

PHYSIOLOGY

Circulation:

Blood circulates around your body to fuel the body with oxygen, the body uses the oxygen and changes it to carbon dioxide (CO₂) the blood then carries this back to the lungs where CO₂ is released and more oxygen taken on. This whole process is known as “*oxidative metabolism*”.

The blood is made mainly of plasma, a clear fluid, red blood cells which give it the color, white blood cells which fight off infection and platelets which help the blood to clot in case of injury.

The *red blood cells* carry the *oxygen* which combines with *haemoglobin* in the cells. Red blood cells are also known as “*erythrocytes*”. The waste CO₂ is carried away in the *plasma*, dissolved as *bicarbonate*.

All this is pumped around by a four chambered pump, the heart. Blood vessels carrying blood away from the heart are called *arteries*. (This is why they spurt out blood rhythmically when punctured as it is pressurized.) Blood vessels carrying blood back to the heart are called *veins*. In between the arteries and veins are small blood vessels where the tissues take on oxygen and release CO₂ - these are called *capillaries*.

Let's look at the route in sequence, starting at the lungs where the blood releases CO₂ and takes on oxygen at the *pulmonary capillaries*.

The newly oxygenated blood is pumped back to the heart by the pulmonary vein and is in turn pumped out through the body via the arteries. These get smaller and smaller until they become capillaries where the oxygen is taken on by the body tissues and the waste CO₂ released. The CO₂ is converted to bicarbonate and carried back in the plasma part of the blood via the veins back to the heart. It is then pumped via the pulmonary artery back to the lungs for re-oxygenation at the pulmonary capillaries, which is where we started.

Respiration:

To get the oxygen to the *pulmonary capillaries* we need to breathe, this is called respiration. To trigger this in the body the build-up of CO₂ causes the *diaphragm* to flex downward making us inhale again. (If you don't believe me try holding your breathe for as long as you can and see what happens to your diaphragm!)

The air passes through the nose which filters the air and the sinuses moisturize the air before it reaches the lungs, when breathing through your mouth, as in diving, this step is bypassed; this is one of the reasons breathing air has to be filtered and why you get dehydrated when diving – always drink plenty of water.

The air then enters the *trachea* via the throat and into the *bronchi*, which are large tubes leading to the lung. These split into smaller and smaller *bronchioles* until

reaching the *alveoli*; small sacs surrounded by the *pulmonary capillaries* where the gas exchange takes place through a thin membrane.

For maximum gas exchange you need to breathe *slowly and deeply*. When you exhale some of the stale air remains in your lungs and passes to the lungs; this is re-inhaled on the next breath.

These are known as *dead air spaces* as no gas exchange can take place here; snorkels and regulators increase dead air space. Shallow, rapid breathing also creates turbulence in the air passages so not as much reaches the alveoli. This causes a build-up of carbon dioxide, known as hypercapnia, which makes the diver feel out of breath. This can also be caused by the diver overexerting himself or a badly maintained regulator which is difficult to breathe from.

Apnea:

This is simply breath-hold diving. When you hold your breath and dive you use the oxygen stored in the tissues, when the waste CO₂ builds up the urge to breathe becomes intolerable and the diver is forced to surface to breathe again and replenish the cells with oxygen.

If the diver *hyperventilates* before the breath hold dive this reduces the residual CO₂ in the lungs and extends the time before the diver gets the urge to breathe. A maximum of 3-4 deep breathes is okay, but any more can lead to *shallow water black-out*.

Excessive hyperventilation reduces the CO₂ levels in the lungs too much (hypocapnia), so that when the diver is underwater the oxygen level drops below critical levels before the CO₂ creates the urge to breath. While the diver is at depth, the partial pressure of the oxygen is greater (remember the physics), when the diver ascends the *partial pressure of the oxygen* drops below the critical level to sustain consciousness; the diver blacks out before reaching the surface due to *hypoxia* (lack of oxygen). This phenomenon is known as *shallow water blackout* and only happens when the diver ascends.

Also in cold water when performing a breath-hold dive, bradycardia (slowing of the heart), reduces circulation speed. This is much more evident in marine mammals than humans, although some free diving experts claim to be able to control this. Remember Jacques Mayol in the Big Blue...

Carotid Sinus Reflex:

The carotid sinus receptors monitor the pressure of arterial blood reaching the brain through the carotid arteries.

Low blood pressure triggers a higher heart rate (*tachycardia*).
High blood pressure triggers a lower heart rate (*bradycardia*).

Receptors will interpret the pressure from an excessively *tight hood*, wet suit neck seal or dry suit neck seal, as high blood pressure.

This *reduces blood flow to the brain* as the heart slows causing the diver to feel light headed if this continues it can lead to unconsciousness.

Carbon Monoxide poisoning:

This is caused by contaminated air from using the wrong lubricants or improper maintenance of the compressor system. Smoking is another source of carbon monoxide (CO). It takes 8-12 hours to flush the CO from your body after smoking just one cigarette. Smoking also destroys the *surfactant* in your lungs. This is the lubricant that prevents the walls of the the lungs from sticking together. If this happens you could have a lung over expansion injury without ever holding your breath.

Carbon monoxide bonds with the haemoglobin 200 times more readily than oxygen does, and doesn't release as easily. When air contaminated with CO is breathed at depth the haemoglobin carries less and less oxygen around the body as the CO bonds with it. However, at depth, the blood still carries enough oxygen dissolved in the plasma by the high partial pressure to meet tissue demands. As the diver ascends and surfaces the plasma cannot carry enough oxygen and the diver blacks out due to *hypoxia*. Signs and symptoms include headache, confusion, narrow vision, *bright red lips and fingernail beds*. Give oxygen and get to medical care.

Oxygen Toxicity:

If you dive on air to greater than 57m or much shallower when using enriched air nitrox mixes. You can have problems with the high partial pressure of oxygen.

There are two types:

- 1) Central nervous system (CNS) toxicity – caused by exposure to oxygen partial pressures greater than 1.4 bar. (57m on air, 33m on EANx32, 29m on EANx36 and just 4m on pure oxygen). Signs and symptoms include: visual disturbances, ear ringing, nausea, twitching, irritability and dizziness (VENTID). But the *most serious symptom/sign is a convulsion usually without warning* which can cause the diver to lose his mouthpiece and drown.
- 2) Pulmonary toxicity – caused by a long exposure to high partial pressures of oxygen. Usually only occurs after a series of multiple dives using enriched air or in technical diving using high concentrations of oxygen for long decompression schedules. Signs and symptoms include burning in the chest and an irritated cough. It usually resolves itself if the diver ceases diving for a few days. It is avoided by following NOAA and DSAT oxygen exposure tables.

Nitrogen Narcosis:

Almost any gas can cause a narcotic effect under pressure. It appears to be related to nerve impulse blockage due to gas dissolved in the nerves. Oxygen has about the same potential so don't expect to be less susceptible when using Nitrox mixes. Expect narcosis to be noticeable at *around 30m*, it varies from one diver to the next and is not predictable. Helium is not narcotic under high pressures which is why it is used in deep technical diving.

Ascending a few metres usually relieves the symptoms which are not directly hazardous – the danger comes from *impaired judgment and co-ordination* which may lead to bad decisions.

Other symptoms can include euphoria (feeling happy), anxiousness, panic, dizziness, tunnel vision.

Decompression sickness:

When under pressure gases will dissolve into liquids, our body is mostly water so when we breathe air at depth it goes into solution in body tissues.

Oxygen is consumed metabolically, but nitrogen is physiologically inert and dissolves into the blood and tissues.

This dissolved gas still exerts pressure which is known as *tissue pressure*. Different tissues absorb and release nitrogen at different rates – slow and fast compartments.

If the diver stays at depth, eventually his body will *saturate* meaning the gas pressures have reached an equilibrium and he can't absorb any more nitrogen.

Calculating different tissue absorption and release is the basis of decompression models for tables and computers.

Recreational dives are too short to reach saturation, but upon ascent from any dive the nitrogen pressure in the tissues is greater than the surrounding pressure, this is called supersaturation.

If the difference between the tissue pressure and the surrounding pressure (*the pressure gradient*) is kept within limits the nitrogen dissolves harmlessly out of the body through respiration.

After some dives *Doppler ultrasound flowmeters* detect silent bubbles in divers – *these are harmless* in themselves but could join up to form larger bubbles if there's too many of them.

If the diver ascends too quickly or misses required decompression stops these small bubbles accumulate and form larger bubbles causing decompression sickness (the bends).

Signs and symptoms of DCS usually take some time after the dive to present, and differ depending on where the bubbles form.

Type 1 DCS: pain only usually in the joints and limbs and skin bends (rashes on the shoulders and upper chest).

Type 2 DCS: life threatening involving the nervous system, numbness, tingling, paralysis, weakness, fatigue, unconsciousness and death.

There are several physiological factors that may predispose a diver to DCS, In other words make it more likely he will get bent even when sticking to tables or a computer especially if near the limits.

These are:

- Fat tissue: Fat releases nitrogen slowly so a fat diver may have more nitrogen after a dive.
- Age: As we get older our circulatory systems become less efficient slowing down the gas exchange.
- Dehydration: This reduces the blood in circulation, slowing nitrogen elimination. Always drink plenty of water while diving.
- Injuries/illness: These could alter or restrict circulation to localized areas where nitrogen isn't eliminated as quickly.
- Alcohol before or after diving: This alters circulation patterns, dilates capillaries and promotes dehydration, all of which alter nitrogen elimination and bubble formation.
- Carbon Dioxide excess: Raised CO₂ levels will alter circulation and gas exchange.
- Cold Water: When the diver gets cold circulation to the extremities is reduced, hindering nitrogen release.
- Heavy Exercise: During the dive this can raise CO₂ levels and accelerate circulation so more nitrogen is absorbed. After a dive it accelerates circulation altering nitrogen elimination.
- Altitude/Flying: Tables and computers are based on surfacing at sea level, if we go to altitude after a dive this increase the pressure gradient and bubbles may form – returning to sea level will not alleviate the bubbles once formed!

Treatment for DCS

First aid Treat all cases as serious, give the patient oxygen, keep the patient lying level on the left side with the head supported, provide primary care and arrange transport to the nearest medical facility.

DCI stands for decompression illness and covers DCS and all lung-overexpansion injuries.

DCS refers only to the condition caused by dissolved nitrogen in the system.

Heat and Cold:

Overheating can cause problems. Firstly, *heat exhaustion*, where the body works at full capacity to cool. Signs and symptoms include: *weak rapid breathing, weak rapid pulse, cool clammy skin, profuse sweating*, dehydration and nausea. Treatment is to get the diver to a cool area, remove exposure suit, and give nonalcoholic fluids, rest until cool.

Secondly, this can lead to *heat stroke*. If heat exhaustion is not treated promptly it can lead to heat stroke which is *life threatening*. The body's cooling system has now completely failed. Signs and symptoms include: *strong rapid pulse, no perspiration/sweating, skin flushed, hot to touch*. Treatment: Cool by whatever means available and get the diver to emergency medical care.

The body responds to heat loss by *vasoconstriction* (reduced blood flow to the extremities) causing finger and toe numbness, then *shivering* as the body tries to generate heat by muscle movement. This signals a losing battle against the cold and is termed *mild hypothermia*. Get the diver to a warm place as soon as possible. If heat loss continues the condition gets worse, it becomes *advanced hypothermia*, which can be life-threatening. The shivering stops. Vasoconstriction stops. The diver may feel warm as blood rushes to the skin. The core temperature drops, mental processes slow, the diver becomes drowsy, then unconsciousness. Coma and death can follow. Advanced hypothermia requires *emergency medical care* as soon as possible.

The Ear:

The ear is divided into the outer, middle and inner ear.

- The *outer ear* consists of the external ear and ear canal, it is open to air/water pressure and channels sound to the eardrum.
- The *middle ear* is separated from the outer ear by the *ear drum* and is sealed against air/water. The ear drum vibrates and passes sound to the *ossicles*, small bones that conduct sound to the inner ear. The middle ear is filled with air and is affected the most by pressure.
- The *inner ear* consists of the *vestibular canals* that control *balance* and the *cochlea* which turns vibrations into nerve impulses sent by the auditory nerve to the brain.
- The ossicles connect to the cochlea at the *oval window* which flexes in and out with the vibrations. The *round window* on the cochlea flexes out when the oval window flexes in, like a water filled balloon.

The middle ear is connected by the *Eustachian tube* to the throat to maintain equilibrium with outside pressure.

As the diver descends pressure pushes in on the eardrum causing discomfort, by equalising the diver forces air up the Eustachian tube to equalise pressure in the

middle ear alleviating the discomfort. Expanding air normally exits from the middle ear through the Eustachian tube easily on ascent.

If the diver does not equalise sufficiently hydrostatic pressure forces blood and fluid into the middle ear until equilibrium is restored. The ears feel full and hearing is reduced and should be checked by a doctor. This is called middle ear squeeze.

If the diver does not equalise and descends faster than fluids can fill the middle ear the eardrum tears due to the pressure. The diver feels a sharp pain, and then relief as the pressure is relieved. When cold water enters the middle ear the diver will experience dizziness or vertigo until the water warms to body temperature. This is because it causes convection in the fluids of the vestibular canals effecting balance.

If the diver delays equalisation, then tries to equalise *forcefully using the Valsalva manoeuvre* (blowing against pinched nostrils from the diaphragm and lungs) pressure on the ear drum presses in on the ossicles which press in on the oval window on the cochlea; the round window flexes out in response. The Valsalva manoeuvre raises pressure in the chest, which causes an increase in pressure in the cochlea (connected by fluid as part of the nervous system). This, plus the transmitted pressure bursts the round window outwards. This injury is known as round window rupture, and is serious and requires medical treatment; the diver may never be able to dive again. To avoid this always use the *Frenzel manoeuvre* (blowing against pinched nostrils but just using the throat muscles to push the air up the Eustachian tube).

If the air cannot escape from the middle ear through the Eustachian tube on ascent a phenomenon known as reverse squeeze occurs. This is usually caused by diving with a cold or using a decongestant that has worn off. Stop or slow ascent and wait for trapped air to work its way out. Swallowing may help or inhaling against pinched nostrils. If ascent continues the eardrum will rupture outward.

Earplugs or a tight wet suit hood create an air space between the plug/hood and the eardrum. When the diver descends the eardrum flexes out towards the plug/hood and can rupture if descent continues. This feels like middle ear squeeze. Avoid by never wearing earplugs and pulling away hood momentarily when equalising on descent.

Let's have some definitions before we continue...

Barotrauma means pressure injury. Baro = pressure. Trauma = injury. It can happen on descent or ascent if an air space is not equalized. An unequalised air space is also called a squeeze.

Other air spaces:

Sinuses are the spaces in the head connected to the nose that filter and moisturise air before it reaches the lungs. They normally equalise with the ears with no problems. Sinus squeeze is usually caused by diving with a cold or congestion. The unequalised sinuses fill with blood and fluid to equalise during the dive – may feel like a sharp pain above the eyes. Upon ascent expanding air pushes blood and fluid

into the nasal cavity and the diver surfaces with blood in his mask. This is usually not serious and heals on its own.

Mask space: This is usually equalised by the diver exhaling into the mask during descent. (That is why you can't dive using goggles). If this is not done the tissues swell, forced into the unequalised mask space, capillaries in the skin and eyes rupture. Looks terrible, but usually clears by itself.

Dry suit space: Squeeze can be caused by not adding enough air to the suit or descending too quickly. Can constrict breathing and caused welts and pinches where the dry suit squeezes.

And finally the biggest air space:

The lungs:

Lung squeeze is caused by a breath-hold descent that reduces lung volume below the residual volume. (This would happen if you went down to over 30m on a breath-hold dive). It can occur shallower if you descend with partially full or empty lungs. It causes fluid to accumulate in the lungs and can be life threatening.

Lung over expansion injuries:

This is what happens if you hold your breath on ascent when using scuba. They can also be caused by lung congestion if diving with a chest cold, or by a local blockage due to loss of surfactant due to smoking. There are four types of injury that can occur:

Air embolism: This is also called arterial gas embolism (AGE). The alveoli and pulmonary capillaries rupture allowing air to enter the bloodstream and flow into the arteries. This is serious and immediately life threatening, the bubbles can lodge anywhere, but usually flow through the *carotid arteries straight to the brain* causing stroke like symptoms, dizziness, confusion, shock, paralysis, personality change, unconsciousness and death..

With all lung over expansion injuries always expect air embolism as this is the worst case scenario, it often occurs with the other types of lung injury.

Pneumothorax: The air from the rupture goes between the lung and chest wall, causing the lung to collapse. Also serious the diver will have chest pain and may cough up blood.

Mediastinal emphysema: The air from the rupture accumulates in the centre of the chest over the heart and interferes with circulation; the diver may feel faint and short of breath, still serious.

Subcutaneous emphysema: The air from the rupture accumulates in the soft tissues under the skin, at the base of the neck. The diver's voice may change and the skin may crackle to the touch.

First aid treatment for these injuries is the same as for DCS, hence the common term DCI to encompass both types of injury. Lie the patient down and administer oxygen, apply primary care and get to a medical facility ASAP.

Note that symptoms of lung over expansion injury usually occur immediately after the dive whereas symptoms of decompression sickness are delayed and can appear 15 minutes after surfacing or even up to 24 hrs before the diver notices symptoms. Micro bubble maximums occur around 35 -40 minutes after surfacing this is the time when most bends will become apparent. But remember this is not an exact science.

