

## Hot Head-Out Water Immersion Acutely Impairs Cerebral Autoregulation in Healthy Participants

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Recurring hot head-out water immersion enhances peripheral vascular function and cerebral blood flow during non-immersion conditions. However, it is not known if an acute bout of hot head out water immersion (HOWI) improves cerebral autoregulation versus thermoneutral HOWI. PURPOSE: We tested the hypothesis that dynamic cerebral autoregulation is greater during and following an acute bout of hot (HOT) vs. thermoneutral (TN) HOWI. METHODS: Seven healthy participants (age: 23±2 y, 2 females) completed two randomized trials consisting of 30 min HOT (39°C) or TN (35°C) HOWI. Beat-to-beat blood pressure (MAP), middle cerebral artery blood velocity (MCAv), and end-tidal partial pressure of  $CO_2$  (PETCO<sub>2</sub>) were recorded continuously. After 5 min of resting baseline, participants breathed through a respiratory impedance device for 5 min to assess cerebral autoregulation using Fourier transformation. Cerebral autoregulation testing was completed pre, 25 min into immersion (during), and immediately post HOWI. **RESULTS:** MAP, MCAv, PETCO<sub>2</sub>, gain, and phase were not different between HOT and TN at pre (P>0.14 for all). MAP was different between HOT and TN during (77±6 vs 91±9 mmHg; P<0.01) and post (81±9 vs 92±12 mmHg; P=0.03). MCAv was not different between HOT and TN during (63±8 vs 65±11 cm/s; P=0.28) or post (58±10 vs 62±11 cm/s; P=0.22). PETCO<sub>2</sub> was different between HOT and TN during  $(42\pm2 \text{ vs } 44\pm4 \text{ mmHg}; P=0.04)$  but was not different at post  $(39\pm3 \text{ vs } 40\pm4 \text{ mmHg}; P=0.13)$ . Gain was different between HOT and TN during (1.0±0.2 vs 0.9±0.2 cm/s/mmHg; P=0.04) but was not different post (1.0±0.2 vs 0.9±0.2 cm/s/mmHg; P=0.15). Gain increased from pre in HOT during (P=0.03) but was not elevated post (P=0.15). Gain was not different from pre in TN during (P=0.95) or post (P=0.95). Phase was not different between HOT and TN during (12±7 vs  $12\pm5^\circ$ ; P=0.48) or post (8±12 vs  $11\pm7^\circ$ ; P=0.30). Phase was not different from pre in HOT or TN during (HOT: P=0.79 or TN: P=0.70) or post (HOT: P=0.70 or TN: P=0.74). CONCLUSION: These preliminary data indicate that the ability of the cerebral vasculature to buffer changes in blood pressure during hot HOWI is impaired compared to nonimmersion conditions and thermoneutral HOWI in healthy participants.

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