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Decompression Illness Advice

Background Information

The increasing popularity of SCUBA diving and growth of commercial diving has increased the incidence of decompression illness (DCI). As more people of varying ages and fitness dive more often, helped by developments in technology to go deeper and for longer, then doctors will see more cases of this condition.

At our Hyperbaric Chamber in London we see many cases of DCI in divers who have observed all the rules and stayed within their tables or computer algorithms, but still develop DCI. No diver, diving school or independent instructor should think that they are immune to DCI.

Here we explain how it can develop, how it is diagnosed and how it is treated.

Pathophysiology

Direct effects of increasing pressure occur only on the gas filled spaces in the body. The human body is primarily made of water, which is non-compressible and transmits pressure evenly. However, the gases in hollow organs - lungs, middle ear, sinuses, poorly filled teeth, bowels, and those dissolved in the blood - are at the mercy of pressure changes.

The physical behaviour of gases is governed by the following 3 gas laws. They define the physics and problems involved in descending and ascending in water.

To understand how DCI can occur and how it is treated, a diver needs to understand these 3 laws.

Boyles Law

The volume of a given mass of gas is inversely proportional to the pressure being exerted on it (temperature remaining steady).

For every 10 metres of descent the pressure increases by one atmosphere (atm). Therefore, total lung volume during a breath-hold dive at 10 metres is one-half that at the surface. At 20 metres it is 1/3, at 30 metres it is 1/4 and at 40 metres it is 1/5. On surfacing these figures are reversed. However when breathing compressed gases as in diving the mass of gas in the lungs is increased to fill the normal volume. An ascent from 30 metres to the surface without venting (exhaling) would cause the gas, in already full lungs, with minimal ability, to expand further to increase its volume to three times normal with the greatest change occurring in the last 10 metres where it would double. This is the key law to explain pressurisation and depressurisation issues and injuries.

Daltons Law

As a diver descends the total pressure of breathing air increases in accordance with Boyle's Law; therefore, the partial pressures of the individual components of the breathing air are increased proportionally. As the individual descends deeper under water, nitrogen dissolves in the blood and is carried to all body tissues until a new equilibrium is reached. Long before this however Nitrogen at the higher partial pressures in blood alters the electrical properties of cerebral nerve cell membranes, causing an anaesthetic effect termed nitrogen narcosis. For every 15 metres of depth this is roughly equivalent to one alcoholic drink. At 50 metres divers may experience alterations in reasoning, memory, response time, and other problems such as idea fixation, overconfidence, and calculation errors.

During descent the partial pressure and hence the amount of dissolved oxygen increases. Breathing 100% oxygen at 2.8 atmospheres absolute (1.8 atm or 18 metres) may cause oxygen toxicity in as little as 30-60 minutes. At 100 metres, the normal 21% oxygen in compressed air can become toxic, because the partial pressure of oxygen is approximately equal to 100% at 10 metres. For these reasons deep divers (usually professional or military, but increasingly sport divers as well) use specialised mixtures that replace nitrogen with helium and allow for varying percentages of oxygen depending on depth. The percentage is small and provides a partial pressure which supports life and strenuous activity without inducing oxygen toxicity

Henry’s Law

With increasing depth, nitrogen in compressed air equilibrates through the alveoli of the lungs into the blood and thence into the tissues. Over time nitrogen dissolves and accumulates initially in the mainly aqueous tissues or those with a high rate of blood flow e.g. the brain, and progressively in the lipid or fatty component of tissues. On longer dives some or all tissues become 'saturated' and will not take up any more nitrogen. As an individual ascends, there is a lag before saturated tissues start to release nitrogen back into the blood. It is this delay that creates problems.

When a critical amount of nitrogen is dissolved in the tissues, reduction of pressure caused by ascending induces the dissolved gases to 'outgas' and form small but myriad bubbles in tissue cells, tissue spaces and blood. Ascending too quickly causes the dissolved gases - nitrogen - to return to gas form more quickly increasing the number and size of the bubbles and while still in the blood or tissues causing local damage which may be felt as symptoms of DCI Further reductions in pressure through flying or ascending to a higher altitude also contribute to bubble formation. The average airline cabin is pressurised only to 8000 feet or 0.8 atm. If a person flies too soon after diving, this additional decrease in pressure may be enough to precipitate bubbling or enlarge any bubbles already in existence. With or without the effects of flying If the bubbles are in the blood in some divers paradoxical embolisation may occur through a PFO.

Types of DCI

A reduction in pressure while ascending at the end of a dive will release dissolved gas (principally nitrogen) from solution in the tissues and blood and consequently form bubbles in the body.

DCI results from the effects of these bubbles on organ systems. The bubbles may disrupt cells and cause loss of function. They may act as emboli and block circulation especially in the capillaries. They may cause mechanical compression and stretching of the blood vessels and nerves.

Additionally, the blood-bubble interface acts as a foreign surface, activating the early phases of blood coagulation and the release of substances from the cells lining the blood vessels causing vasoconstriction which can worsen the effects of a blocked vessel. If not managed properly the blood bubble interface promotes an inflammatory reaction that may lead to permanent damage.

Most DCI comes on in the first few hours after the end of a dive. Some starts before leaving the water. Most cases have developed symptoms within 24 hours. Curiously it may take an affected diver 6 - 8 days or more to recognize the symptoms and seek advice and treatment.

DCI is divided into 2 categories. Type I was mild and described pain only, bends and skin bends. Type II included everything else. Now it still seems useful to use the old names but since the lines between Type I and Type II are blurred by considerable overlap, the classification is not so useful and a description of the symptoms, their speed of onset and their rate of progress are of more use to rescue authorities and to hyperbaric physicians and chamber operators. All DCI should be treated as serious even though the actual symptoms may appear mild. DCI can now be thought of as having 3 categories:

1. **Type I Typically pain in a joint or joints. Can be very painful or just niggly (hence niggles). This is uncommon in sport divers unless dives have been long. Do not ignore other symptoms such as extreme tiredness or loss of sensation placing this sort of bend in a more serious category. Resist the temptation to describe it as a mild bend.**
2. **Type II (serious) All symptoms in this category should be regarded as serious even though they seem mild. They should all be treated. A full description of the range of symptoms appears below.**
3. **Pulmonary Barotrauma and Arterial gas embolisation (AGE).**

Type I DCI

Type I DCI is characterised by
(1) Mild pains that begin to resolve within 10 minutes of onset (niggles)
(2) Pain in a joint or joints with the consequent loss of function of the joint is the classical bend named in parody of the fashionable Grecian Walk of the late 19th Century It occurs in up to 70-85% of patients with DCI.
The pain is often described as a dull, deep, throbbing, toothache-type pain, usually in a joint or tendon area but also in tissue. The shoulder is the most commonly affected joint in divers after a shallower than 40 metre dive, whereas the knees are affected more in deep divers. The pain is initially mild and slowly becomes more intense. Because of this, many divers attribute early DCI symptoms to overexertion or a pulled muscle.

Upper limbs are affected about 3 times as often as lower limbs. The pain of Type I DCI may mask neurological signs that are hallmarks of the more serious Type II DCI.

Skin bends, rashes, mottling, itching and lymphatic swelling have frequently been included in the Type I canon, However modern experience suggests that they are serious symptoms in their own right and should always be investigated and treated. They are described here for convenience.

(3) "skin bends" that cause itching or burning sensations of the skin; or

Skin rash, which generally is a mottled rash causing marbling of the skin or a violet coloured rash which is most often seen on the chest and shoulders. On rare occasions, skin has an orange peel appearance.

It is important that this is not confused with other causes of a rash whilst diving. A suit squeeze will generally have a different pattern and look more like bruising, whilst a neoprene contact dermatitis will be in areas where a suit rubs, such as the neck or cuffs.

(4) Lymphatic involvement is uncommon and usually is signaled by painless pitting oedema. This usually starts on the chest and will tend to move down the trunk over the next few days, finishing in the lower legs. At any stage if the thumb

is pressed into the swelling for 15 -30 seconds it will leave an impression. The mildest cases involve the skin or the lymphatics.

(5) Some authorities consider anorexia and excessive fatigue after a dive as manifestations of Type I DCI. We see them as serious symptoms requiring investigation.

Type II DCI

Type II DCI is characterised by nervous system involvement pulmonary, lung symptoms and circulatory problems such as hypovolaemic shock. Pain is reported in only about 30% of cases. Because of the anatomical complexity of the central and peripheral nervous systems, signs and symptoms are variable and diverse. Symptom onset is usually immediate but may be delayed as long as 36 hours.

Nervous System

The spinal cord is the most common site for Type II DCI; symptoms mimic spinal cord trauma. Symptoms may start within a few minutes to several hours after the dive and may progress to paresis, paralysis, paraesthesiae, loss of sphincter control. Girdle pain around the upper abdomen or the lower trunk is a common first symptom while higher up the cord, neck and arm symptoms may predominate. This form of DCI can be progressive and dynamic and does not follow typical peripheral nerve distribution patterns. This strange shifting of symptoms confuses the diagnosis, differentiating DCI from traumatic nerve injuries.

Other common symptoms include headaches or visual disturbances, dizziness, tunnel vision, and changes in mental status such as confusion and disorientation, loss of short-term memory and some cognitive dysfunction.

Ears

Labyrinthine or inner ear DCI (the staggers) causes a combination of nausea, vomiting, vertigo, and nystagmus in addition to tinnitus and partial deafness. Labyrinthine disturbances not associated with other symptoms of DCI should be viewed as cases of barotrauma.

Lungs

Pulmonary DCI (the chokes) is characterised by (1) inspiratory burning and substernal discomfort, (2) non-productive coughing that can become paroxysmal like a coughing fit, and (3) severe respiratory distress. This occurs in about 2% of all DCI cases and can end in death. Symptoms can start up to 12 hours after a dive and persist for 12-48 hours.

Circulatory System

Hypovolaemic shock commonly is associated with other symptoms. For reasons not yet fully understood, fluid shifts from intravascular to extravascular spaces. The problems of tachycardia (rapid heart beat) and postural hypotension (dizziness when you suddenly sit or stand up) are treated by oral rehydration, if the patient is conscious or by an Intravenous infusion if unconscious. Effective treatment of DCI requires full correction of any dehydration.

Thrombi or clots may form from activation of the early phases of blood coagulation and the release of vasoactive substances from cells lining the blood vessels. The blood-bubble interface may act as a foreign surface causing this effect.

Occasionally pain originally thought to be in the shoulder joints may be due to adverse effects on the cardiac circulation mimicking heart attacks.

Abdominal Pains

These should always be treated as serious symptoms and are usually due to spinal cord damage. It is important to keep an eye on urinary output however.

Pulmonary Barotrauma/Arteroa; Gas E,bolisation

Pulmonary overpressurisation, for example during a breath holding ascent, can cause large gas embolisation when rupture into the pulmonary veins allows alveolar gas to enter systemic or arterial circulation. Gas emboli can lodge in coronary, cerebral, and other systemic arterioles. These gas bubbles continue to expand as ascending pressure decreases, thus increasing the severity of clinical signs. Symptoms and signs depend on where the emboli travel.

Coronary artery embolisation can lead to myocardial infarction or abnormal rhythms. Cerebral artery emboli can cause stroke or seizures.

Differentiating cerebral AGE from Type II neurological DCI is usually based upon suddenness of symptoms.

AGE symptoms typically occur within 10-20 minutes after surfacing. Multiple systems may be involved. Clinical features may occur suddenly or gradually, beginning with dizziness, headache, and profound anxiousness. More dramatic symptoms of unresponsiveness, shock, and seizures can occur quickly. Neurological symptoms vary, and death can result. Central Nervous System DCI is clinically similar to AGE; since the treatment of either requires recompression, differentiating between them is not of great importance.

Pneumothorax & Mediastianal Emphysema

These two conditions resulting from pulmonary overpressure are characterized by shortness of breath and usually follow uncontrolled or poorly controlled ascents. The former may be suspected from uneven movement of the chest and hyper-resonance on percussion of the chest wall. The shortness of breath is often relieved by assuming a sitting position and oxygen. Mediastinal emphysema can be diagnosed by the crackles felt at the root of the neck. Other than reassurance this requires no treatment. The diagnosis in both conditions must be confirmed by a hyperbaric or trauma physician and appropriate investigations done.

How To Diagnose DCI

History: When you feel unwell or have any symptoms after a dive it is essential to remember that the symptoms or signs are pressure-related until proven otherwise, usually, by a diagnostic or therapeutic recompression. Therefore, the doctors will ask you about pressure exposure as an aid to the diagnosis. The following specifics about the dive will be required or elicited:

- Where was the dive location (e.g. ocean, lake, river, quarry or cave)? some dive holidays or dive boats will provide dives continuously over seven days or more. This may be important information.
- When did you first have any symptoms? Did you have any symptoms after earlier dives?
- What was the rate of ascent and the depth that you dived to?
- What were the approximate times spent at specific depths?
- What work did you do during the dive (consider currents, distance swam, water temperature, and primary activity [e.g. wreck diving, recovery])?
- What gases did you use (compressed air, rebreathing equipment, mixed gases)?
- What was your physical condition before, during and after the dive (e.g. fatigue, alcohol intake, fever, vertigo, nausea, overexertion, pulled muscles)?
- Was first aid delivered (e.g. oxygen, positioning, medications, fluids)?

You will be asked about the following symptoms:

- General symptoms of profound fatigue or heaviness, weakness, sweating, or malaise.
- Musculoskeletal symptoms of joint pain, tendonitis, back pain, or heaviness of extremities
- Mental status symptoms of confusion, unconsciousness, changes in personality
- Eye and ear symptoms of loss of vision in a particular area, double vision, tunnel vision, blurring, tinnitus, or partial hearing loss
- Skin symptoms of itching or mottling
- Lung symptoms of shortness of breath, non-productive cough, or blood in your sputum
- Cardiac symptoms of inspiratory, substernal, or sharp or burning chest pain
- Gastrointestinal symptoms of abdominal pain, faecal incontinence, nausea, or vomiting
- Genitourinary symptoms of urinary incontinence or urinary retention
- Neurological symptoms of loss of sensation (general or over a joint), paresis, paralysis, migrainous headache, vertigo, abnormal verbalisation, or unsteady gait
- Lymphatic symptoms of swollen lower limbs

Physical

Physical exam findings will include the following:

- General - fatigue, shock
- Mental status - disorientation, mental dullness
- Eyes - visual field deficit, pupillary changes, air bubbles in the retinal vessels, or nystagmus
- Mouth - Liebermeister's sign (a sharply defined area of pallor in the tongue)
- Pulmonary - rapid respiratory rate, respiratory failure, respiratory distress, or haemoptysis (coughing up blood in the sputum)
- Cardiac - rapid heart beat, low blood pressure, abnormal cardiac rhythms, or Hamman's sign (crackling sound heard over the heart during ventricular contraction)
- Gastrointestinal - vomiting
- Genitourinary - urinary bladder distension, decreased urinary output
- Neurological - loss of sensation, loss of power, anal sphincter weakness, fitting, or poor co-ordination and wobbly gait, especially on heel to toe walking.
- Musculoskeletal - subjective joint pain without objective findings, or decreased range of motion (ROM) due to muscle splinting of involved joint or tendon
- Lymphatic - lymphoedema where there is swelling of the tissues due to the blocking of the lymphatic system.
- Skin - itching, mottling/marbling, violet colour, cyanosis, or pallor
- Diagnostic techniques - Pain, frequently musculoskeletal, is present in a substantial number of DCI cases. In one series 57.8% two specific techniques that can aid the practitioner in diagnosing DCI are:

    - Placing a large BP cuff over the area of pain and inflating it to 150-250 mm Hg. In the patient with nitrogen bubbling in the joint or tendons, this increase can force some of the nitrogen back into solution, resulting in a temporary decrease in pain.
    - Milking the muscle toward the affected joint may increase pain by pushing more nitrogen bubbles toward the joint.

Differentiating between AGE and DCI

- AGE - (1) Any type of dive can cause AGE, (2) onset is immediate (< 10 - 20 min), and (3) neurological deficits primarily manifest in brain but may affect only the spinal cord.
- DCI - (1) Dive must be of sufficient duration to create a gas load in, but not necessarily saturate tissues, (2) onset is latent (0-36 h), and (3) neurological deficits manifest in spinal cord and brain.

Causes of DCI

- Predisposing causes for DCI
    - Inadequate decompression or surpassing no-decompression limits
    - Inadequate surface intervals (failure to decrease accumulated nitrogen)
    - Flying or going to higher altitude soon (12-24 hours) after diving. This increases the pressure gradient.
- Individual physiological differences
    - Obesity (nitrogen is lipid or fat soluble)
    - Fatigue
    - Age
    - Poor physical condition
    - Dehydration
    - Illness affecting lung or circulatory efficiency
    - Prior musculoskeletal injury (scar tissue decreases diffusion)
- Environmental factors
    - Cold water (vasoconstriction decreases nitrogen off-loading)
    - Heavy work (vacuum effect in which tendon use causes gas pockets)
    - Rough sea conditions
    - Heated diving suits (leads to dehydration)
- Divers who have been chilled on decompression dives (or dives near the no-decompression limit) and take very hot baths or showers may stimulate bubble formation.
- Decompression tables: Decompression sickness may occur even if the decompression tables and no-decompression limits are strictly observed. The decompression tables and no-decompression limits list the maximum time allowed for a dive based upon the maximum depth achieved. The limits take into consideration nitrogen saturation of lipid tissues. Once nitrogen has saturated tissues, a standard ascent to the surface with decreasing ambient pressure can allow nitrogen to bubble out of solution according to Henry's law. Once the no-decompression limit has been passed, one or more decompression stops are required during ascent to allow delayed diffusion of nitrogen out of the lipid tissues back into the blood. Nitrogen is then exhaled through the lungs.

These tables also include calculations based upon the surface interval between dives and residual nitrogen off-loading during the time between dives. The original tables have 3 problems:

1. The tables are based on young, healthy and fit, Navy volunteers. Since many civilian divers do not fit this profile, the tables have limitations.
2. The rapidly expanding use of dive computers takes into account the actual time spent at each depth. This allows for more time under water and removes a built-in factor that helps keep divers in the conservative range.
3. There is an increasing number of casual divers.

The best defence against DCI is diving conservatively. However, divers may still develop DCI.

Today, diving experts recommend a safety stop at 3 or 5 metres for 3-5 minutes. For no-decompression dives, this is essentially a decompression stop. Since the largest change in pressure per metre occurs in the last 5 metres just under the surface, this stop allows for additional nitrogen equilibration and off-loading.

Avoidance

It's a pretty basic rule of thumb but anything that:

1. Increases the gas load
2. Decreases off-gassing

Can contribute toward getting DCI. Tips for DCI avoidance are based on this. So remember the following:

Avoidance Tips

- Avoid exercise before and after a dive. Exercise before will make your tissues hungry for oxygen and so load them with nitrogen on a dive, Exercise after will increase the rate of nitrogen washout causing more and bigger bubbles.
- Alcohol will dehydrate you the next day, make sure you drink plenty of water before sleeping if you have been over doing the cocktails the night before.
- It's better to be too warm than too cold on a dive, especially the deco and safety stops. If you are cold your peripheral blood vessels shut down so slowing down their nitrogen release.
- Research has shown there may be a link with women diving whilst menstruating and getting DCI. Dive conservatively if it is that time of the cycle.
- The best way to dehydrate is to sit top deck of a liveaboard, drink coffee and forget about the wind dehydration factor. So don't do it. If your urine is pale yellow, you are OK, if it is that dark Van Gogh sunflower yellow, drink more water.
- Diarrohoea will reduce your circulating fluid volume so reducing your capacity to off-gas after a dive.

Treatment of DCI

Pre-Hospital Care:

All dive boats should carry 100% oxygen for use in acute DCI.

- Extricate from water and immobilise if trauma is suspected.
- If DCI is suspected or certain administer oxygen by mask.
- If the diver is conscious then rehydrate with non alcoholic fluids at a rate of 1 litre each hour.
- Never give the anaesthetic gas Entonox to a diver with DCI. As It contains nitrous oxide which worsens symptoms by increasing bubble size.
- Arrange transport eg ambulance, coastguard helicopter, to nearest emergency department and /or hyperbaric facility. Keep all diving gear with diver. Diving gear may provide clues as to why the diver had trouble (e.g., faulty air regulator, hose leak, carbon monoxide contamination of compressed air).
- Perform CPR and ACLS if required as well as needle decompression of the chest if tension pneumothorax is suspected.
- Keep the patient lying flat, and DO NOT put them in the head down position as this can worsen brain swelling.

Emergency Department Care:

- Continue to administer 100% oxygen to wash nitrogen out of the lungs and set up an increased diffusion gradient to increase nitrogen off-loading from the body.
- Keep lying flat.
- Aggressive resuscitation, and chest tube thoracotomy, if indicated.
- Administer IV fluids for rehydration until urinary output is 60-120 mls/h. Rehydration improves circulation and perfusion.
- Administer aspirin for its antiplatelet activity if there is no active bleeding. Treat patients for nausea, vomiting, pain, and headache.
- Contact closest hyperbaric facility (or DAN for referral) to arrange transfer and try to keep all diving gear with the diver. The diving gear may provide clues as to why the diver had trouble (e.g., faulty air regulator, hose leak, carbon monoxide contamination of the compressed air).
- Patients with apparently mild symptoms can have dramatic improvement and complete resolution.on oxygen alone. This improvement should not dissuade the practitioner from referring and transfer for HBO and recompression as relapses have occurred with worse outcomes permanent physical or mental disability.

HBO Treatment:

- There are several types of hyperbaric chambers ranging from small monoplace (single person) chambers to multi-place and complex multiple lock-out chambers capable of simultaneous treatment several patients with their attendants. Some chambers have the facilities to maintain critical care monitoring and mechanical ventilation.
- The basic theory behind HBO therapy is to first repressurise the patient to a depth where the bubbles of nitrogen or air are made smaller and the gas redissolves into the body tissues and fluids. Then, by breathing higher concentrations of oxygen, always with short air breaks a larger diffusion gradient is established. The patient is slowly brought back to surface atmospheric pressure. This allows gases to diffuse gradually out of the lungs and body.
- Treatment tables govern the exact combination of timing and depths. Modern diving treatment tables were developed primarily by the US Navy with some minor modifications by the US Air Force. In the UK we use similar Tables devised by the Royal Navy. The commonest table to initiate treatment is called Table 62. This takes 4 hours to complete. With early recognition and treatment over 75% of patients improve. If there are still symptoms after the first treatment then retreatment is indicated. The retreatment table is called Table 60 and takes about 1 hour to complete. Retreatment will continue on a daily basis until there is full symptom resolution or there is what we call a plateau in treatment where there seems to be no more change in symptoms from one retreat to the next.
- Even with significant delays in recognition and treatment of DCI, positive results are obtained. These delays can be up to 10 weeks!
- An important issue is transport of the patient to the closest hyperbaric facility. This is frequently accomplished by land transport; however, occasionally air transportation is required. Helicopter transport necessitates the pilot maintaining altitude at < 500 feet. Fixed-wing transport should be limited to aircraft that can maintain cabin pressure at surface 1 atm (e.g., Lear Jet, Cessna Citation, military C-130 Hercules).

DCI Denial

Having treated many patients over the years DCI denial is a real factor in divers.
A medical cliche is that "common things are common, rare things rare".

If you have been diving the week before and develop a joint pain, tingling, fatigue of numbness, then do not assume it is anything other than DCI.

It will not be a rare neurological illness; it is most likely to be a bend.

It's not that you have wrenched your shoulder lifting kit; assume it's a bend.

It's probably not a rare tropical bug; assume it's a bend.

Until proven otherwise.

Due to the nature and unpredictability of nitrogen out of solution in the body, DCI must be treated as an emergency and treated quickly

Follow Up

Admission is only indicated at an institution with HBO capability.

Complications: Residual paralysis, myocardial necrosis, and other ischaemic injuries may occur if recompression is not carried out immediately. These may occur even in adequately treated patients.

Mental effects without other neurological deficits are quite common and include short term memory loss, excessive fatigue, inability to concentrate, loss of cognitive functions and surprisingly itolerance to alcohol. If established this may be known as the *Dysexecutive Syndrome*

Prognosis:

Early symptom recognition and its acceptance prompt diagnosis, and appropriate treatment are keys to a positive outcome with all DCI. With these, a success rate of greater than 75-85% can be achieved.

What Happens Next

Flying after recompression:

Patients with joint pain, skin or lymphatic DCI who have completely resolved all symptoms after recompression can fly 24 hours after exiting the chamber.

Patients with neurological or multisystem DCI who have complete resolution with their first treatment should not fly for 72 hours after recompression.

If a longer recompression than a Table 62 has been used then 72 hours is the limit.

If there are still residual symptoms after finishing treatment then 72 hours is the limit and then only after consultation with a diving medical specialist.

Diving after recompression:

Divers who have had a successfully treated pain only DCI can return back to the water from 7 days after treatment has finished subject to the agreement of the treating physician, We do recommend that the original guidelines are for fit Naval divers, and we suggest that in others that they leave diving for at least 4 weeks after treatment of mild DCI.

Divers who have had mild tingling or numbness, fully treated on a Table 62 should wait for at least 4 weeks after recompression. This is the time, though, for Sport divers, professionals could dive again after 14 days.

If there has been incomplete recovery after initial recompression, or the DCI was more serious there should be a minimum period of 4 weeks off for fit professionals and 6 weeks for Sport divers. Return to diving should again be subject to the advice of and agreement with the treating physician who will be able to explain the long-term risks.

If there has been a serious AGE there may be doubt over whether a diver should ever dive again, and that decision should be left to a diving medical specialist.

Costs

Consultation and Treatment for DCI at LDC is free.

The costs are reimbursed by the NHS, in all cases, except where the diver is not entitled to NHS treatment e.g non-EU citizens who are non-UK resident.

Courtesy of London Diving Chamber www.londondivingchamber.co.uk

Tel: 020 7806 4000 (Ext 4445) / Emergency Number: 07940 353 816