The utility and safety of hypoxia experiences for rebreather divers

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Abstract
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Background: Aircrew training often includes an hypoxic experience aimed at improving symptom recognition and self-rescue in a subsequent hypoxic event. Similar training has been advocated for rebreather divers. We investigated the effect of a prior hypoxic experience on actual and perceived cognitive function during subsequent hypoxia and measured the physiological responses to severe progressive hypoxia.
Methods: Twenty-five subjects underwent two hypoxic hypoxia experiences (trials one and two) approximately five weeks apart. Subjects breathed 5.5% oxygen whilst performing a playing card recognition test. The primary endpoint was the time taken to make three consecutive errors in the card recognition test (time of useful consciousness, TUC). Secondary endpoints were the total number of errors made, accuracy of error recollection and physiological variables.
Results: Mean (SD) TUC was 166 seconds (37) and 169 s (35), and subjects made 8.9 (2.4) and 7.8 (2.0) errors in trials one and two respectively. Error recall was identical between trials with participants failing to recall 6 (3) and 6 (2) errors made in trials one and two respectively. Across both trials mean nadir arterial blood and cerebral oxygen saturations were 52% and 49% respectively. The mean (SD) increase in heart rate was 42 (16) beats·min^{-1}.
Conclusion: An hypoxic experience did not improve cognitive performance or subject insight into performance in a second exposure five weeks later. Hypoxia imposes a significant physiological stress which may be hazardous in unscreened, non-medically supervised subjects. Hypoxia experience training is not recommended for rebreather divers at this time.

Introduction

Unanticipated severe hypoxia can occur in both aircrew and scuba divers with disastrous consequences. In aviation, hypoxia may arise because of cabin decompression or failure of oxygen (O_2) supplementation devices in unpressurised aircraft.[1] In diving, hypoxia may arise because of inadvertent breathing of an hypoxic gas mix at shallow depth, or failure of closed circuit 'rebreather' devices to maintain a safe inspired PO_2 (> 21 kPa) in the breathing loop.[2] In both settings the precipitating circumstances may not be obvious, distracted victims may fail to perceive encroaching symptoms of hypoxia, and consequent decrements in cognitive performance (and ultimately loss of consciousness) can lead to fatal accidents.

In aviation, there is a well-established practice of conducting periodic training experiences to familiarize aircrew with the symptoms of impending hypoxia. Participants are decompressed in a hypobaric chamber to pressures equivalent to high altitude (typically around 37.6 kPa, 25,000 feet) whilst wearing masks which deliver supplemental O_2. Removal of the masks exposes the subjects to hypobaric air and, therefore, hypobaric hypoxia. The demonstration of consequent failure both in simple cognitive tasks and in following instructions is considered a valuable illustration of the dangers of hypoxia. Several studies have demonstrated that an individual's hypoxic symptoms remain relatively constant between widely separated exposures (years).[3–5] Based on these experimental observations, coupled with some weak but supportive real-world evidence,[6] it is believed that better hypoxia symptom recognition prepares participants to recognize impending hypoxia and, thus, intervene in a timely manner in any subsequent event. In addition, although there is no substantive hypothesis that would explain better cognitive performance during hypoxic events occurring after previous hypoxia training, one small study reported a 10–20% higher (but statistically insignificant) probability of retaining useful consciousness in the latter stages of eight minutes (min) of hypobaric hypoxia conducted eight months after a prior hypoxic training exposure.[7]

Use of hypoxic training in aviation has motivated advocacy for hypoxia experiences for divers using closed circuit rebreathers.[8] It has been suggested that hypoxic experiences

could take place in dive training facilities or even private homes with participants breathing on a rebreather (effectively a closed-circuit breathing loop) with no oxygen addition until significant symptoms occur.[8] No established rebreather diving training agencies presently recommend this practice, the utility of which is uncertain and its safety questionable.

A recent negative study of creatine loading as a means of prolonging useful cognitive function during hypoxia conducted in our laboratory (unpublished observations) has provided certain insights into the value and safety of hypoxic experiences for rebreather divers. First, the question could be addressed as to whether a hypoxic exposure could result in prolongation of useful cognitive function during a subsequent hypoxic event. Second, observations were made of arterial blood and cerebral oxygen saturations and the cardio-respiratory responses during severe progressive hypoxia in humans. Such data are surprisingly difficult to find in the medical or physiological literature. This provided insights into the potential for medical complications of hypoxic training experiences in typical rebreather divers. Finally, the study afforded the opportunity for discussion of the complexity and difficulty in investigating whether a prior hypoxic training experience does or does not improve the chances of effective self-rescue during a subsequent real-world hypoxic event.

Methods

The study protocol was approved by the University of Auckland Human Participants Ethics Committee (reference 019199). The parent study was an investigation of the effect of creatine loading on cognitive performance during hypoxia. The relevant data will be reported independently. However, the lack of any effect of creatine on cognitive performance allowed us to re-evaluate the data as an investigation of the effect of a prior hypoxic exposure on cognitive function during subsequent hypoxia, and to appraise the physiological data in the context of conducting hypoxia experiences for rebreather divers. For simplicity, we will henceforth omit references to creatine administration from the narrative.

TRIAL DESIGN

This was an interventional cohort study that took place at the Exercise Metabolism Laboratory, University of Auckland from July to September 2017. Twenty-five subjects underwent cognitive function testing during two hypoxia experiences conducted approximately five weeks apart (a total of 50 hypoxia exposures).

PARTICIPANTS

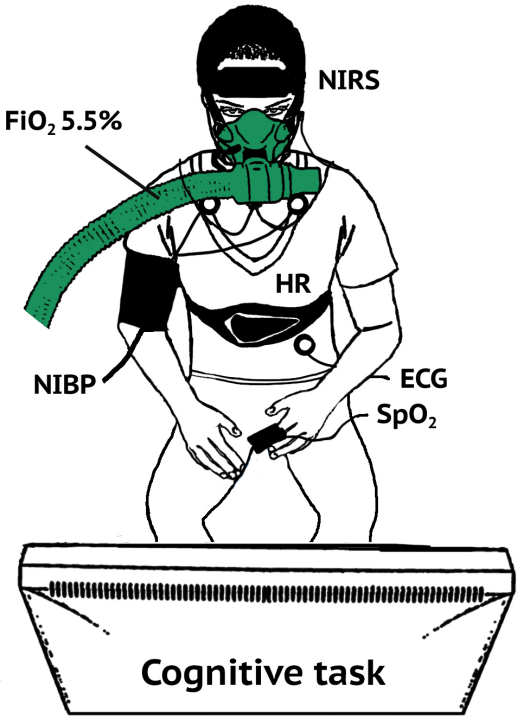
Subjects were 25 volunteers (15 male), mean age 28 years [11 SD], range 20–57 solicited from the local student and diving communities. All subjects received a participant information sheet, a verbal explanation of the study, and provided written informed consent. They completed a pre-participation medical screening questionnaire designed to exclude those with known cardiac, metabolic, neurological or respiratory disease or associated risk factors. The questions were similar to those on a standard pre-participation screening form for scuba diving and were chosen by a specialist anaesthesiologist (SJM).

EXPERIMENTAL SETUP

Subjects were comfortably seated and monitoring was established as follows: 3-lead electrocardiogram (standard limb lead II displayed), finger pulse oximetry (SpO_2; arterial haemoglobin oxygen saturation), and end tidal carbon dioxide (CO_2) (all components of an S5 anesthesia monitoring system; GE Electronics, USA); continuous measurement of inspired O_2 and CO_2 concentrations (via a ML206 Gas Analyzer, ADInstruments, Dunedin, New Zealand); ventilation (tidal volume and respiratory rate) via a Respiratory Flow Head (MLT1000 L, ADInstruments, New Zealand) and single-channel near infra-red spectroscopy (NIRS) (PortaLite, Artinis, The Netherlands) with optodes placed over the left pre-frontal cortex (Fp1 position, international 10–20 system).[9] NIRS is a non-invasive continuous measurement of cerebral (pre-frontal cortex) oxygenation influenced mainly by haemoglobin O_2 saturation in the cerebral venous blood. As in its use during clinical monitoring of patients, both the absolute O_2 saturation value and the percentage reduction from baseline saturation were utilized to measure the impact hypoxia on cerebral oxygenation. Blood pressure was measured via an automated non-invasive blood pressure cuff (NIBP) (GE Electronics, USA) before and after the experiment, but not during the trial because the discomfort of periodic cuff inflation would have been distracting. All experiments were attended throughout by a specialist anesthesiologist (SJM) for safety purposes.

Subjects breathed via a tight-fitting oronasal mask (7450, Hans Rudolpf Inc., USA) connected to a two-way valve (2700, Hans Rudolph Inc., USA) with the delivery of inspired gas controlled by a pneumatic controller and balloon-type valve (8200, Hans Rudolph, USA) which could be switched to deliver either room air or a premixed gas (5.5% O_2 94.5% nitrogen (N_2); inspired oxygen (PiO_2) 38.7 mmHg (SD 1.3)) drawn from a Douglas bag. Hypoxic gas mixtures were prepared within 10 min of each experiment by combining compressed medical grade O_2 and N_2 (BOC, Auckland, New Zealand) in 150 L Douglas bags. The O_2 fraction within each bag was verified using two independent gas analyzers (ML206 Gas Analyzer, ADInstruments, Dunedin, New Zealand). Prior to each experiment, the mask seal was confirmed by leak-free negative pressure generation by the subjects. Maintenance of the seal during the experiment was verified by continuous monitoring of the PiO_2. The experimental set-up is depicted in Figure 1.

Figure 1
Illustration of the experimental setup. ECG – electrocardiography; FiO_2 – fraction of inspired oxygen; NIBP – non invasive blood pressure cuff; NIRS – near infrared spectroscopy; SpO_2 – finger oxygen saturation; HR – heart rate



Cognitive function testing was via a card recognition protocol in which playing cards between numbers 4 and 10 (inclusive), of all four suits and with numbers removed were displayed in front of the subject on a bright LCD screen. The card changed every four seconds (s) irrespective of the accuracy of the subject's answer or absence of an answer. Subjects were required to verbalize the number and suit of each new card. Subjects completed a familiarization version of the test on four occasions under normoxic conditions, in order to confirm perfect test-retest reliability (r = 1) and 100% accuracy.

EXPERIMENTAL PROCEDURE

Each exposure began with the subject breathing air via the mask. The mask gas supply could be switched between air and the hypoxic mix without moving the mask using the balloon-operated valve (8200, Hans Rudolph, USA). The dead space in the hose supplying the mask from the gas switching point was 880 ml and would be effectively cleared within two breaths of switching (< 10 s). A two-minute preliminary period of card recognition (during air breathing) was conducted to establish task baseline measurements and provide a final familiarization with the task. Upon error-free completion of the preliminary task any questions that arose were answered before starting the hypoxic exposure.

The hypoxic exposure began with a three second countdown followed by switching from air to 5.5% O_2 breathing, at which time the card recognition test and measurement of time of useful consciousness (TUC) began. The subject continued breathing 5.5% oxygen until he or she made three consecutive card recognition errors. An error was defined as wrongly identifying the suit of the card and/or the card value, or failure to provide any answer within the 4 second period over which the card was displayed. When three consecutive errors occurred, the inspired gas was switched back to air and the TUC recorded. The card recognition test was continued during recovery of normoxia until SpO_2, ventilation, and heart rate recovered to pre-test levels. All experiments were videoed and later reviewed to verify the results recorded in real time.

Five minutes after completion of the protocol, subjects were asked how many errors they recalled making during the test, and they completed a post-trial questionnaire* adapted from the aviation literature in which the severity of potential hypoxia symptoms was rated on a 125 mm visual analogue scale (VAS) from "*not at all*" to "*severe*" where severe was further defined as meaning "*greatest intensity possible*".[10]

OUTCOMES AND ANALYSIS

The primary endpoint in each exposure was the TUC, defined as the duration from switching to the hypoxic gas supply to committal of the third of three consecutive card recognition errors. Data are presented as mean (standard deviation) and with ranges, as appropriate. The mean times of useful consciousness in the two hypoxic exposures were compared, as were the mean number of errors actually made with the mean number of errors that the subjects recollected making in the two exposures. Finally, the mean severity ratings of individual hypoxia symptoms between the two trials were compared. Comparisons of TUC, errors made, errors recalled, and ratings of symptom severity between the first and second hypoxic experiences were made using two-tailed paired *t*-tests (Prism 7, Graphpad, USA). A *P*-value ≤ 0.05 was considered to indicate statistical significance. No corrections for multiplicity were applied.

Results

All 25 enrolled subjects completed two hypoxia exposures (referred to as trial one and trial two) separated by an average 39 day interval (range 35–42 days). There were no adverse events.

* **Footnote:**
The post-trial questionnaire is available on request to the authors from n.gant@auckland.ac.nz

Figure 2
Time of useful consciousness (TUC) (seconds) for individual subjects in two hypoxic experiences (Trials 1 and 2) separated by approximately five weeks; group means ± SD shown beneath

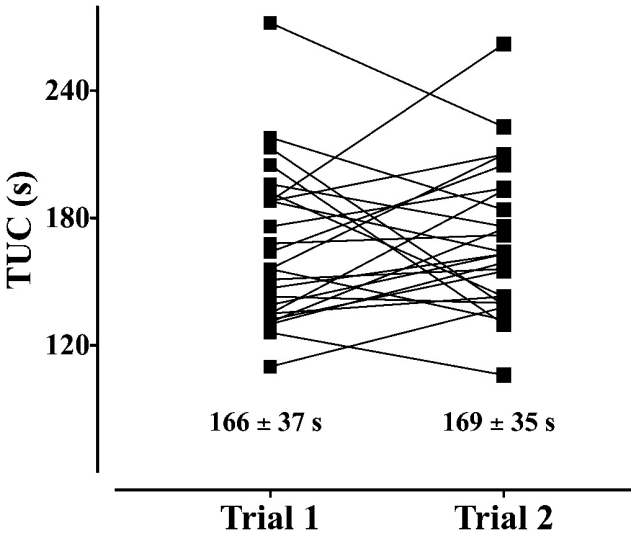
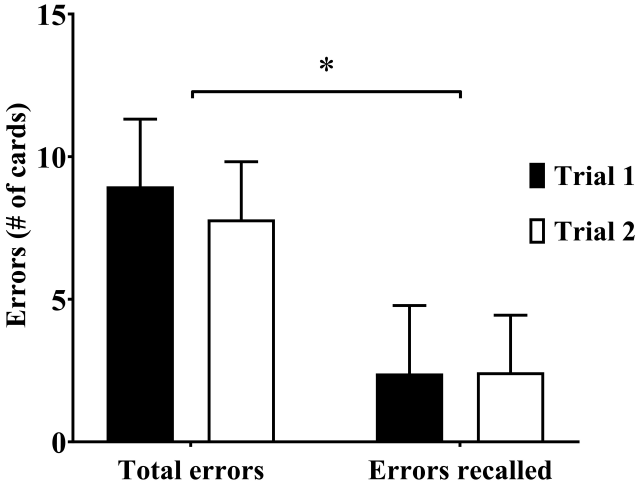


Figure 3
Mean (SD) card identification errors (total errors) made and errors recalled by the subjects in trials 1 and 2; * $P = 0.087$ for number of errors and $P = 0.91$ for recall of errors between the two trials



TUC

The TUC for individual subjects in trials one and two is shown in Figure 2. Mean TUC was 166 s (SD 37) and 169 s (SD 35) respectively ($t_{24} = 0.38$, $P = 0.70$). On average, subjects made 8.9 (SD 2.4) and 7.8 (SD 2.0) errors in trials one and two respectively ($t_{24} = 1.79$, $P = 0.087$) (Figure 3). Subjects exhibited poor perception or recall of the number of errors they made whilst hypoxic. Recall bias was virtually identical between trials with participants, on average, failing to recall 6 (SD 3) errors made in trial one and 6 (SD 2) errors made in trial two ($t_{24} = 0.12$, $P = 0.91$) (Figure 3).

SYMPTOM PERCEPTION

Perception of the 24 individual hypoxic symptoms listed in the post-trial symptoms questionnaire was very similar between trials one and two. The mean ± SD visual analogue scores for each symptom in each trial are shown in Figure 4. There were no statistically significant differences between trials in the ratings of symptom severity for all 24 symptoms (all $P > 0.05$).

PHYSIOLOGICAL CHANGES

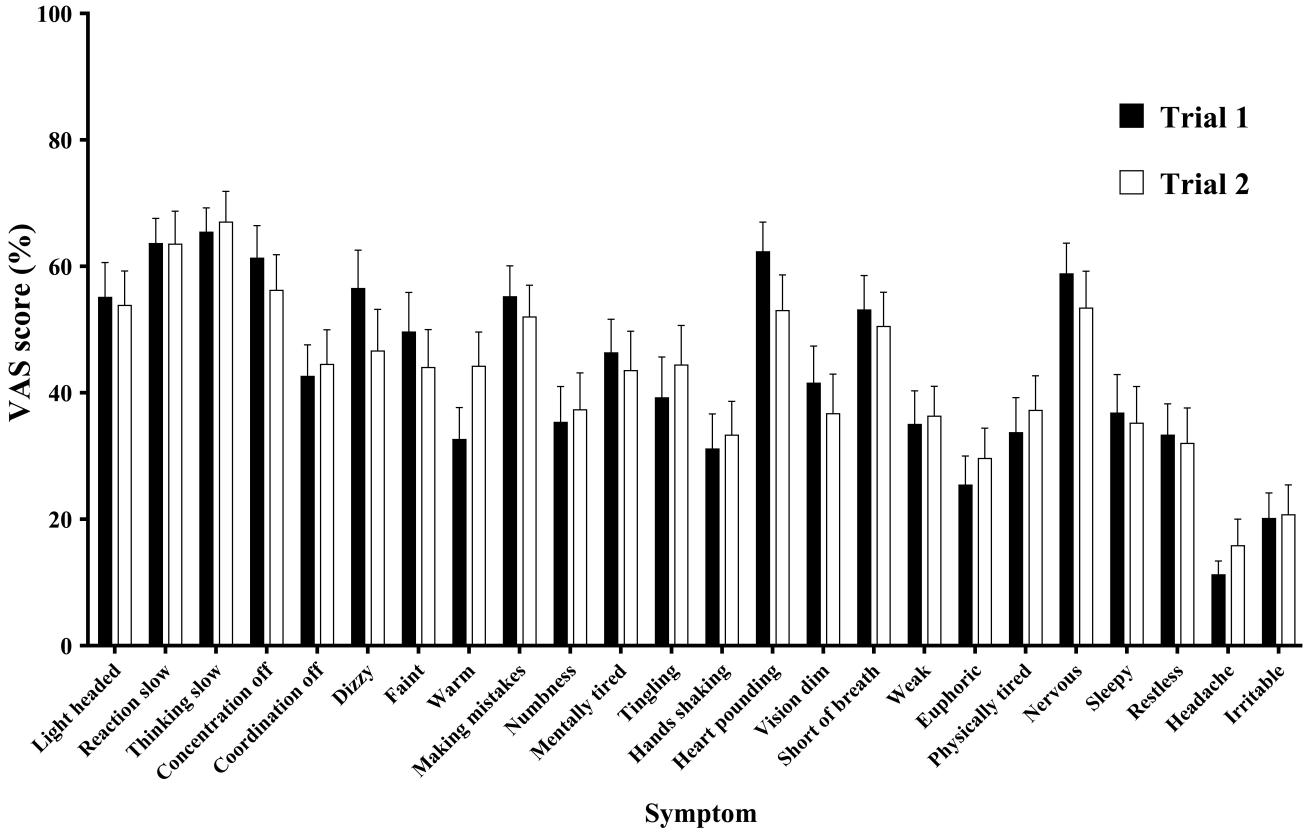
The physiological changes associated with breathing 5.5 % O_2 are shown in Figure 5. By the end of TUC mean heart rate and minute ventilation had increased by a mean of 42 beats·min^{-1} (SD 16) and 10.0 L·min^{-1} (SD 5.1), respectively. Conversely, SpO_2 reduced by 48% (SD 16). Subjects typically exhibited a nadir SpO_2 near 50% at the end of TUC. Prefrontal cortex tissue O_2 saturation (designated 'tissue saturation index' in Figure 5) decreased on average by an absolute value of 16 % (SD 4), but this represents a 25% decrease from the initial mean baseline value of 63%. The mean nadir in pre-frontal cortex tissue O_2 saturation was below 50%.

Discussion

Subjects were exposed to two episodes of severe hypoxia separated, on average, by 39 days and there was no evidence that the first exposure resulted in greater TUC during the second. There was extremely poor post-exposure insight into cognitive impairment during hypoxia, and no evidence that this improved after the second exposure. The lack of any difference in TUC between the two exposures helps clarify the finding of the one previous small study of the effect of repeat hypobaric hypoxia exposures on cognitive performance.[7] That study reported a statistically insignificant trend to (10–20%) higher probability of retaining useful consciousness during the latter stages of eight min of severe hypobaric hypoxia conducted eight months after a prior hypoxic exposure. However, there is no obvious physiological hypothesis which would predict that a hypoxic experience would improve TUC during subsequent hypoxia. One could speculate that a process of psychological adaptation or learning to cope with the impairment might have a positive influence, but our data confirm this does not occur (at least in the context of a one-month interval between events), even when subjects are well aware that they are experiencing hypoxia for a second time.

Consistent with previous studies in sequential widely separated exposures to hypobaric hypoxia,[3–5] the present study found that ratings of severity of potential hypoxia symptoms remained relatively constant between the two exposures. Largely on the basis of this previously reported within-subject consistency of hypoxic symptoms, hypoxia experience training has been recommended for flight crews

Figure 4
Mean (SD) symptom severity ratings during trials one and two recorded using a visual analogue scale (VAS); 0% = asymptomatic; 100% = most severe possible



for the purpose of facilitating appropriate early responses to hypoxia events in flight. The implicit assumption is that knowledge of one's 'hypoxic symptom signature' occurring in a prior hypoxic exposure could result in earlier symptom recognition and initiation of self-rescue in a second hypoxic event.

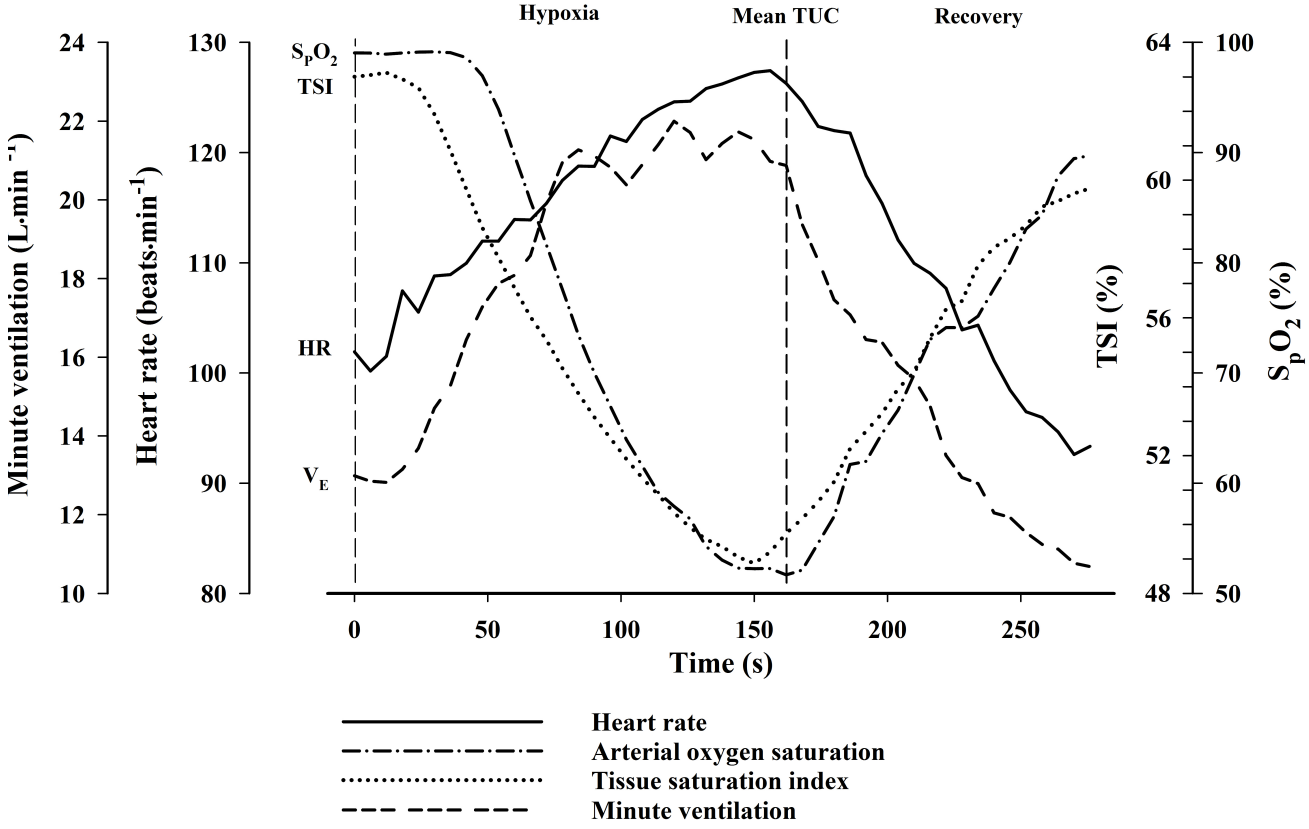
This assumption may be correct, but it has not been proven. A review of in-flight hypoxic events showed a dramatic difference in the incidence of loss of consciousness between aircrew who had received hypoxia training and untrained passengers.[6] However, there are other differences between trained aircrew and passengers (and their in-flight circumstances) that could at least partly account for this result. Our finding of very poor recall of errors during hypoxia is clear evidence of failure to accurately perceive the severity of impairment during hypoxia, or failure to form accurate memories of it, or both. Similarly, our subjects generally gave only mid-range VAS ratings of those hypoxia symptoms related to cognitive function despite their invariably severe objective cognitive impairment. These findings raise the suspicion that prior knowledge of hypoxic symptoms might not help a task-loaded and significantly distracted aviator or diver to self-rescue during a subsequent hypoxic event as reliably as seems to be believed.

This question is of high relevance to both rebreather diving and aviation but resolving it in an experimental setting would require a complex study. One would have to begin with subjects randomized into groups receiving initial hypoxic training (Group H – hypoxia) or not (Group N – no hypoxia). Then after some predetermined interval, both groups would then need to be randomized again to perform an objectively measurable and highly distracting task during either hypoxia or normoxia while blinded to their hypoxia/normoxia allocation, with an instruction to perform a secondary self-rescue task if they perceived impairment during the test. One would then compare the timing and execution of the self-rescue task in those Group H and Group N subjects randomized to hypoxia on the second exposure. The use of a valid task providing similar levels of motivation and distraction to flying a plane or operating a rebreather in the dynamic underwater environment would be a crucial component of such a study, as would blinding participants to their allocation (normoxia or hypoxia) in the second exposure. The difficulty in conducting such a study probably explains why it has not been done to date.

Our cautious interpretation of the benefits of hypoxia familiarization training should not be interpreted as suggesting that we disagree with current practices within

Figure 5

Mean responses from 50 hypoxic hypoxia exposures showing changes in arterial oxygen saturation of haemoglobin (S_pO_2) measured by pulse oximetry; TSI – near infrared cerebral tissue saturation index; V_E – minute ventilation; HR – heart rate; the hypoxia phase (ending at the point of mean TUC) represents the period of breathing 5.5% oxygen and the recovery phase represents the period of recovery during air breathing



aviation. We see little potential for harm in the aviation industry practice of exposing properly screened and medically supervised aircrew to hypobaric hypoxia as a training exercise. Experience has proven that there is little risk and there may be benefits (albeit incompletely quantified at this time).

However, in respect to rebreather diving we do not believe these benefits have been adequately demonstrated to recommend hypoxia experiences in diver training facilities or in divers' homes. Unlike the highly supervised aviation setting, there is no experience or safety data for hypoxia exposures provided to unscreened and non-medically supervised divers and based on first principles there are multiple risks. First, failure to terminate the exposure quickly enough could result in loss of consciousness with associated complications such as loss of airway patency or aspiration of stomach contents into the lungs. Second, the physiological changes seen with breathing 5.5 % O_2 were predictable though nevertheless dramatic. The induction of a significant tachycardia at a time when blood oxygen carriage is extremely poor, as demonstrated in our data (Figure 5), effectively constitutes a myocardial stress test, and in an unscreened population there is an unknown, and possibly unacceptable risk of precipitating an ischaemic myocardial event or dysrhythmia.

LIMITATIONS

First, as in the only previous study of hypoxia training which provided blood oxygenation data,[10] measurement of blood oxygenation was with pulse oximetry. Pulse oximetry may become less accurate (though usually not grossly so) during severe hypoxia[11] and recordings in the study were not verified against a gold standard method such as arterial blood gas measurements (P_aO_2). However, the latter is invasive and, we would argue, unnecessary for the primary purpose of the study. The endpoint was functionally severe cognitive impairment rather than a specific P_aO_2 or S_pO_2. In addition, NIRS measurements are considered valid during hypoxia, and absolute values < 50% or a 20% fall from individual baseline values are commonly considered as intervention triggers in clinical practice.[12] The subjects often crossed either or both thresholds. We believe that it is valid to characterize these hypoxic exposures as severe.

Second, unlike attempts to demonstrate adaptation to cognitive impairment in other settings such as inert gas narcosis,[13] only the effect of a single exposure was evaluated. The possibility cannot be excluded that subjects might learn to cope better with hypoxia over a greater number of exposures. However, such a finding would be of doubtful practical value because training programmes with multiple exposures are unlikely to be considered acceptable.

Third, the definition of TUC used was the duration from switching to the hypoxic gas supply to the point where three consecutive card recognition errors were made. This included a very short initial period (< 10 sec) of clearance of non-hypoxic dead space gas from the gas supply tubing. Therefore, the true TUC during hypoxic gas breathing is correspondingly shorter. However, since the goal was to compare a measure of performance between two standardized sequential trials rather than to explicitly define a true hypoxic TUC, this small error is inconsequential.

Finally, as alluded to above, neither our study nor others conducted previously provide a definitive answer to the key question of whether prior hypoxic experience enhances the chances of effective self-rescue in a subsequent hypoxic event. Our conclusions in respect of the value of such experiences for rebreather divers must therefore be regarded as interim, pending the conduct of a definitive study.

Conclusions

A prior hypoxic experience did not improve cognitive performance or subject insight into performance in a second exposure five to six weeks later. Therefore, it is unlikely that cognitive acclimation or learning contributes to the ability to self-rescue during hypoxia, and any benefit of hypoxic training must lie solely in improved symptom recognition. However, at the present time there is no definitive proof that such training enhances self-rescue during hypoxia. With this in mind, we conclude that the potential risks associated with both hypoxic loss of consciousness and the physiologic changes that occur during hypoxia training are sufficiently concerning that we do not recommend such training for rebreather divers.

References

1 Cable GG. In-flight hypoxia incidents in military aircraft: causes and implications for training. Aviat Space Environ Med. 2003;74:169–72. PMID: 12602449.

2 Mitchell SJ, Doolette DJ. Recreational technical diving part 1. An introduction to technical diving. Diving Hyperb Med. 2013:43:86–93. PMID: 23813462.

3 Smith AM. Hypoxia symptoms in military aircrew: long term recall vs. acute experience in training. Aviat Space Environ Med. 2008:79;54–7. PMID: 18225780.

4 Woodrow AD, Webb JT, Wier JS. Recollection of hypoxia symptoms between training events. Aviat Space Environ Med. 2011;82:1143–7. PMID: 22195396.

5 Johnston BJ, Iremonger GS, Hunt S, Beattie E. Hypoxia training: symptom replication in experienced military aircrew. Aviat Space Environ Med. 2012;83:962–7. PMID: 23066618.

6 Island RT, Fraley EV. Analysis of USAF hypoxia incidents January 1976 through March 1990. In: Proceedings of the 31st Annual SAFE Symposium. Creswell, OR: SAFE Association; 1993. p.100–2.

7 Izraeli S, Avgar D, Glikson M, Shochat I, Glovinski Y, Ribak J. Determination of the "time of useful consciousness" (TUC) in repeated exposures to simulated altitude of 25,000 ft (7,620 m). Aviat Space Environ Med. 1988;59:1103–5. PMID: 3202796.

8 Sutton D. Dry hypoxia training for rebreather divers. Discussion thread on website CCR Explorers [Online]. [cited 2018 July 02]. Available from: https://www.ccrexplorers.com/community/threads/dry-hypoxia-training-for-rebreather-divers.14442/.

9 Klem GH, Lüders HO, Jasper HH, Elger C. The ten-twenty electrode system of the International Federation. The International Federation of Clinical Neurophysiology. Electroencephalogr Clin Neurophysiol Suppl. 1999;52:3–6. PMID: 10590970.

10 Singh B, Cable GG, Hampson GV, Pascoe GD, Corbet M, Smith A. Hypoxia awareness training for aircrew: a comparison of two techniques. Aviat Space Environ Med. 2010;81:857–63. PMID: 20824992.

11 Severinghaus JW, Naifeh KH, Koh SO. Errors of 14 pulse oximeters during profound hypoxia. J Clin Monit. 1989:5:72–81. PMID: 2723709.

12 Scheeren TWL, Shober P, Schwarte LA. Monitoring tissue oxygenation by near infrared spectroscopy (NIRS): background and current applications. J Clin Monit Comput. 2012;26:279–87. doi: 10.1007/s10877-012-9348-y. PMID: 22467064. PMCID: PMC3391360.

13 Rogers WH, Moeller G. Effect of brief repeated hyperbaric exposures on susceptibility to nitrogen narcosis. Undersea Biomed Res. 1989;16:227–32. PMID: 2741255.

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Conflicts of interest

Simon J Mitchell is Editor of *Diving and Hyperbaric Medicine*. He took no part in the peer-review and decision-making processes for this paper, which were managed entirely by the Associate Editor, Associate Professor F Michael Davis. There were no other conflicts of interest.

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