Arterial Blood Gases profiles during Breath-Hold Diving

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Background

Adaptations during voluntary breath-hold diving have been increasingly investigated since these athletes are exposed to critical hypoxemia during the ascent. However, only a limited amount of literature explored the pathophysiological mechanisms underlying this phenomenon with direct measurements. With this study we measured arterial blood gases before, at depth, and after a breath-hold dive in real conditions, to explore the variations of oxygen (O2) and carbon dioxide (CO2) developed by these athletes.

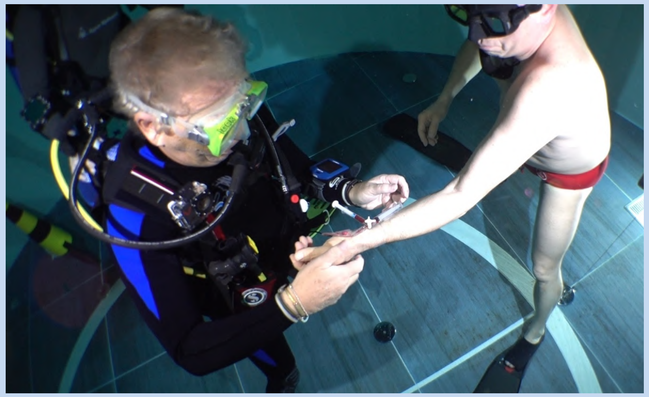

Fig. 1: arterial blood sampling at -42 m (-137.8 ft)

Methods

Six well-trained breath-hold divers were enrolled for the experiments held at the "Y-40 THE DEEP JOY" pool (Montegrotto Terme, Padova, Italy). Before the experiments, an arterial cannula was inserted in the radial artery of the non-dominant limb. All divers were involved in two experimental sessions. In the first, arterial blood samples were obtained: at rest before submersion (Pre); at depth during a sled-assisted dive at -40 m; and 2 minutes after the end of the dives. In the second session, the divers performed: a breath-hold while moving at the surface using a sea-bob (Post-SUR); a sled-assisted breath-hold dive to 42 m (Post-DP); and a breath-hold dive to 42 m with fins (Post-DP-EXE). Arterial blood samples were obtained at rest and at the end of each breath-hold, before the resumption of normal breathing.

Results

No diving-related complications were observed, and the dives had similar durations. The reduction of arterial partial pressure of oxygen (PaO2) from 96.2±7.0 mmHg (mean±SD) at rest to 64.5±4.7 mmHg after breath-hold at surface (attached to the sea-bob; Post-SUR) reflected oxygen consumption by basal metabolism and a minimal energy expenditure needed to remain in contact with the sea-bob. Interestingly, PaO2 dropped after the sled-assisted dive (39.8±8.7 mmHg; Post-DP) and especially after the dive with fins (31.6±17.0 mmHg; POst-DP-EXE) (**Fig 2**). At the bottom of the sled-assisted dive, PaO2 reached a mean value of 197.8±103.7 mmHg (**Fig 3**). The high SD in this sample is related to extremely low values found in two subjects, in which a paradoxical hypoxemia at depth was noted (respectively: 75 and 61 mmHg). Overall, such changes in PaO2 could be explained by the ventilation/perfusion mismatch and right-to-left intrapulmonary shunt caused by atelectasis at depth, in association with oxygen consumption by normal metabolism, and exacerbated by physical effort. Of note, one subject reached a PaO2 of 18 mmHg at the end of the dive with fins but without symptoms, thus reflecting intense adaptations of trained breath-hold divers to extreme hypoxemia.

The arterial partial pressure of CO2 varied somewhat, without reaching extreme values. Mean values at depth showed a slight increase but within normal ranges (42.7±12.3 mmHg) also after each step (38.2±3.0 mmHg at rest; 31.4±3.7 mmHg after the sled-assisted dive; 36.1±5.3 after the dive with fins) (**Fig 2**).

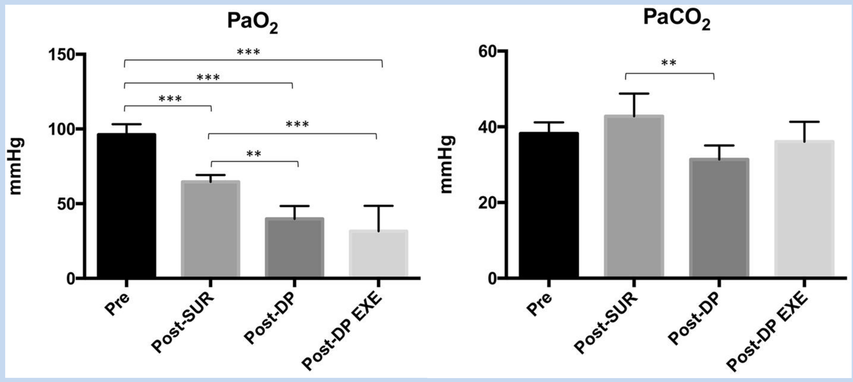

Fig. 2: arterial blood sampling before (pre) and after breath-holding. Asterisks stands for statistically significant differences between conditions

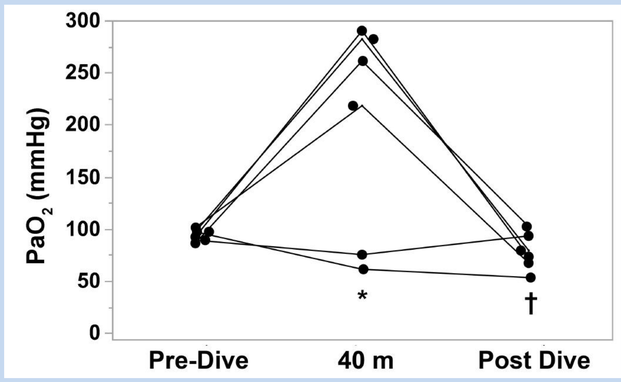

Fig. 3: arterial blood sampling before (pre), at depth, and 2 mins after the dive. *P < 0.05 vs. Pre-Dive; †P < 0.05 vs. 40 m.

Conclusions

We confirmed that the arterial partial pressure of oxygen reaches hazardously low values at the end of breath-hold, especially after the dive performed with voluntary effort, and that some subjects show a paradoxical hypoxemia at depth probably related to pulmonary atelectasis. The implications of our experiments shed light on clinical unanswered questions regarding hypoxic syncope and the risk of sudden death in breath-hold divers.