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Advanced Knowledge Series: Carbon Dioxide Retention

Posted on 14/06/2016 by Sophie Fraser in Dive Medicine

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Introduction

This is the second in a series of articles that will appear in Dive New Zealand/Pacific with the aim of enhancing knowledge of select practically important issues in diving physiology and medicine. In the first article (Feb/Mar issue) I discussed some important aspects of basic carbon dioxide (CO₂) physiology. In this one, I will extend the discussion into an important applied aspect of diving knowledge: the prevention of oxygen toxicity. But first, a quick recap.

Recap

In the first article we discussed how CO_2 is produced in the tissues during the utilisation of oxygen. CO_2 is eliminated by breathing, and the more we breathe the more CO_2 is eliminated. This process of elimination is very precisely controlled by the brain to keep CO_2 in the body at a stable level. If CO_2 levels rise, the brain will 'drive' more breathing to bring CO_2 back to normal and vice versa. This is a completely automatic function. We touched on how this normal process of CO_2 control can be disturbed in diving because of an

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increase in the work required to breathe. The work of breathing increases because we are respiring a denser gas through a regulator or rebreather. In some people more than others, when the work of breathing rises, the brain seems less sensitive to rising levels of CO_2 and will avoid performing extra work to keep CO_2 levels normal. Thus, when under water, divers are prone to having CO_2 levels rise, particularly when exercising and when the work of breathing is high. We refer to this as ' CO_2 retention'.

The hazards of CO₂ retention

Rising blood CO₂ ('hypercapnia') is a problem in diving for several reasons. First, it can cause unpleasant symptoms such as headache, anxiety and shortness of breath. Collectively, these manifestations are often referred to as 'CO₂ toxicity'. The second reason high levels of CO₂ are a problem in diving is that CO₂ can precipitate other diving-related problems. In particular, CO₂ is a narcotic gas and high CO₂ levels will substantially worsen nitrogen narcosis. Similarly, high levels of CO₂ are known to significantly increase the risk of cerebral oxygen toxicity, which can manifest as a seizure with little or no warning. The mechanism for this is probably that high CO₂ levels cause a substantial increase in blood flow to the brain, thus increasing the brain's exposure to oxygen.

CO₂ and oxygen toxicity

Concerns about oxygen toxicity are particularly relevant to nitrox and technical divers who frequently breathe a substantially elevated pressure of oxygen (PO₂). These divers are taught to monitor their oxygen exposure using tables that are in some ways analogous to dive tables. The oxygen exposure is a function of the PO₂ breathed, and the duration of exposure to that pressure. Thus, for a given PO₂, the table will tell the diver the recommended maximum time for a 'safe' exposure. As you might imagine, as the respired PO₂ increases, the duration of 'safe' exposure is reduced and vice versa.

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Since the oxygen exposure accumulates during the dive, it follows that the greatest cumulative exposure occurs near the end of the dive. In technical diving the exposure peaks toward the end of the shallowest decompression stop. On some very deep dives the decompressions are very long and oxygen exposures inevitably exceed the recommended maximums. This creates anxiety amongst divers who naturally worry about the possibility of a seizure caused by oxygen toxicity.

I have often been asked for my opinion on the degree to which we should worry about exceeding recommended oxygen exposures during decompression. The truth is that the oxygen exposure limits are somewhat arbitrary and not proven by data. Moreover, oxygen toxicity seizures are very rare during technical diving decompressions, so we can't be doing anything markedly wrong. I have always believed that so long as the maximum inspired PO₂ is kept reasonable (ideally not more than 1.3–1.45 bar) and the diver is effectively resting, then the risk of a seizure would be small. Equally, I have always been a little reluctant to be dismissive of the risk because there are some unanswered questions, one of which is whether there is a tendency to retain CO₂ during decompression stops at shallow depths. If such a tendency exists, then I would feel less comfortable about exceeding recommended oxygen exposures during decompression. No one had ever investigated divers for CO₂ retention during decompression in real-world dives.



The CO₂ retention during decompression study

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A diver just arrived at surface has their end tidal CO_2 measured by the author. Photo by Pete Mesley.

good estimate of CO_2 levels in the body (and therefore a good indication of whether CO_2 is being retained) is provided by measuring the CO₂ in the expired breath at the end of exhalation. This is referred to as the 'end tidal CO₂ measurement'. Unfortunately, no one has perfected a means of obtaining this measurement in immersed divers on actual real-world dives. However, on a recent trip to Bikini Atoll we went second best by measuring the end tidal CO₂ in rebreather divers completing decompression immediately upon surfacing after direct ascent from the final decompression stop. We did this following 34 dives completed by 18 divers, and we repeated each diver's measurement at rest on the boat later for comparison. The surfacing divers had an average end tidal CO₂ of 36.8mmHg (normal is 35–45) and the measurements after rest on the surface averaged 36.9mmHg.¹ Thus we concluded that there was no general tendency to retain CO₂ during decompression.

This result is reassuring. It does not, of course, exclude the possibility of oxygen toxicity after long exposures to a high PO₂ when resting on decompression, but it does suggest that one of the principle risk factors (high CO₂) is usually not present. Divers have to make their own choices about risk when it comes to the PO₂ they inspire during decompression. Many technical divers breathe 100% oxygen from 6m (where the inspired PO₂ would therefore be 1.6bar). There are very few documented seizures associated with this practice, which is perhaps not so surprising given our study's findings. I personally prefer to go on 100% at my shallowest stop, which is usually 3–4.5m. The reduced inspired PO₂ at these depths (PO₂ = 1.3–1.45 bar) will inevitably further reduce the risk of oxygen toxicity.

Future articles in this series

The third article in this series will discuss our recent Aucklandbased study in which we investigated whether divers could tell if a rebreather scrubber was working properly by simply breathing on the loop.

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Mitchell S.J., Mesley P., Hannam J.A. End tidal CO₂ in recreational rebreather divers on surfacing after decompression dives. *Aerosp Med Hum Perform*. 2015; 86(1): 41–45.

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