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ORIGINAL ARTICLE

Arterial blood gases in divers at surface after prolonged breath-hold

Gerardo Bosco[1] · Matteo Paganini[1] · Alex Rizzato[1] · Luca Martani[1] · Giacomo Garetto[2] · Jacopo Lion[1] · Enrico M. Camporesi[3] · Richard E. Moon[4]

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Abstract

Purpose Adaptations during voluntary breath-hold diving have been increasingly investigated since these athletes are exposed to critical hypoxia during the ascent. However, only a limited amount of literature explored the pathophysiological mechanisms underlying this phenomenon. This is the first study to measure arterial blood gases immediately before the end of a breath-hold in real conditions.

Methods Six well-trained breath-hold divers were enrolled for the experiment held at the "Y-40 THE DEEP JOY" pool (Montegrotto Terme, Padova, Italy). Before the experiment, an arterial cannula was inserted in the radial artery of the non-dominant limb. All divers performed: a breath-hold while moving at the surface using a sea-bob; a sled-assisted breath-hold dive to 42 m; and a breath-hold dive to 42 m with fins. Arterial blood samples were obtained in four conditions: one at rest before submersion and one at the end of each breath-hold.

Results No diving-related complications were observed. The arterial partial pressure of oxygen (96.2 ± 7.0 mmHg at rest, mean ± SD) decreased, particularly after the sled-assisted dive (39.8 ± 8.7 mmHg), and especially after the dive with fins (31.6 ± 17.0 mmHg). The arterial partial pressure of CO_2 varied somewhat but after each study was close to normal (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).

Conclusion 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. Critical hypoxia can occur in breath-hold divers even without symptoms.

Keywords Arterial blood gas · Blood gas analysis · Breath-hold diving · Physiology · Underwater

Abbreviations

BE-ecf	Base excess of extracellular fluid
HCO_3^-	Bicarbonate
$PaCO_2$	Arterial partial pressures of carbon dioxide
PaO_2	Arterial partial pressures of oxygen
POST DP	Arterial blood sampling after a breath-hold sled-assisted dive
POST DP-EXE	Arterial blood sampling after a breath-hold dive with fins
POST SUR	Arterial blood sampling after breath-holding while moving at the surface
PRE	Arterial blood sampling out of the water at rest
SaO_2%	Arterial hemoglobin-oxygen saturation
TCO_2	Total carbon dioxide
TLC	Total lung capacity

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✉ Matteo Paganini
paganini.mtt@gmail.com

[1] Master in Diving and Hyperbaric Medicine, Department of Biomedical Sciences, University of Padova, Via Marzolo, 3, 35131 Padova, Italy

[2] ATIP Center for Hyperbaric Medicine, Padova, Italy

[3] TEAMHealth Research Institute, Tampa General Hospital, Tampa, FL, USA

[4] Department of Anesthesiology, Center for Hyperbaric Medicine and Environmental Physiology, Duke University Medical Center, Durham, NC, USA

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Introduction

In recent years, an increasing number of publications have described the interactions of the human body with immersion at depth (Bosco et al. 2018b). Specifically, adaptations to the underwater environment during voluntary apnea (breath-hold) have been increasingly investigated to better understand the impact of such extreme environments on humans and other diving mammals. Safety concerns have been raised in light of the number of breath-hold divers who have been injured, sometimes fatally, during training or competitions (Fitz-Clarke 2006; Lindholm 2007).

After the seminal study of Lin et al. (1974), breath hold breaking points have been divided into physiological and conventional. Data obtained on the termination of voluntary apnea clarified that the breaking points are variable and subjective among the population (Parkes 2006). Interestingly, trained subjects can overcome the physiological breaking point until the development of critical hypercapnia or hypoxia (Fitz-Clarke 2018). For this reason, breath-hold divers—both at high depths or during extended dives (Lindholm and Lundgren 2009) —are exposed to ascent hypoxia, the most dangerous consequence of apnea that seriously threatens these athletes with loss of motor control or drowning (Craig 1961). Despite this, there are few published observations, most of which consist of analysis of alveolar gas by sampling exhaled air in simulated dry conditions (Craig and Harley 1968; Otis et al. 1948; Schaefer and Carey 1962), static wet apnea (Lindholm and Lundgren 2006), simulated wet dives at 20 m (Linér et al 1993), or after sea deep dives (Ferretti et al. 1991). These experiments suggest that divers experience loss of motor control or loss of consciousness when the end-tidal partial pressure of oxygen drops below 25 mmHg (Lindholm and Lundgren 2006), typically when the athlete ascends from about 10 m to the surface (Lindholm and Lundgren 2009). However, data obtained from such brief breath-hold dives or dry simulations are not necessarily predictive of what actually happens at a deeper depth (Olsen et al. 1962; Qvist et al. 1993). Only two studies have analyzed arterial blood gases during breath-hold apnea at increased ambient pressure (Bosco et al. 2018a; Muth et al. 2003). In our previous study, we confirmed for the first time in a real scenario the theoretically predicted hyperoxia at the bottom, induced by the rise in the ambient pressure (Bosco et al. 2018a). Unfortunately, we were unable to perform an arterial blood sample before the breaking of breath-hold at the surface, due to the time already spent at the bottom for the sampling. With the current experiment, we aimed to measure arterial blood gases in athletes performing breath-hold diving in real conditions, immediately before the end of breath-hold.

Methods

Subjects

Six well-trained (5 years of high-level experience, with a typical dive duration of 200 s and maximal depth of 50–60 m for all) healthy breath-hold divers (5 males, 1 female) were enrolled and medically screened. Inclusion criteria were to have no history of orthopedic, cardiovascular, renal, or metabolic disorders. Exclusion criteria were an allergy to local anesthetics, abnormal coagulation, alterations of the arterial vascularization of the upper limbs, or vasculopathy.

Experimental design

The experimental protocol was approved by the Human Ethical Committee (n° HEC-DSB/03-18) of the Department of Biomedical Science of the University of Padova and adhered to the principles of the Declaration of Helsinki. Written informed consent was obtained from all divers before enrollment in the study.

This study included repeated measures performed under four different experimental conditions. Fourteen days before the beginning of the testing sessions, a familiarization meeting was organized to ensure that all the divers knew the protocol and could complete the scheduled program. In particular, the divers were instructed about the experimental procedures to ensure their correct execution. The experiment took place at the world's deepest pool "Y-40 THE DEEP JOY" with a water temperature of 31.5 ± 0.5 °C located in Montegrotto Terme (Padova, Italy).

Before submersion, an arterial cannula was inserted in the radial artery of the non-dominant limb with the aid of local anesthesia. After a first arterial sampling at rest, all divers performed a standardized warming up consisting of two breath-hold dives: one to 10 m and the other to 25 m. Before each breath-hold, the athletes stayed at rest for at least 20 min to minimize any possible influences from the previous attempt, and then achieved relaxation through a 5-minute long controlled, non-forced ventilation at 8–9 breaths per minute (inspiratory-to-expiratory ratio of 1:2 with normal tidal volumes as perceived by each diver) and a final breath reaching total lung capacity (TLC). Neither deliberate hyperventilation nor lung-packing were performed. In the POST conditions, all blood samples were drawn immediately before inspiration, with the face still submerged. The four conditions (in order) were:

1. Blood sampling out of the water at rest, before the controlled ventilation and before any breath-hold attempts. In this phase, athletes were not hyperventilating (PRE);

2. Blood sampling after breath-holding while moving at the surface. The subject held an instructor using a propulsive sea-bob (POST SUR) (Fig. 1). The duration for this dive was determined using the mean duration of the dives from our previous experiment (Bosco et al. 2018a);
3. Blood sampling after a sled-assisted breath-hold dive to 42 m (POST DP) (Fig. 2);
4. Blood sampling after a breath-hold dive to 42 m with the diver descending and ascending with fins (Fig. 3). (POST DP-EXE). Five out of six divers completed this condition.

Fig. 1 First experimental condition (POST SUR). The subject is performing a breath-hold at surface. An instructor is driving a sea-bob while the subject is attached to him

Fig. 2 Second experimental condition (POST DP). The subject is performing a sled-assisted breath-hold dive to 42 m together with an instructor

Fig. 3 Third experimental condition (POST DP-EXE). The investigators are performing the blood sample after a breath-hold dive to 42 m with the diver descending and ascending with fins. The subject keeps her face underwater until the end of the sample as in every POST condition

During 2, 3 and 4 above, divers were accompanied by a professional instructor performing breath-hold diving too. Subjects performed such conditions in the specified order without randomization, since the 20-min rest minimized any influence from each previous attempt and subjects were not at risk of inductive bias being well-trained athletes.

Procedures for arterial access and blood collection are explained below and were performed by two anesthesiologists, an emergency physician, and a hyperbaric medicine physician as principal investigator.

Arterial access and blood collection

Arterial catheters were placed as previously reported (Bosco et al. 2018a). Briefly, under local anesthesia, an arterial cannula was inserted into the radial artery of the non-dominant limb (Fuhrman et al. 1992). After successful positioning, the arterial catheter was fixed to the skin using an adhesive band and then connected to a circuit with Luer Lock-type connectors to prevent leakage, assembled in a proximal–distal order by:

- one rigid plastic connection, single lumen, 10 cm in length;
- one lockable three-way stopcock;
- one 2.5 ml heparinized plastic syringe for standard blood gas sampling, on the first stopcock port;
- one 10 ml plastic syringe filled with 3 ml of 0.9% NaCl solution, on the second stopcock port, used both for arterial blood aspiration before sampling ("dead space") and for flushing and wash-out of the arterial line after the sample collection.

The circuit was filled with 0.9% NaCl solution and gas bubbles carefully removed. In the case of disconnection, the subject was trained to turn the stopcock to prevent bleeding.

Blood collection consisted of the following steps:

1. Opening of the second stopcock port;
2. Slow aspiration of at least 5 ml of arterial blood with the plastic syringe;
3. Holding the plunger of the syringe firmly, rotation of the three-way stopcock to open the first stopcock port;
4. With the first channel opened, slow aspiration of at least 2 ml of arterial blood into the heparinized syringe;
5. Holding the plunger of the syringe firmly, rotation of the three-way stopcock to close the first port and open the second, to flush the circuit;
6. Closing all the channels with a 45°-intermediate position of the lockable three-way stopcock.

Arterial blood samples were analyzed within 1 minute using an analyzer present on site (*i-STAT Alinity system*, Abbott Diagnostics, IL, USA). Measurements included pH, arterial hemoglobin-oxygen saturation (SaO_2%), arterial partial pressures of oxygen (PaO_2) and carbon dioxide ($PaCO_2$), bicarbonate (HCO_3^-), base excess of extracellular fluid (BE-ecf), total carbon dioxide (tCO_2), and lactate. Times of descent, ascent, and break of breath-hold were recorded. At the end of the experiment, the arterial cannula was removed under aseptic conditions, and a compression bandage applied for 2 h. The insertion area was monitored over the next 2 days for complications.

Statistical analysis

The Kolmogorov–Smirnov test was employed to check data normality distribution. One-way ANOVA for repeated measures was performed to investigate dependent variables at the four different time-points. Then, pairwise comparisons Tukey–Kramer HSD post hoc testing was used. Data analysis was performed using the software JMP Pro 13.1 (SAS Institute, Cary, NC, United States). Data are presented as mean ± standard deviation. Significant level for differences was set to $p < 0.05$.

Results

Five of six divers (characteristics are reported in Table 1) completed this study in all conditions. One diver did not complete the last condition because he reported pain at the site of arterial cannulation and the arterial cannula was removed. All subjects completed the trials with no neurological symptoms, loss of consciousness, or evidence of pulmonary barotrauma. The circuit remained intact during all the tests, and no complications were reported after the experiment.

Breath-hold time typically ranged between 94 and 127 s (see Table 2 for a more detailed description). Performance duration (i.e., time necessary to perform the ascent and the descent phase), ABG time (i.e., time at which samples were drawn), and break of breath-hold time (the time at which subject returned to breathing) are reported in Table 2. Blood gas analysis results are shown in Table 3, and depicted in Fig. 4. Levels of PaO_2 progressively and significantly decreased between the PRE and all POST conditions. POST SUR PaO_2 was significantly higher than POST DP ($p < 0.01$) and POST DP-EXE, ($p < 0.001$). $PaCO_2$ was significantly higher in the POST SUR (42.8 ± 6.0 mmHg) than POST DP (31.4 ± 3.7 mmHg, $p < 0.01$). There were variable changes in arterial lactate after the descent, with POST DP-EXE value significantly higher than POST SUR ($p < 0.001$), POST DP ($p < 0.001$), and PRE ($p < 0.001$).

Table 1 The mean values ± SD, ($n = 6$) for age, body mass, height, and Body Mass Index (BMI) are indicated

Subject	Age (years)	Mass (kg)	Height (m)	BMI (kg/m^2)	Blood pressure at rest (systolic/diastolic; mmHg)
1	29	74	1.81	22.59	135/85
2	42	79	1.82	23.85	110/70
3	50	63	1.70	21.80	110/70
4	53	80	1.80	24.69	130/70
5	38	78	1.91	21.38	130/85
6	38	63	1.70	21.80	125/90
Mean	42	73	1.79	22.68	123/78
SD	9	8	0.08	1.32	11/9

Values of blood pressure registered at rest before starting the trials are also reported

Table 2 The table provides the specific time-profile during breath-hold dive at the surface (SUR), after the 42-m dive with sled assistance (DP), and after the 42-m dive with fins (DP-EXE). ABG: arterial blood gas

Subject	POST SUR			POST DP			POST DP-EXE		
	Performance duration (s)	ABG (s)	Breath-hold end (s)	Performance duration (s)	ABG (s)	Breath-hold end (s)	Performance duration (s)	ABG (s)	Breath-hold end (s)
1	100	100	106	87	95	95	93	95	93
2	96	96	99	89	102	108	na	na	na
3	92	92	95	90	113	116	111	125	127
4	96	96	99	94	111	113	102	113	113
5	92	92	96	96	107	111	108	123	108
6	90	90	94	99	113	115	110	119	119
Mean	94.3	94.3	98.2	92.5	106.8	109.7	104.8	115.0	112.0
SD	3.7	3.7	4.4	4.6	7.2	7.7	7.5	12.1	12.8

Table 3 Mean (± standard deviation) and ranges (min–max) of arterial partial pressure of carbon dioxide ($PaCO_2$), arterial oxygen saturation (SaO_2%), arterial partial pressure of oxygen (PaO_2), pH, bicarbonate (HCO_3^-), total carbon dioxide (tCO_2), and lactate (Lac)

	Pre		POST SUR		POST DP		POST DP-EXE	
	Mean ± SD	Range (min–max)	Mean ± SD	Range (min–max)	Mean ± SD	Range (min–max)	Mean ± SD	Range (min–max)
pH	7.43 ± 0.02	7.38–7.45	7.40 ± 0.05	7.31–7.44	7.46 ± 0.04	7.42–7.51	7.43 ± 0.05	7.39–7.50
$PaCO_2$ (mmHg)	38.2 ± 3.0	35.2–42.4	42.8 ± 6.0	36.7–50.8	31.4 ± 3.7	26.1–34.9	36.1 ± 5.3	31.3–43.1
PaO_2 (mmHg)	96.2 ± 7.0	83.0–102.0	64.5 ± 4.7	56.0–68.0	39.8 ± 8.7	26.0–49.0	31.6 ± 17.0	18.0–61.0
HCO_3- (mmol/L)	25.2 ± 2,2	22.9–29.2	26.4 ± 1.9	24.9–30	22.2 ± 1.6	20.0–24.8	24.0 ± 3.0	19.6–26.7
SaO_2%	97.7 ± 0.5	97.0–98.0	92.0 ± 2.1	89.0–94.0	75.8 ± 11.4	57.0–85.0	55.6 ± 24.1	27.0–91.0
tCO_2 (mmol/L)	26.2 ± 2.1	24.0–30.0	27.5 ± 1.9	26.0–31.0	23.3 ± 1.8	21.0–26.0	25.2 ± 3.1	21.0–28.0
Lac (mmol/L)	0.64 ± 0.38	0.41–1.39	0.79 ± 0.17	0.65–1.08	1.18 ± 0.17	0.96–1.39	2.09 ± 0.35	1.74–2.59

Discussion

The results of this study have revealed blood gases immediately after the end of a breath-hold, both at the surface and during a 42 m dive.

As expected, PaO_2 decreased after each breath-hold, with significant variation among the different experimental conditions (Table 3). The reduction of PaO_2 after POST SUR reflected oxygen consumption by basal metabolism and a minimal energy expenditure needed to remain in contact with the instructor/sea-bob system. In 1996, Sasse sampled arterial blood of eight subjects performing a dry breath-hold from functional residual capacity on room air, and demonstrated a mean drop of 49.8 mmHg in PaO_2 after 35 (± 10.1) s (Sasse et al. 1996). In spite of a longer breath-hold at surface (94.3 ± 3.7 s), we registered a lesser degree of PaO_2 reduction that has two possible explanations. First, we hypothesize that the diving reflex could have slowed the drop in PaO2 during the breath-hold through bradycardia, blood shift, and spleen contraction as widely demonstrated before (Bosco et al. 2018b). Second, reaching TLC, our free-divers had more air available than the subjects in Sasse's experiment, thus providing more oxygen to be stored in the blood.

Since POST SUR and POST DP conditions had similar durations of breath-hold (see Table 2) and no physical effort—except for the comparable and minimal muscular activity needed to hold the instructor/sea-bob system or the sled, respectively—the former can be considered a control. PaO_2 values considerably fell after the sled-assisted breath-hold dive at 42 m (Table 3). The statistically significant difference in PaO_2 between POST SUR and POST DP conditions can be explained by the effects exerted by environmental pressure on the breath-hold divers during the latter. In fact, the subjects inhaled the same FiO_2 (on room air), attempted not to deliberately hyperventilate, and reached TLC before the breath-hold in all conditions. We thus attempted to maintain pre-experiment conditions the same, although we cannot exclude the possibility that blood

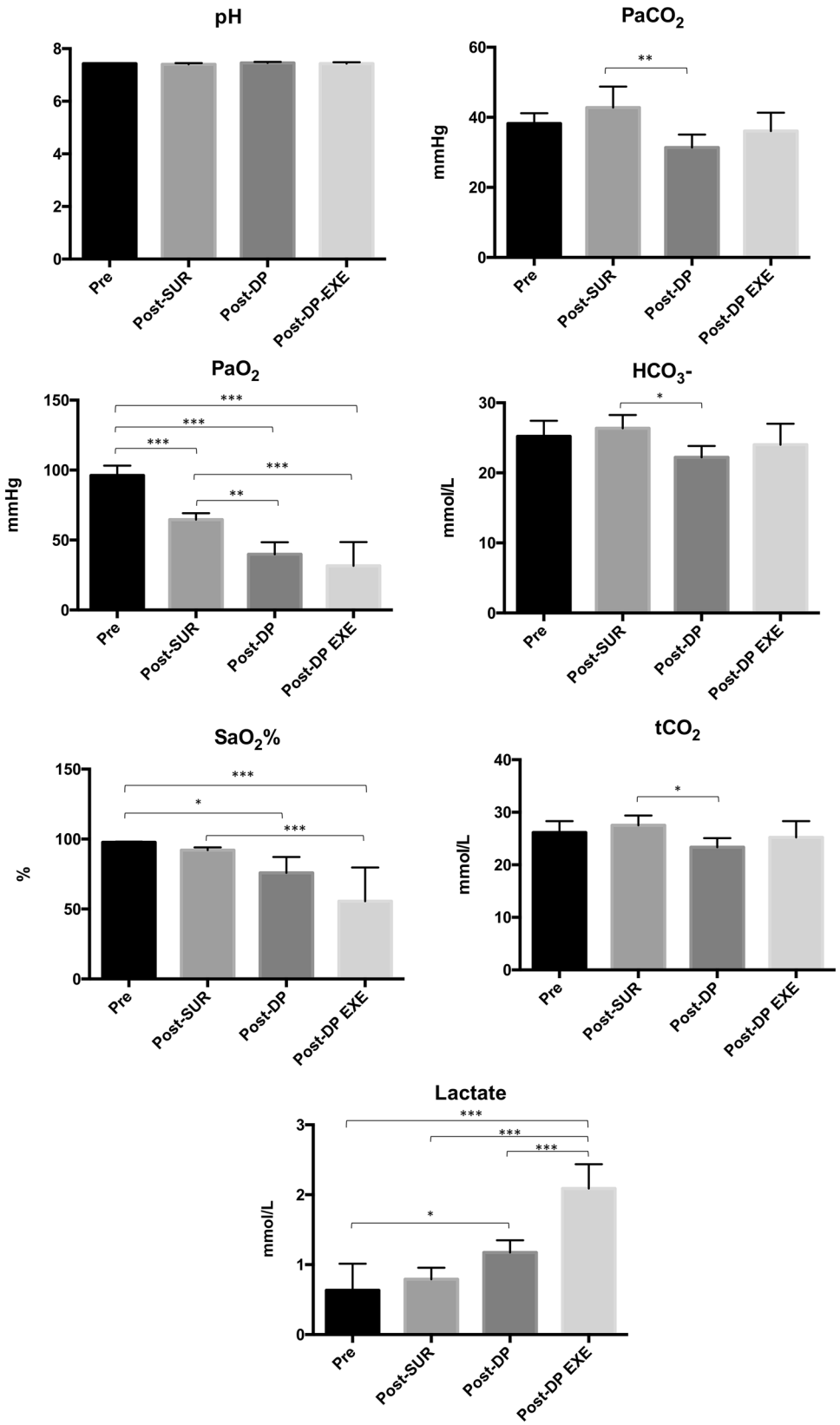

Fig. 4 Comparisons between the different time-point (PRE: sampling at rest; POST SUR: sampling after breath-holding at surface; POST DP: sampling after the sled-assisted breath-hold dive to 42 m; and POST DP-EXE: sampling after the breath-hold dive to 42 m with fins) are reported for all the parameters of arterial blood gas analysis. Vertical bars represent standard deviation. *Significantly different ($p < 0.05$), **significantly different ($p < 0.01$), and ***significantly different ($p < 0.001$). PaO_2 arterial partial pressures of oxygen, $PaCO_2$ arterial partial pressures of carbon dioxide, HCO_3- bicarbonate, $SaO_2\%$ arterial hemoglobin-oxygen saturation, tCO_2 total carbon dioxide

gases before POST SUR and POST DP were not identical. Although not entirely excludable, an unintended increased O_2 consumption due to muscular contraction while diving is improbable with the use of the sled that limited physical efforts. However, Linér et al. (1993) observed higher oxygen consumption at depth, which they attributed to an increase in cardiac output due to the reduction in transthoracic pressure at depth. Another possible explanation for our findings may be the ventilation/perfusion mismatch and right-to-left intrapulmonary shunt caused by atelectasis at depth as previously described (Bosco et al. 2018a; Fitz-Clarke 2018), in association with oxygen consumption by normal metabolism (Muth et al. 2003).

The introduction of exercise during the last breath-hold dive attempt caused a dramatic drop in mean PaO_2 (Table 3: POST DP-EXE), the lowest values of the experiment. When compared to POST DP, exertion and increased oxygen consumption rate could explain these lower values and could be related to an increased risk of critical and fatal injuries during breath-hold (Craig 1976; Lindholm 2006).

Despite PaO_2 values as low as 18.0 mmHg (SaO_2% as low as 27%) measured in POST DP-EXE, no one lost consciousness or had any adverse effects, which might reflect already described hypoxic adaptations in this well-trained population (Ferretti et al. 1991). However, this combination of findings confirms the risk of ascent hypoxia encountered during breath-hold diving. Future experiments should focus on demonstrating or rejecting the possible association between increased risk of injuries and exertion during breath-hold diving.

$PaCO_2$ levels varied somewhat, but at the end of the breath-hold were remarkably close to normal values (Table 3). This is consistent with end-tidal measurements in ama divers reported by Hong et al. (1963), even if our experiment accounted for longer and deeper dives. A significant difference was found between mean $PaCO_2$ in POST SUR and POST DB conditions (Fig. 4), but the value of this finding is questionable. In fact, it is known that arterial PCO_2 values are highly influenced by respiratory rates and volumes. Despite performance of a standard preparation before each attempt—with controlled ventilations and without deliberate hyperventilation or lung-packing—it is possible that unnoticed variations in rate or volume while preparing could have resulted in the observed difference. If anticipation of the 42 m dives resulted in unintended predive hypocapnia this could explain why arterial PCO_2 was lower after the dives than after breath-holding at the surface. Higher arterial PCO_2 after the fin-propelled dives compared with POST DP is consistent with higher CO_2 production rate. Future experiments should take into account this problem, reducing a possible source of error using for example spirometers or detecting end-tidal/arterial CO_2 partial pressures.

Overall, even with different experimental conditions, the values of PaO_2 and $PaCO_2$ observed at the break of breath-hold match those measured in earlier studies. For example, after a wet, static apnea in a hyperbaric chamber, Muth found a comparable reduction in PaO_2, while $PaCO_2$ exhibited moderate variation (Muth et al. 2003). Similarly, in our previous study, O_2 and CO_2 showed the same trend at the end of the breath-hold dive, albeit the results were limited by initiating breathing before the blood sampling (Bosco et al. 2018a). With the current experiment, we chose to avoid the blood sampling at the bottom to gain time to perform an arterial blood sample at the surface, thus removing the confounding effect as in the previous experiment (Bosco et al. 2018a). Our previous study on six subjects demonstrated that $PaCO_2$ and PaO_2, sampled at 5 ATA, were indeed proportionally increased (Bosco et al. 2018a).

The slight elevation of blood lactate after POST DP-EXE is consistent with muscular activity, and did not occur in the other conditions without exertion (Table 3).

The generalizability of these results is subject to certain limitations. For instance, the number of subjects enrolled was limited due to feasibility but comparable with previous studies in the field. While PaO_2 in this study was such that no one lost consciousness or experienced loss of motor control, it should be emphasized that these subjects were all experienced breath-hold divers. Given this limitation, results may not reflect performance or gas exchange in a naïve subject. On the other hand, the breath-hold durations observed (less than 2 min) are not at an elite level, and thus our findings may not be applicable to the general population of breath-hold divers. Finally, arterial blood was not sampled immediately before each dive, assuming the PRE sample as a control at rest. We attempted to control for this important limitation by a standardized preparation before each attempt, although there could have been some variability in PaO_2 and $PaCO_2$ values as previously explained.

Unlike open water breath-hold dives where water temperate is generally cold at depth, these experiments were performed in warm water. To our knowledge there are no studies of the effect of water temperature on gas exchange in breath-hold divers. Cold water on the face may induce bradycardia (and hence reduced cardiac output), which could conceivably attenuate the rates at which $PaCO_2$ and PaO_2 rise and fall at maximum depth. Cold water might induce shivering, which would increase oxygen consumption and carbon dioxide production. On the other hand, it could be speculated that temperature would have a minimal effect since most open water dives are brief (no more than a few minutes in duration) and divers wear thermal protection (except on the face).

Conclusion

Breath-hold divers are at risk of serious adverse events while practicing this extreme sport. Especially during the ascent phase, low O_2 levels were to date only estimated to cause severe neurologic symptoms. With this study, for the first time we measured arterial blood gases in real environmental conditions and confirmed that the PaO_2 reaches hazardously low values at the end of a breath-hold dive, especially after physical exercise. These results represent a warning regarding the dangers of prolonged breath-hold, and more research is needed to understand how critical hypoxia can be prevented in breath-hold divers.

Author contributions GB, EC, RM, and MP conceived and designed the experiments. GB, AR, LM, GG, MP, and JL performed the experiments. MP, RM, and AR analyzed the data. GB contributed the materials. GB, AR, MP, EC, and RM wrote the paper. All authors read and approved the manuscript.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

References

Bosco G, Rizzato A, Martani L, Schiavo S, Talamonti E, Garetto G, Paganini M, Camporesi EM, Moon R (2018a) Arterial blood gas analysis in breath-hold divers at depth. Front Physiol 5(9):1558. https://doi.org/10.3389/fphys.2018.01558

Bosco G, Rizzato A, Moon RE, Camporesi EM (2018b) Environmental physiology and diving medicine. Front Psychol 2(9):72. https://doi.org/10.3389/fpsyg.2018.00072

Craig AB (1961) Causes of loss of consciousness during underwater swimming. J Appl Physiol 16:583–586. https://doi.org/10.1152/jappl.1961.16.4.583

Craig AB Jr (1976) Summary of 58 cases of loss of consciousness during underwater swimming and diving. Med Sci Sports 8(3):171–175

Craig AB, Harley AD (1968) Alveolar gas exchanges during breath-hold dives. J Appl Physiol 24(2):182–189. https://doi.org/10.1152/jappl.1968.24.2.182

Ferretti G, Costa M, Ferrigno M, Grassi B, Marconi C, Lundgren CE, Cerretelli P (1991) Alveolar gas composition and exchange during deep breath hold diving and dry breath holds in elite divers. J Appl Physiol 70(2):794–802. https://doi.org/10.1152/jappl.1991.70.2.794

Fitz-Clarke JR (2006) Adverse events in competitive breath-hold diving. Undersea Hyperb Med 33(1):55–62

Fitz-Clarke JR (2018) Breath-hold diving. Compr Physiol 8(2):585–630. https://doi.org/10.1002/cphy.c160008

Fuhrman TM, Pippin WD, Talmage LA, Reilley TE (1992) Evaluation of collateral circulation of the hand. J Clin Monit 8(1):28–32

Hong SK, Rahn H, Kang DH, Song SH, Kang BS (1963) Diving pattern, lung volumes, and alveolar gas of the Korean diving woman (ama). J Appl Physiol (1985) 18(3):457–465

Lin YC, Lally DA, Moore TO, Hong SK (1974) Physiological and conventional breath-hold breaking points. J. Appl Physiol 37(3):291–296. https://doi.org/10.1152/jappl.1974.37.3.291

Lindholm P (2006) Physiological mechanisms involved in the risk of loss of consciousness during breath-hold diving. In: Lindholm P, Pollock NW, Lundgren CEG (eds) Breath-hold diving. Divers Alert Network, Durham, pp 26–31

Lindholm P (2007) Loss of motor control and/or loss of consciousness during breath-hold competitions. Int J Sports Med 28(4):295–299. https://doi.org/10.1055/s-2006-924361

Lindholm P, Lundgren CE (2006) Alveolar gas composition before and after maximal breath-holds in competitive divers. Undersea Hyperb Med 33(6):463–467

Lindholm P, Lundgren CE (2009) The physiology and pathophysiology of human breath-hold diving. J Appl Physiol 106(1):284–292. https://doi.org/10.1152/japplphysiol.90991.2008

Linér MH, Ferrigno M, Lundgren CE (1993) Alveolar gas exchange during simulated breath-hold diving to 20 m. Undersea Hyperbaric Med 20(1):27–38

Muth CM, Radermacher P, Pittner A, Steinacker J, Schabana R, Hamich S, Paulat K, Calzia E (2003) Arterial blood gases during diving in elite apnea divers. Int J Sports Med 24(2):104–107. https://doi.org/10.1055/s-2003-38401

Olsen CR, Fanestil DD, Scholander PF (1962) Some effects of apneic underwater diving on blood gases, lactate, and pressure in man. J Appl Physiol 17:938–942. https://doi.org/10.1152/jappl.1962.17.6.938

Otis AB, Rahn H, Fenn WO (1948) Alveolar gas changes during breath holding. Am J Physiol 152(3):674–986. https://doi.org/10.1152/ajplegacy.1948.152.3.674

Parkes MJ (2006) Breath-holding and its breakpoint. Exp Physiol 91(1):1–15. https://doi.org/10.1113/expphysiol.2005.031625

Qvist J, Hurford WE, Park YS, Radermacher P, Falke KJ, Ahn DW, Guyton GP, Stanek KS, Hong SK, Weber RE, Zapol WM (1993) Arterial blood gas tensions during breath-hold diving in the Korean ama. J Appl Physiol 75(1):285–293. https://doi.org/10.1152/jappl.1993.75.1.285

Sasse SA, Berry RB, Nguyen TK, Light RW, Mahutte CK (1996) Arterial blood gas changes during breath-holding from functional residual capacity. Chest 110(4):958–964

Schaefer KE, Carey CR (1962) Alveolar pathways during 90-foot, breath-hold dives. Science 137(3535):1051–1052