Original articles

Hypoxia signatures in closed-circuit rebreather divers

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Abstract
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Introduction: Faults or errors during use of closed-circuit rebreathers (CCRs) can cause hypoxia. Military aviators face a similar risk of hypoxia and undergo awareness training to determine their 'hypoxia signature', a personalised, reproducible set of symptoms. We aimed to establish a hypoxia signature among divers, and to investigate their ability to detect hypoxia and self-rescue while cognitively overloaded.
Methods: Eight CCR divers and 12 scuba divers underwent an initial unblinded hypoxia exposure followed by three trials; a second hypoxic trial and two normoxic trials in randomised order. Hypoxia was induced by breathing on a CCR with no oxygen supply. Subjects pedalled on a cycle ergometer while playing a neurocognitive computer game to simulate real world task loading. Subjects identified hypoxia symptoms by pointing to a board listing common hypoxia symptoms, and were instructed to perform a 'bailout' procedure to mimic self-rescue if they perceived hypoxia. Divers were prompted to bailout if peripheral oxygen saturation fell to 75%, or after six minutes during normoxic trials. Subsequently we interviewed subjects to determine their ability to distinguish hypoxia from normoxia.
Results: Ninety-five percent of subjects (19/20) showed agreement between unblinded and blinded hypoxia symptoms. Subjects correctly identified the gas mixture in 85% of the trials. During unblinded hypoxia, only 25% (5/20) of subjects performed unprompted bailout. Fifty-five percent of subjects (11/20) correctly performed the bailout but only when prompted, while 15% (3/20) were unable to bailout despite prompting. During blinded hypoxia 45% of subjects (9/20) performed the bailout unprompted while 15% (3/20) remained unable to bailout despite prompting.
Conclusions: Although our data support a normobaric hypoxia signature among both CCR and scuba divers under experimental conditions, most subjects were unable to recognise hypoxia in real time and perform a self-rescue unprompted, although this improved in the second hypoxia trial. These results do not support hypoxia exposure training for CCR divers.

Introduction

A closed-circuit rebreather (CCR) is a self-contained diving unit that allows a diver to recycle or conserve the oxygen in their exhaled breath while removing the carbon dioxide with a chemical scrubber. CCRs have proliferated over the past several years in part due to increased commercial availability and advantages for certain applications over open-circuit scuba. For recreational and scientific divers, CCRs offer the advantages of longer dive times, deeper dives, increased wildlife encounters, and the ability to explore more remote locations. CCRs also offer increased stealth by minimising exhaled bubbles which provides a distinct tactical advantage in military applications. CCR diving also carries an estimated mortality risk of approximately 10 times that of recreational scuba diving, with hypoxia as one of the leading causes of reported CCR diving injuries or fatalities.[1–3] One study reported that of the recreational CCR deaths between 1998 and 2010 with a known cause, 17% were due to hypoxia.[2]

Like CCR divers, aviators can experience hypoxia with potentially fatal consequences if their cockpit depressurises while in flight or their supplemental oxygen systems

fail. Among these aviators, acute hypobaric hypoxia can present with a variety of symptoms including psychomotor (incoordination, tremors), cognitive (concentration, confusion, memory loss), visual impairment (blurred vision, colour/light intensity changes), psychological (anxiety, depression, euphoria), dyspnoea, paraesthesia, headache, dizziness, tachycardia, and loss of consciousness.[4–6] Significant interpersonal variation in the order, severity, and speed of onset of hypobaric hypoxia symptoms occurs. Interestingly, the intrapersonal manifestation of hypobaric hypoxia symptoms on repeated exposures appears reproducible and serves as a 'hypoxia signature'. Most aircrew experience a high level of agreement between the dominant symptoms experienced during acute hypoxia and those they recall from previous hypoxia exposures (training or real events).[4,5,7,8] Among military aircrew, this forms the basis for hypoxia awareness training at fixed intervals (typically 3–6 years) in a hypobaric chamber.

To our knowledge, using hypoxia signatures to train divers to recognise their symptoms and perform a self-rescue bailout procedure has not been fully investigated. We sought to investigate these questions more thoroughly while mimicking diving conditions with concomitant exercise and mental distraction among groups of subjects who dive with CCRs or only scuba. We hypothesised the following:

1. During gradual onset hypoxia, the majority of cognitively distracted subjects will recognise their hypoxia signature and then perform a bailout procedure without any prompting or alarm when blinded to the breathing gas mixture.
2. Subjects trained as CCR divers will perform a bailout procedure without prompting significantly more often than subjects trained as scuba divers only.
3. Performance of a bailout procedure without prompting improves with a second exposure to gradual onset hypoxia while using a rebreather.

If hypoxia signature training were to prove effective, CCR divers may be able to decrease their risk of hypoxia-associated accidents and fatalities, increasing the safety of CCR diving.

Methods

The study protocol as approved by the institutional review board at the University of California, San Diego (Protocol #161414).

EXPERIMENTAL PROTOCOL

Using posted fliers at dive shops, and announcements at diving clubs and professional diving organisations, we recruited experienced, healthy male and female scuba and CCR divers, with ages between 18–60 years old. We obtained informed consent from all participants. We aimed to recruit 30 subjects split evenly between scuba and CCR divers, but we were only able to recruit 21 subjects before the COVID-19 pandemic began and delayed experimental trials indefinitely.

Subjects underwent a total of four experimental trials in a single day. The first trial was an unblinded trial of the experimental set-up with hypoxia. We induced gradual onset hypoxia by starting with a normoxic oxygen mix (air) and then shut off the addition of oxygen to the breathing loop, mimicking a real life CCR malfunction. Each subject gradually consumed the oxygen in the breathing loop, eventually leading to a hypoxic inhaled gas mixture. This trial served as an unblinded training trial where subjects experienced hypoxia in a safe, controlled environment supervised by practicing emergency medicine physicians with rescue airway equipment and supplemental oxygen immediately available.

We used a Scubaforce (Mönchengladbach, Germany) SF2 rebreather, regularly maintained and serviced, equipped with three Analytic Industries Model PSR 11-39-XD oxygen sensors (Pomona, CA, USA). Soda lime scrubber (Sofnolime 797, Molecular Products Inc., Louisville, CO, USA) was used to remove carbon dioxide (CO_2) from inhaled gas. Subjects breathed from a standard diving mouthpiece and used a nose clip. A gas analyser (MediPines AGM100 Innovative Respiratory Monitor, Yorba Linda, CA, USA) sampled CO_2 and oxygen (O_2) levels from a port drilled into the CCR mouthpiece. After the unblinded hypoxia trial, subjects performed three additional trials; two normoxic control trials and one hypoxic experimental trial. In normoxic trials the fraction of O_2 in the rebreather loop was maintained at 21% by the investigators. In hypoxia trials the O_2 supply to the CCR was isolated. The order of the three additional trials was randomised, and subjects were blinded to the gas they were breathing (normoxic vs hypoxic). All trials ended when the subjects desaturated to 75%, six minutes elapsed (for normoxic trials), or if the subjects felt that they were experiencing an emergency and performed the self-rescue protocol. The self-rescue protocol (a 'bailout') required the subjects to turn a ball valve by pulling on a lever, simulating switching to a bailout gas on a CCR. If the subjects desaturated to 75% (hypoxic trials) or 6 minutes elapsed (normoxic trials), investigators prompted the subjects to bailout with a written sign.

After performing the bailout, investigators removed the mouthpiece and allowed the subjects to recover at least 10 minutes between trials. If subjects failed to perform the bailout, investigators rapidly removed the mouthpiece to prevent loss of consciousness or motor control. This decision was based on investigators' clinical assessment of each subject's reaction to the written prompt to bailout. For example, if a subject's eyes were not moving to read the written bailout sign or the subject made no purposeful hand movement, investigators immediately removed the mouthpiece and encouraged the subject to take deep breaths of room air. No subject lost consciousness.

Figure 1

Study protocol; CCR – closed circuit rebreather; $EtCO_2$ – end tidal carbon dioxide; EtO_2 – end tidal oxygen; SpO_2 – peripheral oxygen saturation

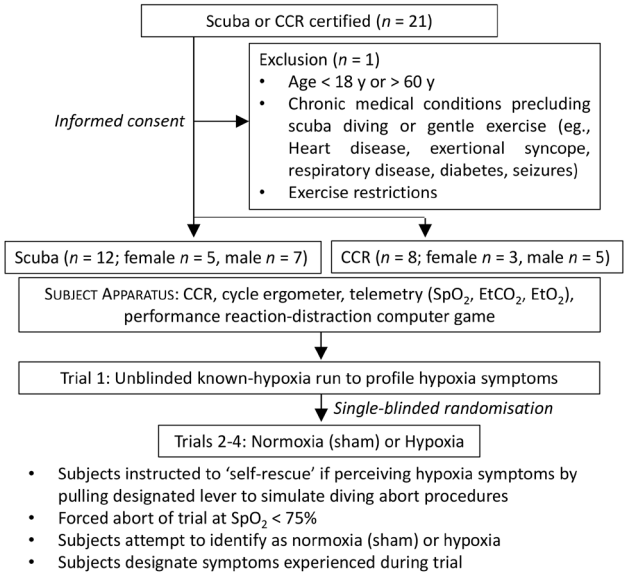
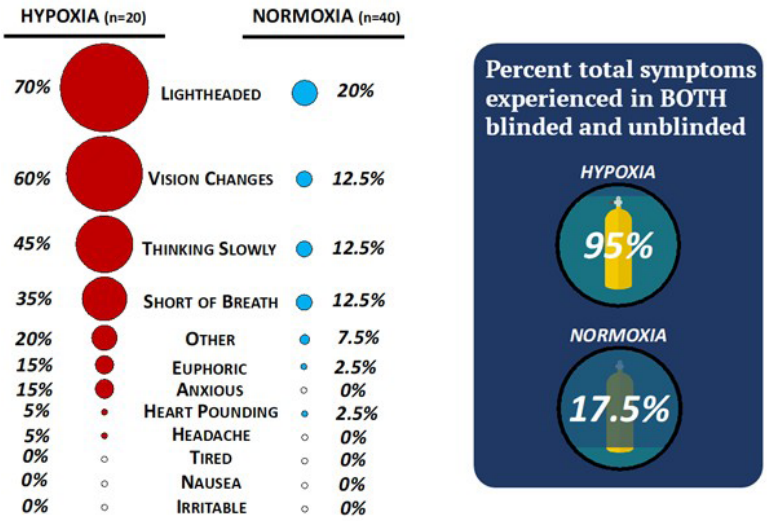


Figure 2

Reported hypoxia symptoms in descending order of frequency of occurrence at left. Percentage of blinded trials with recurrence of the symptoms reported during the unblinded hypoxia trial at right



During each trial, subjects pedaled a cycle ergometer set to 5W to simulate the attention needed for underwater finning without producing a large increase in metabolic rate. They were monitored with a finger pulse oximeter. While pedaling, each subject also played a distracting computer-based neurocognitive test ('Go/No-Go', Automated Neuropsychological Assessment Metrics (ANAM), Vista LifeSciences Inc., Parker, CO, USA) to simulate cognitive task loading underwater. Investigators also instructed the subjects to point to symptoms they were experiencing on a board listing common hypoxia symptoms. After each trial finished, investigators interviewed subjects to determine their perception of which gas mix they breathed (normoxic vs hypoxic), their recall of symptoms, and how the blinded trial compared to the unblinded hypoxia trial. The experimental protocol is summarised in Figure 1.

OUTCOMES AND ANALYSES

The presence of a hypoxia signature under the gradual onset hypoxia condition was investigated. We compared each subject's symptoms which they identified in real-time between their unblinded and blinded hypoxia trials. We reported the frequency of these symptoms as well as

Figure 3
Blinded gas identification among scuba and closed circuit rebreather (CCR) divers after completion of each experimental trial

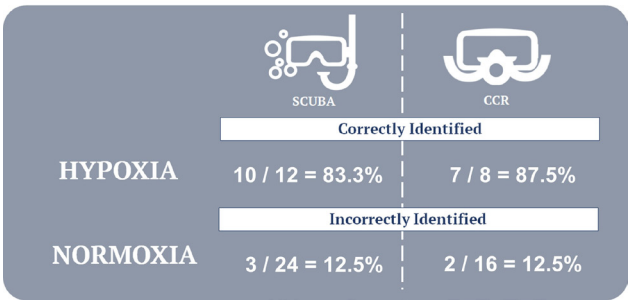
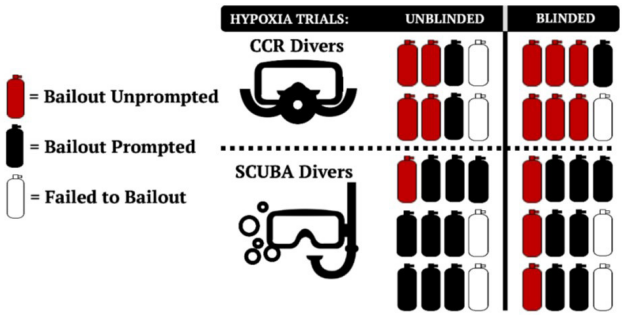


Figure 4
Performance of the bailout procedure with and without prompting during both hypoxia trials. Prompting occurred only when oxygen saturation reached 75%; CCR – closed circuit rebreather



the percentage of blinded hypoxia trials where subjects reported the same symptoms that they experienced during their unblinded hypoxia trial.

The subjects' ability to identify their breathing gas mixture (normoxia vs hypoxia) in interviews after the blinded experimental trials was recorded. We also compared the percentage of correct and incorrect identification of hypoxic and normoxic gases between CCR trained divers and scuba divers.

The subjects' ability to recognise their hypoxia signature symptoms and then perform the bailout procedure without any external prompting or alarm was measured. We reported the number of subjects who performed the bailout without prompting, those who required prompting and performed the bailout afterward, and those who were unable to perform the bailout despite prompting. As part of the analysis for these data, we performed the following:

- A comparison of the CCR trained divers with scuba divers in hypoxia signature recognition and then bailout without any prompting or alarm.
- A comparison of oxygen saturations at the time of bailout between subjects who correctly performed the bailout without prompting and those who required prompting or were unable to perform the bailout. We performed two-sided *t*-tests comparing the saturations of those who performed the unprompted bailout and those who did not (either required prompting to bailout or were unable to bailout) with significance defined as $P < 0.05$.

Lastly, the existence of a training effect with repeated exposure to hypoxia in a single day was investigated by comparing the number of subjects who correctly performed the bailout procedure during the blinded experimental hypoxic trial versus during the unblinded initial introductory hypoxic trial.

Results

We recruited 21 subjects and excluded one due to age. All 20 subjects included in the study completed all trials and none suffered any complication such as loss of consciousness. The CCR group (n = 8) consisted of five males and three females while the scuba group (n = 12) consisted of seven males and five females. Neither group contained subjects who reported any chronic medical condition or prior hypoxia training, and no subject in either group reported a history of decompression illness.

During the experimental trials, the most commonly reported symptoms, regardless of gas mixture, were lightheadedness, vision changes, thinking slowly, and shortness of breath (Figure 2). Following blinded hypoxia trials, nearly all subjects (19/20, 95%) reported recurrence of symptoms experienced during unblinded hypoxia trials. The one subject who did not report the recurrence was a scuba diver. Among the blinded normoxia control trials, some subjects reported similar symptoms compared to the earlier unblinded hypoxia trial in 7/40 trials (17.5%, seven unique subjects) (Figure 2).

During the debriefing interview following each blinded trial, subjects correctly identified the gas mixture in 51/60 (85%) trials. Of those 60 blinded trials, subjects correctly identified 17/20 (85%) hypoxia trials and 34/40 (85%) normoxia trials (Figure 3). Grouped according to their diving history, 10/12 (83.3%) scuba divers correctly identified the blinded hypoxia trial, and 7/8 (87.5%) CCR divers also correctly identified the blinded hypoxia trial. Scuba divers incorrectly identified normoxia as hypoxia in 3/24 trials (12.5%, three unique subjects), while CCR divers incorrectly identified 2/16 normoxia trials as hypoxia (12.5%, two unique subjects).

Among all divers during the unblinded hypoxia trial, only 5/20 (25%) subjects performed the bailout unprompted based on the perception of hypoxia symptoms, and four of these subjects were CCR divers (Figure 4). Of the remaining subjects, 11/20 (55%) correctly performed the bailout only when prompted, while 3/20 (15%) were unable to bailout despite prompting. One CCR diver performed the bailout procedure unprompted but incorrectly during their unblinded hypoxia trial. For subjects who correctly performed the unprompted bailout during the unblinded hypoxia trial, the peripheral oxygen saturation (SpO_2) averaged 80% (SD 5.2),

at the time of bailout (81.3% (5.12) for CCR vs 75% for the 1 scuba subject). This mean SpO_2 of 80% (5.2) was not significantly different than the SpO_2 = 75% endpoint we used in our protocol for those subjects who required prompting to bailout or were unable to bailout (*t*-test, $n = 5$, $P = 0.10$).

In the blinded hypoxia trial, 17/20 (85%) subjects performed the bailout procedure, but only 9/20 (45%) subjects did so unprompted (Figure 4). This represented an increase of four subjects in comparison with the unblinded hypoxia trial where only 5/20 (25%) subjects correctly performed the bailout procedure unprompted. One scuba diver stated in their post-trial interview that they were aware of the need to bailout but continued the trial and then forgot to pull the lever. Of the three subjects who failed to perform the bailout procedure despite prompting, only one was a CCR diver. Among the CCR subjects, 6/8 (75%) correctly performed the bailout procedure unprompted during the blinded hypoxia trial. They represented 6/9 (66.7%) subjects that correctly performed the bailout procedure unprompted and based solely on their recognition of symptoms during the blinded hypoxia trial. Seventy-five percent of CCR subjects correctly performed the bailout procedure unprompted compared to 3/12 (25%) scuba diver subjects. During the blinded hypoxia trial, the average SpO_2 of the subjects who correctly performed the unprompted bailout was 78.6% (SD 4.1) at the time of bailout (78.8% (4.4) for CCR vs 78% (4.0) for scuba). The SpO_2 for these subjects who correctly performed the unprompted bailout during the blinded hypoxia trial was 78.6% (4.1); significantly different to the 75% endpoint we used in our protocol for those subjects who required prompting to bailout or were unable to bailout (*t*-test, $n = 9$, $P = 0.03$).

During the blinded normoxia trials that served as sham controls, subjects performed the bailout procedure unnecessarily in 3/40 trials (two unique subjects), misidentifying normoxia as hypoxia. None of these trials involved CCR subjects. These subjects, believing themselves to be dangerously hypoxic, had an average SpO_2 of 94.3% (3.3). The typical reason subjects gave in the post-trial interview was that they performed the bailout procedure due to perceived changes in the breathing resistance of the experimental CCR that we attributed to the counter-lung. The remaining subjects terminated their trials, after 6 minutes according to our protocol, with an average SpO_2 of 97.6% (1.5), consistent with normoxia.

Additionally, we measured end tidal CO_2 throughout the trials to demonstrate isocapnia. CO_2 levels in subjects undergoing the hypoxia trials averaged 37.94 (3.4) mmHg, while those undergoing the normoxia trials averaged 39.2 (2.8) mmHg.

Discussion

Hypoxia can have an insidious and deleterious effect on CCR divers, sapping them of both motor function and cognitive ability. The development of hypoxia while using a CCR is particularly dangerous since the onset of symptoms is gradual as the diver consumes the available oxygen in the breathing circuit. Divers may overlook subtle symptoms of hypoxia due to a lack of awareness leading to a lack of problem recognition and failure to correct the problem. This may be compounded by underwater tasks or nitrogen narcosis.[1,9] Even if the hypoxia is recognised by the diver, as symptoms progress, the diver may quickly be incapacitated and unable to correct it. If left uncorrected, hypoxia will lead to a rapid loss of consciousness under water and subsequent drowning or death.

A potentially fatal hypoxic breathing loop may result from numerous causes such as the breathing mixture becoming hypoxic by over-dilution with hypoxic diluent gas, failure of the fuel cells to sense hypoxic gas levels, mechanical failure of the solenoid valve controlling the gas mixture, forgetting to open the oxygen tank valve or turn on the electronics prior to diving, and improper diluent gas selection.[1,10–12] In a CCR, oxygen is mixed with a diluent gas to maintain a constant partial pressure of inhaled oxygen (PO_2) regardless of depth. In order to maintain a constant PO_2, galvanic fuel cells measure the breathing loop PO_2, which is reported to the diver on their display. Unfortunately, these fuel cells have a finite lifespan, and failure may be difficult to predict. Due to this fact, most CCRs utilise three cells to measure PO_2, which are interpreted by the computer's algorithm. Many of these algorithms use voting logic where the computer averages the three cells' PO_2 readings, unless one of the three cells varies significantly in which case the computer ignores it. In an electronic CCR as the PO_2 drops below a set point defined by the diver, oxygen is added into the loop through the opening of an electronic solenoid valve. Alternately, if the PO_2 becomes elevated, most CCR models will not add diluent but rather wait for the diver's metabolism to consume the excess oxygen. In a CCR without electronic controls, the diver must perform these gas changes manually. CCR systems are typically set up to include an alarm to prompt the diver to look at their display if the PO_2 varies from the set point.[11,13]

With this study, we aimed to determine if subjects had a reproducible set of symptoms, the 'hypoxia signature', during gradual onset hypoxia as well as the ability to detect hypoxia during a simulated dive and then perform a self-rescue bailout procedure. With 95% agreement between unblinded and blinded hypoxia trials, the data support the presence of hypoxia signatures under our experimental conditions. The subjects also exhibited isocapnia with measured end tidal CO_2 levels all within normal limits across all subjects and trials.

A recent study reported a cohort of subjects exposed to hypoxia twice, approximately five weeks apart, and found no differences between the severity of various hypoxia symptoms during each trial using a visual analog scale (VAS) in interviews five minutes after the hypoxia exposure.[14]

These results support the idea of a hypoxia signature. However, the comparisons made in the VAS score were made between trials five weeks apart with the subjects grouped together versus a comparison between VAS scores on an individual subject basis. Furthermore, the purpose of that study was to determine if hypoxia training could affect the time of useful cognitive function as well as to characterise physiological parameters in the subjects who breathed a hypoxic gas mixture of 5.5% O_2 while performing a card recognition protocol. Our study provides additional insights into the presence of a hypoxia signature in divers and begins to examine its usefulness as a potential training mechanism. Our protocol consisted of a more gradual hypoxic stress where each subject breathed down the CCR loop from room air to a hypoxic concentration all while maintaining isocapnia. This approach mimics the insidious, real-world scenario where a CCR malfunctions and fails to add additional oxygen into the breathing loop. This gradual onset also distinguishes hypoxia in CCR diving from hypoxia seen in aviation which is typically more abrupt in onset in both reality and training.

Additionally, our study sought to examine if a diver could not only recognise their hypoxia signature but perform self-rescue. Recognition of the hypoxia signature provides no safety benefit if the diver is unable or unwilling to perform self-rescue by a bailout procedure. We had hypothesised that with its more gradual onset, our hypoxia protocol would allow the subjects more time for recognition of symptoms and then more time for corrective action, leading to a majority of subjects able to perform the bailout procedure unprompted. However, our results do not support this. While the large majority of subjects correctly differentiated the blinded hypoxia trial from sham trials during debriefing interviews, 55% of all subjects still did not bailout unprompted during the blinded hypoxia trial, contrary to what we expected. We anticipate that these subjects would have had serious adverse effects or died under analogous diving conditions. In fact, we suspect that our findings overestimate divers' ability to bailout due to the artificial nature of a laboratory setting and that our subjects knew we were studying hypoxia. This is an alarming finding given that current CCR equipment may not effectively alert the diver if multiple oxygen sensors fail. This failure is distinct from improper calibration. Dive time, humidity, high temperature, and life cycle can produce inaccurate millivolt potentials in oxygen sensors, which can lead to a 'false high' partial pressure calculation. Thus, oxygen is not injected, and hypoxia can result despite 'normal' readings. This has significant implications for checklist development and implementation.

Furthermore, among those subjects who performed the bailout unprompted, the SpO_2 levels were still quite low, even though the SpO_2 levels for the blinded hypoxia trial were significantly elevated in comparison to those of subjects who did not perform the bailout without prompting. These levels correspond to the steep portion of the oxyhaemoglobin dissociation curve, where small decreases in the partial pressure of O_2 correspond to large decreases in SpO_2, indicating a narrow time frame in which a subject could correct their hypoxia before becoming incapacitated. We predict that such low oxygen saturations would lead to cognitive deficits and impaired divers, risking both their lives and the lives of their dive buddies under real world conditions.

When analysing the two subject groups, the CCR divers outperformed scuba divers at correctly performing the bailout procedure unprompted during the blinded hypoxia trial (75% vs 25%). The improved performance by the CCR divers would seem to support our initial hypotheses regarding the gradual onset of hypoxia allowing for increased recognition and increased bailout but the small number of subjects in the CCR group may represent a sampling bias that is not generalisable to the greater population of CCR divers. The improved bailout performance in this group may be due to increased familiarity with the experimental equipment, increased awareness of their responses to breathing from an external device, increased ability to manage cognitive distractions, or some other effect from their dive training or other prior experience.

Comparing the unblinded hypoxia trial and the blinded hypoxia trial, we observed a nearly two-fold increase in the number of subjects that recognised their hypoxia signatures and performed the bailout unprompted. This increase may represent a training effect stemming from the first, unblinded trial which many of the subjects were keenly interested in experiencing. Aerospace researchers have shown that individuals without hypoxia awareness training are unlikely to recognise these symptoms and appreciate their insidious onset. One study reviewed 656 incidents of in-flight hypoxia within the US Air Force from 1976 to 1990 and found a large difference in the number of aircrew who experienced a loss of consciousness based on whether or not they had received hypoxia training, suggesting a beneficial hypoxia training effect.[15] This retrospective review however did not formally test the efficacy of the training protocol, and other differences between the two groups could explain the observation.

Although a training effect is certainly possible among the divers in our study, our experimental protocol did not test for this effect and other factors may explain the increase in subjects performing the bailout unprompted. For example, during their debriefing interviews, many of the subjects reported that they suppressed the desire to perform the bailout procedure during our unblinded hypoxia trial. They wanted to deliberately push their physiological limits to experience as profound a level of hypoxia as they could. Since the unblinded hypoxia trial served as a baseline of the performance of the bailout without prompting, this desire to push physiological limits artificially worsened the subjects' baseline and may account for the difference in performance between the unblinded and blinded hypoxia trials, negating

any evidence of a true training effect. Other possible factors such as a better understanding of the experimental protocol with repeated trials, increased familiarity with the experimental equipment, or a combination of multiple factors could explain the improvement we observed between hypoxia trials. Importantly, we do not feel that our results support a single day of hypoxia exposure training for CCR divers or that this protocol will benefit their ability to perform self-rescue. Whether a training benefit will occur during a repeat bout of testing of the ability to recognise one's hypoxia signature and then perform the bailout procedure without prompting remains unanswered. Mitchell et al recently took the first steps investigating these problems, finding that a "*hypoxic experience did not improve cognitive performance or subject insight into performance in a second exposure five weeks later*".[14]

Nonetheless, hypoxia exposure during CCR training may still be useful in demonstrating to the CCR diver the insidious and life-threatening danger of the condition. If a diver can recognise their hypoxia signature but cannot perform a bailout self-rescue, the diver should dedicate their efforts to reduce the likelihood of developing hypoxia in the first place. Furthermore, our finding that 3/20 (15%) subjects were unable to perform the simple self-rescue intervention during the blinded hypoxia trial, despite receiving a written command to bailout upon reaching 75% oxygen saturation, is alarming. This underscores the paramount importance of preventing hypoxia as well as the need for detection and prompting to bailout at a much less severe degree of hypoxia. Efforts to decrease the risk of hypoxia include properly maintaining gear, formulating clear dive plans, adhering to the buddy system, and using robust pre-dive checklists.

Future efforts will aim to repeat these trials in the same subjects after one year or more to determine if this first set of trials has provided a training benefit and whether the hypoxia signature remains reproducible. Additionally, we hope to perform this experimental protocol under hyperbaric conditions to investigate whether a normobaric hypoxia signature is reproducible and can act as a surrogate for the underwater environment.

LIMITATIONS

This study was performed in a laboratory under normobaric conditions and may not completely mimic real world diving activity. The subjects' upright posture, lack of a wet suit or dry suit, lack of face mask, and absence of thermal stressors all may have effects in actual dives for which we did not account in this study. Real world scenarios likely will induce even worse performance than what we observed in our study. The monitoring we performed using a fingertip pulse oximeter, furthermore, represents a delayed measure of tissue hypoxia, particularly in the brain. Fingertip pulse oximeters can additionally be negatively affected by factors such as vasoconstriction, fingernail polish, and skin tone. In addition, we were unable to complete our original plan for 30 subjects due to the COVID-19 pandemic to ensure sufficient diversity, especially regarding gender among our subject groups. Nonetheless, the data presented here are rather provocative with a clear difference in the performance of the unprompted bailout by CCR divers versus scuba divers as well as in the performance improvement between the unblinded and blinded trials. Thus, we suspect an additional five subjects in each group would not significantly change the study conclusions regarding our hypotheses.

Conclusions

Although our data support a normobaric hypoxia signature among divers under our experimental conditions, 55% of our diver subjects (11/20) were unable to recognise hypoxia in real time and perform self-rescue only hours after an unblinded demonstration of hypoxia symptoms. Further study is needed to determine the intrapersonal reproducibility of the hypoxia signature over time and under hyperbaric conditions. Additionally, further investigation is required to determine the ability to train the recognition of one's hypoxia signature and, in turn, if that recognition can lead to higher self-rescue rates among divers.

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