INSUFFICIENT CUTANEOUS VASOCONSTRICTION LEADING UP TO AND AT THE ONSET OF SYNCOPAL SYMPTOMS IN THE HEAT STRESSED HUMAN

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Background- Neural control of cutaneous vascular conductance (CVC) is critical for the maintenance of blood pressure during an orthostatic challenge in the heat stressed human. This project tested the hypothesis that under such conditions inadequate cutaneous vasoconstriction contributes to heat stress-induced orthostatic intolerance. Procedure- Data were analyzed from 39 heat stress orthostatic challenges (from 30 subjects) in which the subject experienced syncopal symptoms resulting in test termination. Each subject was instrumented for the measurement of internal temperature (telemetry pill or pulmonary artery catheter), forearm skin blood flow (laser-Doppler flowmetry), arterial blood pressure (Finometer or brachial artery catheterization), and heart rate. CVC was calculated as skin blood flow/mean arterial blood pressure*100. While heat stressed (average increase of internal temperature: 1.2±0.3 °C, mean±SD; range: 0.69 to 1.97 °C), subjects were exposed to lower-body negative pressure (n=34) or 70° upright tilt (n=5). Data were averaged from the period while subjects were normothermic, while heat stressed immediately prior to the orthostatic challenge, and at 5 sec increments for the 2 min period preceding syncopal symptoms and subsequent cessation of the orthostatic challenge. Results- Whole-body heat stress significantly increased heart rate (57±10 to 88±16 bpm) and CVC (30±20 to 156±54 CVC units) without altering mean arterial blood pressure (83±7 to 82±6 mmHg). Immediately prior to termination of the orthostatic challenge due to syncopal symptoms, mean arterial pressure was reduced to 56±9 mmHg (P<0.001). Although at test termination CVC was significantly decreased to 136±55 units relative to before the orthostatic challenge, this value was ~4 fold greater than CVC when subjects were normothermic (30±20 CVC units; P<0.001). Conclusions- Minimal reductions in CVC during an orthostatic challenge, accompanied with profound hypotension, suggest that insufficient cutaneous vasoconstriction contributes to reduced orthostatic tolerance in heat stressed humans. Project supported by NIH HL61388 and HL84072.