

Pathophysiology, treatment and aeromedical retrieval of SCUBA – related DCI

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Introduction

The aviation and underwater environments both present unique demands on human physiology. Humans are well adapted to live on land, usually at low altitudes. With the invention of self contained underwater breathing apparatus (SCUBA) humans have been exposed to an environment which has significant physical, physiological and psychological effects on the human body. An aeromedical clinician should have a sound knowledge of these effects as aeromedical retrieval of injured divers is relatively common. When victims of SCUBA accidents are transported via aircraft the patient can be exposed to further injury. This article explains the physics and physiology involved in SCUBA diving and discusses the more common diving related problems. The aeromedical transfer of patients suffering from decompression illness (DCI) is discussed with transport recommendations provided. In addition, there are controversies involved in both aviation and underwater medicine. Several of these issues are also discussed.

Clarence: Methought I saw a thousand fearful wracks; a thousand men that fishes gnawed upon; wedges of gold, great anchors, heaps of pearl, inestimable stones, unvalued jewels, all scattered in the bottom of the sea. Some lay in dead men's skulls; and in the holes where eyes did once inhabit there were crept, as t'were in scorn of eyes, reflecting gems, that woo'd the slimy bottom of the deep, and mocked the dead bones that lay scatter'd by.

The Keeper: Had you such leisure in the time of death to gaze upon these secrets of the deep?
William Shakespeare - King Richard 111¹.

Definitions

Decompression illness (DCI)

The term decompression illness (DCI) refers to a group of conditions which may arise when there is a reduction in ambient pressure on the body². This can occur when an aviator travels to altitude - either in an aircraft or a decompression chamber. However,

it more commonly arises following a SCUBA dive to depth³. DCI comprises decompression sickness (DCS), which is caused by the formation of bubbles of inert gas (nitrogen) within body tissues, and arterial gas embolism (AGE) which occurs with the entry of gas into the arterial circulation. AGE may occur either as the result of pulmonary barotrauma (PBT)⁴, or when the venous bubble load exceeds the ability of the pulmonary vascular bed to act as a filter of evolved gas^{5,6}. Alternatively AGE may occur when venous gas bubbles move directly from the venous to the arterial circulation via a right to left shunt, such as occurs with a patent foramen ovale (PFO)^{6,7,8,9}. Cerebral arterial gas embolism (CAGE) is the most serious form of AGE. Sub-atmospheric DCI is that group of illnesses that may arise during or following exposure to sub-atmospheric pressure, as may occur during flight and within a hypobaric chamber².

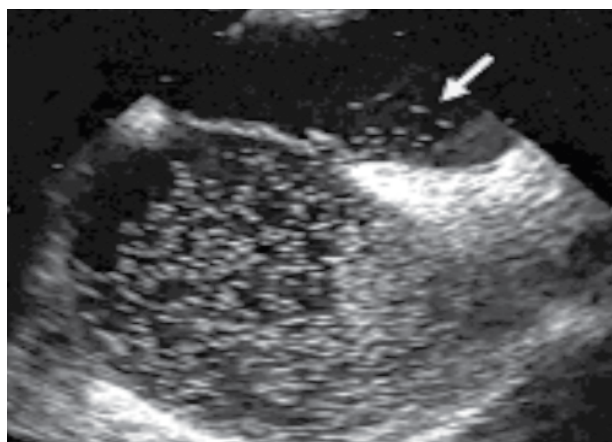


Figure 1. Bubble-contrast injection during Valsalva manoeuvre. The "flap" of the foramen ovale is opened and bubbles are seen crossing from the right atrium to the left atrium (arrow) - the same phenomenon occurs in DCI, permitting VGE to enter the arterial circulation.

<http://www.emedicine.com/ped/topic2494.htm#section-pictures>

Decompression sickness (DCS)

Decompression sickness (DCS) is a multi-system condition arising from the evolution of gas molecules that are normally dissolved in tissues¹⁰. The principle component of these bubbles is the physiologically inert gas nitrogen. Nitrogen bubble formation usually

occurs as a result of inadequate elimination of dissolved inert gas during the ascent from a SCUBA dive^{4,5,11}. Venous bubbles may form de novo or result from the intravascular release of tissue bubbles⁵. When the venous gas emboli reach the lungs they are usually filtered at arteriole level. However, if the pulmonary arterial pressure (PAP) rises - which is usual after SCUBA use - the bubbles may pass into the arterial circulation⁶. These bubbles then travel to vital organs such as the nervous system and lodge in arterioles and capillary beds, leading to the development of DCS symptoms. Bubbles can also arise spontaneously in tissues, and these are called autochthonous bubbles. Autochthonous bubbles are more likely to form in tissues with high gas content and poor perfusion (such as spinal cord white matter, adipose tissue and periarticular tissue)⁵. DCS may also present with constitutional symptoms including headache, fatigue, malaise, anorexia and pain which is poorly localised¹⁰.

Pulmonary barotrauma (PBT)

Pulmonary barotrauma (PBT) occurs during SCUBA diving when expanding gas in the alveoli is unable to escape through the airways^{11,12}. When a SCUBA diver who has been breathing compressed gas at depth ascends, the gas within the lungs must be allowed to escape. If the diver fails to exhale, or if there are local pockets of trapped gas in cysts or bullae, the trapped volume of air will expand in accordance with Boyle's Law. This leads to tearing of the lung parenchyma and egress of air into either the:

- Pulmonary venous system;
- Perivascular sheaths, causing mediastinal emphysema; or the
- Pleural cavity, causing pneumothorax^{4,11}.

PBT is thought to be more likely to occur between adjacent expanding areas of the lung which have non-heterogenous compliance^{6,13,14}. Pulmonary barotrauma is most likely to occur close to the surface, as the greatest rate of pressure change occurs in this zone¹². PBT has been reported following use of SCUBA at depths as little as 1 metre of sea water (1 msw)⁴.

Arterial gas embolism (AGE)

Arterial gas embolism arises following PBT. The gas which enters the pulmonary veins is rapidly returned to the left side of the heart and then redistributed according to buoyancy around the body - most commonly into one of the middle cerebral arteries⁶. AGE most commonly gives rise to neurological symptoms, with a range of reported symptoms including:

- Subtle alterations of higher level functioning;
- Motor and sensory abnormalities;
- Paralysis;
- Seizures;
- Unconsciousness; and
- Death⁴.

The injury pattern from AGE is typically triphasic, with a sequence of:

1. Temporary neurological dysfunction, followed by
2. A period of recovery; then
3. Further deterioration due to emboli occlusion of vessels, endothelial extravasation, platelet activation, coagulopathy and brain lipid peroxidation⁶.

Studies of cerebral artery air emboli (CAAE) have shown that injury severity and the rate of bubble absorption is proportional to the bubble size - with microscopic bubbles taking shorter times to be absorbed than macroscopic bubbles¹⁵.



Figure 2. Fatal arterial gas embolism in a diver who was brought to the surface after running out of air at 200 feet of sea water. Black arrows mark air casts of the aorta, axillary, carotid arteries and left ventricular outflow tract.

From: Barratt: Neurologist, Volume 8(3), May 2002.186-202

Historical considerations

Bert and Haldane

Paul Bert and J.S. Haldane are considered the fathers of diving medicine. Bert showed that DCS was due to the formation of gas bubbles in the body. Further he suggested that DCS could be averted if the diver rose to the surface gradually. He also demonstrated that the pain from DCS could be relieved by a return to depth. Haldane made the observation that a diver could be recovered from depth, provided he stopped in stages to allow absorbed nitrogen to pass out of the body. Haldane developed the first dive tables⁵.

Decompression sickness history

Robert Boyle exposed experimental animals to the effects of hyperbaric and hypobaric conditions, and produced the first description of DCS in 1670, from observations of a viper subjected to hypobaric pressure⁵. During the 17th century in England, the first pressure vessels large enough to hold people were constructed. Air was pumped into these vessels and they were strong enough to hold air under pressure. Patients with a variety of disparate medical conditions were “treated” in these chambers. With the development of caissons in bridge construction, caisson disease or the “bends” was described. The bridge workers were working in the air filled caissons at depths of up to 70 feet (or four Atmospheres Absolute - ATA). The workers developed joint pain due to nucleation of dissolved nitrogen in their joints¹⁶. The death rate from DCS incurred during caisson work was high, and it was Moir in 1896, who developed recompression treatment - effectively reducing the fatality rate from 25% to 2%⁵.



Figure 3. Cunningham's pressure chamber for arthritis, Kansas City 1925. Patients were placed in the chamber for up to a week, and meals were served at a dining table.

Photo - Waters RM. Cunningham Theory Revisited. *Anesthesia & Analgesia*, 44(4): 469-473, July/August 1965.

In 1921 Dr Orville Cunningham built a multi-place hyperbaric chamber which was 64 feet in diameter. This chamber contained compressed ambient air at pressures up to 50 psi *above* atmospheric pressure. Patients were placed inside for up to seven days (including a two day recompression schedule). The chamber was used to treat a variety of diseases which were thought due to an unknown anaerobic organism. One condition specifically mentioned was “post-menopausal arthritis”^{16,17,18}.

During World War II there was intensive research into both aviation and diving medicine. This research probed the boundaries of human endurance in high-altitude flight and deep sea diving. Military involvement in aviation and diving medicine remains strong to this day^{19,20,21}.

Physics, physiology and pathology

Decompression sickness

Henry's law states that the concentration of a gas in solution is proportional to the partial pressure of that gas at the gas - solution interface. Breathing gases in SCUBA diving are compressed to pressures equal to the surrounding water pressure, and therefore the partial pressure of gases in the mixture increase. Tissues take up oxygen and nitrogen at elevated partial pressures, and if these partial pressures are high enough the diver can experience oxygen toxicity or nitrogen narcosis. Nitrogen is absorbed at differing rates by tissues depending on local blood supply and the solubility of the gas in those particular tissues. Some tissues are described as “fast” tissues, permitting faster absorption of nitrogen in them and examples include renal tissues and grey matter. “Slow” tissues take up nitrogen at a slower rate, and examples include cartilage and fat. The longer a SCUBA diver spends at depth the greater the amount of nitrogen that will be dissolved in their body tissues. Given sufficient time, some tissues will be fully saturated^{5,10,11,12,17}.

Upon ascent this process of gas uptake is reversed. In an ideal situation, dissolved nitrogen will remain in solution, as it travels down a concentration gradient to the lung, where it is exhaled as gaseous nitrogen. In reality, this is rarely achieved, with nitrogen bubbles being formed almost routinely upon ascent, especially in “slow” fat containing tissue and within the venous circulation^{5,10}. Doppler studies have demonstrated that venous gas emboli are frequently found in SCUBA divers^{6,8,14}. Usually the nitrogen bubbles remain “silent”, and the dive concludes without any reported symptoms. The greater the bubble load, the more likely the diver will become symptomatic. The venous gas emboli (VGE) are usually filtered out by the pulmonary vasculature, at the arteriolar level^{6,8}. The presence of a PFO, atrial septal defect (ASD) or pulmonary arteriovenous fistula all increase the likelihood of VGE entering the arterial circulation^{12,22}. The incidence of PFO has been reported as between 27 to 30% in the normal population^{7,8}. The presence of a PFO increases the risk of a diver developing DCS for a given dive profile by a factor of 2.5²³.

The pathophysiology of the clinical presentation of DCS is explained by considering the initial effects of bubbles, whereby they cause mass effect in tissues such as articular cartilage (joints) and nervous tissue (such as the spinal cord). The bubbles obstruct venous outflow and occlude arteries, as well as causing direct injury to the vascular endothelium during their transit. In addition, bubbles cause secondary biochemical effects, including activation of platelets, complement, leucocytes and the clotting cascade.

Vascular permeability is commonly disturbed leading to haemoconcentration, disturbance of microvascular flow and a breakdown in the blood brain barrier^{4,8,11}.

DCI was classically divided into Type I (non-serious/pain only) involving limbs or joints, itch, skin rash and localised swelling, and Type II (serious) involving the CNS, inner ear, lungs and heart. DCI is now described based on the clinical manifestations of the disease^{2,5,24-26}. The classification describes DCI in terms of:

1. Acute or chronic;
2. Evolution of symptoms and signs (eg static, progressive, relapsing, spontaneously resolving);
3. Organ system(s) involved (eg neurological, musculoskeletal, skin, respiratory); and
4. Whether there is any evidence of barotrauma².



Figure 4. A diver in a Mark IV diving helmet and surface-supplied air, prepares to descend.

Photo - unknown

Arterial gas embolism

Arterial gas embolism can occur:

1. During heavy VGE loading - when the lung filter cannot absorb the nitrogen bubble load;
2. When there is a right to left cardiac shunt; and
3. When there has been pulmonary barotrauma.

Boyle's Law describes the inverse relationship between gas volume and pressure. A SCUBA diver with a lung volume of 6 litres at ten metres depth (10 msw) will

have air within his lungs at 2 atmospheres pressure (2 ATA). If he ascends to the surface without exhaling, there will now be just 1 ATA, consequently causing a doubling of volume to 12 litres. As this is not possible the diver will suffer from pulmonary barotrauma. Therefore exhalation must occur as the diver ascends to prevent rupture of lung tissue⁴.

When the lungs are fully distended the alveolar pressure is 50cm H₂O above ambient pressure. Experiments conducted on fresh human cadaver lungs in the 1960's showed that transpulmonary pressure differentials of 80 to 110 cm H₂O were sufficient to tear lung parenchyma⁴. When lung tissue is subjected to dynamic changes during pressure flux, adjacent areas of lung with non-heterogenous compliance may be sites of lung disruption. The type of pulmonary lesion that typically underlies AGE has not been identified⁶.

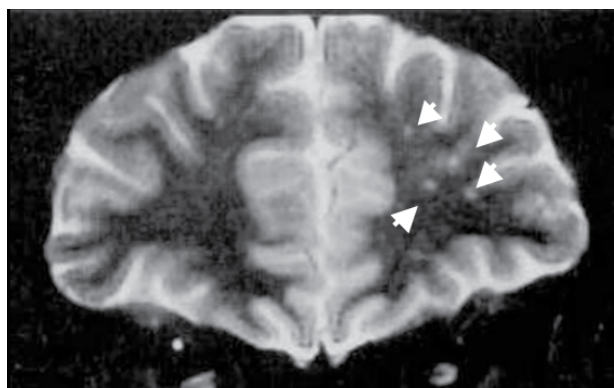


Figure 5. Coronal section magnetic resonance image showing brain lesions in a diver with decompression illness. Lesions arrowed.

From - Leach: BMJ, Volume 317(7166) October 24, 1998. 1140-1143.

Localised or generalised gas trapping may also lead to PBT and AGE^{27,28}. Lung bullae or cysts are a significant risk for barotrauma and the British Thoracic Society states that these conditions are contraindications for SCUBA use²⁹. Emphysematous blebs are also thought to be sites where PBT and AGE may occur during SCUBA use³⁰. The recommended method for detecting pulmonary abnormalities following PBT is spiral high resolution computed tomography (HRCT)³⁰. Some authors have even recommended that chest CT should be used as a screening tool during the initial diving medical conducted for professional divers^{31,32,33}.

Arterial bubbles rapidly distribute according to buoyancy and usually pass to the cerebral circulation, affecting the middle cerebral and vertebrobasilar arteries¹¹. Whilst VGE are usually trapped and resorbed in the lungs, arterial emboli pass through the cerebral circulation fairly rapidly, with over 80% of emboli passing from the arterial to the venous side of the brain within several cardiac

cycles⁶. The reason for this is the higher systolic pressure found in the systemic circulation when compared to the pulmonary circulation. In addition, the venous end of cerebral capillaries is almost twice the diameter of the arterial end (9 versus 5 microns), and the bubbles are thought to be “sucked” through the capillaries into the veins. Whilst the bubbles are transiting the cerebral circulation, they cause endothelial injury via the mechanisms previously listed. This results in neurological symptoms during the next four to five hours, despite the bubble having passed. The bubbles will remain in the cerebral circulation for a much longer interval if the CAGE victim has concurrent hypotension⁶. Treatment of hypotension should be aggressively managed to minimise the duration of symptoms, and to decrease the risk of permanent neurological dysfunction.

Treatment of DCI

Emergency treatment

The treatment of DCS and AGE has converged in recent years⁸. DCI should be suspected whenever a diver experiences pain or neurological symptoms following a dive^{2,11,12,30}. DCI can occur even following very short dives and occurrence commonly occurs despite dive tables being followed – the so called “undeserved DCI”¹¹. The principles of emergency treatment are:

- Rescue;
- Resuscitation;
- Supine posture;
- 100% oxygen; and
- Fluid loading^{2,4-6,11}.

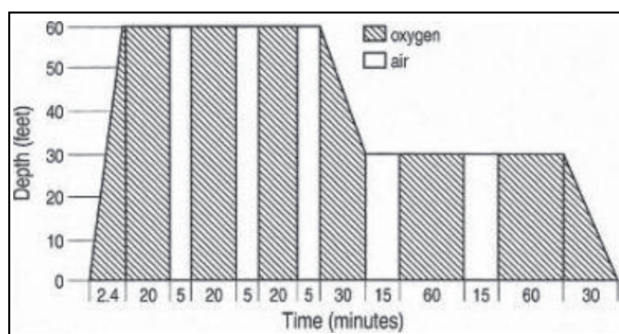


Figure 6. U.S.Navy Treatment Table 6 is the most common treatment for sport divers with DCI. Extensions, or additional oxygen breathing periods, can be added at 60 and 30 feet of sea water as needed.

From Barratt: Neurologist, Volume 8(3), May 2002. 186-202.

There is often co-morbidity due to barotrauma, hypothermia and aspiration, and these should also be borne in mind⁶. The use of oxygen at the highest inspired concentration aids in inert gas elimination by increasing the gradient between blood and tissues,

thus reducing the size and increasing the elimination rate of nitrogen^{2,4}. Nitrogen bubbles contained within the arterial circulation cause obstruction to blood flow (ischaemic hypoxia). The early washout of nitrogen bubbles minimises the hypoxic interval from vascular obstruction, as well as decreasing the time of interaction between the nitrogen bubbles with the vessel endothelium⁸. Hyperoxygenation of the blood also augments oxygenation of underperfused tissue¹². Glucose containing intravenous solution should be avoided as there is evidence to suggest that this may worsen neurological dysfunction. Transport should be provided to take the DCI victim to the nearest hyperbaric facility. If fixed wing aeromedical transfer is contemplated the flight should be conducted utilising a sea-level cabin pressure. If rotary wing transfer is used, the flight path should attempt to remain under 500 feet altitude².

Decompression therapy and adjunctive therapy

Recompression in a recompression chamber (RCC), of proven or suspected DCI is the definitive treatment, and the sooner recompression is begun the better the prognosis⁴. Divers who have had transient symptoms consistent with AGE or Type II DCI should undergo recompression to wash out any nitrogen bubbles that may remain in so-called “silent areas”⁸. The usual protocol is US Navy Table 6, which initially takes DCI victims to 18 msw (2.8 ATA) and lasts for 4 hours 45 minutes³⁴. Oxygen is provided at a concentration of 100% for 25 minute intervals, with 5 minute spells on air mix. This helps to minimise the risk of oxygen toxicity⁴.

The goals of recompression are to compress bubbles, facilitate bubble resorption and increase oxygen delivery to the tissues¹¹. The value of hyperbaric oxygen therapy in AGE and DCS is that any intravascular bubbles causing obstruction will be made smaller in accordance with Boyle’s Law. The bubbles will move to smaller vessels, or pass into the venous circulation. This minimises the extravascular tissue damage and decreases the endothelial reaction from the presence of the bubble. Hyperbaric oxygen at 2,250mmHg (300kPa or 3ATA) improves the arterial oxygen tension (PaO₂) to 2,025 mmHg (270 kPa) and tissue PO₂ to 398 mmHg (53 kPa). This greatly increases cellular oxygen supply³⁵. Hyperbaric pressure tends to collapse the bubble when it adds to the existing collapsing pressures exerted by bubble surface tension and tissue forces. Once these pressures overcome the sum of the partial pressures of the bubble gases (N₂, O₂, CO₂, and H₂O), the bubble will collapse and return to solution¹⁵.

Many adjuncts to recompression have been trialed including corticosteroids, anticoagulants, anti-

inflammatory agents and diazepam. None of these have proved directly beneficial for DCI treatment although diazepam was found useful in preventing and controlling oxygen convulsions³⁶. Intravenous lignocaine infusions have been trialled in combination with hyperbaric oxygen therapy (HBOT) and several case reports have shown dramatic improvements in refractory DCI recompressions³⁷.

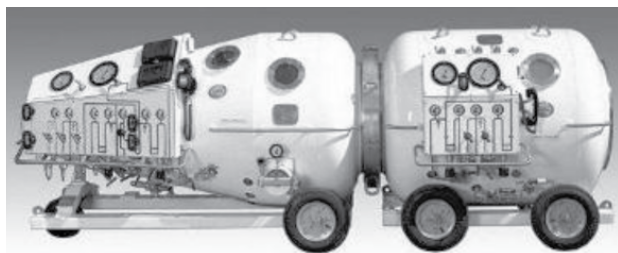


Figure 7. The Cowan dual chamber Transportable Recompression Chamber System (TRCS). The module is approved for flight in RAAF C-130 airframes. The unit is loaded either by using the wheels or via forklift. The Unit has a transfer lock and a recompression chamber.

Photo - Cowan Manufacturing, Australia.

Aeromedical considerations

DCI and the physiological stressors of flight

Divers and subjects who have been in sub-atmospheric hypobaric chambers may develop symptoms and signs of DCI for the first time when exposed to the hypobaric conditions of flight^{2,3,5}. Commercial flight typically provides ambient cabin pressures of 6,000 to 8,000 feet. Following SCUBA use, divers are generally advised to avoid air travel for a minimum of 24, and preferably 48 hours following any dive requiring decompression stops⁵. Dive computers can also calculate the no-flying interval; and these devices may compute shorter time intervals again. Exposure to sub-atmospheric ambient pressures may precipitate bubble formation and the first symptoms of DCI can occur when the diver is remote from medical assistance. Thus whenever possible, transfer of DCI victims should be conducted in aircraft that can maintain sea level cabin pressures.

The barometric pressure at MSL is 760mmHg, and this falls to 565mmHg at 8,000feet³⁸. This results in a reduction of PaO₂ from 95 to 56 mm Hg. DCI victims being transported at altitude will have decreased oxygen gradients to sustain marginally hypoxic tissues. An interesting corollary of decreasing ambient pressure, is that there will then be a relatively increased gradient to assist in eliminating retained nitrogen. Any benefit from this would be overshadowed by the heightened risk of further nitrogen bubble formation.

DCI and the physical stressors of flight

The principle physical stressor of flight is due to vibration. Rotary wing airframes and certain military fixed wing airframes produce high levels of vibration. Vibration assists in a process called tribonucleation, which results in bubbles of gas precipitating out of solution - exactly what we do not need in a DCI victim. Attention should be given to positioning the DCI patient in an area of the aircraft that has less vibration³⁹. This is usually away from the propeller line. Additional measures can be undertaken by using vibration attenuating stretcher mounts and foam vacuum mattresses (that are kept full of air); however, these clinical recommendations are not evidence based. The patient should also minimise movement, and remain supine during the flight.



Figure 8. A series of photos of a fluid filled container sitting on the floor of an airborne C-130 J AME aircraft. The photo to the left shows the pronounced vibration in the propeller line of the aircraft. The photo on the right demonstrates minimal disturbance in the aft end of the fuselage, adjacent to the DCI patient. Consideration should be given to place the DCI patient stretcher in the position with the least vibration.

Photos J. Stephenson.

DCI and the psychological stressors of flight

Air transportation is an unpleasant experience for many passengers and patients. The neuropsychological changes found in some DCI victims may be compounded by the aeromedical transfer. An explanation of the expected stressors of flight, and ongoing reassurance minimises this discomfort.

Aeromedical airframes – some considerations

Short haul aeromedical transfer (including rotary wing)

If required, primary transfer (forward AME) of a DCI victim can be undertaken. Usually this will be performed by light aircraft and helicopters. The maintenance of a sea level cabin may be problematic in light aircraft, and is usually not possible in helicopters (the Bell 222 and the Bell/Agusta BA609 tiltrotor being two exceptions to this). Vibration is pronounced in rotary wing airframes, and access to the patient is limited. Helicopter flights, at low altitude

(below 500 feet), are hazardous, especially when there are human factors influencing the flight-making decisions. Aeromedical helicopter transfers have a poor accident record⁴⁰. The requirement to conduct the mission at low altitude will only add to the risk of accident. The urgency of the transfer can be better assessed if the desk officer (aeromedical evacuation organising officer) organising the aeromedical transfer discusses the clinical scenario with the destination recompression chamber facility. Patients in remote locations with significant DCI symptoms require urgent transportation to a recompression chamber (RCC). Their movement priority will justify transportation in suboptimal aircraft.

Long haul (strategic) aeromedical transfer

Once a DCI victim has reached a RCC there is little indication to transport him further unless there are concurrent injuries. Further movement of the patient would not be considered appropriate unless there were over-riding clinical needs that could not be addressed at that RCC. Aeromedical transfer of a DCI victim requires a sea – level cabin pressure. Many aircraft are unable to provide this option.



Figure 9. AME of DCI victims often involves flight across vast distances to remote coral atolls. Tagula Island, D'Encastreaux Channel PNG, en-route to retrieve a DCI victim.

Photo - Cowan Manufacturing, Australia.

Fuel, range and altitude penalties

Aircraft maintaining sea-level cabins will use significantly larger amounts of fuel for the same distance travelled. The aircraft will have a lower than usual flight ceiling imposed by the sea-level cabin pressure. The maintenance of sea-level cabin pressure causes additional strain on the fuselage. In addition, the compressors will be unable to maintain high differential cabin pressures above this ceiling. Typical fuel penalties are in the vicinity of 30% additional fuel use, which in turn, limits take-off cargo limits,

range and speed. Typical ceilings for a C-130J are in the range of 16,000 to 18,000 feet (when a sea-level cabin is maintained). Flight at lower altitudes is not only less economical, it is slower and there will be more turbulence. An integral part of monitoring a DCI patient in-flight is to also monitor the cabin pressure. The cabin pressure should be checked at regular intervals and recorded on the patient observation record.

Recompression in-flight

There are a variety of monoplace and twin chamber recompression chambers available. They are expensive and heavy. They are usually only maintained by specialist providers such as offshore oil platforms and navies. The chambers require special aircraft loading devices and must be soundly secured. Aircraft would require a prior rating approval by aircraft engineering licensing authorities. Chambers are able to be placed in helicopters and large fixed wing airframes. Monitoring of patients in chambers is problematic in flight. Access is very difficult in twin chambers, and obviously impossible in a mono-chamber without decompressing the patient.



Figure 10. The Cowan TRCS loaded into arescue helicopter, NSW Australia.

Photo - Cowan Manufacturing, Australia.

Air travel post DCI

There is considerable variation in the advice given to DCI victims post RCC treatment. Studies have shown an increased rate of developing DCI with altitude provocation post- diving, and the risk is higher with reduced pre-flight surface intervals. A study by Barnes, Bryson and Dowse revealed that advice provided by RCC facilities to treated DCI patients on the minimum pre-flight interval varied between 72 hours and six weeks⁴¹. It would be prudent to wait at least two weeks before undertaking a commercial flight after successful DCI treatment. In more severe

cases a longer interval may be justified. The Australian Defence Force (ADF) recommends that individuals with musculoskeletal pain only DCI should not fly as passengers or aircrew for a minimum of 72 hours following complete resolution of symptoms². Patients with more severe DCI have their aeromedical disposition confirmed by consultation. With such a wide range in recommendations, a scientifically based standard policy is required following proper trials.

Controversies in hyperbaric medicine and aeromedical retrieval

DCI occurrence when using tables correctly

The aeromedical desk officer should be aware that DCI may occur in divers even when they are diving within the accepted dive tables⁴². Any symptoms that occur upon surfacing from a dive should be considered as possibly being DCI. The occurrence of DCI in divers following tables is commonly termed “undeserved” DCI¹.

The use of helicopters for aeromedical retrieval of DCI victims

Although helicopters pose a theoretical risk of exacerbating DCI by altitude and vibration exposure, it is essential that a diver with DCI reach a recompression facility. The time to reach decompression influences the prognosis of a diver with DCI. Therefore the risks of helicopter retrieval must be balanced against the gains of recompression and in some cases road transportation may be the most appropriate retrieval method if the case is less urgent.

The need for RCC treatment in patients who have recovered spontaneously from DCI

Many patients will recover from DCI symptoms with oxygen treatment only. There have been studies which show that neurological injury may coexist with DCI that appears to be musculoskeletal only⁴³. The aeromedical desk officer should not decline a request for aeromedical transfer, with justification

that the patient has recovered on oxygen alone. Apart from occult bubbles which are yet to declare with symptoms, there may well be delayed onset (third phase) symptoms from CAGE several hours post event.

Presentation times, delays and aeromedical transfer

Divers often present days after their DCI symptoms have appeared. Even with lengthy delays of up to two weeks there are still proven benefits from recompression therapy being instituted⁴². After lengthy delays, most bubbles will have resorbed, however residual symptoms from inflammation have been proven to respond to recompression. Aeromedical transfer to an RCC facility therefore remains a valid request.

Conclusion

Aeromedical retrieval of DCI victims is not an uncommon request for any aeromedical provider. Physicians involved in transferring DCI patients should have a sound knowledge of underwater medicine as well as a thorough appreciation of the stressors of flight. By combining expertise in these two areas aeromedical physicians will be able to transfer DCI victims more safely and efficiently.

Disclaimer

The views, opinions, and/or findings in this report are those of the author and should not be construed as an official policy of the Royal Australian Air Force or the Australian Defence Force.

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