Case studies in physiology: is blackout in breath-hold diving related to cardiac arrhythmias?

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Running title: Arrhythmia associated with blackout

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Abstract

Syncope or “blackout” (BO) in breath-hold diving (freediving) is generally considered to be caused by hypoxia. However, it has been suggested that cardiac arrhythmias affecting the pumping effectivity could contribute to BO. BO is fairly common in competitive freediving, where athletes aim for maximal performance. We recorded heart rate (HR) during a static apnea (STA) competition, to reveal if arrhythmias occur. Four male freedivers with STA personal best (PB) of 349±43s, volunteered during national championships, where they performed STA floating face down in a shallow indoor pool. A non-coded Polar T31 chest strap recorded R-R intervals and a water- and pressure proof pulse oximeter arterial oxygen saturation. Three divers produced STA near their PB without problems, while one diver ended with BO at 5min17s, which was 12s beyond his PB. He was immediately brought up by safety divers and resumed breathing within 10s. All divers attained similar lowest diving HR (47±4bpm), but HR recordings displayed a different pattern for the diver ending with BO. After a short tachycardia the three successful divers developed bradycardia which became more pronounced during the second half of the apnea. The fourth diver developed pronounced bradycardia earlier, and at 2.5min into the apnea HR started alternating between approximately 50 and 140 bpm, until the diver lost consciousness. At resumed breathing, HR returned to baseline. Nadir oxygen saturation was similar for all divers. We speculate that arrhythmia could have contributed to BO, by lowering stroke volume leading to a systolic blood pressure drop, affecting brain perfusion.

New & Noteworthy

Heart rate during prolonged breath-holding until the point of loss of consciousness has not previously been published. The recordings shows that blackout was preceded by a period of persistent alterations in R-R intervals, whereby an ectopic beat followed every normal heartbeat. Explanations for this deviating heart rate pattern could be either premature atrial contractions or premature ventricular contractions following every atrial beat, i.e., bigeminy, which could have compromised cardiac pumping function and caused/contributed to blackout.

Introduction

Breath-hold diving (freediving) is a recreational as well as a competitive sport, whereby voluntary apnea and immersion are combined. Static apnea (STA) is a discipline in competitive freediving where athletes aim to hold their breath for maximal duration under water (1). The physiological responses to apnea involve vagally mediated bradycardia and sympathetically activated peripheral vasoconstriction (2). Previous research studied apneas in a laboratory setting to examine cardiac regulation, and found that apneic duration, the degree of hypoxemia and the magnitude of bradycardia correlated with arrhythmogenesis (3–5). Less is known about heart rate (HR) during STA competitions; arrhythmias were observed once during competition in a freediver (6), and it has been suggested that HR may be affected by competitive stress (7). Freediving may lead to syncope, or "blackout" (BO, (8)), and it is presently unknown if such events are solely caused by cerebral hypoxia arising from depletion of oxygen stores, or if cardiac function is compromised by arrhythmias (9). We therefore aimed to investigate the HR pattern in well trained freedivers during a freediving national pool competition where they engaged in STA of extreme durations, and hypothesized that sudden changes in HR predicted loss of consciousness. A secondary objective was to determine the lowest arterial oxygen saturation (SpO_2) resulting from those apneas.

Methods

PARTICIPANTS AND STUDY PROTOCOL

Four male freedivers (mean ± SD age 41 ± 12 years, height 179 ± 9 cm, weight 70 ± 10 kg, vital capacity 5.7 ± 0.4 L) volunteered to be monitored during their maximal STA attempt at the Swedish national freediving championships. The study protocol had been approved by the Regional Committee for Medical and Health Research Ethics in Umeå, Sweden, and tests were conducted in accordance with the 2013 Declaration of Helsinki with the exemption of publicly pre-registration.

The athletes had been active in the sport for 6 ± 5 years, and currently trained freediving 4.0 ± 1.5 h/week. Their personal best performance in STA before the competition was 349 ± 43 s. The competition was held in a shallow indoor pool with space for the athletes to prepare according to their own routines, which could include stretching, yoga, breathing- and/or meditation exercises. When starting time approached, and the athletes indicated that they were ready, they were equipped with a heart rate chest strap (Non-Coded Polar T-31, Polar Electro Oy, Kemple, Finland) around the thorax below the wetsuit, which transmitted the R-R

intervals to a water- and pressure proof datalogger (10) which was placed in the hood of the wetsuit. A prototype pulse oximeter probe on the forehead was used to record arterial oxygen saturation, and its data was stored on the same data logger (10).

The athlete then entered the water (28 - 29°C, room temperature 27°C) of the shallow pool five minutes before start. Before starting, two freedivers were floating in the supine position with the help of foam objects while the other two freedivers where squatting with their heads above the surface. During the last seconds of the countdown all athletes performed glossopharyngeal breathing or "lung packing" (11) to increase lung volume, whereafter they assumed prone position, submerging their face in the water and holding their breath. All volunteers wore a wetsuit, nose clip, and three wore swim glasses.

Safety routines involved a safety diver present in the water. The athletes were instructed to respond to a small tap by their coach by lifting their index finger to show that they were conscious. When freedivers approached the end of their maximal breath-hold they put their feet down on the bottom, resurfaced and started recovery "hook breathing", a breathing maneuver whereby each exhalation is shortly interrupted to build up intrapulmonary pressure (12). Then followed the "surface protocol" involving removing all facial equipment, showing the "OK" hand gesture and verbally stating "*I am OK*" (13), which was assessed by the judge to deem the result valid. Failure to perform the protocol would signal severe hypoxia and disqualify the athlete.

ANALYSIS

The raw R-R data were exported to a computer for analysis with Kubios software HRV 2.0 (UKU, Kuopio, Finland). The recordings were visualized in a tachogram, which plots the distance of each consecutive R-R interval in milliseconds on the y-axis against the time of recording in seconds on the x-axis. The root mean square of successive differences between normal heartbeats (RMSSD), a measure of parasympathetic activity (14), was calculated and analyzed for baseline (90 to 30 s before start) and the initial response (30 to 90 s after start (15)). Lowest HR was determined as the lowest HR averaged over a 5 s period at any time during the apnea, reflecting the apnea-induced maximal HR reduction (16). The R-R series were also checked for regularity with the help of a Poincaré graph which plots each R-R interval against its previous one, thus each couple of R-R intervals is displayed as one dot in the graph. Depending on the underlying heart rhythm, the graph produced follows a certain

pattern, which can be visually recognized. Typically, the Poincaré graph of a healthy person shows one cluster in the shape of a comet along the line of identity. When the main cluster displays signs of asymmetry, or if clusters exist that are separated from the main cluster, occurrence of rhythm disorders and arrhythmias is indicated (17, 18). For pulse oximetry, the photoplethysmographic (PPG) signals were processed in the following steps to retrieve the SpO_2 values. First, the steps caused by red/infrared light intensity changes were removed. The signals were then detrended using a moving average with a window of 4 s. The R-value was calculated using the Root Square Mean (RMS) method. Two filters were then used to remove unreliable R-values. Values with low perfusion index (PI) were filtered out (19), after which values with fast moving R were filtered out (a conservative laboratory developed method). The SpO_2 values were calculated from R-values using calibration constants a, b and c using the following formula: $SpO_2 = a* R^2 + b*R + c$. Means of the three lowest subsequent SpO_2 values were then used to indicate lowest (nadir) individual arterial oxygen saturation at the end of STA.

Results

Divers 1-3 completed successful STA performances near their personal best (mean 342 ± 34 s), whereas diver four lost consciousness after 317 s, which was 12 s beyond his previous personal best (Table 1). The diver was immediately brought up by safety divers and resumed breathing within 10 s of the "blow-tap-talk" procedure, whereby the safety diver blew air and tapped on the face of the diver and talked calmly to him (20), and he was fully conscious after 20 s. The diver was examined by the responsible AIDA-assigned medical doctor to check that SpO_2 values were in the normal range.

For divers 1-3, mean baseline RMSSD (32 ± 6 ms) did not change across the initial response to apnea (34 ± 6 ms, Table 1). For diver four, baseline RMSSD (30 ms) was similar to those of divers 1-3, but rapidly increased during the initial response to apnea (to 89 ms, Table 1).

Baseline HR in all divers dropped from 97 ± 13 bpm to lowest HR of 47 ± 4 bpm, a decrease by 51 ± 2 % (Table 1). Although divers' lowest diving HR was similar, visual inspection of the tachograms showed different HR patterns across STA (Figure 1).

After a short tachycardia all divers experienced a drop in HR. The bradycardia developed differently throughout the course of STA; in divers one and two HR dropped during the

second half of the apnea, in diver three the bradycardia developed gradually throughout STA, while the fourth diver ending with BO developed an early, pronounced bradycardia.

At approximately 2.5 min into the apnea, the tachogram for diver four showed a distinct pattern alternating between 1250 and 400 ms (approximately a HR of 50 and 140 bpm), clearly identifiable by two lines of data points (Figure 1). This continued until the diver lost consciousness, where the final R-R was alternating between 700 and 400 ms. At resumed breathing, the irregular pattern disappeared and HR returned to pre-dive level.

Inspection of the poincaré plots showed a regular R-R rhythm for the three successful divers, apparent by relatively dense clustered clouds of points (Figure 2). For the diver with BO, two additional separate clusters were visually identified for the second half of the STA (red dots in Figure 2).

All divers had normal SpO_2 values at $\geq$ 95 % during the minute before STA. The apneas resulted in similar arterial oxygen saturation nadirs for all divers (58 ± 4 %; Table 1). All divers recovered to baseline values within 2 min after termination of STA.

Discussion

Heart rate during prolonged breath-holding until the point of loss of consciousness has not previously been published. The recordings shows that BO was preceded by a period of persistent alterations in R-R intervals, whereby an ectopic beat followed every normal heartbeat. Explanations for this deviating heart rate pattern could be either premature atrial contractions (PACs) or premature ventricular contractions (PVCs) following every atrial beat, i.e., bigeminy (21, 22), which could have compromised cardiac pumping function and caused or contributed to BO. All divers experienced bradycardia, which is typical of the "diving response" and is considered protective against hypoxia (1, 2). Although sometimes considered an arrhythmia, our results support the view that bradycardia per se does not lead to loss of consciousness. The similar post-apnea oxygen saturation nadir supports the view that arrhythmias could be a cause of the BO, but it cannot be ruled out that individual sensitivity to hypoxia could also be involved. As all four divers were well trained, these levels of oxygen saturation at above 50 % were not considered extremely low (1).

The current report presents us with two relatively common events in prolonged STA – arrhythmias and unconsciousness – that occurred simultaneously in one diver. The arrhythmias in the diver with BO occurred during the second half of the STA, which is comparable to previous findings where the hypoxic stage of the prolonged apnea was marked by arrhythmogenesis (3, 5). Early afterdepolarizations are more common when heightened vagal tone induces extreme bradycardia and increases action potential duration (23). In our case, the diver with BO experienced a highly augmented vagal drive early on, as evident by the sudden increase in RMSSD, possibly inducing a pro-arrhythmogenic effect on the cardiac tissue. However, since sympathetic discharge most likely increased progressively during the STA due to chemoreflex activation (24), it is possible that an autonomic conflict by sympathetic and parasympathetic co-stimulation synergistically induced the arrhythmias, as has been proposed previously (25, 26).

Although these measurements were obtained through a chest strap transmitter, and we thus could not perform analysis of ECG, the results of our analysis through commonly used methods seem to support this conclusion (18). Even with a complete ECG analysis sometimes is not easy to differentiate these two arrhythmias (21). PACs are considered benign electrical disturbances that usually do not require any treatment, but they can precede more serious arrhythmias like atrial fibrillation (27). On the other hand, PVCs represent potentially more serious arrhythmias triggered by exercise and hypoxia, that can be associated with ischemic heart disease and increased mortality if recurrent (22).

As a result of frequent premature contraction, the filling time of the left ventricle is shortened, which reduces stroke volume and cardiac output (28). We therefore speculate that the current BO case may have been caused, at least in part, by hemodynamic instability induced by a tachyarrhythmia (28). It is worth noting that the bigeminal pattern disappeared upon restoration of breathing, in a similar manner to observations by Costalat et al. (5), where divers had recovered within a minute after resumption of breathing, concomitant with a notable decrease in the number of arrhythmias.

Cardiac arrhythmias may also have occurred in the divers that did not BO, as some parts of the tachograms point to short arrhythmic episodes in these divers. None of these anomalies, however, were as consistent as the deviating pattern in the diver with BO, where the poincaré graph pointed towards a bigeminal pattern (18). Indeed, changes in the intra-thoracic pressure,

as during involuntary breathing movements (IBM's (29)) typically occurring in long apneas, could have influenced beat-to-beat fluctuations (16). However, as we can assume that all these freedivers experienced IBM's to a high degree without showing any deviating patterns, and divers employed similar positions at the water surface, we are inclined to disregard this point.

Further research on cardiac patterns during breath-hold diving is needed, for example to explore the suggested interaction between HR and oxygen saturation (30). Recording cardiac patterns could be particularly important during deep freediving and in recreational freediving, where arrhythmias could potentially be lethal. As previously noted by Shattock and Tipton (25), the frequency of cardiac problems on immersion might be underestimated, and arrhythmias may have been the cause of death in cases where death was ascribed solely to drowning. To confirm our results, HR patterns need to be further investigated in a larger sample and combined with other measurement devices. However, the current study shows that the use of existing underwater technologies in real diving situations deserves further attention, and we believe they may have life-saving capabilities.

Acknowledgement

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Conflict of interest

None

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Figure legends

Figure 1: tachogram of all four divers; apnea start and end marked by dotted lines, black rectangle indicates blackout period for diver 4; red dots for diver 4 correspond to red dots in Figure 2.

Figure 2: poincaré plot for the entire apneic period for each of the divers, whereby the plots for divers 1-3 indicate balanced sympathetic and parasympathetic activity (17), whereas the pattern of the red dots in the plot for diver 4 indicate a bigeminal rhythm (18) during the second half of the apneic period.

Figure 1

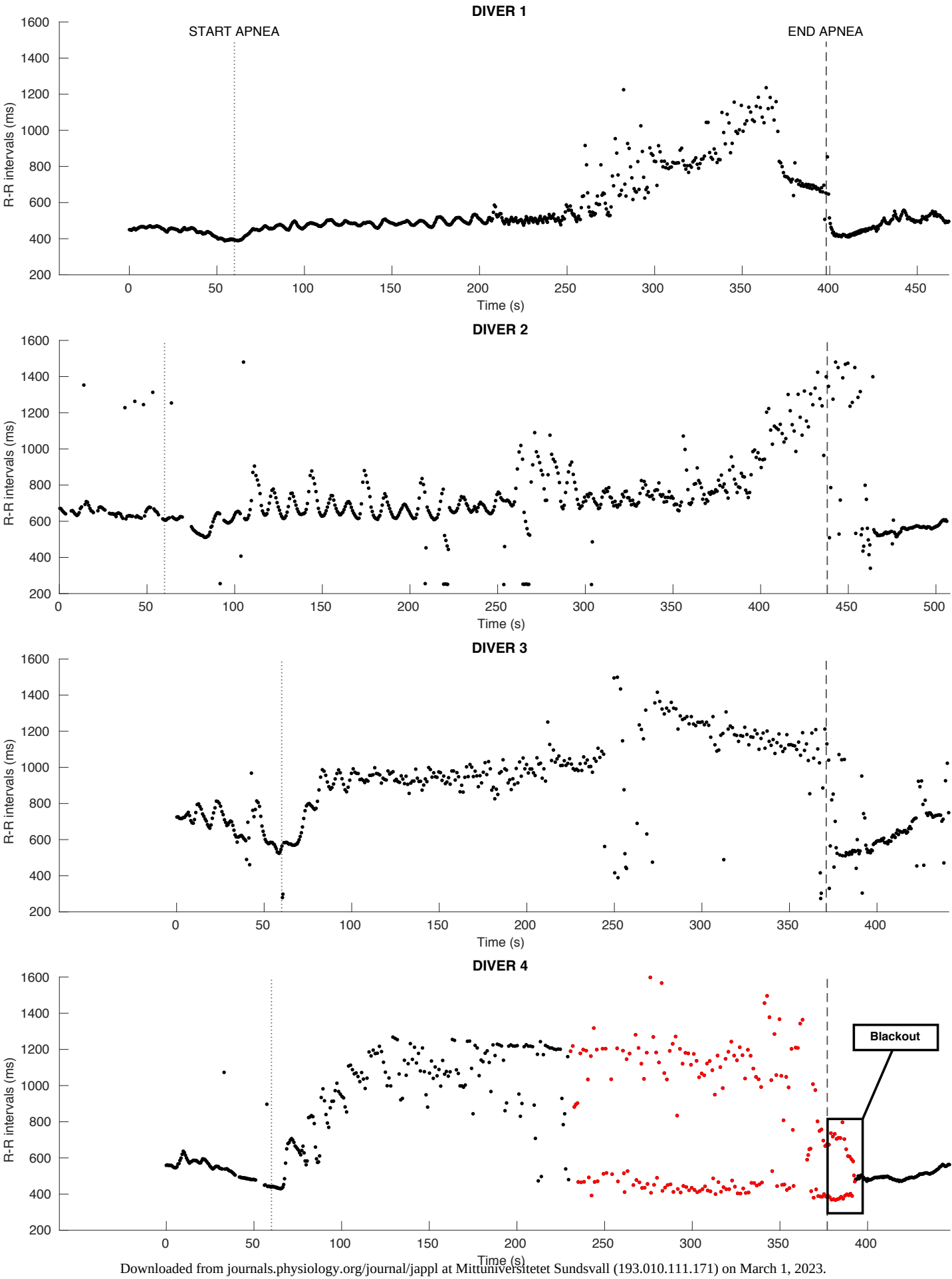


Figure 2

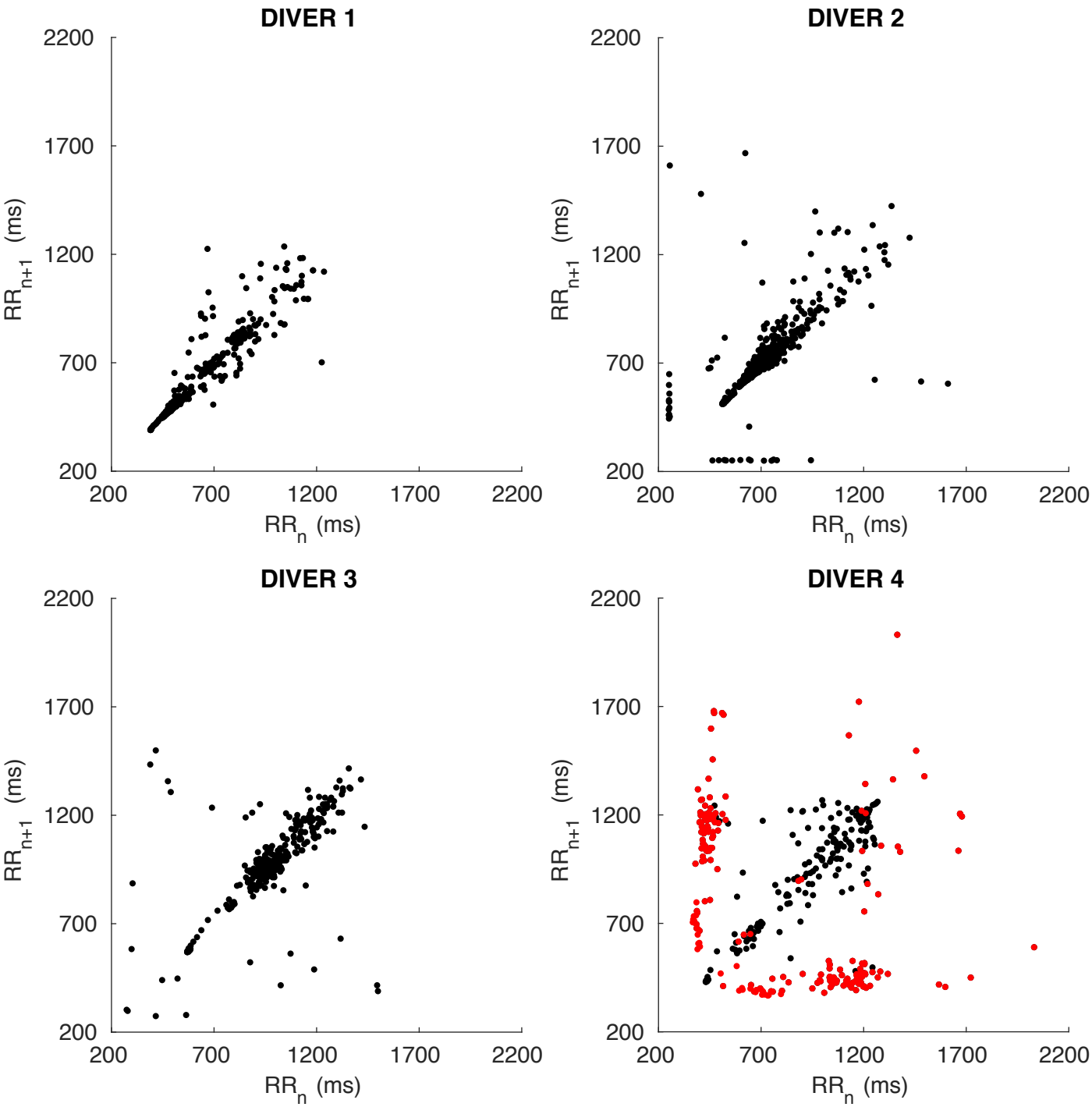


Table 1. *Results of four well trained freedivers engaging in a maximal static apnea during a competition*

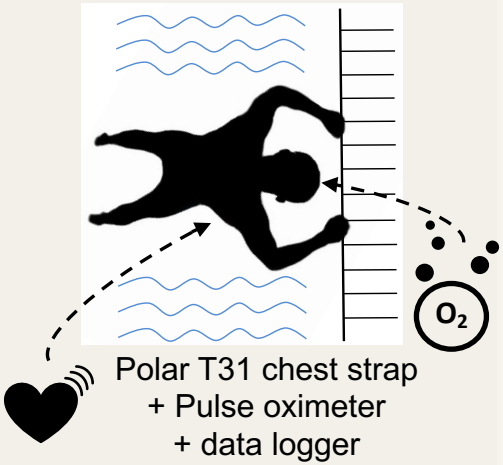
	Diver 1	**Diver 2**	**Diver 3**	**Diver 4**
STA personal best (s)	*375*	*395*	*319*	*305*
STA competition (s)	338	378	311	317
Baseline HR (bpm)	111	92	81	103
Lowest HR (bpm)	52	45	42	48
HR reduction (%)	53	51	48	53
Baseline RMSSD (ms)	29	27	39	30
Initial apnea RMSSD (ms)	27	39	35	89
Nadir SpO_2 (%)	64	57	54	57

STA, static apnea; HR reduction, percental decrease from baseline to lowest heart rate measured at any point during the apnea; RMSSD, root mean square of successive differences between normal heartbeats; Initial apnea, 30 to 90 s after start of apnea; Nadir SpO_2, lowest arterial oxygen saturation value measured by pulse oximetry at the end of apnea.

Case studies in physiology: is blackout in breath-hold diving related to cardiac arrhythmias?

METHODS

4 ♂ well trained breath-hold divers
1 static apnea for maximal time during national competition in shallow indoor pool



Polar T31 chest strap
+ Pulse oximeter
+ data logger

OUTCOME

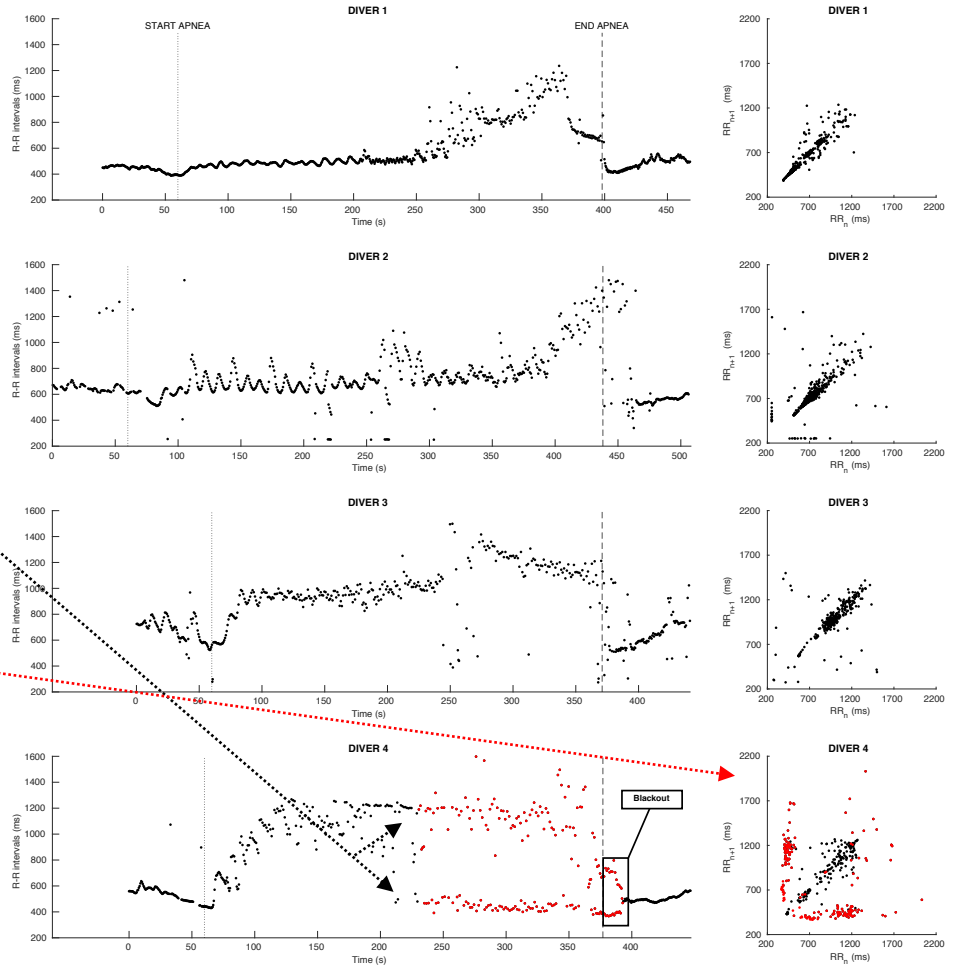
Mean apneic time: 5 min 42 s ± 30 s; **Diver 4 blacked out after 5 min 17 s**

Similar end-apneic oxygen saturation (58 ± 4 %)

Time course of bradycardic response differed between divers, but similar lowest heart rate (47 ± 4 bpm)

Persistant alteration in R-R interval pattern for diver 4, halfway through the apnea

Bigeminal pattern (red dots)



CONCLUSION

We speculate that a tachyarrhythmia could have contributed to the blackout of diver 4, by lowering stroke volume leading to a systolic blood pressure drop, affecting brain perfusion. This highlights the importance and potential of commonplace equipment as a practical tool to improve diving safety and should be investigated further.