Chapter Seven
Decompression Injuries & Emergency Treatment

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Decompression Illness (DCI)

What's in a name? Decompression Illness (***DCI***) is commonly known as Decompression Sickness (***DCS***). However, DCI includes Arterial Gas Embolism (***AGE***) where DCS is only decompression-related. Also, this cluster of afflictions can be prevented. If a patient is suspected of having a DCI and a positive determination cannot be made, have the patient consult a Diving Medical Physician. DCI is broken down into three major categories: DCS Type I, DCS Type II and Pulmonary Over-Inflation Syndromes (***POIS***).

DCS: There is no clear source for DCS although there does seem to be a correlation between inert gas bubbles in the blood and patients who suffer from DCS. It is for this reason that the following theory of DCS is discussed in detail.

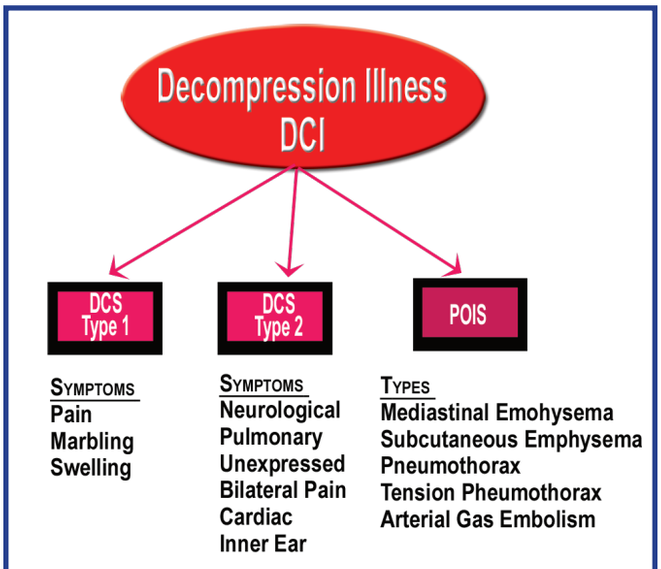


Figure 7-1: Types and symptoms of decompression illness

Tom Mount

Alexander Sotiriou and Jim Holt during a deco stop at Four Sharks Blue Hole, S.Andros Isle, Bahamas

Henry's Law governs DCS in divers. The amount of gas capable of absorption into a liquid at a given temperature is invariably proportional to the Partial Pressure of the gas (***PPgas)***. Only inert gases are of concern to divers with respect to DCS since oxygen will be metabolized prior to absorption. (Concern for high Partial Pressure of O_2 exists, but not when dealing with DCS.) DCS is believed to be a result of inert gas being absorbed into the tissues on compression and while at depth during the dive. They in turn do not have sufficient time to escape during the ascent to the surface. At surface pressure, body tissues are saturated with the inert gas being breathed. As pressure is increased with depth, the partial pressure of the gas inhaled increases. Simultaneously, due to the increased pressure, the body's tissues are capable of absorbing a proportional amount more of the inert gas being breathed. While maintaining a constant increased pressure (at depth), the tissues can absorb an amount of inert gas consistent to the pressure.

As the external pressure is reduced at a decreased depth, tissues begin the process of off-gassing. The tissues are attempting to return to equilibrium equivalent with external pressure by releasing the previously absorbed gas into the blood stream where it is carried to the lungs for filtering. The amount of blood filling the capillary bed at any one time is about 5% of the entire body's blood volume. The capillary bed is the area where the exchange of O_2 and other nutrients with CO_2 and wastes takes place. The exchange can only take place at the capillary bed because

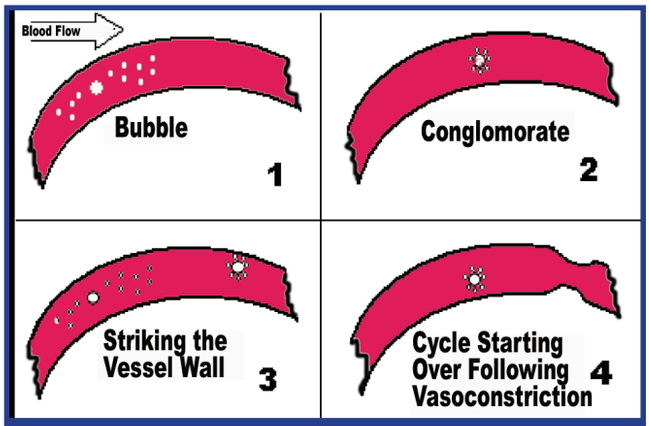
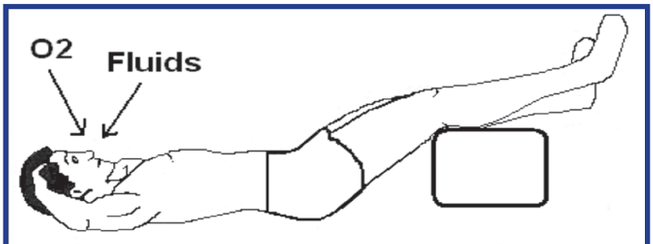


FIGURE 7-2: DECOMPRESSION SICKNESS BUBBLE FORMATION

they are lined with one thin layer of porous endothelial cells capable of allowing solutes smaller than proteins to diffuse between blood and tissue. The endothelial cells are surrounded by a basement membrane which does not interfere with diffusion but serves to hold the capillary together.

When the inert gas solubility capability is exceeded such that the gas is forced out of solution, a bubble is formed to transport the gas out of the system. Upon realizing the presence of the gas bubble, the body immediately sends antibodies to inspect this new foreign body. When it is discovered to be a foreign element to the body, the immune system dispatches phagocytes and leukocytes to attack and remove the bubble by attaching themselves onto the bubble. Another problem associated with the gas bubble trapped in the bloodstream is the surface of the gas bubble tends to attract other particles found in the blood stream such as fat. The result is a large mass consisting of the gas bubble, fat and phagocytes/leukocytes making its way through the blood stream.



Lie flat on back, elevate legs, treat for shock, administer O_2 & fluids, make victim as comfortable as possible.

FIGURE 7-3: TREATMENT FOR DECOMPRESSION ILLNESS

A common misunderstanding is that the bubbles lodge in the veins and block blood flow. While this may be true in the worst case scenarios, it is not the standard manifestation. Usually this conglomerate easily fits through all vessels. The problems arise when the conglomerate moves through the blood stream and ***bounces off the walls***.

When the endothelial cells on the blood vessel walls are damaged in any form by striking or grazing, the body reacts to ensure there is minimal loss of blood. These responses potentially worsen the DCI. The first response is the adhesion of the blood platelets to the exposed collagen fibers (in the wall of the injured vessel), which causes the release of serotonin from platelets resulting in strong vasoconstriction. This process of vasoconstriction and platelet aggregation instigates a vicious cycle which could eclude the vein after a series of conglomerates does its damage.

Categories of DCS: The first significant symptom of DCS is psychological, not physical; ***denial***. Divers believe that, "***This could never happen to me***," which often worsens the more concrete effects of DCS, which are categorized in the list below.

1. **Type I:** This is the less severe of the two types. Even though the symptoms are not very severe, they cannot be ignored. Common symptoms are pain, marbling and swelling

   a. **Pain:** Dull or aching type pain, usually in a joint. Pain origin is non-descript and can normally not be pinpointed, similar to a sprain. It may/may not get worse during movement, but is usually present at rest. It is generally confined to a specific area and is not attributable to another injury

   b. **Marbling:** Skin bends (***Cutis Marmorata***) Condition starts with intense itching and yields way to a bluish gray bruise like discoloration.

POINTS TO REMEMBER

DCS Type 1 Decompression Sickness

- Simple joint pain
- Marbling of the skin
- Swelling of the lymph nodes
- Denial is common

Skin will look marbled or mottled. Symptoms may get progressively worse. Symptoms which start as itching may not lead to marbling; itching alone is not DCS Type I

c. Swelling of the Lymph Nodes: Significant lymph node pain and swelling

2. Type II: Unlike Type I, Type II may not be readily apparent. A diver may feel "funny" or over-tired. Normally these symptoms would not be problematic. However, post dive they pose a significant health risk. Type I symptoms may/may not accompany these symptoms. Many of the symptoms of DCS Type II mirror those of an arterial gas embolism (***AGE***)

a. Unexpressed: These are symptoms such as over tired and weakness. They may become more severe as time progresses. If treatment is not provided, these "minor symptoms" could progress to a severe neurological deficit

b. Neurological: These are any symptoms which may be seen or discovered as a result of a comprehensive neurological assessment. Symptoms include, but are not limited to: numbness, tingling, increased or decreased sensation in an area, muscle weakness

c. Pulmonary: Commonly referred to as ***chokes***. A great deal of inert gas bubbles inundate the vascular area in the lungs. This is intravascular bubbling (***cavitation***). Substernal pain which is aggravated by inspiration along with an irritating possibly productive cough. This is generally accompanied by an increase in breathing rate and may progress to circulatory collapse, unconsciousness and death

d. Inner Ear: Sometimes called ***Staggers***. Tinnitus (ringing in the ears), hearing loss, vertigo, dizziness, nausea, and vomiting are some of the symptoms. Inner Ear DCS is associated with mixed gas diving and during decompression when the diver switched from breathing helium to air. Even though the symptoms are similar, Inner Ear DCS must be differentiated from ear barotrauma. The symptoms of the "***staggers***" may be due to neurological decompression sickness where symptoms of barotrauma may be due to a ruptured TM. A quick check of both ears by a medically trained individual will help to differentiate these problems

e. Cardiac: Very rare. One report of 1st degree AV block which responded to recompression. Symptoms are similar to heart attack or stroke

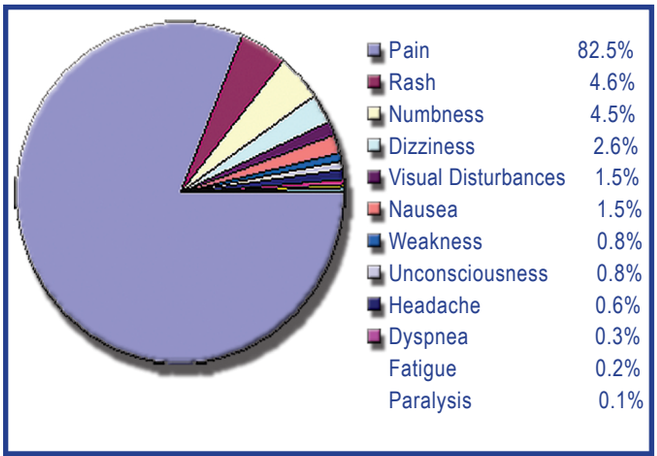


FIGURE 7-4: DECOMPRESSION SICKNESS & THE DISTRIBUTION OF TYPICAL SYMPTOMS

POINTS TO REMEMBER

DCS Type II Decompression Sickness

- **Any symptom following a dive that is not Type I DCS**
- **Similar symptoms to an AGE**
- **Watch for unexpressed symptoms**

PULMONARY OVER-INFLATION SYNDROME (POIS) & EXTRA ALVEOLAR AIR (EAA)

All POISs/EAAs are caused by an over-inflation or a rupture of the alveoli lining which leads to Pulmonary Interstitial Emphysema. It is caused by excessive positive pressure within the lung or some kind of blockage which does not allow the expanding air in the alveoli to escape during a decrease in external pressure. It could manifest itself by a permanently or temporarily congested or blocked brachial tubes, a diver failing to breathe continuously during ascent, or a diver who performs the Valsalva Maneuver on ascent. The route which the escaping gas takes determines the type of POIS and ultimately the treatment.

1. Mediastinal Emphysema: A mediastinal emphysema occurs when the bubble of gas which

escaped from the rupture leaks into the mediastinal tissues in the middle of the chest. It is characterized by substernal pain which may be mild to moderate and is often described as a dull ache or a feeling of tightness across the chest. The pain may become worse with deep inspiration or coughing and may radiate to the shoulder, back or neck

2. **Subcutaneous Emphysema:** A subcutaneous emphysema is a mediastinal emphysema which has leaked upward into the subcutaneous tissues in the neck and lower face. It is characterized by a voice change, crepitating or the feeling/appearance of fullness in the neck, shoulder or collarbone area

3. **Pneumothorax and 4. Tension Pneumothorax:** In a pneumothorax the gas which has escaped, leaks into the space between the chest wall and the lining of the lung. This leak causes a pocket of gas which may cause respiratory distress. If the leak is an isolated incident, the gas will normally be reabsorbed with time. If the leak continues, the pressure within the cavity could force the whole lung or a lobe to collapse. This situation is severe. Indications of a pnuemothorax include a sudden sharp flank pain in the chest followed by breathing difficulty. A tension pneumothorax occurs when the lung collapses completely and presses on the heart. The collapsed lung pushes the heart and its blood vessels to the other side of the chest, and the heart cannot pump normally

5. **Arterial Gas Embolism (*AGE*):** An AGE is caused by entry of gas bubbles into the arterial circulation which then could act as blood vessel obstructions or similarly to any inert gas bubble such as those which come from decompression sickness. These emboli are frequently the result of pulmonary barotrauma caused by the expansion of gas taken into the lungs while breathing gas under pressure and held in the lungs during ascent. The gas might have been retained in the lungs by choice or accident. The organs that are especially susceptible to arterial gas embolism and that are responsible for the life threatening symptoms are the central nervous system (***CNS***) and the heart. In all cases of arterial gas embolism, associated pneumothorax is possible and should NOT be overlooked

Points to Remember

Pulminary Over-Inflation Syndrome

- Mediastinal Emphysema
- Subcutaneous Emphysema
- Pneumothorax
- Tension Pneumothorax
- Arterial Gas Embolism (*AGE*)

Initial first aid is a must. If a person is suspected of having a DCI, ***IMMEDIATELY*** administer fluids, oxygen and transport in supine position (***lying on the spine***) to the nearest hyperbaric facility. A POIS indicates a hole in the alveoli. For this reason, recompression is NOT normally recommended because of the risk of introducing more gas into the blood via the existing hole. AGE's are an exception to that rule because the result of the introduction of additional gas into the blood via the hole in the lung is overshadowed by the severity of the symptoms.

Risk Factors that May Hasten the Onset of DCS

Patent Foramen Ovale (*PFO*)

All fetuses have a hole between the chambers of the heart; the lungs are non-functional in the fetus so the hole allows blood to bypass the lungs. Technically, the hole is called a foramen ovale that is patent (***open***). Normally, this hole seals within 24 hours of birth. Adults have the advantage that blood is transported across the lung capillary bed which is insensitive to bubbles. The fetus does not have this advantage. The fetus does absorb nitrogen across the placenta, but any bubbles that may be formed in the fetus would end up in the fetus's circulation, or possibly in the placenta. This is why woman who think they are pregnant should not dive.

The PFO, or opening in the wall of the heart, is necessary to transfer oxygenated blood via the umbilical cord. However, a PFO can create a myriad of problems if it is found intact or only semi-closed more than 24 hours after birth. This patency can cause a shunt of blood from right to left, but more often there is a movement of blood from the left side of the heart (***high pressure***) to the right side of the heart (***low pressure***). People with shunt lesions are less likely to develop syncope or hypotension with diving than are obstructive valve lesions, but are more likely to develop pulmonary congestion and severe shortness of

breath from the effects of combined exercise and water immersion.

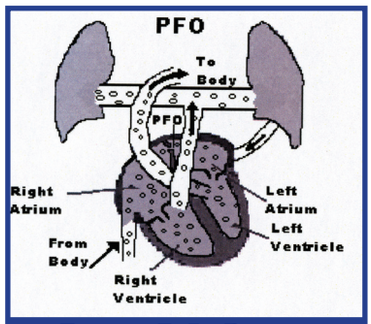


Figure 7-5: Patent Foramen Ovale

Ordinarily, the left to right shunt will cause no problem; the right to left shunt, if large enough, will cause low arterial O_2 tension and severely limited exercise capacity. In divers there is the risk of paradoxical embolism of gas bubbles which occur in the venous circulation during decompression. Intra-atrial shunts can be bi-directional at various phases of the cardiac cycle and some experts feel that a large atrial septal defect is a contra-indication to diving. In addition, a Valsalva maneuver, used by most divers to equalize their ears, can increase venous atrial pressure to the point that a right to left shunt occurs, thereby transmitting bubbles that have not been filtered out by the lungs.

If a diver is concerned or is having some of the symptoms noted above he or she should seek medical attention. Normally a diver who already has dove to in excess of 100 fsw (930 msw) would have had problems before if a significant PFO was present.

Previous DCS or Problematic Areas

People who have been previously exposed to DCS are more likely to have DCS in the same area. The area where the former insult or previous injury was has probably developed scarring. The increase of scarred tissue over the area makes it less wide. When a bubble tries to pass, it may come in contact with the already scarred tissue faster than would have if there was no scar tissue present.

Age

More applicable than the specific numerical age is the fact that the body changes as people age. Increased body fat, degenerative joint disease, alterations in pulmonary function and cardiac disease are among those changes that increase the risk of DCS with age. This may or may not be the driving ideology, however we know that the U.S. Navy dive tables were established using Navy divers. These individuals are 18-25 years old and in top physical condition. Diving within the limits of the Navy dive tables may be ill-advised practice for an older person.

Body Fat

Fat has high nitrogen solubility. High nitrogen solubility increases nitrogen absorption and bubble growth. We also know that the U.S. Navy dive tables were established using Navy divers who are 18-25 years old and in top physical condition. Diving within the limits of the Navy dive tables may be ill-advised practice for a heavier person or someone who is less physically fit.

Post-Dive Exercise

Doppler scores and the likelihood of DCS increase with post dive exercise. The probable reason is the increase of circulation post dive pushes the decompression progress too far. Your body may attempt to off-gas too quickly causing bubbles to form. These bubbles decrease the tissue and arterial inert gas tensions which reduce the elimination rate.

Body Temperature

Cold decreases your body's ability to off-gas. The problem here is most divers start off relatively warm and as the dive progresses become increasing cold. At the point in the dive where the diver ascends to begin decompression they are cold.

Workload at Depth

Presumably this increase of risk is due to an increase in circulation allowing more inert gas to be absorbed.

Hydration Level

This is the largest contributing factor of DCS. Divers are dehydrated due to immersion diuresis as well as sun exposure and decreased fluid intake. This combination reduces circulation and the rate of off-gassing.

In-Water Recompression

No established course exists to certify anyone to perform in-water recompression. This procedure should not even be attempted if the patient is unconscious or vomiting. If

JIM AKROYD

GEN AKROYD INSPECTING A MARK V DIVING HELMET

it must be attempted, a diver should always accompany the patient and observe his or her condition very closely as the incidents of CNS oxygen toxicity increase when the patient is in the water. The patient should use a full-face mask to decrease the problems associated with an oxygen convulsion. The volume of oxygen required to complete this treatment in water is about 300 cu ft. Using a rebreathing apparatus would reduce consumption to about 300 liters, but the patient must be trained in the use of a rebreather. As water removes heat 25 times faster than air, a patient should also have adequate exposure protection. Hypothermia is a major concern.

If there is no recompression chamber in a reasonable proximity, a stricken diver could be placed on 100% oxygen and brought to not greater than 30 fsw (9 msw). There should be some means of ensuring the patient is not able to descend deeper than 30 fsw (9 msw). A flat bottom is best for this purpose, however surge may be a factor if swells are high enough. The patient should stay a minimum of 60 minutes and a maximum of 90 minutes. The patient should ascend to 20 fsw (6 msw) and stay for 60 minutes. Repeat the 60 minute stop at 10 fsw (3 msw). Patients should continue to breathe 100% oxygen en route to the nearest hospital or hyperbaric facility.

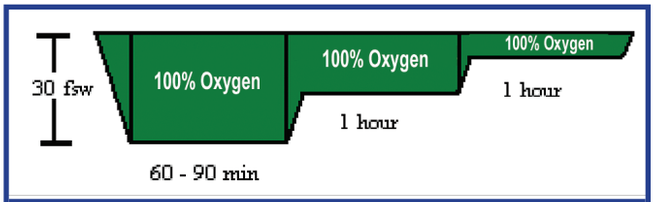


FIGURE 7-6: IN-WATER RECOMPRESSION

THE EFFECT OF COLD ON DCS

As most divers learned in the open water class, water conducts heat away from the body 25 times faster than air. However, heat escapes from the body several ways when in the water. Convection, conduction and evaporation are the methods of heat transfer although respiration is also applicable. Heat loss by convection occurs when warm air surrounding the body is pushed away by moving cool air. While this is not directly applicable to diving, indirectly, divers whose body parts come out of the water for periods of time (such as the head when surfacing) would be susceptible to convection.

A diver can get colder much more quickly when diving than he or she would in the same air temperature. This is due to conduction or the transfer of heat via direct contact. A diver can easily become chilled and then hypothermic in water whose temperature is less than 98.6°F (37°C) because the body is in direct contact with the water.

Courtney Platt & DiveTech

Robert Hew decompressing with squid family at Cobalt coast, Grand Cayman

Heat loss through evaporation is needed to regulate your body temperature in hot weather or when a diver is working hard. In cold conditions, evaporation can quickly suck away warmth, especially if you've been active and then are stationary, like when you are on the bottom working and then while hanging on decompression. Evaporation removes heat (***energy***) from the body as water is converted from liquid to gas. For this reason it is very important for deep divers to wear appropriate thermal protection to include (if wearing a dry suit) underwear that wicks water from the skin.

A primary indication of mild hypothermia in divers is uncontrollable shivering. Other indications include blue color and numbness. More severe signs include lack of coordination, weakness, weak pulse, confusion and death. Prior to a diver becoming cold enough to shiver uncontrollably, they should discontinue diving. Appropriate exposure protection should be used with consideration to water temperature, thermoclines and duration of dive.

Divers that are planning long exposures, such as those found in technical diving, should be adequately protected from the cold. This is particularly important when decompression diving. During a deep dive, the inert gas is absorbed while at depth when the diver is relatively warm because they just started the dive and they are working. The diver consequently becomes cold during decompression because of the reduced work with respect to being on the bottom and the duration of time in the water. When a diver is very cold, the body's protection system will shunt the blood to the extremities and heat the core. Because of the body's natural ability to protect the core, the diver will not have the same circulation to the extremities and there fore will not decompress efficiently which could lead to DCS. Wearing a hood on decompression dives is an excellent method for stopping heat reduction. Rebreather divers maintain a significant advantage with respect to cold temperatures because they are breathing a warm and moist media. Breathing media such as this promotes heat retention.

Conclusion

Since we do not know what the true cause of DCI is, it is difficult to prevent. Some people who have made seemingly innocuous dives have suffered from this malady, albeit others who have "earned" hits by skipping stops have gotten away without being bent. The bottom line with any

Points to Remember

Risk Factors

- **PFO**
- **Previous DCS or areas with previous problems**
- **Age**
- **Body fat**
- **Post dive exercise**
- **Body temperature**
- **Workload at depth**
- **Hydration level (*Largest Contributor*)**

MATTI ANTTILA

WINTER DIVING IN THE ICE & SNOW HAS IT'S OWN SPECIALIZED DECO NEEDS

DCI is that the symptoms should be treated. The cause is not relevant to treatment. The suggestions herein are merely suggestions. Your local Diver's Alert Network or hospital should be consulted to ensure you are affecting the correct type of treatment. None of these treatments should be performed without proper training. Divers should err on the side of safety when it comes to deep decompression and become as learned on the current theories as possible in order to make the best decisions in a bad situation.

MATTI ANTTILA