Arterial Oxygen Saturation and Heart Rate Variation During Breath-Holding: Comparison between Breath-Hold Divers and Controls

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Arterial Oxygen Saturation and Heart Rate Variation During Breath-Holding: Comparison between Breath-Hold Divers and Controls

Abstract

Breath-holding induces cardiovascular responses, notably bradycardia and peripheral vasoconstriction, which are known collectively as the diving response. This response is oxygen-conserving, i.e. an augmented response attenuates arterial oxygen desaturation, and is enhanced by apnoea training. To test this hypothesis, we compared heart rate (HR) and arterial oxygen saturation (\(\text{Sa}_2\)) in breath-hold divers (BHD) and non-divers (ND). Nine BHD and nine healthy ND performed two static apnoeas (for 30 s and 45 s) and two dynamic apnoeas (for 30 s and 45 s) while swimming underwater at 0.7 m s\(^{-1}\). The pool temperature was 26°C. The apnoeas were performed at 60% of forced vital capacity. Heart rate (HR) and \(\text{Sa}_2\) were recorded before breath-holding and at its end and are expressed in % change from rest values (\(\Delta\text{HR}\) and \(\Delta\text{Sa}_2\)). Comparisons between BHD and ND showed that \(\Delta\text{Sa}_2\) were lower in divers after both static apnoeas for 30 s and 45 s (~2.8% vs. ~5.5%; ~3.2% vs. 6.3%; p < 0.05, respectively) and dynamic apnoeas (~6% vs. ~10.1% ~7.2% vs. ~12.3%; p < 0.05, respectively). The change in HR did not differ between the two groups and negative linear relations were found between \(\Delta\text{HR}\) and \(\Delta\text{Sa}_2\) in both divers and ND (\(r = 0.66\) and 0.61, respectively; p < 0.001). Moreover, the slope was lower for the divers (~0.785 vs. ~0.1429; p < 0.001). Our results suggest that apnoea training explained the greater oxygen conservation seen in the divers in both static and dynamic conditions.

Key words

Breath-holding · arterial oxygen saturation · training · bradycardia

Introduction

Breath-holding is known to induce a combination of cardiovascular responses in humans called the diving response [11,19,20,26]. This “diving reflex” includes bradycardia, peripheral vasoconstriction, reduced cardiac output, increased arterial blood pressure and a redistribution of peripheral blood flow to oxygen-sensitive organs [5,7,15,35]. Moreover, arterial oxygen saturation (\(\text{Sa}_2\)) is less reduced with a pronounced diving response, which reinforces the hypothesis of heart and brain protection against the hypoxia of apnoea [1]. These responses have been observed during dry static apnoea in air and with facial and whole body immersion [1,6,18]. However, the extent of the HR and \(\text{Sa}_2\) modifications during exercise has been questioned [8,21,30]. The discrepancies seen in the literature seem to be due in large part to the variety of the research protocols employed [3,22,27,32]. Furthermore, the studies showing a correlation between HR and \(\text{Sa}_2\) have often been undertaken in laboratory conditions [1,2,25].

To elucidate the effects of training per se on the diving reflex, we investigated the effects of breath-holding on heart rate and \(\text{Sa}_2\).

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Bibliography

under static and dynamic conditions. Specifically, we compared the heart rate and SaO₂ responses induced by two different breath-holding durations, both at rest (static apnoea) and during underwater swimming at a set speed (dynamic apnoea), in experienced breath-hold divers and controls.

Material and Methods

Subjects
Eighteen voluntary, non-smoking and healthy men participated in this study. One group was composed of nine breath-hold divers (BHD) and the other group comprised nine non-diving controls (ND). The BHD group had been regularly training in apnoea (5 ± 2 hours week⁻¹ for at least 6.4 ± 4.6 years). The controls had no experience in apnoea but were trained in field sports (3.5 ± 2.1 hours week⁻¹ for at least 9.2 ± 4.8 years). A consent form was signed by all subjects before the experiment.

Measurements
Forced vital capacity (FVC) and forced expiratory volume in one second (FEV₁) were measured (Kit Micro Cosmed, Rome, Italy) before apnoea testing. For each parameter, the best value was chosen from at least three consecutive manoeuvres differing by no more than 5% [31]. HR was recorded continuously (Sport Tester PE-4000 Polar) and the average values were calculated for the period 90–30 s before each apnoea in a prone position in water (HR0) and at the end of the apnoea tests for the last 10 s (HR1). Arterial oxygen saturation (SaO₂) was continuously recorded with a beat-by-beat pulse oximeter placed on the index finger (Biox 3700, Ohmeda, Madison, WI, USA) [4]. For SaO₂, an average value was calculated for the period 90–30 s before apnoea test (SaO₂ T0) and for the 10-s period ending with the nadir SaO₂ value (SaO₂ T1) (Fig. 1).

Protocol
Apnoeas were performed in a 25-m swimming pool. The water temperature was 26°C. During immersion, subjects wore swimming trunks, a mask, fins, and a weight belt to hold them completely under the water. Hyperventilation or Valsalva manoeuvres were forbidden before and during the tests. First, two static apnoeas of 30 and 45 seconds (SA30, SA45) were performed in a prone position, with a 15-minute recovery between them. The subjects then performed a warm-up (200 m) with fins, mask, and snorkel at an underwater swimming velocity of 0.7 m·s⁻¹ [10]. Two dynamic apnoeas of 30 and 45 seconds (DA30, DA45) were next performed at an underwater swimming velocity of 0.7 m·s⁻¹, which was assumed to be moderate muscular effort [10]. These dynamic apnoeas were also separated by a 15-minute recovery period (Fig. 1). The apnoeas (SA and DA) were performed with the same initial lung volume measured before the apnoeas using the spirometer (60% of the forced vital capacity: FVC).

Data analysis
Heart rate and SaO₂ variations (Δ) were calculated as the index of change (direction and magnitude) induced by apnoea (ΔHR = [HR1 – HR0/HR0] - 100; ΔSaO₂ = [SaO₂ T1 – SaO₂ T0/SaO₂ T0] - 100). ΔHR was negative or positive, depending of the HR kinetics (bradycardia or tachycardia). This method minimised the differences between rest and post-apnoea values of HR and SaO₂.

Statistical analysis
We used Statview software (Abacus Concept, Inc., Berkeley, CA, USA) for analysis. A Mann-Whitney test was used to compare the biometric, training, and spirometric characteristics between divers and non-divers. Comparisons of HR and SaO₂ kinetics between divers and non-divers were made by a Wilcoxon test. Spearman correlations were determined between variables. A p-value < 0.05 was considered significant.

Results
No differences between the groups were found for the anthropometric characteristics or FVC and FEV₁, expressed as absolute values or as % of predicted values (Table 1). Moreover, all values

<table>
<thead>
<tr>
<th></th>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>FVC (l)</th>
<th>Predicted values (% of FVC)</th>
<th>FEV₁ (l·s⁻¹)</th>
<th>Predicted values (% of FEV₁)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Divers</td>
<td>26.3 ± 6</td>
<td>76.7 ± 13</td>
<td>177.0 ± 3.2</td>
<td>5.17 ± 0.6</td>
<td>99.9 ± 7.9</td>
<td>4.37 ± 0.3</td>
<td>99.8 ± 6.9</td>
</tr>
<tr>
<td>Non-divers</td>
<td>22.3 ± 1.1</td>
<td>70.8 ± 7.6</td>
<td>174.8 ± 5.4</td>
<td>5.39 ± 0.54</td>
<td>104.5 ± 6.1</td>
<td>4.41 ± 0.52</td>
<td>100.7 ± 10.8</td>
</tr>
</tbody>
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Values are means ± SD. FVC = forced vital capacity; FEV₁ = forced expired volume in 1 second; % FVC and % FEV₁ = percentage of predicted values of FVC and FEV₁, ns = non-significant

Fig. 1 Protocol. Each subject performed static apnoeas (SA) of 30 seconds and 45 seconds and then dynamic apnoeas (DA) for the same durations. The heart rate was recorded before (HR0) and after each apnoea (HR1). The arterial oxygen saturation was measured before (SaO₂ T0) and after each apnoea (SaO₂ T1).
were within the normal range of recommended reference values predicted by the ECCS standard [31].

The change in HR did not differ between BHD and ND after both 30 s and 45 s of SA and DA (Fig. 2). However, ΔHR after SA45 was greater than after DA45 for both BHD and ND (p < 0.01 and p < 0.001, respectively). Moreover, in ND, ΔHR was also greater after DA45 than after DA30 (p < 0.05) (Fig. 2).

Differences in ΔSaO₂ were noted between BHD and ND after both 30 s and 45 s of SA and DA (p < 0.05) (Fig. 3). However, the SaO₂ decrease was greater during DA than SA for both BHD and ND (Fig. 3).

We found a negative correlation between ΔHR and ΔSaO₂ in both BHD and ND (p < 0.001). The slopes were different between the two groups (−0.0785 vs. −0.1429, respectively; p < 0.001) (Fig. 4).

**Discussion**

The present study characterised the kinetics of HR and SaO₂ during calibrated exercise (0.7 m s⁻¹) in an aquatic condition, according to the indications of Delapile et al. [10]. The main findings were a smaller decrease in SaO₂ observed in breath-hold divers after both static and dynamic apnoeas compared with non-divers and a greater decrease in SaO₂ for both divers and non-divers during the dynamic apnoeas.

The validity of the oxicimeter was tested and confirmed by Benoit et al. [4]. They measured SaO₂ values during exercise under severe hypoxic conditions and concluded that the oxicimeter was able to determine SaO₂ with a standard deviation mean of 2%.

Heart rate variations and bradycardia were greater after SA than DA, with no differences between BHD and ND. This result agrees with previous studies on the effect of breath-holding on HR [2, 3, 12, 36]. The diving response observed during the apnoeic tests in dynamic conditions seemed to be a functional adaptation of the cardiovascular system in order to preserve oxygen stores [2, 36]. During these conditions, we observed an antagonist effect between exercise and apnoea on HR. The static apnoeic bradycardia was less marked than the exercise tachycardia and the net result was consequently increased HR during DA. The bradycardia and vasoconstriction were attenuated during DA compared with SA, suggesting higher oxygen consumption and carbon dioxide production. Thus, the moderate tachycardia resulted in a higher metabolic demand on the cardiac and skeletal muscles [26].

Breath-holding time is often used to estimate oxygen-conserving in humans. In this study, breath-holding time only explained the
HR difference between DA30 and DA45 in ND (p < 0.05), but had no effect on HR in any other condition. In fact, the change in HR was lower during the shorter test than the longer test only in ND. This may be explained by the inability of the non-divers to hold their breath long enough for bradycardia to develop fully. However, a close relationship between the magnitude of the diving response and breath-holding time has been observed in groups of subjects with different levels of diving experience [33,34]. In our trained divers, exercise did not increase the diving bradycardia.

We observed smaller SaO2 decreases in BHD than in ND, despite the longer breath-holding time of BHD (p < 0.05). These results confirmed previous data that linked this phenomenon to an enhanced diving reflex in divers [1,2,25]. SaO2 was more greatly reduced after DA than SA for both BHD and ND. In fact, the SaO2 reductions during SA and DA in the divers suggested that the pulmonary O2 uptake was able to cover the metabolic demand. For both DA and SA, SaO2 reached its minimal value around 30 s after the end of the apnoeic test. In agreement with Andersson and Schagatay’s proposal [1], vasoconstriction during apnoea may have been at the origin of the slow decrease in SaO2 over the 30 s following the break in breath-holding. Moreover, although simultaneous oxygen conservation (vasoconstriction due to apnoea) and oxygen consumption (vasodilatation due to exercise) occurred during DA, our SaO2 decreases were related to the end of apnoea and thus to the end of the peripheral vasoconstriction [10,14,16,35].

DA significantly reduced SaO2, an effect that was closely related to the reduction in heart rate. This finding suggests that more pronounced bradycardia could lead to lower oxygen desaturation. In fact, when cardiac output is reduced, oxygen diffusion decreases [23,28,29]. Moreover, BHD presented a smaller SaO2 reduction compared with ND. This result is partly explained by chemoreceptor adaptations. The BHD had a lower ventilatory response to CO2 than ND, which was related to diving experience [9,13,17,24]. This “low CO2 sensitivity” could affect the sensitivity of peripheral and central chemoreceptors and consequently modify the sympathetic nerve stimulation, which provokes an HR increase. This in turn would provoke a cardiac output increase and thus a greater reduction in SaO2.

Conclusions

The diving response in the breath-hold divers was oxygen-conserving, i.e. an augmented response attenuates SaO2 after 30 and 45 seconds of static and dynamic apnoeas. The slopes of the linear regression were significant differences between the two populations.

Acknowledgements

We dedicate this article to Pascal Delapilie, PhD, of the University of Rouen, who succumbed to skiing injuries in the winter of 2002. His passion for the physiology of breath-holding united us around this work.

References
