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# DIVING MEDICINE

## CARBON DIOXIDE AND DIVING

In the December/January issue I presented an article about the limitations on performance of work underwater. One of the issues touched on in that article was the production and elimination of carbon dioxide. This has recently been a “hot” topic on rebreather diving discussion forums because a number of rebreather accidents have been linked to carbon dioxide toxicity. The December / January article prompted a number of email questions regarding carbon dioxide specifically, and so in this article I address that issue in a little more detail.

Carbon dioxide is a waste product of the normal energy producing reactions that consume oxygen in the cells of the body. It is produced in the tissues and carried in blood back to the lungs for elimination in the exhaled breath. Carriage in blood is in three forms. A small amount is dissolved, another small proportion is bonded to hemoglobin or plasma proteins, but most of the carbon dioxide in our blood is carried in a soluble form as bicarbonate, the end product of a chemical combination of carbon dioxide with water. The formation of bicarbonate liberates hydrogen ions in the blood, and although most of the hydrogen ions are buffered by haemoglobin and other proteins, this causes the acidity of the blood to increase. Increased acidity (due to hydrogen ions) is detected by the special “receptors” in the respiratory center of the brain, and the rate and depth of breathing is increased in order to eliminate carbon dioxide until the acidity is reduced to normal levels. In this regard, increasing carbon dioxide levels are recognized as an important stimulus that drives us to increase ventilation of the lungs.

It is critical to understand, at this early stage of the discussion, that the elimination of carbon dioxide from the body is directly related to ventilation of the lungs. Indeed, the relationship between carbon dioxide levels and ventilation can be described by this very simple equation:

$$PaCO_2 = VCO_2 \div VA$$

Where:

$PaCO_2$  = Pressure of carbon dioxide in arterial blood

$VCO_2$  = carbon dioxide production by the tissues

$VA$  = alveolar ventilation

In words, this equation tells us that arterial carbon

dioxide will tend to increase if carbon dioxide production increases or if alveolar ventilation decreases. Obviously, the opposites apply. If the normal balance between production and elimination becomes disturbed, then carbon dioxide can accumulate causing unpleasant and potentially deadly physiological effects.

### Carbon dioxide build up during diving

An excess of carbon dioxide in the blood is called “hypercapnia” and it may occur in diving for a variety of reasons. From the above equation we can predict that carbon dioxide may accumulate if there is an increase in carbon dioxide production or a decrease in ventilation. Whilst this equation applies equally in non-diving and diving situations, there are some “unique” aspects of diving that are important, particularly with respect to ventilation.

If we consider carbon dioxide production first, we can immediately appreciate that any extra exercise associated with diving (just like exercise in any context) will result in increased carbon dioxide production. Perhaps the only unique thing about diving is that breathing itself becomes an increasingly important part of the exercise load at deeper depths. This is because of the increased density of the breathing gas, and the extra work the diver’s respiratory muscles must perform in order to continue shifting the same volumes. Put into context, a diver swimming at 0.5 knots at 5m depth will be performing less work and producing less carbon dioxide than the same diver swimming at exactly the same speed at 30m, because at 30m there is extra work associated with breathing. Now, if we consider ventilation, we start encountering some very interesting problems. Because of the increased

density of breathing gas at greater depths, our ability to ventilate the lungs is reduced; more or less because it is just too hard to move the same volumes as we can at the surface. Indeed, breathing air at 4ATA (30m, 100’) our “maximum voluntary ventilation” (breathing as rapidly and deeply as we can to move as much air as possible) is only 50% that at the surface!! Herein lies the big problem. Whilst it is easy to start working just as hard at depth as we can at the surface (swimming per se is no harder at 30m than it is at the surface), it is entirely possible that we fail to eliminate the increased amount of carbon dioxide produced because we can’t breathe hard enough at this depth (whereas we could at the surface). Under these circumstances of increased production and decreased ventilation (and hence decreased carbon dioxide elimination),  $CO_2$  will start to accumulate.

There are other unique circumstances of diving that can amplify this basic physiological problem:

First, even greater depth-dependent breathing resistance may be imposed by the regulator or other breathing equipment, especially if they are poorly tuned or incorrectly set up. Breathing resistance may also be imposed by a tight exposure suit, buoyancy compensator or harness.

Second, the increased respiratory dead space created by a regulator may allow some expired carbon dioxide to be rebreathed, although this only happens to a small extent with the masks and regulators normally used by recreational divers.

Third, in rebreather diving, any failure of the scrubber to remove all carbon dioxide from the recycled gas will result in the diver inhaling carbon dioxide. This is not accounted for by the above equation, and potentially can cause very dramatic increases in arterial carbon dioxide levels. In a similar vein, if a diver’s air supply has been contaminated with extra carbon dioxide the consequences may be disastrous. In reality however, air contamination with carbon dioxide is vanishingly rare.

Fourth, it has been argued that both nitrogen narcosis and breathing oxygen at higher partial pressures than we are accustomed to at 1ATA may blunt the usual increased

drive to breathe that arises from increased carbon dioxide levels as described above.

Fifth, there is evidence to suggest that an increased drive to breathe (from increasing carbon dioxide levels) coupled simultaneously with increased breathing resistance (such as we see in diving – especially deeper diving) will cause adoption of a disadvantageous pattern of rapid shallow breathing that does not move much “fresh gas” and is only likely to exacerbate the problem. Indeed, this may induce a catastrophic spiral of increasing carbon dioxide levels, and less and less efficient ventilation.

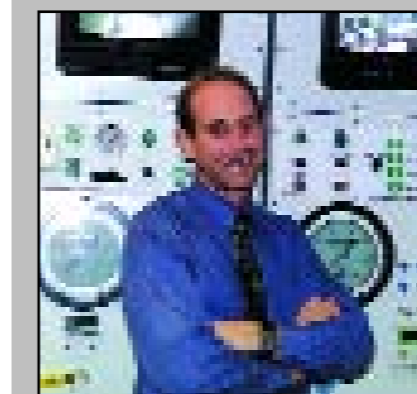
Sixth, some divers intentionally indulge in practices that are likely to promote retention of carbon dioxide. Fixation on reducing air supply consumption and indulging in intentional hypoventilation is the obvious example. So-called “skip breathing” where the diver intentionally holds their breath to reduce consumption is the ultimate expression of this dangerous practice.

Finally, although not unique to diving, certain individuals have a lower ventilatory response to increasing carbon dioxide levels than others and may therefore be at more risk of suffering problems in diving where there are many risk factors promoting hypercapnia as we have seen above. In addition, some research indicates that scuba divers may actually **develop** a reduced responsiveness to carbon dioxide and higher resting carbon dioxide levels than non-divers. Once again, this was more pronounced in certain individuals. It was suggested that this increased tolerance may be a consequence of the higher carbon dioxide levels experienced with some equipment designs during dives.

We can see from the above that there are many circumstances of diving that favor carbon dioxide accumulation, and this is a problem that all divers should be very aware of.

### Carbon dioxide toxicity

Carbon dioxide stimulates breathing, relaxes the smooth muscle in the walls of blood vessels in the brain. At sufficiently high levels, carbon dioxide will cause depression of the nervous system. It follows that as



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carbon dioxide levels increase the first sign is often an increase in the rate of breathing. The rapid breathing caused by hypercapnia can warn a diver of increasing carbon dioxide levels but, if he or she is exercising, the symptom can be masked by the expected increased breathing due to the exercise. As the level continues to increase a throbbing headache, usually at the front of the head, results from the dilation of blood vessels in the brain. The headache can be severe and can last for many hours after a dive. **Increased breathing rate and throbbing headache are the two symptoms of carbon dioxide excess commonly encountered by recreational divers.**

If the level continues to rise, (as it may in a rebreather system with a defective scrubber) dizziness, nausea, confusion, disorientation and restlessness can occur. Unconsciousness may ultimately follow as hypercapnia progresses.

#### **Response to symptoms of carbon dioxide toxicity**

If a diver develops symptoms of carbon dioxide toxicity they should immediately rest, take deep, regular breaths and try to relax. The dive should be terminated and the diver should return to the surface with as little effort as possible.

Elevated concentrations oxygen can be given if available. Divers have sometimes reported an improvement in symptoms after breathing elevated oxygen concentrations. Ordinary analgesics may provide some relief from the headache but it is not uncommon for a "carbon dioxide headache" to persist for many hours after a dive despite the use of analgesic medication.

In the context of rebreather diving, symptoms of carbon dioxide toxicity can mean that diver is simply exerting too much and ventilating insufficiently, just as in open circuit diving. However, as alluded to above, such symptoms may also indicate that there is a problem with the carbon dioxide scrubber. A detailed discussion of this rebreather failure mode is beyond the scope of this article, however there are several potential responses in such a situation. First, the diver could "bail-out" onto an emergency supply of open circuit gas carried for this purpose. Second, the diver could flush the rebreather loop with fresh gas to displace any gas contaminated with carbon dioxide. In a situation where there has been transient carbon dioxide breakthrough (through the scrubber) because of a short period of intense work, this strategy combined with rest and deep breathing is likely to bring the situation under control. Third, a closed circuit rebreather diver could adopt a "semi-closed circuit" mode of operation in which they exhale every third or fourth breath, resulting in the periodic drawing of fresh gas into the rebreather loop. The option that is taken will depend upon the rebreather type and the circumstances under which the carbon dioxide toxicity arises.

#### **Prevention**

A minor degree of hypercapnia is perhaps inevitable in divers, especially those who dive deeper. Some of the extra breathing effort is unavoidable as it is inherent to breathing denser gas through a regulator. However, the likelihood of significant hypercapnia can be minimized by:

Always breathing normally. Never try to conserve air by "skip breathing".

Using a regulator which provides the minimum resistance to breathing. Any regulator must be properly adjusted, maintained, and cleaned to minimize deposits of salt, sand and other foreign bodies which will affect its performance. Some older unbalanced regulators are not suitable for deep diving as they become harder to breathe from as the cylinder pressure drops.

Minimizing exertion during a dive, especially a deep dive.

If using a rebreather, by always replacing scrubber material at the end of its predicted life, and by correctly assembling the "scrubber" system.

The use of less dense helium-containing mixes for deep dives in order to reduce the work of breathing and improve the ability to ventilation efficiently. ■