Underwater pulse oximetry reveals increased rate of arterial oxygen desaturation across repeated freedives to 11 metres of freshwater

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Abstract
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Introduction: Recreational freedivers typically perform repeated dives to moderate depths with short recovery intervals. According to freediving standards, these recovery intervals should be twice the dive duration; however, this has yet to be supported by scientific evidence.
Methods: Six recreational freedivers performed three freedives to 11 metres of freshwater (mfw), separated by 2 min 30 s recovery intervals, while an underwater pulse oximeter measured peripheral oxygen saturation (SpO_2) and heart rate (HR).
Results: Median dive durations were 54.0 s, 103.0 s and 75.5 s (all dives median 81.5 s). Median baseline HR was 76.0 beats per minute (bpm), which decreased during dives to 48.0 bpm in dive one, 40.5 bpm in dive two and 48.5 bpm in dive three (all $P < 0.05$ from baseline). Median pre-dive baseline SpO_2 was 99.5%. SpO_2 remained similar to baseline for the first half of the dives, after which the rate of desaturation increased during the second half of the dives with each subsequent dive. Lowest median SpO_2 after dive one was 97.0%, after dive two 83.5% ($P < 0.05$ from baseline) and after dive three 82.5% ($P < 0.01$ from baseline). SpO_2 had returned to baseline within 20 s after all dives.
Conclusions: We speculate that the enhanced rate of arterial oxygen desaturation across the serial dives may be attributed to a remaining 'oxygen debt', leading to progressively increased oxygen extraction by desaturated muscles. Despite being twice the dive duration, the recovery period may be too short to allow full recovery and to sustain prolonged serial diving, thus does not guarantee safe diving.

Introduction

Breath-hold diving, or 'freediving' is an underwater activity practiced for competition, recreation or as a profession. In competitive freediving, athletes aim to reach a maximum time, distance or depth underwater in one dive.[1–4] The growth in popularity of the competitive sport has led to a rapid increase also of recreational freedivers world-wide.[5] All freediving activity is done on one breath of air only, and divers must rely on physiological responses to conserve oxygen to sustain brain function and work capacity, and if they overestimate their ability they are at risk of hypoxic syncope,[6,7] also called 'blackout'. While competitive freediving is made relatively safe via the presence of safety divers who can bring the diver back to the surface should blackout occur, recreational freedivers are at greater risk; according to statistics from Diving Alert Network (DAN) an average of 51 freedivers per year drown, which may be related to blackout events.[5]

Unlike competition divers, who perform one maximal dive after which they can rest and recover for an unlimited time, recreational freedivers typically perform repeated non-maximal dives to moderate depths with short recovery intervals. But how do freedivers know how to pace their dives to allow full recovery and safe continued diving? There is a 'rule of thumb' that the diver should spend double the dive duration at the surface to fully recover between subsequent dives. However, the serial effects of repeated freedives in a field setting are in fact not well known. Most studies of freediving physiology to date have been done in the laboratory due to the challenges with underwater measurements, and studies of repeated dives with short intervals revealed some serial effects on physiological responses and peripheral oxygen saturation (SpO_2).[8,9] Could changes induced by repeated diving lead up to hypoxic loss of consciousness? To understand if incomplete recovery of oxygen stores between dives could be a factor responsible for blackout in recreational divers, it is necessary to perform research during actual freediving to depth, as the gas

exchange and physiological responses are likely affected by the changes in hydrostatic pressure.

We recently constructed an underwater pulse oximeter enabling continuous measurements of heart rate (HR) and SpO_2,[10] and monitored these variables during deep single dives in competition divers.[11] Our current aim is to use this novel technology in recreational divers to study SpO_2 and HR, to investigate any serial effects in short dives. While SpO_2 will reveal the level of oxygen remaining after each dive, the HR-recordings will show the magnitude of the 'diving response' which conserves oxygen to sustain brain function[12,13] by peripheral vasoconstriction[14,15] and bradycardia.[16,17] By studying the diving response and arterial oxygen depletion during three subsequent dives to 11 metres of fresh water (mfw), a commonly reached depth for recreational divers, we aimed to reveal whether it is true that a diver can safely continue diving as long as the recovery interval is kept double the dive time.

Methods

The study protocol was approved by the Regional Committee for Medical and Health Research Ethics in Umeå. Sweden, (Dnr 2019-05147) and the tests were conducted in accordance with the Declaration of Helsinki. All participants gave their informed, written consent to participate in the study.

PARTICIPANTS

Six male recreational freedivers with a mean and standard deviation (SD) age of 36 (8) years, height 181 (3) cm and weight 89 (13) kg from a local freediving club participated. They had trained freediving regularly for a minimum of one year, and thereby qualified in category 3 in the 5-level categorisation system used to describe training levels of freedivers.[18] All divers were accustomed to repeated freediving and had frequently been to at least 12 meters depth, and were trained in safety procedures.

PROCEDURES

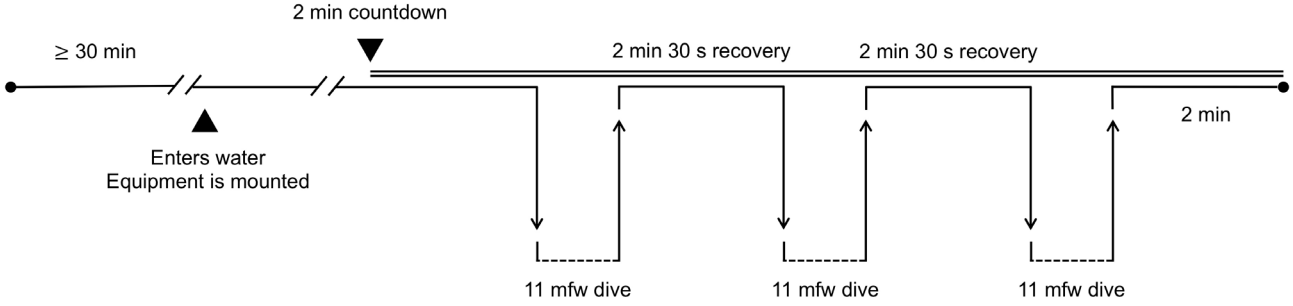
The participants reported to the 11 m deep indoor freshwater diving facility with an ambient air and water temperature of 32°C at least two hours after eating, wearing their own freediving equipment (mask, fins, wetsuit, weights). They were weighted to be neutrally buoyant at approximately 10 mfw. After 30 min of poolside rest, participants performed three consecutive freedives to 11 mfw separated by resting intervals at the surface, with countdown for the following dive. This protocol was aimed to reflect a part of their usual training routine; thus, dive durations were entirely determined by the divers, without receiving any input from the research team. Based on previous experience, the dive time was estimated to be on average about 1 min 15 s and the recovery interval, intended to be of about double the dive duration, was pre-set to 2 min 30 s (Figure 1). Participants were asked to refrain from hyperventilating and 'lung packing' manoeuvres, which are often used by competition divers to enhance dive duration.[19] Dives were conducted with established safety procedures including safety divers.[3] Following a 2-min countdown before each dive, the freediver swam down along a vertical diving line at his own pace and stayed at the bottom of the pool as long as he would do during usual training. Upon resurfacing, divers performed the recovery hook-breathing technique which is part of the safety measures frequently used by competition divers and many recreational freedivers.[19] Diving and interval durations were monitored with a stopwatch and recorded by the underwater depth-time profile of the datalogger.[20]

MEASUREMENTS

Upon entering the water, the participants were equipped with the prototype pulse oximeter for continuous measurements of SpO_2 and HR.[10] For details regarding the method and analysis see previous publications.[10,11] In short, plethysmograms were recorded at 30 Hz, and SpO_2 and HR were extracted from the plethysmograms at a window size of six seconds, and analysed internally every second. The

Figure 1

The experimental procedure consisting of three 11 metres of fresh water (mfw) freedives with 2 min 30 s recovery between the dives. Double line signifies continuous measurements of peripheral oxygen saturation (SpO_2) and heart rate (HR). Dotted line signifies bottom time, which was determined by the diver



two sensor heads were placed on both temples of the diver, and kept in place with medical tape and by the pressure of the hood of the wetsuit. Recording started two minutes before the first dive and continued until two minutes after termination of the third dive.

For the analysis, the results of both sensors were averaged for each data point to obtain one single value for SpO_2 and HR for each second.[10,11] The resulting time series for each dive was then smoothed using a 5-s moving median function. Baseline SpO_2 and HR were determined by calculating the mean over the time period of 90 to 30 s of measurements prior to the first dive.[12] The maximal arterial oxygen desaturation was calculated as the percentage change between the previously established baseline SpO_2 and the lowest SpO_2 value (SpO_{2nadir}).[21] We aimed to identify divers at risk of reaching severe hypoxia by tallying the number of divers reaching SpO_2 less than 75% in dives one to three. The magnitude of the diving bradycardia was calculated as the percent change from baseline HR, and maximal HR-reduction determined by identifying the lowest HR value (HR_{nadir}) of each dive.[21]

Heart rate data for all divers were also identified for the different diving phases for each dive, including baseline, descent (average of a 5 s window around 5 mfw during the descent), bottom (average of the entire period at the bottom), ascent (average of a 5 s window around 5 mfw during the ascent), first recovery period (average of the first 15 s directly after resurfacing) and second recovery period (average of values between 16 and 30 s after resurfacing). Because the duration of dives differed between participants, data for each dive were also expressed for subsequent timepoints representing percentages of total dive time.

STATISTICS

Due to the small sample size, a Friedman test was run to determine if there were differences in dive times and the studied physiological variables across the three dives. Pairwise comparisons were performed with Bonferroni corrections for multiple comparisons. For all data median values and interquartile range are presented, but mean values are also provided in an additional table for clarity. Additionally, a mixed model repeated measures analysis of variance (ANOVA; within factors: diving phase; between factors: dive number) were used to identify if dive number influenced the pattern of HR throughout the dive. Significant interactions or main effects were followed up with simple main effect analyses with pairwise comparisons using Bonferroni correction. For all ANOVA's, effect sizes are presented as partial eta-squared statistic (η^2_p). $P < 0.05$ was considered statistically significant.

To assess the impact of repeated dives on 1) arterial oxygen desaturation during diving and 2) reoxygenation during post-dive surface intervals, two models were constructed to assess the impact across each of three dives. The diving data were treated as the periods of diving, beginning at the onset of apnoea until the presentation of the oxygen saturation minimum at, or immediately after, the termination of diving. Surface intervals were based on the remaining data, extending from the presentation of the oxygen minima to the onset of the next dive. For each category (diving desaturation and surface reoxygenation), two models were considered. Models were constructed for each category separately. Peripheral oxygen saturation was modelled as a function of proportion of the dive (allowing comparison of variable dive durations) or surface interval (0–100%) or time since surfacing in an interaction term with dive number. In each model, the proportion of the dive was included as a continuous smooth term and dive number as a fixed factor. The framework used for the above-described analysis was generalised additive models (GAM), constructed within the 'mgcv' package in R.[22] Additive models were required to allow complex, non-linear relationships between covariates and response metrics. For each model, the final model was chosen by a model minimising the information theoretic criterion (AIC),[23] if their inclusion did not improve the model by two or more ΔAIC. Residual plots were examined for any evidence of violation of model assumptions.

Results

DIVE DURATION

All participants completed the three dives without reporting any symptoms related to hypoxic blackout, nor were there any symptoms of severe hypoxia, i.e., loss of motor control, observed by the researchers. Median (range) dive durations for dives one, two and three were 54.0 (39–83) s, 103.0 (86–113) s and 75.5 (58–122) s ($\chi^2(2) = 2.333$, $P = 0.311$). Median duration of all dives was 81.5 (51–108) s. The resulting median duration of recovery intervals was 151 (150–161) s, thus close to double the dive duration. Median (IQR) and mean (SD) values for the studied variables are presented in Table 1.

PERIPHERAL OXYGEN SATURATION

Median pre-dive baseline SpO_2 was 99.5 (99–100)%. SpO_{2nadir} occurred on average at 7.5 (2–8) s after resurfacing across all dives, thus the circulatory delay was the same in all dives. SpO_2 was significantly different across the diving series ($\chi^2(3) = 16.119$, $P = 0.001$). Post hoc analysis revealed a difference between baseline SpO_2 and SpO_{2nadir} after resurfacing from dive two (83.5 (70–87)%; $P = 0.015$) and three (82.5 (58–95)%; $P = 0.003$), but there were no significant differences between SpO_{2nadir} of dives one, two and three. The number of divers that dropped below an SpO_2 of 75% increased from none during dive one, to two in dive two, to three in dive three.

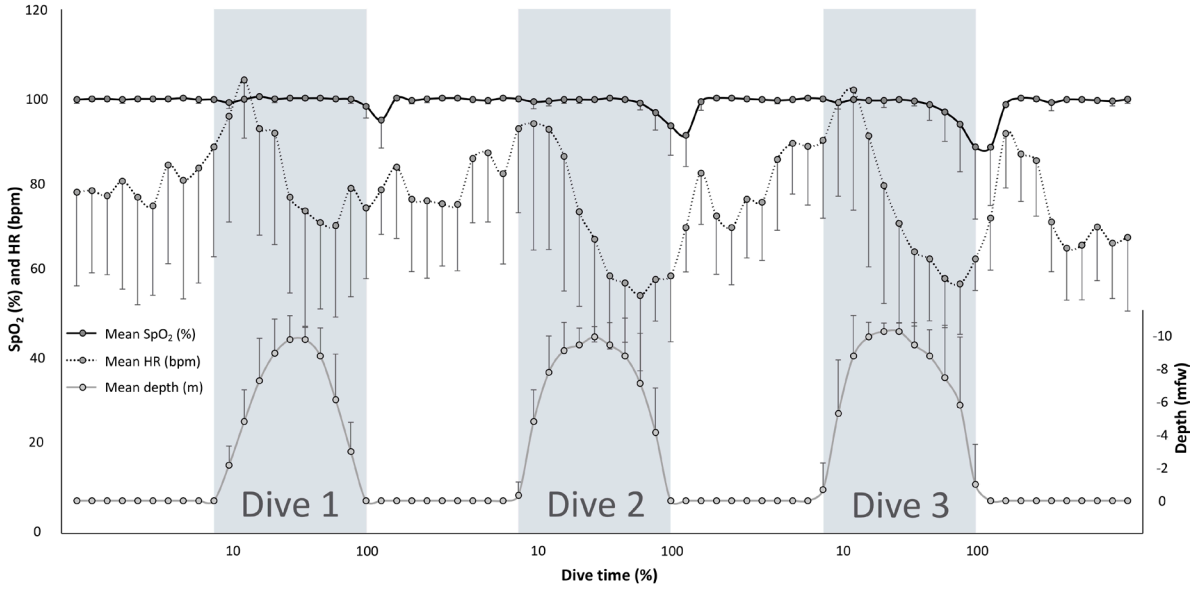
Table 1

Dive durations, pulse oximetry and heart rate results from baseline (pre-dive) and three dives to 11 metres of fresh water (mfw); HR – heart rate; HR_{nadir} – lowest heart rate in the dives; IQR – interquartile range; NA – not applicable; SpO_2 – peripheral oxygen saturation; SpO_{2nadir} – lowest peripheral oxygen saturation in the dives

Parameter	Statistic	Baseline (pre-dive)	Dive one	Dive two	Dive three
Dive duration (s)	Median (IQR)	NA	54 (39–83)	103 (86–113)	75.5 (58–122)
	Mean (SD)	NA	63 (30)	96 (28)	87 (42)
SpO_2 pre-dive SpO_{2nadir} (%)	Median (IQR)	99.5 (99–100)	97 (81–98)	83.5 (70–87)	82.5 (58–95)
	Mean (SD)	100 (1)	91 (11)	80 (14)	76 (22)
Oxygen desaturation (Δ%)	Median (IQR)	NA	3.0 (17.7–1.3)	16.1 (29.0–13.0)	17.1 (41.7–5.5)
	Mean (SD)	NA	8.7 (10.9)	19.7 (14.0)	23.7 (21.8)
HR pre-dive HR_{nadir} (bpm)	Median (IQR)	76 (61–97)	48 (41–69)	40.5 (36–48)	48.5 (36–51)
	Mean (SD)	79 (21)	55 (19)	45 (14)	44 (9)
Maximal HR reduction (Δ%)	Median (IQR)	NA	31.0 (36.8–21.4)	43.0 (48.7–39.5)	45.4 (48.3–42.8)
	Mean (SD)	NA	30 (16)	42 (12)	43 (10)

Figure 2

Mean and standard deviation (SD) heart rate (HR) in beats per minute (bpm), peripheral oxygen saturation (SpO_2 %) and dive depth profile in metres of fresh water (mfw) from six freedivers in three sequential dives to 11 mfw; values are relative to dive time; dive time is expressed as percentage to be able to compare means from all dives which were of different durations



HEART RATE

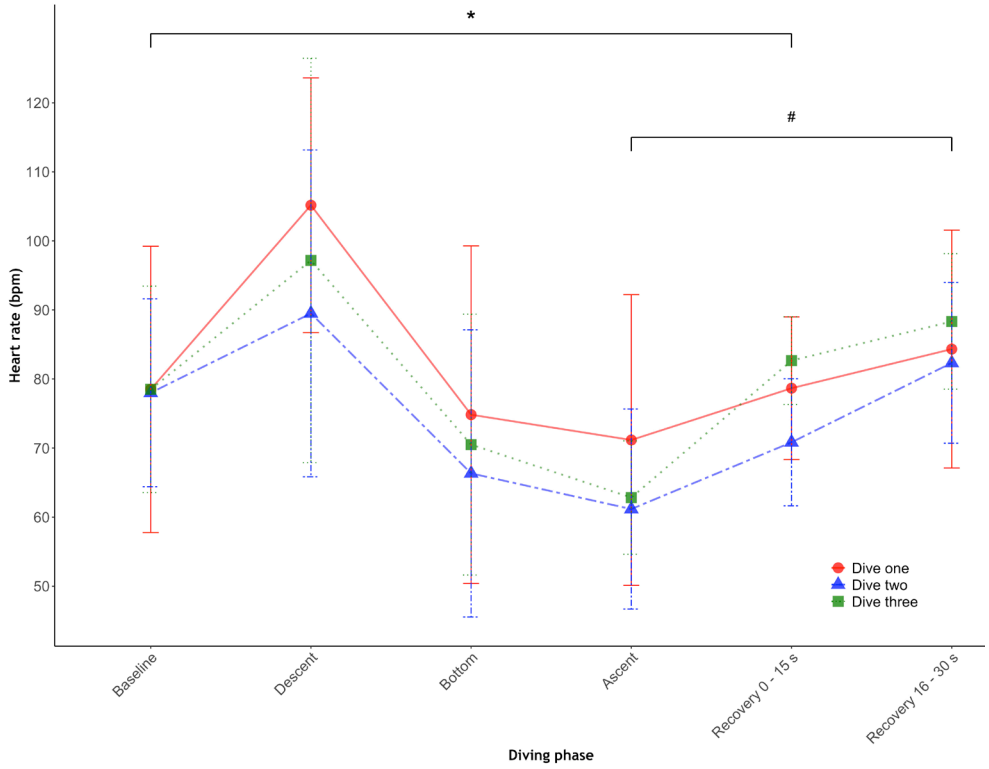
Median pre-dive baseline HR was 76.0 (61–97) bpm. HR was significantly different across the diving series, ($\chi^2(3) = 12.559$, $P = 0.006$). Post hoc analysis revealed a significant difference between baseline HR and HR_{nadir} from dive one (48.0 [41–69] bpm; $P = 0.044$), two (40.5 [36–48] bpm; $P = 0.001$) and three (48.5 [36–51] bpm; $P = 0.005$), but there were no significant differences between HR_{nadir} when the three dives were compared (Figure 2).

DIVING RESPONSE PHASES

Figure 3 displays the HR responses divided into different phases for dives one, two and three. Heart rate responses for the different diving phases shows similar patterns for all divers, regardless of dive number or individual absolute HR, i.e., there were no significant differences in each phase between the dives. There was no significant interaction between dive number and diving phase ($F_{10,75} = 0.485$, $P = 0.895$, $\eta^2_p P = 0.061$), and no main-effect for dive number

Figure 3

Mean (SD) heart rate in beats per minute (bpm) for divers undertaking each dive, with dives divided into phases (baseline, descent, bottom, ascent and recovery); * indicates significant difference between descent phase and all other phases except for the second recovery phase. # indicates significant difference between ascent phase and both recovery phases



($F_{2,15} = 0.524$, $P = 0.602$, η^2_p $P = 0.065$). However, there was a main effect for diving phase ($F_{5,75}$ = 14.046, $P < 0.001$, η^2_p P = 0.484). Heart rate increased from baseline during descent, when divers actively needed to overcome positive buoyancy. When divers reached the bottom of the pool, HR dropped below baseline levels, and continued to drop further during the ascent, where positive buoyancy reduced work ($P < 0.05$ between the descent phase and all other phases except for second recovery phase). Upon resurfacing, HR increased back to baseline levels ($P < 0.05$ between ascent phase and both recovery phases).

OXYGEN DESATURATION AND RESATURATION PATTERNS

The fitted model predicted that SpO_2 for the first half of the dive was similar between dives one, two and three. However, during the second half of the dives the rate of desaturation increased with each subsequent dive (Figure 4 left panel). The maximal model was retained as the best model with lowest AIC, thus, the interaction term between proportion of dive and dive number was retained allowing flexibility in the model to fit different intercepts to each dive, supporting the finding that the patterns of change in SpO_2 between dives were different. Further, the fitted model predicted that the pattern and rate of reoxygenation was similar between recovery surface intervals (Figure 4 right panel). The final model did not retain the interaction term between dive duration and dive number, supporting the observation that the patterns of change in SpO_2 during recovery intervals was not different across the dive series.

Discussion

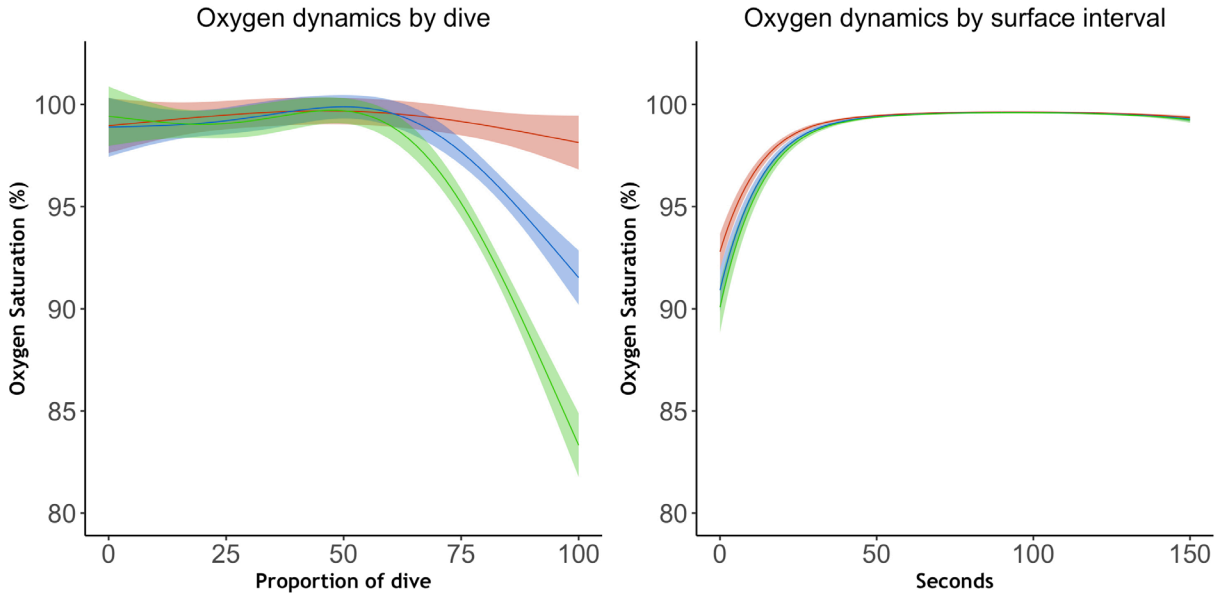
This study is, to our knowledge, the first to use underwater pulse oximetry to examine SpO_2 and HR continuously during repeated diving to moderate depths in recreational freedivers.

PERIPHERAL OXYGEN SATURATION PATTERNS

A progressively increased desaturation occurred across the dive series. Modeling of oxygen desaturation and resaturation rate showed that desaturation rate increased from the first dive through the third dive, despite similar oxygen resaturation patterns during recovery. This seems to imply that oxygen was consumed at a faster rate for each subsequent dive, despite similar magnitudes of the oxygen conserving diving response and despite similar dive durations. We are also unable to relate these differences in desaturation rate to differences in levels of exertion between dives, as depth was the same for each dive, which is also supported by similar HR-response and dive durations. We therefore speculate that the enhanced rate of peripheral oxygen desaturation across the three dives may be attributed to a subsequently increased oxygen extraction in working muscles. It is well known that oxygen uptake recovers more

Figure 4

Left panel – plots represent mean oxygen dynamics for dive one (red), dive two (blue) and dive three (green), shaded areas represent 95% confidence intervals. The changed shape and slope of the curve indicates statistically significant increased rate of oxygen desaturation for each subsequent dive. Right panel – lines represent oxygen dynamics upon resurfacing for dive one (red), dive two (blue) and dive three (green), shaded areas represent 95% confidence intervals, demonstrating similar rates of reoxygenation after all dives



slowly following muscular activity than arterial oxygen concentration.[24] The resting interval between dives was about twice the average dive duration, thus following the 'rule of thumb' taught in freediving schools, but our study shows that this may in fact be too short to allow full recovery for the divers, due to a remaining oxygen debt in their muscles.

Consequently, our findings indicate that the relatively short repetitive freedives to moderate depth, characteristic of recreational freedivers, may induce hypoxaemia at a progressively increased rate across the series. The lowest SpO_2 observed was 47% in one diver during the last dive which lasted 135 s, which is below 50%, a level considered to be associated with blackout in untrained individuals.[1] We speculate that a longer diving series may enhance the oxygen debt further which could involve a risk of blackout.

Even though we are aware of the possible margin of error in these pulse oximetry measurements, a well-known problem associated with all pulse oximetry measurements when oxygen saturation is decreased, these results are similar to data presented by others,[25] from a dive lasting 300 s by an elite diver. This shows that our recreational divers indeed approached very low levels of oxygen saturation in a shorter amount of time, and that some of our divers may have been very close to levels associated with blackout in non-elite divers.

Our data thus shows that factors other than dive duration may be important when aiming to predict oxygen management during repeated diving. These results are highly relevant from a safety perspective as recreational freedivers seem to be at particular risk for hypoxic blackout.[5] Our findings indicate that the pacing of repeated dives is essential to safety, and that the current recommendations are not sufficient.

DIVING RESPONSE PATTERNS

Heart rate traces for all divers and dives had similar features: an increase in HR from baseline levels during the descent phase, when the divers were actively swimming down while overcoming positive buoyancy. During this phase the exercise tachycardia thus dominates over the diving bradycardia, and the cardiovascular diving response is not developed. A contributing factor could be that depth was only 11 mfw, and the descent phase may have been too short to allow the drop in HR due to the diving response, typical of the passive 'free-fall' phase in deep freediving.[3,11] In studies of deeper and longer dives it appears that the two stimuli for exercise tachycardia and diving bradycardia may balance out resulting in an intermediate HR.[11]

Another factor responsible for the high HR during the descent phase in our study could be that the HR reduction, in addition to being triggered by apnoea, is influenced by facial cold-receptors stimulated by the difference between ambient air and water temperature.[26] The fact that both air and water were 32°C in our study may have resulted in a smaller stimulus than in a previous study where air was 26°C and water 22°C.[11]

When the diver reached the bottom and stopped swimming, bradycardia developed, as also observed in deeper dives.[11,27,28]

Compression of the lungs by hydrostatic pressure to half their size at the surface could also have contributed to the drop in HR at the bottom.[29]

During ascent the low HR prevailed in the positively buoyant diver, while in deep diving this phase is associated with an elevation in HR due to the intense work against negative buoyancy.[11] During the initial 15 s after surfacing, HR increased, and this recovery tachycardia continued to increase in magnitude during the following 15 s when breathing had elevated SpO_2, however, not to same extent as patterns previously noted after deep dives.[11]

The percent change from baseline to the lowest HR attained during the dives increased from 30 (16)% during the first dive to 42 (12) and 43 (10)% during dive two and three respectively, however this increase was not statistically significant. A lowest individual HR reduction of 58% was observed in one diver during the second dive. The HR reductions of the latter two dives is comparable to those found in previous studies in competition divers.[11,27]

We would also like to suggest that the tendency for longer dive durations in dives two and three may demonstrate that the initial dive acts as a warm-up, enabling the diver to produce longer apnoeic duration due to the recruitment of spleen red cell stores,[8] or enhancement of other protective reflexes. This should be examined in a larger group of divers, as the small group studied here would possibly lead to type two errors.

LIMITATIONS

A limitation of this study is the small sample size, which reduces statistical power, and only allows preliminary conclusions to be drawn from our results. Also, pulse oximetry is less reliable at low saturation levels, so absolute values in the low range can be numerically less accurate than e.g., blood gas analysis,[30] but their relative values between dives would be reliable. The observation that the response patterns were similar for all divers regardless of their differences in response magnitude could support the generalisation of these findings. Future studies should include a larger sample and more subsequent dives to further evaluate the effects of serial diving.

Conclusions

In the current group of recreational divers, a series of three dives separated by recovery periods of approximately twice the dive duration led to subsequently increased rate of arterial oxygen desaturation, despite attaining baseline SpO_2 values within 30 s of resurfacing. The fact that the rate of oxygen desaturation increased throughout the freediving session did not overtly compromise the safety of these divers, even though some individuals reached extremely low SpO_2 values. However, the combination of progressively increased rate of oxygen desaturation for longer sequential dive sessions could pose a risk of blackout, especially if dives are being prolonged. We therefore speculate that the enhanced rate of peripheral oxygen desaturation across the three dives may be attributed to an increased oxygen extraction in inadequately recovered working muscles. Indeed, in three out of six divers, this led to hypoxia below 75% SpO_2 in the last dive. Despite being twice the dive duration, the recovery period may have been too short to allow full recovery, thus an oxygen debt had accumulated. This suggests that the 'rule of thumb' – that such intervals guarantee safe freediving – may not be reliable for longer freediving sessions. Longer dive series should be studied to see if the rate of oxygen desaturation continues to increase and may cause a risk of hypoxic blackout. To evaluate our results further, it would be helpful if oxygen uptake measurements could be made across serial dives, and if venous oxygen levels could be determined.

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