

Inter-individual Differences in Tolerance to a Simulated Hemorrhage Challenge during Heat Stress: Cerebrovascular Control

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ABSTRACT

A high degree of inter-individual variability exists in heat stress (HS)-induced reductions in orthostatic tolerance relative to normothermia (NT), which may be associated with HS-mediated reductions in cerebral perfusion, and thus mechanisms of cerebrovascular control during hypotensive challenges. This study tested two hypotheses; 1) the magnitude of improvement in cerebral autoregulation (CA) would be negatively correlated with the difference in tolerance to graded lower body negative pressure (LBNP) [assessed with a cumulative stress index (CSI)] during HS relative to NT (CSI_{diff}), and 2) cerebrovascular sensitivity to HS-induced hypocapnia would be positively correlated with CSI_{diff} . Subjects ($N=13$) were exposed to LBNP on two occasions (NT and HS) separated by $>72h$ to assess CSI. On a third day, indices of CA were assessed during NT and HS by spectral and transfer function analyses, and cerebrovascular sensitivity to changes in Pa_{CO_2} was determined during NT, HS, and HS+LBNP (-20 mm Hg; HS_{LBNP}). Estimates of CA were improved during HS compared to NT ($P<0.05$); however, there was no relationship between the change in any index of CA from NT to HS and CSI_{diff} ($P>0.05$). Hyperventilation-induced hypocapnia reduced cerebral vascular conductance (CVCi) during HS and HS_{LBNP} relative to NT ($P<0.01$ for both), but no relationship existed between $\Delta CVCi/torr$ in any condition and CSI_{diff} ($P>0.05$ for all). In summary, HS augments mechanisms of cerebrovascular control to protect against orthostatic challenges; however, individual differences in these responses do not predict tolerance to a simulated hemorrhage when internal temperature is elevated.