

Oxygen-enriched Air Decreases Ventilation during High-intensity Fin-swimming Underwater



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ABSTRACT

Oxygen-enriched air is commonly used in the sport of SCUBA-diving and might affect ventilation and heart rate, but little work exists for applied diving settings. We hypothesized that ventilation is decreased especially during strenuous underwater fin-swimming when using oxygen-enriched air as breathing gas. Ten physically-fit divers (age: 25 ± 4 ; 5 females; 67 ± 113 open-water dives) performed incremental underwater fin-swimming until exhaustion at 4 m water depth with either normal air or oxygen-enriched air (40% O₂) in a double-blind, randomized within-subject design. Heart rate and ventilation were measured throughout the dive and maximum whole blood lactate samples were determined post-exercise. ANOVAs showed a significant effect for the factor breathing gas ($F(1, 9) = 7.52$; $P = 0.023$; $\eta^2_p = 0.455$), with a lower ventilation for oxygen-enriched air during fin-swimming velocities of $0.6 \text{ m} \cdot \text{s}^{-1}$ ($P = 0.032$) and $0.8 \text{ m} \cdot \text{s}^{-1}$ ($P = 0.037$). Heart rate, lactate, and time to exhaustion showed no significant differences. These findings indicate decreased ventilation by an elevated oxygen fraction in the breathing gas when fin-swimming in shallow-water submersion with high velocity ($> 0.5 \text{ m} \cdot \text{s}^{-1}$). Applications are within involuntary underwater exercise or rescue scenarios for all dives with limited gas supply.

Introduction

Ventilation (\dot{V}_E) is a critical factor for the duration and safety of a dive and might be affected by the inspiratory fraction of oxygen (F_{iO_2}) in the breathing gas. This has already been reported in land-based studies for rest and exercise [1–4], but only little work exists for the context of sport-diving with SCUBA (self-contained underwater breathing apparatus) [5–7], where breathing gases with an elevated oxygen

content are commonly used [8] and the amount of gas consumed per breath increases with depth. Several diving-specific factors, like exertion during fin-swimming or physiological adaptations underwater might influence this interaction in an applied setting.

Oxygen enriched air (EAN; i. e. inspiratory oxygen fraction (F_{iO_2}) above 21%) is the second most-used breathing gas besides air in sport-diving, with the main purpose to replace nitrogen (N₂) with

oxygen (O_2). When performing the same dive with EAN instead of air (AIR), N_2 tissue saturation and decompression risks are reduced and no-decompression dive-times prolonged [8, 9]. One detrimental effect of oxygen-enriched air is oxygen toxicity (i. e. inspiratory oxygen partial pressure ($P_I O_2$) > 140 kPa), which is known as a limiting factor for $F_I O_2$ in the mix and maximum depth during the dive [8, 10]. The risk of nitrogen narcosis, which affects responsiveness, well-being, and cognitive performance under high PN_2 [11–14], is reduced when diving with EAN. Thus, diving with EAN as a breathing gas has become extremely popular, especially if many repetitive dives are performed.

A higher $F_I O_2$ and $P_I O_2$, respectively, in the lungs (i. e. due to a raised $F_I O_2$ in the breathing gas) results in a higher arterial oxygen partial pressure ($P_a O_2$) and slightly more O_2 in the blood. In healthy subjects, this increases solely the concentration of physically dissolved O_2 , which is marginal compared to hemoglobin-bound O_2 . However, O_2 delivery is a limiting factor for increased workload and especially for transitions in work rate, like during incremental exercise until exhaustion. In those scenarios, an increased O_2 delivery (from an increase in $P_a O_2$) might be beneficial for O_2 diffusion to the muscle and therefore delay the metabolic acidosis from lactate accumulation. This is in line with several studies reporting decreased lactate accumulation during hyperoxic exercise on the bicycle [15–20]. Furthermore, the over-proportional increase in \dot{V}_E , among other things as a result of metabolic acidosis, might be postponed. This can explain higher peak workloads or longer submaximal exercise times [16, 17, 21–23]. Also, a lower perceived exertion was stated for hyperoxic exercise [24, 25].

Peripheral arterial chemoreceptors, like in the carotid bodies, regulate \dot{V}_E by monitoring PO_2 , PCO_2 , and pH. Whereas hypoxemia leads to an increase of \dot{V}_E during rest, hyperoxia (i. e. higher $P_a O_2$) attenuates the sensitivity of those receptors [1, 4, 26]. Furthermore, a lower heart rate (HR) was observed in hyperoxia during rest [27, 28] as a result of enhanced vascular resistance and during exercise [3], where a reduced sympathetic activation was suspected as the modulating factor at steady-state exercise intensities.

In submersion studies with moderate exercise on a bicycle ergometer, a lower \dot{V}_E was observed at depth (470 kPa ambient pressure) with a $P_I O_2$ of 21 kPa [5], which suggests the sole influence of submersion, and under hyperoxia (175 kPa $P_I O_2$) [5, 6]. These studies observed no effects on \dot{V}_E between 70 kPa and 130 kPa $P_I O_2$, which might be attributed to the counteracting effects of breathing gas density at depth. However, most findings so far are from cycle ergometer experiments in the laboratory or a pressure chamber and did not consider specific sport-diving aspects like exercise modality (i. e. fin-swimming vs. cycling), the body-position in the water (i. e. upright vs. supine), or immersion with potential influences on physiological reactions such as metabolism, blood shift, and breathing resistance. In combination with an increased $F_I O_2$ in the breathing gas (i. e. 56 kPa at 4 m water depth), exercise modality might affect O_2 consumption and whole blood lactate production, at least in transient phases, and therefore the respiratory drive. In turn, factors like \dot{V}_E , HR, perceived exertion, and time to exhaustion (TTE) might be affected in an applied context during incremental exercise. Therefore, former findings need verification in an applied field setting for sport diving.

In this study, we investigated the effects of either AIR (21 % O_2) or EAN (40 % O_2) as breathing gas on \dot{V}_E , HR, lactate concentration [Lac^-], and TTE during underwater incremental fin-swimming. We hypothesized, that \dot{V}_E and HR are lowered for EAN at given fin-swimming speeds. Additionally, lower [Lac^-] after exhaustive underwater fin-swimming was expected.

Materials and Methods

Participants

An a priori sample size calculation (G * Power 3.1.9.2) demanded a total sample size of 10 participants to obtain an effect size $f = 0.5$ and power of $1 - \beta = 0.80$ with a mixed design and a significance level of $\alpha < 0.05$. Eleven healthy, young, and physically-fit sport students participated in the study (see ► **Table 1**). One female participant did not complete the velocity of $0.8 \text{ m} \cdot \text{s}^{-1}$ for both conditions and was excluded from the analysis ($N = 10$; 25 ± 4 years (mean \pm SD); 5 females). Each participant performed two test conditions with incremental underwater fin-swimming until exhaustion, breathing either AIR or EAN. All tests were conducted in an indoor pool ($20 \times 20 \times 5$ meters) in approximately 4 m water depth. A valid medical examination for diving and diving experience of five or more open-water dives was mandatory for participation. All divers were informed about the purpose and design of the study and signed an informed consent form before participation. Termination of participation was possible at all times without reasoning. An ethics committee, following the declaration of Helsinki, approved the study [29].

Study Design

All participants performed two test conditions in a crossover, randomized and double-blind design with a mandatory interval of 2–7 days to ensure full physical recovery. Neither the test conductor nor the participants knew which gas was used in which test. Only the study supervisor knew the organization of test conditions and gas logistics. In one condition, the breathing gas was normal air (AIR), and in the other oxygen-enriched air (40 % O_2 , enriched air Nitrox: EAN). Test conditions differed solely by the breathing gas (AIR, EAN). The diving gear was provided and consisted of a 3 mm wetsuit, a 10 L steel tank, a commercially available buoyancy control device (BCD), a breathing regulator, mask and fins.

The fit2dive-test's underwater exercise parcours [30] was utilized for incremental fin-swimming. The test was developed to enable specific exercise testing for sport divers, taking into account specific locomotion, equipment-induced water drag, and fin-swimming technique. The parcours consisted of a 50 m-long rope that was anchored to the bottom of the pool in the shape of a hexagon. Checkpoints were marked with a buoy to allow for self-controlled fin-swimming velocity with a marching-table and stopwatch. After a mandatory round of slow fin-swimming for familiarization, participants then started swimming under the supervision of the test conductor. Fin-swimming velocity was increased in 150 s-long steps of $0.4 \text{ m} \cdot \text{s}^{-1}$, $0.6 \text{ m} \cdot \text{s}^{-1}$, $0.8 \text{ m} \cdot \text{s}^{-1}$, and $1.0 \text{ m} \cdot \text{s}^{-1}$. The test ended if two consecutive checkpoints could not be reached according to the marching chart. Participants then surfaced together with the test conductor and stated their rating of perceived exertion (RPE) on a scale ranging from "very light" (6) to "full exertion" (20) [31].

► **Table 1** Table shows individual values, mean values, and standard deviation for the number of open-water dives, overall and specific fin-swimming self-stated fitness level, and weekly training hours for all participants (N = 10).

Age [years]	Weight [kg]	Height [cm]	Open-water dives	physical training [h per week]	overall fitness level [self-stated]	fin-swimming fitness level [self-stated]
23	67	175	72	12	good	good
25	78	173	12	10	medium	medium
24	70	170	35	3	medium	medium
23	82	185	25	3	good	good
22	61	168	25	1	medium	medium
21	68	180	14	6	good	good
22	55	167	400	10	good	good
23	78	177	65	10	good	good
27	82	176	5	5	good	medium
36	72	178	14	2	medium	bad
25	71	175	67	6		
4	8	5	113	4		

Measurements

Process during incremental exercise was tracked by the test conductor. The maximum time to exhaustion was noted for every participant and condition. HR and tank pressure was recorded with a heart-rate-belt and pressure transmitter, respectively. Recordings were made every 4 s and stored on the dive computer for later analysis. Before the start of physical exercise, a 3-min HR-baseline measurement was recorded in the supine position at 5 m water depth.

Whole blood [Lac⁻] was determined from earlobe blood samples once before submersion (i. e. after 30 min of rest; baseline) and five times with one-minute intervals after exercise, the first sample taken immediately after the termination of exercise and surfacing with the participants (i. e. approx. 15 s after exercise). The maximum lactate concentration [Lac⁻]_{max} was determined as the highest value out of the five samples taken after exercise. The samples were stored cooled and analyzed the same day in the laboratory.

Data Processing

\dot{V}_E was calculated for ambient pressure using tank-pressure and depth recordings from the diving computer. In our formula, \dot{V}_E [L · min⁻¹] is the amount of gas consumed per minute, ΔP_{tank} [kPa] is the difference in tank pressure throughout 40 s, V_{tank} [L] is the volume of the tank, and P_{depth} [kPa] is the mean of depth reading (recorded every 4 s) throughout 40 s multiplied by 10 kPa + 100 kPa. Means for \dot{V}_E and HR were determined during the last 20 s of every velocity.

$$\dot{V}_E = \Delta P_{\text{tank}} \times V_{\text{tank}} \times P_{\text{depth}}^{-1}$$

The second ventilatory threshold (VT2) was estimated for both conditions by three experienced evaluators as the point of over-proportional increase of \dot{V}_E in relation to HR (see ► **Fig. 1**), marking rapid lactate increase and hyperventilation [32]. VT2 was utilized to gain information on the participants' metabolism during exercise, as we could not perform spiroergometric measurements or take lactate samples underwater.

► **Table 2** shows means and standard deviations for \dot{V}_E , HR, and time at VT2 for the three independent estimations. Figures show means and 95 % confidence intervals. Mean values for \dot{V}_E and HR throughout the exercise were calculated over the last 20 s at rest and for every fin-swimming velocity.

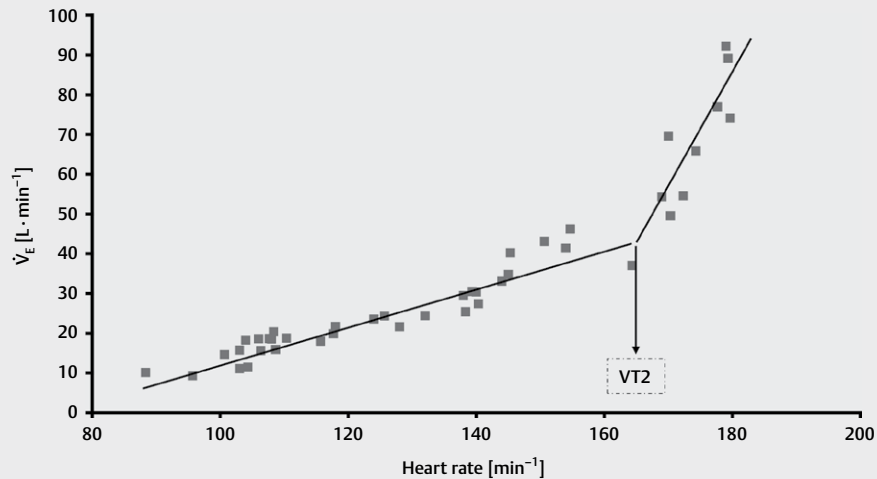
Statistics

Using SPSS statistics 25 (IBM, USA), all variables were checked for a violation of normal distribution using the Shapiro-Wilks test. Alpha was set to 0.05. Two-way ANOVAs with repeated measures on the factors gas (AIR, EAN) and velocity (Rest, 0.4 m · s⁻¹, 0.6 m · s⁻¹, and 0.8 m · s⁻¹) were calculated for HR, \dot{V}_E and [Lac⁻]. Multiple mean value comparisons according to Bonferroni and one-tailed pairwise comparisons were used to investigate significant results. The correlation between [Lac⁻]_{max} and TTE was investigated using the correlation coefficient from Spearman. For main-effects, effect-sizes were estimated using partial eta squared (η^2_p), where values = 0.01 indicate a small effect, values = 0.06 a medium effect, and values = 0.14 a large effect. For post-hoc comparisons, Cohen's *d* was computed as the quotient from the difference between two means and the mean standard deviation, where values between 0.2 and 0.5 indicate a small effect, values between 0.5 and 0.8 indicate a medium effect, and values > 0.8 indicate a large effect [33].

Results

The analysis of \dot{V}_E produced significant main effects for the factor gas ($F(1, 9) = 7.52$; $P = 0.023$; $\eta^2_p = 0.455$) and velocity ($F(1.25, 11.21) = 39.59$; $P < 0.001$; $\eta^2_p = 0.815$). An interaction effect for gas * velocity could not be found ($F(1.51, 13.52) = 2.52$; $P = 0.126$; $\eta^2_p = 0.220$). Post-hoc, values for EAN were significantly lower compared to AIR during the velocities of 0.6 m · s⁻¹ ($P = 0.032$; $d = 1.02$) and 0.8 m · s⁻¹ ($P = 0.037$; $d = 0.47$). Participants reached VT2 at a similar time during exercise (AIR, EAN) with no significant differences for HR and \dot{V}_E (see ► **Fig. 2** and ► **Table 1**).

HR showed a significant main effect for velocity ($F(1.75, 15.74) = 154.27$; $P < 0.001$; $\eta^2_p = 0.945$), but not for gas ($F(1,9) = 0.691$; $P = 0.427$; $\eta^2_p = 0.071$) or gas * velocity ($F(1.58, 14.18) = 0.453$; $P = 0.599$;



► **Fig. 1** Data shows the determination of the ventilatory threshold at the disproportional increase of Ventilation (\dot{V}_E) in ratio to heart rate (HR). Example of one subject.

► **Table 2** Mean values for ventilation (\dot{V}_E [L]) and heart rate (HR [min^{-1}]) were calculated for the last 20 seconds of every velocity (Rest, 0.4, 0.6, and 0.8 $\text{m} \cdot \text{s}^{-1}$). The maximum whole blood lactate [Lac^-] $_{\text{max}}$ [$\text{mmol} \cdot \text{L}^{-1}$] was the highest lactate sample taken after incremental exercise. The maximum time to exhaustion (TTE) was the time accomplished during incremental exercise. The rating of perceived exertion (RPE) was stated directly after exercise. HR, \dot{V}_E , and time for ventilatory threshold (VT2) were estimated by three experienced evaluators. Table shows means \pm standard deviation. * $P < 0.05$ for comparisons of breathing gases (AIR vs. EAN).

	Variable		EAN [40% O ₂]	AIR [21% O ₂]
	Rest	[Lac ⁻]	[mmol · L ⁻¹]	1.31 ± 0.5
HR		[bpm]	96 ± 12	96 ± 13
\dot{V}_E		[L · min ⁻¹]	17 ± 7	18 ± 6
0.4 [$\text{m} \cdot \text{s}^{-1}$]	HR	[bpm]	111 ± 8	108 ± 14
	\dot{V}_E	[L · min ⁻¹]	22 ± 4	22 ± 6
0.6 [$\text{m} \cdot \text{s}^{-1}$]	HR	[bpm]	145 ± 14	140 ± 19
	\dot{V}_E^*	[L · min ⁻¹]	35 ± 7	44 ± 12
0.8 [$\text{m} \cdot \text{s}^{-1}$]	HR	[bpm]	171 ± 10	170 ± 14
	\dot{V}_E^*	[L · min ⁻¹]	63 ± 26	74 ± 25
VT2	HR	[bpm]	149 ± 19	153 ± 13
	\dot{V}_E	[L · min ⁻¹]	34 ± 10	38 ± 10
	Time	[s]	327 ± 74	344 ± 83
Max	RPE	[a.u.]	16 ± 2	15.9 ± 1.7
	TTE	[s]	480 ± 62	480 ± 71
	[Lac ⁻] $_{\text{max}}$	[mmol · L ⁻¹]	6.9 ± 1.2	7.1 ± 1.4

$\eta^2_p = 0.048$). [Lac⁻] did not show any significant variations between EAN and AIR ($F(1, 9) = 0.16$; $P = 0.699$; $\eta^2_p = 0.017$, see ► **Fig. 3**). No difference for TTE or RPE during exercise was found between conditions ($F(1, 9) = 0.037$; $P = 0.852$; $\eta^2_p = 0.004$, see ► **Table 2**).

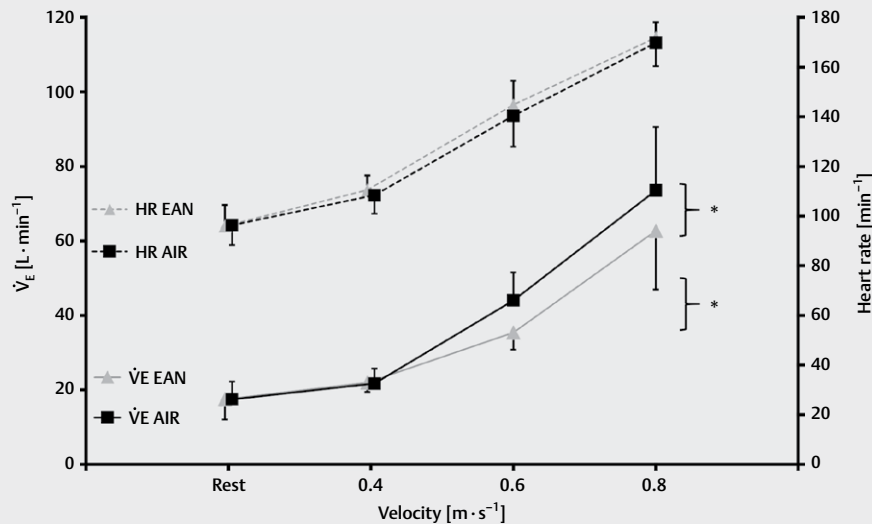
For AIR, Spearman correlation coefficient showed a significant correlation for [Lac⁻] and TTE ($r_s = 0.638$; $P = 0.047$). A correlation with EAN showed no significant effects ($r_s = 0.329$; $P = 0.353$).

Discussion

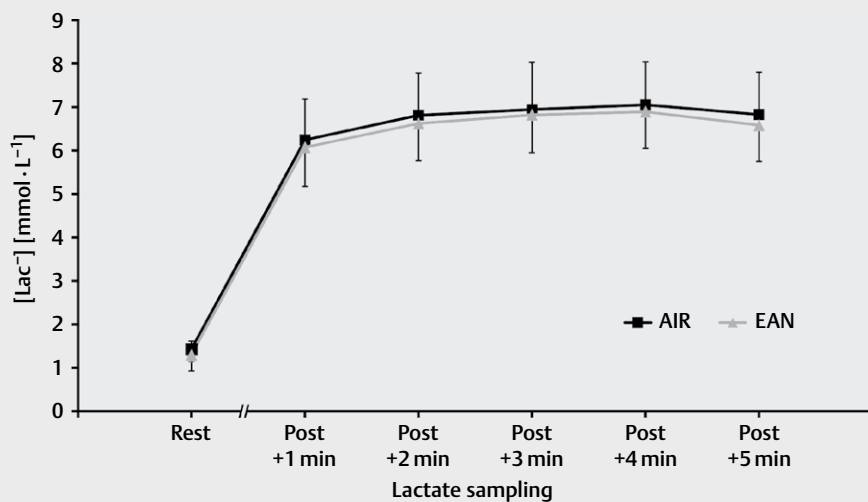
Our results show a significantly lower \dot{V}_E with EAN for the fin-swimming velocities of 0.6 $\text{m} \cdot \text{s}^{-1}$ and 0.8 $\text{m} \cdot \text{s}^{-1}$ in shallow underwater settings compared to AIR. Surprisingly, [Lac⁻] $_{\text{max}}$ after exercise did not show any significant differences between AIR and EAN. We observed no differences or correlations for HR, TTE, or the ventilatory threshold.

Thus, it can be speculated why the effect of a lower \dot{V}_E only shows for high velocities. In normobaric conditions, hemoglobin in arterial blood is typically saturated for 97% or higher. Whereas increased PO₂ has no relevant effects on hemoglobin saturation, the concentration of physically dissolved oxygen increases linearly according to the ambient pressure (Henry's law) [34]. For normobaric conditions, the dissolved O₂ concentration for an alveolar PO₂ of 15 kilopascal (kPa) can be approximated to 3 $\text{mL} \cdot \text{L}^{-1}$ for AIR and 6.8 $\text{mL} \cdot \text{L}^{-1}$ for EAN (i.e. 34 kPa alveolar PO₂), respectively, when assuming a physical solubility for O₂ of 0.2 $\text{mL} \cdot (\text{L} \cdot \text{kPa})^{-1}$ [35]. The inspiratory P_iO₂ for EAN in the present study results in more than two times the physically dissolved O₂ in the arterial blood [O₂_{art}] when compared to AIR (see ► **Table 3**). At 25 m, which is a common water depth in sport diving, breathing EAN leads to an O₂_{art} of 26.8 $\text{mL} \cdot \text{L}^{-1}$.

We assume that during rest and low velocities (i.e. 0.4 $\text{m} \cdot \text{s}^{-1}$), the muscles' oxygen demand is sufficiently covered. With increasing exercise intensity, the delay of cardiovascular adaptations to the working muscles increased O₂-demand then creates a local oxygen deficit [36, 37]. An increased O₂ supply and the accompanying greater amount of oxygen in the blood increase oxygen uptake ($\dot{V}O_2$), and could enhance the diffusion of oxygen to the muscle [20]. As a result, additional glycogen oxidation could attenuate the accumulation of lactate and respiratory acidosis [3, 26, 37]. Hyperoxia (e.g. hyperbaric conditions underwater) might reduce the amount of metabolic anaerobic glycolysis at least during transient phases after increased exercise intensity [38], thus reducing lactate production and maintaining a higher pH-value with a slower



► **Fig. 2** The X-axis depicts rest and the velocities 0.4, 0.6, and 0.8 m·s⁻¹. Means and 95% confidence intervals are shown for heart rate (upper two graphs) and ventilation (\dot{V}_E , lower two graphs) for the conditions normal air (AIR) and oxygen-enriched air (EAN). * P < 0.05 for comparisons of breathing gases (AIR vs. EAN).



► **Fig. 3** Means and 95% confidence intervals for samples of whole blood lactate concentrations [Lac⁻] from the earlobe. Samples were taken once before exercise (Rest) and every minute for five times directly following incremental exercise (Post + x min).

increase in \dot{V}_E from an alleviated respiratory drive [26, 38]. This seems especially relevant with a decreased oxygen deficit during work rate transitions and high-intensity exercise [16, 36, 38], where the acceleration of $\dot{V}O_2$ kinetics must be assumed [38, 39]. These metabolic adaptations and their influence on VT2 could be backed up in future studies involving spirometric measurements.

It should be noted that other studies reported decreases in \dot{V}_E during hyperoxic exercise accompanied by a decrease in blood lactate [20, 40–42], which is inconsistent with our data (see [37] for a review). Differences in [Lac⁻] might become clearer with measurements conducted during exercise, which was not possible in this experimental setup. In this study, maximum values measured *after*

exercise might not reflect the metabolic state *during* exercise. Some authors also emphasized the relevance of individual physical fitness and exercise intensity [20] as well as recruited muscle mass [43] on lactate production during hyperoxic exercise. In line with our findings, Pederson et al. reported only non-significantly lower [Lac⁻] during submaximal exercise involving small muscle groups [44]. Although these differences between underwater fin-swimming and modalities like running and cycling might explain our findings, new technologies for continuous underwater lactate measurements should be employed in future studies.

TTE and RPE, which did not differ between conditions in our study, most likely depend on exercise modality (i. e. running vs. cy-

► **Table 3** Relation of inspiratory and alveolar oxygen partial pressure (PO_2 [kPa]) and the related arterial O_2 concentration [O_{2art}] for AIR (21% O_2) and EAN (40% O_2) as breathing gases. Data were calculated for normobaric (100 kPa) and hyperbaric (140 kPa and 350 kPa, respectively) underwater conditions.

		EAN [40% O_2]		AIR [21% O_2]	
		PO_2 [kPa]	$[O_{2art}]$ [$mL \cdot L^{-1}$]	PO_2 [kPa]	$[O_{2art}]$ [$mL \cdot L^{-1}$]
normobaric [100 kPa]	inspiratory	40	6.8	21.0	3.0
	alveolar	34		15.0	
hyperbaric [underwater] [140 kPa]	inspiratory	56	10.0	29.4	4.7
	alveolar	50		23.4	
hyperbaric [underwater] [350 kPa]	inspiratory	140	26.8	73.5	13.5
	alveolar	134		67.5	

cling), the applied exercise test (i. e. time trials, constant work rate, incremental exercise), exercise duration, and $F_{I}O_2$ [37]. Some studies reported a prolonged TTE and RPE in hyperoxic settings [3, 23, 37, 45], whereas Peeling et al. observed influences on RPE only with $F_{I}O_2 > 100$ kPa [25]. In the present study, work rate stages during incremental exercise were defined by velocity rather than power. In contrast to exercise in the laboratory, water-resistance underwater increases exponentially with velocity, thus making an increase in work rate more difficult, especially during high-intensity exercise. The absence of a prolonged TTE was likely dependent on the individual fin-swimming efficiency and leg strength, and therefore individual physical exhaustion was not reached in every test. Future studies should investigate these effects for different water depths, PO_2 and prolonged constant work rates.

Conclusions

Our results reveal a significantly lower \dot{V}_E for high-intensity fin swimming (i. e. $\geq 0.6 \text{ m} \cdot \text{s}^{-1}$) in shallow water in hyperoxia ($P_{I}O_2 = 56$ kPa) compared to normal air. No effects were observed for $[Lac]_{max}$, TTE, and RPE. Hyperoxic gases like EAN40 (= 40% O_2) are frequently used by sport divers and gas consumption plays a major role in planning and executing dives with a limited gas supply. During a dive at 20 m water depth, our results would suggest 270 L less gas consumed for 10 min of fin-swimming at $0.6 \text{ m} \cdot \text{s}^{-1}$, when using EAN instead of normal air (i. e. 27 bar or ~ 400 psi less in a 10 L dive tank). This velocity can be considered reasonable when swimming against a current. Although exercise intensity, modality, ambient pressure (i. e. increased $P_{I}O_2$ at depth), and accompanying increased breathing gas density must be considered as modulating factors, the use of hyperoxic gases in sport diving could lower \dot{V}_E in shallow water contexts and therefore increase the duration or safety of the dive with higher gas reserves.

Declaration of Helsinki statement

This study followed the rules of the declaration of Helsinki.

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Conflict of interest

The authors declare that they have no conflict of interest.

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