End Tidal CO_2 in Recreational Rebreather Divers on Surfacing After Decompression Dives

Simon J. Mitchell; Peter Mesley; Jacqueline A. Hannam

INTRODUCTION: Deep dives using rebreather devices result in oxygen exposures that carry a risk of cerebral oxygen toxicity. Elevation of arterial CO_2 levels increases this risk. CO_2 retention may occur during the deep working phases of dives, but it has not been investigated in 'real world' dives at the end of resting decompression when oxygen exposures are peaking, often to levels higher than recommended maxima.

METHODS: We conducted an observational field study to measure end tidal CO_2 ($P_{et}CO_2$) in divers surfacing after decompression. Sixteen rebreather divers conducted two dives and two completed one dive (a total of 34 dives) to depths ranging from 44–55 msw. Bottom times ranged from 35 to 56 min and time spent on decompression ranged from 40 to 92 min. The first breaths on reaching the surface after removing the rebreather mouthpiece were taken through a portable capnograph. The $P_{et}CO_2$ was recorded for the first breath that produced a clean capnography trace. $P_{et}CO_2$ measurement was repeated for each subject 2–3 h after diving to give paired observations.

RESULTS: There were no differences between mean surfacing $P_{et}CO_2$ [36.8 mmHg (SD 3.0)] and the mean $P_{et}CO_2$ made later after diving [36.9 mmHg (SD 4.0)]. One subject on one dive returned a surfacing $P_{et}CO_2$ higher than a nominal upper limit of 45 mmHg.

DISCUSSION: We found no general tendency to CO_2 retention during decompression. It is plausible that breaching oxygen exposure limits during resting decompression is less hazardous than equivalent breaches when exercising at deep depths.

KEYWORDS: rebreather dives, CO_2 retention, oxygen exposure limits, oxygen toxicity, diving physiology.

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Some recreational 'technical' divers use rebreather devices which recycle expired gas and thus extend gas supplies to undertake deep dives of long duration.[11] Rebreathers continuously optimize the inspired Po_2 to minimize tissue uptake of inert gases at depth and to accelerate gas elimination during ascent. This strategy helps manage the risk of decompression sickness, but optimizing the inspired Po_2 for decompression increases the risk of central nervous system (CNS) oxygen toxicity whose first manifestation can be loss of consciousness and a seizure. In theory, the risk of oxygen toxicity can be tracked and managed by comparing the duration of exposure to a particular inspired Po_2 to recommended limits.[12] It is common to express oxygen dose as a percentage of the recommended duration of exposure to the inspired Po_2[11] and divers often refer to this as the "CNS percent."

Many of the very deep and/or long dives being performed by rebreather divers mandate long decompressions that result in exposures well in excess of CNS 100%. Indeed, exposures exceeding 200 or even 300% are not uncommon, yet there are few reports of seizures during decompression. This has provoked a debate among divers about the validity of the recommended limits. Not surprisingly, diving physiologists are frequently asked about the significance of these oxygen exposures, and how extreme divers should manage them.

There are physiological grounds for hypothesizing that the oxygen exposure limits may be less applicable to a diver in a state of comparative rest during decompression than to a diver

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exercising at deep depths. In particular there is an extensive literature demonstrating that at least some divers retain carbon dioxide (CO_2) when exercising at depth.[6] This is significant because an elevated P_aCO_2 is a risk factor for CNS oxygen toxicity.[13] CO_2 retention (and the risk of CNS oxygen toxicity) should be less likely in a resting diver decompressing in shallow water, but to our knowledge this has never been investigated in real world dives conducted by rebreather divers. We therefore measured end tidal CO_2 ($P_{et}CO_2$) in rebreather divers immediately at the point of surfacing from decompression dives involving at least 30 min of decompression time. The null hypothesis was that there would be no evidence for CO_2 retention during decompression based either on comparison of the surfacing $P_{et}CO_2$ values to a nominal normal range (35–45 mmHg), or on comparison of those values to 'control' measurements made in the same divers during rest at the surface at least 2 h after the study dive.

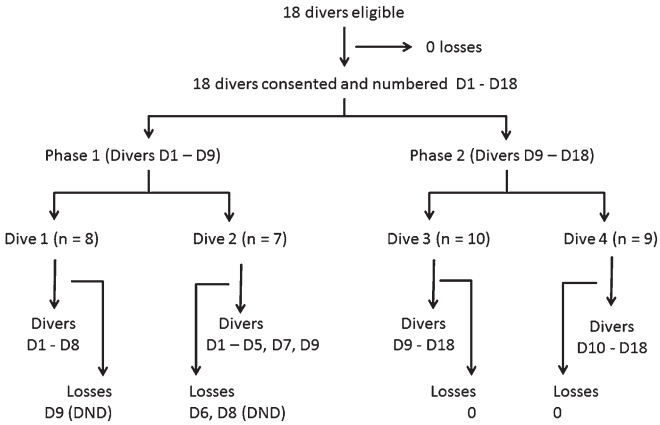

Fig. 1. Structure of the study. Divers 1–9 participated in the first phase of the expedition and divers 9–18 participated in the second phase. Thus, diver 9 was the only participant in both phases. This diver participated in one study dive in each phase. Two divers (D6, D8) did not dive (DND) on one dive and only completed one study dive. D9 did not dive on the first study dive, but was still able to complete a second dive on the second phase of the expedition. All data from all dives were included in the analysis.

METHODS

This was an observational field study that was reviewed and approved by the University of Auckland Human Participants Ethics Committee (reference 012/315). Subjects were 18 consenting rebreather divers on an expedition to Bikini Atoll to dive to the ships sunk during nuclear weapons testing in 1946. This was a sample of convenience, though it is relevant that all eligible divers on the expedition consented to participate. In the consent process we provided information about the study in such a manner that the divers remained naïve to the important physiology; in particular, we avoided disclosing details about the fundamental relationship between CO_2 elimination and pulmonary ventilation prior to conduct of the dives.

The expedition was split into two phases. There were 9 divers present on the first phase and 10 on the second (**Fig. 1**). One diver (designated D9 in Fig. 1) was present for both phases. Two dives were investigated in each phase and all divers present were invited to complete both dives with the exception of D9, who did not participate further after completion of two study dives (Fig. 1).

The dives were chosen based on two criteria. First, the target wreck had to have a permanent mooring line so the expedition vessel would be attached to the wreck itself. This meant descent to the wreck and ascent to the decompression trapeze could be made directly via the mooring line. Second, the weather had to be calm so that conditions for $P_{et}CO_2$ measurement at the boarding ladder were ideal. Divers were not compelled to participate on any dive and two chose not to complete one of the dives for reasons of fatigue or equipment issues (Fig. 1).

No attempt was made to influence the dive plan. The only requirement was that the dive involved at least 30 min of decompression, where decompression was defined as beginning when the diver left the wreck to ascend up the shot line. This duration was chosen, somewhat arbitrarily, as likely to give sufficient time for resolution of any derangement of CO_2 elimination occurring during the period of activity at depth. To be clear, our intent was to measure the typical CO_2 'status' of a diver on decompression; not during activity at the bottom.

All dives were on wrecks lying in a narrow depth range between 49 and 55 msw. All divers were using closed circuit rebreathers set to deliver a constant inspired Po_2 of 1.2 to 1.3 ATA during the bottom phase of the dive and throughout most of the decompression. Some divers reduced the Po_2 to 1.1 ATA at the shallowest decompression stop. The diluent gas was 'trimix' 15:30 (15% oxygen, 30% helium, and 55% nitrogen), giving an equivalent narcotic depth[11] of 30 msw at the deepest possible depth.

We did not attempt to influence the divers' choices in operation of their rebreathers. In this regard, it is notable that rebreathers recycle expired gas and remove the CO_2 in a 'scrubber' canister which contains absorbent material with a finite life. This 'life' may extend beyond the duration of one or two dives (depending primarily on their length), but if used for too long the scrubber will eventually fail to remove all expired CO_2. Carbon dioxide rebreathing could confound our results by encouraging CO_2 retention,[6] so we noted those instances where divers elected to begin a study dive with a partially consumed scrubber.

The parent ship was moored on the wreck and divers descended and ascended the mooring line at the beginning and end of each dive, respectively. A decompression trapeze was provided with bars set at the depths required for the shallowest and longest decompression stops. On completion of their final decompression stop (undertaken according to personal preference between 3 and 6 msw), the divers were instructed to ascend to the immediately adjacent exit ladder and to remove their mouthpiece immediately on reaching the surface. The final ascent typically took 30 to 60 s. One of the investigators waited at the ladder and, immediately the rebreather mouthpiece was removed, presented an EMMA™®

transport capnograph device (Masimo Sweden AB, Danderyd, Sweden) attached to a flexible catheter mount with an internal volume of 25 ml. The divers were instructed to seal their lips around the catheter mount and take 2–4 normal breaths 'in and out' through the device. The end tidal CO_2 was recorded from the first breath which produced a clean capnograph trace of normal morphology, which invariably occurred on the second or third breath. Control readings were obtained between 2–3 h after diving during rest.

We compared the mean $P_{et}CO_2$ readings immediately after diving with those measured 2–3 h later at rest. Since the prevailing conditions on a particular dive could conceivably influence any tendency to retain CO_2, each dive was analyzed as a separate experiment, but the data for all dives were also pooled for analysis. We reanalyzed the pooled data stratified according to whether the diver began the dive with a fresh or partly used CO_2 scrubber. A paired two tailed *t*-test was used to compare postdive and control $P_{et}CO_2$ readings for each dive and for the pooled analyses. A *p*-value of 0.05 or less was considered to indicate statistical significance.

RESULTS

The subject group was comprised of 17 men and 1 woman with an average age of 44.6 (8.7 SD). Subjects had been diving for a median 16.5 yr (7–43 range) and had completed a median 1300 dives (250–5000+ range). This was a cohort of experienced divers. Descriptive data on the four dives are provided in **Table I**. All dives easily exceeded the minimum 30-min decompression duration threshold (range 40–92 min). Dive conditions were relatively consistent, though participants in the second phase of the expedition (when dives 3 and 4 were performed) tended to undertake longer dives with longer decompressions (Table I).

There were no differences between the means of $P_{et}CO_2$ readings recorded immediately on surfacing and 'control' values measured 2–3 h later (**Table II**). The same applied when data for all dives were pooled (also shown in Table II). Similarly, when the pooled data were stratified by dives commenced with fresh CO_2 scrubber material ($N = 22$) versus partially used CO_2 scrubbers ($N = 12$), there was no difference in the mean $P_{et}CO_2$ measured immediately on surfacing (mean $P_{et}CO_2$ of 36.8 mmHg in both conditions). On dive 4, one individual surfaced with an $P_{et}CO_2$ of 48 mmHg, which is outside a nominal normal range of 35–45mmHg. This individual's control $P_{et}CO_2$ after that dive was 39 mmHg.

DISCUSSION

We found no general tendency to CO_2 retention among rebreather divers surfacing from dives involving periods of decompression ranging between 40 to 92 min. This finding is consistent with prior knowledge of CO_2 regulation. CO_2 retention has been demonstrated in exercising divers at moderate depths where work of breathing may be increased by elevated gas density, static lung loads, and external resistance of the underwater breathing apparatus. The relevant mechanisms have been discussed in detail elsewhere.[6,10] In contrast, CO_2 retention should be less likely in the conditions investigated here: long decompressions at shallow depths where divers are at rest and respired gas density is only marginally elevated. In the absence of respiratory impediments healthy subjects will usually not retain CO_2, even during exercise.[4]

While our results are, therefore, not surprising, this was (and remains) an important question. One of the most feared complications of dives involving prolonged exposure to high inspired pressures of oxygen is cerebral oxygen toxicity. This may manifest without warning as a seizure, which frequently leads to drowning. Simple calculations of cumulative oxygen dose based on duration and inspired Po_2 imply that the risk of cerebral oxygen toxicity progressively increases throughout a dive and peaks during decompression.[11] This causes considerable anxiety among rebreather divers, who often perform dives in which recommended exposure limits are exceeded, sometimes markedly. Indeed, to achieve their goals, some extreme

Table I. Characteristics of the Four Study Dives.

	DIVE 1 ($N = 8$)	DIVE 2 ($N = 7$)	DIVE 3 ($N = 10$)	DIVE 4 ($N = 9$)
Depth (msw)				
Mean (SD)	53.5 (1.2)	45.0 (1.3)	50.6 (1.0)	53.7 (1.0)
Range	52 - 55	44 - 47	49 - 52	52 - 55
Bottom time (min)				
Mean (SD)	40.8 (1.0)	39.7 (3.4)	51.9 (2.7)	50.8 (4.0)
Range	40 - 43	35 - 43	47 - 55	46 - 56
Decompression time (min)				
Mean (SD)	57.5 (3.2)	49.6 (5.1)	73.8 (9.9)	74.2 (13.7)
Range	56 - 62	40 - 55	65 - 88	55 - 92
Total dive time (min)				
Mean (SD)	98.1 (3.4)	89.3 (4.2)	125.7 (10.8)	126.1 (16.8)
Range	92 - 102	83 - 96	114 - 140	108 - 147
Water temperature (°C)	30	30	30	30
Current	nil	nil	nil	nil

Bottom time is defined as the time from beginning of descent to the beginning of ascent up the mooring line. Decompression time is defined as the time from beginning of ascent up the mooring line to arrival at the surface (which includes the time spent on decompression stops). Total dive time is the sum of the bottom time and decompression time.

Table II. Mean $P_{et}CO_2$ on Surfacing and at the Control Measurement After Diving, Reported by Dive and for the Pooled Data.

	$P_{et}CO_2$ ON SURFACING (mmHg)	$P_{et}CO_2$ CONTROL (mmHg)	*p*
	MEAN (SD)	MEAN (SD)	
Dive 1 ($N = 8$)	37.5 (3.7)	37.7 (3.5)	0.86
Dive 2 ($N = 7$)	37.6 (5.3)	36.8 (3.0)	0.61
Dive 3 ($N = 10$)	34.7 (1.4)	36.3 (2.5)	0.08
Dive 4 ($N = 9$)	38.1 (4.8)	37.1 (3.1)	0.56
All dives ($N = 34$)	36.8 (4.0)	36.9 (3.0)	0.86

divers have little choice but to essentially ignore such limits. It is widely believed (or at least assumed) that the recommended exposure limits are less applicable to the comparatively restful and shallower decompression phase of a dive than to the active bottom phase at greater depth.

Arterial CO_2 is an important factor in such discussions because an elevated P_aCO_2 is considered a significant risk factor for cerebral oxygen toxicity. Hard human evidence of this is "sketchy,"[13] but the animal data are virtually irrefutable.[1–3] The relevance to humans has been inferred largely from a recognized increase in risk of oxygen toxicity when exercising at depth,[5] and the marked increase in oxygen delivery to the brain which occurs as the P_aCO_2 rises.[9] It follows that the question of whether CO_2 retention occurs during shallow decompression at rest when cumulative oxygen exposures are peaking is important. As discussed above, CO_2 retention may occur when the work of breathing increases and a diver using underwater breathing apparatus even at shallow depths cannot be considered unencumbered from a respiratory perspective. Irrespective of depth, the use of a rebreather imposes static lung loads and an external resistance; all gas flow around the breathing circuit must be generated by the diver's effort. To our knowledge, no one has previously investigated the potential for this milieu to provoke CO_2 retention in the shallow decompression phase of real-world rebreather dives.

This was an observational field study and we made no attempt to influence individuals' diving methods. As previously mentioned, one relevant factor was the divers' use of CO_2 scrubbers that were already partly consumed from use on a prior dive(s). Had we found an apparent tendency to CO_2 retention, one potential confounder would have been failure of the CO_2 scrubber to remove all CO_2 from the inspired gas, resulting in CO_2 rebreathing. For this reason we noted the CO_2 scrubber durations accumulated on previous dives before each study dive because significant previous use would indicate a higher risk of failure. These durations varied between 0–3 h of use, but the numbers can only be meaningfully interpreted in the context of the actual model of rebreather used and its respective scrubber canister size and recommended duration. Since we did not detect any generalized tendency to CO_2 retention, it seems unlikely that scrubber failure was occurring. Nevertheless, for completeness, we reanalyzed the pooled surfacing $P_{et}CO_2$ values stratified according to whether the scrubber material was new at the start of the dive, or whether it had been used on previous dives. The surfacing levels were identical in these groups, further suggesting that scrubber failure did not influence our results. It is notable that the one diver who returned a high $P_{et}CO_2$ after one dive began that dive with a fresh scrubber.

We did not find a general tendency to retain CO_2 during shallow decompression while using a rebreather. Our subjects surfaced with $P_{et}CO_2$ levels that did not reflect CO_2 retention either in terms of the absolute values, or when compared with resting levels measured 2–3 h after diving. However, these results must be interpreted cautiously and some limitations of our study must be acknowledged.

First, despite widespread interest in the problem, there is currently no validated method of directly recording $P_{et}CO_2$ during rebreather dives in the field.[8] We were therefore forced to use end tidal measurements made on the first breaths at the point of surfacing as a surrogate for the CO_2 levels on decompression. This method has been used by one of the authors to demonstrate CO_2 retention in an informal test on a susceptible individual after underwater swimming activity using a rebreather in a pool. In the present study the decompression trapeze was immediately adjacent to the exit ladder, but we cannot exclude the possibility that $P_{et}CO_2$ may have changed in the very brief ascent from trapeze to stairs. It is notable, however, that the most likely precipitant for CO_2 retention during resting decompression (increased work of breathing due to the external resistance of the rebreather and any associated static lung load) would not have materially changed during the ascent. Similarly, the inspired Po_2 (another potential precipitant) would have fallen only minimally from the chosen Po_2 set point of 1.1–1.3 ATA to close to 1 ATA on arrival at the surface. It follows that conditions which potentially precipitate CO_2 retention did not substantially change during the brief ascent.

Second, this is a small study. We doubt that a larger study would draw a different conclusion, but we cannot exclude the possibility. Nor have we excluded sporadic CO_2 retention on decompression by predisposed individuals. Indeed, one diver returned a marginally high postdive reading on one occasion, but our study was not large enough to calculate a valid incidence of such events with any degree of confidence.

Third, these dives took place in relatively benign conditions (warm water, no current) and the results cannot be confidently extrapolated across all decompression scenarios, particularly those involving stress and hard work (though such circumstances are rare). Similarly, the dives were not as long as those where the oxygen exposure exceeds the recommended limits. The highest exposure accumulated in this study was approximately 80% of the recommended maximum. We doubt that a tendency to CO_2 retention unapparent on decompressions lasting up to 90 min would be unmasked by longer durations, but we cannot exclude the possibility.

Finally, it is possible that the normally small difference between $P_{et}CO_2$ and P_aCO_2 in healthy subjects may be exaggerated during 100% oxygen breathing due to an increase in physiological dead space.[14] The mechanism may be hyperoxia-induced pulmonary vasodilation, which redistributes blood flow from

well-ventilated to less well-ventilated lung units. This possible measurement error was recently cited by Gill et al.[7] as a potential explanation for an apparent change in the slope of $P_{et}CO_2$–V_E curves for subjects breathing an inspired PO_2 of 0.21 versus 1.3 ATA. Our surfacing $P_{et}CO_2$ readings may, therefore, underestimate the P_aCO_2 by more than the usual small amount, but this is an unconfirmed hypothesis which (as with the proposal by Gill et al.[7]) would require a study involving simultaneous arterial blood gas sampling and end tidal CO_2 measurement to resolve.

In mitigation of these limitations, we believe the real-world nature of the dives increases their interest value and relevance, and is a strength. We also highlight the fact that, for an unregulated field study, there was a significant degree of standardization of dive parameters, particularly among subjects on each dive (Table I). Finally, the confluence of the results with outcomes predicted on the basis of known physiological principles increases their face validity.

In conclusion, whereas CO_2 retention is relatively common during exercise at deep depths,[6] the vast majority of our subjects resting at a shallow decompression stop did not appear to retain CO_2. It therefore seems plausible that a breach of oxygen exposure limits during resting decompression at shallow depth is less hazardous than the same breach during periods of exercise at deep depths. Our methods do not allow a definitive conclusion to be drawn on this matter. The development of a validated end tidal CO_2 monitor that can be used on rebreathers during diving would allow a more definitive resolution of this issue, which deserves further study.

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Simon J. Mitchell conceived the study and wrote the study protocol. Jacqueline A. Hannam contributed to the protocol, wrote the ethics application, and sponsored the ethics process. Peter Mesley organized the expedition. Mitchell and Mesley collected the data. All authors contributed to the data analysis and writing of the paper.

The authors have no conflicts of interest to declare.

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