

Diving and Carbon Dioxide

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A healthy 44-year-old female with 21 logged dives prepares for a guided drift dive to a depth of 95 feet/29 meters. She doesn't exercise regularly and so is feeling a little apprehensive about diving in a current. As she begins her descent, the diver notices that her rental regulator doesn't deliver air quite as easily as others she's used. She thinks that it may not be adjusted properly, but doesn't have much experience with different regulators so she isn't sure. The diver continues her descent and enjoys the dive despite feeling mildly uncomfortable about her regulator. Toward the end of the dive, she becomes separated from the group. She has to kick against the current for several yards, and by the time she rejoins the group, she is fatigued, having a difficult time catching her breath, and feels the beginnings of a headache. She signals an ascent to the divemaster, who decides to bring the group up together. The group ascends normally and re-boards the boat, where the diver's headache worsens to the point that she is nearly incapacitated. She becomes nauseous and vomits over the side of the boat. The divemaster questions her and diagnoses CO₂ toxicity. The diver's headache resolves over the next six hours, and after a good breakfast the next morning, she reports that she feels fine.

Most of us are aware that our bodies produce carbon dioxide (CO₂) as a byproduct of metabolism, and that this CO₂ is eliminated via the lungs. At rest, the human body produces CO₂ at a rate of about 0.15-0.35 liters per minute (l/min) – in terms of volume, roughly anywhere from half of a small coffee cup to a 12 ounce drink cup at standard temperature and pressure. This can increase to about 4.5 l/m with aerobic exercise.

CO₂ level in the arteries is sensed by specialized nerve centers in the brain called central chemoreceptors. Our brains interpret this information and trigger our respiratory muscles when the CO₂ level reaches a certain point. At atmospheric pressure, healthy lungs will provide enough ventilation to allow the CO₂ level in the bloodstream to remain relatively constant.

Oxygen is carried almost exclusively on hemoglobin molecules in the red blood cells, with a small amount dissolved in the blood plasma. Carbon dioxide, on the other hand, is a little more complex. It is carried in the bloodstream in three ways: as bicarbonate (HCO₃⁻); dissolved in the blood plasma; and, combined with chemicals called amines, bound to hemoglobin (West 2005). Even though only about 10% of the body's CO₂ is dissolved in the plasma, the amount of dissolved CO₂ is considered to be an accurate representation of the overall CO₂ level in the bloodstream. We can measure dissolved CO₂ directly by drawing blood from either a vein or an artery and analyzing it. The level is expressed as a partial pressure, called pCO₂ (partial pressure of CO₂). Normal venous pCO₂ is between 40 and 55 millimeters of mercury (mmHg), and normal arterial pCO₂ is between 35 and 45 mmHg. Arterial pCO₂ is one of the parameters commonly used to assess ventilatory status.

Diving can significantly alter the body's ability to eliminate CO₂. A study conducted at the Duke Center for Hyperbaric Medicine and Environmental Physiology found four variables that can influence arterial pCO₂ during immersed exercise (Cherry, Forkner et al. 2009): depth, which corresponds to gas density; external breathing resistance; individual maximal oxygen consumption (VO₂ max); and individual hypercapnic ventilatory response. Let's look at some of these variables in the context of the fictional case study above.

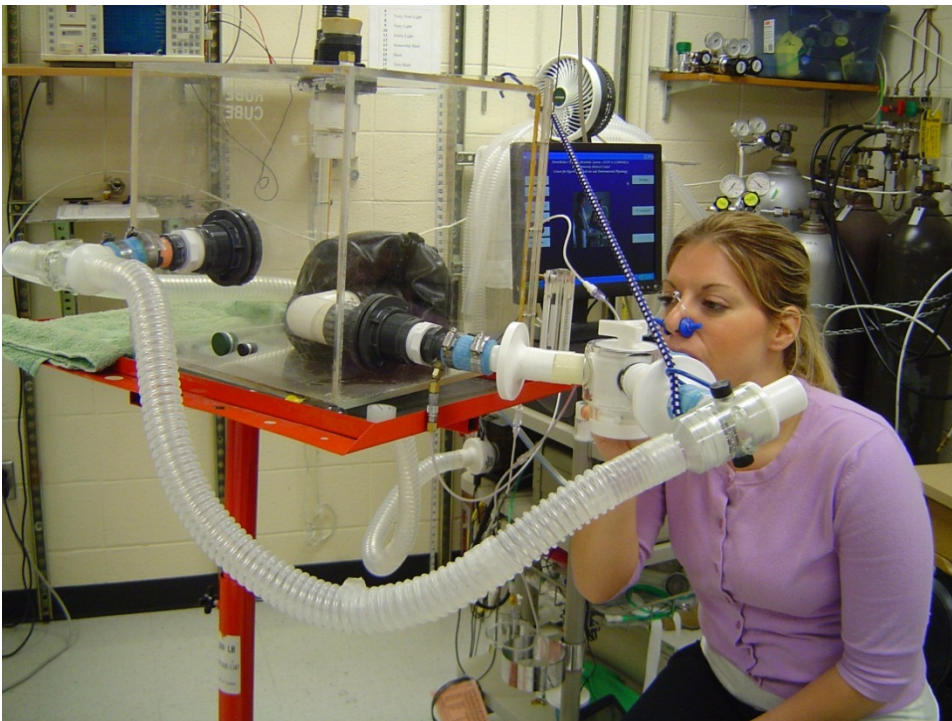
Our diver spent the majority of her dive at about 95 feet/29 meters. This corresponds to a pressure of about 3.88 atmospheres absolute (ATA), or nearly four times atmospheric pressure. Her breathing gas, then, was almost four times as dense as it was on the surface. Because of the flow characteristics of the bronchial tree, this is not readily apparent to a diver who is at rest or swimming slowly. However, when the diver increased her level of exertion by swimming against the current, her respiratory rate increased, along with the velocity of the gas in her airways. Under these

conditions, gas density becomes a significant factor and will limit the diver's maximum voluntary ventilation, or MVV (West 2005) (Hickey, Lundgren et al. 1983). This means, in essence, that the diver may not have been able to breathe quickly enough to eliminate the CO₂ that was building up in her bloodstream.

The diver noticed that she was having some difficulty drawing a breath from her regulator but because of her inexperience, failed to recognize this as an equipment malfunction. She chose to continue the dive rather than abort, and it's likely that her poorly-functioning regulator contributed to her incident. Using a specially-constructed underwater breathing apparatus, Cherry and colleagues demonstrated that increases in breathing resistance sometimes resulted in increased arterial pCO₂ in their test subjects, as measured by the partial pressure of CO₂ in the arteries (Cherry, Forkner et al. 2009).

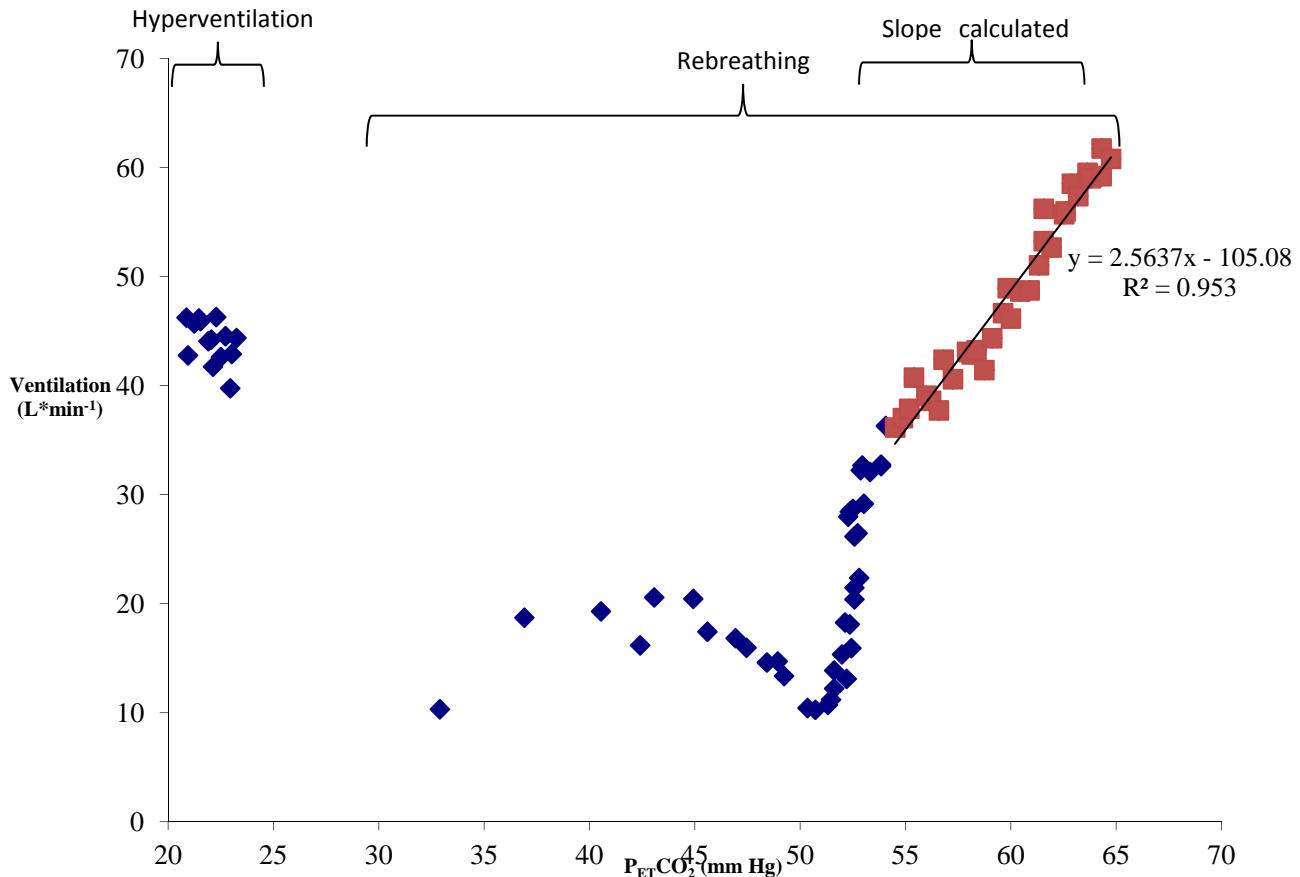
When we assess a patient or test subject's capacity for exercise in the lab, we often perform an individual maximal oxygen consumption test, also called a VO₂ max test. This test directly measures the amount of oxygen consumed by the body at maximal exercise. VO₂ max is expressed in liters per minute (l/min), and is a good indicator of aerobic fitness. The higher an individual's VO₂ max, the greater his or her exercise capacity. Though there is no reason for a healthy diver to undergo a VO₂ max test, all divers should be aware of their own levels of physical fitness and use that knowledge in planning their dives. A typical drift dive does not involve swimming against the current, but our diver from the case study above found herself in a situation where she had to exert herself. Her relatively poor physical condition became a detriment and a possible contributing factor to her CO₂ toxicity.

To quantify individual response to increasing levels of CO₂, researchers and clinicians may measure hypercapnic ventilatory response (HCVR). HCVR is the rate at which an individual's ventilation increases in response to increasing inspired (inhaled) CO₂ levels. The test begins with the subject hyperventilating in room air, after which he or she will inhale from, and exhale into, a large gas bag containing a mixture of 5% CO₂ and 95% O₂. The high oxygen content in the bag prevents hypoxia, while the high CO₂ content begins to provoke the subject's ventilatory response. The CO₂ content of the gas bag will increase as the subject exhales into it.



Duke Hyperbaric Center research assistant Stefanie Martina demonstrates the HCVR apparatus in the lab. Image courtesy Mike Natoli.

The subject's CO₂ level is measured with an end-tidal CO₂ meter, which measures the CO₂ level in the exhaled gas. Normal end-tidal CO₂ ranges from 35 to 45 mmHg. Measurement of the HCVR begins when the diver's end-tidal CO₂ reaches 55 mmHg, and ends when it reaches 65 mmHg. When the results are plotted on a graph, a line is drawn, and the slope of that line indicates the relative strength of the individual's HCVR. A steeper slope indicates a brisker HCVR, and a flatter slope indicates a weaker HCVR.



Sample HCVR graph. End-tidal CO₂ is on the x axis, and ventilation in liters per minute is on the y axis. The small squares are individual ventilation vs. end-tidal CO₂ readings. Image courtesy Mike Natoli.

Though hypercapnic ventilatory response is not addressed in our case study, it can play a significant role in some cases of CO₂ toxicity. Recently, we evaluated a diver who complained of severe, migraine-like headaches after surfacing from his dives. When the diver's HCVR was measured, the resulting graph was nearly flat, which means that his respiratory rate did not increase with increasing levels of CO₂ in his blood. Our physicians concluded that his blunted HCVR was allowing the partial pressure of CO₂ in his arteries to increase to toxic levels during his dives. He has since stopped diving and is awaiting further testing. If a diver experiences one or more unexplained episodes of CO₂ toxicity, an HCVR test may be indicated.

Dr. Cherry's research can be directly applied to good diving practice. Arguably, the most important practical aspect is that no matter how good a diver's physical condition and exercise tolerance are on the surface, gas density and breathing resistance become significant factors under water, and their influence increases with increasing depth. In other words, a diver can't exercise to the same level under water as he or she can on the surface. Still, the better a

diver's physical condition, the better he or she will be able to tolerate the rigors of diving, and the better he or she will be able to cope with unexpected circumstances like those encountered by our fictional drift diver.

Most divers know that skip-breathing can lead to dangerously high carbon dioxide levels, and modern diving classes discourage the practice. In this article, we've examined several other important factors in CO₂ toxicity. Gas density, external breathing resistance, individual exercise capacity, and individual hypercapnic ventilatory response all play a role in a diver's CO₂ level. Awareness of these is critical for divers, especially those who dive beyond recreational depth limits.

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