The Mechanisms of Decompression Illness

- Part 2

Part 2 of a 3-part article by
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The Mechanisms and Symptoms of Bubble Trouble

Decompression illness (DCI) is the most talked about medical problem in diving. In this article we continue our "in-depth" look at this mysterious and unpredictable disease. Last issue, we established that DCI was caused by bubble formation in blood and tissues during or soon after ascent from a compressed air dive. We noted that there were two potential sources of bubbles: formation from dissolved nitrogen; and pressure damage to the lungs. We also noted that bubble formation from dissolved nitrogen occurred in the tissues and venous blood, and that lung pressure damage introduced air bubbles directly into the arteries. That is a very brief summary of a long article, and those who are interested but didn't read it should try to get a copy. This issue we will consider the way in which bubble formation in these various locations cause problems, and the symptoms that are produced.

Not all bubbles cause problems….

There is now a mass of evidence, mainly from Doppler studies of bubbles in venous blood, that most dives result in bubble formation. Interestingly, in most cases, this does not produce symptoms or cause harm to the diver, and accordingly, this phenomenon is commonly referred to as "silent bubbling". The reason for bubble formation becoming symptomatic as opposed to "silent" is not clearly established, but it is almost certainly more likely if there are more bubbles, they are larger, or they are forming in vital tissues. In addition, it seems likely that susceptibility to a given degree of bubbling may vary between individuals, or within individuals from day to day. The reason(s) for this is not clear either, but variation in the vigor of some of the harmful processes initiated by bubbles (see below) may be important.

Problems caused by bubble formation in blood…

Bubbles formed in blood, be it arterial or venous blood (see Figure 1), are recognised as "foreign matter" and their presence turns on a variety of the body's inflammatory processes. These are designed to lead the way in attacking foreign invaders such as bacteria, but unfortunately, they serve no useful purpose in the presence of bubbles. Indeed, a vigorous inflammatory response may actually be harmful. For example, the release of certain chemicals by activated blood cells may cause leakiness in blood vessel walls, and loss of plasma from the blood into the surrounding tissues. This, in turn, may cause the blood to become thicker and to circulate less efficiently through the very small blood vessels. In addition, bubbles can initiate a clotting response, and if this gathers momentum and becomes widespread the patient can become very sick indeed. Thankfully, these dangerous inflammatory and clotting processes appear only in the most serious of DCI cases and are therefore relatively rare. Perhaps the most common symptom of DCI related to bubble-induced inflammation is fatigue and malaise similar to the early symptoms of a "flu-like" illness. It is not surprising that such symptoms should arise in DCI since, as suggested earlier, the same inflammatory processes are activated by bubbles as are activated by the presence of viruses or bacteria, and it is these processes that lead to that feeling of being "off-colour" in both situations.

Once introduced into blood, bubbles will move with the flow until they enter a blood vessel which is small enough to trap them.

In the case of bubbles formed from dissolved nitrogen in the veins, this will be the capillary beds of the lungs. As mentioned in the previous article, it is fortunate that the lungs appear to be an efficient filter for venous bubbles since this prevents them getting through to the arterial side of

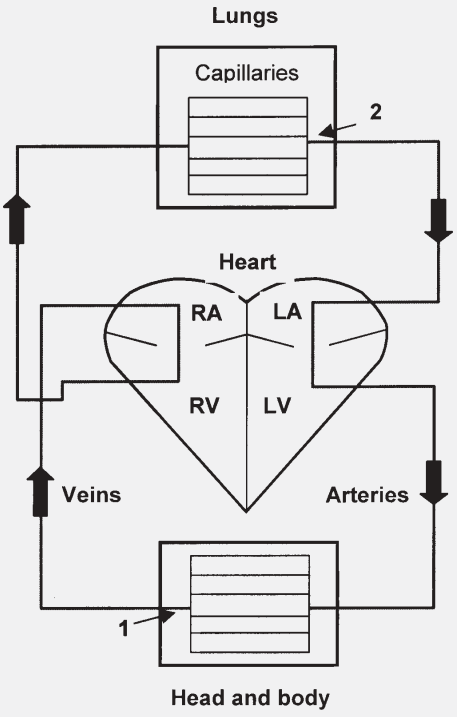

Figure 1. The human circulation. Oxygenated blood ("arterial blood") (red vessels) leaves the lungs and is carried to the left side of the heart. It enters the left atrium (LA), then the left ventricle (LV), which pumps the blood through the arteries to all the various tissues of the head and body. In 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.

the circulation (see Figure 1). However, if there are sufficient bubbles entering the lung capillary bed over a short period this can cause a feeling of chest discomfort and shortness of breath. If this is to happen, it usually does so within the first 30 minutes of a dive, and it is considered a bad sign. If there are sufficient bubbles entering the lung circulation to cause symptoms, this usually means that there are many bubbles forming in other places, and that other symptoms are not far behind.

In the case of bubbles entering the arteries, either because of lung pressure damage or because nitrogen bubbles formed in the veins cross to the left side of the heart through a patent foramen ovale (see previous article), they may end up in the capillaries of virtually any tissue in the body. Unfortunately, because the brain receives a large amount of blood flow, it also receives a significant proportion of any bubbles in the arteries. Moreover, while many tissues are relatively unaffected by the presence of bubbles in their small blood vessels, the brain is very sensitive to any interruption of blood flow.

Thus, when bubbles become trapped in the blood vessels of the brain we usually see rapid onset of dramatic neurological symptoms such as loss of consciousness, disorientation, visual changes, speech problems, weakness, and sensory changes. It is typical for this to occur within minutes of surfacing from the dive. Interestingly, depending on their size, bubbles may trap only very briefly. They commonly dissipate through the brain circulation and flow is restored. The patient may appear to recover at this point and this is frequently and inappropriately interpreted as a sign that nothing further needs to be done. Unfortunately, in their passing, the bubbles often damage the blood vessel walls, and the patient may slowly deteriorate again as inflammatory changes gradually interrupt blood flow again. In addition, there may be more bubbles around which may also enter the brain circulation, especially if the patient is allowed to sit or stand from a recumbent position. It follows that patients who have exhibited rapid onset neurological symptoms soon after a dive should, irrespective of any spontaneous recovery that occurs, be given oxygen, maintained in a horizontal posture, and urgently evacuated to a hyperbaric unit.

Problems caused by bubble formation in tissues…

As discussed in the previous article, bubbles may form from dissolved nitrogen within the tissues themselves. The mechanisms by which they cause damage and the symptoms they produce will vary from tissue to tissue.

One of the most serious forms of DCI is that involving the spinal cord. Although there is some controversy over the way in which bubbles injure the cord, the formation of bubble within the cord tissue itself is one of the most widely accepted mechanisms. It is not difficult to conceive that bubbles forming within this delicate tissue may cause damage by physically disrupting or distorting the delicate nerve fibres that run down its length. The bubbles may also disrupt small blood vessels lying nearby causing bleeding into the surrounding tissue. The injury resulting from these events may incite the same type of self-damaging inflammatory processes in the spinal tissue as was mentioned earlier in relation to bubble formation in the blood. The symptoms will depend upon the degree of disruption and damage, and the level of the cord

involved. If the lower part of the cord is affected, then changes in power and sensation in both legs can be expected. This may range from a little bit of tingling to total paraplegia and loss of sensation. It is common for bladder function and bowel control to be impaired or lost also. If the lesion is high in the cord, both the arms and legs may be involved. Very high lesions are life threatening because the patient's ability to breathe may be compromised. The prognosis for successful treatment of this form of DCI probably depends in part on the degree to which swelling and distortion are responsible for the loss of function, since these processes are reversible once the bubbles resolve. Unfortunately, if irreversible disruption of tissue has occurred, then recovery is unlikely even when bubbles are resolved by recompression.

The most common symptom of DCI, musculoskeletal pain, is also believed to arise from bubble formation from dissolved gas within tissue. Although divers often complain of "pain in the joints", the bubbles responsible for this pain do not form within joints. Rather, they almost certainly form in the pain-sensitive structures such as tendons, ligaments, and the joint capsule, which are either part of, or in close proximity to the joints. Although this causes pain while the bubble is present, these structures are unlikely to suffer any long-term damage as a result of bubble formation within. Thus, musculoskeletal DCI can usually be successfully treated with minimal long-term effects following on.

Some extra notes regarding symptoms….

Some very common symptoms of DCI are musculoskeletal pain and a tingling sensation in the skin. Although tingling does imply some neurological involvement, it is very common for patients to suffer tingling without progressing to more serious forms of neurological DCI. Nevertheless, tingling (or any other neurological symptom) occurring early after diving should always be taken seriously because it may be the first symptom of a progressive spinal problem, and such symptoms mandate urgent discussion of the patient with a diving physician.

Other relatively common symptoms are the constitutional symptoms such as fatigue and malaise, a perception of cognitive impairment, and headache. These are relatively "non-specific" symptoms, meaning that there are many potential causes. It would be unusual to make the diagnosis of DCI based on a headache alone, but interpretation of symptoms is best left to an experienced diving physician.

Symptoms of DCI usually arise within the first few hours of diving, especially if serious manifestations are going to occur. However, it is relatively common for divers to first notice symptoms of milder DCI less precipitously, and perhaps even the next day or later. If symptoms do not arise for some hours after a dive, it is highly unlikely that serious neurological disease is going to occur.

Part 3.

In the next article of this series we will consider the various factors considered as increasing and reducing the risk of DCI. □