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Divers revisited: The ventilatory response to carbon dioxide in experienced scuba divers

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Summary
Purpose: To investigate the ventilatory response to CO_2 in hyperoxia, hypoxia, and during exercise amongst experienced scuba divers and matched controls.
Methods: Two studies were performed. The first investigated the CO_2 sensitivity in rest and exercise using CO_2 rebreathing in hyperoxia at a workload typical for diving with divers ($n = 11$) and controls ($n = 11$). The second study examined the respiratory drive of divers ($n = 10$) and controls ($n = 10$) whilst breathing four different gas mixtures balanced with N_2 (ambient air; 25% O_2/6% CO_2; 13% O_2; 13% O_2/6% CO_2) to assess the combined response to hypercapnia and moderate hypoxia.
Results: Exercise at a load typical for diving was found to have no effect on the ventilatory sensitivity to CO_2 in divers (rest: 1.49 ± 0.33; exercise: 1.22 ± 0.55 [l/min × $mmHg^{-1}$]) and controls (rest: 2.08 ± 0.71; exercise: 2.05 ± 0.98 [l/min × $mmHg^{-1}$]) while differences in sensitivity remained between the groups. Inhalation of the four gas mixtures revealed the tested oxygen pressures caused no significant alteration in the ventilatory sensitivity to CO_2 in divers and controls.
Conclusions: Experienced divers possess a lower ventilatory response to CO_2 which was not affected by exercise or the tested oxygen pressures suggesting a dominant adaptation of central CO_2 sensitivity.

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Introduction

Hypercapnia related symptoms ranging from increased depth and rate of breathing, breathlessness (air hunger), headache, dizziness, mental disorientation to complete unconsciousness are risks associated with diving whilst breath holding or using underwater breathing apparatus [1,2]. Elevated PCO_2 is reported in both diving groups however breath hold diving is associated with prolonged apneusis [3]. Elevation of PCO_2 during a scuba (self-contained underwater breathing apparatus) dive can be the result of a combination of factors including intentional 'skip breathing', unintentional hypoventilation and the use of a semi-closed or closed circuit rebreather [2]. Exposure to even mild levels of hypercapnia has been shown to substantially increase the risks of developing central nervous system oxygen toxicity [4,5]. Earlier studies have compared the ventilatory patterns, lung volume and chemosensitivity of non-divers with either breath hold or scuba divers with varying results [3]. Florio, Morrison and Butt [3] found Royal Naval Clearance Divers possessed a lower mean ventilatory response to hypercapnia compared to non-divers of similar age and build.

There remains an ongoing debate as to whether exercise has an influence on the ventilatory response to PCO_2. Cummin et al. [6] found an increase in the ventilatory response when cycling above 75 Watts whereas Clark and Godfrey [7] found a decrease. Furthermore, Kelley, Owen and Fishman [7,8] and Martin et al. [9] found no change in the ventilatory response to CO_2 with exercise, whilst McConnell and Semple [10] found endurance athletes had a lower ventilatory response to PCO_2 during rest compared to sprint trained athletes. However during exercise their ventilatory response to CO_2 was the same as sprint trained athletes and control subjects. To our knowledge Froeb's [11] research is the only study which has assessed the effects of exercise on the ventilatory response to CO_2 amongst scuba divers. Froeb [11] tentatively concluded a tendency existed for scuba divers to display a diminished ventilatory response to CO_2 during rest however this was not present whilst performing light exercise of 3 km·h on a flat treadmill ($\approx$70 Watts). It is a concerning prospect that few studies have investigated the effects of exercise on the ventilatory response to CO_2 amongst scuba divers as diving is an active activity involving primarily the lower body to propel the diver through the water at an estimated workload of 7 METS [12] If as Clark and Godfrey [7] found exercise lowers the ventilatory response to CO_2 then the risks of CO_2 retention during a scuba dive may be greater than previously thought.

If the divers are found to process a lowered ventilatory response to PCO_2 as we hypothesise, questions still remain as to whether this can be attributed solely to central adaptation of chemosensitivity or whether there is a contribution by the peripheral chemoreflex. As of yet, research with scuba divers has only tested the ventilatory response to CO_2 with hyperoxic conditions. It is regarded in most individuals that hyperoxia (PO_2 = 150 mmHg) effectively silences the peripheral chemoreflex response to CO_2 [13,14]. In order to investigate the influence of the peripheral chemoreflex on the ventilatory response to CO_2, tests need to be performed in hypoxic conditions. Hypoxic stimulation of the peripheral chemoreflex response has been shown to increase the peripheral chemoreflex sensitivity to CO_2 via changes in [H+] at the carotid body [13,15,16]. In this current study we hypothesised that the ventilatory response to hypoxia would not be significantly different between scuba divers and controls as divers are often exposed to hyperoxia. Melamed and Kerem [17] found no impairment in the hypoxic ventilatory response amongst active O_2 divers, ex-O_2 divers and non-diving controls. In order to assess whether the peripheral chemoreflex to CO_2 is altered through scuba diving the ventilatory response to CO_2 with and without the presence of hypoxia was compared. In this study we used a hypoxic mixture of 13% O_2 obtaining an average end-tidal pO_2 of 56.5 $\pm$ 3.99 mmHg. This mixture fits closely with Duffin's [13] recommendation of a hypoxic pO_2 of 50 mmHg being used to add the peripheral response.

Methods

Participants

This study comprises of two experiments which were both approved by the Ethics Committee of Bangor University (Gwynedd, Wales) and carried out in accordance with the Declaration of Helsinki for research on human subjects. Written informed consent was obtained from all subjects prior to testing. Male experienced scuba divers and non-divers were recruited. To be eligible for the scuba diving group, subjects were required to have performed at least 100 dives (Table 1). For inclusion in the control group, participants were required not to had any experience in scuba or breath hold diving. The participants' level of physical activity was collected and assessed through the use of a physical activity questionnaire and the groups matched for age; body mass, height and physical activity (see Table 2).

Table 1 Diving experience of the scuba diving group measured with a diving questionnaire in all studies. All divers used open-circuit breathing apparatus and regularly used enriched air nitrox gas mixtures. Values represented mean $\pm$ SD.

Parameter:	Experiment 1: rest vs. exercise	Experiment 2: hypercapnia and/or hypoxia
N	11	10
Years diving	15.5 $\pm$ 9.0	15.0 $\pm$ 9.2
Number of dives	1045 $\pm$ 1083	1621 $\pm$ 1250
Max depth dived (m)	52.6 $\pm$ 11.7	45.8 $\pm$ 21.11
Common diving depth (m)	26.4 $\pm$ 6.4	28.0 $\pm$ 10.3

Table 2 Physical characteristics of participants. For the categories of physical activity scores 1 = low, 2 = moderate, 3 = high activity. Values represent mean ± SD.

	Experiment 1: resting vs. exercise		Experiment 2: hypercapnia and/or hypoxia	
Parameter	Divers	Controls	Divers	Controls
N	11	11	10	10
Age (yr)	33.5 ± 10.0	27.6 ± 8.0	35.6 ± 9.0	31.8 ± 8.2
Height (cm)	179.9 ± 8.5	178.7 ± 6.0	179.9 ± 7.3	177.3 ± 5.6
Mass (kg)	84.6 ± 18.9	75.8 ± 10.9	82.6 ± 20.2	78.5 ± 7.8
BSA (m^2)	2.0 ± 0.2	1.94 ± 0.13	2.01 ± 0.25	1.95 ± 0.10
FVC (l)	5.13 ± 0.65	5.52 ± 0.54	5.45 ± 0.49	5.49 ± 0.55
FEV_1 (l)	4.29 ± 0.51	4.66 ± 0.47	4.47 ± 0.35	4.41 ± 0.62
Physical activity scores	1.7 ± 0.6	2.0 ± 0.4	2.1 ± 0.7	2.0 ± 0.7

General procedures

The objective of the first experiment was to investigate the ventilatory response to CO_2 amongst experienced scuba divers and non-diving controls during seated rest with blood gases measured in arterialised capillary blood samples from the earlobe and during exercise involving cycling at a workload 'typical' for a scuba dive [12]. If required subjects could split the trial into two visits scheduled for the same time of the day (± 1 h) to exclude for any circadian variability in the respiratory drive [18]. The second experiment compared the ventilatory response to hypercapnia with hyperoxia, normoxia, and moderate hypoxia amongst scuba divers and controls breathing fixed gas mixtures. This was to investigate the contribution of the peripheral and central chemoreflex to the altered CO_2 response in divers. All experiments were performed at room temperature (18–22 °C) with humidity (<70%) with FEV_1 and FVC measured first whilst seated following ATS/ERS guidelines [19].

Experiment 1: Ventilatory response to CO_2 during exercise and rest

Procedures

Subjects performed CO_2 rebreathing in rest or exercise in a counterbalanced randomised order. During the test subjects focused on a non-dramatic movie to avoid conscious control of breathing [20]. Progressive hyperoxic hypercapnia was achieved using a 150 l Douglas bag filled with 100% oxygen. During the test participants were connected to a breath by breath metabolic cart (3B Metalyser®, Cortex Biophysik, Germany) with its volume transducer and gas sampling port attached to a closed circuit rebreathing loop including the Douglas bag. Inspired gases were sampled from the loop via a bespoke valve which allowed CO_2 and O_2 concentrations to be measured by a high flow gas analyser (Servomex 5200, Servomex Group Ltd, England). Both gas analysers were calibrated prior to testing with pre-mixed gases of known composition. Calibration of the Cortex volume transducer was also performed whilst connected to the closed loop system with the Douglas bag disconnected to adjust for any resistance in tubing and valves.

CO_2 rebreathing was stopped when 8% end-tidal PCO_2 was reached. To allow the duration of the test to be uniform between subjects the amount of oxygen required in the Douglas bags were estimated based on the body characteristics using the Harris and Benedict [21] and Weir equation [22] enabling a test duration of 15–20 min during the resting condition. During exercise rebreathing, however, test duration was reduced to 5 min as pilot testing revealed progressive elevation of CO_2 during exercise was not tolerated for longer.

Resting CO_2 rebreathing

Resting minute ventilation was first recorded with the subject connected to the system with the rebreathing bag detached allowing ambient air to be ventilated. This period lasted for at least 8 min to allow ventilation to stabilise prior to the connection of the rebreathing bag. Inspired gases were continuously monitored taken from inspiratory tubing next to the mouthpiece (Servomex Gas Analyser) and end-tidal gas values were measured by the metabolic cart (3B Metalyser®, Cortex Biophysik, Germany). Furthermore, arterialised capillary blood samples from the earlobe were taken from the participants' right earlobe using Finalgon® (Boehringer Ingelheim, Germany) cream to achieve arterialisation. These blood samples were collected prior to rebreathing (ambient) and at five, six and seven percent PCO_2 and analysed using a blood gas analyser, (GEM Premier 3000 Instrumentation Laboratory, UK). Earlobe capillary blood samples have been shown to be valid substitutes to arterial blood samples [23]. Rebreathing was stopped once 8% end-tidal PCO_2 was reached.

Exercise during CO_2 rebreathing

The subjects cycled on an electronically braked cycle ergometer for 5 min at a cadence of 60 rpm with a load of 100 Watts while breathing through the system with the rebreathing bag disconnected. The CO_2 produced during the exercise protocol was not significantly different between the two groups (Scuba: 1.32 ± 1.24 l/min; Controls 1.28 ± 0.17 l/min). The load was chosen to simulate the typical workload of a scuba dive [12]. After 5 min the

rebreathing bag was connected with the experiment continued until 8% end-tidal PCO_2 was reached.

Experiment 2: Ventilatory response to CO_2 during hyperoxia and hypoxia

Procedures

In this experiment the volume transducer and gas sampling port of the metabolic cart (MetaMax® 3B, Cortex Biophysik, Germany) was attached to a two-way valve allowing gas to be inspired from the Douglas bag and expired into the atmosphere. The metabolic cart was calibrated prior to testing with a pre-mixed gas composition of 13% O_2 with 6% CO_2. Calibration of the volume transducer was carried out while connected to the tubing and valve system with the Douglas bag disconnected, allowing adjustment for any resistance generated by the system. A 250 l Douglas bag was filled prior to each experiment with the required pre-mixed gas balanced with N_2 (BOC Ltd, England). These gas mixtures were Mixture 1: ambient air; Mixture 2: 25% O_2/6% CO_2, Mixture 3: 13% O_2 and Mixture 4: 13% O_2/6% CO_2. Six percent CO_2 was chosen to correspond with Eynan et al. [20] study method which used a test procedure later adapted by the IDF Medical Corps of the Israel Naval Medical Institute. 13% oxygen was selected to achieve moderate hypoxia [24]. Ambient air was breathed to allow measurement of baseline minute ventilation. Subjects were blinded to the order of tests. The subjects were seated and connected to the breathing system, breathing each gas mixture until a plateau in minute ventilation was achieved (~5 min). Additionally during the experiment subjects focused on a non-dramatic movie with questions being asked about the movie at the end of the study, this to avoid the subject consciously controlling their ventilation [20].

Data analysis

All measurements are expressed in BTPS with mean ± SD and $p < 0.05$ considered statistically significant. Spida 5 version 2.0.8.-2 software automatically selected the highest measures, according to ATS/ERS guidelines for FEV_1 and FVC [19]. In the first experiment the ventilatory response to $PCO_2$was analysed using linear regression. The slopes of the linear regression characterised the sensitivity to CO_2 for each subject. Statistical analysis was carried out using the Statistical Package for Social Sciences Version 16.0 for Windows® (SPSS Inc., Chicago, IL) with a two-way ANOVA performed on the mean ventilatory response slopes of the two groups with exercise and rest. In the second study a one-way ANOVA compared change from ambient (baseline) minute ventilation between the two groups with each gas mixture. An independent *t*-test then compared the change in minute ventilation of the hyperoxic hypercapnic concentration and the hypoxic hypercapnic concentration between the groups. Furthermore, Pearson *r* was used in both studies to identify existing correlations between increased diving experience and the ventilatory response.

Results

Testing the ventilatory response to CO_2 rebreathing in exercise and rest, the diving group displayed a significant ($p < 0.05$) lower ventilatory response to CO_2 during rest (1.49 ± 0.33 l/min $mmHgCO_2^{-1}$) and exercise (1.22 ± 0.55 l/min $mmHgCO_2^{-1}$) compared with non-diving controls (rest: 2.08 ± 0.71 l/min $mmHgCO_2^{-1}$; exercise: 2.05 ± 0.98 l/min $mmHgCO_2^{-1}$). Both groups revealed no change in ventilatory response slope between rest and exercise conditions. Fig. 1 shows the change in minute ventilation with rest and exercise CO_2 rebreathing and Table 3 shows the

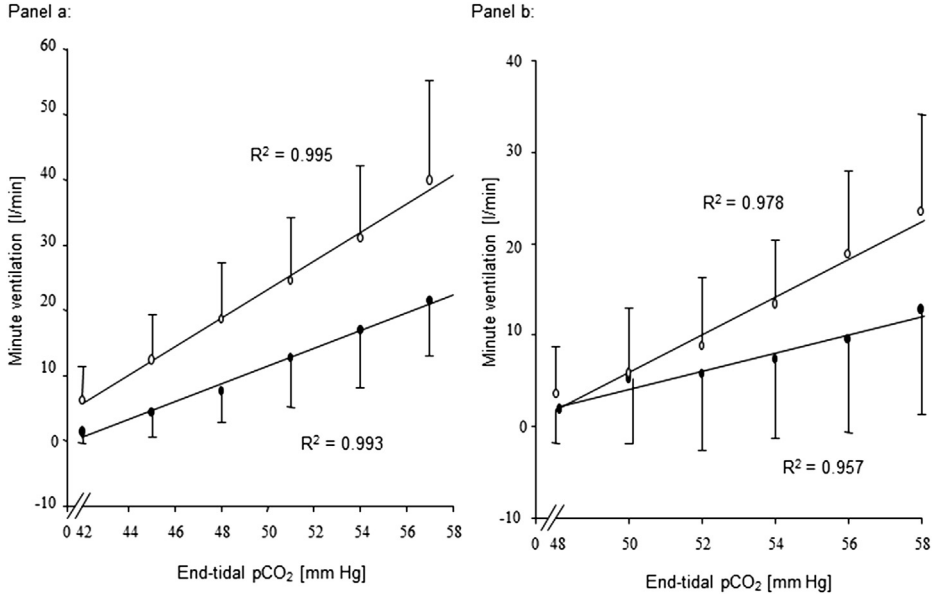

Figure 1 Change in minute ventilation from resting baseline vs. end-tidal PCO_2 during CO_2 rebreathing at rest (panel a) and whilst exercising (panel b) where ● = scuba diving group and ○ = control group. The scuba divers had a significantly lower ventilatory response slope during both rest and exercise ($p < 0.05$). Values represent mean ± SD.

Table 3 Capillary blood gas parameters during ambient and resting CO_2 rebreathing. Values represent mean ± SD.

	Ambient	5%	6%	7%
pH				
Scuba	7.40 ± 0.02	7.36 ± 0.02**	7.34 ± 0.01**	7.32 ± 0.02
Control	7.40 ± 0.02	7.38 ± 0.02	7.37 ± 0.02	7.33 ± 0.3
PCO_2 (mmHg)				
Scuba	43.18 ± 2.40	47.73 ± 2.28**	49.43 ± 2.23**	53.20 ± 2.20
Control	41.55 ± 1.57	44.72 ± 1.74	45.80 ± 1.48	51.14 ± 4.22
HCO_3 std (mmol/L)				
Scuba	26.01 ± 0.91	25.45 ± 0.80	24.97 ± 0.81	25.46 ± 1.29
Control	25.71 ± 0.95	25.70 ± 0.71	25.56 ± 0.78	25.33 ± 0.93
BE (mmol/L)				
Scuba	1.44 ± 1.16	0.57 ± 1.03	−0.04 ± 1.03	0.60 ± 1.64
Control	1.05 ± 1.21	0.90 ± 0.91	0.70 ± 0.99	0.42 ± 1.19
THbc (g/dL)				
Scuba	13.91 ± 0.80	13.93 ± 0.85	14.26 ± 0.91	14.42 ± 1.03
Control	13.93 ± 0.85	13.65 ± 1.01	88.88 ± 0.98	14.06 ± 0.84

*Significantly different means between the two groups by One-way ANOVA represented.
** = $P < 0.01$.

results of the analysed capillary blood samples during resting CO_2 rebreathing.

The second study compared the ventilatory response between scuba divers and controls whilst breathing premixed gas mixtures. The minute ventilation between the divers and controls was not significantly different whilst breathing ambient air (Scuba divers: 9.33 ± 2.94 l/min; controls: 10.32 ± 1.97 l/min).

There was no significant difference in the change in minute ventilation from baseline with the 13% O_2 mixture (Scuba divers: 9.76 ± 3.62 l/min; Controls: 11.58 ± 1.76 l/min). In both the hyperoxic hypercapnic gas condition (25% O_2/6% CO_2; Scuba divers: 18.54 ± 6.72 l/min; Controls: 25.76 ± 6.12 l/min) and the hypoxic hypercapnic gas condition (13% O_2/6% CO_2; Scuba divers: 22.42 ± 7.92 l/min; Controls: 29.88 ± 8.05 l/min) the divers displayed a significantly lower change in minute ventilation from baseline compared to the controls ($p < 0.05$). The change in minute ventilation from resting (baseline) is displayed in Fig. 2. The tested oxygen concentration used in our study did not significantly alter the ventilatory response to CO_2 in both groups suggesting the results are mainly due to a reduction in the central chemosensitivity of the scuba divers. In both experiments no correlation between diving experience and the ventilatory response to CO_2 was found.

Discussion

In both studies only highly experienced scuba divers were recruited. We could show that the respiratory drive to CO_2 was significantly lower in the divers compared with matched controls irrespective of the measured conditions of exercise or rest. Additionally, divers revealed a consistently lower sensitivity to CO_2 even if hypercapnia was combined with moderate hypoxia or hyperoxia. To our knowledge this study is the first to investigate a possible contribution of peripheral chemoreflex for the divers' altered ventilatory response to CO_2.

CO_2 sensitivity

During CO_2 rebreathing the divers exhibited a mean ventilatory response ~40% lower than controls of similar age and build. These findings are comparable to Florio, Morrison and Butt [3] which found a 33% lower CO_2 sensitivity amongst divers using breathing apparatus. Consistent with the findings of Froeb [11] and Florio, Morrison and Butt [3] no difference in vital capacity was observed between the scuba divers and controls.

Whether the ventilatory response to CO_2 amongst scuba divers is inherited or acquired through learning is still debated [3,5,11]. Kerem, Melamed and Moran [25] compared ex-divers, active divers and non-divers measuring end-tidal PCO_2 in rest and exercise found hypercapnic values were almost indistinguishable between the ex-divers and the active divers. Kerem, Melamed and Moran [25] suggested either this characteristic was acquired through training and retained after cessation of diving or was an inherent feature prevalent within the diving population. In our study as well as studies by Froeb [11], Florio, Morrison and Butt [3], and Kerem, Melamed and Moran [25] no correlation has been identified between the number of dives performed and the ventilatory sensitivity to CO_2 amongst scuba divers.

In favour of an acquired component Wood, Fatemian and Robbins [26] found repeated bouts of exercise paired with simultaneous CO_2 inhalations, altered the ventilatory response to exercise suggesting the ventilatory response to CO_2 may be influenced through learning and memory. In view of inheritance Saunders, Leeder and Rebuck [27] found a significant relationship between CO_2 ventilatory sensitivity in young swimmers and their siblings. However Scoggin et al. [28] found non-athletic parents and siblings of long-distance runners displayed a similar decreased ventilatory response to hypoxia but not hypercapnia. Furthermore, Eynan et al. [5] studied novice divers who trained extensively for 1 year (~150 dives) using closed circuit breathing apparatus with oxygen at shallow depths of 3–5 m. It was found the divers did not develop a

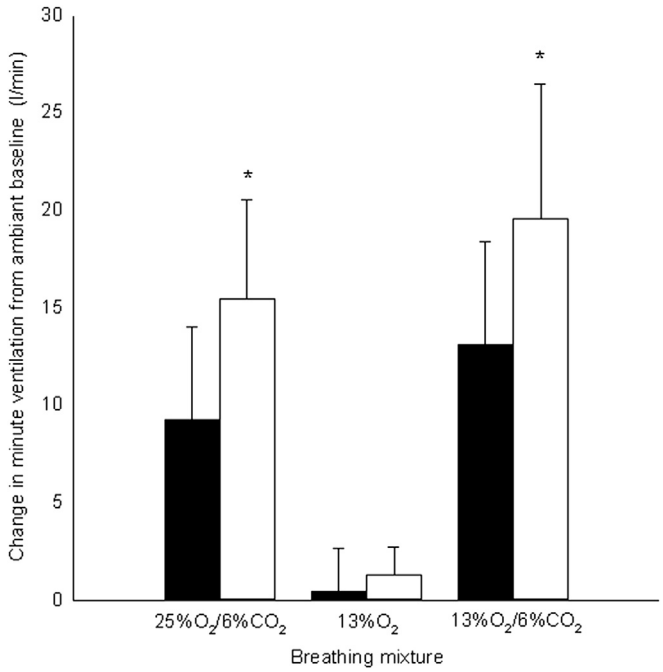

Figure 2 Change in minute ventilation from resting baseline (l/min) with breathing mixture. ■ = scuba divers □ = controls, where significantly different between the groups with each gas mixture, * = $p < 0.05$. Values represent mean ± SD.

tendency to retain CO_2 after this period suggesting, CO_2 retention is not a trait that is acquired during diving in shallow water. These findings may not be applicable to deeper diving as there is an increased gas density with depth resulting in an elevation in the work of breathing and subsequent reduction in ventilation [29]. Eynan et al. [5] suggest that a conditioned breathing pattern may be developed in divers conducting deep dives which may save on the work of breathing but result in an increase in CO_2.

Potential mechanisms which may explain the ventilatory response to CO_2 amongst scuba divers include the development of a conditioned breathing pattern when breathing through a mouthpiece [25]. The resting ventilation however was not significantly different between the two groups in both studies. Another possibility is the divers have a higher setting of chemostat. This implies a higher resting eupneic $PaCO_2$ [25]. The analysed capillary blood samples performed prior to CO_2 rebreathing revealed no significant difference between the groups when breathing air. There is the possibility the decreased ventilatory response to CO_2 is due to a reduced CO_2 build-up around the chemoreceptors caused by vasodilation and higher cerebral blood flow amongst the divers [25]. Slosman et al. [30] investigated 215 healthy recreational divers and reported a negative influence of dive depth on cerebral blood flow. They suggest scuba diving may have long-term negative neurofunctional effects when performed in extreme conditions such as cold water, with more than 100 dives per year and with maximal dive depth below 40 m. However the divers in our studies however regularly dive below this stated 40 m threshold.

Effects of exercise on CO_2 sensitivity

In this study exercise at the tested workload had no modifying effect on the ventilatory response to CO_2 revealing a persistent difference between divers and controls, contradicting Froeb's [11] findings. Froeb showed a disappearance of the difference in CO_2 response between divers and controls performing light exercise of 3 kmh on a flat treadmill (≈70 W). The chosen workload during CO_2 rebreathing in our study is estimated to be relevant to scuba diving [12]; moreover, Martin et al. [9] also reported no change in the ventilatory response to CO_2 during exercise amongst athletes. Differences in economy between the groups of divers and controls might have contributed to the loss of differences in ventilatory response to CO_2 in Froeb's study.

Effects of moderate hypoxia on CO_2 sensitivity

Synergistic effects of O_2 and CO_2 on ventilation has been shown to be based on carotid body response [31]. Even under euoxic/normocapnic ventilation the carotid body is suggested to play an important role in the control of ventilation and hyper-additive peripheral—central interaction for the combined response to O_2 and CO_2 has been reported [32].We hypothesised that an alteration in gain of the carotid body chemoreflex could be an influencing factor for the adaption of CO_2 response in divers. While there is so far no evidence for a central O_2—CO_2 interaction, we combined moderate hypoxia with hypercapnia to investigate a possible contribution of the carotid bodies for the reduced CO_2 response in divers. If divers would display a reduced gain for the combined response to CO_2 and O_2 of the carotid bodies, the ventilatory response to CO_2 in hypoxia versus hyperoxia should be reduced in divers compared with non-divers. However, in this study we could observe that the differences in ventilatory CO_2 response in hypoxic and hyperoxic conditions between divers and controls were unchanged, suggesting that the altered ventilatory response in experienced divers is a central adaptation.

Additionally, the finding that the ventilatory response to moderate hypoxia was not significantly different between the two groups supports this notion. These findings correspond with Melamed and Kerem [17] which also found no difference in the peripheral chemoreflex amongst non-divers, active O_2 divers and ex-O_2 divers.

Modifications of the respiratory drive in divers

In each study no correlation was found between CO_2 sensitivity and diving experience, a finding which is in agreement with other studies [3,11] leading to the suggestion that the changes in CO_2 sensitivity is achieved in a comparably short time or the sensitivity is inherited with individuals who are sensitive to CO_2 not likely to continue diving as a leisure or professional activity. There is of course the possibility the response to CO_2 is both acquired through learning and inherited. Walterspacher et al. [33] postulated successful breath hold diving is dependent on the magnitude of trainable adaptation to increased CO_2 levels as well as genetics. Apnoea training, involving repeated breath holds with short recovery periods, have also been shown to increase the time in withstanding the respiratory drive, contributing to prolonged breath hold duration [34].

Adaptations have been shown to occur in a short time amongst clinical populations which may also further increase our understanding of the modification of the ventilatory adaptation amongst scuba divers. Obstructive sleep apnoea patients are frequently exposed to nocturnal bouts of hypoxia and hypercapnia implicated to induce alterations in the central and peripheral chemoreceptors [35]. Likewise patients with Chronic Obstructive Pulmonary Disease (COPD) are also reported to process an attenuated ventilatory response to hypercapnia/hypoxia [36,37] unfortunately however, in COPD it is impossible to determine whether the reduced ventilatory response is due to an impaired respiratory central drive, as the ventilatory response is correlated with the mechanical limitations of COPD [37,38].

Conclusion

Scuba divers possess a lower ventilatory response to CO_2 and low intensity exercise does not modify the CO_2 sensitivity in divers and controls. The lowered ventilatory response seen amongst scuba divers seems limited to adaptation of the central chemoreceptors as there is no change in the difference between divers and non-divers in ventilatory drive in the hypoxic and hyperoxic CO_2 response. Moreover, these findings highlight the risks of CO_2 retention amongst scuba divers and provide additional support for the testing of the ventilatory response to CO_2 with scuba divers as achieved by the Israel Naval Medical Institute for physiological training [20].

Ethical standards

The experiments comply with the current laws of the country in which they were performed.

Conflicts of interest

The authors declare that they had no conflict of interest.

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