Safety proposals for freediving time limits should consider the metabolic-rate dependence of oxygen stores depletion

Charlotte Sadler[1], Kaighley Brett[1], Aaron Heerboth[1], Austin R Swisher[2], Nader Mehregani[2], Ross Touriel[1], Daniel T Cannon[2]

[1] *Department of Emergency Medicine, University of California, San Diego, USA*
[2] *School of Exercise and Nutritional Sciences, San Diego State University, San Diego, USA*

Corresponding author: *Dr Charlotte Sadler, Department of Emergency Medicine, University of California, San Diego, USA*
csadler@ucsd.edu

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Abstract
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Introduction: There is no required training for breath-hold diving, making dissemination of safety protocols difficult. A recommended breath-hold dive time limit of 60 s was proposed for amateur divers. However, this does not consider the metabolic-rate dependence of oxygen stores depletion. We aimed to measure the effect of apnoea time and metabolic rate on arterial and tissue oxygenation.
Methods: Fifty healthy participants (23 (SD 3) y, 22 women) completed four periods of apnoea for 60 s (or to tolerable limit) during rest and cycle ergometry at 20, 40, and 60 W. Apnoea was initiated after hyperventilation to achieve $P_{ET}CO_2$ of approximately 25 mmHg. Pulse oximetry, frontal lobe oxygenation, and pulmonary gas exchange were measured throughout. We defined hypoxia as $SpO_2 < 88\%$.
Results: Static and exercise (20, 40, 60 W) breath-hold break times were 57 (SD 7), 50 (11), 48 (11), and 46 (11) s ($F_{[2.432, 119.2]} = 32.0$, $P < 0.01$). The rise in $P_{ET}CO_2$ from initiation to breaking of apnoea was dependent on metabolic rate (time × metabolic rate interaction; $F_{[3,147]} = 38.6$, $P < 0.0001$). The same was true for the fall in SpO_2 ($F_{[3,147]} = 2.9$, $P = 0.03$). SpO_2 fell to < 88% on 14 occasions in eight participants, all of whom were asymptomatic.
Conclusions: Independent of the added complexities of a fall in ambient pressure on ascent, the effect of apnoea time on hypoxia depends on the metabolic rate and is highly variable among individuals. Therefore, we contend that a universally recommended time limit for breath-hold diving or swimming is not useful to guarantee safety.

Introduction

Breath-hold diving, also known as freediving, is a popular recreational and competitive sport. Freediving is all diving done between two breaths of air and without the use of any breathing apparatus or external gas supply.[1] In addition to a stand-alone competitive sport, breath-hold diving is also done as a part of snorkelling, spearfishing, and swimming. Breath-hold divers may compete in various disciplines, including maximum time and depth. Outside of recreational and competitive sport, breath-hold diving is common across all ages in a variety of leisure time activities taking place in swimming pools, lakes, and oceans.

Loss of consciousness and death from hypoxia is a concern during breath-hold diving and swimming and occurs due to low brain tissue oxygen partial pressure (PO_2). At depth, the increased hydrostatic pressure results in an elevated alveolar PO_2 sufficient to maintain consciousness, at least for a limited period of time. However, during ascent the alveolar PO_2 falls with the reduction in hydrostatic pressure in addition to the reduction from normal O_2 consumption. The arterial PO_2 follows suit and unconsciousness may result prior to or just after reaching the surface, which may result in drowning. This is compounded by depletion of O_2 stores due to muscular activity and suppression of the drive to breathe due to low arterial partial pressure of carbon dioxide (PCO_2) following hyperventilation. Hypoxic syncope may occur even in very shallow breath holds, such as in a pool.

Unlike scuba diving, which is regulated and requires training and certification for participation, there is no such prerequisite for participation in breath-hold diving. Although there are many organisations that provide courses and certifications in breath-hold diving, they are not mandatory. As a consequence, it is difficult to disseminate safety rules or protocols to the public, unless they choose to take a formal breath-hold diving class. Thus, any rule that is proposed must be fairly simple to disseminate and implement. At a joint meeting of the Undersea and Hyperbaric Medical Society (UHMS) and the Divers Alert Network (DAN) in 2006, one presenter proposed to set a limit of 60 seconds (s) duration as a safety measure for amateur breath-hold diving.[2]

Although this was not officially adopted or recommended by the aforementioned organisations, we wanted to further investigate this proposition, as a simple, widely applicable safety rule could potentially be very useful. However, a fixed time limit recommendation likely cannot account for variable metabolic rate and oxygen store depletion. For example, hypoxia may not set in during 120 s of static apnoea, yet < 40 s of apnoeic exercise may be sufficient to elicit dangerous hypoxia.[3–5] We aimed to measure the effect of work rate on arterial and tissue oxygenation during apnoeic exercise. We hypothesised that the effects of apnoea on arterial and tissue oxygenation are dependent on metabolic rate.

Methods

The protocol was approved by the Institutional Review Boards at San Diego State University and University of California, San Diego and complied with the Declaration of Helsinki.

PARTICIPANTS

Fifty healthy volunteers were recruited: 22 women; age 23 (SD 2) years (y); body mass index (BMI) 23.5 (2.8) $kg \cdot m^{-2}$; height 163 (7) cm; weight 62.0 (9.1) kg, and 28 men: 23 (3) y; 23.9 (3.9) $kg \cdot m^{-2}$; 179 (8) cm; 76.2 (13.7) kg. Inclusion criteria included age 18–30 y, non-pregnant, non-smokers and ability to participate in physical activity. Exclusion criteria included a history of heart disease, syncope, asthma, vertigo, or exertional chest pain or shortness of breath. Volunteers provided written informed consent and were screened for cardiovascular risks with the Physical Activity Readiness Questionnaire (PAR-Q) prior to beginning any physical activity. A brief intake form was also filled out by the participants to collect information on their prior dive experience and exercise habits. Of the 50 volunteers, one participant had previous free dive experience. Freediving was not formally defined, but instead the responses reflected the participants' own opinion of their activities. However, it was likely that most had engaged in breath-hold swimming and diving during recreational activities. Forty-eight out of 50 participants exercised at least once a week and reported 3.2 (SD 1.6) sessions of exercise per week. There were no differences between the sexes in age, BMI, or reported frequency of exercise sessions per week.

PROTOCOL

Volunteers completed four periods of apnoea for 60 s (or to their tolerable limit if < 60 s) sequentially during seated rest and cycle ergometry at 20, 40, and 60 W (Excalibur Sport PFM, Lode BV, Groningen, NL). Each apnoea was preceded by hyperventilation and subjects were instructed to begin breath-holding when they achieved an end tidal PCO_2 ($P_{ET}CO_2$) of 25 mmHg. This practice is commonly used to extend apnoea duration and was done in an attempt to mimic real world conditions, as well as increase the likelihood that the participants would be able to complete the full 60 s of apnoea. At the initiation of the apnoeic period, participants were instructed to complete a near maximal inspiration and also encouraged to close their glottis. They had been instructed on this technique during the orientation to the experiment. The volunteers were fitted with a nose-clip and remained on the mouthpiece (80 mL dead space) for the duration of the apnoea. At 60 s, or at the limit of tolerance, the participants were encouraged to perform a deep expiration nearly to residual volume as this aided in a confident single-breath measurement of the end tidal gas fractions. Each trial (including the static trial) was preceded and separated by a 3 min period of cycling at 20 W while breathing normally, which we considered active recovery. Pulse oximetry, frontal lobe oxygenation, and pulmonary gas exchange were measured throughout the hyperventilation and apnoeic periods. Based on clinical experience, and for the sake of safety, we chose to define hypoxia as a peripheral oxygen saturation (SpO_2) of < 88% and trials were stopped if SpO_2 dropped below this value. The mouthpiece was removed during the rest periods for comfort.

MEASUREMENTS

Respired gases and ventilation were measured breath-by-breath with a commercial metabolic measurement system (VMAX Encore, Vyaire, Yorba Linda, CA, USA). The system was calibrated immediately prior to each experiment. A 3 L syringe (Hans Rudolph Inc., Shawnee, KS, USA) was used to calibrate the mass flow sensor from ~0.2 to 8.0 $L \cdot s^{-1}$, mimicking flow rates expected at rest and during exercise. The CO_2 and O_2 analysers were calibrated using gases of known concentrations (O_2 26.0 and 16.0%; CO_2 0.0 and 4.0%). Cardiac function was monitored continuously using a 12-lead ECG (GE Cardiosoft, GE Healthcare, Chicago, IL, USA) and a pulse oximeter to measure SpO_2 was applied to the middle or ring finger (Nonin Medica Inc, Plymouth, MN, USA). While SpO_2 was monitored throughout the apnoea, the baseline and nadir of SpO_2 was used for analysis.

NEAR-INFRARED SPECTROSCOPY

Near-infrared spectroscopy (NIRS) was used to measure frontal lobe oxygenation during static and exercising apnoea. The measurement technique relies on the known absorption characteristics of oxygenated haemoglobin (HbO_2) and deoxygenated haemoglobin (HHb) when NIR light is directed into tissue.[6,7] The NIRS device (NIRO-200, Hamamatsu Photonics KK, Hamamatsu, Japan) consisted of a laser diode light source, and a photodiode to detect the returned NIR light after passing through the tissue under interrogation. The NIRS probe (consisting of one fiber optic emission optode and two detection optodes) was secured over the frontal lobe with care to avoid the sinus cavities. The probe was affixed using double-sided adhesive tape and an elastic bandage to minimise ambient light contamination and movement of the probes. The

probes were enclosed in a black rubber housing with a fixed emission/detection distance (detectors were 4 and 5 cm from the emitter) for measurement of the relative absorbance of each chromophore by spatially resolved spectroscopy (SRS). Source light was provided at three wavelengths (775, 810 and 850 nm) and detection sampled at 2 Hz to calculate the tissue oxygenation index (TOI). The SRS method is thought to allow signal loss, due to light scatter, to be better accounted for in calculation of TOI ($[HbO_2]/[HbO_2+HHb]$, expressed as a percentage) and the signal is therefore proportional to chromophore concentration.[6,7]

STATISTICAL ANALYSES

A two-factor repeated measures analysis of variance (ANOVA) was used to test the effect of apnoea duration (time, 2 levels: pre/post apnoea) and metabolic rate (condition, 4 levels: 0, 20, 40, 60 W). A one-factor repeated measures ANOVA was used to test difference in apnoea duration across the static and exercise conditions. Bonferroni-corrected *post hoc t*-tests were used in case of a main effect in the omnibus test. All data were analysed using the Prism v7, GraphPad (GraphPad Software Inc., San Diego, CA, USA). Results are reported as mean (SD).

Results

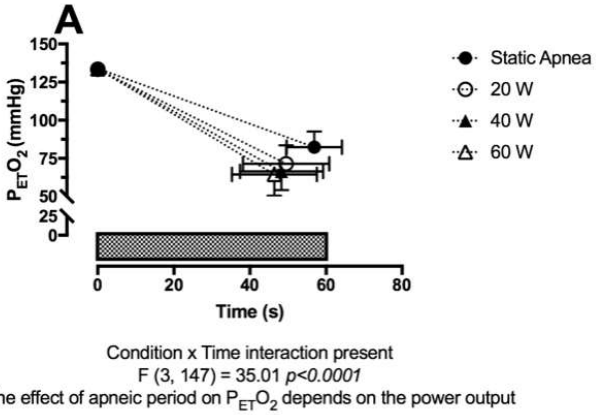
All of the participants completed the protocol, though not every participant was able to complete a full 60 s of apnoea. Static and exercise (20, 40, and 60 W) breath-hold break times were 57 (7), 50 (11), 48 (11), and 46 (11) s ($F [2.432, 119.2] = 32.0$, $P < 0.01$). Mean $P_{ET}CO_2$ at the initiation of apnoea was identical across the four conditions (all at 23 [2] mmHg). No trials were discontinued due to $SpO_2 < 88\%$, and all of the apnoeas < 60 s were ended voluntarily by the participant.

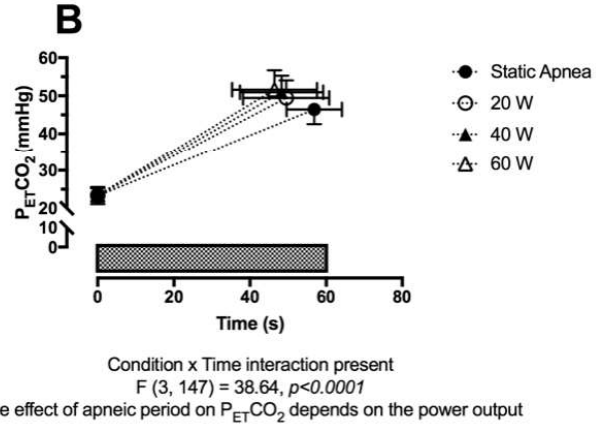
The effect of apnoea time on end tidal PO_2 ($P_{ET}O_2$), $P_{ET}CO_2$, SpO_2, and heart rate was dependent upon the power output (Figure 1A–C, $P_{ET}O_2$ $F [3,147] = 35.01$, $P < 0.0001$, $P_{ET}CO_2$ $F [3,147] = 38.6$, $P < 0.0001$, HR $F [3,147] = 9.6$, $P < 0.0001$; Figure 2A, SpO_2 $F [3,147] = 6.36$, $P = 0.0004$). Frontal lobe TOI was not affected during the apnoeic period (Figure 2B).

In total, there were 14 episodes of hypoxia (defined as $SpO_2 \leq 88\%$): six in the 20 W trial; five in the 40 W trial; and three in the 60 W trial. No episodes of hypoxia were recorded during the static apnoea. These 14 episodes occurred in eight participants. No oxygen saturations ≤ 88% were observed during the apnoea, rather they occurred in the 30 s period after the apnoea ended. The lowest oxygen saturation observed was 82%. The lowest saturation values occurring after completion of apnoea is due to transit delay to capillary bed in the finger. These nadir SpO_2 values are what appear in the analysis and in Figure 2A. Similar phenomena were present in other breath-hold studies.[4] No participants experienced loss of consciousness or any neurologic signs

Figure 1

Gas exchange and heart rate during static and exercising apnoea. Shaded rectangle along the x-axis represents the target breath-hold duration of 60 s. Panel A: $P_{ET}O_2$ measured immediately prior to, and at the first expiration following apnoea. A condition x time interaction was present ($F [3,147] = 35.01$, $P < 0.0001$), thus the effect of apnoeic time on $P_{ET}O_2$ depended on the power output. Panel B: $P_{ET}CO_2$ measured immediately prior to, and at the first expiration following apnoea. A condition x time interaction was present ($F [3,147] = 38.6$, $P < 0.0001$), thus the effect of apnoeic time on $P_{ET}CO_2$ depended on the power output. Panel C: Heart rate (HR) measured at initiation and completion of the apnoeic period. A condition x time interaction was present ($F [3,147] = 9.6$, $P < 0.0001$), thus the effect of apnoeic time on heart rate depended on the power output





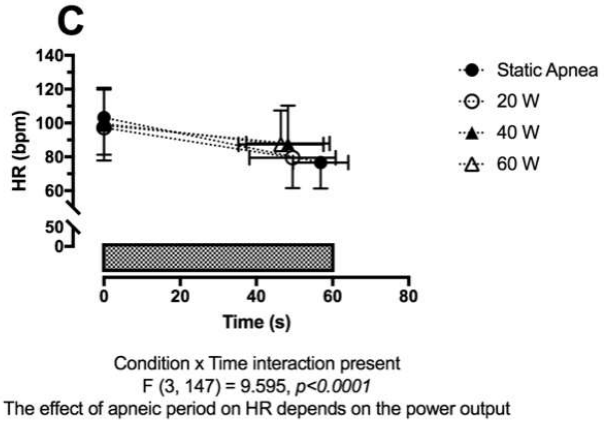
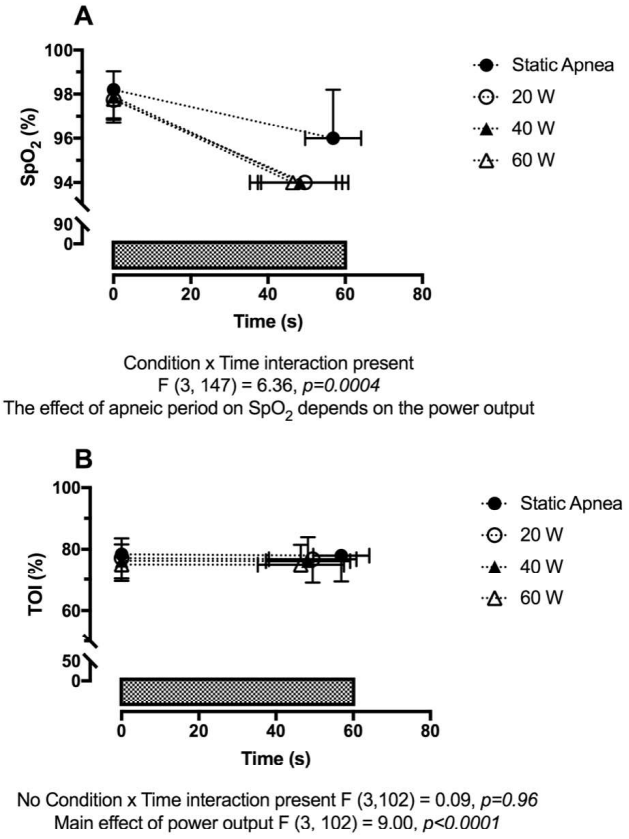


Figure 2
Pulse oximetry and near-infrared spectroscopy data during static and exercising apnoea. Shaded rectangle along the x-axis represents the target breath-hold duration of 60 s. Panel A: SpO_2 at initiation and SpO_2 following the apnoeic period. A condition x time interaction was present (F [3,147] = 6.4, P = 0.0004), thus the effect of apnoeic time on S_pO_2 depended on the power output. Panel B: Tissue oxygenation index (TOI), as measured with near-infrared spectroscopy, was not affected by apnea duration (F [1,34] = 0.7, P = 0.4)



or symptoms and the only subjective symptoms reported were presyncope ('seeing stars' as described by one subject).

Discussion

The study aimed to measure the effect of work rate on arterial and tissue oxygenation during apnoeic exercise. Unsurprisingly, the effects of apnoea on $P_{ET}O_2$, $P_{ET}CO_2$, SpO_2, and heart rate were dependent upon the power output and, therefore, the metabolic rate. While it is not advised to interpret main effects in the presence of an interaction, there does appear to be a dose response in most variables to that of work rate (Figure 1). SpO_2 appears to be the exception (Figure 2A) and this may be due to the relatively narrow range of work rates included in our study design. All power outputs were in the moderate intensity domain and this was intentional seeing as heavy and very-heavy intensity work is rather difficult to sustain during apnoea. However, we want to reiterate that making interpretations for dose-response across the main effect of metabolic rate is speculative.

Brain oxygenation was unaffected by apnoea or work rate, owing to apparently robust autoregulation of brain perfusion. Apnoea duration was reduced in a step-wise manner to increasing power output – also not a surprising finding. While not all participants were able to complete the desired 60 s apnoea across the exercise conditions, there were no instances of loss of consciousness or dangerously low SpO_2.

The primary finding reinforces the concept that depletion of O_2 stores during apnoea is dependent on metabolic rate. This argues against the rationale for assigning a fixed time limit for apnoeic swimming or diving. The problem becomes much more complex when hydrostatic pressure changes are considered, such as when a diver descends and ascends during the activity.

THE PUBLIC HEALTH PROBLEM

According to the most recent DAN incidence report, there were 300 breath-hold diving accidents reported between 2010–2013 and 243 (81%) were fatalities. It is unlikely that this represents the true number of incidents, as there is significant under reporting and other obstacles to collecting these data. Nevertheless, there are a substantial number of accidents. It is probable that near-fatalities and accidents are even further under reported than deaths. In the cases reported, the activities described were snorkelling (56%), spear fishing (18%), freediving (16%) and collecting (10%). In a detailed subset of 162 cases, the disabling injury most commonly reported was hypoxic blackout (40 cases, 25%).[8] While there is no national surveillance programme for hypoxic loss of consciousness in water, these events are common even in community pools.[9] These cases occur even in guarded swimming pools, with dangerous underwater breath-holding behaviours cited as a common factor in these incidents.[10] The behaviours cited included: hyperventilation prior to breath-hold and training for static (motionless or still) apnoea and dynamic (swimming or diving) apnoea.[9] Men 16–20 yr of age are at particularly high risk for these behaviours.[10]

OBSTACLES FOR SAFETY RECOMMENDATIONS

An obstacle to developing recommendations or rules for breath hold diving is the wide variability in work rate and duration when complications arise. For example, breath holds longer than 79 s were associated with increased likelihood of blackouts in Hawaiian fisherman[11] whereas people at rest may not become hypoxic even with a breath-hold of up to 120 s.[4]

Next, measures of arterial oxygen saturation (SaO_2) when complications arise is also variable.[5,12] Based upon blood gas measurements from competitive breath-hold divers, a PaO_2 of approximately 30 mmHg is needed to maintain consciousness on the surface after a dive.[13,14] A PaO_2 of 30 mmHg typically corresponds to an SaO_2 of 55–60%. The value is dependent on the allosteric modifiers present

in the blood. Arterial desaturation of 20–50% is possible following 40 s of apnoea while exercising at 100 watts.[5] In another example, 80 s breath hold at two atmospheres absolute pressure with exercise resulted in SaO_2 of 58% with notable impairment.[12] Power to 60 W was constrained at maximum and did not have any incidences of SpO_2 < 88% during the apnoeas. Yet, swimming and diving can require higher power and greater deoxygenation.[15] For example, greater oxygen desaturations are present with higher output (120 W) corresponding to swimming speeds of ~0.5 $m \cdot s^{-1}$.[16] This is not a surprising result and is in agreement with our demonstration that O_2 stores depletion is metabolic rate-dependent. Without carefully defining the metabolic work and O_2 demands, a dive time limit alone, is unlikely to be useful.

A final barrier for safety recommendations is the variability in hydrostatic pressure effects. As in the example above, critical PO_2 in diving studies is that value necessary to maintain consciousness until surfacing. However, this is only the value upon reaching the surface – the PaO_2 at depth is hydrostatic-pressure dependent. Hypoxia of ascent is the primary threat and occurs as alveolar PO_2 is reduced due to the steep fall in hydrostatic pressure.[13] The present study was conducted at 1 atmosphere pressure and could not take account of the effects of hydrostatic pressure change on alveolar, arterial, and tissue PO_2 and PCO_2. Biological variation also complicates critical blood gases. Breath-hold divers' blood gases at a depth of 40 m show expected hyperoxia followed by hypoxia upon surfacing.[17] However, even this behaviour was not uniform. Compression atelectasis and V/Q mismatch at depth followed by subsequent reversal of this phenomenon while ascending may be responsible for the variability in blood gas measurements.[17] Loss of consciousness may also occur just after reaching the surface due to the circulatory delay in the oxygen reaching the brain tissue. Without considering the hydrostatic pressure changes involved in a dive, a time limit may be of limited utility.

ABSENCE OF ADVERSE EVENTS IN THIS EXPERIMENT

An important limitation in the design of this study is that of a sample not powered or designed to test safety. While we did not measure any instances of hypoxia within the 60 s apnoeic period, hypoxia was observed in 14 trials after the cessation of apnoea. The hypoxia definition of SaO_2 88% is, by definition, an arbitrary cutoff and it is not suggested that this be universally used as a definition of hypoxia or danger.

In this study, brain tissue oxygenation was consistently maintained across apnoeic time and metabolic rate. Further, brain oxygenation did not change in proportion to SpO_2 across the range of values achieved. The disparity is due to the superb autoregulation for perfusion in the brain.[18] Whilst there was a main effect of metabolic rate on brain oxygenation, the differences were of no physiologic consequence. These findings are in contrast to trained breath-hold divers performing maximal breath-hold in which cerebral oxygenation is challenged due to the extreme duration of apnoea (> 240 s).[19] Oxygen consumption in trained divers is also decreased, likely due to the development of a more pronounced diving reflex (and subsequent bradycardia).[20] Training prolongs the physiologic break point (onset of involuntary ventilatory activity), secondary to a decreased ventilatory response to increased $PaCO_2$.[21] Clearly, the risk for hypoxia is much greater in divers capable (on average) of > 240 s breath-hold.[19] This is, again, independent of any additional complexities from hypoxia of ascent.

SAFETY RECOMMENDATIONS FOR THE FUTURE

It may not be feasible to recommend an apnoeic time limit for amateur breath hold divers in a recreational setting. Guidance, tools or training are needed to make freediving safer. For example, in the setting of underwater rescue, a 40 s maximal breath hold time was recommended.[1] The limit was based on a study of repeated breath-hold dives (divers were recreational freediving instructors) to 5 m and 8 m for varying durations (maximum of 45 s). This recommendation relies on the mean nadir oxygen saturation of 89% for 45 s dives, with the lowest recorded saturation of 71%.[1] A conservative time limit such as this one could be imposed that would mitigate most of the dangers discussed above. However, apnoea time should not be a primary prescribed safety variable. The depletion of O_2 stores during apnoea is dependent on the metabolic rate (therefore, work rate) and may be highly variable amongst individuals. It seems an impossible task to develop multiple sets of rules or time limits based on different theoretical scenarios with many variables.

LIMITATIONS

Order effect is a serious limitation of our study design. Each volunteer performed the trials in the same order (i.e., static, 20 W, 40 W, and 60 W) with a 3 min active recovery period between each trial. This may have affected the volunteers' success in completing the 60 s of apnoea. It is not possible to speculate whether the order effect increased or decreased the apnoea time. While VO_2 gain (VO_2 per unit power: ~10 $mL \cdot min^{-1} \cdot W^{-1}$ for cycling) is surprisingly rigid[22] an order effect on apnoea durations may have underestimated the strength of the metabolic-rate dependence in the studied variables. That is, high power output trials resulted in shorter breath hold times and, if anything, this may have constrained the magnitude of disturbances to O_2 stores, end tidal measurements, etc. Finally, order effect does present a small risk for a priming effect on the metabolism. However, there is strong evidence that this priming effect is only present when including the heavy and very-heavy intensity domains and is thus unlikely in this study design.[23–25]

Finally, it was not possible to account for the effects of immersion and the diving reflex, which could potentially mitigate some of the effects of pressure on oxygen consumption referenced above. The diving reflex slows the heart rate and decreases myocardial oxygen demand, thereby reducing whole body $\dot{V}O_2$ and slowing time to hypoxia. Thus, oxygen saturation during apnoea decreases less with facial immersion, which could have a protective effect against hypoxia in the water. Facial immersion alone slows oxygen consumption to a greater degree than apnoea alone.[26] There are additional effects of immersion and temperature, such as peripheral vasoconstriction, that were not accounted for in this study.

Conclusions

The effect of apnoea on O_2 stores depletion was, as expected, dependent on metabolic rate, independent from the added complexities of a fall in static pressure on ascent. Therefore, it is contended that a time limit for amateur, recreational breath-hold diving may not guarantee safety.

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