Original articles

Effect of a prior hypercapnia experience on recognition of hypercapnia in divers: a randomised controlled study

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Abstract
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Introduction: Rebreather diving carries an increased risk of hypercapnia. Hypercapnia can cause impaired cognition, breathlessness, and increase the risk of oxygen toxicity. We investigated whether a prior unblinded hypercapnia experience, compared to reading about hypercapnia symptoms, would improve divers' ability to recognise hypercapnia and initiate self-rescue.
Methods: Forty divers were recruited and randomised to receive either an unblinded hypercapnia experience (partial pressure of end-tidal carbon dioxide [$P_{ET}CO_2$] of 8.5 kPa) or an information leaflet explaining hypercapnia symptoms. At least one month later, participants in each group were further randomised to undergo blinded exposure to hypercapnia or normocapnia, allocated at 3:1. The primary outcome was the proportion of participants who self-initiated bailout prior to reaching $P_{ET}CO_2$ 8.5 kPa. Continuous cardiorespiratory data ($P_{ET}CO_2$ and $P_{ET}O_2$, tidal volume, respiratory rate, minute ventilation, heart rate, and blood pressure) were also recorded. Subjective symptoms associated with hypercapnia were assessed with a visual analogue scale.
Results: Thirteen of 15 participants (87%) who received the unblinded hypercapnia-experience self-initiated bailout compared to 10/15 information leaflet participants (67%) ($P = 0.149$). There was no difference in cardiorespiratory physiology parameters at bailout between the groups. Shortness of breath, light-headedness, and disorientation were the most intensely reported symptoms. Approximately half (47%) of participants who received a hypercapnia training experience had a correlated symptom response during their subsequent hypercapnia testing session.
Conclusions: Although no significant training benefit was shown, becoming familiar with the sensations associated with hypercapnia under appropriate supervision could be useful to rebreather divers both recreationally and within occupational settings.

Introduction

Rebreather diving, where gas is recirculated with carbon dioxide (CO_2) removed and oxygen (O_2) added to maintain a constant partial pressure of O_2 'setpoint', is common in recreational and occupational settings (e.g., military divers). Advantages include reduced gas usage, especially at greater depths, prolonged dive time, and staying undetected.[1] However, rebreather faults or user error can result in CO_2 rebreathing and hypercapnia.[2] Hypercapnia may also be provoked in diving by perturbation of respiratory control (reduced ventilatory responsiveness to rising CO_2 levels) caused by a combination of increased work of breathing and exercise.[3] Moreover, there is considerable inter-individual variability in this tendency, with some individuals more prone to allowing CO_2 levels to rise without increasing ventilation; often deemed 'CO_2 retainers'.[4]

Symptoms of hypercapnia include impaired cognitive function[5] and breathlessness.[6] Moreover, an elevated partial pressure of arterial CO_2 ($PaCO_2$) can also increase the risk of oxygen toxicity via cerebral vasodilation and consequent enhanced oxygen delivery to the brain,[7] potentially causing seizures and death.[8] The narcotic effects of CO_2 can occur independently of nitrogen narcosis or in an additive or synergistic manner.[9] It can be difficult to differentiate the symptoms of hypercapnia from normal dive phenomena, e.g., increased breathing resistance or breathlessness on exertion.[10,11] Hence, symptoms alone are typically not reliable for detecting hypercapnia.[12]

Despite the well-established dangers of a hypercapnic episode at depth, there continues to be a technical difficulty in developing a reliable method of detecting causative hazards or hypercapnia itself while diving.[13] So-called temperature sticks in rebreather scrubbers only detect a potential failure of the scrubber.[14] Similarly, inhaled CO_2 monitors can only detect the presence of CO_2 in the inspired gas.[15] Neither technology can detect hypercapnia arising from dysregulation of respiratory control. Underwater capnography has not yet been developed,[13] and is challenged by confounding of near-infrared CO_2 sensors in the 100% humidity environment of a rebreather loop.[16]

The aim of this study was to investigate whether an open-label (unblinded) experience of hypercapnia symptoms would improve divers' recognition of hypercapnia and ability to perform a self-rescue in a subsequent blinded hypercapnia exposure.

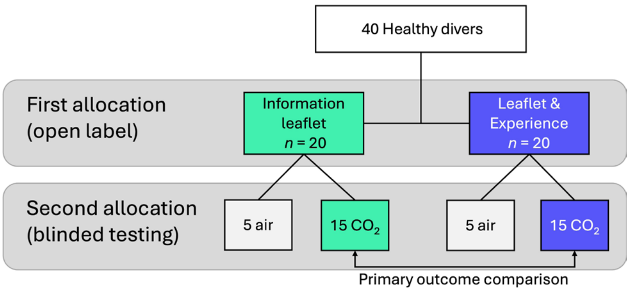
Methods

The study protocol was approved by the Health and Disability Ethics Committee, New Zealand (21/NTB/102) and was registered with the Australian New Zealand Clinical Trials Registry (U1111-1266-1320, http://www.anzctr.org.au/, RRID:SCR_002967). All participant data, informed consent, randomisation, and questionnaires were managed in a local secure database in RedCap.[17]

STUDY DESIGN

This single-blind randomised study was performed at the Exercise Physiology Laboratory at the University of Auckland, from April to August 2024. All participants received an information leaflet detailing the basic physiology of hypercapnia and frequent hypercapnic symptoms (*Appendix 1). In addition, they were randomly allocated into two groups: either receiving an unblinded hypercapnia experience (hypercapnia experience group) or not (information leaflet group). At least one month later, participants attended a blinded exposure where they underwent either a hypercapnic rebreathing protocol or breathed room air, allocated 3:1 (thus 15 from each group were exposed to hypercapnia, while five breathed room air) (Figure 1).

Figure 1
Study design; all participants were blinded to the intervention (CO_2 / hypercapnia or air exposure) during blinded testing visits



PARTICIPANTS

Forty healthy divers aged 18–55 years were recruited. Participants were deemed eligible following screening with the Diver Medical Screening Committee Diver Medical Participant Questionnaire.[18] All participants provided written informed consent. Study participants were excluded if they: were currently engaging in recreational or psychoactive drug use, had a history of mental illness, consumed greater than 21 alcoholic drinks per week, consumed more than five cups of coffee per day or equivalent caffeine consumption, were currently smoking or vaping, had previously participated in a hypercapnia study, or had significant freediving experience.

EQUIPMENT CONFIGURATION

A closed-loop breathing circuit was built from an O$_2$ptima closed-circuit rebreather (DiveRite, Lake City, USA), a Sentinel rebreather bailout valve (VR Technology, Poole, UK), and ADInstruments parts (ADInstruments, Dunedin, New Zealand) (Figure 2). The CO_2 scrubber cartridge was removed from the scrubber canister. A Petrel 2 rebreather controller (Shearwater Research, Richmond, Canada) continuously measured PO_2 within the circuit to maintain an inspired PO_2 of 30 kPa, thereby preventing a hypoxic inspired gas. Participants breathed through a mouthpiece connected to a bacterial filter (Elliptical filter, Alerkan Healthcare, Ankara, Turkey). The automatic diluent valve and bailout valve were connected to an air cylinder. Respiratory tubing (MLA1011A, ADInstruments) connected the rebreather parts through SP0143 3-way stopcocks (ADInstruments), which allowed for switching between room air and the rebreathing circuit. Respiratory filters increased the breathing resistance in room air mode to match the resistance in rebreather mode. During the blinded hypercapnia exposure, a series of four MLA246 4.7 L mixing chambers (ADInstruments) was added, so that rebreathed levels of CO_2 would not increase too precipitously.

A sampling line, positioned just distal to the mouthpiece filter, continuously sampled inspired and expired gas,

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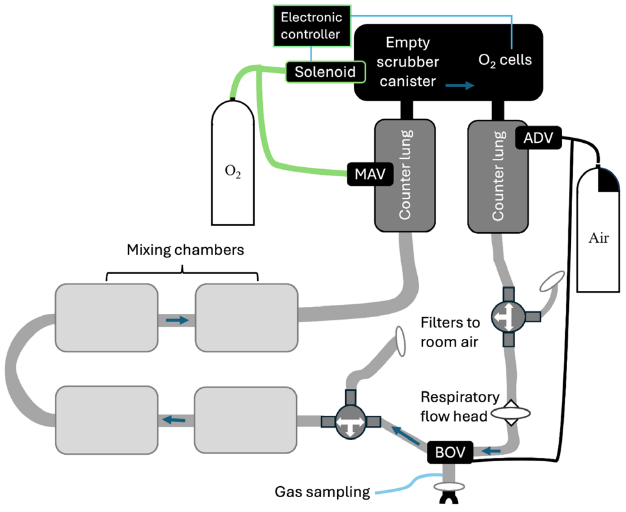
***Footnote:** Appendix 1 is available online on our website https://www.dhmjournal.com/index.php/journals?id=416

Figure 2

Experimental rebreathing circuit set-up; the three-headed arrows represent the three-way manual stopcocks that allowed the switch between rebreathing (depicted) and breathing room air (turn counterclockwise). Mixing chambers in series were added for the blinded hypercapnia exposures only. ADV – automatic diluent addition valve; BOV – bailout valve; MAV – manual addition valve



via a desiccant cartridge (MLA604), Nafion drying tube (MLA0343), and in-line filter (MLA0110), to a ML206 respiratory gas analyser (all ADInstruments) set to 200 ml·min^{-1} for measurement of CO_2 and O_2. The gas analyser was calibrated with room air and a hypoxic/hypercapnic gas mixture prior to each measurement. Ventilation was measured with an inline MLT1000L respiratory flow head and FE141 spirometer (both ADInstruments). The flow head was zeroed and calibrated with a MLA5530 3L syringe (ADInstruments) filled with room air prior to each measurement. Participants were instrumented for continuous measurement of heart rate (electrocardiogram, lead II ECG, BioAmp, FE321, ADInstruments), ear clip pulse oximetry (MLT320/E, ADInstruments), and non-invasive blood pressure (the latter in the unblinded experience only) with finger photoplethysmography (Finapres Nova, Finapres Medical Systems, Enschede, Netherlands), all sampled continuously at 1 kHz using Powerlab 16/35 and acquired via LabChart Pro 8.1.24 (ADInstruments).

EXPERIMENTAL PROCEDURE

All participants wore a nose clip to ensure closed-loop integrity. The closed-circuit rebreather was initially filled with an enriched oxygen mixture with PO_2 around 30 kPa. Each exposure began with room air breathing via the circuit, after which the stop-cocks were switched to the closed-circuit rebreather without the participants' knowledge. At the end of each exposure, the stopcocks were returned to open-circuit, while participants recovered for at least two minutes or until heart rate and the end-tidal pressure of carbon dioxide ($P_{ET}CO_2$) returned to baseline.

Unblinded hypercapnia experience

Twenty participants performed a visual attention task[19] while seated, breathing from the experimental setup. A baseline two minutes of room air breathing was observed followed by rebreathing in rebreather mode. Rebreathing was performed until either $P_{ET}CO_2$ reached 8.5 kPa, or the participant's symptoms became intolerable and they requested to cease the experience.

Blinded hypercapnia or air exposure

Cycle ergometry (Velotron Dynafit Pro, Seattle, WA, USA) was included to simulate the mild respiratory effort that is associated with normal underwater finning. The cycle ergometer was set to a workload estimated to elicit 30% of each participant's expected maximal exercise capacity, with the capacity calculated based on height, weight, age, and sex.

Participants were seated on the cycle ergometer and fitted with a virtual reality (VR) headset (HTC Vive Pro Eye, Taoyuan, Taiwan). This was programmed with a virtual reality dive to provide a level of task focus/distraction with some relevance to diving. The task was to count orcas swimming past using a manual click-counter.

After one minute stationary and two minutes of cycling while breathing room air and without the subjects' knowledge, the circuit was either switched to the closed-circuit rebreather mode to induce hypercapnia or remained on room air. Participants were instructed to 'bailout' by operating the bailout valve if they perceived symptoms of hypercapnia or if the heads-up display (HUD) turned from green to red in the VR environment. This was activated if a hypercapnic participant reached a $P_{ET}CO_2$ of 8.5 kPa without bailing out, or if breathing room air, after six minutes.

OUTCOME MEASURES

The primary outcome measure was the proportion of self-initiated versus HUD-prompted bailout among hypercapnic participants from the information leaflet and hypercapnia experience groups. Secondary outcomes included $P_{ET}CO_2$, inspired O_2 pressure, tidal volume, respiratory rate, minute ventilation (all breath-by-breath in 10-second averages), heart rate, blood pressure (unblinded experience only) (all beat-to-beat in 10-second averages), and time from the switch to breathing on the closed loop to rescue (during the unblinded training experience) or bail-out (during the blinded exposure). Self-reported hypercapnia symptoms experienced on a 0–100 visual analogue scale (VAS), as well as the first recognised symptom, were recorded within five minutes after each exposure.

STATISTICAL ANALYSIS

Descriptive statistics were reported as mean and standard deviation (SD) or median (range) where appropriate.

Normality of data was evaluated with the Shapiro-Wilk test. The difference in the proportion of participants in the information leaflet versus the hypercapnia experience group who performed a self-initiated bailout was analysed with a Chi-square test, with Cohen's w to calculate an effect size. Differences in cardiorespiratory outcome measures between the information leaflet and unblinded hypercapnia experience groups were analysed with independent *t*-tests and reported as mean difference with 95% confidence intervals (95% CI). These parameters were compared for a 10-second interval at the end of both the baseline and the hypercapnia periods. For the 15 participants who completed both the unblinded hypercapnia experience and the hypercapnia exposure, symptom consistency between both exposures was assessed with Pearson correlation. All data were analysed with SPSS Statistics version 27.0 (IBM, Armonk, NY, USA), with α set at 5%.

Results

Forty participants completed the study. Table 1 describes participant characteristics. The information leaflet group appeared to be more experienced (greater number of dives, years of diving experience, and more rebreather divers), but this was considered unlikely to be a confounding influence following analysis of the ventilatory response to hypercapnia stratified by diving experience. The mean time interval between the unblinded hypercapnia experience and the blinded hypercapnia exposure was 46 days (range 28–76).

None of the participants from either the information leaflet or unblinded hypercapnia experience groups who were subsequently randomised to receive room air in the blinded exposure performed a self-initiated bailout; thus, there were no false positives.

In the assessment of the primary outcome, 13/15 (87%) participants in the unblinded hypercapnia experience group performed a self-initiated bailout compared to 10/15 (67%) participants in the information leaflet group ($P = 0.149$, effect size = 0.264, Figure 3). Two of 15 (13%) participants in the unblinded hypercapnia experience group and 5/15 (33%) participants in the information leaflet group required a HUD prompt to bailout when the $P_{ET}CO_2$ reached 8.5 kPa. All participants bailed out appropriately in response to this prompt.

Cardiorespiratory responses during the unblinded hypercapnia experience and the blinded hypercapnia exposure are shown in Table 2. One participant was excluded

Table 1

Characteristics of the study participants; SD – standard deviation

Parameter	**Experience group *n* = 20**	**Information leaflet group *n* = 20**	**Total *n* = 40**
Age (mean years, range)	32 (18–53)	36 (23–54)	34 (18–54)
Female *n* (%)	9 (45)	6 (30)	15 (38)
Body Mass Index, kg.m^{-2} (mean, SD)	25.8 (4.1)	28.1 (5.7)	27.0 (5.0)
Ethnicity *n* (%)			
NZ European	9 (45)	10 (50)	19 (48)
Māori	1 (5)	1 (5)	2 (5)
Chinese	2 (10)	1 (5)	3 (8)
Other	8 (40)	8 (40)	16 (40)
Highest level of education *n* (%)			
Secondary school	5 (25)	4 (20)	9 (23)
Bachelors	9 (45)	5 (25)	14 (35)
Masters	5 (25)	6 (30)	11 (28)
PhD or other doctorate	1 (5)	5 (25)	6 (15)
Diving history			
Number of dives (median, range)	98 (3–600)	150 (5–2,000)	102 (3–2,000)
Years of diving experience (median, range)	7 (< 1–15)	12 (1–34)	8.5 (< 1–34)
Diving certification *n* (%)			
Open-circuit recreational	17 (85)	16 (80)	33 (83)
Open-circuit technical	2 (10)	1 (5)	3 (8)
Closed-circuit rebreather	1 (5)	3 (15)	4 (10)

Figure 3

Bailout outcome based on prior hypercapnia experience ('Training') or information leaflet allocation; the 10 participants who received room air rather than hypercapnia are shown in the figure to demonstrate that no false positives (i.e., a diver performing a bailout when they were not hypercapnic) occurred; HUD – head-up display

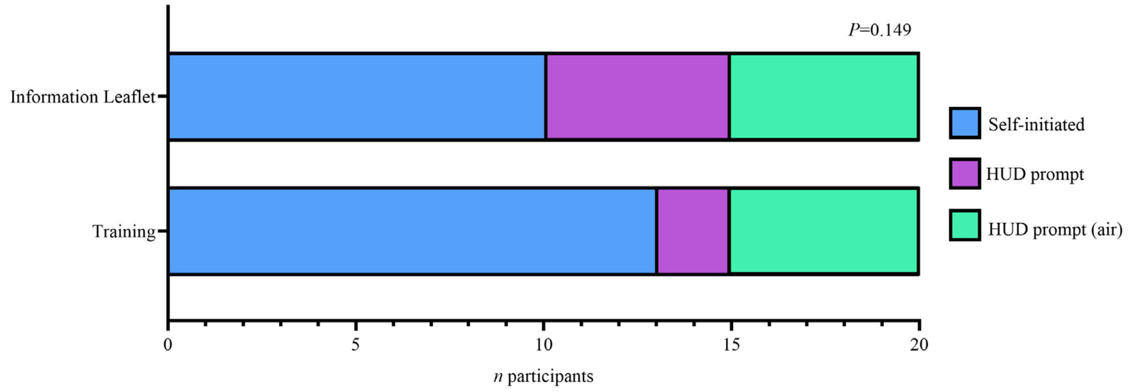


Table 2

Cardiorespiratory responses to hypercapnia during baseline and final 10 s of the unblinded hypercapnia experience (n = 19) and the blinded hypercapnia exposure for the hypercapnia experience group (n = 13) and the information leaflet (n = 15) group; room-air breathing controls and data from two subjects were excluded – see text. Data are presented as mean (standard deviation) and mean differences with 95% confidence intervals (CI). Mean difference is between groups during the final 10 s of the blinded hypercapnia exposure. $P_{ET}CO_2$ – end-tidal partial pressure of carbon dioxide; P_IO_2 – inspired partial pressure of oxygen

Group / parameter	**Unblinded experience**		**Blinded hypercapnia exposure**			
	Baseline ***n* = 19**	**Hypercapnia** ***n* = 19**	**Baseline stationary** ***n* = 20**	**Baseline cycling** ***n* = 20**	**Hypercapnia** ***n* = 13**	**Mean difference (95% CI)**
$P_{ET}CO_2$ (kPa)						
Experience group	5.1 (1.0)	8.2 (0.5)	5.2 (0.6)	5.8 (0.7)	8.0 (0.5)	0.1 (-0.3 to 0.5)
Information leaflet group			5.3 (0.6)	5.8 (0.5)	8.1 (0.6)	
P_IO_2 (kPa)						
Experience group	21.0 (0.2)	31.1 (1.1)	20.8 (0.3)	20.8 (0.3)	29.5 (0.7)	0.5 (-0.1 to 1.0)
Information leaflet group			21.1 (0.2)	21.3 (0.7)	29.9 (0.6)	
Tidal volume (L)						
Experience group	0.9 (0.4)	2.2 (0.5)	0.9 (0.2)	1.6 (0.4)	2.2 (0.6)	0.2 (-0.3 to 0.6)
Information leaflet group			1.1 (0.3)	1.7 (0.3)	2.3 (0.6)	
Respiratory rate (breaths.min^{-1})						
Experience group	12 (6)	19 (7)	13 (3)	14 (3)	19 (6)	3 (-8 to 1)
Information leaflet group			13 (4)	13 (4)	15 (6)	
Minute ventilation (L.min^{-1})						
Experience group	11.2 (8.9)	41.3 (17.6)	12.4 (4.1)	20.7 (5.7)	37.4 (7.3)	1.7 (-12.6 to 9.1)
Information leaflet group			13.9 (2.7)	22.2 (8.7)	35.7 (17.8)	
Heart rate (beats.min^{-1})						
Experience group	77 (13)	91 (20)	74 (26)	100 (33)	111 (38)	2 (-27 to 31)
Information leaflet group			85 (17)	107 (17)	113 (36)	
Blood pressure (mmHg)						
Systolic blood pressure	120 (35)	163 (18)				
Diastolic blood pressure	68 (22)	93 (12)				
Mean arterial pressure	86 (26)	117 (13)				

Figure 4

Matchstick figure showing individual ventilation (V_E) responses from stationary baseline to cycling baseline, and final 10 s of hypercapnia in the 15 participants from each initial group allocated to hypercapnia in the blinded exposures; the topmost line (a hypercapnia experience group participant) was classified as an outlier due to an extreme psychological response

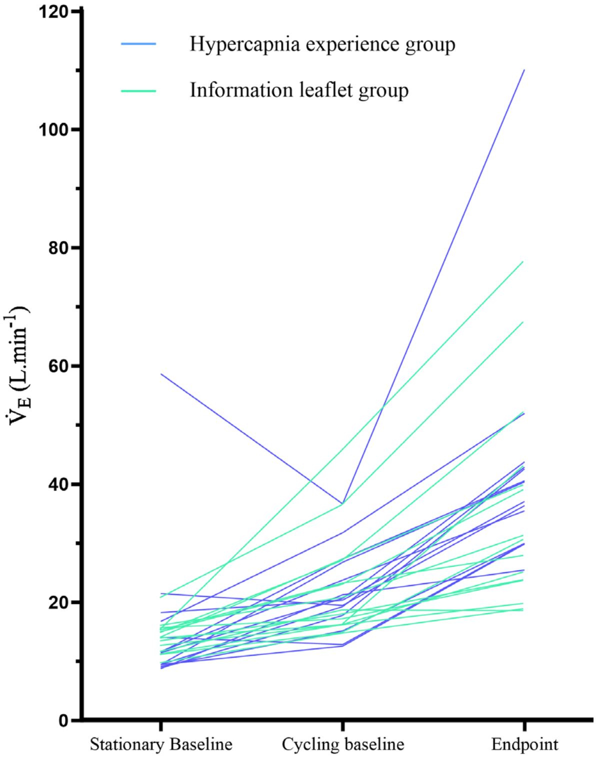
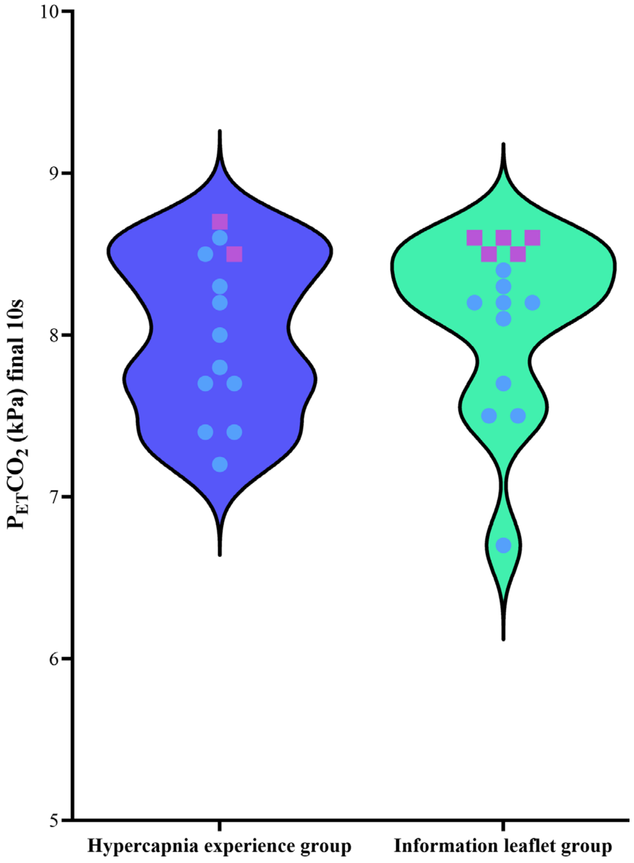


Figure 5

Violin plot of the end tidal CO_2 ($P_{ET}CO_2$) during the final 10 s of the blinded hypercapnia exposure, for the hypercapnia experience and information leaflet groups; blue circles represent self-initiated bailouts, while purple squares indicate those that required a head-up display prompt



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from the physiological data (for both exposures) due to a disproportionate psychological response at baseline and during hypercapnia (top blue line in Figure 4). Hypercapnia was associated with an increase in all parameters. Divers in the hypercapnia experience and information leaflet groups had similar cardiorespiratory responses at bailout (Figure 5, Table 2).

During the unblinded hypercapnia experience, one experiment was terminated after breathing from the circuit for 10 minutes due to technical difficulties (a crack in the tube, causing a plateau in the accumulation of CO_2), while four participants requested to stop the unblinded hypercapnia experience early due to symptom intolerance.

In the blinded hypercapnia exposure, those who performed a self-initiated bail-out had a lower $P_{ET}CO_2$ than those needing a HUD-prompt (mean difference 0.7 kPa, 95%CI: -1.1 to -0.2, Table 3). Participants perceived hypercapnia and bailed out at $P_{ET}CO_2$ as low as 6.7 kPa, with a mean of 7.9 ± 0.5 kPa for those who self-initiated the bailout (Table 3). However, there was no difference between the groups.

Shortness of breath was most frequently reported as the first and most intense symptom across all hypercapnia exposures (unblinded and blinded), regardless of initial group allocation (columns 1–3 Figure 6). The first symptom reported during the blinded exposure across both groups was shortness of breath (15/30, 50%), followed by a feeling of warmth (4/30, 15%), light-headedness (4/30, 15%), visual disturbance (1/30, 3%), and nausea (1/30, 3%). Of those who received hypercapnia in both the unblinded experience and the blinded exposure, 7/15 (47%) experienced similar symptoms (and intensity of symptoms) between both sessions ($P < 0.05$), while 8/15 (53%) did not.

Discussion

This study did not show a significant improvement in recognition of hypercapnia and self-initiated bailout after the unblinded hypercapnia experience compared to receiving the information leaflet only. There was a small difference between groups in the proportions of participants who self-initiated bailout during the blinded hypercapnia exposure (87% in the hypercapnia experience group versus 67% in

Table 3

Cardiorespiratory responses during the final 10 s of the blinded hypercapnic exposure (room-air breathing controls and data from two subjects were excluded – see text) comparing the hypercapnia experience group (HE) and the information leaflet group (IL), divided between those performing a self-initiated bailout and those needing a head-up display (HUD) prompt. Data are presented as mean (SD) and mean differences with 95% confidence intervals (CI). BPM – breaths per minute (respiratory rate) or beats per minute (heart rate); $P_{ET}CO_2$ – end-tidal partial pressure of carbon dioxide; P_IO_2 – inspired partial pressure of oxygen

Group	Self-initiated HE (*n* = 12) IL (*n* = 10)	Mean difference (95% CI)	HUD HE (*n* = 1) IL (*n* = 5)	Mean difference (95% CI)
$P_{ET}CO_2$ (kPa)				
Experience training	7.9 (0.5)	0.1 (0.5 to -0.4)	8.5	-0.1 (0.1 to -0.3)
Information leaflet	7.9 (0.5)		8.6 (0.1)	
P_IO_2 (kPa)				
Experience training	29.4 (0.8)	-0.4 (0.3 to -1.0)	29.9	-0.3 (1.4 to -1.9)
Information leaflet	29.8 (0.7)		30.2 (0.6)	
Tidal volume (L)				
Experience training	2.2 (0.6)	-0.2 (0.3 to -0.8)	2.2	0.0 (1.5 to -1.4)
Information leaflet	2.4 (0.6)		2.2 (0.5)	
Respiratory rate (BPM)				
Experience training	19.0 (6.6)	3.3 (8.9 to -2.4)	13.6	-0.5 (15.2 to -16.1)
Information leaflet	15.8 (6.0)		14.1 (5.1)	
Minute ventilation ($L.min^{-1}$)				
Experience training	38.1 (7.3)	-0.8 (12.3 to -13.9)	29.9	0.5 (31.0 to -30.1)
Information leaflet	38.8 (20.4)		29.4 (10.1)	
Heart rate (BPM)				
Experience training	109.6 (39.9)	-2.0 (35.2 to -39.2)	125.6	9.9 (54.7 to -34.9)
Information leaflet	111.6 (43.7)		115.7 (14.7)	
Time to 'rescue'/bail-out (min)				
Experience training	2.74 (0.67)	0.2 (0.8 to -0.3)	3.8	0.5 (3.4 to -2.4)
Information leaflet	2.51 (0.58)		3.4 (1.3)	

the information leaflet group), which might indicate that, for some divers, hypercapnia exposure training is useful. Nevertheless, the lack of a substantial training effect imparted by undertaking the unblinded experience may reflect the inherently unpleasant nature of hypercapnia, meaning that most participants will recognise it even if not previously exposed. This is also supported by the finding of no difference in the cardiorespiratory response between participants who self-initiated their bail-out. This contrasts with our recent finding in a similar study of a significant training effect for prior exposure to hypoxia, whose symptoms are more subtle and perhaps less appreciable on the basis of written material alone.[20]

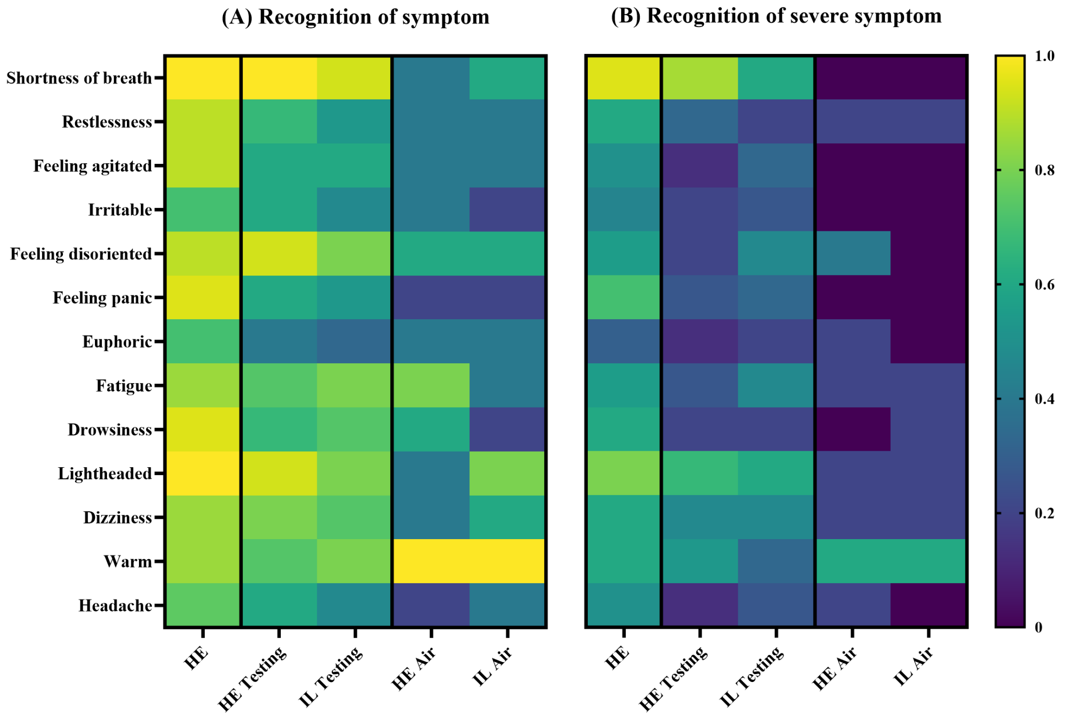
These findings align with previous work from our lab showing that during a five-minute 'pre-breathe', 15/20 divers were able to detect a fully absent scrubber in the rebreather apparatus.[11] However, 18/20 (90%) of divers were unable to detect a partially-failed scrubber in the rebreather set-up, as the inspired carbon dioxide levels were lower (but still elevated).

A previous study of hypercapnia exposure as a training strategy found that only 34% of 213 non-trained divers could detect a P_ICO_2 of < 3.6 kPa, while 90% of 186 trained divers could.[21] Moreover, hypercapnia-trained divers were able to detect CO_2 at lower levels ($P_{ET}CO_2$ 6.3 [SD 0.7] kPa) compared to non-trained divers (7.2 [SD 1.2] kPa). Those positive results could be due to the large sample size compared to our much smaller population, but more importantly, their unblinded hypercapnia exposure session took place immediately before the testing session.

Shortness of breath, light-headedness, and disorientation were the most intensely reported symptoms based on visual analogue scale results. Symptoms were more severe

Figure 6

Self-reported symptom heatmap; (A) shows the reporting of a symptom (visual analogue scale score ≥ 5/100), and (B) shows the reporting of severe symptoms (visual analogue scale score ≥ 50/100). Yellow indicates all participants recognised this symptom (A) or recognised it as severe (B), while dark blue indicates no participant recognised this symptom (A) or as severe (B). HE – unblinded hypercapnia experience; HE Testing – unblinded hypercapnia experience group exposed to hypercapnia during the blinded testing session; IL Testing – information leaflet group exposed to hypercapnia during the blinded testing session; HE or IL Air – participants from the hypercapnia experience or information leaflet groups breathing room air during the blinded testing session



in the unblinded experience session, potentially due to the masking by cycling in the blinded exposure. As expected, the symptom experience was more severe and specific to hypercapnia in the hypercapnia group versus those who were breathing room air, indicating that our blinding was effective. During the blinded exposure, the prior hypercapnia experience group more often recognised and experienced more severe symptoms compared to the information leaflet group, indicating a potential training effect, as the $P_{ET}CO_2$ range was similar in both groups.

The present study has several limitations. Most importantly, it may have been underpowered to demonstrate significance for a real but small-to-medium training effect. In addition, the study environment contrasts with diving, where submergence, higher pressures, and dense gas affect breathing in ways that could reduce or exaggerate any benefit of prior hypercapnia training.[22] For example, hyperoxia (via a suppression of the carotid body's response to hypercapnia) or nitrogen narcosis might reduce symptom experience,[3,4] whereas immersion augments chemosensitivity,[23] and cold water can intensify symptom experience.[24]

In contrast, the study is strengthened by the inclusion of a control group, who did not perform a training exposure (but received related educational material), and the further inclusion of five participants from each group who continued to breathe room air during their blinded ('testing') session. There were no false positives in any of these ten exposures, suggesting that despite understanding the purpose of the study, the weight of expectation was appropriately calibrated among our participants. Moreover, rather than performing a step change in inspired CO_2, a progressive increase in CO_2 was imposed. This is a more ecologically valid model of elevation of inspired CO_2 when diving a rebreather. Our study had a one-month period between both sessions, allowing for some settlement of the experience before the blinded exposure. This contrasts with previous research that had both sessions on the same day.[21,25] Lastly, we mitigated some confounding factors that may influence the potential for premature detection of a hypercapnic stimulus outside of normal dive conditions by having the participants exercise on a cycle ergometer at an intensity similar to casual finning. This exercise was designed to elicit an increase in ventilation and a feeling of warmth that would mask these hallmark hypercapnia symptoms, as would be the case underwater.

Although the results from this study showed a minimal effect of prior hypercapnia exposure in improving recognition of a subsequent hypercapnic event, we remain open-minded about the concept of hypercapnia exposure as an educational strategy for rebreather divers. It remains possible that prior experience of hypercapnia symptoms may help in symptom recognition during diving. In addition, experience of the unpleasant 'breathless' nature of hypercapnia would at least engender a respect for the problem, demonstrate the elevated gas consumption likely to accompany hypercapnic events after bailout onto open circuit scuba, and motivate attention to avoidance strategies such as proper replacement of CO_2 absorbents and avoiding hard work and dense gas at extreme depths. Importantly, unlike hypoxia which could result in loss of consciousness, controlled hypercapnia exposures in a dry environment are unlikely to be harmful in healthy individuals, especially if simple precautions are taken. If done, we would advise that hypercapnia exposures be performed under experienced supervision, for instance, as part of a rebreather diver training course. A small $P_{ET}CO_2$ sensor,[26] should also be included at the mouthpiece to reassure that the $P_{ET}CO_2$ remains within clearly safe limits (for example, < 8.5 kPa).

Currently, there is no reliable method of detecting rising arterial CO_2 during a dive.[27] The present research indicates that, if warned, divers are still capable of performing a self-initiated bail-out at $P_{ET}CO_2$ of 8.5 kPa (albeit in a dry lab environment), while Warkander et al. showed some divers could become incapacitated at levels marginally higher than this.[4] Therefore, if underwater capnography were developed, bailout warnings should be given well in advance of 8.5 kPa. Our results provide some reassurance that with warnings prior to this level, divers can successfully perform a self-rescue bailout procedure adequately. However, we should note that previous research has already shown an increase in reaction time at a $P_{ET}CO_2$ of 7.3 kPa.[5]

Conclusions

We report that divers who underwent an unblinded hypercapnia experience were not better at recognising hypercapnia and bailing out from a rebreather during a subsequent simulated dive compared to those who read about hypercapnia symptoms from an information leaflet. However, our study may have been underpowered to prove a small to medium training effect based on the predefined primary outcome. There was also no difference in the physiological response between the two groups at bail-out. We remain sympathetic to the idea that becoming familiar with the symptoms associated with hypercapnia, particularly the feeling of shortness of breath, through undergoing a hypercapnia experience with knowledgeable supervision in a dry, safe environment may be valuable in rebreather diver training.

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Conflicts of interest and funding

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