The Mechanisms of Decompression Illness

Part 1 of a 3-part article by Dr. Simon Mitchell ***- Part 1***

Few subjects are more central to diving medicine than decompression illness, the most important diving medical disorder. We are all taught about the disorder and its potentially serious consequences during diver training courses but the treatment given to the subject by the diver training organisations is relatively superficial. Recognising its importance, many divers want to know more; those few extra details that might help them improve their diving practice, make things a little safer, or simply satisfy their curiosity. To help with all of these, the following series of three articles in the next issues of Alert Diver SEAP will consider the topic of decompression illness in more detail than you will usually see. In this first article, I will explain the sources of the bubbles that are the central cause of this puzzling disorder. In my next article, we will examine how these bubbles cause damage, and what symptoms result. Finally, in the third article we will discuss the various potential risk factors for decompression illness, and how the risk can be minimised.

A simple definition….

Decompression illness is a disorder caused by bubbles forming in body tissues or in the blood; in other words, in places where they shouldn't be. These bubbles cause damage by a variety of mechanisms that I will return to in the next issue. How do these bubbles form?

There are two potential sources of bubbles…

There are two potential sources of bubbles, and even though these sources are quite distinct (see below), we refer to the adverse effects of bubbles from either source under the "umbrella" term "decompression illness" (DCI). This is because, not surprisingly, it is often difficult to tell which source of bubbles has caused a patient's problem. So what are these two sources of bubbles?

Bubbles can form in tissues and blood from dissolved gas…

The first source is bubble formation from nitrogen that has been dissolved in tissues during the dive. As you have been taught in your most basic training course, during a dive, air is breathed at increased pressure, and therefore more of the nitrogen molecules from the air can dissolve into the blood. Take a few moments to study the very simplified human circulation diagram (Figure 1) and read the explanatory caption. The nitrogen enters the blood in the lung capillary bed (top of diagram), and after passing through the left heart, is distributed to the tissues via the arteries. In the capillary beds of the head and body, nitrogen leaves the blood and diffuses into tissues. The deeper the dive, the faster the nitrogen is taken up from the air we breathe, and the longer the dive, the more time it has to accumulate in the tissues.

During the subsequent ascent, pressure falls and less nitrogen can remain dissolved in the tissues. As pressure falls the nitrogen diffuses out of tissues and into the venous blood in the capillary beds of the head and body (see the circulation diagram). It is carried in the veins back to the lungs for elimination. Ideally, this elimination process occurs fast enough to dissipate the nitrogen molecules without bubble

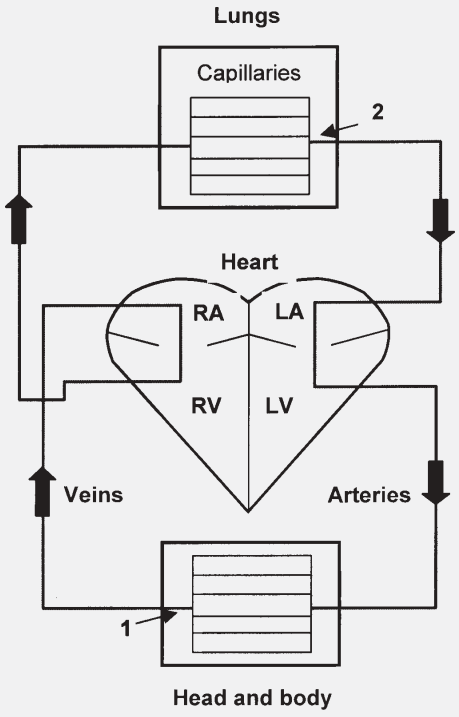

Figure 1. The human circulation. Oxygenated blood ("arterial blood") (red vessels) leaves the lungs and is carried to the left side of the heart. It enters the left atrium (LA), then the left ventricle (LV), which pumps the blood through the arteries to all the various tissues of the head and body. In these tissues the blood flows through thin walled capillaries, and across these thin walls, oxygen in the blood is exchanged for carbon dioxide in the tissue. De-oxygenated blood ("venous blood") (blue vessels) passes back to the right heart in the veins and is then pumped to the lungs. Once again, in the lungs the deoxygenated blood flows through thin walled capillaries where carbon dioxide in the blood is exchanged for oxygen in the tiny alveoli (air sacs) of the lung tissue. See text for explanation of points 1 and 2.

formation. However, in tissues that cannot get rid of their nitrogen very quickly, the pressure of dissolved nitrogen will exceed the ambient (surrounding) pressure at some point during the ascent (a process commonly referred to as "supersaturation"), and the molecules of dissolved nitrogen may not be able to "resist" the physical stimulus to form a bubble.

It is common to hear mention of "fast" and "slow" tissues in discussions of these processes. These terms refer to the speed with which a particular tissue takes up and eliminates nitrogen. This is determined by the amount of blood the tissue receives (more blood flow usually means that nitrogen uptake and elimination is faster) and other factors such as the relative solubility of nitrogen in the tissue and blood. As a rule, very fast tissues are quite resistant to bubble formation because although they take up nitrogen quickly, they usually get rid of it quickly enough to prevent bubble formation, so long as the ascent is made at the correct rate. It's the medium – fast tissues, such as spinal cord white matter, that often cause the problem in typical short duration sport dives. They can accumulate a large nitrogen load reasonably quickly, but might not lose it quickly enough during the ascent, especially if the bottom time was long and the ascent a little fast. "Slower" tissues such as tendons are often not a problem after a short dive, even if it is deep, because they don't have enough time to accumulate significant quantities of nitrogen. However, they become more important during long dives, or repetitive dives, when nitrogen can build up over a long time.

Bubble formation from dissolved gas can occur both within the tissues themselves and in the blood. Here's an important point: bubbles forming in the blood do so in the capillaries on the venous side of the head and body circulation (see point 1 on the diagram). If you think about it carefully, this is not surprising, since during ascent all the nitrogen molecules from the tissues are "trying" to get back to the lungs to be eliminated as described earlier. They enter the blood in the capillary beds of the head and body and seem prone to forming bubbles there. These bubbles are then carried back to the right side of the heart in the veins (follow it on the diagram) and then to the capillary bed of the lungs. The lung capillary bed is the first network of small blood vessels where these bubbles are likely to trap. Indeed the lung appears to be a good filter for venous bubbles, and seems able to tolerate a significant proportion of its capillaries being obstructed by bubbles without adverse effects. This is fortunate since without this "lung capillary filter" many of these bubbles might find their way into the arterial circulation (the red side of the diagram) where they can do more harm (see later). The fact that blood bypasses the lungs in the circulation of a foetus (and is therefore not filtered in this way), is one of the concerns over diving during pregnancy.

As an aside, when various references mention "Doppler bubble monitoring", it is the detection of these venous nitrogen bubbles using Doppler ultrasound to which they refer. From the above discussion it should be obvious why bubble counts made using this technique do not always correlate with the risk of DCI. As I have mentioned, most of these venous bubbles will be filtered out in the lung capillary bed and may do no harm. In contrast, the real villains might be the bubbles forming in the tissues themselves, and Doppler techniques cannot count these bubbles.

Bubbles can be introduced to the blood by damage to the lungs…

The second potential source of bubbles in DCI is the introduction of air bubbles to the arterial circulation because of lung over-expansion. This has nothing to do with dissolved nitrogen, or time and depth for that matter. Indeed, this problem can arise during ascent from depths as shallow as 1 – 2 metres. Open water trainees are always taught that the most important rule in scuba diving is "to breathe normally at all times; never hold your breath". This is because any air trapped in the lungs during an ascent (by holding the breath for example), will expand as pressure decreases. If there is sufficient over-expansion of the lung it may rupture some of the small airways and the associated blood vessels. Such damage is referred to as pulmonary barotrauma. This, in turn, may result in the introduction of air bubbles to the lung capillary circulation (point 2 on the diagram). These bubbles are then carried back to the left side of the heart from whence they are pumped out into the arteries. You can see from the circulation diagram that the first network of small blood vessels where these bubbles are likely to trap is the capillary beds of various tissues in the head and body. Some of the organs potentially affected in this way, such as the brain, are much less tolerant of bubbles blocking blood vessels than are the lungs. Indeed, bubbles arriving in the circulation of the brain may cause stroke-like symptoms. These arterial bubbles are therefore considered very dangerous.

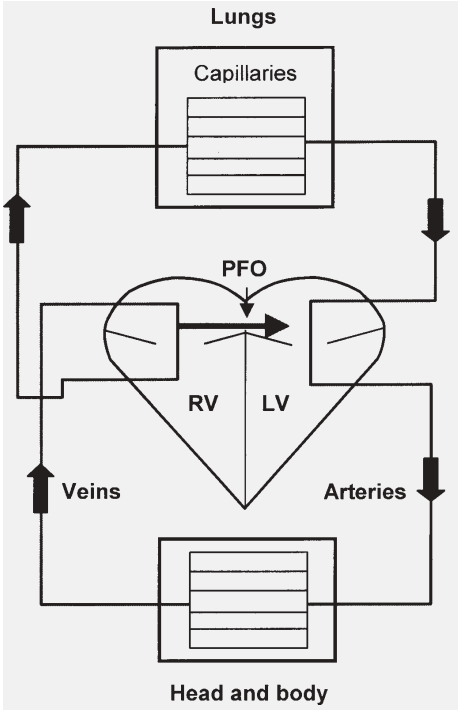

Figure 2. The human circulation showing a persistent patent foramen ovale (PFO). Compare this to Figure 1. In this situation a communication exists between the right and left atria of the heart, and blood may potentially move from the venous side of the circulation to the arterial side (blue arrow). This "shunting" blood may carry venous bubbles that would normally be filtered out by the capillary bed of the lungs into the arterial circulation where they are potentially more dangerous. In reality, most PFOs are small and may shunt very little, if at all. Small PFOs are of doubtful significance in diving, but there is a clear association between large shunting PFOs and DCI.

Venous bubbles can become arterial bubbles….

Considering that the lung filters venous bubbles while arterial bubbles can block blood vessels in vital structures such as the brain, it might seem fair to conclude that the bubbles forming from dissolved nitrogen in the veins are less dangerous than bubbles introduced to the arteries by lung pulmonary barotrauma. However, several studies have shown that if the diver has a persistent **"patent foramen ovale" (PFO)**, a hole between the two upper chambers of the heart which usually closes at birth, it may

allow venous nitrogen bubbles to cross into the arterial circulation, bypassing the lung capillary "filter" (see Figure 2). When this happens, the venous nitrogen bubbles can, in theory, cause all the same problems as air bubbles introduced to the arteries by pulmonary barotrauma.

Not surprisingly therefore, it is often difficult to tell which of the two sources of bubbles is responsible for the symptoms in some cases of DCI. This is the main reason why we have tended to drift away from the old terminology for these problems. Previously we used "decompression sickness" (DCS) to describe problems related to bubbles formed from dissolved nitrogen, and "arterial gas embolism" (AGE) to describe problems related to air bubbles introduced to the arteries by pulmonary barotrauma. Give the obvious potential for diagnostic confusion, it seems most sensible to refer to both processes under the single decompression illness label.

How do bubbles cause problems...

In the second article of this series we will go on to look at how bubbles in tissue and blood cause problems, and the nature of the symptoms they produce.

□

ABOUT THE AUTHOR

Dr. Simon Mitchell has been Director of the Diving Medicine Unit at the Royal New Zealand Naval Hospital, Auckland and at the Wesley Centre for Hyperbaric Medicine in Brisbane Australia. He is an accomplished medical researcher, technical diver, dive instructor and underwater photographer.

DAN SEAP FINANCES

DAN SEAP is a non-profit association, and, as such, is subject to an annual financial audit. In the 2002-03 financial year, the gross takings were AUD$1,026,447, with an operating surplus of $103,723.

Despite the turmoil and decreased economic, diving and travel activity within the Asia-Pacific Region, we managed to maintain income and achieve a good, although reduced operating surplus. However, the final months of the financial year were far more subdued and we are expecting a substantial reduction in income and surplus in the next financial year.

As planned, much of the previous surplus was spent on the building of a dedicated training area within our main offices, and several research grants to which we had committed.

The current surplus is expected to be used to help offset the anticipated decline in revenue for the 2003-04 year.

Following is an outline of revenue and expenses for the past financial year:

REVENUE

Source	%
Membership & insurance	60
Product sales	19
Training	15
Other	6

EXPENSES

Source	%
Product	16
Wages	27
Membership	24
Building	8
Post & freight	7
Advertising	4
Telephone & hotlines	2
Travel	1
Other	13