An Introduction to Clinical Aspects of Decompression Illness (DCI)

Costantino Balestra

ABSTRACT. Decompression Illness (DCI), Decompression Sickness (DCS), Dysbaric Illness (DI), disorder, syndrome are terms associated with the clinical signs or symptoms originally generated by a reduction of absolute pressure surrounding the patient. For 100 years the definition of the “disease” is a matter of “disputes” or “consensi”. We understand nowadays that it is not enough to know how to cure evident clinical manifestations, but also to reduce or virtually eliminate the primary physical cause for the physiological damages: the gas separation phase from saturated tissues – stationary or circulating bubbles. To achieve this goal, research is more oriented on the decompression procedures or the diver pre-conditioning (heat exposure, physical activity, whole body vibration, antioxidant medication, oxygen breathing, hyperbaric oxygen therapy, hydration or dehydration) and post-conditioning (different decompression procedures or models, deep stops, shallow stop followed by a deeper one, post exposure hydration, speed of ascent, exercise during decompression). Some factors that were believed to be crucial, such as patency of the cardiac Foramen Ovale or gender, are considered less important than modified decompression procedures that are studied today with sharper technology.

INTRODUCTION

Decompression Illness (DCI) is a complex condition that can appear with a wide variety of signs and symptoms. Any significant organic or functional decrement in individuals who have been exposed to a reduction in environmental pressure must be considered as possibly being DCI until proven otherwise. This applies to acute, sub-acute and chronic changes related to decompression and may be related to acute clinical symptoms or to situations that may develop subclinically and insidiously. It is in fact generally accepted that subclinical forms of DCI, with little or no reported symptoms, may cause changes in the bones, the central nervous system and the lungs (Kelemen, 1983; Shinoda et al., 1997; Wilmshurst and Ross, 1998).

Generally, a disorder is a physical derangement, frequently slight and transitory in nature. A disease is considered a condition of an organ, part, structure, or system of the body in which there is abnormal function resulting from genetic predisposition, diet, or environmental factors. A disease is typically a more serious, active, prolonged and deep-rooted condition. DCI should be considered a disorder due to a physical primary cause that can transform into a disease unless adequate and timely action is undertaken to abort or to minimize the pathophysiological effects of bubbles on the body tissues.

The predominant physical cause of DCI is the separation of gas in the body’s tissues, due to inadequate decompression, leading to an excessive degree of gas supersaturation (Kumar et al., 1990). Rapid decompression (rate of ascent or omission of decompression stops) is a primary cause of gas separation in tissues (Figure 1).

The most obvious prevention strategy for DCI is, therefore, determining and observing appropriate ascent and decompression procedures (Marroni and Zannini, 1981; Vice President of DAN Europe (Research and Education), Haute Ecole Paul Henri Spaak, Environmental and Occupational Physiology Laboratory, 91, Avenue C. Schaller, 1160 Auderghem, Belgium

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Marroni et al., 2001). Unfortunately, the recommendations for decompression are largely empirical and not always reliable. This is confirmed by the finding that more than half of the DCI cases managed by Divers Alert Network (DAN) worldwide over the past several years have not been associated with an obvious violation of decompression procedures, dive table or dive computer limits; they have been “unpredictable”. This has led to a search for other contributing factors to the development of DCI, such as a Patent Foramen Ovale, in an effort to explain the wide variation in individual susceptibility to DCI. Other factors include complement activation in the presence of gas bubbles as well as an uncertain relationship between gas bubbles, blood cells, and the capillary endothelial lining in response to bubble presence and development of DCI.

The manifestations of DCI are sometimes trivial and subtle. These are likely to be ignored or denied by individual divers, training organizations, and emergency physicians unless they are made aware of them and offered specific information on the manifestations of DCI. However, there appears to be growing evidence that under-reported, under-estimated and under-treated signs and symptoms of DCI may result in permanent organic or functional damage so that raising the level of suspicion amongst divers and physicians alike becomes increasingly important.

Although the presence of Doppler-detectable gas bubbles in the blood is not necessarily predictive of clinically evident DCI, the appearance of DCI in the absence of detectable pulmonary artery and venous bubbles is rare. There is even growing experimental and clinical evidence that suggests that asymptomatic “silent” bubbles in the body may be causing cellular and biological reactions that release secondary potentially damaging biochemical substances in the blood.

DEFINING DCI

The previously adopted classification criteria, i.e., Type I or Type II DCS (decompression sickness) and AGE (Arterial Gas Embolism), are inadequate in that they presume that the underlying pathophysiology is fully appreciated. Unfortunately, there is great disparity in the application of this classification of DCI amongst specialists when asked to define similar cases of decompression disorders using this traditional classification. Consequently, a descriptive form of classification has appeared that uses the common term “DCI”, followed by a description of the clinical signs and symptoms and their onset and development characteristics. The latter has been considered both more universally understandable and simpler to teach. It also shows a much higher degree of correlation among specialists describing the same DCI cases. For purposes of clarity and consistency, DCI refers to disorders of decompression that are clearly due to DCS or where the origin or embolized gas cannot be definitively attributed to pulmonary barotrauma. Where the cause of arterial embolization is the direct consequence of pulmonary overexpansion, the term AGE is used.

Epidemiologically, there is universal consensus among the international diving medical community that the incidence of DCI is generally very low and that there is no significant gender-related susceptibility. There is also consensus that neurological manifestations are by far the most common form of DCI amongst recreational divers.

Many yet unknown aspects of DCI are the subject of ongoing international studies. These include: the relationship between gas separation and DCI injury, the relationship between clinical symptoms and the severity of the disease, the relationship between initial clinical onset, treatment results, and permanent sequelae, the reason for the large variation in individual susceptibility to DCI, the lifespan of gas bubbles; and the true incidence of DCI.

DCI HISTORY

Boyle (1670) demonstrated that DCI could be produced in a reptile by a sudden lowering of atmospheric pressure. The first clinical recording of DCI was in compressed-air workers. Triger (1845) reported that two men had suffered “very sharp pain” in the left arm and another had pain in the knees and left shoulder 30 minutes after emerging from a seven-hour exposure at pressure (the pressure could have ranged between 2.4 and 4.25 atm). Although not knowing what it was, Triger also reported the clinical treatment for DCI as “rubbing with spirits of wine soon relieved this pain in both men and they kept working on the following days”. Pol and Watelle (1854) wrote that they were “justified in hoping that a sure and prompt means of relief would be to recompress immediately, then decompress very carefully”. Yet it was only many years later that their advice was heeded.

In 1878, Paul Bert demonstrated that the cause of DCI was dissolved nitrogen going into gas phase in body tissues and that this bubble formation was responsible for symptoms. Bert also highlighted the existence of “silent bubbles” in the venous blood. He understood that recompression was the key treatment of value and that it should be applied promptly. He also used oxygen at one atmosphere following very rapid decompression.
and observed that cardiopulmonary symptoms, but not spinal cord paralysis, could be relieved by normobaric oxygen breathing (Bert et al., 1943).

Moir (1896) published his work on the 1889 excavations of the Hudson River tunnel. Facing a tragic fatality rate of 25% of the employed workers due to DCI, he installed a recompression chamber at the work site. Following this intervention there were only a further two deaths out of 120 men employed over the following 15 months. Moir wrote:

With a view to remedying the state of things an air compartment like a boiler was made in which the men could be treated homeopathically, or reimmersed in compressed air. It was erected near the top of the shaft, and when a man was overcome or paralyzed, as I have seen them often, completely unconscious and unable to use their limbs, they were carried into the compartment and the air pressure raised to about 1/2 or 2/3 of that in which they had been working, with immediate improvement. The pressure was then lowered at the very slow rate of one pound per minute or even less. The time allowed for equalization being from 25 to 30 minutes, and even in severe cases the men went away quite cured.

Unknownly, Moir was recording both the means of prevention and treatment of DCI. Variations of his techniques, now called surface decompression, are currently still used.

Even though few subsequent publications appeared on recompression treatment for the next 30 years, it was the widely accepted notion that, to be effective, recompression should commence promptly followed by slow decompression. These principles remain in effect to this day even though the pressures used, breathing gases applied, and rates of decompression observed, have undergone much modification.

**ETIOLOGY AND PATHOPHYSIOLOGY OF DCI**

DCI is a disease with protean clinical manifestations. It follows the appearance of gas bubbles produced by the excessively rapid lowering of ambient pressure. This reduction enables inert gas dissolved in tissue to enter the gas phase causing the formation of gas bubbles in tissues and body fluids.

The clinical syndrome is known by a multitude of names including decompression sickness, DCI, decompression injury, caisson disease, bends, chokes, staggers, dysbarism and gas bubble injury. Although arterial gas embolism is usually associated with pulmonary barotraumas, decompression bubbles can also lead to embolization if there is shunting between the venous drainage and systemic circulation (e.g., intracardiac and intrapulmonary shunting). This blurs the boundaries between decompression sickness and arterial gas embolism, which is why the term DCI was created. Many languages do not differentiate between "sickness" and "illness" so that the terms "dysbarism", "dysbaric illness", or "dysbaric injury" have become equivalent terms for DCI. Clinical settings of DCI include diving, aviation, hyperbaric oxygen therapy (i.e., nurses, chamber assistants, and medical personnel), caisson work and tunneling under pressure.

**PREDISPOSING FACTORS**

Whereas the primary factor causing DCI is undisputedly the reduction in ambient pressure causing a rapid inert gas desaturation of tissues, several factors have been identified that can increase an individual’s susceptibility to DCI.

**EXERCISE**

Exercise during exposure to increased ambient pressure (during the bottom phase of the dive) appears to increase the incidence of DCI. The probable explanation is that increased perfusion during exercise leads to a corresponding increase in inert gas uptake, which must be subsequently eliminated during decompression.

Exercise during ascent has differential effects. During decompression stops, mild exercise appears to be helpful. On the other hand, increased activity during pressure change appears to increase the DCI risk. At least three mechanisms may help to explain this effect:

1. The formation of gas micronuclei. Rapidly flowing blood, especially in the area of vessel bifurcation, may create foci of relative negative pressure through a venturi effect. Molecules of gas from the surrounding supersaturated blood may then diffuse into these foci down a partial-pressure gradient. The resulting localized collections of small numbers of gas molecules called micronuclei are thought to act as a nidus for further bubble growth formation;
2. Increased local CO₂ production by exercising muscle may play a role since CO₂ is a highly diffusible gas that could contribute to the formation of gas micronuclei. Even small increases in FCO₂ seem to increase the incidence of DCI. The mechanism of this effect is not clearly understood; and,
3. Increases in core body temperature due to increased muscle activity may reduce the solubility of gas in body tissues leading to bubble formation.

However, very recent research results are questioning some of these assumptions regarding exercise and diving, in particular that of exercise prior to diving. The latter appears to lower DCI incidence depending on when the exercise is performed. The explanation of these findings is still hypothetical, although nitrous oxide seems to be protective when it is produced by an increase in physical exercise 20 hours before diving (Wisloff and Brubakk, 2001; Wisloff et al., 2003; Dujic et al., 2004; Wisloff et al., 2004). There is an association between recent local musculoskeletal injuries and an increased incidence of DCI at or near the site of the injury. The mechanism responsible for this phenomenon is unclear. Changes in local perfusion and increased gas micronuclei formation in injured tissue are postulated mechanisms.
COLD WATER

Diving in cold water tends to increase the incidence of DCI. Inert gas uptake is generally not affected because the exercising diver is usually warm and has increased tissue perfusion due to exercise (Martini et al., 1989; Gerriets et al., 2000). However, as the diver cools during the dive and at the safety or decompression stops, the diver’s tissues experience a reduction in blood flow due to the cold and an increase in solubility tends to retain more gas. As the diver rewarms after the dive, the excess gas may be released as bubbles.

AGE

Advancing age increases the incidence of DCI for reasons that are not yet clearly known but may be related to the reduction in pulmonary function or the reduction of tissue microvascularization.

DEHYDRATION

Dehydration was reported as a factor that increases the risk of DCI during studies on aviators during World War II. The mechanism is again unclear. Changes in the surface tension in serum favoring bubble formation have been postulated. Anecdotal reports suggest that prior alcohol ingestion increases the incidence of DCI, possibly through this mechanism. Some recent papers add insight on the mechanism and advocate new approaches, considering hydration of the tissues more important than plasmatic volume or surface tension (Gempp et al., 2009).

FATIGUE

As with alcohol, there is anecdotal evidence suggesting that significant fatigue preceding a dive increases the incidence of DCI. It is uncertain whether the fatigue is a subtle indicator of some unidentified biochemical factor or a non-specific warning of general hemodynamic factors.

PATHOGENESIS OF DCI

VASCULAR OBSTRUCTION

Vascular obstruction by bubbles or bubble-formed complexes may occur in the systemic or pulmonary circulation, a most important element in the pathogenesis of DCI. Vascular obstruction may occur as bubbles enter the circulation from supersaturated tissues and slow down venous return or due to embolization of vascular beds by bubbles formed elsewhere. Such disturbances may be clinically invisible in non-critical areas such as fatty tissue, but may be life-threatening in critical organs such as the central nervous system and heart.

Diffuse peripheral vascular obstruction and stasis with resultant tissue hypoxia or anoxia may lead to metabolic acidosis and hypovolemia due to increased capillary permeability. Acidosis and hypovolemia may considerably impair cardiovascular function. Vascular obstruction of pulmonary capillaries, secondary to embolization of bubbles or bubble-formed complexes in venous blood, results in increased pulmonary vascular resistance, bronchiolar constriction and peribronchiolar oedema. These changes may lead to alterations in ventilation-perfusion ratios with resultant arterial hypoxemia, a condition called the chokes.

BLOOD-BUBBLE INTERACTIONS: COAGULATION

Much attention has been devoted to the possible consequences of blood-bubble interaction. Bubbles are thought to be capable of activating Hageman Factor (Factor XII) with activation of coagulation, contributing to vascular obstruction. Bubbles constitute a foreign element in the blood and activate the complement and coagulation cascades. They may even cause “denaturation” of lipoproteins with the release of large quantities of lipid. Electron-micrographic studies in animals have shown vascular obstruction by a complex that appears to be composed of a gas bubble surrounded by a layer of lipid, to which platelets are agglutinated. This and similar observations have given rise to a variety of experimental work investigating inter alia the possible usefulness of anticoagulants in DCI. To date there is no firm experimental evidence to indicate that disseminated intravascular coagulation occurs in DCI, nor that routine anti-coagulation is therapeutically useful. Enhanced coagulation at local sites in tissue, however, may contribute to the pathogenesis of DCI. Coagulation Factor XIIa is, however, capable of triggering the reaction of the complement system. The sequence of reactions of this system produces factors that increase capillary permeability and factors that are chemotactic to leukocytes. Factor XIIa is also capable of activating the Kinin-Bradykinin System with liberation of bradykinin and histamine. Bradykinin may cause local pain. Both are capable of increasing capillary permeability.

LOCAL VERSUS VASCULAR BUBBLES

There is little reason to doubt that the localized pain in a joint is the result of local gas formation. Webb et al. (1944a; 1944b) and Ferris and Engel (1951) showed that gas could be seen in periarticular and perivasular tissue spaces. They also demonstrated a correlation between the presence of gas and the occurrence of localized pain. The effectiveness of local pressure in relieving such pain, such as by inflating a blood-pressure cuff, adds legitimacy to the hypothesis. Importantly, DCI often occurs simultaneously in several sites and limb pain may distract physicians from more sinister neurological abnormalities (Figure 2).

While bubbles within tissues are clearly a cause for concern, significant numbers of venous gas emboli may be recorded without any clinical manifestations. In fact, precordial Doppler
detection of bubbles in the right ventricle or pulmonary arteries is not considered to have significant positive predictive value for DCI. However, high degrees of bubbling are associated with an increased risk of developing symptoms. On the other hand, Brubakk et al. (1984) have not observed DCI symptoms in individuals with no bubbles in the pulmonary artery and in the muscles of the thigh. Nevertheless, Nishi (1993) has reported that DCI is always accompanied by bubbles, if all monitoring sites are considered. Brubakk et al. (1991) have argued that Doppler detection, which is performed at intervals, may miss occasional bubbles and that the exceptional cases that have presented with DCI symptoms in the absence of Doppler bubble signals, may have fallen in this category (Table 1).

**Biochemical Effect of Vascular Bubbles**

Gas bubbles affect cells and disrupt biochemical processes as has been demonstrated by *in vitro* studies. Thorsen et al. (1986) showed that gas bubbles are associated with the aggregation of thrombocytes. The degree of aggregation seems to be independent of the gas content of the bubble, but rather is related to its surface properties. Independently, Ward (1967) and Bergh et al. (1993) have also reported that gas bubbles activate complement *in vitro*, and that the response is unrelated to the content of the bubble. This also supports the hypothesis that the bioactive properties of bubbles are related to their surface characteristics. Ward (1967) and Bergh et al. (1993) also differentiated between “sensitive” and “non-sensitive” individuals, depending on the degree of complement activation in response to bubbles. The latter was also related to clinical manifestations of DCI. Individuals with low C5a levels before dives produced many gas bubbles and a single air dive seemed to reduce C5a levels suggesting that gas bubbles may activate both C5a and C5a receptors. This phenomenon has been confirmed by Stevens et al. (1993) in divers up to 14 hours after treatment for DCI.

Complement activation (Kilgore et al., 1994) triggers the activation of neutrophils and the formation of multiple membrane-attack complexes (MAC) that eventually lead to cellular destruction. This also causes the leukocytes to adhere to the endothelium as they circulate over damaged endothelium. Such neutrophil activation has been demonstrated during decompression (Benestad et al., 1990).

C5a activation may be related to some of the skin changes seen in DCI: erythema, edema and infiltration of inflammatory cells (Swerlick et al., 1988). Another important effect of C5a is vasoconstriction and blood flow reduction (Martin et al., 1988). If circulation of blood is restricted during decompression, gas elimination is similarly reduced leading to possible critical supersaturation local bubble formation. Post ischemic hyperemia is not seen, possibly due to C5a activation, leukocyte adherence or even persisting vascular or perivascular bubbles (Bergh et al., 1993).

Vik et al. (1990) also observed that pulmonary changes in pigs following decompression were similar to those observed after complement activation. Lungs exposed to significant amounts of bubbles for approximately 100 minutes after decompression developed considerable leukocyte invasion. Complement activation was therefore considered to be the most important mechanism for acute lung injury (Ward, 1967; Ward et al., 1995).

Certain pulmonary function changes have been observed in divers. These include a reduction in carbon monoxide diffusion capacity and compliance (Thorsen et al., 1986). They are believed to support the growing evidence that inflammatory processes may follow decompression. In fact, the reduction in carbon monoxide diffusion capacity is rapid and is associated with the development of bubbles (Dujic et al., 1992; 1993).

Vik et al. (1990) consider the lungs to be a primary target organ for gas bubbles that are probably exposed to their effects in all decompressions. Indeed, the concept of the lungs serving as a “bubble trap” has been purported for many years, but we are only now looking at the impact that this function has on the “filter” itself. Although removal of bubbles by the lungs prevents more harmful distribution of bubbles to the arterial system, the process of doing so also has consequences for the lung. In addition, the filtering mechanism is not foolproof: if the gas load on

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<th>Bubble grade</th>
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**TABLE 1. Doppler grades (precordial) in different studies and DCI incidence (Modified after Brubakk et al., 1991).**
the lungs is large, the filtering capabilities of the lungs will be exceeded and gas will enter the arterial circulation. An increase in pulmonary artery pressure of only about 30% is considered sufficient to cause arterialization of venous gas bubbles.

Central nervous system alterations in DCI are believed to result from multiple mechanisms, including intra- and extravascular (tissue) bubbles (Francis et al., 1990). Vascular bubbles do not seem to be an important pathophysiological feature of spinal cord DCI. In a group of 10 amateur and 10 professional divers, five of whom had neurological DCI, no changes could be seen (Morild and Mork, 1994). However, the same authors reported changes in the endothelial layer of the brain ventricles in a group of divers (Morild and Mork, 1994; Mork et al., 1994).

Brubakk (1994) has entertained the possibility that this may not so much be evidence of intravascular gas bubbles in the brain as it may be indicative of an increase in venous pressure due to venous gas embolism of the lung interfering with venous return. Another possible explanation may be gas bubbles in the spinal fluid adhering to the lining of the ventricles and causing changes in the adjacent endothelium. Chryssanthou et al. (1977) has indeed shown that animals exposed to decompression show changes in the integrity of the blood-brain barrier and Broman et al. (1966) has also confirmed that even short contact between gas bubbles and endothelium (i.e., 1-2 mins) leads to such changes. Further studies in rabbits have shown that bubble-endothelium contact causes endothelial damage and progressive reduction of cerebral blood flow and function (Helps and Gorman, 1991).

**CLINICAL MANIFESTATIONS OF DCI**

There is ongoing controversy about the best way to classify decompression disorders. Until 1990, these disorders were divided into DCS and AGE. DCS was then divided into two broad categories based on the severity of symptoms and the associated treatment regimens. However, today we recognize that certain forms of DCS may be the result of paradoxical or even frank AGE. Therefore, in the application of the traditional classification that follows, a modifier (DCS or DCI) is added to indicate where such pathophysiological ambiguity exists. Certain manifestations of decompression disorders are known never to be associated with arterial gas embolism and therefore can confidently be classified as decompression sickness. These include limb “bends” and lymphatic DCS.

Time of symptom onset in cases (all manifestations):

- 50% occur within 30 minutes of surfacing;
- 85% occur within 1 hour of surfacing;
- 95% occur within 3 hours of surfacing;
- 1% are delayed more than 12 hours; and,
- Some symptoms have been reported to begin as late as 24 hours and more after surfacing and even longer if there is subsequent altitude exposure (e.g., flying or mountaineering).

### 1. TYPE I

**PAIN ONLY - DCS**

In recreational compressed-air diving the upper extremities are involved three times more often than the lower limbs. This is reversed for caisson workers and in commercial saturation diving. The pain can range from slight discomfort to a dull, deep, boring or even unbearable pain. It is usually unaffected by movement of the joint and there may be overlying edema and regional numbness.

**LYMPHATIC MANIFESTATIONS - DCS**

The lymphatic manifestations of DCS presumably result from obstruction of lymphatics by bubbles. The manifestations can include pain and swelling of lymph nodes, with lymphedema of the tissues drained by the obstructed lymph nodes. New data are suggesting that normobaric oxygen may improve the flow of lymph and may assist in resolving inert gas bubbles contained within the lymphatic system or even to remove some tiny micronuclei that can behave like proteins and thus be captured by the lymphatics (Balestra et al., 2004). Oxygen preconditioning is a known factor to reduce venous gas emboli post diving, which may be explained by micronuclei elimination by the lymphatic system. Some recent results show a reduction in post-diving bubble grades after a known boosting of lymphatic captation by whole body vibration (Figure 3); more evidence is needed to test this hypothesis.

**CUTANEOUS MANIFESTATIONS - DCI**

Itching is common following decompression from dry chamber dives where the skin is in direct contact with the chamber atmosphere rather than with water. This condition, sometimes called “diver’s lice”, is thought to be the result of gas dissolving directly into the skin and causing cutaneous irritation and the release of histamines with subsequent itchiness upon decompression. This is not a true or systemic form of DCI and does not require recompression. On the other hand, itchiness of the skin following a dive in which the skin was wet, is more likely to be true cutaneous DCI. Note that some in-water dives are performed in drysuits. Under these conditions the skin is in direct contact with compressed gas and “diver’s lice” may appear. However, this is usually accompanied by some degree of skin rash or visible skin change.

*Cutis marmorata* is a form of DCI which is thought to result from a complex interaction between bubbles, venous congestion and the immune system. It usually manifests itself as bluish-red “blootches”, frequently affecting the upper back and chest. Prominent linear purple markings are also frequently observed. These manifestations are a systemic form of DCI and suggest significant bubble formation that may also be affecting other areas of the body. As a result, prompt recompression is
recommended, which usually leads to prompt resolution. This sign frequently heralds more serious forms of DCI and there is a statistical association with PFO even if the physiopathological link is currently not fully understood.

2. TYPE II DCI

PULMONARY DCI

This is a syndrome usually presenting with a triad of symptoms:

a) Substernal pain that usually burns and progressively increases. Initially, the pain may be noted only when coughing or with deep inspiration. Over time the pain may become constant;

b) Cough that is initially intermittent and provoked by cigarette smoking (Behnke’s sign). Paroxysms of coughing may become intractable; and,

c) Progressive respiratory and dyspnea.

The manifestations of pulmonary DCI are believed to result from the combined effects of gas emboli in the pulmonary artery and obstruction of the vascular supply to the bronchial mucosa. Untreated pulmonary DCI may be fatal.

NEUROLOGICAL DCI

Although the exact mechanisms of neurological DCI are not fully understood, they are believed to include embolism, autochthonous (i.e., spontaneous interstitial bubble formation), venous stasis and immunological phenomena. These mechanisms have different latencies and show different responses to recompression. The neurological manifestations of DCI are therefore unpredictable, and any focal neurological symptom or sign may be a manifestation of its presence. Any neurological abnormality following a dive should always be assumed to be of central origin and treated accordingly.

CEREBRAL DCI

Brain involvement in DCI appears to be especially common in high altitude aviators (i.e., flying in excess of 25,000 feet in unpressurized aircraft). In this group pulmonary venous gas embolism is also common and hypoxia and positive-pressure breathing may facilitate the transfer of bubbles or immunological products into the systemic circulation. Not surprisingly, a migraine-like headache accompanied by visual disturbances is a common manifestation of DCI. In divers, brain involvement usually presents more overtly with stroke-like symptoms. Collapse with unconsciousness is a rare presentation of DCS but common in AGE. If it does occur, it represents a very grave form of DCI.

SPINAL CORD DCI

Paraplegia is a “classic” symptom of DCI in divers and clearly represents spinal cord involvement. Bladder paralysis with urinary retention and fecal incontinence frequently accompany such paraplegia. Recent years have seen a decline in both the proportion and absolute number of cases of serious paralysis in recreational divers (from 13.4 percent in 1987 to only 2.9 percent in 1997). Similarly, loss of consciousness has dropped from 7.4 percent to 3.9 percent during the same period. The incidence of loss of bladder function has dropped from 2.2 percent to 0.4 percent during this period (DAN Diving Accident Reports). Interestingly, the reduction in severe neurological symptoms has not been balanced by a proportional rise in pain-only or skin manifestations. Rather, there has been an unexpected appearance of mild, ambiguous neurological manifestations, such as paresthesia or tingling, which appear to respond well to oxygen administration and recompression.

INNER EAR DCI

Audiovestibular DCI is a not uncommon manifestation of CNS involvement. Usually both the cochlea and vestibular apparatus are involved and the presenting symptoms include tinnitus, deafness, vertigo, nausea, vomiting, and ataxia. Nystagmus may be present on physical examination. It is not clear whether the situation depends predominantly on bubble formation in the perilymph or is due to embolization of the auditory vestibular Inner ear DCI is a serious medical emergency and must be treated immediately to avoid permanent damage. Since the nutrient arteries of the inner ear are very small, rapid reduction in bubble diameter, with immediate 100% oxygen

FIGURE 3. Bubble score after 30 min. of whole-body vibration preconditioning 2 hours before the dive (25 m/25 min), n = 6.
administration and prompt recompression is essential.

**Shock - DCI**

Shock occasionally occurs in DCI and is usually associated with serious pulmonary manifestations indicating a hyperacute form of DCI. Multiple mechanisms may contribute to the pathogenesis of shock in DCI, including loss of vascular tone from spinal cord involvement, myocardial depression from hypoxemia and acidosis, pulmonary embolization, and hypovolemia due to increased capillary permeability resulting in loss of plasma water and hemoconcentration.

**Back, Abdominal, or Chest Pain - DCI**

Pain in these areas, in contrast to limb pain, should be considered with great attention, as it frequently represents spinal cord involvement.

**Extreme Fatigue - DCI**

Fatigue, out of proportion from that normally occurring after a dive, has long been regarded as a serious manifestation of DCI. However, the biochemical and pathophysiological mechanism of this symptom is unknown.

**NEW DCI CLASSIFICATION**

The simple classification into Type I and Type II DCI implies that the different categories are well defined disease entities and that there is reasonable agreement about the classification (Brubakk, 1994; Brubakk and Eftedal, 2001).

However, two separate studies (Kemper et al., 1992; Smith et al., 1992), showed considerable uncertainty between experts using the classical classification system. For instance, cerebral DCI could not in many cases be distinguished from arterial gas embolism or vestibular barotrauma. Other studies have shown that solely articular symptoms are rare, as they are usually accompanied by central nervous system symptoms (Vann et al., 1993; Freiberger et al., 2002). Extreme fatigue can be classified as a minor symptom, but could also be a sign of subclinical pulmonary embolism (Hallenbeck et al., 1975). Francis and Smith (1991) therefore suggested the currently widely adopted term “DCI”, to include the two previously used definitions of decompression sickness and arterial gas embolism. They also suggested avoidance of the classifications of Type I and Type II, while adopting instead a descriptive classification method according to the clinical manifestations (signs and/or symptoms) and their evolution in time. Using this classification scheme, a very high degree of concordance between different specialists was possible (Pollard et al., 1995). Francis and Smith (1991) proposed the following Classification Table for Decompression Injuries (Table 2), which is a useful guideline to correctly describe the various possible manifestations of a decompression disorder.

**DCI TREATMENT**

Air was nearly always used as the breathing gas and oxygen treatment was not really explored until Yarbrough and Behnke's (1939) preliminary experiments. The early treatment theory was conceptually homeopathic in trying to decide how deep to take the injured diver. The original depth of the dive was used as a guide. For example, if decompression from a depth of 40 meters caused the symptoms, recompression to the same pressure should alleviate them. However, there were controversies as others thought that the situation of the diver should lead the decision and that the depth of relief should mark the initial treatment pressure. Still others argued that bubbles may be compressed but never disappear and should always be assumed to be present. For these reasons the recommendation was to

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8 • THE FUTURE OF DIVING
compress the patient to the depth of relief plus at least one atmosphere. The rationale was that if a bubble became extremely small, surface tension may cause it to collapse and disappear. It was generally realized that bubbles remaining in the tissues and circulation would continue to take up inert gas, as more nitrogen was absorbed during the recompression treatment. In the 1924 edition of the U.S. Navy Diving Manual, a suggested recompression treatment procedure was first published, but more than 50% of the treatments were unsuccessful. The U.S. Navy published treatment tables again in 1942 without much improvement in the results.

Van Der Aue and Behnke (1945) experimented with better treatment methods that resulted in the publication of the U.S. Navy Air Recompression Tables I to IV that became the universal standard for the next 20 years. The outcome improvement over the previous approach was dramatic and over 90%. The principles of these tables were:

- Recompression to depth of relief plus at least one atmosphere. In practice, this meant going to a minimal depth of 30 meters;
- A maximum treatment depth of 50 meters. This depth was considered a good compromise between optimal recompression of any bubble while minimizing nitrogen narcosis risk and subsequent decompression;
- The use of a 12-hour stop at 9 meters before surfacing. Theoretically, this “overnight soak” was intended to allow all the tissues to saturate or desaturate to the 9-meter level, from where, according to Haldane, direct decompression to the surface would be safe; and,
- The use of intermittent oxygen breathing during the last hours of treatment.

These tables were at first considered very successful, with a failure rate on the initial recompression of only 6% in 1946. Despite their great length, from 6 hours 20 minutes for Table I to 38 hours 11 minutes for Table IV, they represented the only available therapeutic solution at the time. In 1963, however, the observed failure rate for serious symptoms cases was 46% on the initial recompression, and it became 47.1% in 1964. The reason for the failure of these tables, which had initially been so successful, was that more and more civilian recreational scuba diving cases were being treated. The civilians had often dived in total ignorance of decompression requirements, not to speak about the pre-treatment intervals, which were significantly longer than with the military divers.

Goodman and Workman (1965) started investigating the use of oxygen at moderate depths (2.8 ATA) for the treatment of DCI. Oxygen treatment had first been suggested by Behnke and Shaw (1937). At that time, however, the U.S. Navy Bureau of Medicine and Surgery was concerned that oxygen breathing in the chamber may be dangerous and not “sailor proof” and that the risks of oxygen toxicity and fire were too great (Kindwall, 1998). For this reason Behnke’s excellent results were ignored. End (1937) had also noted that treating DCI with compressed air was inefficient and had introduced oxygen breathing with excellent results in over 250 cases of DCI in compressed air workers (Kindwall, 1998). The first 52 cases treated with the “new” Goodman and Workman oxygen schemes showed that at least 30 minutes at the maximum depth of 18 meters and a total oxygen breathing treatment time of 90 minutes could be considered “adequate” treatment, allowing for a 3.6% failure rate. Treatment schedules were lengthened to two hours and four hours of oxygen breathing, with air breathing intervals of 5 to 15 minutes, to avoid oxygen toxicity. The two- and four-hour schemes were called U.S. Navy Table 5 and 6 respectively and were officially published in 1967. The most significant conceptual difference, with respect to the previous approach, was the importance given to the time of relief, instead of the depth of relief (Kindwall, 1998). Compression ceased to be limited to the scope of reducing the bubbles until they could disappear and then decompressing the diver according to a safe profile.

The concept of a real “therapeutical” treatment scheme was introduced where compression is just the vehicle for a therapeutical drug (oxygen) that exerts its treating action during the entire treatment schedule while also offering a double tool for the reduction of the offending gas bubbles, both by pressure and by a favorable diffusion gradient. Serial treatment of DCI had been advocated by many, until, at a meeting of the North Sea offshore diving groups at the Royal Society of Medicine in London in 1976, a consensus was reached that if the diver had residual symptoms after the initial treatment, daily hyperbaric oxygen treatment should be continued for at least two weeks or until the patient’s signs and symptoms had plateaued (Elliott et al., 1974a, b; Elliott and Moon, 1993; Eke et al., 2000).

Ancillary pharmacological treatment to recompression began to be emphasized in the late 60’s and 1970’s. In 1979, the Undersea Medical Society organized a workshop on the management of severe and complicated cases of DCI, where the importance of hydration, steroids, heparin, aspirin and other agents were discussed. Several modifications to treatment schemes were then introduced, such as the Comex Table 30, using mixed gas at a maximum pressure of four atmospheres, and the concept of saturation treatment. This was first introduced by Miller et al. (1978) with saturation treatment to start at four atmospheres while the patient breathed oxygen at 0.35 to 0.5 bar.

Currently, many different treatment protocols are in use, while the U.S. Navy Table 6 probably enjoys the most universal use. Available evidence indicates that this table is adequate in the majority of cases where treatment is initiated immediately (DAN Diving Accident Reports). Unfortunately there is often considerable delay in initiating treatment, and many of the secondary effects of the bubbles on blood and tissues become important. Kelleher et al. (1996) has shown that initial treatment is effective in only about 66% of the cases. Leitch and Barnard (1982) and Leitch (1985) have demonstrated, however, that none of the alternative proposed protocols, including saturation
decompression, are superior to U.S. Navy Table 6.

The severity of symptoms should not be the only variable considered. Heliox and trimix dives may differ from air dives due to the differences in partition coefficients of helium and nitrogen in the tissues (Brubakk et al., 1986). The use of different gas mixes, particularly heliox for the treatment of DCI, also for compressed air diving, is controversial. Some reports seem to indicate that helium is beneficial. Air bubbles in tissue have been observed to disappear faster from the spinal cord if heliox is used instead of pure oxygen at 1 ATA, but the reverse is true at the pressure of 2.8 ATA (Hyldegaard and Madsen, 1989; Hyldegaard et al., 1991; Hyldegaard and Madsen, 1994). Brubakk (1994) believes, having shown an increased shunt in the lung and a reduction of gas elimination at increased oxygen tensions, that there is actually little benefit in using high oxygen tensions and that lower tensions may be more advantageous.

Over the years, many attempts have been made to improve the treatment of DCI with certain drugs, generally, with little success. Some of these possibilities have not been sufficiently studied and may deserve further attention, such as the use of fluorocarbons, which have a higher solubility for nitrogen than plasma. Lutz and Herrmann (1984) were able to substantially reduce the mortality of rats undergoing rapid decompression from 8 ATA when fluorocarbon was infused after decompression. A further point that deserves attention and study is complement activation and its effect on the leukocyte-endothelium adhesion, which appears to have a certain role in DCI, and for which drugs could have a role. In 1994, the European Committee for Hyperbaric Medicine organized its first European Consensus Conference, where DCI was one of the topics. In 1996 a second, more specific Consensus Conference was organized, the theme of which was “The Treatment of Decompression Accidents in Recreational Diving”. Following both Conferences, and after extensive presentations by leading international experts, the two International Juries formulated recommendations that have since been adopted as the current standards for the definition and treatment of DCI in Europe.

CONCLUSION

DCI is generally considered a rather benign condition, if adequate treatment is promptly started, with a success rate in excess of 80%. There is universal consensus that 100% oxygen should be administered immediately as the single most important first aid treatment of any DCI case related to surface-oriented diving and that rehydration is a valuable adjunct during diving first aid treatment in the field.

Hyperbaric treatment should be started within the shortest possible timeframe from surfacing or from the onset of the first DCI signs and symptoms. Hyperbaric treatment tables using 100% oxygen at environmental pressures not exceeding 2.8 bars, with various depth/time profiles, demonstrate very good results in more than 80% of the treated cases. There is no significant evidence that any other therapeutical schemes provide better results and would therefore be preferable as the hyperbaric treatment of first choice for DCI related to surface-oriented diving.

It is accepted by many specialists that the use of high pressure (generally 4 bars maximum) treatment tables using a gas mixture of 50% Helium and 50% Oxygen may prove highly effective and provide good results in the cases that do not quickly and satisfactorily respond to the standard low pressure hyperbaric oxygen treatment tables.

Although conclusive scientific evidence suggesting the use of any pharmacological treatment other than oxygen is missing, the administration of adjunctive fluid therapy is considered very important and generally recommended by diving-hyperbaric medicine specialists, whereas the role of other drugs, such as steroids and anticoagulants, although widely used without any apparent adverse effect, is still controversial.

The continuation of hyperbaric oxygen therapy, combined with a specific rehabilitation protocol in neurological cases when the initial DCI treatment tables are not totally successful, is considered important and there is growing scientific evidence that it can significantly contribute to eventually achieving a better functional recovery.

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