Original research

Cardiovascular and hemodynamic responses on dryland vs. immersed cycling

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ABSTRACT

Objectives: To investigate the effect of water immersion on oxygen uptake ($\dot{V}O_2$) and central hemodynamic responses during incremental maximal exercise at the same external power output (P_{ext}) and recovery on an immersible ergocycle vs. a dryland ergocycle.

Design: Cross-over design study.

Methods: Twenty healthy participants (32 ± 7 years; 173 ± 6 cm; 71.7 ± 9.7 kg) performed maximal incremental exercise tests while pedalling either immersed on immersible ergocycle (Hydrorider®) or on dryland ergocycle (Ergoline 800S; Bitz, Germany). Initial P_{ext} of dryland ergocycle protocol was set at 25 W and increased by 25 W every minute until exhaustion. P_{ext} on immersible ergocycle was controlled by pedalling rate (rpm). Initial rpm was set at 40 rpm and was increased by 10 rpm until 70 rpm and thereafter by 5 rpm until exhaustion. Gas exchange and central hemodynamic parameters were measured continuously during exercise and a 5-min recovery period. Reported $\dot{V}O_2$, stroke volume, cardiac output ($\dot{Q}$) and arteriovenous difference (C(a-v)O$_2$) were compared.

Results: During exercise on immersible ergocycle, $\dot{V}O_2$ and C(a-v)O$_2$ were lower ($P < 0.0001$) whereas stroke volume and $\dot{Q}$ were higher ($P < 0.05$) relative to a dryland ergocycle exercise of equivalent P_{ext}.

Conclusions: During exercise and recovery in immersion, ($\dot{V}O_2$) and arteriovenous difference were reduced in healthy young participants, while stroke volume and cardiac output were increased for the same P_{ext}. During the recovery, central hemodynamics responses remained higher in immersible ergocycle.

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1. Introduction

Exercise training on an immersible ergocycle (IE) is becoming more popular as it appears to be suitable for different groups of individuals: athletes, sedentary participants, overweight and obese subjects and even the elderly. However, water immersion can affect hemodynamic and oxygen uptake ($\dot{V}O_2$) responses during deep water running or during exercise on IE.[1,2] During exercise on IE, the external hydrostatic pressure increases venous return and central blood volume, which raises the stroke volume, cardiac preload and cardiac output with concomitant reduction in heart rate and peripheral vascular resistance.[3,4] In addition, the density of water is greater than the density of air and increases the drag during locomotion[5] and pedalling.[2] Previous studies showed that immersion reduces $\dot{V}O_2$ max during deep water running or treadmill[1,6] and during immersed cycling for submaximal and maximal intensities.[7,8] However, while comparing central hemodynamic responses at the same level of $\dot{V}O_2$ or at an apparent same submaximal external power output (P_{ext}), these studies did not take into account the water external force exerted on the legs as a function of pedalling rate. The P_{ext} during exercise on IE is dependent on drag forces on pedals, paddles, rods and legs and their tangential velocity from pedalling rate (rpm).[9–11] We recently calculated that pedalling in water requires an approximately 15% higher P_{ext} when compared to the same pedalling rate on an equivalent ergocycle on dryland.[10,11] According to our knowledge, $\dot{V}O_2$ and hemodynamic responses have never been compared at the same level of P_{ext} in

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water and on dryland, nor have they been compared during recovery under these two conditions. Hence, the first aim of this study was to compare $\dot{V}O_2$, central hemodynamic and arteriovenous difference responses during incremental maximal exercise on DE vs. IE at the same external power output (P_{ext}) in healthy young participants. The second aim was to study the central hemodynamic during recovery after IE vs. DE.

2. Methods

Twenty healthy young participants (32 ± 7 years, 18 males and 2 females) (see Table S1 in Supplementary material) were included. Body and fat mass were measured with bio-electrical impedance (Tanita, model BC418, Japan). Inclusion criterion was age over 18 years. Exclusion criteria were: (1) inability to perform a maximal cardiopulmonary exercise test and (2) any documented cardiovascular, pulmonary, musculo-skeletal or metabolic diseases. All participants performed a maximal continuous graded exercise test in a laboratory room of constant air temperature ($21\,°C$) and in a thermoneutral swimming pool ($30\,°C$).[1,12] All subjects provided written informed consent. The study was approved by the Research Ethics Committee of the Montreal Heart Institute.

Participants performed a maximal incremental exercise test in random order on IE (immersion to the chest level) and DE with similar external power output (P_{ext}) while measuring gas exchange (Cosmed K4b^2, Cosmed, Rome, Italy) and central hemodynamic responses (PhysioFlow®, Enduro model, Manatec, France). Initial P_{ext} of DE protocol was set at 25 W and increased by 25 W every minute until exhaustion. Pedalling rate was at a minimum cadence of 60 rpm. The P_{ext} on IE was controlled by pedalling rate as previously reported.[3,10,11] Briefly, the external forces during exercise on an IE are caused primarily by the mechanical components of the pedalling system (paddles, pedals and rods)[11] and by leg movement drag (calf, foot and thigh) that is dependent on the surface area of the lower limbs and pedalling rate (rpm).[10,14] The P_{ext} expressed in watts (W) was calculated by multiplying the total net force (F) overcoming the resistance of the system movement (pedalling system and legs) by the velocity (m/s) of pedal displacement.[2,9,10] The following general fluid equation was used to determine the total net F[9–11]

$$F = \frac{1}{2}\rho A v^2 Cd$$

where ρ is the density of water (at $30\,°C = 995.7$ kg/m^3), A is the projected area (m^2) in the direction of the movement for all segments involved (legs, paddles, rods and pedals), v is the velocity of the pedal (m/s) according to pedalling rate from 40 to 100 rpm, and Cd is the drag coefficient of every element shape (lower limbs, paddles, rods and pedals). Initial pedalling rate was set at 40 rpm (corresponding to an external power of 25 W) and was increased by 10 rpm until 70 rpm and thereafter by 5 rpm until exhaustion.[10,11,15] Physiological responses to exercise on DE and IE up to chest immersion were then compared for each participant at the same P_{ext}. Gas exchange was measured continuously at rest, during exercise, and after exercise cessation. Calibration of the Cosmed flow module was accomplished by introducing a calibrated volume of air at several flow rates with a 3-liter pump.[13] Gas analyzers were calibrated before each test using a standard certified commercial gas preparation (O$_2$: 16%; CO$_2$: 5%) according to manufacturer's specifications and previous published methodology.[1,13] Data were measured every four respiratory cycles during testing and then were averaged every 15 s for minute ventilation ($\dot{V}E$, in L min^{-1} BTPS), oxygen uptake ($\dot{V}O_2$, in L min^{-1}, STPD), carbon dioxide production ($\dot{V}CO_2$, in L min^{-1} STPD).[1] Maximal exercise tests on IE and DE lasted until the attainment of one of the two primary maximal criteria: (1) a plateau of $\dot{V}E$ despite an increase

in cadence, (2) R.E.R. > 1.1, or one of the two secondary maximal criteria: (1) measured maximal heart rate attaining 95% of age-predicted maximal heart rate, (2) inability to maintain the required workload, (3) subject exhaustion with cessation caused by general fatigue that required exercise cessation. The highest $\dot{V}O_2$ value reached during the exercise phase of each test was considered as the $\dot{V}O_2$ peak.[1] Each of the two tests were separated by one week.

Central hemodynamics was measured continuously at rest, during exercise and after exercise cessation using impedance cardiography. Data were averaged every 15 consecutive heartbeats[13] for cardiac output (in L min^{-1}), stroke volume (in mL), heart rate (in beats min^{-1}), ejection fraction (in %), end-diastolic and systolic volume (in mL), contractility index (in arbitrary units) and systemic vascular resistance (in dynes s^{-1} cm^{-5}). The use of this non-invasive technique during dry and immersed exercise in adults has been previously published.[3,13,16] Ejection fraction was calculated according to the Capan formula: EF (%) = $0.84 - (0.64 \times$ pre-ejection time)/ventricular ejection time.[17] Contractility index (CTI) was calculated according to the following formula CTI (arbitrary unit) = dZ/dT_{max}.[17] Arteriovenous difference (C(a-v)O$_2$) was calculated according to the Fick principle: C(a-v)O$_2$ = $\dot{V}O_2/\dot{Q}$.[13] Blood pressure measurement was done manually on the upper arm, with a sphygmomanometer (WelchAllyn, USA) at the elbow crease over the brachial artery, according to recommended guidelines.[18]

Results are presented as mean $\pm$ standard deviation except where otherwise indicated. Normal Gaussian distribution of the data was verified by the Shapiro–Wilk test. A two-way ANOVA (condition $\times$ time) with repeated measures was performed to test the null hypothesis that physiological response during a maximal graded exercise test was similar when performed on dryland or in water. Multiple comparisons were made with the Bonferroni post hoc test. Statistical significance was set at the $P < 0.05$ level for all analyses.

3. Results

Comparison of $\dot{V}O_2$ and hemodynamic responses measured during exercise on IE and DE are presented in Table 1. The P_{ext} max achieved was 267 ± 12 W. Both $\dot{V}O_2$ and C(a-v)O$_2$ were significantly lower during exercise on IE ($P < 0.001$, $g = -0.25$ and -0.87 respectively). By contrast, stroke volume (SV) and cardiac output ($\dot{Q}$) were significantly higher during exercise on IE ($P < 0.05$, $g = 0.59$ and 0.20 respectively). There was no effect of water immersion on other parameters. There was no interaction ($P > 0.05$) for any variable except for C(a-v)O$_2$ ($P < 0.001$).

Comparison of $\dot{V}O_2$ and hemodynamic responses measured after exercise cessation on IE and DE are presented in Table 2. As presented in Fig. 1, we found an interaction effect for $\dot{V}O_2$, EF, ESV and contractility index ($P < 0.05$). During recovery, $\dot{V}O_2$ and C(a-v)O$_2$ were significantly reduced in water ($P < 0.001$, $g = -0.41$ and -0.67), whereas SV, EF and contractility index were higher ($P < 0.05$, $g = 0.64$, 0.71 and 0.19 respectively).

4. Discussion

The principal new findings of this study are that: (1) Compared to dryland ergocycle exercise (DE), $\dot{V}O_2$ and arteriovenous difference were significantly reduced during exercise on IE at the same P_{ext} whereas cardiac output and stroke volume were increased on IE in healthy young participants. (2) During recovery, $\dot{V}O_2$ and C(a-v)O$_2$ remained reduced during immersion whereas stroke volume, ejection fraction and cardiac contractility remained higher. This study is the first to compare $\dot{V}O_2$, central hemodynamics and C(a-v)O$_2$ at the same P_{ext} intensity levels during immersed and dry

Table 1
Comparison of oxygen uptake and hemodynamic parameters during exercise on immersed (IE) and dryland ergocycle (DE).

Parameters		Rest	25 W 40 rpm	50 W 50 rpm	75 W 60 rpm	125 W 70 rpm	200 W 80 rpm	W_{max} (267 ± 12 W)	P-value Condition effect
$\dot{V}O_2$ (mL/min^{-1})	IE	360 ± 97	512 ± 194‡	817 ± 244‡	1203 ± 243*	1630 ± 319‡	2256 ± 272‡	2636 ± 577‡	<0.0001
	DE	402 ± 131	782 ± 203	1033 ± 193	1298 ± 213	1858 ± 253	2601 ± 296	3370 ± 708	
HR (beat/min^{-1})	IE	69 ± 9	85 ± 10	93 ± 9	107 ± 11	127 ± 13	153 ± 12	166 ± 11	0.1784
	DE	75 ± 17	88 ± 10	97 ± 12	112 ± 13	131 ± 13	154 ± 14	175 ± 13	
SV (mL)	IE	84 ± 14*	92 ± 12*	97 ± 13*	105 ± 13‡	109 ± 15‡	108 ± 17*	109 ± 16*	0.0166
	DE	74 ± 11	84 ± 12	90 ± 13	91 ± 13	95 ± 12	97 ± 15	101 ± 17	
$\dot{Q}$ (L min^{-1})	IE	5.8 ± 1.1	7.8 ± 1.0	9.0 ± 1.1	11.2 ± 1.6†	13.7 ± 1.6‡	16.6 ± 2.5‡	18.3 ± 3.2	0.0244
	DE	5.4 ± 1.3	7.5 ± 1.3	8.7 ± 1.3	9.7 ± 1.6	11.8 ± 1.2	14.8 ± 1.8	17.6 ± 2.9	
C(a-v)O$_2$ (mL/100 mL)	IE	6.36 ± 1.84‡	6.77 ± 3.10*	9.31 ± 3.25*	10.96 ± 3.02*	11.86 ± 2.67‡	13.87 ± 2.80*	14.62 ± 2.72‡	<0.0001
	DE	7.07 ± 1.62	10.48 ± 2.74	12.03 ± 2.49	13.62 ± 3.00	15.73 ± 2.31	17.64 ± 2.39	18.75 ± 2.75	
EF (%)	IE	69 ± 8	71 ± 9	73 ± 9	75 ± 9	75 ± 10	73 ± 12	71 ± 11	0.0861
	DE	62 ± 10	66 ± 11	68 ± 11	69 ± 11	69 ± 11	68 ± 9	68 ± 9	
EDV (mL)	IE	123 ± 24	132 ± 26	136 ± 26	142 ± 28	147 ± 31	154 ± 41	153 ± 43	0.8864
	DE	121 ± 26	138 ± 33	141 ± 29	140 ± 26	143 ± 27	147 ± 33	149 ± 26	
ESV (mL)	IE	39 ± 16	39 ± 19	37 ± 18	36 ± 19	38 ± 22	45 ± 32	45 ± 31	0.2916
	DE	47 ± 20	48 ± 23	47 ± 26	45 ± 22	46 ± 22	48 ± 21	48 ± 20	
Contractility Index (A.U.)	IE	196 ± 78	201 ± 70	239 ± 79	256 ± 84	264 ± 88	239 ± 105	226 ± 95	0.1360
	DE	136 ± 54	187 ± 96	206 ± 106	217 ± 101	221 ± 90	205 ± 64	204 ± 75	
SBP (mmHg)	IE	113 ± 10	113 ± 9	117 ± 9	122 ± 8	129 ± 8	146 ± 14	152 ± 15	0.3179
	DE	111 ± 6	111 ± 6	115 ± 9	120 ± 12	133 ± 13	155 ± 14	166 ± 18	
DBP (mmHg)	IE	69 ± 6	70 ± 6	70 ± 5	70 ± 7	71 ± 8	72 ± 9	72 ± 8	0.1031
	DE	72 ± 6	72 ± 6	72 ± 6	70 ± 5	73 ± 6	76 ± 8	78 ± 8	
SVR (dyne s^{-1} cm^{-5})	IE	1270 ± 482	924 ± 231	799 ± 180	670 ± 165	561 ± 116	516 ± 160	489 ± 140	0.602
	DE	1330 ± 331	911 ± 201	786 ± 124	727 ± 117	630 ± 77	555 ± 94	493 ± 67	

Values are mean ± SD. IE: immersed ergocycle; DE: dryland ergocycle; P_{max}: maximal power output; $\dot{V}O_2$: oxygen uptake; HR: heart rate; $\dot{Q}$: cardiac output, SV: stroke volume; C(a-v)O$_2$: arteriovenous difference; EF: ejection fraction; EDV: end diastolic volume; ESV: end systolic volume, A.U.: arbitrary unit; SBP: systolic blood pressure; DBP: diastolic blood pressure; SVR: systemic vascular resistance. Condition effect (immersions vs. dryland).

* $P < 0.05$.
† $P < 0.01$.
‡ $P < 0.001$.

Table 2
Comparison of oxygen uptake and hemodynamic parameters during recovery on immersed (IE) and dryland ergocycle (DE).

Parameters		1 min	2 min	3 min	4 min	5 min	P-value Condition effect
$\dot{V}O_2$ (mL/min^{-1})	IE	1532 ± 514‡	874 ± 505†	546 ± 375‡	448 ± 293*	406 ± 214*	<0.0001
	DE	2140 ± 509	1287 ± 306	932 ± 258	665 ± 204	585 ± 212	
SV (mL)	IE	106 ± 17*	110 ± 16‡	104 ± 19	104 ± 19*	99 ± 18*	0.0248
	DE	95 ± 12	95 ± 11	95 ± 17	91 ± 11	89 ± 110	
$\dot{Q}$ (L min^{-1})	IE	14.8 ± 3.0	13.3 ± 2.9	11.4 ± 3.0	10.7 ± 2.6	10.1 ± 2.4	0.2402
	DE	14.2 ± 2.1	12.2 ± 1.8	10.6 ± 1.8	9.6 ± 1.6	9.2 ± 1.6	
C(a-v)O$_2$ (mL/100 mL)	IE	10.50 ± 4.12†	6.33 ± 3.22‡	4.77 ± 2.59	4.19 ± 2.34†	3.99 ± 1.88‡	<0.0001
	DE	14.83 ± 3.41	10.46 ± 2.89	8.89 ± 2.62	6.91 ± 2.23	6.38 ± 2.43	
EF (%)	IE	76 ± 10*	77 ± 9†	75 ± 8*	75 ± 9*	73 ± 8*	0.0460
	DE	69 ± 9	69 ± 10	69 ± 11	68 ± 9	67 ± 9	
EDV (mL)	IE	141 ± 28	144 ± 23	138 ± 26	140 ± 32	138 ± 30	0.7011
	DE	139 ± 23	140 ± 26	139 ± 27	135 ± 24	135 ± 24	
ESV (mL)	IE	34 ± 20	33 ± 16	34 ± 15	36 ± 19	38 ± 18	0.2388
	DE	44 ± 19	45 ± 22	44 ± 22	44 ± 20	45 ± 20	
SBP (mmHg)	IE	149 ± 13	143 ± 12	136 ± 14	130 ± 14	127 ± 14	0.1466
	DE	154 ± 21	145 ± 17	143 ± 18	134 ± 13	129 ± 13	
DBP (mmHg)	IE	67 ± 9	65 ± 9	63 ± 7	61 ± 8	64 ± 6	0.0527
	DE	70 ± 8	67 ± 8	68 ± 7	68 ± 7	66 ± 7	
Contractility index (A.U.)	IE	297 ± 120*	304 ± 119*	282 ± 108*	265 ± 89*	238 ± 73*	0.0342
	DE	220 ± 73	220 ± 81	220 ± 89	208 ± 72	193 ± 63	
SVR (dyne s^{-1} cm^{-5})	IE	559 ± 198	602 ± 195	694 ± 279	710 ± 311	744 ± 291	0.7195
	DE	552 ± 95	611 ± 111	719 ± 167	762 ± 170	777 ± 172	

Values are mean ± SD. IE: immersed ergocycle; DE: dryland ergocycle; $\dot{V}O_2$: oxygen uptake; HR: heart rate; $\dot{Q}$: cardiac output, SV: stroke volume; C(a-v)O$_2$: arteriovenous difference; EF: ejection fraction; EDV: end diastolic volume; ESV: end systolic volume, A.U.: arbitrary unit; SBP: systolic blood pressure; DBP: diastolic blood pressure; SVR: systemic vascular resistance. Condition effect (immersions vs. dryland):

* $P < 0.05$.
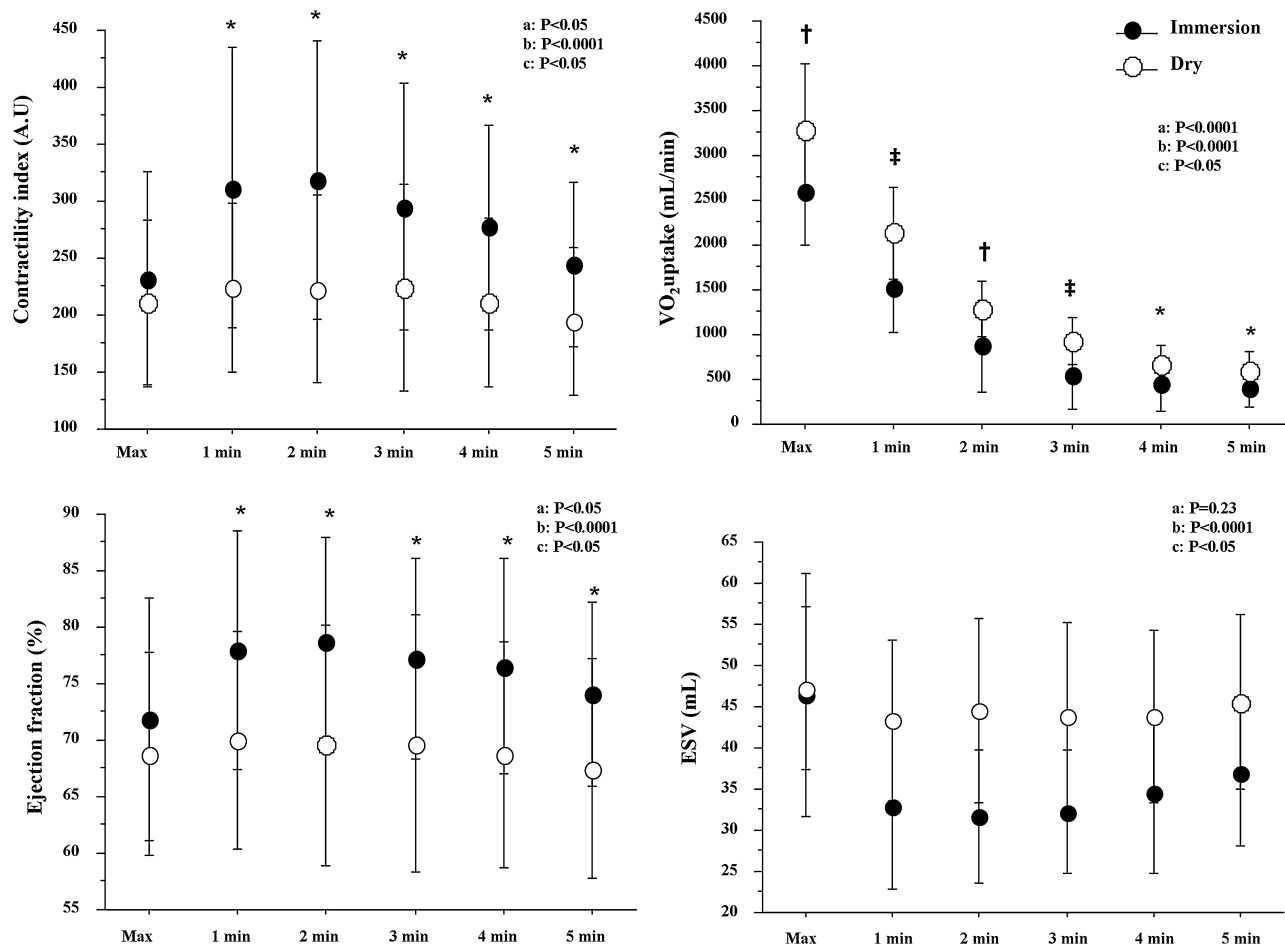† $P < 0.01$.
‡ $P < 0.001$.
§ $P < 0.0001$.

Fig. 1. Recovery kinetics for $\dot{V}O_2$, ejection fraction, end-systolic volume (ESV), and contractility index in dryland and immersion. (a) Condition effect, (b) time effect, (c) interaction effects (condition × time). Difference revealed by post hoc analysis (*$P<0.05$, †$P<0.01$, ‡$P<0.001$).

conditions. Moreover, we report for the first time in man, post-exercise central hemodynamic responses after immersed cycling exercise.

Our findings regarding the lower $\dot{V}O_2$ and C(a-v)O_2 and higher cardiac output and stroke volume at submaximal and maximal levels (SV only) during IE vs. DE are in agreement with one previous study performed on IE[3] and another during swimming vs. running.[19] Furthermore, with respect to $\dot{V}O_2$ peak, our results agree with one study[20] but disagree with two others[12,21] suggesting no difference in $\dot{V}O_2$ peak during IE and DE. This discrepancy in results may potentially be explained by small sample size in previous studies, the fact that calculation of the P_{ext} in water did not take into account the water drag force on the legs or pedalling system or the exercise protocol used.[12,21]

The lower C(a-v)O_2 during exercise explains in great part the reduced $\dot{V}O_2$ at sub and maximal levels we observed during IE. In humans, thermoneutral immersion at rest was shown to increase skeletal muscle blood flow in the legs,[22] suggesting a hyperperfusion state of leg skeletal muscle at the start of exercise (rest). Therefore, we speculate that immersion may cause hyperperfusion also during exercise, increasing muscle blood flow and reducing red cell transit time, thereby decreasing muscle oxygen diffusion. Regarding cardiac output and stroke volume, our results agree with previous studies demonstrating higher values during exercise at submaximal[3,7,12] and maximal levels during IE.[12] Previous studies[3,12] have compared hemodynamic responses during exercise on IE at the same level of $\dot{V}O_2$, however results could be biased given that $\dot{V}O_2$ is affected by immersion and that the P_{ext} was not

equivalent.[10,11] Only the studies of Brechat et al.,[7,8] attempted to compare the cardiovascular responses at an equivalent P_{ext} (120 W) during IE and DE, and found a higher $\dot{V}O_2$, cardiac index and stroke index during IE. However, in those two studies, the P_{ext} was measured by an immersible power torque and the drag water resistance on the legs (foot, calf, and thigh) was not taken into account and has been shown to confer an additional external power expenditure of approximately 15%.[10,11]

As demonstrated in previous studies[3,12,23] the higher cardiac output during immersion depends mainly on the augmentation of stroke volume. The determinants of cardiac output are heart rate, preload, afterload and contractility.[12,23,24] Previous studies during exercise (at the same $\dot{V}O_2$ level)[12,23] demonstrated an increase of diastolic and systolic diameters as well as end diastolic and systolic volume during immersion[12] suggesting an increased cardiac preload (via the Frank Starling mechanism). In our study, immersion tended to increase ejection fraction ($P=0.08$); we may speculate that the increase in stroke volume might be due to a combination of a reduced afterload and/or increased contractility given that end-diastolic volume (preload) was not increased (Table 1).

Blood pressure and systemic vascular resistance were not different between the two conditions. Previous studies have demonstrated either no difference[4,12] or a reduction of these parameters during IE.[25] The reduction of resting systemic vascular resistance during immersion appears to be due to stimulation of the cardiopulmonary baroreceptors by the central shift of blood volume and with a concomitant reduction of sympathetic activity.[3,25]

During the recovery, $\dot{V}O_2$ and C(a-v)O_2 remained lower during immersion while stroke volume and ejection fraction were higher. We believe that this is due to persistent hyperperfusion of lower limb skeletal muscles during recovery with continued increased muscle blood flow and reduced red cell transit time, thereby decreasing muscle oxygen diffusion. Furthermore, the increased stroke volume during recovery in immersion could be mediated by increased left ventricular contractility (increased contractility index) compared to exercise on dryland condition given the absence of any differences in end systolic or diastolic volumes (Table 2). Heart contractility is known to be increased by two mechanisms that include: (1) beta-adrenergic stimulation, (2) myocardial fiber length/sensitization of the contractile protein to cytosolic calcium concentration.[24] Because immersion reduces sympathoadrenergic activity[4,25] after exercise, our increased contractility index may be explained by an increased sensitivity of cardiac contractile proteins to calcium.[24]

5. Conclusions

During exercise and recovery in immersion, $\dot{V}O_2$ and C(a-v)O_2 were reduced in healthy young participants. We may believe that the reduced muscle O_2 extraction during immersion could occur due to increased blood flow and hyperperfusion of lower limb skeletal muscle, reducing red cell transit time and thereby decreasing muscle oxygen diffusion. In parallel, during immersed cycling exercise, stroke volume and cardiac output were improved for the same P_{ext}. This may be due to a combination of decreased afterload and/or increased contractility. During recovery, immersion increased stroke volume, ejection fraction and contractility presumably via an increased sensitivity of cardiac contractile proteins to calcium. Further studies are needed to understand by which mechanisms $\dot{V}O_2$ is decreased during water exercise on IE at the same external power output (P_{ext}) relative to exercise on DE.

Practical implications

- Immersion favors higher cardiac responses during incremental exercise and its recovery.
- Exercise in immersion may represent a more important stress to the cardiac system and could be beneficial for cardiac adaptations during exercise training program.
- Exercise training in immersion could also be interesting during athlete's rehabilitation program to maintain and/or improve cardiac function more rapidly.

Acknowledgements

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.jsams.2014.08.005.

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