Measurement of Renal Hemodynamics by Doppler Ultrasound during Sympathetic Activation while Heat Stressed

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Abstract

Introduction: P-aminohippurate clearance demonstrates that passive heat stress increases renal vascular resistance (RVR) in proportion to increases in core temperature. Exercise, a sympathetic stimulus, also increases RVR, but this increase is attenuated during heat stress. Additionally, passive heat stress attenuates the vascular response to the cold pressor test (CPT), a non-exercise sympathetic stimulus. However, the effect of passive heating on renal vascular responsiveness to a non-exercise sympathetic stimulus is unknown.

Purpose: Test the hypothesis that passive heat stress attenuates increases in RVR during the CPT compared to normothermia.

Methods: Twelve healthy adults (age: 22 ± 1 y, 4 women) completed a CPT by immersing their right hand in an agitated ice slurry mixture for 2 min during normothermia (Normothermia), and after intestinal temperature increased by 1.2 ± 0.1°C following passive heating with a water perfused suit (Heated). Mean arterial pressure (MAP), finger photoplethysmography, heart rate (HR), 3-lead ECG, and renal blood velocity (RBV) were measured at 1 min pre-CPT, and at 1 and 2 min into the CPT (T1 and T2). RBV was assessed in the distal segment of the right renal artery during the same phase of the respiratory cycle via Doppler ultrasound (GE Vivid 7 Dimension). The coronal approach was utilized using a linear-array transducer with a 2.5–3.5 MHz pulsed frequency, and participants assumed the left lateral recumbent position. All measurements were obtained by the same sonographer with the focal zone set to the artery’s depth, and the transducer held in the same location at an insonation angle <60°. RVR was calculated as MAP/RBV and RVR and RBV were averaged across 2–4 beats. Data are presented as the absolute change from pre-CPT (mean ± SD) unless otherwise stated.

Results: Pre-CPT, MAP was lower (77 ± 9 vs 86 ± 9 mmHg, P=0.01) and HR was higher (100 ± 12 vs 96 ± 7 bpm, P=0.01) in Heated. RBV was lower (Heated: 31.1 ± 4.8 Normothermia: 34.2 ± 6.3 cm/s, P=0.056), but RVR did not differ (Heated: 2.6 ± 0.6, Normothermia: 2.7 ± 0.7 mmHg/cm • s, P=0.33) between Normothermia and Heated pre-CPT. Elevations in MAP (T1: 20 ± 11; T2: 25 ± 11 mmHg) and HR (T1: 16 ± 9; T2: 13 ± 7 bpm) during Normothermia differed from the MAP (T1: 5 ± 3; T2: 5 ± 5 mmHg) and HR (T1: 1 ± 10; T2: 1 ± 10 bpm) response evoked by the CPT during Heated (P<0.01). Changes in RBV during the CPT did not differ between Heated and Normothermia at T1 (Heated: 1.3 ± 6.2; Normothermia: -3.1 ± 6.6 cm/s, P=0.76) or T2 (Heated: 1.6 ± 6.0; Normothermia: -0.3 ± 7.5 cm/s, P=0.73). Changes in RVR did not differ between Heated and Normothermia at T1 (Heated: 0.3 ± 0.6; Normothermia: 0.9 ± 1.0 mmHg/cm • s, P=0.06), but were lower in Heated at T2 (0.0 ± 0.5 vs 0.9 ± 1.1 mmHg/cm • s, P=0.01).

Conclusion: These data indicate that passive heat stress attenuates Doppler ultrasound derived measures of renal vascular responsiveness to the CPT, a non-exercising sympathetic stimulus.

Purpose

Test the hypothesis that passive heat stress attenuates increases in RVR during the CPT compared to normothermia.

Methods

Protocol

• Twelve healthy adults (age: 22 ± 1 y, 4 women) completed a CPT by immersing their hand in an agitated ice slurry mixture for 2 min, during normothermia (Normothermia) and after passive heating (Heated).

• Passive heating involved increasing intestinal temperature by 1.2 ± 0.1°C with a water perfused suit.

• Data were analyzed at Pre-CPT (Pre), and 1 min and 2 min into the CPT.

• Data are reported as a change (Δ) from Pre (mean ± SD).

Dependent Variables

• Mean arterial pressure (Photoplethysmography)

• Heart rate (3-lead ECG)

• Renal blood velocity (Doppler ultrasound):
  • Assessed in the distal segment of the right renal artery during the same phase in the respiratory cycle within a subject.
  • The coronal approach was utilized with the participant in the left lateral recumbent position.
  • A 2.5–3.5 MHz pulsed frequency linear-array transducer positioned at the same anatomical location and held at <60° insonation angle.

• Intraoperator CV = 5.0 ± 0.8%

• Renal vascular resistance (mean arterial pressure / renal blood velocity)

Results

Conclusions

• These data indicate that increases in RVR evoked by the CPT are attenuated during passive heat stress.

• Thus, passive heat stress blunts renal vascular responsiveness to sympathetic activation.

• Although unlikely based on renal angiography data, it remains unclear if these observations are confounded by changes in renal artery diameter at the measurement site.

• Notably, if the renal artery was vasoconstricted, the current findings would likely be underestimating the attenuated renal vasculature responsiveness to sympathetic stimulation during heat stress.