A Simple Guide to Decompression Illness sorts it all out in one short booklet. It offers everything we need to know and more, in terms any diver can understand.

John Liddiard - Diver Magazine

A SIMPLE GUIDE TO DECOMPRESSION ILLNESS

This book will enable divers to have a better understanding of some of the medical problems and illnesses associated with diving. After reading this book you will be able to fully recognize each of the individual conditions and have a clear understanding of the best action to take in the event of decompression illness.

- For all levels of experience from novice to instructor
- **▼** Topics explained in clear non-medical language
- 🗹 Guide to initial treatment and first aid 🤝 🔓
- ✓ Includes emergency action charts and recompresssion tables
- Contains emergency contact numbers & Recompression chambers



Lee Griffiths has extensive experience working within the diving & hyperbaric industry. His positions held include Diving Safety Manager and Dive Accident Facility Manager. In 2003 he won an Award for Improving Dive Safety in South East Asia by the Divers Alert Network. He currently works at Whipps Cross University Hospital London and has over 1000 hours logged operating hyperbaric chambers.



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A SIMPLE GUIDE TO DECOMPRESSION ILLNESS

Foreword by John Lippmann

Lee Griffiths

aquapress

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by Lee Griffiths

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Foreword

One of the biggest on-going challenges facing Divers Alert Network (DAN) worldwide is to educate divers about the prevention, recognition and management of decompression illness (DCI). Divers are introduced to the signs and symptoms of DCI during their initial dive certification training, and these are discussed again in various forums, texts, magazines and certain training programs. However, despite this, a surprising number of divers still fail to realize, or maybe, fail to accept, that the symptoms they are experiencing after a dive could be DCI. Decompression illness needs to be considered as a possible cause of any signs or symptoms appearing within 24 to 48 hours after a dive, unless this can be specifically ruled out by a doctor or dive medic who is trained in dive medicine.

Although most cases of DCI in recreational divers are relatively mild and respond to treatment, serious symptoms can and do occur, and may persist. Prompt oxygen first aid and recompression will minimize the likelihood of permanent injury, however, up to 30% of divers have residual symptoms of varying severity after treatment.

Unfortunately, most divers, and many dive professionals, appear to lack adequate knowledge of important diving medical and fitness-to-dive issues, including the prevention and management of DCI. It is important for divers and dive professionals alike to realize that they do not "know it all" when they finish their training. They should be made aware that there is much more to learn, no matter what level they are at, and should be given the "hunger" and tools to continue their education.

This book, written by Lee Griffiths, is one such tool. Lee, a highly experienced dive medic, managed a very busy recompression facility in Southern Thailand for several years, and regularly assisted in the diagnosis and treatment of DCI. In this book, Lee systematically and thoroughly discusses the various "pressure injuries" faced by divers, from ear barotraumas to decompression illness, and he does so in a manner that will be understood by most divers.

This book is valuable reading for all divers.

John Lippmann

Executive Director, Divers Alert Network S.E. Asia-Pacific

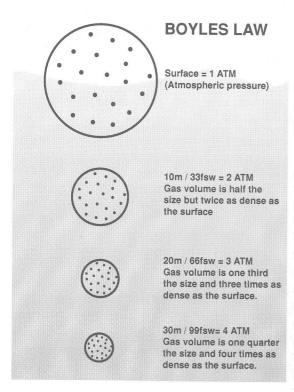
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Effects of Pressure/Mechanism of Injury

Effects of Pressure

When a diver submerges, the ambient pressure he or she is exposed to increases because of the weight of the water. The deeper the diver goes, the more water is above them, there is more weight, hence there is more pressure exerted upon the diver. Because of this, it is important that divers are familiar with the basic gas laws of physics, the most important of which is Boyle's law.



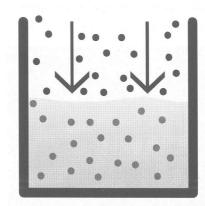
At a depth of 10m (33ft) the diver is exposed to twice as much pressure as compared to the surface at sea level. Since most body tissues are comprised mainly of liquid they are not affected by pressure changes (a liquid is incompressible). However, gases and mixtures of gases (air/heliox/nitrox) are compressible, and any gas-filled spaces of the body are directly affected by pressure changes in accordance to Boyle's law, i.e. at

10m(33ft) a gas volume is half the size it would be at the surface but will become twice as dense, at 20m(66ft) one third the volume and three times as dense and so on. Being under this amount of pressure and having our gas-filled organs squeezed is not usually a serious problem within recreational diving limits. The main concern is when the diver ascends and the gases within these spaces start to re-expand.

Mechanism of Injury

Mechanism of injury is a descriptive term used in medicine to describe how an injury occurred. A driver in a car crash may have fractured ribs. The mechanism of injury may be due to sudden deceleration and the driver's chest striking the steering wheel.

In diving illnesses, the mechanism of injury will vary depending on where the gas volumes are within the body, how they got there and the level of disruption they cause.



HENRY'S LAW

"The amount of gas dissolved in a given volume of fluid is proportional to the surrounding pressure acting upon that fluid"

The mechanism of injury for cases of DCI involves the expansion of gases and the formation of bubbles within the body. Henry's law (another important gas law) states that the amount of gas dissolved in a given volume of fluid is proportional to the surrounding pressure acting upon that fluid. This means that liquids can all hold dissolved gas. If the pressure upon the liquid is increased, as when a diver descends, more gas can be dissolved into solution. When the pressure is released, as when a diver ascends, excess gas can no longer be held in solution and is released to form gas bubbles. This in itself can be a mechanism of injury as bubbles can become trapped, block or

damage blood vessels, impair nerves and cause damaging bio-chemical changes in the blood.

Other mechanisms of injury can result from differences between gas pressure within body organs and cavities and the ambient, or the external pressure of the water, acting upon the body, as in a barotrauma of descent and ascent.

Barotrauma of Descent

Most divers know this condition as a squeeze. It results from a compression of gas in an enclosed space as the pressure increases with descent underwater. Air in non-collapsible body cavities (i.e. ears and sinuses) is compressed. It is possible to have a barotrauma of descent in a collapsible body cavity i.e. the lungs but it is indeed rare due to the relative elasticity of the lungs and constant equalization due to breathing. It may be experienced by free (breath hold) divers who dive to deep depths (in excess of 30m/100ft).

Usually barotraumas of descent are felt in the ears and sinuses but can affect any body air space, or added air space like diving masks and suits.



The word barotrauma is actually a combination of two words.

Baro - pertaining to pressure i.e. Barometric

Trauma - meaning damage to tissues by force or violence

Suit squeezes can occur when a pocket of air under a fold or fitting of a dry suit is compressed and results in the skin being pinched.

Mask squeeze occurs when the gas enclosed in a divers mask is compressed upon descent. More commonly found in new divers and free divers this condition is initially felt as discomfort around the eyes and

cheeks and bridge of the nose. If left, this can lead to pain and headache and in severe cases bruising and swelling around the eyes. This can be prevented in most cases by gently exhaling through the nose during descent.

Thoracic (lungs) squeeze as mentioned is rare. This may occur at deeper depths when the lungs are compressed to a volume of around 25% of the original volume at the surface. The current world record for a free dive is well in excess of 100m (300ft). This suggests that there may be other factors at work that are not fully understood. It is believed that it is possible to pool large volumes of blood in the chest during such dives to compensate for the reduction in lung volume.

A thoracic squeeze is characterized by initial bleeding and oedema (swelling) of the soft tissues of the lungs; this is felt as chest pain and possible breathlessness. If it continues, haemorrhage into the gas filled spaces of the lungs will occur. The diver will feel this as increased chest pain, breathlessness and explains why many professional free divers feel that they can taste blood at the deeper depths.

External ear squeeze is extremely rare but can possibly occur when there is a blockage within the ear canal. This can be caused by excess wax, otis externa (swelling due to an ear infection), a tight fitting hood or ear plugs.



A typical example of a mask squeeze showing bleeding into the whites of the eye

This then creates an air space between the blockage and the ear drum (tympanic membrane). As the diver descends and equalizes the middle ear to the ambient pressure, the tympanic membrane balloons outwards (as it would in a reverse block). This can also cause damage to the outer ear canal. This causes the diver to feel pain and sometimes nausea and vertigo (feeling sick and a sensation of rotation). If the descent is continued, bleeding into the ear canal may occur or even cause the eardrum to burst or become perforated.

Middle ear squeeze is the most common medical problem associated with diving. A squeeze of this type occurs when the Eustachian tube is blocked and fails to allow air into the middle ear, or a failure to equalize the middle ear in general. As the diver descends the ambient pressure increases pushing on the ear drum causing it to balloon inwards (opposite to an external ear squeeze or reverse block) this is both painful and potentially dangerous.

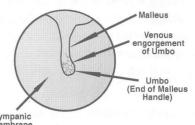
To avoid this, the diver must match the pressure inside the middle ear to the ambient pressure outside. This can be achieved by yawning, swallowing or a gentle Valsalva manoeuvre (blowing against pinched nostrils). This should be done often before pain or discomfort is felt. If large pressure differences are allowed to build up, as is the case when descending with un-cleared ears, the Eustachian tube collapses making it impossible to equalize the middle ear.



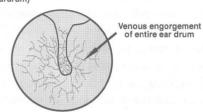
A doctor uses an otoscope to examine a divers ear. The diagrams on the next page show what the doctor can expect to see if the eardrum has been injured.

The actual amount of trauma to the middle ear can be graded using a system called the teed scale. Generally speaking there are five grades of trauma, grade five being the most severe.

TEED SCALE



Tympanic Membrane (Eardrum)

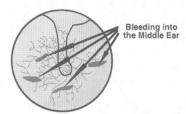


Grade 1

Pain with venous engorgment (veins fill with blood) on the ear drum around the area where the malleus ossicle touches the eardrum. (this can be seen on evaluation by a physician)

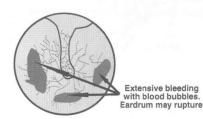
Grade 2

Venous engorgment and swelling of the entire ear drum.



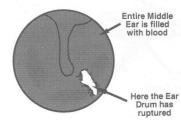
Grade 3

bleeding into the ear drum



Grade 4

Extensive bleeding into the middle ear with the appearence of blood bubbles (the eardrum may rupture)

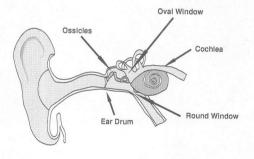


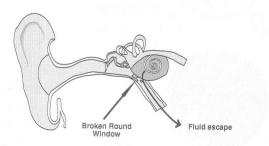
Grade 5

The entire middle ear fills with dark blood and appears completely red and may also rupture

With further descent there is discomfort, pain, nausea and vertigo. If the tympanic membiane ruptures pain relief will occur almost immediately. If water is allowed to enter the middle ear then a condition called caloric vertigo may occur. This form of vertigo is due to a thermal imbalance within the inner ear with one side being cooler than the other. The sensation can be very powerful and will not be alleviated until the water warms to body temperature.

Inner ear barotraµma is usually caused by a forceful Valsalva manoeuvre. Valsalva manoeuvres should always be gentle. Forceful Valsalva's are not recommended ard are used by divers who are having trouble clearing their ears. Rather than using a forceful valsalva it is recommended that a diver re-ascend flightly to elevate any pain or discomfort. One of the effects of a Valsalva manoeuvre is an increase in intracranial pressure conducted through the inner (fluid filled) ear by reducing venous return to the heart. The combination of increased intracranial pressure and decreased middlê ear pressure can cause the round window (and on occasions the ovel window) of the inner ear to rupture. This is also known as an inner ear bafotrauma. This may require delicate surgical repair by an ear nose and throat specialist (ENT).





Excessive and forceful 'Alsalva manoevres (pinching the nose and blowing through it) can cause the round window of the inner ear to rupture.

Sinus squeezes occur when the passages that vent the sinuses into the nasal cavity are blocked. This can be due to mucus, allergies or inflammation. Sinus squeeze presents as an increasing pain behind the nose and cheeks and or frontal headache. If left unequalised and descent is continued, it can cause intense headache and bleeding into the nasal cavity. This is common in novice divers who only notice the problem when they surface and find blood around their face or in the mask. If there are no other symptoms upon surfacing, this is unlikely to cause any medical problems, as the bleeding would have usually stopped by then. However infection may set in and this may need to be treated.

In general sinus and ear squeezes from diving are not serious because as the diver descends and feels pain they do not proceed any further but automatically ascend to relieve the pain therefore preventing serious damage, unless of course it is some form of uncontrolled descent. If symptoms such as dizziness, loss of hearing or ringing in the ears (known as tinnitus) continue then the diver should seek medical advice.

The more serious forms of ear and sinus squeeze i.e. tympanic membrane rupture, are more closely associated with submarine escape, due to the extremely rapid compression required for such an escape.

Barotrauma of Ascent

Barotrauma of ascent occurs through the reverse process of descent. If the diver has equalized the body air spaces during descent, upon ascent the gas inside these air spaces will expand due to the decrease in ambient pressure.



Inexperienced divers who panic may forget training and bolt for the surface.

In most cases these gases naturally expand and are released from the body without complication. If however these air spaces are blocked, either before or during ascent, then the pressure inside these air spaces will increase because the trapped gas cannot escape. The trapped gas will then push against body tissues in some cases causing the same type of injury as a barotrauma of descent. This is sometimes known as a reverse block.

Barotraumas of ascent are also responsible for injuries that are far more dangerous and potentially life-threatening.

Reverse blocks are not as common as squeezes but are seen from time to time especially in divers that are constantly changing depths over short periods of time for example instructors with students.

Reverse blocks occur within the middle ear and sinuses and signs and symptoms can be similar to middle ear and sinus squeeze. Reverse blocks should always be suspected when the previously mentioned symptoms occur during ascent. The causes of obstruction can be the same as for a squeeze i.e. build up of mucus, swelling etc. Reverse blocks can occur when a diver uses certain medications (usually decongestants) prior to diving. These medications can wear off during a dive and cause congestion that may trap expanding gases in the sinuses and middle ear.

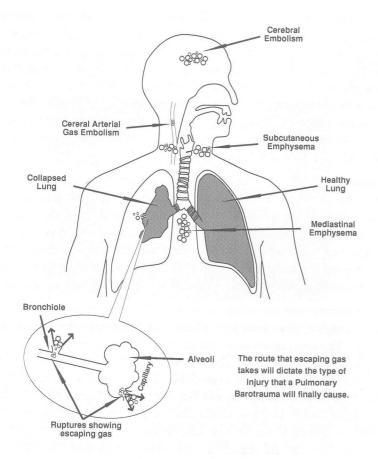
There are some other symptoms that are not as common in ear squeezes as they are in reverse blocks. When the ascent in an upright position is continued with a reverse block the pressure build up within the middle ear on the affected side will increase, but will remain the same as ambient pressure on the unaffected side. This pressure difference can cause a condition called alternobaric vertigo, a severe feeling of rotation that can be accompanied by nausea and vomiting. This should be distinguished from caloric vertigo which is caused by temperature differences as opposed to pressure differences.

This usually disappears after a few minutes and relief is often accompanied by a squeaking sound as the trapped gas escapes, or if the diver can redescend to equalize the pressure in both ears. If it occurs at depth it can be potentially hazardous as the diver may loose all sense of direction. Reverse blocks can be minimised by slow descents and adequate equalisation techniques. Divers should also avoid diving with colds or other forms of congestion and should avoid taking decongestants to relieve congestion.

Sinus barotraumas can occur at any time if the gas pressure inside the sinus cavities expands to the point where it causes damage to the surrounding tissues. This may present as an extremely painful sensation combined with a feeling of fullness in the sinus area. More advanced symptoms may include numbness or tingling (paresthesia) in the face and teeth. This is due to the barotrauma causing swelling that affects a cranial nerve. Divers who suffer a sinus barotrauma may also complain of tasting blood and, on occasion, spitting blood. This can also be found in some ear barotraumas. Treatment is often decongestants and antibiotics for possible infection. Divers with this condition should refrain from further diving until cleared to dive by a doctor familiar with diving medicine i.e. a DMO.

It is possible, but extremely rare, that during a reverse block of the sinus the bony wall of the sinus can rupture. This can allow expanding gas to enter the cranial cavity (skull) and place pressure on the brain. This condition is known as pneumocephalus.

This will lead to symptoms commonly associated with severe head injuries and can also lead to infection of the brain itself. The escaping gas from a sinus rupture can escape anywhere on the face or the orbital socket (the hole in which the eye sits). This is known as orbital emphysema. Expanding gas trapped in a poorly prepared tooth cavity filling or in tooth decay will place pressure on the pulp of the tooth, which in turn can press on the underlying nerve causing pain and headache.



Pulmonary Barotrauma (Pulmonary Over-Inflation Syndrome)

The more serious forms of barotrauma of ascent are seen in the pulmonary system (the lungs). The most common cause is when a diver ascends with a closed glottis (holds their breath). Other causes can be pathological (due to disease) and cause involuntary breath holding such as asthma or any chronic obstructive pulmonary disease (COPD).

The lung tissues can stretch to accommodate minor gas expansion and do have a degree of elasticity. However, if the pressure within the lungs increases too much, the soft tissues will be unable to cope with the expansion of gas and will rupture allowing gas to escape its natural confines.

The route that the gas takes upon escape and where it ends up will determine the type of injury sustained. In general, the expansion of trapped air within the lungs is initially felt as a gradually increasing chest pain, difficulty breathing, hoarseness or voice change and can include subcutaneous emphysema. This is simply known as a pulmonary barotrauma. These can be the initial signs and symptoms of what many divers call a lung over expansion injury but not necessarily.

It is thought that the majority of lung ruptures occur at the base of the alveoli where the tissue is generally thinner and weaker. Many physicians now believe that ruptures may occur more often in the bronchioles as they are not as elastic and do not stretch as much as the alveoli. It is possible for expanding gas to rupture the pleura (the tissue that surrounds the lungs). It is important to note that a pressure differential of just 60 -75. mmHg (0.8 -1.0 MSW) is sufficient to rupture lung tissue. This means that a diver can be injured after breathing from a scuba tank at the bottom of a shallow pool and simply standing up whilst holding his or her breath.

Mediastinal emphysema

If the route of the escaped gas takes it into the mediastinum (the gap between the lungs where the heart is) then this will cause mediastinal emphysema, also known as pneumomediastinum. Small amounts of gas in this area are usually asymptomatic (produce no signs or symptoms). If the tissues of the mediastinum are stretched too much by a substantial amount of gas, then a moderate to mild retrosternal (behind the sternum or breastbone) chest pain may be felt. This may or may not be accompanied by a sense of fullness in the chest or throat, a rapid heart rate and low blood pressure. If the gas pressure is excessive then this can severely impair the heart by pushing against it and interfering with its proper function. The gas can some times escape into the sack around the heart known as the pericardium but this is uncommon.

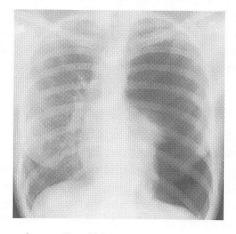
Subcutaneous emphysema

Closely linked to mediastinal emphysema is subcutaneous emphysema. Subcutaneous simply means under the skin. Emphysema is a medical term meaning an accumulation of air in the organs or tissues. This occurs when the gas from the mediastinal emphysema tracks upward to the neck area and on occasion the subcutaneous tissues of the head. Some times the gas

can scatter throughout the tissues of the chest this is sometimes called interstitial emphysema. This condition can also occur after an operation hence its other name of surgical emphysema.

Subcutaneous emphysema is not usually painful, and may only be noticed by swelling and a crackling beneath the skin (this is called crepitation) in the neck when turning the head or doing up a collar. There may also be a difficulty swallowing and people may notice a voice change. These types of conditions do not require recompression on their own, but may accompany much more serious decompression illnesses that do.

Pneumothorax / Tension Pneumothorax (Collapsed lung)



This frontal x-ray shows a collapsed left lung. It is much darker in colour than the right.

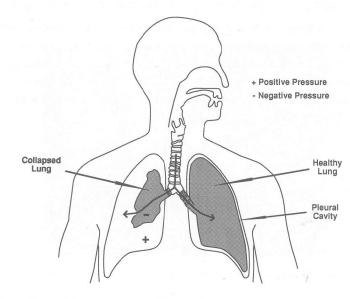
A pneumothorax is more common in conventional trauma than in a diving accident but it can happen. As mentioned previously, when the lung ruptures it may rupture at the base of the alveoli where the tissues are weaker. If the gas escapes by rupturing the lining of the lungs (pleura) this will force gas into the pleural cavity (the space between the lungs and the chest). This is a pneumothorax.

To help explain this injury it may be necessary to understand a little more about the physiology of the lungs. Both lungs are surrounded by separate pleural potential cavities. These potential cavities are normally void of any air and are an actual vacuum. This vacuum sticks the lungs to the chest wall and diaphragm in the same way that two wet panes of glass will stick together. When the diaphragm contracts and flattens on inhalation,

this cavity enables the lungs to inflate, and on exhalation allows them to deflate.

If this cavity is filled with air, or excess fluid, then this will impair the ability of the lungs to inflate.

It is possible for a pneumothorax to occur without any trauma, known as a spontaneous pneumothorax which can be caused by coughing or yawning and stretching aggressively at the same time, This is more common in tall, thin young men who have weak lung tissues or enlarged alveoli. It is worth noting that smoking and taking recreational drugs such as cocaine can in some people weaken the lung tissues and causes a spontaneous pneumothorax. People who have experienced a spontaneous pneumothorax are considered unfit to dive.



Pneumothorax: If the pressure on the injured side builds up too much it will push the collapsed lung against the heart impairing it's function.

Most healthy people who dive have large circulatory and ventilatory reserves and therefore most pneumothoraces are small and not life threatening. A pneumothorax is characterized by chest pain, which is made worse by taking a deep breath and difficulty breathing. A more substantial pneumothorax may lead to cyanosis (blueness of the extremities i.e. lips and fingernail beds), increased heart rate (tachycardia), a reduced movement of the chest on the affected side and a

tracheal shift (the windpipe moves off centre) although this is a late sign.

A pneumothorax can become life-threatening if it occupies a large part of the pleural space on the affected side or becomes a "tension pneumothorax".

A tension pneumothorax is a true emergency. It occurs when the air is allowed into the pleural space on inhalation but cannot escape back into the lung on exhalation, usually due to a flap of tissue acting as a one-way valve. Every time the casualty inhales the pneumothorax increases in size and further collapses the lung on the affected side. If left untreated, it pushes on the mediastinum, shifting it across and compressing the lung on the unaffected side. The increasing pressure on the affected side also compresses the arteries and veins going to and from the heart, which decreases cardiac output (the amount of blood the heart is able to pump).

Signs and symptoms of this type of injury include high anxiety, chest pain, cyanosis, difficulty breathing which gets worse, rapid heart rate, tracheal shift (the wind pipe moves to one side), distended neck veins and unequal expansion of the chest (tension does not fall with respiration). Divers with a pneumothorax cannot be recompressed until the pneumothorax is treated and the lung re expanded.

Arterial Gas Embolism (AGE)

If the gas from a pulmonary barotrauma of ascent escapes into the pulmonary blood vessels this will form bubbles in the oxygenated blood (arterial blood) which will travel to the heart then away to other body organs. These bubbles will flow with the arterial blood until they get lodged in the small arterioles blocking blood flow to any organs or tissues beyond the blockage. This often results in a dramatic onset of symptoms typically just prior to, immediately upon surfacing or just after.

There are two organs that are particularly susceptible to such an embolism, the heart and brain (cerebral arterial gas embolism, CAGE). This is one of the more serious forms of decompression illness. AGE is considered different to decompression sickness, but now it is thought of more along the lines of a mechanism of injury causing acute neurological decompression illness rather than a completely separate illness.



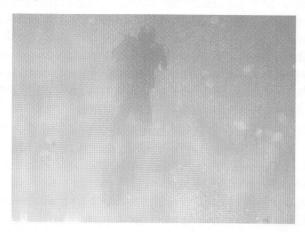
If a diver collapses immediately after diving assume the worst until proven otherwise.

AGE can cause death immediately upon surfacing, but is more likely to present as a decreased level of consciousness or complete unconsciousness within minutes of surfacing. AGE should be suspected in any diver who collapses after a

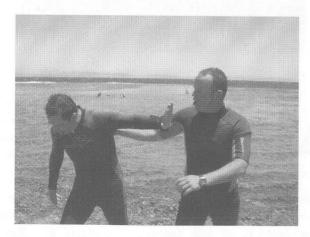
dive; even if they appear to completely recover. Relapses can occur later.

Symptoms of CAGE can closely mimic those of a stroke and may include paralysis or muscular weakness, usually affecting one side of the body, confusion, inability to talk (aphasia), blindness or other visual disturbances, severe headache, vertigo, nausea and convulsions. If the gas

manages to block the coronary arteries this may cause cardiac arrest (heart stops) or symptoms similar to a heart attack.



Commonly AGE presents with neurological symptoms because the diver usually ascends head first allowing the bubbles to move upward through the arterial circulation blocking blood flow to the brain. This is why divers suspected of having a pulmonary barotrauma of ascent should not be allowed to stand, even if they feel capable of doing so. They should lie down and remain flat unless they have difficulty breathing where they may find it more comfortable and easier to breath if they are in a semi recumbent position (sat up slightly).



It is best not to allow a diver with a suspected AGE to walk even if they feel capable of walking.

Decompression Illness (decompression sickness)

The bends, caissons disease, diver's paralysis are some of the names used to describe decompression illness. This was probably first described by Sir Robert Boyle (Boyle's law) in 1670 when he described his famous experiment in which he depressurised a viper in a vacuum noting that a bubble grew in the snake's eye. Strangely enough this disease was at first not noticed or associated with diving but in compressed air workers in bridge caissons and tunnels, hence the name 'caissons disease'. The word 'bends' first came to light in the 1870's during the Brooklyn bridge project. The project employed over 600 workers and recorded 110 cases of decompression sickness (including the main architect Washington Roebling), 3 of whom died.

After decompression, the workers had to walk up a large staircase to get to the surface. Workers often described intense joint and limb pain. They were often described as 'doing the bend' due to the posture they adopted when exiting the caisson to try to alleviate the pain. This was similar to a fashionable posture that elegant women of the time would walk with known as 'the Grecian bend'. This was shortened by workers and called the bends.

To understand decompression sickness in the traditional sense we have to understand the mechanism of injury, as previously mentioned. During compression (diving) inert gas (nitrogen/helium) from the breathing mix is dissolved into solution within the body. The greater the pressure (depth) the more gas can be dissolved from the blood into the tissues.

During decompression (ascent) the inert gas moves in the opposite direction from the tissues into the blood where it is carried to the lungs and exhaled. If this is done in a controlled manor i.e. if depth, time, decompression stop and safe ascent guidelines are followed then the inert gas tension does not reach a sufficient level of super saturation for symptomatic bubbles to form. This is true for the majority of divers, however there are secondary factors that influence the rate at which inert gas can safely be released by the body and can increase the risk of decompression illness. Such factors include, age, illness, injury, fatigue, amount of body fat, temperature, dehydration, medications and general tolerance to bubble formation can all influence the rate at which the body eliminates excess inert gas. This means that a diver does not have to do anything wrong to get decompression illness. There are only two ways to guarantee that a diver does not get decompression illness; the first is if the

diver does not go diving, the second is that if he does go diving he doesn't come back up!



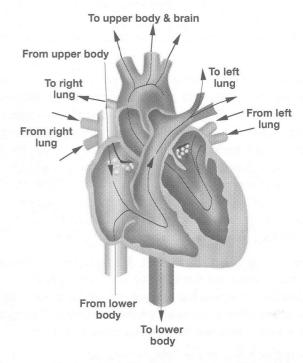
Don't share computers, a computer can only give accurate data to the person who is wearing it. If the diver in the picure was diving with his buddy but just a couple of metres deeper, the information on his buddies computer will not be relevant to this diver.

If the decompression is inadequate or incomplete, or if the ascent is too rapid then the body will not be able to eliminate this inert gas quickly enough. This increases the risk of dangerous bubbles forming in the blood and tissues. Secondary factors such as dehydration or injury and illness as well as becoming cold during the dive do affect off gassing, and may also contribute to bubble formation.

The human body is normally capable of tolerating a certain amount of bubbles within it. For example, bubbles in the venous blood are effectively removed from circulation by the lungs. Studies have shown that such bubbles exist in up to 80% of divers who have no symptoms of DCI these bubbles are known as silent bubbles. In general venous blood (de-oxygenated blood travelling from the tissues back to the heart and lungs) can tolerate a certain amount of gas bubbles well and combined with the lungs are excellent filters of gas bubbles. It is possible for the bubble burden to overwhelm the pulmonary filter and not be exhaled if the bubble burden is too great. This means the bubbles may transit the venous blood and enter the arterial blood. These bubbles will travel through the arterial blood until they reach small blood vessels and more than likely block them, or damage them as they pass through causing swelling and activating the clotting process of the blood. This damage in itself will reduce the amount of blood being able to flow through the fine blood

vessels and can cause symptoms.

As you can see we are now back to AGE but through a different mechanism of injury (not by barotrauma but by bubble formation and growth). This shows that for practical reasons the previous distinction between DCS and AGE was a little artificial and the term decompression illness, encompassing the two, is being used more frequently to describe these events.



A PFO can cause a 'shunt' and force bubbles back into arterial blood (blood going to the tissues)

The shifting of venous gas bubbles to arterial blood does not necessarily happen just in the lungs. As venous blood enters the upper chambers of the heart it is possible for it to pass into arterial circulation by means of a slight defect in the septum (the thin wall of tissue separating the upper two chambers of the heart, the right chamber carrying venous blood, left chamber carrying arterial blood). This defect, or potential hole, is known as a patent foramen ovale (PFO) and it is estimated that it can found in approximately 25-30% of the adult population. The exact consequences of a PFO on an individual diver are not fully understood, but it is believed that a PFO may be responsible for certain 'undeserved neurological hits'.

How inert gas bubbles damage tissues is not fully understood and has yet to be clarified. There are theories that bubbles may disrupt the actual structure of tissues or interrupt tissue microcirculation, it is also thought that bubbles may interfere with the biochemical activity of the tissue. It is also known that bubbles can damage the lining of blood vessels causing them to leak as mentioned earlier. Bubbles may form in some types of tissue with little effect causing no symptoms of DCI and doing little damage such as adipose (fat) tissues. However, other tissues are much more sensitive to bubble formation which can lead to tissue dysfunction. This is particularly true of nervous tissue, as nerve cells cannot reproduce themselves if destroyed.

Decompression illness can interfere with the proper function of most body tissues and organs and thus the signs and symptoms of decompression illness can vary widely. The previous system for identifying and recording cases of decompression illness relied on grouping it according to the anatomical site and presumed mechanism of injury (how the gas got there). Previously Decompression illness (DCI) was grouped into 2 areas and labelled as decompression sickness (DCS). DCS Type 1 (mild skin and pain only), DCS Type 2 (serious, Neurological symptoms) A third type (DCS Type 3) was sometimes used to describe an arterial gas embolism and DCS. It was also known as biphasic DCS.

These terms are still sometimes used, but it is now recognized that symptoms may coexist and that a person may have Type One, Two and Three DCS at the same time. It is also recognized that a person may have type one symptoms that progress to type two symptoms. This meant that the system was of limited use from a diagnostic point of view.

The newer system relies on a descriptive protocol:

- (A) Onset: (After the dive)
- Acute (rapid)
- Chronic (slower, over a longer period of time)

(B) Evolution:

- Progressive (getting worse)
- Static (staying the same)
- Spontaneously improving (getting better)
- Relapsing (getting worse after getting better)

(C) Manifestation:

- Pain
- Neurological (CNS/PNS)
- Pulmonary (lungs)
- Cutaneous (skin)
- Lymphatic (lymph nodes/glands swelling)
- Constitutional (non specific i.e. headache, nausea, etc.)

The newer system may describe the illness as this:

Acute, progressive, neurological decompression illness.

Onset

Evolution

Manifestation

Acute

Progressive

Neurological

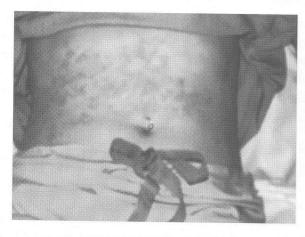


The onset of classical decompression illness symptoms is usually in the order of 20 minutes to 48 hrs post dive with the majority of serious symptoms occurring within the first four hours after diving. This does not mean that serious symptoms cannot occur after four hours. Most diving physicians agree that in most cases of DCI, symptoms appear to start within 24 hours after surfacing from a dive.

The symptoms of DCI can be categorized into six groups.

- Pain
- ii) Neurological
- iii) Pulmonary
- iv) Cutaneous
- v) Lymphatic
- vi) Constitutional

Pain is a very common manifestation of DCI, usually presenting as a deep ache in the limb or joint, which may begin during decompression or upon completion of a dive. Following a bounce or no stop/no deco dive the upper limbs tend to be involved rather than the lower limbs. In saturation divers it is the lower limbs that appear to be affected. Minor pains that seem to move from joint to joint are often referred to as 'niggles'. Limb and joint pain in saturation divers is usually felt in the lower extremities and knees. There is usually no sign of injury in the area that the pain is felt but occasionally there may be some skin rash over the affected area. Usually the more classical signs of skin and muscle injury such as swelling, tenderness and warmth to touch are not seen.



Cutis mamorata is a skin rash seen usually around the abdomen and indicates a potentially serious case of decompression illness.

Pain only DCI can resolve completely without treatment with 12-72 hours, but a physician must evaluate a patient to rule out any other complications such as neurological involvement as pain is often just one sign or symptom of many others. This is necessary as pain only cases can progress to much more serious conditions.

Neurological symptoms can be minor and major ranging from numbness and tingling, which is another common symptom, to loss of consciousness and paralysis, visual disturbances, sensory defects and higher function abnormalities such as mood swings and memory loss. DCI can affect both the peripheral nervous system (nerves) and the central (brain and spinal cord) nervous system.

Spinal cord involvement with neurological DCI is not uncommon. This

often presents with other symptoms. These symptoms are commonly associated with deep, short dives with rapid ascents. Symptoms may occur shortly after surfacing. About half of the more serious cases start to experience symptoms within ten minutes, but neurological symptoms can occur later and be just as serious.

Onset of symptoms may, but not always, start with girdle pain shortly followed by pins and needles, numbness, muscular weakness and paralysis. Using a neurological survey it is possible to locate the point within the spinal cord that is being affected. It is also possible for spinal cord involvement to affect sphincters especially within the bladder. This is often first noticed as an inability to urinate and progresses to a distended (enlarged) bladder. It is worth noting that less than 10% of serious cases show symptoms more than four hours after a dive and that most initial symptoms seem to start within 6 hours. These are just statistical observations.

The audiovestibular system is another sub class of neurological DCI resulting from bubble formation within the inner ear. Bubbles may form in any part of the fluid filled inner ear including the auditory nerve. Symptoms include nausea, vertigo, tinnitus (ringing in the ears) and nystagmus and loss of hearing. All of these symptoms are common in ear barotraumas, which is why a physician should examine any diver suffering from ear barotrauma for signs of DCI unless the dive profile makes it highly unlikely.

Pulmonary DCI, often referred to as the 'chokes' is associated with very deep dives or omitted decompression where a large gas burden is present. As previously mentioned the venous blood is a very good filter of gas bubbles. If however the decompression after such a dive is rapid or omitted then bubble formation in venous blood becomes too great and the lungs cannot eliminate such a large amount of inert gas. This causes a venous gas embolism of the pulmonary circulation. Although this is rare, especially in recreational divers, the symptoms usually start about 30 minutes post dive, but can be within minutes of surfacing and can be similar to those of a pneumothorax. The symptoms usually start with central chest pain or tightness of the chest, unproductive coughing which taking deep breaths or smoking may aggravate. Breathlessness and cyanosis and shortly after signs of shock will follow. If the condition progresses the patient will deteriorate rapidly with cardiovascular collapse, unconsciousness and death will follow. Pulmonary

decompression illness is rarely seen in 'undeserved hits'.

Cutaneous DCI (where bubble growth has occurred under the skin) and on its own is not considered life threatening. This is usually manifested as severe itching that develops into a rash that may turn the skin marbled or mottled. Usually, this is located around the shoulders and trunk.

On occasion the lymph nodes or lymph glands as they are also know, can become enlarged due to DCI this can produce a swollen feeling in the throat. The skin can feel thicker than normal and may have a pitted appearance similar to that of an orange peel.

There are a number of non-specific symptoms commonly associated with DCI. These are called constitutional symptoms e.g. fatigue, headache, anorexia, nausea and vomiting, and a general feeling of being unwell or lethargic. These are not regarded as minor symptoms and must be taken seriously.

Quite often though, the only way to confirm the diagnosis of DCI is to recompress the patient, to see if there is an improvement.

Initial Treatment and first aid

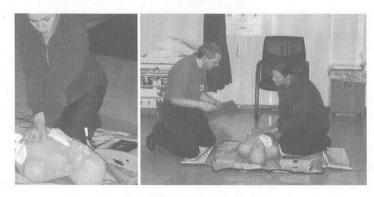
One of the main factors influencing the final outcome of a case of DCI is whether the condition is recognized and if prompt first aid is given.



DCI is mainly confined now to compressed gas divers, but tunnel workers and high altitude pilots as well as astronauts are also susceptible to the condition. Relatively speaking this is a very small group within society; this means that many

people trained in standard first aid will not recognize or know how to correctly manage DCI.

Even trained medical professionals will have a limited knowledge of the illness and the treatment it requires; and will more often than not seek to consult with physicians familiar with the condition. Many paramedics and nurses unfamiliar with DCI may even look to other divers in a group for advice. This makes the initial recognition and first aid even more crucial.



General First Aid training is an important skill for any dive professional.

Once DCI is suspected the initial steps towards managing it should be the same as for any other first aid situation. Namely, remove the victim from the water as quickly as possible. If necessary commence CPR or EAR, the later can even be started in water if the situation dictates e.g. the boat is too far away. This would obviously be a worst case scenario and standard first aid following the ABC's would apply until professional medical help arrived or the diver recovered significantly enough to be able to breathe oxygen unassisted. Divers that have had a forced ascent or suspected pulmonary barotraumas should not be permitted to stand or walk even if they feel capable. Keeping the victim lying flat may help slowdown or stop any bubble flow towards the brain. The only exception would be if

the diver is experiencing breathing difficulties, they may be able to breath easier lying semi-recumbent.



A typical divers emergency oxygen unit. This one is portable with a simple demand valve system attached.

The use of oxygen in a diving accident cannot be over emphasised. Thus all diver training agencies recommend that, as a minimum, dive leaders/instructors should be trained in the use of oxygen in a diving accident. Most reputable dive operators world wide will provide oxygen and someone trained in its use on their dive boats. If possible oxygen given by demand valve (a system similar to scuba gear) is by far the best option to give a diving accident casualty. This allows the highest possible concentration of oxygen to be inspired. It is not always possible however or practical to give a diver oxygen by demand valve. There are other options such as non re-breather masks. These masks can be given to a diver who finds it difficult to use a demand valve. The down side to this is that it will not supply as much oxygen to the diver and because the oxygen is on a constant flow this will waste some of it. Most oxygen administration units will allow both types to be used simultaneously allowing two divers to be treated from a single unit. Oxygen can also be given during resuscitation which makes the resuscitation effort on its own much more efficient regardless of the person being a victim of a diving accident or not.



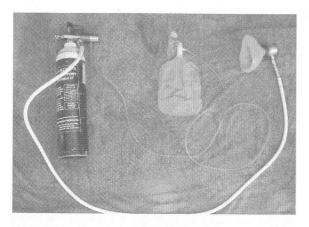
The best all round option for most diving accidents is to give oxygen via a demand valve.



If the diver has difficulty breathing or finds a demand value difficult to use. A non rebreather mask can be used and the diver can be sat up.

To fully understand how important the use of oxygen is we need to go back to the mechanism of injury of a diving accident and to understand how the body uses oxygen.

As we know most DCI's involve a formation and expansion of an inert gas that blocks blood flow and causes damage, swelling and lack of oxygen to cells. It is important to any first aider to administer oxygen as quickly as possible to reverse this as much as possible and restrict the amount of permanent damage done. The use of oxygen in a diving accident aims to allow any inert gas bubble to 'off-gas' much faster than it would if the patient were to breathe only air. It also aims to reduce any swelling that may be present. This is true for non diving accidents as well as diving accidents.



An O2 unit showing both demand valve and non rebreather systems

At one ATM of pressure the oxygen that we breathe in the air is carried in the blood mostly by the red blood cells. Almost all of these cells are approximately 95 to 98% full of oxygen in a normal healthy person.

This is when the oxygen content in the air is approximately 21% such as at sea level. So if we substitute the breathing gas, in this case air, for 100% oxygen; then the cell will quickly become completely full. The excess oxygen is now carried in the plasma (the actual blood fluid) this makes the blood much more efficient in carrying oxygen to hypoxic tissues even if less blood is actually getting to a damaged area.

In fact, under hyperbaric conditions, this process can make the red blood cells redundant and is one of the reasons that hyperbaric oxygen is used in carbon monoxide poisoning and severe blood loss.



A major contributing factor of DCI is dehydration. Divers should drink plenty of water before and after diving.

Another form of therapy that should always be considered is the giving of oral fluids or, if possible, I.V fluids (a drip). Diving in itself can dehydrate; and divers with a suspected decompression illness should be assumed to be dehydrated. DCI can cause up to two litres of water to leave the bloodstream through damaged blood vessels. Oral fluids should only be given if the diver is conscious and should be stopped if the diver cannot pass urine and their bladder has become distended or painful. Ideally isotonic sports drinks are the best to give such as Gatorade as these drinks will also supply some of the valuable salts that are also lost.

More recently, there has been talk of 'in water' recompression following a diving accident. This has been researched by some doctors and scientists especially in Australia. The main point in the use of in water recompression is that specialist in-water recompression equipment must be used as well as special dive tables and procedures. A second diver would also be needed to accompany the injured diver.

There are various problems with in water recompression such as the length of time for a treatment underwater, and the large stores of oxygen that would be required. This could expose the patient to further dangers such as hypothermia. There is also the possibility of oxygen toxic seizures which could prove fatal in a diver that is using scuba equipment. The other problems would come with medical complications and the fact that no other adjunctive treatment such as I.V fluid or oral fluid can be given. Finally in water recompression may delay getting a diver to professional medical treatment. There are reports of successful in water recompression and there are also reports where divers have died during the attempt that may have survived standard first aid and evacuation to a recompression facility.

In water recompression may have its place in the management of a remote diving accident but does require specific training and equipment. It would also need to be considered if the accident plan showed that an evacuation to a recompression chamber was not possible within a reasonable time frame and that the patient's condition is serious enough to warrant the risk. In water recompression is not considered a first aid procedure.

The use of surface oxygen however, has very few risks and does allow inert gas to off gas much faster initially that in water recompression using air only.

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As previously mentioned many healthcare professionals will have little if any knowledge of diving accidents. When a diving accident victim is handed over to medical personnel it is important to advise them on all the treatment and first aid

given as well as details of the accident. They should be advised on local dive accident hotlines and that under no circumstances should Entonox (a trade name for an analgesic gas called nitrous oxide) be given as this can expand gas bubbles. Also, no dextrose should be given either orally or as part of an I.V fluid until a diving physician has been consulted. Normal saline is the best all round I.V fluid that can be given safely to a diving accident victim. As a general rule no analgesic (pain killer) as this may mask certain symptoms. Physicians familiar with diving medicine may wish to give some pain relief usually to treat minor residual symptoms that have not resolved with recompression.

Many dive safety and training organizations provide dive accident management flow charts which help guide the first aider and even medical personnel in the correct course of action. All dive professionals should be familiar with the signs and symptoms of DCI and the correct first aid to be given. If they are working they should be familiar with the local dive accident management plan as well as local dive accident hotlines such as DAN/DES.

An appendix can be found at the back of this book giving lists of dive accident hotlines as well as oxygen administration training organisations and their contact numbers. It also gives types of courses available and amount of hours the courses last

Diagnosing DCI

Many divers believe that only a trained diving physician can diagnose a decompression illness. This it not true. Any physician can diagnose this illness. If they are not trained in diving medicine it is recommended that they contact a competent diving physician or one of the many dive accident 'hotlines' that are now available 24 hours a day, seven days a week e.g. DAN.

Diagnosing DCI starts with simple facts and a series of simple questions that need to be answered before deciding on what further testing or treatment can be given. Obviously the physician will want to know the chief complaint, which is the main problem the patient has, as well as any other symptoms. One of the simplest and most important questions however is: Has the person been diving using a compressed gas either underwater or in a hyperbaric environment?



It's not just the reflexes in the knees that are tested. Here a doctor tests the divers bicep reflex. Other reflexes include the tricep, ankle, wrist, base of the foot and sometimes under the scrotum.

This is one of the first and simplest questions a diving medical officer should ask. If the answer is yes then the next question should be how long after the exposure did the first symptoms occur? The relative time frame that most diving physicians will be looking for is up to 48 hours after a

dive. A vast majority of initial symptoms (over 90%) normally occur within 24 hours. They may also be vague, forgotten or attributed to something other than diving by the patient.

If the initial onset of symptoms seems to have occurred after a period of 48 hrs the physician may look towards other causes that may be non-diving related. He or she may look to eliminate them before considering it to be a possible DCI.

The physician will also want to know about the patients general and pre dive health. This may include:

- 1. Any allergies the patient has.
- 2. Any medications the patient is taking.
- 3. Any significant previous medical conditions or injuries that the patient may have suffered in the past.
- 4. The last time they ate or drank, and if so what and how much?
- 5. The general events leading up to the dive i.e. was the patient feeling ill the night before or suffered from lack of sleep?
- 6. Has the patient been drinking alcohol or taking recreational drugs in the previous 24-48 hours?

When it comes to the dive itself there is the obvious things such as:

- 1. Amount of dives that day and previous few days.
- 2. Type of diving i.e. no decompression or decompression diving.
- 3. The gas being breathed and mixes used.
- 4. Dive profiles and any complications such as rapid ascent or omitted decompression.

There may be other questions such as:

- 1. The type of dive table or computer used and if any decompression stops or safety stops were made.
- 2. Whether the dive was strenuous or cold.
- 3. Whether any narcosis was experienced at any time.
- 4. Were any symptoms experienced during the dive and if so at what point?

- 5. The level of experience the diver has and what was the purpose and type of dive i.e. teaching or wreck diving.
- 6. Was the dive a typical type of dive for the patient and did anything unusual or dangerous occur i.e. did anyone run out of air.

The medical staff may also wish to speak to any witnesses or dive buddies and may want to examine any computers or equipment that the diver was using.

They will also want to know if any first aid or oxygen was given and if so what was the response? Did the patient's symptoms improve, stay the same or get worse?

Other questions would include; is there anything in the patient's past that could explain the symptoms? Has the patient been exposed to anything that may cause similar symptoms or injured themselves in any other way that may explain the problem? If so are these injuries or illnesses known to mimic decompression illness and can they be quickly confirmed by other means or testing?

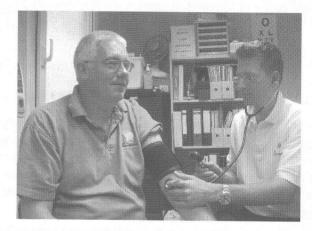




DCI can impair skin sensitivity. Here a Doctor asks the diver to close her eyes as she is lightly touched with both sharp and blunt objects. The diver is asked if she can feel the difference between the two.

A physician should make a simple vital signs survey (VSS) also known as taking base line observations; as this will give a very general view of the patient's medical condition and may give the physician some idea of how seriously ill a patient is. The survey takes just a couple of minutes and includes the patient's temperature, pulse, respiratory rate and blood pressure. It may also explain certain symptoms that a patient has i.e. a

patient having low blood pressure may explain why they would be suffering from severe tiredness and general weakness. Patients with high blood pressure may experience numbness and tingling as well as headache and visual problems; this may also be true for patients who have high respiratory rates through hyper ventilation.



A vital signs survey (VSS) is a basic but essential part of the examination of a diver.

This includes blood pressure.

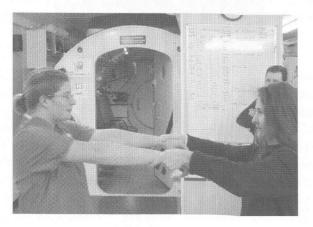
Time is important when dealing with suspected DCI and the attending physician must weigh up the "risks and benefits" of further testing for other causes of the symptoms as opposed to treating the patient quickly for decompression illness. If other possible conditions are thought to be potentially less debilitating than DCI then the physician may elect to treat the patient with recompression therapy, whilst either waiting for further test results, or just to simply "rule out" decompression illness. This may require more than a single treatment in a recompression chamber. If other possible causes of the symptoms are potentially more dangerous than DCI, such as a heart attack, then the physician may attempt to treat that condition first or in conjunction with recompression. Obviously, medical staff will try to treat multiple conditions together if possible and will always endeavour to treat patients as quickly as possible to minimise the long term effects of an illness as well as suffering. Certain treatments however, may need to be delayed until a firm diagnosis is confirmed; this is because the wrong treatment or treatment not given in a correct order may be potentially harmful to a patient. This is certainly true of a Pneumothorax, as patients with this condition cannot be safely recompressed until it has been treated.



Pneumothorax is often confirmed by a chest x-ray commonly known as a CXR; it can also be confirmed in a serious unstable patient by listening to the chest sounds with a stethoscope and percussion. Percussion is the term used to describe how a physician or medic taps the chest with his fingers to listen for different sounds that may be dull or hollow. Pneumothorax does require a

tube to be inserted into the chest wall to relieve it prior to recompression. This procedure or a similar one can be performed in a chamber in a critical emergency but most physicians will want this done outside the chamber if possible.

Another test that is quick and may be used is a blood sugar test. This will help in the possible diagnosis of hypo and hyperglycaemia which can mimic certain symptoms of decompression illness. It also helps in evaluating some patient's medical conditions like diabetes and whether they can be recompressed quickly or if they require treatment or some sugar prior to recompression. Certain other blood tests such as a complete blood count (CBC) can also be carried out to check for infection and red blood cells. The later may indicate how dehydrated a patient is. Finally in cases where chest pain is present a physician may take an echocardiogram (ECG) to rule out a possible heart attack. It is important to realise that although these tests can be done they will not confirm or rule out DCI.



Here a Doctor quickly tests strength in a divers arms.

By far the gold standard used to effectively complete the diagnosis is a neurological survey performed by the physician or under the supervision of a physician. This is still the best method of making a diagnosis of DCI as most symptoms are nerve related.

This survey tests the patients central and peripheral nervous systems for any abnormalities.

The test is split up into several areas such as:

- 1. Higher functions like memory, thought processes, mood and speech.
- 2. Cranial nerves like eye movements, hearing, facial movements and sensations on the face.
- 3. Muscle power to test for any weakness in major muscle groups.
- 4. Reflexes such as knees, elbows, biceps and feet.
- 5. Co ordination, balance and gait (how a person walks).
- 6. Skin sensitivity to such things as to light touch and pin prick as well as to temperature.
- 7. Deep sensitivity to vibration and pressure.



This test checks a divers balance. The diver must have their eyes closed as they perform the test.

The physician will also want to know of any difficulties in urination or defecation and may ask the patient to go to the toilet if possible. The evaluation takes on average between 30 minutes and an hour to complete dependent on the physician and how far he or she wishes to evaluate a certain area. The test is usually non invasive which means that physician does not need to insert anything into the body to complete the test. Some of the tests and questions asked during the test may seem odd to the untrained eye. The physician may ask the patient to do such things as subtract seven from 100 and keep subtracting until told to stop. They may ask the patient to clench their teeth and smile, stand with one foot in front of the other with arms folded and eyes closed; the list goes on. Although the tests are basic and may not seem relevant at the time to the patient, they are important and may find problems that the patient was not aware of. They are also used as a baseline as after, and during treatment, these tests may be repeated to check for improvements, relapses or deterioration.

Unfortunately there is no "magic wand" that can diagnose decompression illness with a simple test. Some divers are lead to believe that Doppler ultra sound scanners can be used to diagnose decompression illness. This is not true for several reasons; firstly it takes a trained and experienced technician to interpret the readings. In diving, Doppler can only detect bubbles in the blood stream and not in the other tissues of the body. Up to 80% of divers have bubbles in the blood stream after a dive but very few suffer decompression illness; this is another reason that Doppler cannot be used to diagnose decompression illness. It is sometimes used in cases of omitted decompression in divers with no symptoms to assess if recompression is needed due to a large bubble load. If the patient has symptoms this is not necessary as the physician will be guided by the symptoms and not the findings of a Doppler.

Of course some cases are obviously recognisable as decompression illness and do not require much initial evaluation (due to severity) before the patient is recompressed. Others may mimic decompression illness but be quickly recognised and treated by the attending medical staff. Ultimately, diagnosing decompression illness may involve classic medical detective work. The physician may have to use his or her personal knowledge and experience as well as that of others. He or she will evaluate the evidence by careful scrutiny of the incident or dive. They will also make note of time frames and symptoms as well as the response to first aid and oxygen. They will weigh up the possibility of coincidence and that the symptoms

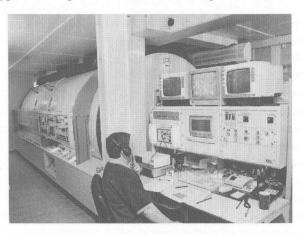
are caused by something not related to the dive. They will evaluate the results of all tests completed and if necessary recompress the patient to find out if they respond to treatment. This is called therapeutic diagnosis or rule out treatment.

Many diving physicians believe that any symptom that is encountered within 48 hours after a dive must be assumed to be caused by the dive until proven otherwise. Likewise any diver experiencing any ill health or symptoms after a dive that where not present before diving should seek medical advice from a doctor or local recompression/hyperbaric unit. At the very least they should be encouraged to contact one of the many 24 hours dive accident emergency numbers available world wide such as DAN/DES/INM.

Medical treatment of DCI and recompression.

There is still only one definitive treatment for decompression illness and that is effective recompression in a controlled environment. Although the provision of normobaric (surface) oxygen is very important as a first aid measure it is not and should not be considered as the only treatment necessary, even if all symptoms are resolved.

Effective recompression in a hyperbaric chamber in most cases is the only realistic treatment for decompression illness. Hyperbaric chambers vary in size and design as well as capability. Hyperbaric chambers are simply large containers that are designed for human occupancy and can be pressurised to equivalent depths of sea water using various gases. Generally speaking hyperbaric chambers can be broken down into two distinct types: Mono-place chambers and multi-place chambers.



A typical Mulitplace Chamber.

Mono-place chambers are small and are intended to fit a single person inside. The benefits are that they are easily installed, much more portable and require minimal manning by staff. They can also be much more cost effective to run than the larger multiplace units. The majority of the chamber being made of a clear acrylic material allows the patient to see outside. Some patients however do not like being in such a small space with limited movement at all, regardless of the acrylic.



A standard Monoplace Chamber

From a technical perspective mono-place chambers differ from multi place chambers in many ways. Firstly, they are usually pressurised by using 100% oxygen (although some manufacturers and technicians can provide the chamber with the capability to be pressurised with air) and the patient breathes this gas in the chamber as the treatment gas. Second; most mono-place chambers are limited on depth and can only be pressurised on average to no more than 3 ATA (20m 66ft). This limits their operational capability and means that patients in these chambers cannot be recompressed on tables that require a depth deeper than around 18m or 60fsw. In certain ways these chambers will give the attending physician fewer options when treating a patient. Another disadvantage of these chambers is the fact that the patient is alone in the chamber and medical staff cannot get direct 'hands on' access to the patient during treatment; so evaluating the patient during treatment is limited as are any necessary medical interventions. Communications with the patient is often via an intercom system.

Mono-place chambers are usually hospital based and used to treat other conditions that require hyperbaric oxygen therapy (HBOT). Most diving physicians would prefer to use a multi-place hyperbaric chamber for the treatment of decompression illness. The use of mono-place chambers in the treatment of decompression illness is usually because a multi-place chamber is either unavailable or several hours away. In some regions, like the United States, Mono-place chambers are common and are used to routinely treat cases of decompression illness. These chambers are usually hospital based and can allow some monitoring of the patients vital signs



Inside large chamber.



Inside small chamber.

Multi-place chambers in themselves can vary in size, design and capability but in general multi-place chambers have the ability to hold more than one patient, are pressurised with air and the therapeutic gas is breathed through a tight fitting mask or hood. A third type of chamber known as a duo-place chamber (because it can only take two people) is also classed as a multi-place chamber. They also have the ability to allow access during treatment to the patient via an attendant who accompanies

the patient into the chamber. They also allow medical equipment and personnel into the chamber as and when necessary, without interruption to treatment via a separate air lock that is independent of the main treatment compartment where the patient and attendant are situated. Many have a specialised small air lock called a med lock which is used to quickly place equipment into the main treatment compartment



Chambers can be manually operated or operated by computer.



Manual controls

These chambers have the ability to treat more than one patient, and give the attending physician more options on treatment depth as these chambers can often go to equivalent depths of 50m plus (165fsw). This also means that different therapeutic gas mixes can be given to the patient.

The down side to multi place chambers is the relative high costs involved and that more staff are required to operate the chamber (an absolute minimum would be two, one inside attendant and one operator). In general though, a team of three is deemed the minimum for treatment using a multi place chamber; one attendant, one operator and one supervisor.

After the attending physician makes his or her diagnosis and believes it is decompression illness they will prescribe a treatment table, which is a dive profile that is specific for therapeutic use in a hyperbaric chamber. These treatment tables can last several hours and in some extremely rare cases even days! A more detailed example of some of these tables can be found in the appendices to this book. It is worth mentioning at this stage that if the patient's symptoms worsen during the initial treatment table then it may be modified by extending the length of time or changed completely.

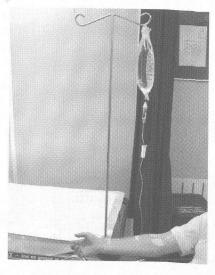


Small medical locks allow things to be placed into Multiplace chambers without having to bring the chamber to the surface.

As well as the treatment in the hyperbaric chamber, the doctor may also prescribe some other medications to either assist in the treatment of DCI or to treat other conditions that the patient may be suffering from. Such treatments that assist with recompression are called adjunctive therapies and may include I.V fluids, decongestants, or anti-convulsive drugs such

as Diazepam. Some physicians may give other drugs such as Lidocaine or steroids for very severe cases of decompression illness.

In all cases of decompression illness, the patient is assumed to be dehydrated as well as suffering from DCI. Another concern during treatment would be to ensure the patient has enough fluids as dehydration can complicate DCI symptoms when the blood thickens and cannot get into the small capillaries. If the patient can drink they will be encouraged to drink fluids such as water or oral rehydration solutions. In some cases the medical staff may place the patient on an I.V (drip) of normal saline to rapidly rehydrate the patient.

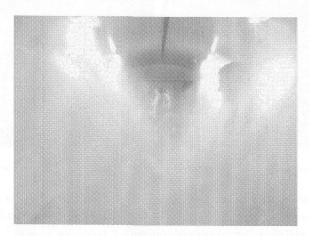


In the more serious cases of DCI medics and doctors my aggressively rehydrate the diver using intravenous infusion (a drip). Usually normal saline.

Where possible all the procedures are explained to the patient in detail as well as any possible complications that may arise. If the patient has any questions the staff are on hand to answer them prior to or during treatment.

The patient will often be required to sign consent forms and may be asked to shower and remove any personal items they have. They will also be required to change into cotton clothing that will be supplied by the chamber staff prior to treatment.

The main reason for this is safety. Any and all sources of potential ignition or fuel will be removed prior to treatment. Each chamber has its own rules



Fire suppression systems (sprinkler systems) are one of the most important safety features of any multiplace chamber.

When the treatment starts, the patient will be required to equalise; as they would for a normal dive. If they are having difficulty equalising then decongestants may be given. In severe cases where a patient cannot equalise a small hole can be made into the ear drum to allow the middle ear to equalise without the need for any such techniques like valsalva being necessary. In most cases the descent just needs to be made a little slower.

In many modern hyperbaric chambers an effective climate control system keeps the temperature at a comfortable level. In some older chambers the patient may experience some temperature changes most notably during descent and ascent where the temperature may rise and fall considerably especially if the descent is quick. This is not a cause for alarm and the temperature quickly returns to normal when the chamber reaches the treatment depth.





The two main methods used to supply oxygen to the patient. A simple face mask and a hoodtent.

Once at the treatment depth the patient will be placed on the therapeutic gas either by breathing the environmental gas if in a mono-place chamber or by putting on a mask or hood in a multi-place chamber.

The preferred gas for therapeutic use is 100% oxygen. If the depth is greater than 18m (2.8 ATA) a heliox or even a Nitrox mix is used, normally 50% Oxygen and 50% Nitrogen/Helium. Most dive professionals will appreciate that breathing 100% oxygen at depths over 6m is not recommended and viewed as highly dangerous. This is due to the toxic effect of oxygen at high partial pressures (as when we dive) that can cause convulsions and lead to drowning. The main reasons that it can be used in a hyperbaric chamber are that the chamber is dry with a breathable air source. This means that a patient who experiences a convulsion will not drown. Secondly the patient is at rest and not swimming like they would be during a dive. This makes convulsions less likely as the person is not exercising. During the treatment the patient will be given 'air breaks'. This breaks up the amount of time the patient is breathing oxygen and helps prevent complications due to the toxic effect of oxygen. In Mono place chambers where the atmospheric gas is the treatment gas, the patient will breathe air through a mask that is inside the chamber with them.

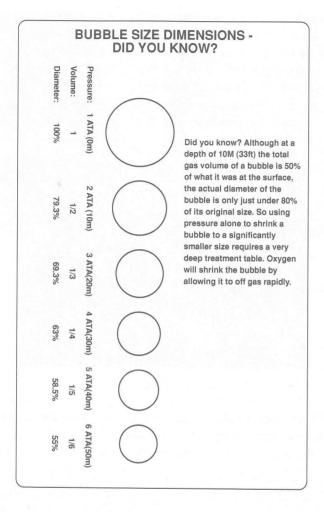
The routine in the chamber is very simple and some chambers have entertainment systems to help pass the time but generally speaking the patient is asked to relax and follow the instructions of the attendant or physician. In multi place chambers the patient may receive a little privacy when they wish to go to the toilet (a very common occurrence especially towards the end of treatment) as multi place chambers can allow people to use a small commode or urinal in the entry lock. Some multi place chambers even have a specialist toilet in them. Mono place chambers, due to their size, have limited room for movement but urinals and bedpans can be placed inside and a sheet or covering can be placed over the chamber itself to provide a little privacy.



Loading a Monoplace Chamber.

Basically the treatment aims to do two main things. The first is to stop the condition from progressing and getting worse. The second is to repair the damage that has been done to tissues and nerves.

In many cases when dealing with sport divers there is a delay in seeking medical advice and the damage may be done and the bubbles gone. This does not mean that recompression will be useless. Recompression will enable oxygen to get to damaged areas and repair them much quicker than normal and may repair areas that would otherwise not be able to heal. The hyperbaric oxygen will also greatly reduce any micro swelling that may have occurred and thus allow blood to flow through microcirculation.



If the patient is recompressed early enough then the treatment depth will also enable the bubbles to be shrunk. The main effect on the bubble will be by rapid off gassing by breathing oxygen which will shrink the bubble much faster and thus help prevent any long term damage.

As one eminent hyperbaric physician once said "Are we treating the bullet, or the bullet hole?"

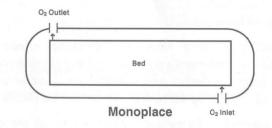
After treatment the patient may be assessed again to check for any changes in his or her condition. Patients are normally admitted to hospital or kept at the clinic as an inpatient at least for the first 24 hours after the initial treatment regardless of how minor the symptoms are or have been.

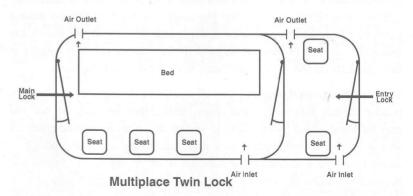
This is so that any problems that may arise can be dealt with quickly.

Even when treating people as out patients (patients that go home after treatment) the chamber staff may ask the patient to stay for a while after the treatment has finished just to ensure that there are no major relapses or deteriorations.

Most patients require more than a single recompression and as time progresses the attending physician may change the recompression treatment table as the patient improves or stabilises. The recompression treatments are usually around 12 to 24 hours apart, but initially may be very close together or even back to back for very severe cases.

Patients are made aware of things they should not do whilst being treated with recompression therapy; such as going to altitude, flying, diving or drinking alcohol. This is to allow the body to heal itself without risk of further complication or relapse.





Many patients may feel a little strange or light-headed after treatment, this is not uncommon and usually disappears after a few minutes. The patient may just require a drink or something to eat. It is worth noting that although the patient has finished the treatment for the day the hyperbaric oxygen will continue to repair damage for a few hours after treatment.

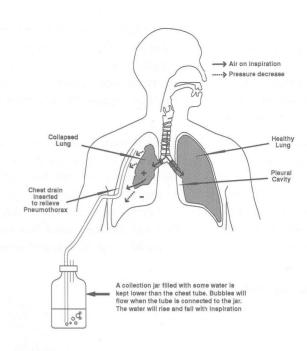
Treatment usually continues until the patient has fully recovered and has no symptoms of decompression illness or until there have been little or no improvements over a period of one or two successive treatments. This is called a 'plateau'. In most cases, more than 70 % of sport divers will have a complete recovery upon completion of the recompression therapy. For others the minor residual symptoms may subside and disappear over time. Any symptoms or disabilities that are present about a year after treatment has finished are usually permanent. Some of the key factors in why up to 30% of sport divers still have residual symptoms seem to be:

- a) Failure to recognise symptoms of decompression illness.
- b) Delay from symptom onset until recompression.
- c) Lack of first aid and oxygen administration given on the scene.
- d) Denial that the symptoms are caused by decompression illness and causing further delay.
- e) Continued diving with symptoms.

Those patients suffering from severely debilitating effects of decompression illness after treatment may be referred to a physiotherapist or neurologist for further evaluation and rehabilitation. In these cases there may be need for counselling for both the family and the patient.

Once treatment is completed, the patient is examined by the attending physician and will be told when they may fly or go to altitude, and if they can return to diving. Usually the physician will want to see the patient again in a few days or weeks time, dependent on how severe the illness was and how well the patient responded to the treatment. It is usually at this consultation that a final clearance to return to diving is given.

It is worth remembering that not all forms of diving related illnesses require recompression. Ear and sinus barotraumas certainly do not and may be complicated by exposure to further pressure. Most emphysema's do not require recompression as a rule unless they are large enough to cause severe pain or impair the function of other organs. As mentioned before pneumothorax is treated with a surgical procedure. A patient with this condition, that has other forms of decompression illness that do require recompression, must have the pneumothorax treated prior to going into a chamber.



In order to relieve a pnuemothorax the air that has built up within the chest that has collapsed the lung needs to be released by inserting a tube into the chest between the ribcage and the lung. This tube is then connected to a simple one way valve which is then connected to a collection bag or jar.

Summary

What you have just read is a very basic introduction to the many different ways in which decompression illness and barotraumas caused by diving can affect a diver. We have also spoken, in a very general way, about how DCI is treated.

Debate and research on the diagnosis and treatment of DCI still goes on, both in the medical and diving industries. It will no doubt change and be updated quicker than many divers will realize. Decompression illness in all its forms is still not fully understood by many in the diving and medical communities. The subject is filled with rumour, speculation and 'old wives tales'. The one thing that is for certain is that the more people that go diving, the more people will get decompression illness.

This may sound like doom and gloom but we have to look at this from the bigger picture. DAN reasearch has shown that a DCI occurs in approximately 1 in 10,000 dives and the majority of them are not life

threatening. This does not mean that symptoms should be ignored. Any diver having symptoms after a dive that are not readily explainable should contact a doctor familiar with diving medicine. What many in the diving medical community find is that most recreational divers who go to them for advice, do so not because they have symptoms, but because the symptoms do not go away.

In a perfect world, all divers should stick to their levels of competence and not go beyond the limits of their training. They should be fit to dive generally as well as being fit and well on the day, medical check ups aren't a bad idea either. In many countries they are mandatory for professional divers. They should not go to their limits, limits are there to stay away from, not work to. Finally if they have any concerns about their health or have symptoms after diving they should speak to a doctor as soon as possible.

Safe diving everyone!

Glossary

ACUTE ONSET: Symptoms appearing over a short period of time.

ADJUNCTIVE THERAPY: Treatment that is given as a secondary or parallel treatment

AGE: Arterial Gas Embolism; a gas bubble that travels in arterial blood until it gets lodged or otherwise damages blood vessels

ALVEOLI: Small sacs situated within the lungs where carbon dioxide and oxygen are exchanged between inspired air and capillary blood.

ANALGESIC: Pain killers i.e. Paracetamol, Pethadine, Morphine, Nitrous Oxide

ARTERIAL BLOOD: Blood carrying oxygen from the lungs to the heart, then from the heart to the body tissues.

ATA. Atmospheres absolute; the pressure in ATM including the atmospheric air pressure.

ATM: Atmospheres; a unit of measurement for pressure

ASYMPTOMATIC: Having no symptoms

BAROTRAUMA: Injury due to pressure.

BIOCHEMICAL: The chemistry of living organisms and of vital processes.

BOUNCE DIVE: A no decompression dive.

CARDIOVASCULAR: Pertaining to the heart and blood vessels

CHRONIC: Persisting for a long time.

CORONARY ARTERIES: Arteries that surround the heart, supplying it with blood enabling it to function.

CPR: Cardio Pulmonary Resuscitation

CUTANEOUS: Pertaining to the skin.

CVA: Cerebrovascular accident, a stroke.

CYANOSIS: A bluish coloration of the skin due to low oxygen in the blood.

DAN: Divers alert network, an international non profit organisation committed to providing insurance, advice, research and training in areas

of diving medicine to sports divers.

DES: Divers emergency service, a 24 hour emergency call centre based in Australia and New Zealand providing medical advice to both divers and medical professionals in the South East Asia Pacific region.

DCI: Decompression Illness; a modern term used to describe both AGE and DCS

DCS: Decompression sickness; an older term used to describe symptoms caused by inert gas bubbles forming in tissues through supersaturation.

DMO: Diving Medical Officer; A diving physician

EAR: Expired Air Resuscitation.

ENTRY LOCK: Part of a multi place hyperbaric chamber that allows people and equipment to enter a chamber without having to surface it by depressurisation.

EMBOLISM: A blockage of a blood vessel by some form of plug (Air, Fat, Clot) brought by the blood stream.

EMPHYSEMA: Accumulation of air in body tissues or organs other than the Lung.

FSW: Feet of Seawater

GAS TENSION: The partial pressure of a gas within body tissues or blood.

HBO/HBOT: hyperbaric oxygen/hyperbaric oxygen therapy

HELIOX: A gas mixture of Helium and Oxygen

HEMOGLOBIN: A component of red blood cells that carries oxygen.

HEMORRAGHE: Bleeding

INERT GAS: gas that is not used by the body i.e. nitrogen or helium.

INM: Institute of Naval Medicine, UK military medical branch responsible for diving medicine

INTRAVENOUS FLUID (I.V): Fluids given by a drip that is connected to a vein by a needle or catheter.

LYMPH NODES: A small mass of tissue where lymphocytes are produced which are part of the immune system and filtering system.

MAIN LOCK: The main part of a multi place hyperbaric chamber where the treatment is given.

MANIFESTATION: The general way in which a disease may present signs and symptoms.

MECHANISM OF INJURY: The way in which an injury occurred.

MEDIASTNUIM: The area between the lungs containing the heart, trachea, oesophagus and large blood vessels.

MED LOCK: A small air lock on a multi place hyperbaric chamber that allows small items to be rapidly placed into and out of the main lock.

MICROCIRCULATION: The flow of blood through the very fine blood vessels

mmHG: millimetres of mercury, a unit of measure for pressure.

MSW: Metres of Seawater

NAUSEA: An unpleasant sensation in the abdomen often with a feeling of imminent vomiting.

NITROX: A mixture of Oxygen and Nitrogen

NORMOBARIC OXYGEN: Oxygen given at the present atmospheric pressure

NYSTAGMUS: Involuntary rapid movement of the eye.

PLATEAU: The point in treatment where no further progress is made

PLEURAL CAVITY: The potential space between each lung and the chest wall

PULMONARY SYSTEM: Pertaining to the lungs, airways and its blood supply.

RESIDUAL SYMPTOMS: Symptoms that remain after treatment has been completed.

SATURATION (GAS): States where by the tissues have the maximum amount of gas load permissible at a given ambient pressure.

SPONTANEOUS: Immediately without due cause.

SUBCUTANEOUS: Below the skin.

SUPERSATURATION: States where by the tissues have more gas load than the surrounding pressure will allow and leads to off gassing.

TINNITUS: A noise in the ears such as ringing, buzzing, roaring or clicking

THERAPUTIC DIAGNOSIS: Using a treatment to confirm an illness.

THERAPUTIC GAS: The Gas used to treat patients in a hyperbaric chamber usually oxygen or Heliox/Nitrox.

VALSALVA MANOEUVRE: Equalizing the ears by pinching the nostrils and blowing through the nose.

VENOUS BLOOD: Blood returning to the heart usually deoxygenated from the body tissues but also oxygenated blood from lungs to the heart.

VERTIGO: A sense of rotation.

VESTIBULAR: Pertaining to the semi circular (vestibular) canals within the inner ear.

APPENDIX i

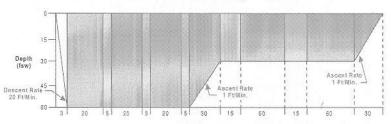
Recompression Tables

The following mentioned tables are the most commonly used for diving accidents there are others however and some hyperbaric units may modify tables to suit.

USN TT6 (US Navy Therapeutic Table 6) RN 62 in the UK

- Descent rate 20 ft/min.
- Ascent rate Not to exceed 1 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
- 3. Time on oxygen begins on arrival at 60 feet.
- If oxygen breathing must be interrupted because of CNS Oxygen Toxicity, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption
- Table 6 can be lengthened up to 2 additional 25-minute periods at 60 feet (20 minutes on oxygen and 5 minutes on air), or up to 2 additional 75-minute periods at 30 feet (15 minutes on air and 60 minutes on oxygen), or both.
- 6. Tender breathes 100 percent O₂ during the last 30 min. at 30 fsw and during ascent to the surface for an unmodified table or where there has been only a single extension at 30 or 60 feet. If there has been more than one extension, the O₂ breathing at 30 feet is increased to 60 minutes. If the tender had a hyperbaric exposure within the past 12 hours an additional 60-minute O₂ period is taken at 30 feet.

Treatment Table 6 Depth/Time Profile

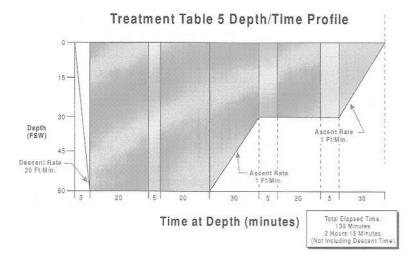


Time at Depth (minutes)

Total Flapsed Time: 285 Minutes 4 Hours 45 Minutes (Not Including Descent Time)

USN TT5 (US Navy Therapeutic table 5) RN 61 in the UK

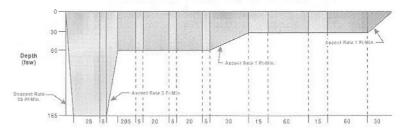
- Descent rate 20 ft/min.
- Ascent rate Not to exceed 1 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
- 3. Time on oxygen begins on arrival at 60 feet.
- If oxygen breathing must be interrupted because of CNS Oxygen Toxicity, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption.
- Treatment Table may be extended two oxygen-breathing periods at the 30-foot stop. No air break required between oxygen-breathing periods or prior to ascent.
- Tender breathes 100 percent O₂ during ascent from the 30-foot stop to the surface. If the tender had a previous hyperbaric exposure in the previous 12 hours, an additional 20 minutes of oxygen breathing is required prior to ascent.



USN TT6A (US Navy Therapeutic Table 6A)

- 1. Descent rate 20 ft/min.
- Ascent rate 165 fsw to 60 fsw not to exceed 3 ft/min, 60 fsw and shallower, not to exceed 1 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
- Time at treatment depth does not include compression time.
- Table begins with initial compression to depth of 60 fsw. If initial treatment was at 60 feet, up to 20 minutes may be spent at 60 feet before compression to 165 fsw. Contact a Diving Medical Officer.
- If a chamber is equipped with a high-O₂ treatment gas, it may be administered at 165 fsw and shallower, not to exceed 3.O ata O₂
 - Treatment gas is administered for 25 minutes interrupted by 5 minutes of air. Treatment gas is breathed during ascent from the treatment depth to 60 fsw.
- 6. Deeper than 60 feet, if treatment gas must be interrupted because of CNS oxygen toxicity, allow 15 minutes after the reaction has entirely subsided before resuming treatment gas. The time off treatment gas is counted as part of the time at treatment depth. If at 60 feet or shallower and oxygen breathing must be interrupted because of CNS oxygen toxicity, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption.
- Table 6A can be lengthened up to 2 additional 25-minute periods at 60 feet (20 minutes on oxygen and 5 minutes on air), or up to 2 additional 75-minute periods at 30 feet (60-minutes on oxygen and 15 minutes on air), or both.
- 8. Tenders breathes 100 percent O₂ during the last 60 minutes at 30 fsw and during ascent to the surface for an unmodified table or where there has been only a single extension at 30 or 60 fsw. If there has been more than one extension, the O₂ breathing at 30 fsw is increased to 90 minutes. If the tender had a hyperbaric exposure within the past 12 hours, an additional 60 minute O₂ breathing period is taken at 30 fsw.
- If significant improvement is not obtained within 30 minutes at 165 feet, consult with a Diving Medical Officer before switching to Treatment Table 4.

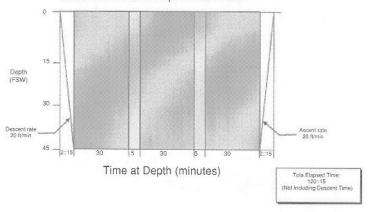
Treatment Table 6A Depth/Time Profile



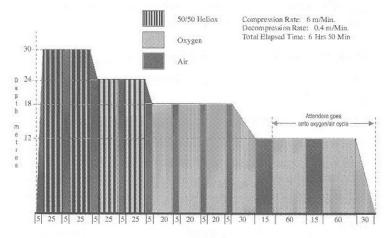
USN TT9 (US Navy Therapeutic Table 9)

- Descent rate 20 ft/min.
- Ascent rate 20 ft/min. Rate may be slowed to 1 ft/min depending upon the patient's medical condition.
- 3. Time at 45 feet begins on arrival at 45 feet.
- If oxygen breathing must be interrupted because of CNS Oxygen Toxicity, oxygen breathing may be restarted 15 minutes after all symptoms have subsided. Resume schedule at point of interruption.
- Tender breathes 100 percent O₂ during last 15 minutes at 45 feet and during ascent to the surface regardless of ascent rate used.
- If patient cannot tolerate oxygen at 45 feet, this table can be modified to allow a treatment depth of 30 feet. The oxygen breathing time can be extended to a maximum of 3 to 4 hours.

Treatment Table 9 Depth/Time Profile



MODIFIED COMEX 30



Time (minutes)

APPENDIX ii

Divemasters dive accident assessment chart

A simple chart that can be used by divemasters and instructors to compile important information about a diving accident victim.

DIVE ACCIE	DENT /	ASSE	SSME	TV			
DATE:	TIME		L0	CATIC)N:		
Diver's name:				Age:		Sex:	
Date and time o	f last div	e:					
Symptom onset							
Any symptoms					i tranning.		
Any symptoms					dluluu		
	·		for the I		No.		
	Dive 1	S:I	Dive 2	S:I	Dive 3	S:I	Dive 4
Depth							
Time							
Deco required						2000	
Deco omitted							
Fast ascent							
Cold or strenuous							
Name of Table of Pre dive health of the Any significant processing Symptoms: (circle)	was diver, ti	ical his	drated, ill or i	njured, ta	gies?	ion etc)	
02 given Y/N? First aid/fluids g					. What m	ethod?)
Divers condition ☐ Improved ☐				ged \Box	Relapse	d	0.

CRITICAL/S	SERIO	US DIVE	ACCIDE	NT ASSES	SSMENT
DATE:	TIMI	E:	LOCATIO	N:	
Diver's name:		**************	Age:	Sex	C
Date and time of	of last di	ve:			
Symptom onse					
		cle answers			
CPR required ?	Y/N	For how lo	ong?	Succes	sful? Y/N
DEFIB? Y/N				Succes	
	Diver		Diver Unco		
If diver regaine				1973-75-75-75-75-15-15-15-15-15-15-15-15-15-15-15-15-15	nioun?
•					
Level of consci					
For I	Pulse rate	Pulse strength	Breathing Rate	Breathing strength	Skin colour
initial					
+15min					
+30min					
+45min					
+1 Hr			91770		
+1hr15min				5.53	19.7
+1hr30min					
Can diver move (if suspected AGE do Any weakness					7
If yes explain					
Does Diver hav	e difficu	lties in urina	ting? Y/N		
Any strange ski	in sensa	tions or lack	of feeling/n	umbness? Y/	N
If yes explain					
Any balance pr	oblems (or dizziness'	Y/N		
If yes explain					
Any vision/hear	ring/spec	ech/memory	problems?	Y/N	
If yes explain					

If no treatment or oxygen given please still tick one of the above

Any other problems headache/nausea/personality changes etc? Y/N

APPENDIX iii

Emergency action card

This chart is left blank and can be used as a template. It can be copied and made waterproof. The most likely emergency situations that divemasters and instructors may come across in their area of operation can be assessed and a simple contingency plan can be written on the card, one card for each scenario. The first card is an example of how to use it. All areas are different and not all boats and staff are the same. A thorough risk assessment is advised of the dive sites, boats and weather conditions likely to be encountered as well as liasing with local emergency services for advice prior to completing your action cards.

EMERGENCY ACTION CARD: 1 (MAN OVERBOARD)

P.1 Immediate/ Within 5 minutes	SHOUT "MAN OVERBOARD" THROW FLOATATION PRESS MOB FUNCTION ON GPS STOP BOAT PERSON TO POINT AT VICTIM CONTINUOUSLY	CONTACT 1/2 contact 1/2 if victim not round in s minutes
P.2 Within 5-10 minutes	IF AT NIGHT ILLUMINATE WITH TORCH/FLARES IF VICTIM IN SIGHT, TURN 180' IF NOT "WILLIAMSON TURN" DECIDE APPROACH SIDE PORT OR STARBOARD HAVE SOMEONE READ! WITH HEAVING LINE TO THROW TO VICTIM CUT ENGINE PRIOR TO RECOVER!, REMOVE VICTIM AS QUICKLY AS POSSIBLE	CONTACT CONTACT 1/2 IF RECOVERY NOT POSSIBLE OR DELAYED CONTACT 3 ADVISE ON SITUATION HAVE THEM INFORM COASTGUARD
P.3 Within 10-15 minutes	MOVE VICTIM TO WARM AREA ASSES AND TREAT FOR HYPOTHERMIA AND DROWNING TREAT ANY OTHER SERIOUS INJURIES	CONTACT CONTACT 1/2 IF VICTIM REQUIRES MEDICAL TREATMENT OR EVACUATION
P.4 15 minutes onwards	, IF NECESSARY ARRANGE EVACUATION (IF NOT ALREADY DONE SO) , TREAT ANY MINOR INJURIES , PREPARE FOR EVACUATION (IF NECESSARY) , RECORD EVENTS , STAND DOWN AND ASSESS THE GENERAL SITUATION	CONTACT CONTACT S UPDATE THEM ON SITUATION ADVISE THEM ON YOUR PLANS. IF NECESSARY STAND DOWN COASTGUARD

	4.0,04	Method A	Method B
	1. COASTGUARD	VHF CH 16	TEL: 020 123456
EMERGENCY CONTACTS	2. POLICE EMERGENCY	TEL.: 111	TEL: 020 654321
	3. COMPANY OFFICE	YHF CH 2	TEL: 020 456789

EMERGENCY ACTION CARD: 1

P.1	2 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1	CONTACT
Immediate/ Within 5 minutes	PRIORITY 1	17 5 35.5
P.2	2	CONTACT
Within 5-10 minutes	PRIORITY 2	II y y incasion in
		1100
P.3	1 2 2 2	CONTACT
Within 10-15 minutes	PRIORITY 3	
	the contract of	
P.4		CONTACT
15 minutes onwards	PRIORITY	
	2 9	

Method A

Method B

EMERGENCY CONTACTS

2.

1.

3.

APPENDIX iv

Emergency numbers

Here is a list of the more common dive accident phone numbers world wide.

Divers Alert Network

(World wide including USA and Canada, Europe, Japan, Philippines, Malaysia, Southern Africa

DAN America: +1 919 684 8111

DAN Latin America: +1 919 684 9111

DAN Europe: +39 06 4211 8685

DAN Japan: +81 3 3812 4999

DAN Southern Africa: (from within South Africa 0800 020 111) + 27 11

254 112

DAN South East Asia Pacific from within Philippines: 02 815 9911

DAN South East Asia Pacific from within Malaysia: 05 930 4114

For 24 hour advice throughout the rest of the South East Asia region and Australia/New Zealand call the DAN supported hotlines below:

Divers Emergency Service (DES)

Australia, New Zealand and South east Asia Pacific

Diver's Emergency Service (DES) (Freephone Australia only 1 800 0888 200)

(Outside Australia +61 88 212 9242)

Diver's Emergency Service (DES) (New Zealand): 0800 4DES 111

SSS Recompression Chamber network:

(Thailand, Mexico, Ecuador, Papua New Guinea, Belize, Bahamas)

Thailand

Koh Samui: (077) 427 427 Phuket (076) 342 518

Bahamas

Nassau: (242) 362 5765

Belize

Ambergris Caye: (011) 501 226 2851

Papua New Guinea

Port Moresby: (675) 325 9599

Mexico:

Ecuador:

Cancun (998) 8871 688 Playa Del Carmen (984) 8731 365 Cozumel 1-800-700-2666 (Mexico only), Baha (624) 1433 666 Merida Yucatan (999) 948 3441

rioriaa racatair (>>

Galapagos (593) 5526 911

Hyperbaric health hyperbaric chambers

Philippines, Micronesia, Fiji, Papua New Guinea, Solomon Islands, Malaysia, Vanuatu

Philippines

Batangas City: 6343 723 8388

Micronesia

Chuuk (Truk): 691 330 2318

Palau: 680 488 2552

Fiji

Suva: 679 313 444

Papua New Guinea

Port Moresby: 325 6633

Solomon Islands

Honiara: 677 950 42

Malaysia

Ipoh: 5242 8533

Vanuatu

Port Vila: 678 25566

Independent emergency numbers

Cayman Islands, Egypt, Indonesia, Maldives, Singapore, South

Cayman Islands

Grand Cayman: 949 29 89

Maldives

Bandos medical centre: 960 44 00 88

Egypt

Sharm El Sheikh: (12) 212 42 92 El Gouna (Hurghada): (12) 218 75 50

Alexandria: (203) 546 25 12

Indonesia

Bali: 361 227 911

Maldives

Bandos medical centre: 960 44 00 88

Singapore

Singapore: 750 55 44

South Africa

Pretoria: 012 334 2567 Capetown: 021 671 8655 Durban: 031 268 5000

United Kingdom

England & Wales

Institute of Naval Medicine (INM) duty doctor: 07831 151523

Scotland

Aberdeen Royal Infirmary: 01224 6818181

South West England

Diving Diseases Research Centre (DDRC): 01752 209999

London & the South East

London Hyperbaric Medicine (LHM): 07736 898066

APPENDIX v

Further reading and training

Below is a list of relevant courses that may be of use to sports diving professionals.

Oxygen provider/Oxygen therapy courses

Basic courses:

BSAC

Oxygen administration course

A one day course containing both practical and theory lessons in the use of Oxygen in diving accidents. Open to BSAC club members. Entry to this course is usually for Ocean (club) divers and upwards but may be open to others at the discretion of the BSAC rescue skills examiner.

DAN America/DAN Europe/DAN SEAP/DAN Southern Africa

Oxygen first aid for scuba diving injuries

A simple straight forward course lasting around half a day lectures include basic physiology, signs and symptoms of decompression illness, O2 safety, O2 equipment and use, resuscitation review using oxygen. DAN SEAP also provide CPR training.

Advanced oxygen courses:

DAN America/DAN Europe/DAN SEAP/DAN Southern Africa

Advanced oxygen first aid for scuba diving injuries

Students must already have a certificate in basic oxygen provision in diving accidents before enrolling on this course. The course covers the use of more advanced equipment including bag valve masks and positive pressure resuscitation valves.

AED courses:

DAN America/DAN Europe/DAN SEAP/DAN Southern Africa

AED's for scuba diving/AED for aquatic emergencies

Two separate, simple courses lasting up to a day covering the uses of

automated external defibrillators, both practical and theory sessions are included as well as CPR reviews. Students should have knowledge of basic CPR prior to attending.

Hazardous Marine life injuries courses:

DAN America/DAN Europe/DAN SEAP/DAN Southern Africa

All DAN regional centres offer a half day course in the recognition and treatment of marine life injuries including; Jelly fish stings, venomous bites, fish spine injuries and large predator injuries.

General first aid courses:

DAN SEAP

The only DAN region, at the moment, to offer courses in general first aid. Courses range from basic CPR and emergency first aid to senior first aid and workplace first aid.

Course duration ranges from four to 24 hours. All courses are accredited with the nationally recognised training scheme in Australia and various workplace authorities in Australia.

PADI Emergency First Response (EFR)

PADI EFR courses are broken down into basic modules consisting of Primary care courses (CPR), Secondary care courses (First aid) and now include introduction to AED modules as well as a care for children module specialising in the initial management of sick and injured children ranging from ages one to eight. EFR also provide a first aid at work program recognised by the HSE in UK. The EFR program is recognised by OSHA in the United States and meets the requirements as laid down by the U.S Coast guard and the boy scouts of America.

Diver Medical Technician (DMT) courses:

IMCA

The International Maritime Contractors Association (IMCA) has set out guidelines for DMT courses that IMCA accredited training establishments are required to follow. The DMT course is an advanced course mainly aimed at the commercial diving sector. The course covers basic first aid as well as oxygen first aid for diving accidents. The course also covers more

advanced subjects and certain paramedic skills. In general the course is two weeks long and is open to recreational divers as well as commercial divers. Recreational divers will not receive the IMCA accredited certificate but will receive certification as a DMT.

NBDHMT

The National Board of Diving and Hyperbaric Medical Technicians (NBDHMT) in the United States offer national certification for those who have completed accredited DMT courses in the U.S. These courses are broken down into DMT (basic) and DMT (advanced). The basic course is approximately 40 hours and includes rapid field neurological testing and chamber operations. The advanced module includes training in advanced paramedic interventions.

Those wishing to participate on these courses should have a basic first aid certificate it is also recommended that they have some form of rescue diver certification or equivalent. Those wanting NBDHMT certification must possess an EMT basic or Paramedic Licence.

ADAS

There are six training establishments in Australia and New Zealand that provide ADAS (Australian Diver Accreditation Scheme) approved DMT courses. These courses are approximately three weeks in duration and are aimed at the commercial diving sector in Australia and New Zealand but are open to recreational divers. The first module is a workplace first aid certificate the other modules include advanced airway management I.V infusion and other paramedic skills as well as advanced dive accident management and recompression.

Recompression chamber awareness courses:

PADI

PADI provide a simple course run at participating hyperbaric centres that give a basic introduction to recompression and provide a 'dry dive' in a chamber. The course is normally no longer than a day.

Contacts:

All DAN courses

DAN America www.diversalertnetwork.org

DAN Europe www.daneurope.org DAN Southern Africa www.dansa.org DAN SEAP www.danseap.org

PADI courses

www.padi.com

BSAC courses

www.bsac.com

EFR courses

www.emergencyfirstresponse.com

DMT courses

UK

Diving diseases research centre (DDRC) Plymouth www.ddrc.org

The underwater centre Fort William www.theunderwatercentre.co.uk

USA

For a full list of providers contact NBDHMT www.NBDHMT.org

Australia/New Zealand

Royal Adelaide hospital (Adelaide) www.rah.sa.gov.au/hyperbaric/courses.

Recommended Reading

For those who wish to read more and have a greater understanding of diving medicine:-

Cole, Bob. The Decompression Matrix. Dive Info. 2003

Diving Diseases Research Centre. *The DDRC Underwater Accident Manual*. AquaPress. 2005

Edmonds, Carl. Diving & Subaquatic Medicine. Hodder Arnold. 2005

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Lippmann, John. Oxygen First Aid for Divers. J. L. Publications. 1994

Haux, Gerhard F. K. History of Hyperbaric Chambers. Best. 2000

Merritt, David. Mending the Bends. Best. 2002

Supervisor of Diving. US Navy Diving Manual. AquaPress. 2001

Wienke, Bruce R. Basic Decompression Theory & Application. Best. 2003

Wienke, Bruce R. Reduced Gradient Bubble Model in Depth. Best. 2003

APPENDIX vi

GAS LAWS

Boyle's Law

"The pressure of a fixed mass of gas is inversely proportional to its volume if the temperature is constant"

If you put external pressure on a gas in a flexible container e.g. like taking a balloon underwater, it gets smaller, if you release the pressure it gets bigger. The change in size is proportional to the change in pressure applied to it as long as the temperature of the gas remains the same. (Barotrauma)

Dalton's Law

"The total pressure exerted by a mixture of gases is equal to the sum of the partial pressures exerted by each of the gases"

Air at 1 ATM contains about 79% nitrogen (N2) and 21 % Oxygen (O₂) so the total pressure of the mixture is 1 ATM. Therefore, 0.21 of an ATM is O₂ and 0.79 of an ATM is N2. Add the two together and you get 1 ATM. At 2 ATM everything would be doubled, so N2 would be 0.79 x 2 = 1.58 and O₂ would be 0.21 x 2 = 0.42. Add those two numbers together and you get 2 ATM. (CNS Oxygen toxicity)

Henry's Law

"The amount of any given gas that will dissolve into a liquid at any given temperature is a function of the partial pressure of the gas that is in contact with the liquid and the solubility of the gas in the particular liquid"

All liquids can hold gas in solution without releasing it and making bubbles. The amount of gas the liquid holds in solution without making bubbles is dependent on how easily the particular gas can dissolve into a liquid and the partial pressure of the gas that is touching the liquid. The higher the partial pressure the more gas can be dissolved into the liquid

If you increase the general pressure on a gas mixture you will increase the partial pressure. If you release the pressure, the partial pressure will drop

and the gas will start to come out of the solution and may form bubbles. **Decompression illness (DCI/DCS)**

Charles' Law

"At a constant pressure, the volume of a gas varies directly with the absolute temperature"

As long as a gas can expand in a flexible container e.g. a balloon, if the temperature of the gas is heated the size of the balloon (volume) will increase. If the gas is cooled the size will decrease.

Gay-Lussac's Law

"The pressure of a confined gas at a fixed volume is proportional to the temperature"

If the volume of a gas does not change e.g. like in a scuba tank, the pressure will increase if the temperature rises and decrease if the temperature falls.

Contributors:

John Lippmann



John Lippmann has been involved in researching, teaching, writing and consulting on diving and accident management for the past 30 years. He has specialised in certain areas including resuscitation, first aid, dive rescue and various aspects of decompression. His books, many of which have been published worldwide, include The DAN Emergency Handbook, Deeper Into Diving, The Essentials of Deeper Diving, Scuba Safety in Australia, Oxygen First Aid, First Aid & Emergency Care, Automated External Defibrillators and First Aid for Marine Animal Injuries.

John is currently the Executive Director and Director of Training of the Divers Alert Network S.E. Asia-Pacific (DAN SEAP which he founded over 10 years ago in an effort to improve the safety of scuba diving within the Asia-Pacific.

Klaus Torp



Klaus Torp is an assistant professor of anesthesiology at Mayo Clinic College of Medicine and at the Uniformed Services University of the Health Sciences. He is a diplomate of the American Board of Anesthesiology and board certified in Undersea and Hyperbaric Medicine by the American Board of Preventive Medicine since 2002.

He completed fellowship training in critical care medicine and received fellowship training at the Duke University's F.G. Hall Hyperbaric Medicine and Environmental Physiology Center in 2000/2001.

He graduated from the NOAA/UHMS/USRF Physicians' Training In Diving Medicine Course in 2000, after which he went into diving recompression chamber practice in remote locations in South East Asia and Central America. Since 2002, he serves as a faculty member at the NOAA/UHMS/USRF Physicians' Training In Diving Medicine Course lecturing on Diagnosis and Treatment of Decompression Illness. He is a member of the adjunctive therapy committee of the Undersea and Hyperbaric Medical Society. He has lectured on various hyperbaric and diving medicine topics in Europe, Asia and America. Besides his clinical expertise, he is a researcher in diving medicine in collaboration with the Naval Medical Research Center in Bethesda, Maryland.

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London Hyperbaric Medicine; Page 9, 28, 31, 45, 46, 47 top picture, 51, 52 Hood tent only, 53.

SSS Thailand, Page 10, 37, 48.

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