Original articles

Effects of inspiratory muscle training versus high intensity interval training on the recovery capacity after a maximal dynamic apnoea in breath-hold divers. A randomised crossover trial

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Abstract
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Introduction: After a maximal apnoea, breath-hold divers must restore O_2 levels and clear CO_2 and lactic acid produced. High intensity interval training (HIIT) and inspiratory muscle training (IMT) could be employed with the aim of increasing recovery capacity. This study aimed to evaluate the relative effects of IMT versus HIIT on recovery of peripheral oxygen saturation (SpO_2), and also on pulmonary function, inspiratory muscle strength, lactate and heart rate recovery after a maximal dynamic apnoea in breath-hold divers.
Methods: Fifteen breath-hold divers performed two training interventions (IMT and HIIT) for 20 min, three days per week over four weeks in randomised order with a two week washout period.
Results: IMT produced a > 3 s reduction in SpO_2 recovery time compared to HIIT. The forced expiratory volume in the first second (FEV_1) and maximum inspiratory pressure (MIP) were significantly increased in the IMT group compared to HIIT. The magnitude of these differences in favour of IMT was large in both cases. Neither training intervention was superior to the other for heart rate recovery time, nor in peak- and recovery- lactate.
Conclusions: IMT produced a reduction in SpO_2 recovery time compared to HIIT after maximal dynamic apnoea. Even a 3 s improvement in recovery could be important in scenarios like underwater hockey where repetitive apnoeas during high levels of exercise are separated by only seconds. IMT also improved FEV_1 and MIP, but no differences in lactate and heart rate recovery were found post-apnoea between HIIT and IMT.

Introduction

During dynamic apnoea with fins, breath-hold divers compete to dive as deep as possible while oxygen (O_2) and carbon dioxide (CO_2) levels are progressively reduced and increased respectively. A major concern among divers is to surface with a safety margin to avoid hypoxic syncope or blackout; however, hypoxic incidents can be triggered even after the diver has surfaced.[1]

After a maximal apnoea, the diver attempts to recover O_2 levels and clear CO_2 and lactic acid produced. We hypothesised that the speed and success of the recovery depend on respiratory function to some extent; thus, in this study we analysed two types of training that have been shown to improve respiratory and physical function.

High intensity interval training (HIIT)[2] has become popular in recent years, and is characterised by increasing aerobic and anaerobic capacity[3] or increasing skeletal muscle oxidative capacity[4] in a short time period. In addition, inspiratory muscle training (IMT) is an effective method to increase pulmonary gas flow and capacity. IMT produces an increase in the strength and endurance of the specific musculature likely to facilitate optimal exchange of O_2/CO_2.[5] Previous studies suggest that IMT increased lung volumes,

work capacity, and power output in healthy subjects;[6,7] as well as improved physiologic responses in hypoxic exercise.[8] Furthermore, IMT attenuates the human respiratory muscle metaboreflex,[9] leading to a delay in the reduction of blood flow in the limbs, as a consequence of the fatigue of the inspiratory muscles, suggesting better blood flow redistribution after apnoea. HIIT and IMT have beneficial effects on lactate levels and recovery after exercise.[2,10,11]

This study aimed to analyse the effects of IMT versus HIIT on recovery of peripheral oxygen saturation (SpO_2) after maximal dynamic apnoea in breath-hold divers. Our second aim was to investigate the effects of these types of training on pulmonary function, inspiratory muscle strength, lactate recovery and heart rate recovery in the same population.

Methods

The study was approved by the Ethics Committee of La Salle University Centre for Advanced Studies (CSEULS-PI-215/2018) and was conducted following the ethical standards of the Declaration of Helsinki. The study was registered in clinicaltrials.gov, identifier NCT04084535. Divers were informed of the procedures, including benefits and risks, prior to signing the informed consent document, and all provided written informed consent before enrolment.

DESIGN

A single-blind, randomised, crossover trial was conducted. To ensure blinding, an external individual who was not involved in the study allocated participants to each group using GraphPad Software© (1:1 simple randomisation), and the intervention allocations were adequately concealed in sealed envelopes. The participants were not blinded and were allocated into 1 of 2 groups: (1) IMT; or (2) HIIT. The assessor was blinded to the allocation schedule, and the participants were specifically asked not to discuss the intervention with the evaluators. The washout period was two weeks after which the individual began the alternate training mode.

PARTICIPANTS

Participants were recruited from three Spanish freediving centers (Madrid Zaragoza, and Barcelona) from September 2019 to December 2019. Breath-hold divers were eligible to participate if: over 18 years of age; without co-morbidities that limited their ability to participate in exercise programmes or to practice voluntary apnoea; and members of the Spanish Federation of Underwater Activities (FEDAS). The exclusion criteria were participation in another exercise programme during the study period, and inability to attend at least 80% of the intervention sessions.

TRAINING PROCEDURES

The IMT included a four-week home training protocol consisting of 20 minutes (min), three days per week for all sessions. It was performed in a sitting position with a noseclip. Participants were instructed to perform fast and forceful inspirations and were encouraged to achieve maximal inhalation/exhalation with every breath. To provide inspiratory resistance a Powerbreath Classic Competition threshold pressure device (POWERbreathe International Ltd; Southam, Warwickshire, UK) was used. The training load was performed with the percentages of 50% (100 breaths in 10 min) and 80% (30 breathes in 10 min) maximum inspiratory pressure as described in Appendix 1*.

In the other group, the HIIT consisted of a swimming programme of 20 min, three days per week for four weeks. The training was applied under supervision, with the intensity monitored by the participant, who rated the perceived fatigue using the rating scale of perceived effort (RPE).[12,13] The swimming programme consisted of 20 min of interval training: a 1-min high-intensity work interval at about 19 points on the RPE followed by a one-min moderate-intensity work interval at about 12 points on the RPE.

OUTCOMES

All outcome measures were assessed pre-intervention and post-intervention (before and after the four-week programme). The assessments were conducted in a quiet, temperature-regulated, humidity controlled location, close to a swimming pool (24°C (SD 1), relative humidity ~90%).

Baseline measurements including weight, height, spirometry, maximum respiratory pressures, heart rate, SpO_2 and basal end-tidal carbon dioxide (E_TCO_2) were obtained before the maximal dynamic apnoea attempt. Spirometry was assessed with a portable spirometer (Spirobank II Basic, MIR, Rome, Italy) according to the American Thoracic Society/European Respiratory Society task force statement.[14] Maximum respiratory pressures were measured using a device that applies an inspiratory/expiratory load (Micro-RPM™, Care Fusion, San Diego, CA). This measurement was performed in a sitting position with the nose occluded with a nose clip. Divers were asked to perform a forceful and deep inspiration/expiration maintained for longer than 1 s after a complete expiration/inspiration. This was performed at least three times until there was < 20% variability between measurements, and the highest value was recorded.[15] Basal heart rate and SpO_2 were detected by a finger probe pulse oximeter (Nonin® Model 9847, Nonin Medical, Inc.). The basal E_TCO_2 was recorded every 5 seconds (s) over 2 min by the Nonin 9847 series hand-held pulse oximeter/CO_2 detector with semi-quantitative E_TCO_2 bar graph readout using the endotracheal tube adapter placed in the participant's mouth in the supine position.[16]

***Footnote:** Appendices 1 and 2 are available on the DHM journal website for viewing.

Next, the divers were asked to perform maximal dynamic apnoea with bi-fins in a 25 m pool when ready. A safety diver accompanied the divers during the attempt, as is done during competitions. Heart rate, SpO_2 and E_TCO_2 were recorded every 5 s during the 1 min recovery time. The primary outcome was the length of time to return to 95% SpO_2 during the 1 min recovery time after maximal dynamic apnoea. During the measurement, a signal indicated good perfusion with a green light or poor perfusion with a red light. Only data indicating good finger perfusion were analysed. At the same time as pulse oximetry, blood lactate concentrations were measured by analysing capillary blood samples taken from finger pricks using a Lactate Scout point of care analyser (EKF Diagnostics, Cardiff, UK) and at 3 min and 10 min during the passive recovery. The outcome measure to establish lactate recovery was the difference between the maximum value registered and the value at 10 min. Finally, breath-hold divers can also experience diaphragm contractions when they dive owing to the increasing level of CO_2 in the blood and the accompanying urge to breathe. Therefore, we asked participants to count the number diaphragm contractions during the maximal dynamic apnoea in order to evaluate training adaptations.

SAMPLE SIZE

The sample size was designed to detect which training model (IMT or HIIT) produced SpO_2 recovery in less time. For this purpose, in accordance with the recommendations established for crossover designs, a paired Student's *t*-test, with a power of 80% and an alpha error of 5% was chosen. A large effect size (d = 0.8) was used to detect clinically relevant differences.[17] In addition, the sample initially established was increased by 20%, given losses are common in longitudinal studies. Hence, a total sample of at least 15 participants was assessed as being required.

DATA ANALYSIS

A P-value < 0.05 was considered statistically significant. The Shapiro-Wilk test showed a normal distribution of the data except for the number of contractions during apnoea and the lactate assessment (peak and recovery). The statistical analyses were performed according to the procedures described by Wellek and Blettner for crossover designs.[18] The carry-over effects were assessed to confirm they were negligible. Specifically, for the parametric data, a paired Student's *t*-test was used to assess residual and period effects, whereas the Wilcoxon test was used for nonparametric data. The carry over effects were assessed by comparing within-subject sums of the results from both periods through an unpaired Student's *t*-test or the Mann-Whitney U test.

A comparison of the changes between baseline and post-intervention due to IMT versus those produced by HIIT was used to assess treatment effects. Specifically, a paired Student's *t*-test was used for parametric data, and a Wilcoxon test was used for nonparametric data. Effect sizes were established according to Cohen's method (Cohen's d): small (0.20–0.49), medium (0.50–0.79) or large (≥ 0.8).[19]

Furthermore, an intention-to-treat analysis was performed to include missing data and to protect the randomisation. Currently, there is no standardised method to handle missing outcomes.[20] Hence, the average change obtained in the corresponding training intervention was used to replace the missing outcomes. In addition, to carry out the intention-to-treat analysis, the participants were required to perform at least three of the four planned evaluations. Otherwise, the participant's data was not included in the analysis.

Results

Sixteen breath-hold divers were included in the study. Two weeks after starting the study, a participant suffered a trauma that required immobilisation; he was not included in the analysis. Therefore, the sample ultimately analysed consisted of 15 participants (three women and 12 men), of which one was included as intention-to-treat because, for personal reasons, they could not attend the last day of assessments.

The mean age, weight and height of the 15 divers were 36 (SD 9) years, 75 (12) kg and 176 (8) cm. The prior personal best in dynamic apnoea with fins ranged from 60 to 141 m (mean 100 (SD 21) m). At rest and before the dives, all freedivers had a basal SpO_2 above 95% and a basal E_TCO_2 of 30 mmHg; the latter being a function of the measuring device's ability to only provide incremental E_TCO_2 readings of 2, 6, 10, 20, 30, 50, or 75 mmHg. The mean maximal distance during dynamic apnoea recorded in the first attempt was 82 m (SD 20) and the range was 50–120 m. Throughout all study dives individual divers performed the distance recorded in their first attempt.

RESPIRATORY VARIABLES, SPEED, AND NUMBER OF DIAPHRAGM CONTRACTIONS DURING APNOEA

There were no carry-over effects for any of the variables assessed (Table 1). The forced expiratory volume in one second (FEV_1) (mean difference [95% CI] 0.22 L [0.06 to 0.38]) and maximum inspiratory pressure (MIP) (mean difference [95% CI] 13.05 cmH_2O [0.39 to 25.71]) were significantly increased after IMT compared to HIIT (Table 2). The magnitude of these differences in favour of IMT nearly met the threshold classification for 'large' in both cases (FEV_1, d = 0.8; MIP, d = 0.73). Neither training was superior to the other for the rest of the respiratory variables: forced vital capacity (FVC) (mean difference [95% CI] -0.005 L [-0.24 to 0.23]); and maximum expiratory pressure (MEP) (mean difference [95% CI] -2.7 cmH_2O [-16.45 to 11.05]), nor for speed (mean difference [95% CI] -0.03 $m \cdot s^{-1}$ [-0.07 to 0.01]) and number of contractions during apnoea (Z-value = –0.903; P-value = 0.367).

Table 1

Descriptive data for all variables at baseline (Pre) and end (Post) of each training period, as well as assessment of carry-over effects. Data are presented as mean (SD). Note: a) = Residual effects: Pre 1 Total vs. Pre 2 Total; b) = Period effects: Post 1 Total vs. Post 2 Total; c) = Carry-over effects (Post 1 + Post 2): AB vs BA. AB sequence = inspiratory muscle training (IMT) followed by high intensity interval training (HIIT); BA sequence = HIIT followed by IMT; Contract. = diaphragmatic contractions; FEV_1 = forced expiratory volume in the first second; FVC = forced vital capacity; HR-R = recovery time of heart rate; Lactate-M = maximum blood lactate; Lactate-R = blood lactate recovery; MEP = maximum expiratory pressure; MIP = maximum inspiratory pressure; SpO_2-R = recovery time of peripheral oxygen saturation

Parameter		Period 1		Period 2		Mean diff. (95% CI) or Z-value; *P*-value
		Pre	Post	Pre	Post	
Physical recovery after apnoea						
SpO_2-R (s)	AB	13.22 (8.53)	9.71 (5.02)	12.14 (5.87)	11.51 (6.28)	a) -0.96 (-3.7 to 1.78)
	BA	6.00 (3.25)	7.25 (2.44)	8.75 (3.41)	6.75 (4.27)	b) -0.57 (-2.35 to 1.21)
	Total	9.37 (7.1)	8.40 (3.92)	10.33 (4.87)	8.97 (5.66)	c) 7.22 (-2.53 to 16.98)
HR-R (s)	AB	29.14 (15.75)	29.29 (15.62)	32.86 (12.59)	30.53 (12.36)	a) -3.53 (-7.83 to 0.76)
	BA	37.00 (14.40)	38.5 (15.42)	40.38 (13.06)	40.25 (13.41)	b) -1.51 (-7.95 to 4.92)
	Total	33.33 (15.05)	34.2 (15.69)	36.87 (12.97)	35.71 (13.44)	c) -18.94 (-47.89 to 10.02)
Lactate-M ($mmol \cdot L^{-1}$)	AB	4.07 (1.68)	4.03 (2.46)	3.84 (2.69)	3.87 (2.04)	a) Z = -0.786; *P* = 0.432
	BA	3.68 (1.21)	3.63 (1.29)	3.94 (1.70)	3.9 (1.61)	b) Z = -0.346; *P* = 0.729
	Total	3.86 (1.41)	3.81 (1.86)	3.89 (2.13)	3.89 (1.76)	c) Z = -0.463; *P* = 0.643
Lactate-R ($mmol \cdot L^{-1}$)	AB	1.00 (0.61)	0.94 (1.03)	0.97 (0.82)	1.05 (0.65)	a) Z = -0.157; *P* = 0.875
	BA	0.84 (0.52)	0.83 (0.44)	1.06 (0.77)	1.08 (0.84)	b) Z = -1.254; *P* = 0.210
	Total	0.91 (0.55)	0.88 (0.75)	1.02 (0.76)	1.06 (0.73)	c) Z = -0.290; *P* = 0.772
Respiratory assessment						
FVC (L)	AB	5.27 (1.26)	5.36 (1.31)	5.27 (1.35)	5.29 (1.17)	a) 0.09 (-0.15 to 0.33)
	BA	5.97 (0.57)	6.08 (0.64)	5.81 (0.91)	5.86 (0.91)	b) 0.15 (-0.09 to 0.39)
	Total	5.64 (0.99)	5.74 (1.04)	5.56 (1.13)	5.59 (1.04)	c) -1.29 (-3.52 to 0.93)
FEV_1 (L)	AB	4.26 (1.04)	4.45 (1.07)	4.22 (1.17)	4.29 (1.01)	a) 0.14 (-0.05 to 0.33)
	BA	4.96 (0.44)	4.75 (0.29)	4.73 (0.66)	4.82 (0.65)	b) 0.04 (-0.14 to 0.22)
	Total	4.63 (0.83)	4.61 (0.74)	4.49 (0.93)	4.57 (0.85)	c) -0.83 (-2.57 to 0.91)
MIP (cmH_2O)	AB	134.71 (28.22)	161.14 (30.52)	154.14 (30.59)	160.9 (33.88)	a) -8.8 (-19.06 to 1.46)
	BA	161.50 (14.96)	161.75 (22.43)	161.00 (16.85)	168.5 (15.81)	b) -3.49 (-10.1 to 3.13)
	Total	149.00 (25.39)	161.47 (25.51)	157.80 (23.57)	164.95 (25.15)	c) -3.92 (-59.64 to 51.8)
MEP (cmH_2O)	AB	173.71 (32.90)	194.14 (41.11)	180.00 (34.54)	197.07 (38.00)	a) -6.47 (-14.92 to 1.98)
	BA	196.25 (54.10)	216.00 (56.32)	202.88 (52.53)	214.63 (48.12)	b) -0.63 (-11.62 to 10.36)
	Total	185.73 (45.42)	205.80 (49.37)	192.20 (45.06)	206.43 (43.11)	c) -39.41 (-141.8 to 62.96)
Assessment during apnoea						
Speed ($m \cdot s^{-1}$)	AB	0.97 (0.18)	0.99 (0.19)	0.99 (0.20)	1.02 (0.20)	a) -0.03 (-0.08 to 0.01)
	BA	0.89 (0.09)	0.94 (0.11)	0.93 (0.09)	0.96 (0.10)	b) -0.02 (-0.06 to 0.01)
	Total	0.93 (0.14)	0.96 (0.15)	0.96 (0.15)	0.98 (0.15)	c) 0.11 (-0.22 to 0.44)
Contract. (*n*)	AB	16.43 (9.68)	18.57(14.65)	17.00 (9.38)	13.71 (12.05)	a) Z = -0.704; *P* = 0.482
	BA	11.88 (7.68)	10.00 (9.61)	12.13 (9.86)	11.63 (8.14)	b) Z = -0.786; *P* = 0.432
	Total	14.00 (8.67)	14.00 (12.56)	14.4 (9.63)	12.6 (9.83)	c) Z = -0.347; *P* = 0.728

PHYSICAL RECOVERY AFTER APNOEA

Once again, there were no carry-over effects for any of the physical recovery variables assessed (Table 1). The comparison between the interventions for the physical recovery variables is shown in Table 2. The IMT intervention showed a statistically significant reduction in SpO_2 recovery time compared to the HIIT intervention [IMT intervention (SpO_2 recovery = –2.71 (SD 5.04) s) vs HIIT intervention (SpO_2 recovery = 0.37 (3.76) s] (Table 2). Specifically, IMT reduced the SpO_2 recovery time obtained with the HIIT by more than 3 s [mean difference (95% CI) -3.08 s (-5.72 to -0.43)], which implies a difference of moderate-large magnitude (d = 0.69). Neither training was superior to the other in terms of heart rate recovery time [mean difference (95% CI) 0.28 (-5.78 to 6.35) s], nor in peak lactate level (Z-value = 0.683; *P*-value = 0.494) and recovery lactate level (Z-value = –0.369; *P*-value = 0.712).

Table 2

Comparison of the changes between baseline and post-intervention after inspiratory muscle training versus those after high intensity interval training. Data are presented as mean (SD). Contractions = diaphragmatic contractions; FEV_1 = forced expiratory volume in the first second; FVC = forced vital capacity; HIIT = high intensity interval training; HR-R = recovery time of heart rate; IMT = inspiratory muscle training; Lactate-M = maximum blood lactate; Lactate-R = blood lactate recovery; MEP = maximum expiratory pressure; MIP = maximum inspiratory pressure; SpO_2-R = recovery time of peripheral oxygen saturation; * = $P < 0.05$

Parameter	Δ IMT intervention	Δ HIIT intervention	Intervention effects Mean difference (95% CI); d or Z-value; *P*-value
Physical recovery after apnoea			
SpO_2-R (s)	-2.71 (5.04)	0.37 (3.76)	-3.08 (-5.72 to -0.43); d = -0.69*
HR-R (s)	-0.01 (0.66)	-0.29 (10.88)	0.28 (-5.78 to 6.35); d = 0.04
Lactate-M ($mmol \cdot L^{-1}$)	-0.04 (0.60)	-0.01 (0.64)	Z = -0.683; P = 0.494
Lactate-R ($mmol \cdot L^{-1}$)	-0.02 (0.88)	0.03 (0.43)	Z = -0.369; P = 0.712
Respiratory assessment			
FVC (L)	0.07 (0.27)	0.07 (0.24)	-0.005 (-0.25 to 0.23); d = 0
FEV_1 (L)	0.14 (0.28)	-0.08 (0.27)	0.22 (0.06 to 0.38); d = 0.8*
MIP (cmH_2O)	16.33 (21.09)	3.29 (13.76)	13.05 (0.39 to 25.71); d = 0.73*
MEP (cmH_2O)	15.8 (19.20)	18.5 (18.67)	-2.7 (-16.45 to 11.05); d = 0.14
Assessment during apnoea			
Speed ($m \cdot s^{-1}$)	0.02 (0.06)	0.05 (0.04)	-0.03 (-0.07 to 0.01); d = -0.59
Contractions (*n*)	0.73 (7.87)	-2.53 (8.12)	Z = -0.903; P = 0.367

Discussion

This study analysed the effects of IMT versus HIIT on recovery after maximal dynamic apnoea. According to the literature, both interventions could contribute to restoring O_2 levels and clearing the CO_2 and lactic acid produced during apnoea through various mechanisms;[3–5] however, to our knowledge this is the first study comparing IMT versus HIIT in terms of recovery after maximal dynamic apnoea in breath-hold divers. The IMT intervention led to a significant reduction in SpO_2 recovery time compared to the HIIT intervention; in addition, FEV_1 and MIP were significantly increased after the IMT intervention compared to HIIT. The results obtained in our study suggest the superiority of IMT in terms of improved post-apnoea recovery.

Measuring lactate concentrations after maximal apnoea can provide relevant information on the contribution of the anaerobic pathway, lactate clearance, or acidosis tolerance.[21] Previous studies suggested that an effort is considered maximal when it fulfils the following requirements: maximal HR is reached, respiratory quotient (CO_2/O_2) is greater than 1.2, the RPE is close to 20 and blood lactate exceeds 8 $mmol \cdot L^{-1}$;[22,23] but in our study, only two divers reached levels higher than 8 $mmol \cdot L^{-1}$. In previous studies performed with breath-hold divers,[24,25] values of 2.3 $mmol \cdot L^{-1}$ in static apnoea and 7.1 $mmol \cdot L^{-1}$ in dynamic apnoea have been reached. Also, no differences were found in lactate recovery during post apnoea recovery between HIIT and IMT. This outcome could have been due to the low peak lactate concentrations observed in the participants. In previous studies, both HIIT and IMT have demonstrated an influence on post-exercise lactate clearance at high intensity.[26,27] Cited studies suggested that physical training would improve lactate buffering during a 10 min-recovery after exercise; however, in our study (unlike previous studies) lactate recovery was measured after 10-min recovery from maximal voluntary apnoea. It might be that blood flow centralisation (induced by diving reflex or metaborreflex), could modify the metabolic buffering response of the system. Future studies should clarify this controversy. On the other hand, in this study the lactate concentration reached post-apnoea had a stronger association with swimming speed than with hypoxia levels.

Previous studies[28,29] have shown a strong association between apnoea training and vital capacity (VC) increase. This increase was up to 2 L in a trained breath-hold diver versus an untrained diver, for the same height and age; but, in our study, there were no changes in VC after interventions. As suggested by other studies,[30,31] divers (who have significant motor control in their respiratory musculature, thoracic and pleural flexibility, and experience in swimming in hyperbaric conditions) are highly trained, making it difficult to further increase their VC with IMT or HIIT. A 2013 meta-analysis reported that divers and swimmers would

not benefit in respect to VC because they are already close to their optimal condition.[32] Previous studies,[6,7] reported an increase in MIP and VC after IMT; however, in these studies the training duration was doubled (eight weeks vs. four weeks in our study). Nevertheless, similar studies previously demonstrated an increase in MIP after six or four weeks.[8,10] Another conclusion from relevant studies is that only high-intensity IMT (80% of MIP at baseline) increased VC.[6,7] In our study, an increase in MIP and FEV_1 was found after IMT, indicating that IMT is an effective intervention to improve respiratory functionality even in highly trained participants.[33] However, HIIT did not produce improvement in MIP or FEV_1. These results are different when compared to a previous study,[34] which showed no changes for FEV_1 but an increase in MIP after HIIT.

To analyse the recovery after apnoea, the main outcome was monitored by pulse-oximeter to detect the time to reach an $SpO_2 > 95\%$ in divers.[35] In the present study nadir SpO_2 (mean (SD); minimum) values were 70% (15); 32% in HIIT and 72% (16); 33% in IMT. To recover, the breath-hold diver must perform ventilations that allow a rapid and effective restitution of normoxia to reduce risk of a blackout. The present results showed a faster SpO_2 recovery after IMT, suggesting that specific IMT might produce greater ventilation efficiency in the first post-apnoea breaths.

Pulse oximetry is the most common method employed to reflect the first few minutes of oxygenation recovery after submersion; however, this method failed to record the initial 10–15 s after surfacing. This delay was due to the time needed to dry and heat the finger. With the use of pulse oximetry, there is also a delay in nadir O_2 saturation compared to more central measurements. However in our situation, the delay might have been beneficial, given the values we obtained at 20–30 s might represent events that occur at a point closer to the end of the dive. In addition, E_TCO_2 was determinated during the recovery period after the maximal apnoea, but the device only can display the following data: 2, 6, 10, 20, 30, 50 ,75 mmHg CO_2. All divers registered > 75 mmHg during the first breaths, however, it was unable to determine the CO_2 levels precisely after apnoea, suggesting that a more accurate device to measure E_TCO_2 is needed in future research.

Conclusions

IMT showed a decrease in SpO_2 recovery time compared to the HIIT intervention after maximal dynamic apnoea. Although one could question the practical significance of a three-second improvement in recovery of the SpO_2 with IMT compared to HIIT, a difference of this magnitude could be important in extreme performance activities like underwater hockey where heavy apnoeic exercise is performed during repetitive dives separated only by seconds. Regarding secondary outcomes, an improvement in FEV_1 and MIP were achieved, but no differences in lactate and heart rate recovery were found post-apnoea between HIIT and IMT. Thus, IMT appears to be an effective training intervention for divers aiming to improve oxygen recovery after a maximal apnoea.

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