# Chapter 13



# DECOMPRESSION PHYSIOLOGY and SUSCEPTIBILITY

Decompression Sickness (DCS) is an illness caused by the effects of gas coming out of solution to form bubbles in the body after diving. It is due to the effect of Henry's Law (see Chapter 2) following diving exposures. Understanding decompression theories is difficult if not impossible, so the average diver may well bypass most of this chapter, if he is not technically inclined.

In sport divers the main gas formed in bubbles is nitrogen ($N_2$) because these divers almost always breathe air. However, the same principles apply to other inert gases, such as helium (He), which may be breathed by deep commercial and technical divers.

## GAS UPTAKE

When a diver breaths air from scuba equipment at depth, $N_2$ is breathed at an increased partial pressure. Because gas diffuses from areas of high concentration (high partial pressure) to areas of lower concentration, $N_2$ is taken up from the lungs by the blood and transported around the body and into the tissues. The greater the depth, the greater the partial pressure of $N_2$, and therefore the amount of $N_2$ absorbed. Early in the 20$^{th}$ century, Haldane applied this concept to decompression.

The speed of $N_2$ distributing to the tissues depends on the their blood flow. Tissues with high metabolic needs such as the brain, heart, kidneys and liver receive most of the blood pumped from the heart. They will also receive most of the $N_2$ carried in the blood and will have a **rapid $N_2$ uptake.** Such tissues are termed "**fast tissues**" because of their fast $N_2$ uptake.

Because blood passing through the lungs immediately equilibrates with any change in inspired $N_2$ partial pressure, blood is the fastest tissue of all.

Other tissues such as ligaments, tendons and fat, with a relatively small blood flow, have a relatively slow $N_2$ uptake. These tissues are termed **"slow tissues"**. Between the two are tissues of intermediate blood flow such as muscle. Some organs, such as the spinal cord, have both fast and slow tissue components. The rate of uptake of $N_2$ in a tissue is **exponential** i.e. it varies depending on the amount of gas already taken up by the tissue. As the tissue takes on gas, the uptake slows because the partial pressure gradient decreases.

The filling of a scuba cylinder is an example of an exponential process. When an empty cylinder is connected to a high-pressure source, the cylinder initially fills quickly, but the flow slows as the pressure in the cylinder increases and approaches that of the gas source.

The uptake of gas in any tissue is initially rapid but slows with time. Accordingly, it may take a long time for a tissue to become fully saturated with gas, but fast tissues become saturated sooner than slow tissues.

Since the exponential uptake takes a long time to reach completion, even if it starts rapidly, the concept of **tissue "half times"** is used to compare tissues. The half time is the time taken for a tissue to reach half its saturation level. A fast tissue may have a half time as little as a few minutes, while a slow tissue may have a half time of some hours.

## GAS ELIMINATION

$N_2$ is eliminated in a reverse of the uptake process. As the diver ascends there is a reduction in the partial pressure of $N_2$ in the air he breathes, allowing blood to release $N_2$ into the lungs. The decrease in the blood level of $N_2$ causes $N_2$ to diffuse into the blood from the tissues. Fast tissues naturally unload $N_2$ quicker than slow tissues.

Theoretically, tissues should lose $N_2$ exponentially, and most decompression tables are calculated on this assumption. While large amounts of $N_2$ are lost initially, the process slows with time and it may take 24 hours or longer for all the $N_2$ taken up during a dive to be released. Diving again during the time of $N_2$ elimination will mean that the diver will start his second dive with a **$N_2$ retention** in some tissues. Adjustments are provided in the decompression schedule to allow for this and are incorporated as the **repetitive dive tables.**

If there is diminished circulation to a tissue during decompression, gas elimination will be reduced and thus bubble formation will be more likely.

In practice, even during routine conservative dives, bubbles of $N_2$ frequently form in the blood and tissues, interfering with $N_2$ elimination. It has been estimated that as much as 5% of $N_2$ taken up by the body after some dives is transformed into bubbles on decompression. These are often termed "**silent bubbles**" since they usually do not produce any symptoms. They do however have a profound and unpredictable influence on the decompression requirements for repetitive diving, because it takes much longer to eliminate gas bubbles in tissues than it does gas in solution.

# SATURATION

When tissues are subjected to an increased partial pressure of inert gas during a dive, they take up dissolved gas in accordance with Henry's Law. However, there is a limit to the amount of gas which can be dissolved by a tissue exposed to any given partial pressure of gas (i.e. depth of dive). When this limit is reached the tissue is said to be **saturated**.

Our bodies are normally saturated with $N_2$ at atmospheric pressure and contain about one litre of dissolved $N_2$. If a diver were to descend to 20 metres (3 ATA) and remain there for a day or more, his body would take up the maximum amount of $N_2$ possible at that pressure and would then be saturated at that depth. His body would now have about 3 litres of $N_2$ dissolved in it.

Once the body is saturated with inert gas at a given depth, it will not take up more of that gas, no matter how long the diver spends at that depth. Consequently, once the diver is saturated the decompression requirement does not increase with time. This economy of time is exploited in **saturation diving**, when the diver is kept at depth for very long periods of time (days, weeks, months) but then needs only the same lengthy decompression.

# BUBBLE FORMATION

The process of bubble formation can be demonstrated easily by opening a bottle of beer (or champagne, depending on taste and income). In a carbonated beverage $CO_2$ is dissolved in the liquid at a high pressure, which is then maintained by the lid. When the lid is opened, the pressure over the liquid becomes atmospheric and the partial pressure of $CO_2$ in solution exceeds the critical limit for bubble formation, causing bubbles to form. This could be avoided if the pressure was reduced slowly (decompressed).

During ascent, the pressure surrounding the diver (the environmental pressure) is reduced. Eventually, the pressure of $N_2$ dissolved in the tissues may become greater than the environmental pressure. The tissue is then said to be **supersaturated**.

The tissues are able to tolerate a certain degree of gas supersaturation. Nevertheless, Haldane explained that if the pressure of $N_2$ in the tissues exceeds the environmental pressure by a critical amount, then bubble formation is likely. The pressure differential needed to cause this varies between tissues but with most scuba diving it equals or exceeds 2 : 1 (i.e. the partial pressure of inert gas in the tissues should not be more than twice the environmental pressure). This explains why DCS under recreational diving conditions is unlikely after an isolated dive to less than 10 metres — the pressure at 10 metres is 2 ATA, while the pressure at the surface is 1ATA – a 2:1 ratio..

Gas bubbles in the tissue and blood are the cause of DCS. The exact mechanism of bubble formation is complex. It is likely that microscopic gas spaces (**bubble nuclei**) exist in all body fluids and that these form a nucleus for bubble formation during decompression.

Bubbles can form in any tissue in the body including blood. The pressure in each bubble will be the same as the environmental pressure (if it was not, the bubble would expand or contract until it was) and the bubble size is governed by Boyle's Law as the pressure changes.

At the onset of DCS, the pressure of $N_2$ in the tissues is supersaturated (greater than the environmental pressure) so there is an immediate diffusion (pressure) gradient of $N_2$ which then diffuses into any bubbles (or nuclei) present, causing them to expand.

A bubble of DCS contains mainly $N_2$ if the diver has been breathing air, but the other gases present in the tissues, such as carbon dioxide ($CO_{2)}$, oxygen ($O_2$) and water vapour, also diffuse into it.

Once a bubble has formed its behaviour depends on several factors. Any increase in pressure such as diving or recompression will reduce its size while any decrease in pressure such as ascent in the water, over mountains or in aircraft, will expand it. The bubble will continue to grow in any tissue until the $N_2$ excess in that tissue has been eliminated. Once this has occurred (which may take hours or days) the bubble will begin to decrease in size but it may take hours, days or weeks to disappear. In the meantime the bubble can damage the tissues around it.

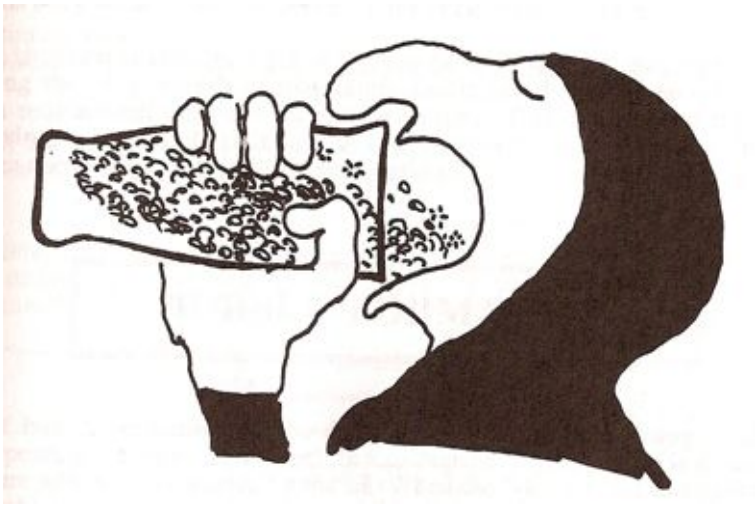


**Fig. 13.1**

There is good evidence that bubbles frequently form in tissues and blood of recreational divers after routine no-decompression dives, even when the tables have been faithfully followed. These bubbles do not usually cause symptoms but certainly cause doubt about the validity of both the decompression tables and dive computer algorithms.

Tissue damage by a bubble results from several factors. Bubbles in the blood obstruct blood vessels in vital organs such as the brain, while bubbles forming in the tissues may press on blood vessels and capillaries, obstructing their blood flow. Bubbles in the blood can also stimulate the clotting process causing the blood to clot in the blood vessels, obstructing blood flow to vital organs, and reducing the ability of the remainder of the blood to clot adequately. Many other biochemical and physiological changes with ill-defined sequelae occur in the tissues and blood vessels during both decompression and DCS. In the brain, spinal cord and other tissues, bubble pressure in or on nerves may interfere with nervous system functioning.

# DIVE PROFILES

The type of dive has a significant bearing on where and when bubble formation takes place. **Short deep** dives (i.e. deeper than 30 metres) **tend to cause bubbles in the fast tissues** (blood, brain and spinal cord) while **long shallow dives tend to produce bubbles in the slow tissues** (like the joints). Long deep dives cause bubbles everywhere.

This distribution occurs because:

- in short dives, only the fast tissues take up enough $N_2$ to form bubbles on ascent and
- after shallow dives, fast tissues eliminate their relatively modest $N_2$ excess before a critical pressure differential develops.

It can thus be seen why it is important to ascend slowly from all deep dives. The slower the ascent rate, the longer the time for fast tissues to eliminate $N_2$ through the lungs, before a critical $N_2$ pressure-differential develops. Slow tissues are more affected by the total exposure (duration + pressure effects) and are more influenced by this and the duration of staging.

Diving folklore contains a myth that a diver using a single 2000 litres (72 cu. ft) tank cannot develop DCS. The air supply available was said to limit the diver to safe dive profiles. This is not true. For example, A single dive to 20 metres, may last 30-40 minutes, within the no-decompression time given by most tables, but it is not entirely safe. Remember, as mentioned previously, that even a single dive in excess of 10 metres can produce DCS.

The myth may become more apparent for deeper dives. For example, a single 2000 litre tank will give around 10 minutes duration for a 50 metre dive. According to most decompression tables, a 10 minute dive to 50 metres will require 10 minutes of decompression — but there may be no air remaining to complete these stops. Even if there was sufficient air, dives to this depth have a significant risk of DCS despite the tables being followed correctly.

# FACTORS INFLUENCING DCS

DCS is unpredictable. In general, anything that increases blood flow to an organ will increase the rate of $N_2$ loading. Anything that interferes with blood flow from an organ will reduce the capacity to off-load $N_2$. These alterations may explain some of the possible predisposing factors that increase the likelihood of DCS.

## ❑ Depth/duration.

Any dive deeper than 10 metres can produce DCS although in general, the deeper the dive, the more gas absorbed, the greater the risk. The longer the dive at any one depth, the more gas absorbed (until saturation), the more the DCS risk.

## ❑ Individuals.

Some people are to be more susceptible to DCS than others. Even an individual may vary in susceptibility at different times, and DCS can develop after a dive profile which has been safely followed on many previous occasions. Others frequently get DCS despite conservative diving.

## ❑ Adaptation.

Repeated dives to similar depths over a period of time reduce the incidence of DCS. This may be due to the elimination of bubble nuclei. A diver returning to these dives after a 2 week break loses the benefits of this adaptation or acclimatisation.

## ❑ Age.

**Older divers** tend to be more predisposed to DCS (an old diver can be defined as anyone older than the senior author of this text). This age factor probably comes into play after the 3rd decade.

## ❑ Obesity.

This appears to be a predisposing factor probably due to increased $N_2$ solubility (4-5 : 1) in fat compared to water (obesity is defined as anyone heavier than the biggest author). This may be relevant for those with a BMI of > 25.

## ❑ Debilitation.

Factors causing the diver to be unwell such as **dehydration, hangover** or **exhaustion** tend to predispose to DCS. Fatigue (pre-dive) is observed in some cases.

## ❑ Injury.

DCS, particularly involving the musculo-skeletal system and joints, is more likely with recent bruises, strains or chronic injuries.

## ❑ DCS.

A previous episode of DCS, especially if it was unexpected from the dive profile, or if it damaged tissue (as in neurological DCS), makes the diver predisposed to similar subsequent episodes.

## ❑ Patent Foramen Ovale.

One reason for some people to have an increased susceptibility to DCS is that they have a small hole in their heart. All of us had a hole in our heart when we were a foetus. In about a third of the population some remnant of this hole remains, it is called a patent foramen ovale, or PFO. These people have an increased susceptibility to DCS, the likely reason is that bubbles that would normally be trapped in the lungs without causing symptoms pass through the hole, by-passing the lung filter, and on to other parts of the body, where they cause noticeable symptoms. However, the risk from a PFO is not great enough for it to be appropriate to test all divers for it, and repair of the hole is probably more dangerous than diving with it.

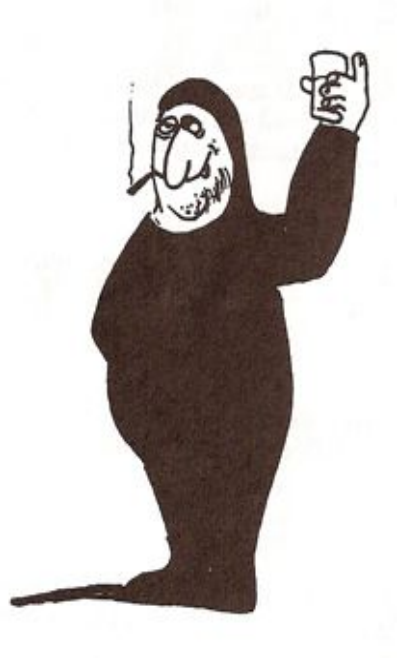
## ❑ Cold.

Diving in cold conditions makes DCS more likely, especially when the diver is inadequately insulated. More precisely, coldness during the dive inhibits inert gas uptake (because of restricted circulation) but allows more $N_2$ to dissolve in body fluids — whilst coldness during decompression inhibits inert gas release. Theoretically, it may be better to be cold during the dive and warm on decompression, unless bubble formation occurs. Warming will then reduce gas solubility and increase bubble growth and DCS.

The association between cold exposure and DCS is complex and contentious. During decompression and post-diving the cold environment may cause peripheral constriction of blood vessels and more bubble formation. Alternately, taking hot showers also tends to cause increased bubble formation and DCS.

## ❑ Alcohol and other drugs

It has been observed that divers who over-indulge in alcohol, or perhaps take other **drugs** or medications, may be especially susceptible to DCS. In the case of alcohol, especially taken the night before, the effects may be due to the associated dehydration or the vascular dilatation (remember the throbbing headache and "hangover"), increasing $N_2$ take-up.



## ❑ Exercise.

This also is complex and contentious. Some even claim that exercise 2-24 hours before diving, and even after diving, may reduce bubble formation. At least in rats.
Strenuous exercise during a dive is likely to increase the $N_2$ uptake by increasing blood flow to muscles, increasing gas uptake and favouring DCS development. Gentle exercise during decompression, by promoting circulation from the tissues probably aids in $N_2$ elimination. The effects may depend on whether bubbles or bubble nuclei have already formed. Strenuous exercise after the diver has returned to the surface makes the development of DCS, particularly in the musculo-skeletal system, more likely by promoting bubble formation. Strenuously activity, such as by shaking a beer can before opening it, aptly illustrates this phenomenon. During the first hour or two after a dive, particularly when there has been a large $N_2$ uptake, it is probably best to rest quietly as this is the period of maximal $N_2$ elimination.

**Fig. 13.2**

## ❑. Physical Fitness.

The less physically fit the diver, the more likelihood of DCS, probably because more energy is used and more blood flow is required for the same outcome – transporting more $N_2$.

## ❑ Gender.

There is some evidence that women have a higher incidence of DCS for certain dive profiles. There are subtle differences in physiology and body composition which could explain this. The decompression tables in current use only evolved after extensive testing on men alone (see Chapter 8).

## ❑ Dive profile.

**Deep dives** (greater than 18 metres), **long dives, decompression dives** and any dives exceeding the **limiting line** (in RN based tables) all have a higher incidence of DCS.

**Square wave profiles** (remaining at the maximum depth for all the dive) are probably more hazardous than an equivalent $N_2$ load produced by **multi-level diving**, if the levels are at diminishing depths ("forward dive profiles"). Reverse dive profiles are even more dangerous.

## ❑ Reverse Dive Profiles

Divers are advised to dive from deep to shallow ("forward dive profiles"). The should dive their deep dive first in repetitive dives, and dive to progressively shallower depths when multi-level diving. If this order is not followed ("reverse dive profiles"), DCS is more likely.

## ❑ Rapid ascents.

These allow insufficient time for $N_2$ elimination from fast tissues, thus encouraging bubble formation.

## ❑ Multiple ascents.

Multiple ascents during a dive imply multiple decompressions and often involves rapid ascents. Bubbles in the blood (fast tissue bubbles) are likely to form during these ascents. The bubbles may not be adequately filtered by the lungs, passing along into the tissues, or may be reduced in size during the second or subsequent descent, allowing them to escape through the pulmonary filter into the tissues. DCS is then more likely.

## ❑ Repetitive dives.

Each repetitive dive begins with a $N_2$ load of some degree from the previous dive. Since bubble formation even after routine dives is common, a repetitive dive will often start with the diver carrying $N_2$ bubbles from the previous dive. $N_2$ elimination is less rapid from bubbles than it is from the same amount of gas in solution. These bubbles will be supplemented by $N_2$ taken up during subsequent dives, and make DCS more likely.

The algorithms used in dive computers are less accurate, and less validated, when used for repetitive diving.

Also with repetitive dives there may be the residual physiological effects of the previous dive, increasing the likelihood of decompression sickness. These physiological effects may include a lower body temperature, dehydration from immersion and recent exercise.

A very short surface interval may avoid some bubble formation in some dives, but if bubbles have developed, the longer the surface interval the safer the repetitive dive.

## ❑ Flying after diving.

The jet age often finds divers flying home after a dive holiday within hours of their last (sometimes literally) dive. International airliners are pressurised to an altitude of about 2000 metres (6500ft.) above sea level. This means a pressure reduction on the diver of about 25% with a corresponding increase in the degree of $N_2$ supersaturation as well as a corresponding increase in the size of any bubbles he may be carrying. The increase in size of critical bubbles may be sufficient to provoke symptoms or aggravate existing symptoms.

The DCIEM recommendation is "whenever possible it is inadvisable to fly above 600 metres in any aircraft within 48 hours of completing any dive. Travelling by vehicle over mountain ranges or hills can expose divers to the same dangers as flying and should be avoided in the same way for 24 hours. If flying after diving is considered essential, flying may be carried out after 24 hours but the increased risk of DCS must be borne in mind."

## ❑ Dive computers.

Using dive computers that are based on largely invalidated **theories** (as opposed to practical diving and decompression table experience) may result in a diver getting much more time underwater while diving — and in the recompression chamber during treatment. Both can be included in his log book if he survives.

They can be made safer by employing the advice given later (see Chapter 14).

## ❑ Multi-Factorial Effect.

Often there is more than one factor increasing the likelihood of DCS. Thus in one large Australian series over half the cases engaged in multiple dives, deep dives (greater than 30metres) and/or had ingested alcohol within 8 hours. Another 20% were precipitated by aviation exposure. Thus many of these divers would have had at least 2 factors increasing their likelihood of DCS.

**Note:** The senior (elder) author believes that the only explanation for most cases of DCS lies in the random application of Chaos Theory, which he also does not understand, or string theory which no-one understands.