Analyser position for end-tidal carbon dioxide monitoring in a rebreather circuit

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Key words
Carbon dioxide, hypercapnia, rebreathing, rebreathers/semi-closed circuit, rebreathers/closed circuit, physiology, simulation

Abstract
(Ineson A, Henderson K, Teubner D, Mitchell S. Analyser position for end-tidal carbon dioxide monitoring in a rebreather circuit. *Diving and Hyperbaric Medicine*. 2010;40(4):206-9.)
Introduction: A diving rebreather currently nearing release incorporates an infra-red CO_2 analyser at the end of the exhale hose and uses the expired gas CO_2 measurement made at this position to detect hypercapnia. This configuration may allow exhaled anatomic and mouthpiece dead space gas to mix with alveolar gas in the exhale hose thus falsely lowering the CO_2 measurement, especially at low tidal volumes.
Methods: A test circuit was constructed using a typical rebreather mouthpiece and exhale hose connected into an anaesthetic machine breathing loop. True end-tidal PCO_2 was measured in gas sampled from the mouth and compared breath-by-breath to the PCO_2 measured in gas sampled at the end of the exhale hose. Two subjects each completed 60 breaths at tidal volumes of 500, 750, 1000, 1500 and 2000 ml.
Results: There was a small (≤ 0.21 kPa) mean difference between true end-tidal CO_2 and end-of-hose CO_2 at tidal volumes of 1000 ml or more. However, at lower tidal volumes, the mean difference increased and, at 500 ml, it was 1.04 kPa and 0.70 kPa in subjects 1 and 2 respectively.
Conclusion: Measurement of the peak exhaled PCO_2 at the end of a rebreather exhale hose may provide a reasonable estimation of the true end-tidal CO_2 at large tidal volumes, but may significantly underestimate the true end-tidal CO_2 at low tidal volumes.

Introduction

In a new recreational diving rebreather, an infrared carbon dioxide (CO_2) analyser has been placed at the distal end of the exhale hose to measure “end-of-breath CO_2” and to warn of hypercapnia. We undertook this simple study specifically to investigate the potential for exhaled anatomic dead space gas to mix with alveolar gas during passage along an exhale hose, thus diluting the CO_2 measured at the analyser.

Methods

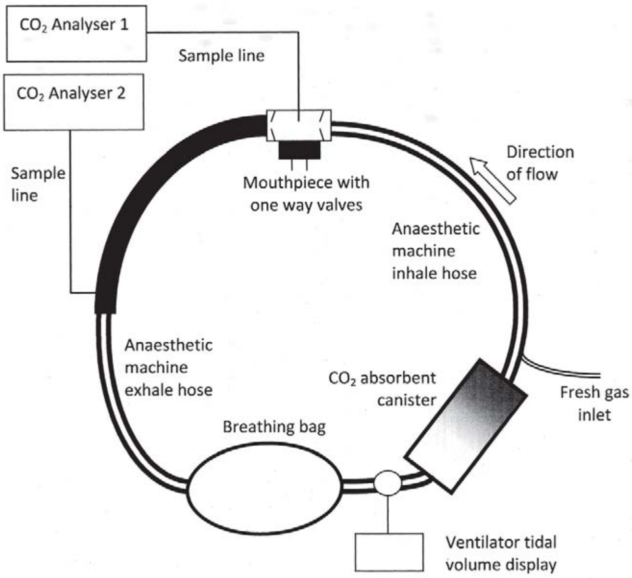
This study was approved by the Northern X Regional Ethics Committee of the New Zealand Ministry of Health. A test circuit (Figure 1) was constructed using a rebreather mouthpiece (Jetsam Technologies, Port Moody, Canada) with an internal volume of 40 ml, connected to a rebreather exhale hose (Carleton Technologies, New York, USA) with an internal volume of 340 ml. The inhale port of the mouthpiece and the end of the rebreather exhale hose were connected to the inhale and exhale hoses (respectively) on an Aestiva 7900 anaesthetic machine (GE Healthcare, Madison WI, USA) to establish a breathing circuit in which exhaled CO_2 was removed by an absorbent canister and tidal volume was measured by the circuit ventilator.

Gas sampling lines were plumbed into the rebreather mouthpiece to measure the ‘true’ end-tidal CO_2, and into the end of the rebreather exhale hose to approximate the CO_2 analyser position on the new rebreather (Figure 2). These lines were connected to identical infra-red CO_2 analysers (Datex-Ohmeda, Helsinki, Finland) on separate Aestiva anaesthetic machines. The analysers continuously draw gas through the sampling lines (200 ml min^{-1}) with a 2.5 s sampling delay and <400 ms delay in measurement, accurate to ± 0.2 vol%. The analysers are programmed to automatically zero every 60 minutes. Fresh gas flow into the circuit was nitrox 50 at 1 L min^{-1}. The anaesthetic machines and analysers were set up and checked by a qualified anaesthetic technician.

A resting subject breathed on the circuit with the nose pinched shut, using the digital readout on the ventilator to volitionally control tidal volume. Breath-by-breath peak CO_2 measurements were recorded simultaneously from the two positions for tidal volumes 500 ml, 750 ml, 1000 ml, 1500 ml and 2000 ml. Breaths were ‘accepted’ for each of these tidal volumes if they were between 450–550, 700–800, 900–1100, 1400–1600 and 1900–2100 ml respectively. Recordings were undertaken for each tidal volume in three groups of 20 breaths, giving 60 breaths per tidal volume per subject. The breathing rate was determined by subject comfort and no attempt was made to influence this. Two subjects completed this protocol on separate days.

The mean (± standard deviation) differences between ‘true’ end-tidal PCO_2 and the ‘end-of-hose’ PCO_2 were calculated for each tidal volume and each subject. Between-

Figure 1
Stylised layout of the experimental breathing circuit and monitoring devices



subject comparisons of these mean differences were made using a Student t-test (STATA 10SE, Statacorp, Texas). A *P*-value of less than 0.05 was taken to indicate statistical significance.

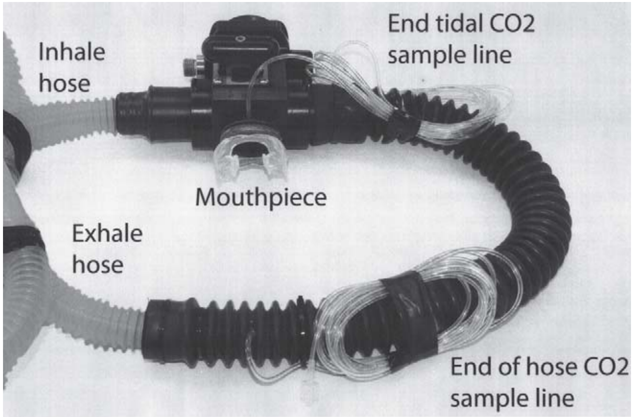
Results

Subject characteristics are reported in Table 1. The mean differences between true end-tidal PCO_2 and the PCO_2 measured at the 'end-of-hose' position were inversely related to tidal volume (Table 2), though at tidal volumes 1000 ml and above they were small and relatively constant. There were subtle but significant inter-subject differences at all tidal volumes except 750 ml. The subjects are reported separately.

Discussion

Hypercapnia is an important hazard in diving. A predisposition to CO_2 retention in divers has been documented in multiple studies which were reviewed recently.[1] This may result from perturbation of the respiratory response to rising arterial PCO_2 under conditions of increased work of breathing caused by breathing dense gas or the use of scuba equipment that imposes high breathing resistance or significant static lung loads. The resulting tendency to relative hypoventilation can lead to hypercapnia. Divers using rebreathers, in which gas is recycled around a closed-circle circuit, face the unique risks of failure in the valves which ensure unidirectional flow and of CO_2 absorbent failure. The CO_2 absorbent has a finite capacity which may become exhausted. If the absorbent canister fails, then the diver will rebreathe CO_2 and may become hypercapnic. Although there are no reliable data to describe the relative importance of hypercapnia as a disabling agent in accidents, there is abundant anecdote describing 'near misses', and there are examples of carefully investigated injuries or deaths that were strongly linked to hypercapnia.[2,3]

Figure 2
Rebreather mouthpiece and exhale hose showing sampling line positions for end-tidal and end-of-hose CO_2 sampling and connections to anaesthetic circuit



Until recently, attempts to detect impending hypercapnia were limited to the use of a temperature measurement device in the absorbent canister to monitor progress of the exothermic CO_2 absorbent reaction through the canister. These devices, colloquially referred to as 'temp sticks', are now deployed in a number of rebreathers and their efficacy in predicting scrubber exhaustion and CO_2 break-though has been reported.[4] However, although a temp stick may predict absorbent failure, it will not warn a diver that they are becoming hypercapnic. This would require direct measurement of CO_2 in the expired gas in a manner analogous to that used in anaesthesia.

Devices to measure PCO_2 in a mix of gases by using its unique absorbance of infra-red light are incorporated into anaesthetic monitoring systems. The measurement can be made directly by an analyser device placed on the breathing circuit at the entrance to the airway, or by sampling gas from

Table 1
Subject characteristics
(% of predicted forced vital capacity in brackets)

	Subject 1	Subject 2
Gender	Male	Female
Age (yr)	52	48
Height (cm)	186	164
Weight (kg)	87	66
Body mass index	25.1	24.5
Forced vital capacity (L)	5.97 (107%)	3.9 (107%)

Table 2
Mean (sd) 'true' end-tidal CO_2, 'end-of-hose' CO_2, the difference between them (kPa) and the 95% CI for the difference for the two subjects over the range of tidal volumes; *n* per subject for each tidal volume = 60; means rounded to one decimal place

Tidal volume (ml)	500	750	1000	1500	2000
Subject 1					
True end-tidal CO_2	6.0 (0.23)	6.0 (0.16)	6.1 (0.23)	5.8 (0.27)	5.6 (0.21)
End-of-hose CO_2	5.0 (0.51)	5.7 (0.20)	5.9 (0.23)	5.6 (0.23)	5.4 (0.07)
Difference	1.0 (0.53)	0.3 (0.08)	0.2 (0.08)	0.2 (0.07)	0.2 (0.07)
95% CI	0.90–1.18	0.28–0.34	0.19–0.23	0.18–0.22	0.19–0.23
Subject 2					
True end-tidal CO_2	4.8 (0.07)	4.5 (0.10)	4.8 (0.26)	3.7 (0.037)	3.6 (0.37)
End-of-hose CO_2	4.1 (0.12)	4.2 (0.11)	4.6 (0.26)	3.5 (0.36)	3.4 (0.32)
Difference	0.7 (0.10)	0.3 (0.07)	0.2 (0.06)	0.1 (0.05)	0.2 (0.10)
95% CI	0.67–0.73	0.28–0.32	0.16–0.20	0.11–0.13	0.61–0.19

the same point and carrying it back to an analyser remote from the circuit. Either strategy provides a continuous measurement of the PCO_2 in gas entering and leaving the airway.

Selecting a sampling point at the entrance to the airway imparts several crucial advantages. First, inspired gas can be analysed to ensure it is free of CO_2, thus warning of any CO_2 rebreathing. Second, the anatomical dead space gas will pass distally in the circuit early during exhalation, and the end-tidal gas can therefore be assumed to represent mixed alveolar gas. Since alveolar gas is in equilibrium with dissolved arterial gases, the PCO_2 measured in the end-tidal gas can be used as an approximation of arterial PCO_2. In fact, the end-tidal CO_2 usually underestimates the true arterial PCO_2 by a small amount due to a contribution to mixed alveolar gas by well-ventilated, poorly perfused alveolar units that contain little CO_2, but in healthy individuals the difference is small enough that it is typically ignored for most purposes.

Infra-red CO_2 detection devices are now sufficiently small, moisture-resistant, and economical of power to allow deployment in rebreather circuits. For the reasons described above, such a device would ideally be deployed at the entrance to the airway (in the rebreather mouthpiece) to allow measurement of both the inspired CO_2 and the end-tidal CO_2. Unfortunately with current devices, this would render the mouthpiece too bulky. Moreover, drawing a sample from the mouthpiece to a remote analyser would require a pump with high power consumption.

Placement of the analyser at the distal end of the exhalation hose avoids these problems, but may allow mixing of anatomical and mouthpiece dead-space gas with the alveolar gas within the exhale hose prior to measurement of the 'end-of-hose' PCO_2. Since the total dead-space volume is approximately 200 ml (150 ml anatomical dead space and 50 ml mouthpiece dead-space), this would have little impact on the measured end-of-hose PCO_2 after a large tidal volume exhalation. However, at smaller tidal volumes the dilution effect might become significant.

Our study confirmed these suspicions. There was a small and physiologically insignificant difference between the true end-tidal CO_2 and the end-of-hose CO_2 during respiration at larger tidal volumes (≥ 1000 ml), but this difference became larger at lower tidal volumes; averaging 1 kPa (7.5 mmHg) in one subject and 0.7 kPa (5.3 mmHg) in the other at 500 ml tidal volume. This between-subject difference may be accounted for by a larger dead space volume in the larger, male subject. Although a relatively small difference in gas partial pressure, 1 kPa (7.5 mmHg) is potentially a physiologically significant difference in arterial PCO_2. If a warning device underestimated the end-tidal CO_2 by 1 kPa (7.5 mmHg) during an episode of hypercapnia, then this could delay an appropriate response and make incapacitation more likely.

Interpretation of this finding is difficult because little is known about tidal volumes in typical diving situations. A number of studies of exercise in controlled underwater laboratories have reported tidal volumes as part of their datasets.[5–7] These data suggested that tidal volumes less than 1000 ml are not often encountered under the conditions imposed by the various experiments, at rest or during exercise.

Taken together with our findings, this suggests that an end-of-hose CO_2 measurement may be an adequate indicator of hypercapnia in the majority of circumstances. Nevertheless, an event has been reported in which an exercising, immersed diver was subjected to an increase in breathing resistance and responded by increasing respiratory rate and reducing tidal volumes (to approximately 600 ml).[8] The diver appeared completely unaware of a consequent rapid rise in the end-tidal CO_2 and became incapacitated almost without warning. A similar event was described in a second diver but respiratory volumes were not reported. It is in this type of 'low tidal volume, progressive hypercapnia' scenario that inaccuracy in end-of-hose CO_2 measurement might be important. It is plausible that such scenarios are more common than we realise as part of the fatal pathway in hypercapnic events during diving.

It must be acknowledged that this simple study has several limitations and must be interpreted cautiously. Firstly, we did not utilise the mouthpiece and exhale hose from the new rebreather in which end-of-hose CO_2 monitoring will occur. Differences in volume and geometry may result in less mixing in this device's exhale hose, but it is also possible there may actually be more mixing, especially since it appears to have a greater diameter than the components used in this study. Secondly, the study was conducted with normocapnic subjects and we did not replicate the hypercapnic conditions that the end-of-hose analyser is in place to detect. We believe our method was adequate for the purpose of demonstrating a difference between true end-tidal CO_2 and end-of-hose CO_2, but we do acknowledge that no hypercapnic test was performed. Thirdly the study was not performed under real diving conditions. Diving may increase anatomical dead space if tidal breathing is conducted at higher lung volumes, a ventilation pattern which is known to be adopted during respiration with dense gas.[9,10] If this occurred, the dilution effect may be greater than we have demonstrated. Finally only two subjects were studied. Nevertheless, all humans have anatomical dead space and all rebreathers have mouthpiece dead space. Our findings are thus predictable from physiological first principles. Studying a larger number of subjects possessing a wider range of physical characteristics might further define the magnitude of effect but it would be very unlikely to change our fundamental conclusion.

Conclusions

Our results confirm that dilution of alveolar gas by dead-space gas does occur in the exhale hose of a rebreather during low tidal volume breathing. In respect of estimating end-tidal CO_2 by analysing gas at the end of the hose, this dilution is likely to be insignificant during breathing at tidal volumes that are thought to be typical of many diving scenarios. Nevertheless, it is possible that significant under-estimation of end-tidal CO_2 could occur when CO_2 measurements are made at the end-of-hose position during low tidal volume breathing unless some form of compensation is built into the interpretation algorithm.

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