Oxygen Toxicity and CCR Diving

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I recently had the privilege of spending five days in Florida with Lamar Hires, the owner of Dive Rite. That was the longest time we have managed to spend together since he 'certified' me as a full cave diver in 1988 (I already had over 100 exploratory cave dives in Canada). Although I am an Inspiration CCR IT (I have been diving the Inspiration since 2000 and the Megalodon since 2005), I did the full Optima CCR course with Lamar as he had other students to train. During this time Lamar and I had hours to chat and it quickly became apparent that there are some serious mistakes being made by CCR divers as a result of their lack of understanding of oxygen (O2) toxicity in the CCR diving environment. Lamar asked me to write this article to address some of those mistakes. Other articles that I have written on oxygen toxicity and diving are posted on this website. I strongly suggest you stop and read them now as you need to know what they say to fully understand this article.

Oxygen toxicity is a consequence of biochemical damage that occurs in cells as a result of oxygen free radicals. Whenever oxygen is present, oxygen free radicals are formed. Our cells have several mechanisms to inactivate oxygen radicles and to repair the damage that they cause. These defenses are able to keep ahead of the damage at normal partial pressures of oxygen (pO2) but they fall behind when the pO2 exceeds about 0.45 atmospheres (ata).

At pO2s of 0.45 to 1.3 ata the lungs are usually the first tissue in the body to show the effects of oxygen damage. Mild cough and painful inspiration progress to uncontrollable cough and very painful inspiration. Exposure times of "days" are usually required to experience symptoms. Levels of discomfort that will be tolerated by a diver will completely heal in about 4 weeks.

At pO2s of 1.3 to 1.6 ata divers can experience oxygen damage to the eyes (hyperbaric induced myopia) where the diver becomes near-sited. This usually requires exposure times in the range of 30 or more hours over 10 or so days. I had one diving companion who developed this problem after 33 hours CCR diving using a pO2 set point of 1.3 ata over 11 days. The myopia largely resolves over a few months but the diver may be left with a small permanent visual change, and they may be more susceptible to recurrence of the problem in the future.

At pO2s of 1.3 to 1.6 ata divers can also experience convulsions (CNS toxicity). The risk of convulsing is related to the pO2, the time of exposure, the work level, the level of carbon-dioxide, and individual variation. The problem is that the risk of convulsing is highly variable in the same person from day to day. What this means is that you might tolerate a very high O2 exposure without problem on one day but convulse at a relatively low O2 exposure another day. There is absolutely NO WARNING before the convulsion starts and if you are in the water when you have a convulsion you will most likely drown or embolize.

NOAA has come up with a conservative set of exposure limits that will protect most divers most of the time. However, these limits are designed for open circuit bounce dives and NOT for CCR diving. Many CCR divers are using procedures based on assumptions that are NOT physiologically correct.

For example, many CCR divers push the pO2 limits to reduce the amount of required decompression. The bottom line is that a small increase in the pO2, say from 1.3 ata to 1.4, 1.5, or even 1.6 ata will only remove a few minutes from your decompression time while drastically increasing your risk of an O2 convulsion. Most CCR manufacturers recommend that you NEVER have a pO2 in the breathing loop of more than 1.3 ata. I have been strongly supportive of this philosophy since I started CCR diving in 2000.

As a result of hyperbaric induced myopia, some CCR divers are using a pO2 of 1.2 ata or even less as their maximum on any dive. Certainly if you are going to dive more than three hours in one day or if you are going to be doing several consecutive days of CCR diving you need to reduce the pO2 to 1.2 or even to 1.1 ata avoid O2 toxicity. I know a few CCR divers who never use a pO2 of more than 1.0 ata.

The biggest mistake many CCR divers make is to elevate the pO2 at the end of the dive, during decompression. The logic is that they are at rest and therefore the risk of an O2 convulsion is reduced. This logic is correct, but it fails to consider several other factors.

CNS O2 toxicity is a result of cumulative damage in the cells. At the end of a CCR dive that requires decompression a significant amount of damage has occurred. If you then increase the pO2 you will increase

the rate of damage and you will dramatically increase the risk of suffering an O2 convulsion, even if you are at rest.

I was absolutely convinced of this point in 2000 when I started diving CCR and flatly refused to perform this procedure even though the VERY senior instructor (not Lamar) on my CCR Trimix course strongly recommended it. Since then I have reviewed several CCR fatalities where death was almost certainly as a result of an O2 convulsion secondary to pushing the pO2s.

I stated previously that you may do the same dive with high pO2s many, many times without problem and then suffer a seizure on the next dive. However, there are several reasons CCR divers are more likely to suffer an O2 seizure than OC divers.

While diving CCR the diver is often exposed to the maximum pO2 for the entire dive. Diving OC the diver is exposed to the maximum pO2 only when they are at the maximum depth of the dive and during the first decompression stop after a gas switch.

While diving CCR the diver is often exposed to an elevated partial pressure of carbon-dioxide (pCO2). There are several reasons all divers are exposed to elevated pCO2 but when diving CCR there are more reasons and the elevation of CO2 can be greater. Failure of the one-way values sometimes occurs (usually not installed correctly) but by far the most common reason is failure of the CO2 absorbent due to a number of problems that are almost always the diver's fault. Diver's don't pack the absorbent correctly, it settles during a long car or boat ride, divers remove and then refill the canister with the same absorbent, channeling can occur, etc. but most commonly divers simply dive too long on one fill to try and save a few dollars.

So let's return to the practice of elevating the pO2 during decompression. Not only is the brain at the highest risk of convulsing due to the accumulated damage that occurred during the dive, but the pCO2 is most likely to be elevated as the absorbent is partially or mostly used up.

So why does pCO2 matter so much in O2 toxicity? Quite simply, pCO2 controls the blood flow to the brain. As the pCO2 rises, the blood flow to the brain is increased. As the blood flow to the brain is increased, more O2 (and O2 radicals) will be delivered to the brain even if the pO2 remains constant! More O2 radicals results in more damage to the cells. On top of this, if the diver then also increases the pO2 ……. is it any wonder that they convulse?

I have to cover one final point and that is 'air breaks'. The risk of CNS O2 toxicity can be dramatically reduced if the diver breathes a gas mixture with a reduced pO2 for 5 minutes after every 20 to 25 minutes of breathing a gas mixture with a higher pO2. While sitting in a dry chamber breathing 100% O2 at 2.0 ata, the diver can breathe O2 for twice as long before developing a specific level of pulmonary O2 toxicity if they breathe air (pO2 0.4 ata) for 5 minutes after every 20 minutes of O2. During the 5 minute "air break" the number of O2 radicals is dramatically reduced. As a result, the cells 'catch up' and repair some of the damage that occurred while the diver was breathing a higher pO2.

Theoretically it is quite easy to do this while diving (switch to an OC regulator on a tank of air or normoxic trimix if you are shallow enough) but practically this is fairly difficult to do while diving. In addition, it is very challenging to sort out your decompression obligation if you are frequently switching gas mixtures.

So what is the bottom line? Taking all of the physics and physiology into consideration, understanding oxygen toxicity as well as anyone (there is still a lot we don't understand) and remembering how many CCR divers have died (many almost certainly as a result of O2 toxicity) I have the following recommendations.

CCR divers should NEVER have a pO2 in the loop greater than 1.3 ata. There have been a few well documented convulsions in divers with a pO2 of 1.3 ata but I am not aware of any at lower pO2s. Therefore, a very good argument can be made to never have a pO2 in the loop greater than 1.2 ata.

If you are going to be doing more than 3 hours diving in one day, or diving CCR for several days in a row, the pO2 should be set at 1.2 ata or less, starting with the first dive! The CO2 absorbent must be managed properly and if you decide to 'push' the times a bit, ensure your pO2s are reduced to 1.2 ata or less. Certainly the 'pre-package' absorbent used in the Optima eliminates many of the problems commonly encountered with loose absorbent.

These recommendations should result in a low, but not zero risk of an O2 induced seizure.

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