Cardiovascular responses to water immersion in humans: impact on cerebral perfusion

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1School of Sport Science, Exercise and Health, The University of Western Australia, Crawley, Western Australia; 2Centre for Heart, Lung and Vascular Health, School of Health and Exercise Sciences, Faculty of Health and Social Development, University of British Columbia, Kelowna, Canada; and 3Research Institute for Sport and Exercise Science, Liverpool John Moores University, Liverpool, United Kingdom

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Carter HH, Spence AL, Pugh CJ, Ainslie P, Naylor LH, Green DJ. Cardiovascular responses to water immersion in humans: impact on cerebral perfusion. Am J Physiol Regul Integr Comp Physiol 306: R636–R640, 2014. First published February 19, 2014; doi:10.1152/ajpregu.00516.2013.—Epidemic increases in cerebrovascular perfusion and shear stress may have beneficial impacts on endothelial function that improve brain health. We hypothesized that water immersion to the level of the right atrium in humans would increase cerebral perfusion. We continuously measured, in 9 young (mean ± SD, 24.6 ± 2.0 yr) healthy men, systemic hemodynamic variables along with blood flows in the common carotid and middle and posterior cerebral arteries during controlled filling and emptying of a water tank to the level of the right atrium. Mean arterial pressure (80 ± 9 vs. 91 ± 12 mmHg, P < 0.05), cardiac output (4.8 ± 0.7 vs. 5.1 ± 0.6 l/min, P < 0.05) and end-tidal carbon dioxide (PeTCO2, 39.5 ± 2.0 vs. 44.4 ± 3.5 mmHg, P < 0.05) increased with water immersion, along with middle (59 ± 6 vs. 64 ± 6 cm/s, P < 0.05) and posterior cerebral artery blood flow velocities (41 ± 9 vs. 44 ± 10 cm/s, P < 0.05). These changes were reversed when the tank was emptied. Water immersion is associated with hemodynamic and PeTCO2 changes, which increase cerebral blood velocities in humans. This study provides an evidence base for future studies to examine the potential additive effect of exercise in water on improving cerebrovascular health.

Subject Characteristics

Nine young, healthy recreationally active (≥2 h of physical activity per week) males were recruited (24.6 ± 2.0 yr). The participant’s average heights and weights were 1.74 ± 0.09 m and 76 ± 9 kg, respectively, with the average body mass index (BMI) being 25.0 ± 1.7 kg/m². Subjects had no history of cardiovascular, musculoskeletal or metabolic disease, did not smoke, or take medication. Women were excluded from this study due to the effects of estrogen on hemodynamic and vascular variables.

Experimental Procedures

Subjects arrived at the laboratory after having fasted for a minimum of 8 h and abstained from alcohol, caffeine, and vigorous exercise for at least 24 h. Upon arrival, subjects were seated and instrumented (~30 min). They were then positioned in a tank (1.4 m diameter, 1.55 m height, 2.386 liters) in a standing position with their arms resting comfortably on a platform at heart level. Subjects were asked to remain as stationary as possible throughout the experiment and to avoid excessive movement. This experimental approach avoided the potential for confounding effects of movement into, or out of, the tank on hemodynamics and CBFV. After a 10-min baseline period of quiet rest, three submersible water pumps (KPA 600A; Grundfos, South Australia) filled the tank at a constant rate with euthermic water (30°C) to the level of the right atrium (RA), a process that was completed in 7 min. This water temperature was selected on the basis of experiments in our laboratory which indicated that basal skin temperatures average 30.1 ± 1.6°C. The subjects remained immersed at the level of the RA for 10 min while remaining in a stationary standing posture, after
which time the pumps were reversed and water rapidly evacuated at a constant rate. Subjects attended the laboratory wearing shorts and a tee-shirt. The average ambient air temperature throughout the testing sessions was 26.0 ± 3.4°C. Before the commencement of baseline recording, after instrumentation, tee-shirts were removed for the duration of the testing session. Data were measured and recorded continuously throughout the entire protocol.

Experimental Measures

Systemic hemodynamics. A Finometer PRO (Finapres Medical Systems, Amsterdam, The Netherlands) was used to measure changes in mean arterial pressure (MAP), heart rate (HR), cardiac output (CO), and stroke volume (SV) via photoplethysmography. These data were exported to a data acquisition system PowerLab (LabChart 7, ADInstruments, Sydney, Australia) in real-time. The finger cuff was placed around the middle finger of an arm supported at right atrium level on a platform. The subject was instructed not to move their arm or finger during recording. All summary data were time averaged for 1–2 min.

Assessment of middle and posterior cerebral artery velocities. Middle and posterior cerebral artery velocities (MCAV and PCAV, respectively) were measured using a 2-MHz pulsed ST3 transcranial ultrasound system (Spencer Technologies, Seattle, WA). Search techniques adopted to identify the MCA and PCA are described in detail elsewhere. (2) A Marc 600 headframe (Spencer Technologies) was secured to allow for adjustments to the insonation angle until an optimal M-mode image was found. Raw analog MCAV and PCAV cerebral velocity traces were exported from PowerLab to LabChart for post hoc analysis. All data at respective time points were averaged for 1–2 min.

End-tidal carbon dioxide (PetCO2) was collected in seven subjects via a sampling tube connected to a Hans Rudolph mask and was measured by an online gas analyzer (ML206; ADInstruments).

Assessment of common carotid artery diameter and velocity. After the 10-min baseline period, common carotid artery (CCA) diameter and velocities were simultaneously recorded throughout the protocol using a 10-MHz multifrequency linear array probe attached to a high-resolution ultrasound machine (T3000; Terason, Burlington, MA). Recording began following optimization of the longitudinal B-mode image of the lumen-arterial walls. Concurrently, Doppler velocity assessments were collected using the lowest possible insonation angle (always <60°). Subjects’ arms were supported at heart level throughout the test.

Analysis of artery diameter and flow were performed using custom-designed edge-detection and wall-tracking software, which is independent of investigator bias and has previously been comprehensively described (8, 43). From synchronized diameter and velocity data, blood flow (the product of lumen cross-sectional area and Doppler velocity) was calculated at 30 Hz. Reproducibility of diameter measurements using this semiautomated software is significantly better than manual methods, reduces observer error and bias significantly, and possesses an intraobserver CV of 6.7% (43).

Statistics

Statistical analysis was performed using SPSS 19.0 (SPSS, Chicago, IL) software. Repeat-measure ANOVAs were performed with post hoc analysis t-tests used where significant values were found. Statistical significance was assumed at P < 0.05. All data are reported as means ± SD unless stated otherwise.

RESULTS

Middle and Posterior Cerebral Artery Velocities

A repeated-measures ANOVA revealed a significant increase in MCAV from baseline throughout the protocol (P < 0.05, Fig. 1A). Post hoc analysis revealed MCAV increased significantly during filling to the hip, upon immersion to the RA, and after 5 and 10 min at RA level (all P < 0.05). Once the tank was emptied, MCAV returned to baseline levels (P < 0.05). Similarly, PCAV increased (P < 0.05) with differences evident during filling to the hip, upon immersion to the RA, and after 5 and 10 min at RA level (P < 0.05). Consistent with the MCAV response, PCAV returned to baseline levels once the tank was emptied (P < 0.05, Fig. 1B). PetCO2 values increased during immersion (P < 0.05) with differences evident upon immersion to the RA and after 5 and 10 min of
Continuous immersion at the RA (all \( P < 0.05 \), Fig. 1D). However, no change in respiratory rate was evident throughout the immersion protocol (\( P = 0.97 \), Table 1).

**Impact of Water Immersion on Systemic Hemodynamics**

Repeated-measures ANOVA revealed a significant increase in MAP from baseline throughout the protocol (\( P < 0.05 \), Fig. 1C). Post hoc analysis indicated that MAP was significantly elevated above baseline during filling (at the level of the hip), upon immersion to the RA, and after 5 and 10 min of immersion (all \( P < 0.05 \)). Once the tank was emptied, MAP decreased significantly compared with immersion data (\( P < 0.05 \). In keeping with MAP data, CO increased (\( P < 0.05 \)), with differences evident after 10 min of immersion (\( P < 0.05 \), Table 1). CO decreased as a result of tank emptying and was not significantly different from baseline levels after 5 min.

SV also increased (\( P < 0.05 \)), with differences during immersion to the hip, upon immersion to the RA, and after 5 and 10 min at RA level (\( P < 0.05 \), Table 1). However, SV returned to baseline values 5 min postemptying. HR decreased during immersion (\( P < 0.05 \)) with significant differences during filling (hip), upon immersion to the RA, and after 5 and 10 min at RA level (all \( P < 0.05 \)) (Table 1). Once the tank was emptied, HR returned to baseline values.

**Common Carotid Artery Diameter and Blood Flow**

Common carotid diameter increased significantly (\( P < 0.05 \)) with differences upon immersion to the RA and after 5 and 10 min (all \( P < 0.05 \)) (Table 1). However, there was no significant change in common carotid flow throughout the protocol (Table 1).

**Correlations With Cerebral Blood Flow Velocities**

Correlations and multiple regression analysis were performed using change from baseline values at each data collection point. Significant correlations existed between changes in MCAV and changes in MAP (Fig. 2, \( r = 0.32, P < 0.05 \)), changes in PCAV and MAP (\( r = 0.50, P < 0.01 \)), changes in \( \text{PETCO}_2 \) and MCAV (\( r = 0.71, P < 0.001 \)), and changes in \( \text{PETCO}_2 \) and PCAV (\( r = 0.68, P < 0.001 \)). Multiple regression analysis using \( \text{PETCO}_2 \) and MAP as predictors of MCAV produced \( R^2 = 0.509, F = 16.60, P < 0.001 \). This analysis revealed only \( \text{PETCO}_2 \) added significantly to the MCAV prediction (\( P < 0.001 \)), whereas MAP did not (\( P = 0.95 \)).

**DISCUSSION**

The aim of the present study was to comprehensively examine the cardiovascular response to graded water immersion in standing subjects using a rapid filling and emptying protocol. The tank was filled with euthermic water (30°C) to avoid any reflex responses elicited by cold or heat and to provide a model allowing for the direct and accurate examination of hydrostatic effects per se. Our principle finding is that water immersion at rest has significant impacts on hemodynamic variables and \( \text{PETCO}_2 \), which are associated with increases in CBFV.

Two important stimuli that can elicit changes in CBFV are blood \( CO_2 \) levels and MAP. In the current study we observed significant changes in CBFV that were consistent, and correlated with, changes in MAP. Traditionally, on the basis of steady-state measures made in patients with hypertensive and hypotensive disorders, CBFV was considered to be autoreg-

<table>
<thead>
<tr>
<th>Variable Baseline</th>
<th>Hip 0–1 min</th>
<th>3–5 min</th>
<th>8–10 min</th>
<th>Rest</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac output, l/min</td>
<td>4.8 ± 0.7</td>
<td>4.7 ± 0.5</td>
<td>5.1 ± 0.8</td>
<td>5.9 ± 0.6</td>
</tr>
<tr>
<td>Stroke volume, ml</td>
<td>62 ± 9</td>
<td>70 ± 9*</td>
<td>81 ± 8*</td>
<td>83 ± 7*</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>78 ± 12</td>
<td>67 ± 12*</td>
<td>63 ± 11*</td>
<td>60 ± 10*</td>
</tr>
<tr>
<td>Carotid artery diameter, mm</td>
<td>5.74 ± 0.49</td>
<td>5.99 ± 0.36*</td>
<td>6.05 ± 0.43*</td>
<td>6.12 ± 0.34*</td>
</tr>
<tr>
<td>Carotid artery flow, ml/min</td>
<td>458 ± 162</td>
<td>486 ± 139</td>
<td>490 ± 132</td>
<td>503 ± 127</td>
</tr>
<tr>
<td>Respiratory rate, breaths/min</td>
<td>15 ± 3</td>
<td>15 ± 5</td>
<td>16 ± 5</td>
<td>14 ± 5</td>
</tr>
</tbody>
</table>

Values are means ± SD. \( n = 7 \) for respiratory rate variables. *Significantly different from rest at \( P < 0.05 \).
lated such that relatively constant levels were maintained across a wide range of MAP (60–150 mmHg) (23). However, subsequent technical advances revealed that CBFV can respond rapidly to dynamic changes in MAP, a concept termed dynamic cerebral autoregulation (1, 40). In contrast to previous studies, which have either assessed CBFV responses to static or dynamic stimuli, the present study utilized a novel approach that assessed the impact of both rapid and sustained changes in MAP on CBFV. Interestingly, we observed no evidence of autoregulation in response to either rapid or sustained filling in the current study. However, the relationship between MAP and CBFV must be also considered in the context of increases in PETCO2. It is known that water immersion alters the mechanics of respiration. For example, the centralized shift in blood volume to the thorax along with the ascent of the diaphragm has been shown to significantly reduce functional residual volume and increase pulmonary capillary blood volume (3, 7, 15), whereas a further study reported no change in tidal volume during immersion in 25–30°C water (20). In the current study, water immersion was associated with an increase in PETCO2. Breathing rate did not change significantly throughout the immersion protocol, suggesting that changes in tidal volume may have been responsible for the rise in PETCO2. Future studies are required to elucidate the casual pathways. Importantly, the changes in PETCO2 were correlated with those in CBFV, and a multiple regression analysis revealed it was the strongest predictor for changes in MCAV. We can therefore not exclude the possibility that increases in PETCO2 masked autoregulatory responses to changes in MAP. For example, it is known that even small elevations in PETCO2 can reduce the effectiveness of cerebral autoregulation (4). Regardless of the mechanism(s) responsible, our principle finding was that water immersion increased CBFV, and this is the important functional outcome of the study.

While our principle interest was changes in CBFV, our experiment provides an insight into the central hemodynamic effects of water immersion. There have been numerous studies examining the effects of water immersion on cardiovascular variables; however, there are notable discrepancies in their findings. For example, several studies have reported increases in CO following water immersion (5, 7, 10, 15, 27, 33, 42, 44), whereas others have reported decreases (37) or no change (21, 29). HR responses are similarly conflicting, with studies reporting increases (5, 7, 10, 11, 29, 42, 44) and decreases (14–16, 22, 27, 33, 37, 38). Finally, MAP responses have also reported increases (5, 33), decreases (25, 32), or no change (10, 16, 31). These conflicting results are related to differences in methodology, in particular posture, the duration of immersion, and water temperature, as highlighted in a study by Park et al. (33), all of which may elicit reflex changes. In the present study we aimed to clarify the hydrostatic impacts of water immersion per se in upright humans by performing a comprehensive integrative examination of the systemic cardiovascular response to immersion in euthermic water. By employing a filling and emptying protocol we were able to demonstrate reversibility, reinforcing the validity of our measures. We also used independent techniques to measure related variables. For example, cerebral velocities were assessed by transcranial Doppler, whereas common carotid blood flows were assessed using linear array duplex ultrasound. The concordance between these measures reassures us that our data are robust. We also observed increases in CO, SV, and MAP as a result of water immersion, which were reversed when the tank was emptied. It is presumed that a cephaloid redistribution of blood volume as a result of immersion increases preload and SV, in accordance to the Frank-Starling mechanism. The decrease in HR we observed is likely to be a reflex response to the increase in MAP. Our finding of changes in CCA diameter during immersion is consistent with baroreflex elicitation.

A limitation of the present study is our cardio- and cerebrovascular responses to water immersion were collected in young healthy individuals, and our results are therefore not necessarily translatable to other population or clinical groups, i.e., the elderly or heart failure groups.

Implications

Our findings of increased CBFV during water immersion at rest raise the possibility of acute and chronic impacts of water immersion on cerebrovascular function. There is evidence for basal vascular nitric oxide-mediated tone in the cerebrovasculature (13) and exercise improves cerebrovascular endothelial function by upregulating endothelial nitric oxide synthase expression in animals (13, 17). Acute increases in blood flow and shear in peripheral arteries modulate endothelial function (41), and chronic episodic exposure to shear stress induces endothelial adaptation (30, 41). We speculate that water immersion may amplify effects on CBFV, including those associated with exercise training.

Perspectives and Significance

This study indicates that euathmic water immersion in resting humans increases MAP, PETCO2, and, consequently, CBFV. Given that increases in blood flow and shear in the peripheral vasculature are potent stimuli for endothelial adaptation, our observation that CBFV increases during water immersion has potential implications for the use of aquatic exercise as a modality that promotes brain health.

GRANTS

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s). The results of the present study do not constitute endorsement by the American College of Sports Medicine.

AUTHOR CONTRIBUTIONS


REFERENCES


