

# OXYGEN TOXICITY AND CCR/RE-BREATHER DIVING

*A couple of years ago I 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 had already done 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 serious mistakes being made by rebreather divers as a result of their lack of understanding of oxygen (O<sub>2</sub>) toxicity in the rebreather diving environment. Lamar asked me to write an article to address some of those mistakes. This article builds on prior articles that I have written on the topic of oxygen toxicity that have appeared on the Dive Rite blog.*

**Oxygen toxicity is a consequence of** the biochemical damage that occurs in cells as a result of oxygen free radicals. Whenever oxygen is present, oxygen free radicals are formed. The number of radicals is directly related to the partial pressure of oxygen (pO<sub>2</sub>). Our cells have several mechanisms to inactivate oxygen radicals and to repair the damage that they cause. These defenses are able to keep ahead of the damage at normal partial pressures of oxygen but they fall behind when the pO<sub>2</sub> exceeds about 0.45 atmospheres (ata).

**At pO<sub>2</sub>s 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 four weeks.

**At pO<sub>2</sub>s of 1.3 to 1.6 ata divers can** experience oxygen damage to the eyes (hyperbaric induced myopia) where the diver becomes near-sighted. 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 pO<sub>2</sub> set point of 1.3 ata over 11 days. The myopia usually resolves over a few months but the diver may be left with a small permanent visual change, and they may be more susceptible to this problem in the future.

**At pO<sub>2</sub>s of 1.3 to 1.6 ata divers can** also experience convulsions (CNS toxicity). The risk of convulsing is related to the pO<sub>2</sub>, 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 O<sub>2</sub> exposure without problem on one day but convulse at a relatively low O<sub>2</sub> 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 rebreather diving. Many rebreather divers are using procedures based on assumptions that are NOT physiologically correct.

**For example, many rebreather divers** push the pO<sub>2</sub> limits to reduce the amount of required decompression. The bottom line is that a small increase in the pO<sub>2</sub>, 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 O<sub>2</sub> convulsion. Most CCR manufacturers recommend that you NEVER have a pO<sub>2</sub> 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 pO<sub>2</sub> 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 rebreather diving you need to reduce the pO<sub>2</sub> to 1.2 or even to 1.1 ata to avoid O<sub>2</sub> toxicity. I know a few rebreather divers who never use a pO<sub>2</sub> of more than 1.0 ata.

**The biggest mistake many rebreather** divers make is to elevate the pO<sub>2</sub> at the end of the dive, during decompression. The logic is that they are at rest and therefore the risk of an O<sub>2</sub> convulsion is reduced. This statement is correct, but it fails to consider several other factors.

**CNS O<sub>2</sub> toxicity is a result of** cumulative damage in the cells. At the end of a rebreather dive that requires decompression a significant amount of damage has occurred. If you then increase the pO<sub>2</sub> you will make the rate of damage even greater and you will dramatically increase the risk of suffering a convulsion, even if you are at rest.

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**I was absolutely convinced of this point** in 2000 when I started diving CCR and flatly refused to perform this procedure even though the instructor on my CCR Trimix course (not Lamar but someone very senior in the community) strongly recommended it. Since then I have reviewed several rebreather fatalities where death was almost certainly as a result of an O<sub>2</sub> convulsion secondary to pushing the pO<sub>2</sub>s, at least one during decompression.

**I stated previously that you may do the** same dive with high pO<sub>2</sub>s many, many times without problem and then suffer a seizure on the next dive. In addition, there are several reasons rebreather divers are more likely to suffer an O<sub>2</sub> seizure than OC divers.

**When diving a rebreather the diver is** usually exposed to the maximum pO<sub>2</sub> for the entire dive. Diving OC the diver is exposed to the maximum pO<sub>2</sub> only when they are at the maximum depth of the dive and during the first decompression stop after a gas switch.

**While diving a rebreather the diver is** often exposed to an elevated partial pressure of carbon-dioxide (pCO<sub>2</sub>). There are several reasons all divers are exposed to elevated pCO<sub>2</sub> but when diving a rebreather there are more reasons and the elevation of pCO<sub>2</sub> can be greater. Failure of the one-way valves in the breathing loop sometimes occurs (usually not installed correctly) but by far the most common reason is failure of the CO<sub>2</sub> 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 pO<sub>2</sub> 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 pCO<sub>2</sub> may be elevated as the CO<sub>2</sub> absorbent has been partially used up.

**So why does pCO<sub>2</sub> matter so much in** O<sub>2</sub> toxicity? Quite simply, pCO<sub>2</sub> controls blood flow to the brain. As the pCO<sub>2</sub> rises, blood flow to the brain is increased. As blood flow to the brain is increased, more O<sub>2</sub> (and O<sub>2</sub> radicals) will be delivered to the brain even if the pO<sub>2</sub> remains constant! More O<sub>2</sub> radicals results in more damage to the cells. On top of this, if the diver also increases the pO<sub>2</sub> ... is it any wonder that they convulse?

**I have to cover one final point and that** is 'air breaks'. The risk of CN O<sub>2</sub> toxicity can be dramatically reduced if the diver breathes a gas mixture with a reduced pO<sub>2</sub> for five minutes after every 20 to 25 minutes of breathing a gas mixture with a higher pO<sub>2</sub>. While sitting in a dry chamber breathing 100% O<sub>2</sub> at 2.0 ata, the diver can breathe O<sub>2</sub> for twice as long before developing a specific level of pulmonary O<sub>2</sub> toxicity if they breathe air (pO<sub>2</sub> 0.4 ata) for five minutes after every 20 minutes of O<sub>2</sub>. During the five minute 'air break' the number of O<sub>2</sub> 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 pO<sub>2</sub>.

**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 a rebreather. 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 fairly well (there is still a lot we don't understand) and remembering how many rebreather divers have died (many almost certainly as a result of O<sub>2</sub> toxicity) I have the following recommendations.

**Rebreather divers should NEVER have a** pO<sub>2</sub> in the loop greater than 1.3 ata. There have been a few well documented convulsions in divers with a pO<sub>2</sub> of 1.3 ata but I am not aware of any at lower pO<sub>2</sub>s. Therefore, a very good argument can also be made to never have a pO<sub>2</sub> in the loop greater than 1.2 ata.

**If you are going to be doing more than** three hours diving in one day, or diving a rebreather for several days in a row, the pO<sub>2</sub> should be set at 1.2 ata or less, starting with the first dive!

**The CO<sub>2</sub> absorbent must be managed** properly and when the absorbent has been used for a while the pO<sub>2</sub> in the breathing loop should be reduced (not elevated for decompression). Certainly the 'pre-packaged' absorbent used in the Optima and other rebreathers eliminates many of the problems commonly encountered with loose absorbent.

**These recommendations should result in a low, but not zero risk of an O<sub>2</sub> induced seizure while diving rebreathers.**

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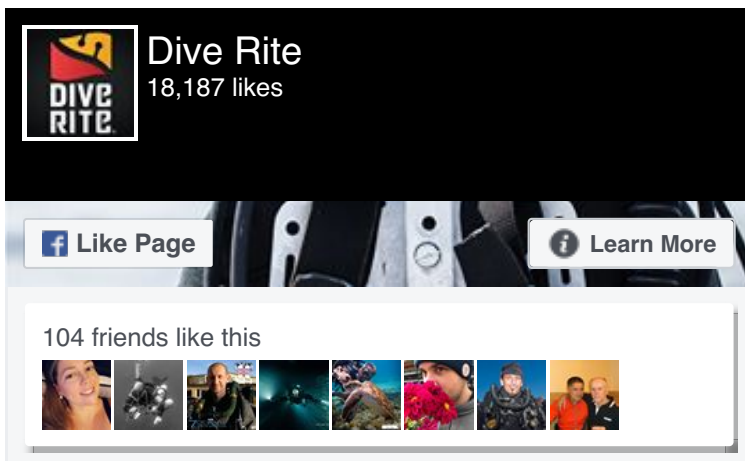
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