, is not known. It may be the result of damage to the endothelium or the capillaries supplying blood to the bone (see Chapter 29).
3. Motor disorders.
4. Peripheral neuropathy.
5. Patients who have suffered DCS have a higher incidence of vascular diseases.
6. Neuropsychological deficits

Monoplace vs Multiplace Chambers

Most recompression facilities available to divers are two compartment multiplace chambers. The use of monoplace chambers for DCS has been controversial, because examination of the casualty is not possible and most cannot be compressed beyond 3 ATA. However compression to 6 ATA is unnecessary in DCS following surface-orientated diving, especially when treatment is delayed. Monoplace chambers can be used for the treatment of DCS under the following circumstances:

- Diving accident victims who arrive at a hyperbaric facility where only a monoplace chamber is available should be accepted for treatment.
- Monoplace chambers should be equipped to provide air breaks using a mask and an external source of compressed-air to allow USN Tables 5 and 6 to be followed.

Air breaks are possible in a Sechrist model 2500-B monoplace chamber and the equipment is easily fitted into other monoplace designs.

Use of Oxygen vs Other Gas Mixtures

Oxygen treatment has disadvantages, because it cannot be used at pressures greater than 2.8 ATA because of oxygen toxicity and air breaks are therefore used at this pressure. In fact, convulsions may occur after even 20 minutes at this pressure and there may also be severe vasoconstriction which may cause worsening of symptoms. Because of the fire hazard raised by pure oxygen, compressed air is still commonly used for the treatment of DCS in workers in compressed-air working, but the results are inferior to those obtained using oxygen treatment. Oxygen treat-

Table 10.2
Therapeutic Table: COMEX 30A

Depths (Meters)	Time (min)	Gas Breathed Patient	Attendant	Total Time (min)
30	40	50/50	Air	40
30 to 24	5	Air	Air	45
(5 min/m)	25	50/50	Air	40
24	5	Air	Air	75
24	25	50/50	Air	100
24 to 18	5	Air	Air	105
(5 min/m)	25	50/50	Air	130
18	5	Air	Air	135
18	25	O_2	Air	160
18	5	Air	Air	165
18	25	O_2	Air	190
18 to 12	5	Air	Air	195
(5 min/m)	25	O_2	Air	220
12	10	Air	Air	230
12	45	O_2	Air	275
12	10	Air	Air	285
12	45	O_2	O_2	330
12	10	Air	Air	452
12	45	O_2	O_2	385
12	10	Air	Air	395
12 to surface	24	O_2	O_2	419
(Rate = 2 min/m)				

Table 10.3
Therapeutic Table: COMEX 30A

Depths (Meters)	Time (min)	Gas Breathed Patient	Attendant	Total Time (min)
30	60	Air	Air	60
30 to 24	6	Air	Air	66
(1 min/m)				
24 to 21	60	Air	Air	126
(20 min/m)				
21 to 18	66	Air	Air	192
(22 min/m)				
18 to 15	72	Air	Air	264
(24 min/m)				
15 to 12	78	Air	Air	342
(26 min/m)				
(2 tablets 5 mg Valium on arrival at 12 m)	18			
12	10	Air	Air	352
12	40	0_2	0_2	392
12	10	Air	Air	402
12	40	0_2	0_2	442
12	10	Air	Air	452
12	40	0_2	0_2	492
12	5	Air	Air	497
12 to surface	24	0_2	0_2	521
(Rate = 2 min/m)				

ment in recompression has the following advantages over air:

- A large gradient for nitrogen elimination
- No further addition of nitrogen
- Tissue oxygenation is improved even without full restoration of blood flow
- Reduced blood sludging
- Improved WBC filterability

In commercial diving it is essential to avoid the use of compressed-air in the recompression therapy of helium and oxygen mixture divers because the addition of nitrogen may cause dramatic worsening of symptoms and even death because of gas countertransport (James 1981). For cases presenting after surfacing the oxygen tables may be used but for deeper recompression therapy helium and oxygen mixtures must be used. This practice has now been incorporated into US Navy procedures (USN Manual 1993). Considerable commercial experience has been gained in the use of 50/50 heliox on the Comex 30 table for both air and heliox divers (Table 10.2). In contrast to oxygen at 2.8 ATA no cases of deterioration in divers have been recorded using heliox in recompression therapy. This is now fully supported by extensive animal experimentation with direct observation of bubbles in tissue. The only contrary experimental data has been from a severe experimental models where following the use of heliox a transient in pulmonary arterial pressure was seen. However this has not been a problem in the therapy of human decompression sickness and James (1988) has stated that the advice first given by the US Navy in 1959 to use helium-oxygen mixtures is still current in the US Navy. Helium and oxygen mixtures can be used in place of air on any of the USN air recompression tables and are the preferred choice for recompression beyond 2.8 ATA.

Role of Drugs

1. IV Saline The only effective "drug" intervention proven to be effective in decompression sickness is rehydration. Wells (1978) demonstrated a reversal of the sludging of blood. His studies failed to demonstrate benefit from other plasma expanders and the Dextrans may provoke allergic reactions.
2. Steroids. As in spinal cord injury there is evidence that steroids may be beneficial when edema is present.
3. Intramuscular diclofenac sodium, a nonsteroidal anti-inflammatory agent, has been used to relieve the residual pain of DCS.
4. Lidocaine, a sodium-channel-blocking agent used clinically as an antiarrhythmic and local anesthetic, can be used as a neuroprotective agent in DCS. Clinical evidence of efficacy in DCS is limited to anecdotal reports. Expeditious administration of lidocaine may be justified

in severe neurologic DCS after patient counseling and consent.

5. Nitric oxide (NO)-donating agents. This is based on the hypothesis that exogenous NO administration or pharmacological up-regulation of NO may reduce DCS risk and severity by decreasing bubble formation, reduction of bubble-mediated inflammatory and coagulation cascades and protection of endothelial integrity (Duplessis & Fothergill 2008). Some of these effects can be achieved by statins, which are approved for treatment of hypercholesterolemia. Statin-mediated lipid reduction may reduce bubble generation via alterations in plasma rheology and surface tension. Use of NO-donor medications such as isosorbide mononitrate and nitroglycerine should be investigated for te treatment of DCS.
6. Lekotrienes. Zafirlukast and zileuton, which are 5-lipoxygenase inhibitors, can reduce inflammatory responses to DCS in rats (Little & Butler 2008).

Hydration of the patient is very important, but saline or balanced electrolyte solution should be used rather than 5% dextrose solution, which may aggravate CNS edema.

Platelet antagonists such as aspirin can reduce platelet aggregation surrounding microbubbles. Substances that increase the intracellular levels of cyclic AMP seem most promising in this respect. There has been no significant human experience with heparin which may promote hemorrhage. In experimental animals perfluorocarbon emulsion (FC-43) combined with 100% oxygen breathing has been shown to provide hemodynamic and neurological protection in DCS. On balance it seems unlikely that pharmacological strategies will become available for the management of DCS and reliance must be placed on early recompression therapy with high partial pressures of oxygen and helium oxygen mixtures.

Importance of Early Treatment

The importance of early treatment has been established beyond dispute in military and commercial diving experience and has been emphasized by Bayne (1978). Fifty consecutive cases of DCS in US Navy divers were reviewed after recompression therapy. There was no mortality or obvious morbidity. The common factors in these cases were as follows:

1. Medical screening and conditioning were strict.
2. Physicians and divers were acquainted with the signs and symptoms.
3. The interval between onset of symptoms and recompression was short.
4. There was aggressive diagnostic and therapeutic use of HBO.
5. There was judicious use of adjunctive measures such as intravenous fluids and dexamethasone.

Immediate hyperbaric treatment is the main factor in ensuring complete recovery from severe DCS.

Once DCS is treated, current guidelines recommend an observation period of at least 6 h for patients with neurological symptoms in case of relapse. Surveys have shown a symptom relapse rate as high as 38.5%, with half of those occurring in the first 24 h. A short-term observation unit is recommended for monitoring of these patients. A retrospective study of patients presenting with DCS at a major hyperbaric facility showed that of 102 consecutive patients with DCS who receiving HBO, 42 (41.2%) had neurological sequelae; 10 required more than one treatment for refractory symptoms or relapse; 38 received up to three treatments, which can be done within the time requirements of short-term observation (Tempel *et al* 2006). Therefore, short-term observation units would provide a safe and efficient disposition for patients after receiving HBO.

Delayed Treatment

Patients with residual symptoms of DCS who present several days following the exposure, can also benefit from HBO treatment, with complete resolution of their symptoms. Therefore, DCS cases should be treated with HBO whenever they are seen, even as late as 2 weeks days after the first symptoms.

A transportable recompression rescue chamber (TRRC) has been suggested as an alternative to delayed treatment TRRC for one person can be used for the rapid initiation of treatment and evacuation in severe scuba-diving accidents. This chamber has also been used for evacuation, although a two-compartment chamber (one compartment for the victim and one for the attendant) is better.

There is some risk is involved in transport as gas bubbles may expand as altitude is increased in an aircraft and hypoxia will be worsened. However some aircraft can maintain sea level conditions at altitude and the Swiss air-rescue service can transport a monoplace hyperbaric chamber in a helicopter. The ideal transportable hyperbaric chamber should be two compartment and fully equipped for ancillary treatment. Some chambers are available for surface transport and can be modified and fitted into a boat or a helicopter so that the patients can be treated while they are being transported to a regular medical facility for further care.

Treatment of Residual Neurological Injury and SPECT Brain Imaging in Type II DCS

The first DCS case where HMPAO – SPECT brain imaging was used to identify viable brain tissue and document the response to HBO at pressures lower than those used conventionally for the treatment of DCS was reported by

Harch *et al* (1993). Since then a total of 13 divers who had type II DCS or cerebral arterial gas embolism were managed by this approach (Harch *et al* 1994a). HMPAO-SPECT scans were done after test exposure to HBO at 1.5 or 1.7 ATA for 90 min. The initial scans were abnormal in all cases. "Tailing" HBO treatments at low pressures (1.5–2 ATA) were continued with primary HBO treatment for DCS in 9 of these and 4 with delays of 4 to 86 days. Neurological improvement was correlated with improvements shown on SPECT scans. A case history with illustrations of SPECT is shown in Appendix 2. This approach has now been adopted by the US Navy and it is essential to recognize that this therapy is addressing hypoperfusion due to edema by utilizing the vasoconstrictive properties of oxygen at increased dosage and is not directed at persisting gas phase.

Risk Factors for DCS

The following risk factors for DCS have been recorded for air diving:

1. Obesity. Obesity increases the risk of DCS. Divers who are more than 20% in excess of ideal weight, according to standard tables, should be prohibited from diving until they have reduced their weight to acceptable levels.
2. Early, retrospective reports of the incidence of altitude decompression sickness (DCS) during altitude chamber training exposures indicated that women were more susceptible than men. In a recent study, no differences in altitude DCS incidence were observed between the sexes (Webb *et al* 2003). Women are at a higher risk of developing altitude-related DCS during their menstrual periods.
3. Sensitivity to complement fixation. Individuals who are more sensitive to complement activation by alternate pathways are more susceptible to DCS.
4. High serum cholesterol levels and hemoconcentration predispose to bubble formation.
5. Moon *et al* (1989) examined 37 patients with a history of DCS, using bubble contrast, two-dimensional echocardiography, and Doppler imagery. Bubble contrast showed right-to-left shunting through the patent foramen ovale in 11 (37%) of these patients, as compared with a 5% incidence in 176 healthy volunteers detected using the same technique. Persons with a patent foramen ovale and a cardiac right-to-left shunt have an increased risk of developing neurologic complications even after recreational scuba diving in shallow water (Schwerzmann & Seiler, 2001). The presence of a foramen ovale, therefore, is a risk factor for the development of DCS in divers, because it allows the passage of venous emboli into the systemic circulation. The fetus may be at risk of DCS in a pregnant diver. The pulmonary filter is not functioning in the fetus and the bubbles generated by either the fetal or the placental tissues will pass through the foramen ovale into the fetal arterial circulation, where they can proceed to embolize the brain, the spinal cord, and other organs.
6. A prolonged stay under pressure followed by rapid decompression.
7. Heavy exercise or other stress at depth.
8. Flying after diving and a rapid ascent to high altitude.

Several decompression tables, some of them computerized, are available for the guidance of divers. Essentially all calculations assume that the additional gas taken up remains in solution and gas equilibrates in the lung to ambient conditions. Both of these assumptions are now known to be untrue. In effect decompression tables contrive to minimize bubble formation and allow the safe elimination of the bubbles formed.

The hypothesis that number of bubbles evolving during decompression from a dive, and therefore the incidence of DCS, might be reduced by pretreatment with HBO, has been tested in rats (Katsenelson *et al* 2007). HBO pretreatment was shown to be equally effective at 304, 405 or 507 kPa, bringing about a significant reduction in the incidence of DCS in rats decompressed from 1,013 kPa. this method has not yet been tested in humans.

Conclusions

The evidence is now conclusive that gas phase forms during most decompressions and adherence to published "no-decompression" limits or decompression tables does not eliminate the risk of decompression sickness. Air diving beyond 30 msw (4 ATA) is associated with a greatly increased risk of decompression sickness.

Early recognition and prompt management of a patient with DCS is essential and early recompression treatment reduces the incidence of late complications. Recompression with a high partial pressure of oxygen is recommended for the initial treatment and US Navy tables 5 and 6 are the most widely used in surface-orientated diving. When required, further recompression should be undertaken using helium and oxygen mixtures. Adherence to good diving practice and the recognition of risk factors for DCS are important to reduce the incidence of this disease.