TACSM Abstract –

Hyperthermia does not alter baroreflex control of heart rate during central hypovolemia associated with simulated hemorrhage.

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ABSTRACT

Baroreceptors modulate arterial blood pressure through neural control of cardiac output and peripheral vascular resistance. Hyperthermia reduces tolerance to central hypovolemia, however hyperthermia itself does not alter baroreflex control of heart rate or muscle sympathetic nerve activity. The combined influence of hyperthermia and profound central hypovolemia on baroreflex function remains unknown. This study tested the hypothesis that baroreflex sensitivity (as indexed from Δ heart rate /Δ blood pressure) would be reduced in hyperthermia compared to normothermia during central hypovolemia. Twelve healthy males (32 ± 5 y) underwent central hypovolemia to pre-syncope, which was induced via progressive lower body negative pressure (LBNP), during normothermia and, on a separate day (randomized, counter balanced) during hyperthermia (+1.2 ± 0.1°C increase in internal temperature). Baroreflex sensitivity was assessed during the final full stage of LBNP completed, and during a 30 second period immediately preceding any bradycardia prior to pre-syncope. LBNP during hyperthermia reduced tolerance by ~58% relative to LBNP during normothermia (normothermia: 72mmHg ±20 vs hyperthermia: 42mmHg± 13; $P<0.001$). During the final full LBNP stage, baroreflex sensitivity was not different between thermal conditions (hyperthermia: 1.9 ± 1.6 bpm/mmHg, normothermia: 2.4 ± 1.5 bpm/mmHg, $p=0.46$). Likewise, just prior to pre-syncope baroreflex sensitivity between thermal conditions was not different (hyperthermia: 1.0±1.3 bpm/mmHg, normothermia: 1.5±1.1 bpm/mmHg; $p=0.10$). These data indicate that during profound central hypovolemia baroreflex control of heart rate is unaffected by hyperthermia. Thus, reductions in the capacity to tolerate central hypovolemia while hyperthermic is not related to altered baroreflex control of heart rate.

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