In: Pollock NW, Sellers SH, Godfrey JM, eds. Rebreathers and Scientific Diving. Proceedings of
NPS/NOAA/DAN/AAUS June 16-19, 2015 Workshop. Durham, NC; 2016.

Respiratory Physiology of Rebreather Diving

Gavin Anthony[1], Simon J. Mitchell[2]*

[1] QinetiQ, Gosport, Hampshire, United Kingdom
[2] Department of Anaesthesiology, University of Auckland, Auckland, New Zealand
sj.mitchell@auckland.ac.nz
* corresponding author

Abstract

The use of rebreathers imposes a number of stresses on the respiratory system that frequently provoke retention of carbon dioxide (CO_2) during diving. The most important physiological mechanism leading to CO_2 retention is a derangement of the control of breathing which is usually responsible for subconsciously adjusting lung ventilation to keep the arterial CO_2 (P_aCO_2) at a normal level. When the work of breathing increases during diving there is a tendency for this breathing control system to become insensitive to rising P_aCO_2. An elevated P_aCO_2 can cause unpleasant and dangerous symptoms, increase inert gas narcosis, and predispose to cerebral oxygen toxicity. It follows that strategies to mitigate the risk of CO_2 retention in rebreather diving are important. These include minimising the work of breathing through appropriate rebreather design, taking account of respired gas density when planning rebreather dives, minimising physical exertion (particularly when deep), and meticulous attention to equipment preparation and adherence to best practice guidelines for replacement of CO_2 absorbent material.

Keywords: diving, rebreather, carbon dioxide, breathing, ventilation, hypercapnia

Introduction

The principal function of the lungs is to bring venous blood and gas in the lung alveoli into close proximity so that carbon dioxide (CO_2) in the blood may be exchanged for oxygen (O_2) in the alveoli. In healthy individuals the lungs are remarkably efficient at this task, and ventilation (the volume of gas moved in and out of the alveoli per unit time) is 'automatically' controlled (see below) to maintain adequate oxygenation (an arterial blood PO_2 [P_aO_2] between 80 and 100 mm Hg) and normal CO_2 levels ('normocapnia' – an arterial blood PCO_2 [P_aCO_2] around 38±7.5 mm Hg [2SD]).

During diving the inspired PO_2 is almost always elevated to planned and safe levels of 'hyperoxia'. Thus, in the absence of equipment malfunction or diver error, hypoxia or symptomatic hyperoxia are unexpected. In contrast, both immersion and the use of rebreathers (or other underwater breathing apparatus) impose challenges to maintenance of normal respiratory control and CO_2 homeostasis. As a result, a P_aCO_2 higher than normal (hypercapnia) is frequently encountered in the absence of any error or equipment related problem. This is important because hypercapnia can augment inert gas narcosis, increase the risk of oxygen toxicity, and produce unpleasant symptoms such as shortness of breath, confusion, anxiety, and ultimately unconsciousness.

This article will focus on the physiological mechanisms which may lead to hypercapnia during diving. It will begin with a brief account of normal CO_2 physiology. It will then examine the reasons why the work of breathing may increase when a diver is immersed using rebreathers, and the physiological basis for this to cause hypercapnia. Finally, it will examine the strategies divers may use to mitigate these physiological challenges. With the target scientific diver audience in mind, the article is deliberately written in a didactic style and does not assume detailed prior knowledge. It is not intended as a comprehensive academic work on the subject. Such treatments can be found elsewhere (Doolette and Mitchell 2011).

Normal CO_2 Physiology

Carbon dioxide is a by-product of metabolism of oxygen in cells. It is a volatile acid and will produce unwanted biochemical derangements (and symptoms as mentioned above) if levels in the body are allowed to increase. CO_2 diffuses from tissues to venous blood and is carried to the lungs where it diffuses from blood to alveoli and is breathed out. Maintenance of the diffusion gradient that drives this process is entirely dependent on movement of fresh gas in and out of the lungs ('ventilation'). Thus, greater ventilation will remove more CO_2 from the alveoli, thus maintaining an increased partial pressure gradient for CO_2 diffusion from the venous blood. Conversely, less ventilation will remove less CO_2 from the alveoli and less CO_2 will be removed from the blood. The crucial message here is that the amount of CO_2 eliminated from the body is directly proportional to ventilation. The relevant processes are depicted in Figure 1.

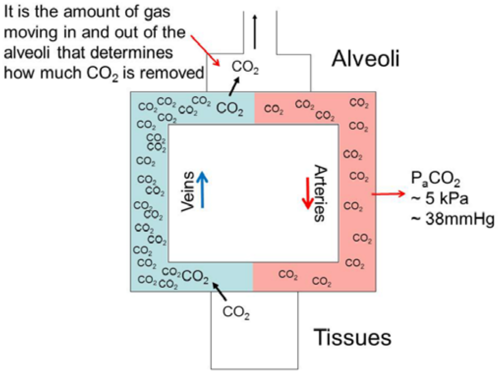

Figure 1. Depiction of the process of production and elimination of CO_2.

It can be deduced from Figure 1 that the production of CO_2 in tissues and its removal by the lungs are processes that must be balanced. If ventilation of the lungs is inadequate ('hypoventilation') CO_2 levels will increase, and if ventilation is excessive ('hyperventilation') then CO_2 levels will decrease. The process of balancing CO_2 elimination by the lungs with production by the tissues is mediated through control of ventilation by the respiratory controller in the brain stem. Although maintenance of adequate oxygenation would seem intuitively more important than CO_2 regulation, and although both hypoxia and hypercapnia do provoke the respiratory controller to increase breathing, it is the P_aCO_2 that is widely accepted as the primary effector. The respiratory controller indirectly monitors arterial CO_2 levels through sensing of the pH of the cerebrospinal fluid (which is directly influenced by P_aCO_2). If CO_2 levels increase then the respiratory controller will drive increased ventilation to remove more CO_2 and vice versa. The controller generally 'defends' a P_aCO_2 around 38 mm Hg ± 7.5 mm Hg (2SD) (5.1 kPa ± 1 kPa [2SD]), though as will be seen below, this can be disturbed in diving. This is a substantial oversimplification of a complex and incompletely understood process, but it is adequate for the purposes of this discussion.

The most common derangement of this system during diving is that there may be inadequate ventilation and an increase in P_aCO_2; a process often referred to as 'CO_2 retention'. The obvious question is 'what causes divers to hypoventilate thus allowing the P_aCO_2 to rise?' The answer is not simple or even fully understood, but a significant contribution to the process occurs because of the increase in the work of breathing that occurs during diving. Thus, in the following section we briefly consider the causes of increased work of breathing in diving.

Causes of Increased Work of Breathing in Diving

There are multiple factors that increase the physical effort required to move gas in and out of the lungs during diving.

Immersion effects

Immersion may cause changes in the mechanical properties of the lungs if the chest is exposed to a different external pressure than the pressure inside the airways. For example, consider a diver upright in the water using open-circuit scuba. The regulator supplies gas at a pressure equating to the ambient pressure at the depth of the second stage (mouthpiece). Since the diver's airways are connected to this regulator, the pressure inside the airways is therefore the same as the ambient pressure at the depth of the mouth. The lungs themselves (remember the diver in this example is upright) are slightly deeper than the mouth and they are therefore exposed to an external water pressure that is slightly higher than the pressure inside the airways. This difference in pressure 'across' the lung (the pressure within the airways being slightly less than the pressure on the outside of the lung) is called a 'negative static lung load' or 'hydrostatic imbalance'. The relative negative pressure inside the lung airways encourages blood to engorge the relatively distensible lung blood vessels, and this renders the lung stiffer than normal. Put another way, the lung's compliance is reduced meaning that more muscular effort would be needed to move the same amount of gas in and out. A negative static lung load also exists when a rebreather diver with a back-mounted counterlung is swimming in a horizontal position. In this setting, the airways are in continuity with (and contain gas at the same pressure as) the counterlung, which is sitting at a slightly shallower depth (and lower pressure) than the lungs themselves.

Static lung loads can vary according to the type of equipment (open- or closed-circuit), the position of the counterlung in the latter, and the orientation of the diver in the water. It is beyond the scope of this article to discuss the various combinations of circumstances that may arise. Suffice it to say that under some commonly encountered circumstances, static lung loads (and particularly negative static lung loads) can increase the work of breathing during diving as described.

Equipment-related resistance

The use of underwater breathing apparatus imposes an external resistance to breathing. It is intuitively apparent that this would be potentially important in a rebreather. In using a rebreather all of the energy required to propel gas through the hoses, various connectors, and the CO_2 scrubber, must be provided by the diver's own effort. In this regard, the design of the rebreather (and in particular considerations like the geometry of the gas flow path, diameter of hoses, and type of CO_2 absorbent canister) can make a substantial difference to the work of breathing. Not surprisingly, there are recommended standards for maximum work of in underwater breathing apparatus. Relevant standards and testing of rebreathers in this regard are discussed in more detail by Anthony (2009).

Gas density

One of the most important influences on work of breathing in diving is the increase in density of respired gas that occurs as depth increases. Since any underwater breathing apparatus will supply gas at ambient pressure, the density of the respired gas increases in direct proportion to depth. Increases in gas density result in a parallel increase in the resistance to flow of the gas through the diver's own airways, and in rebreather diving there is also the extra effort of moving dense gas through the hoses, connectors and CO_2 scrubber of the unit. Under these circumstances, the associated increase in the work of breathing can be substantial.

Another relevant phenomenon profoundly affected by gas density is a reduction in the maximal ventilation that can be achieved even when a diver is consciously attempting to move as much gas as possible in and out of the lungs. For example, in dry chamber experiments it has been shown that the maximum amount of air a subject can move in and out of the lungs in one minute is approximately halved (compared to the surface) at 100 ft (30 m, 4.0 ATA) (Camporesi and Bosco 2003).

This 'ceiling' on ventilation performance appears related to the physiological phenomenon known as 'dynamic airway compression', and it is explained as follows. During maximal breathing effort, the muscles of the chest wall and diaphragm create a positive pressure inside the chest in order to force gas out of the alveoli and outward through the airways as quickly as possible. However, as gas passes out along the airway, the pressure inside airway falls due to frictional forces of the gas on the airway walls. At some point during a forced exhalation this pressure drop inside the airway is sufficient that the raised pressure inside the chest exceeds the pressure in the airway, and the airway starts collapsing. This limits the outward gas flow through the airway, and this restriction on outward flow then becomes the limiting factor in how much gas can be moved in and out of the lungs each minute.

This actually occurs in air breathing at 1.0 ATA, but the limitation begins at such high flow rates that is does not significantly hamper work performance (except perhaps in extreme exercise). However, when breathing a dense gas underwater the resistance to flow is much higher and a significant pressure drop inside the airway as gas flows outwards occurs at much lower flow rates. Thus, the airway will begin to collapse at low flow rates, and this limits breathing to a much greater extent than seen during air breathing at 1.0 ATA. Indeed, it has been shown that if extremely dense gas is breathed, a diver might not be capable of moving much more gas in and out of their lungs than during normal breathing sitting at rest (Wood and Bryan 1969). Such situations would be unlikely to be encountered in properly planned dives, but it is possible (see below). A more detailed and illustrated explanation of this phenomenon can be found in the DAN Technical Diving Workshop Proceedings (Mitchell 2009).

Physiological Mechanisms of Hypercapnia in Diving

Having briefly considered the causes of increased work of breathing in diving, the discussion moves on to an explanation of how this increase in work may result in hypercapnia.

With reference to the earlier discussion of control of ventilation, it would be expected that if the P_aCO_2 began to rise (for example, when a diver starts to exercise and produces more CO_2), then the respiratory controller would automatically increase ventilation in order to remove more CO_2 and bring the P_aCO_2 back to normal levels. This is indeed the classically described ventilation response in experiments using very low resistance breathing equipment where the P_aCO_2 is forced to rise by introducing CO_2 to the inhaled gas so that no matter how much the subject breathes, they cannot return the CO_2 to normal. Under these circumstances, curves plotting end-tidal CO_2 (an indirect measurement of P_aCO_2) and ventilation typically show an approximately direct linear relationship.

However, there is some degree of inter-individual variability in the ventilation response to rising CO_2, and this can be markedly exaggerated if there is an unusual increase in the work required to increase ventilation (as is the case in diving). Under these circumstances, it is as though the respiratory controller is confronted with a choice: Either to perform the extra work required to maintain a normal P_aCO_2, or to avoid the extra work and allow the P_aCO_2 to rise. There appears to be a spread of individual responses between these two extremes. This is illustrated in Figure 2 which shows end-tidal CO_2 vs ventilation 'curves' for 15 subjects who were breathing on a rebreather circuit with no CO_2 scrubber in place (Deng et al. 2015). In this setting there was no removal of exhaled CO_2 and consequently there was substantial CO_2 rebreathing. The arterial CO_2 was forced to rise no matter how hard the subjects breathed. It is also contextually important that the diving rebreather used in this experiment imposed an increase in the work of breathing that was greater than normal. The Figure 2 'curves' are lines interpolated between points plotted from measurements of end-tidal CO_2 and ventilation made 30 s after starting to breathe and on termination of breathing on the circuit. All of these subjects voluntarily terminated breathing within a five-minute period citing 'shortness of breath' among their symptoms. The remarkable feature of the data is the variability in individual ventilation responses. Some subjects did not increase ventilation at all (indeed in some it actually decreased) whereas others exhibited a more classical linear increase in ventilation as the end-tidal CO_2 rose.

The implication of these data (and those of others that have examined the underlying mechanisms in more detail (Poon 1987; 1989)) is that in some divers at least, there is a tendency for the respiratory controller to prioritize the avoidance of respiratory work over maintaining the P_aCO_2 at normal levels when the work of breathing is increased. Put another way, during diving an increase in the work of breathing may provoke a 'naturally occurring flaw' in control of breathing such that the P_aCO_2 may rise simply because the diver does not breathe enough to eliminate the CO_2 that they are producing. This is the most plausible mechanism for the frequent finding of CO_2 retention in divers using underwater breathing apparatus (UBA), especially during exercise.

A second physiological mechanism for hypercapnia during diving relates to the potential for respiratory limitation by dynamic airway compression described above. It is plausible that if sufficiently dense gas was breathed a diver could find themselves in a situation where they would be unable to ventilate sufficiently to maintain a normal P_aCO_2 even at minimal levels of exercise, and even if they tried hard to do so. The principle of this mechanism is illustrated in Figure 3.

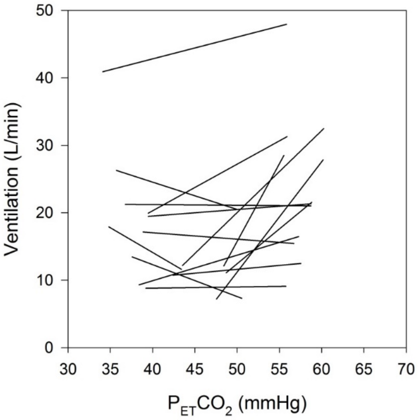

Figure 2. Indicative end-tidal CO_2 – ventilation curves for subjects breathing on a rebreather circuit with no CO_2 scrubber. Breathing was voluntarily terminated by the subjects when they developed symptoms of CO_2 toxicity (including a perception of shortness of breath in all cases). Each subject is represented by a straight line linking two paired measurements of end-tidal CO_2 and ventilation: the first made at 30 s after starting to breathe on the circuit, and the second on voluntary termination of the breathing period. $P_{ET}CO_2$ (end-tidal CO_2) is a conveniently measured approximation of the P_aCO_2. Reproduced with permission from Deng et al. (2015).

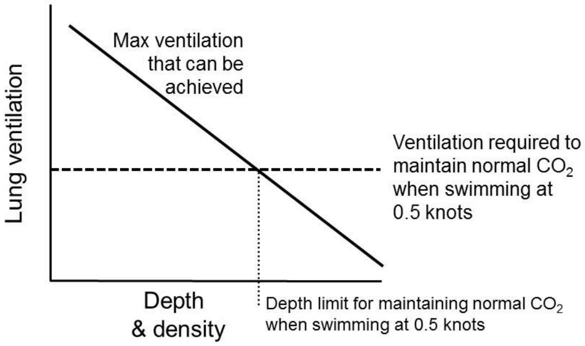

Figure 3. Notional depiction of the relationship between maximum possible ventilation and increasing depth and respired gas density. If the maximum possible ventilation falls below the ventilation required to eliminate the CO_2 produced (and therefore to maintain a normal P_aCO_2) at a given level of exercise, then the P_aCO_2 must inevitably increase. See text for further explanation.

In reference to Figure 3, the amount of ventilation (gas movement in and out of the lungs) required to keep the P_aCO_2 normal at a given level of exercise (nominally to swim at 0.5 knots) does not change as depth increases. However, as depth and the respired gas density increase, the maximum ventilation that can be achieved decreases because of the onset of dynamic airway compression at progressively lower flow rates through the airways. If the diver progresses deeper than a depth where they can produce the ventilation required to keep the P_aCO_2 normal, then the P_aCO_2 must inevitably rise. To make matters worse, the rising P_aCO_2 may trigger increased breathing effort which will only serve to produce more CO_2 because once dynamic airway compression occurs, no amount of extra effort will improve ventilation volumes. There is one published event in which there is reasonable supporting evidence for involvement of this mechanism, which occurred on a rebreather dive to a depth of 265 m (869 ft) (Mitchell et al. 2007).

For completeness, we observe that 'non-physiological' problems related to equipment (such as an absent, incorrectly installed or expired scrubber canister, or malfunctioning one way valves in the rebreather mouthpiece) are also potential causes of hypercapnia during rebreather diving. All of these result in some degree of CO_2 rebreathing, and if CO_2 is inhaled the diffusion gradient for elimination of CO_2 from venous blood to lung alveoli is diminished. If a large amount of CO_2 is rebreathed this can lead to a catastrophic impairment of CO_2 elimination with rapid development of symptoms of hypercapnia, but even a relatively small amount of inhaled CO_2 is potentially problematic because the associated impairment of CO_2 elimination will compound the physiological predispositions to hypercapnia described above.

Mitigation of the Risk of Hypercapnia During Rebreather Diving

At a practical level, the most important question arising from this discussion is 'what steps can be taken to mitigate the risk of hypercapnia during rebreather diving'? There are several possibilities.

Manipulation of static lung load

There is some evidence that a negative static lung load is the least desirable condition from a physiological perspective in rebreather diving, and that mildly positive static lung loads are best tolerated during hard work underwater (Thalmann et al. 1979). In a horizontal diver these conditions would be produced by back and front-mounted counterlungs respectively. However, choosing a counterlung configuration based primarily on concerns about static lung load may be ill-advised because the lung load will vary according to the diver's orientation in the water. For example, while a back-mounted counterlung would produce a negative static lung load in the horizontal position, it would be largely neutral in the upright position. In theory, over the shoulder counterlungs should produce the least extreme and least variable static lung load, but they have their own set of disadvantages such as cluttering the space around the diver's front and head.

Minimising equipment-related breathing resistance

All underwater breathing apparatus, including rebreathers, should be designed with the goal of reducing their external breathing resistance as much as is practicable. Other than choosing a device with good related design and testing characteristics there is little that divers can do in this regard. However, on a cautionary note, divers should take great care with making any modifications to a rebreather that might alter the geometry or resistance of the gas flow path. Common examples include departures from manufacturer-recommended grade of CO_2 absorbent material, the incorporation of extra oxygen cells for independent PO_2 monitoring, changing mouthpiece configuration, and changing the composition of any moisture pad material.

Consideration of gas density in diving planning

Most rebreather divers are very familiar with specialised dive planning strategies like calculating a maximum operating depth for a gas in order to avoid an unsafe inspired PO_2, or calculating an equivalent narcotic depth in planning the helium content of trimix to avoid unacceptable levels of nitrogen narcosis (Mitchell and Doolette 2013). In contrast, one almost universally overlooked dive planning strategy related to work of breathing is the use of gas density calculations to avoid breathing gases with unacceptably high density at depth. In no small part this situation prevails because there have been no definitive guidelines on acceptable gas density in diving.

There is a paucity of related data, though a recent analysis of a dataset of human testing records for UBA provides some potentially valuable insights upon which some preliminary guidelines can be based. Among other things, QinetiQ is a UBA testing house located near Portsmouth in the UK. Over some 20 years hundreds of manned test dives have been undertaken utilising ethics committee approved protocols which incorporate graded levels of underwater work for evaluating performance of a range open-circuit, semi-closed, and closed-circuit UBA. These dives have been conducted over depths ranging from 4 to 80 m (13 to 262 ft), using a range of gases including oxygen, air, nitrox and heliox. Throughout these tests a standard set of endpoints have been used to define 'dive failure' including: (any of) equipment or monitoring failure, diver unable or unwilling to continue because of dyspnoea (shortness of breath) or exhaustion, and an end-tidal CO_2 >8.5 kPa (64 mm Hg) over five consecutive breaths. The latter is indicative of significant CO_2 retention to a level associated with sudden incapacitation in the diving setting (Warkander et al. 1990).

Although this program of testing was not designed to specifically answer questions about tolerable gas density, the wide range of gas densities that were incidentally used has facilitated an evaluation of the proportion of work-loaded rebreather dive failures due to end-tidal CO_2 >8.5kPa stratified according to the gas density breathed. These data are reported in Figure 4. With the dual caveats that the trials were not specifically designed to answer this question and that the number of dives at the higher densities is comparatively small, there is a clear signal that near a respired gas density of 6.0 $g{\cdot}L^{-1}$ there is an upward inflection in the risk of dangerous CO_2 retention during working rebreather dives. A similar analysis of dive failures in open-circuit underwater breathing apparatus trials produced a virtually identical result.

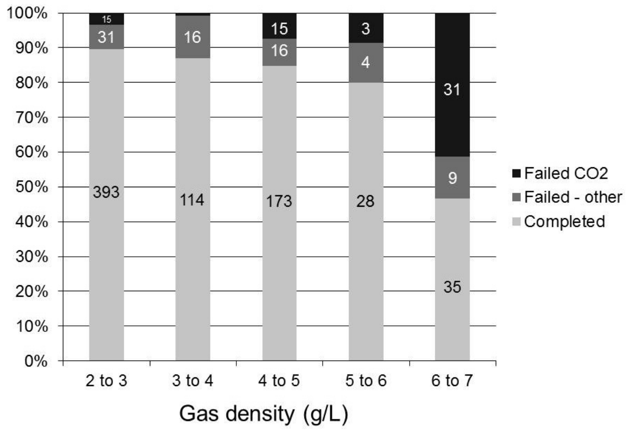

Figure 4. The proportion of rebreather test dives ending in failure due to an end-tidal CO_2 >8.5 kPa (black) and other causes of failure (dark grey) stratified by respired gas density. Figures refer to numbers of dives. At respired gas densities >6 $g{\cdot}L^{-1}$ there is a sharp increase in the risk of dive failure, with most failures being caused by dangerous levels of CO_2 retention.

For the purposes of planning rebreather dives and in the current absence of more definitive or contradictory data, it seems prudent to recommend an ideal maximum gas density of 5.2 $g{\cdot}L^{-1}$ (equivalent to air diving at 31 m [102 ft]) and an absolute maximum of 6.2 $g{\cdot}L^{-1}$ (equivalent to air diving at 39 m [128 ft]). Implementation of such a recommendation will require an appreciation of how to calculate gas density for a given respired gas at a given depth. Such calculations begin with knowledge of the density of air and the individual components of gas mixes at 1.0 ATA (Table 1).

Gas	Density ($g{\cdot}L^{-1}$)
Hydrogen	0.090
Helium	0.179
Nitrogen	1.251
Oxygen	1.428
Air	1.293

Table 1. Gas density in $g{\cdot}L^{-1}$ for common diluent gases, oxygen and air at 1.0 ATA. Data from Doolette and Mitchell (2011).

Calculation of the density of air at depth is a simple process of multiplying its density at 1.0 ATA by the ambient pressure at the target depth. For example, the density of air at 30 m (99 ft) is given by 1.293 $g \cdot L^{-1}$ x 4.0 ATA = 5.17 $g \cdot L^{-1}$.

Calculation of density for a mixed gas is achieved by using simple proportions to calculate the density of each component at 1.0 ATA, summing the components, and multiplying this sum by the ambient pressure in ATA at the target depth. For example, consider trimix 16:50 (16% oxygen, 50% helium, 34% nitrogen) intended for use at 70 m (230 ft) where the ambient pressure is 8.0 ATA. Calculating density for each component at 1.0 ATA we use the fraction of gas x its density at 1.0 ATA, thus, substituting in values from Table 1:

0.16 x density of oxygen (1.428) = 0.23 $g \cdot L^{-1}$
0.50 x density of helium (0.179) = 0.09 $g \cdot L^{-1}$
0.34 x density of nitrogen (1.251) = 0.43 $g \cdot L^{-1}$

The sum of the products of these calculations is 0.75 $g \cdot L^{-1}$ for density at 1.0 ATA. If this is then multiplied by 8.0 ATA for the ambient pressure at the planned depth we get 6.0 $g \cdot L^{-1}$. Therefore, in respect of gas density this would be an acceptable (but less than ideal) mix at this depth.

Moderating expectations of work capacity at depth

Unsurprisingly (given the above discussion) it is widely recognised among experienced divers that as depth increases there should be a corresponding moderation of expectation of work capacity. Hard work (with an inevitable increase in CO_2 production) is best avoided on a rebreather at any time, but this is particularly so at increased deep depths where the respired gas density is likely to be trending toward (or exceeding) the ideal limit. There are many practical strategies which help with reducing work at depth including exhibition of basic dive skills (such as maintenance of good buoyancy control and good trim/streamlining in the water), intelligent task planning, and the use of assistive technology such as diver propulsion vehicles. However, the use of such strategies is not a substitute for minimising the work of breathing in a UBA and strategic planning of gas density because events such as an emergency situation requiring extra work, or failure of a diver propulsion vehicle can occur unexpectedly.

Detection of CO_2 rebreathing

We earlier acknowledged the potential for CO_2 rebreathing to be caused by an absent, incorrectly installed or expired scrubber canister, or by malfunctioning one way valves in the rebreather mouthpiece. The strategies to prevent and detect such problems are issues of rebreather diving technology and practice rather than physiology. Nevertheless, for completeness, we will briefly discuss them there.

The cornerstone of preventing CO_2 rebreathing during use of a rebreather is meticulous adherence to manufacturer guidelines on both CO_2 absorbent duration and preparation of the rebreather before diving. Function of the mushroom valves in the rebreather mouthpiece should be checked every time the rebreather is assembled and the unit should not be used if the valves appear to be leaking. Great care must be taken with packing absorbent into the CO_2 scrubber canister to ensure that subsequent settling of the material does not result in a loose pack and channelling of gas through pathways of low material density. Similarly, the scrubber canister must be carefully installed in the rebreather avoiding any error that might result in gas bypassing the canister. Various rebreathers have easily avoidable but known vulnerabilities in this regard, and users must be aware of these.

As a final check of these good practices, rebreather divers are taught to conduct a five minute 'prebreathe' of the unit prior to entering the water. The prebreathe has multiple goals, but one of them (and the one upon which the five-minute duration is predicated) is the detection of symptoms of CO_2 toxicity should there be any error in preparation or assembly that allows rebreathing of CO_2. The efficacy of this strategy was recently tested in a randomised single blind study in which divers prebreathed a rebreather which either had a normal scrubber, a completely absent scrubber, or a partial failure of the scrubber allowing bypass of a significant amount of CO_2. The subjects were asked to terminate the prebreathe as they would in the real world if they developed symptoms of CO_2 toxicity. Twenty trials were undertaken in each condition. As expected, no diver terminated the prebreathe when breathing on a circuit with a normal scrubber. However, only 10% (2/20) were able to detect symptoms (and thus terminated) in the partial failure condition despite an inspired PCO_2 of 20 mm Hg. A much higher proportion (75%) detected the complete absence of the scrubber, but remarkably, 25% did not despite developing an end-tidal CO_2 greater than 60 mm Hg. Thus, it was concluded that while a prebreathe is a vital part of evaluating a rebreather before diving (for example, to check that the oxygen addition system is functioning), it cannot be relied upon to reveal problems with the CO_2 scrubbing function of the unit. Based on reports of a stressed or breathless appearance of some subjects who did not terminate during CO_2 rebreathing in the Deng et al. (2015) study, it could be concluded that peer observation of the prebreathe might improve its sensitivity. Whilst such a strategy might be applied successfully in a disciplined military or scientific diving setting, it is unlikely to be considered practical or executed diligently in 'mainstream' technical diving.

CO_2 sensors placed on the inhale limb of the rebreather circuit (downstream of the CO_2 scrubber) are a relatively recent innovation that represent a potential solution to the problem of detecting CO_2 bypassing the scrubber. These are only available on a small number of rebreathers at this time and there is limited experience with their use in the field. It is too early to make definitive recommendations on their use.

References

Anthony TG, Diving re-breathing apparatus testing and standards UK/EU perspective. In: Vann RD, Mitchell SJ, Denoble PJ, Anthony TG, eds. Technical Diving Conference Proceedings. Durham, NC: Divers Alert Network; 2009, pg. 218-36.

Camporesi EM, Bosco G. Ventilation, gas exchange, and exercise under pressure. In: Brubakk AO, Neuman TS, eds. Bennett and Elliott's Physiology and Medicine of Diving, 5th ed. Edinburgh, UK: Saunders, 2003: 77-114.

Deng C, Pollock NW, Gant N, Hannam JA, Dooley A, Mesley P, Mitchell SJ. The five minute prebreathe in evaluating carbon dioxide absorption in a closed-circuit rebreather: a randomised single-blind study. Diving Hyperbaric Med. 2015; 45: 16-24.

Doolette DJ, Mitchell SJ. Hyperbaric conditions. Comprehensive Physiol. 2011; 1: 163-201.

Mitchell SJ, Cronje F, Meintjes WAJ, Britz HC. Fatal respiratory failure during a technical rebreather dive at extreme pressure. Aviat Space Environ Med. 2007; 78: 81-6.

Mitchell SJ. Respiratory issues in technical diving. In Vann RD, Mitchell SJ, Denoble PJ, Anthony TG, eds. Technical Diving Conference Proceedings. Durham, NC: Divers Alert Network; 2009, pg. 12-37.

Mitchell SJ, Doolette DJ. Recreational technical diving part 1. An introduction to technical diving. Diving Hyperbaric Med. 2013; 43: 86-93.

Poon CS. Ventilatory control in hypercapnia and exercise: Optimization hypothesis. J Appl Physiol.1987; 62: 2447-59.

Poon CS. Effects of inspiratory resistive load on respiratory control in hypercapnia and exercise. J Appl Physiol. 1989; 66: 2391-99.

Thalmann ED, Sponholtz DK, Lundgren GEG. Effects of immersion and static lung loading on submerged exercise at depth. Undersea Biomed Res. 1979; 6: 259-90.

Warkander DE, Norfleet WT, Nagasawa GK, Lundgren CE. CO_2 retention with minimal symptoms but severe dysfunction during wet simulated dives to 6.8 atm abs. Undersea Biomed Res. 1990; 17: 515-23.

Wood LDH, Bryan AC. Effect of increased ambient pressure on flow volume curve of the lung. J Appl Physiol. 1969; 27: 4-8.

QUESTIONS AND DISCUSSION

AUDIENCE MEMBER: What is that depth again?

SIMON MITCHELL: It is not a depth. It is a density. So there are multiple ways you could achieve a particular gas density. You could have a light gas at a very deep depth or a heavier gas at a shallow depth. Irrespective of the depth the important thing is the gas density. Depth (or more correctly ambient pressure) is relevant in that it determines density for a given respired gas, but it is not relevant to these results.

JEFF BOZANIC: Did you measure PCO_2 during the tests for all your subjects?

SIMON MITCHELL: Yes.

JEFF BOZANIC: What was the highest level you were able to measure?

SIMON MITCHELL: The normal level of inhaled CO_2 is zero. It went up to over 50 mm Hg. By the end of a five-minute prebreathe with no scrubber our subjects had an end-tidal CO_2 of around 8.5 kPa, over 60 mm Hg, who had no idea (Deng et al. 2015). Unbelievable, is it not? Even with substantial ventilation increases a lot of these divers did not know. In terms of the ventilation increase, the tidal volume increased more than the rate.

DAVE CONLIN: Two questions. One, the separation between your counterlungs and your actual lungs, do you get to a point at depth where that difference in pressure because it is a small difference in the overall pressure, that your body is physiologically experiencing, is inconsequential? If you are at 33 ft (10 m), 25 cm is a lot different in pressure.

SIMON MITCHELL: I see, you are asking is there context with depth in determining a static lung load? No -- 20 cm of water is always 20 cm of water, no matter where you are in the water column.

DAVE CONLIN: My other question is if your body is a CO_2 retainer and your body does not care about the CO_2 it is retaining, so to speak, what is the problem?

SIMON MITCHELL: That is a good question. I did not think to mention it because high CO_2 becomes a problem in everyone once it reaches a certain point. The trouble is that CO_2 retainers do not see it coming. Dan Warkander and Barbara Shykoff at NEDU have done a lot of work on this. What they see with CO_2 retainers is that CO_2 creeps up during exercise at depth with an elevated breathing resistance. The divers are peddling away underwater indicating "I am fine." Then, all of a sudden, they either pass out or stop responding to hand signals. They pass a threshold and the lights go out. So, eventually it happens to