

Effect of Acute Heat Exposure on the Pressor Response to a Voluntary Hypoxic Apnea: A Cross-tolerance Study

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Category: Doctoral

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ABSTRACT

The pressor response induced by a voluntary hypoxic apnea is exaggerated in individuals with obstructive sleep apnea and is strongly correlated to sympathetic overactivity. Acute heat exposure alters neural control of blood pressure, but its effect on the pressor response to a voluntary hypoxic apnea has never been explored. **PURPOSE:** To test the hypothesis that acute heat exposure attenuates the pressor response to a voluntary hypoxic apnea, and thereby manifest as a form of physiological cross-tolerance. **METHODS:** Eight adults (3 females, 26 ± 2 yrs) were exposed to passive heat stress (water perfused suit) sufficient to increase body core temperature by 1.2°C . Voluntary hypoxic apneas were performed in duplicate before acute heat exposure (pre-heat) and in recovery when body core temperature returned to $\leq 0.3^\circ\text{C}$ of baseline. Participants breathed gas mixtures of varying FiO_2 (21%, 16%, and 12%; randomized) for 1 min followed immediately by a 15 s end-expiratory apnea. Beat-by-beat arterial blood pressure (Finometer) and arterial oxygen saturation (finger pulse oximetry) were measured throughout. The pressor response was calculated as the difference between baseline mean arterial pressure and the peak response following each apnea. **RESULTS:** The change in arterial oxygen saturation during each apnea did not differ from pre-heat to recovery (FiO_2 21%, pre-heat $0 \pm 1\%$ vs. recovery $0 \pm 2\%$; FiO_2 16%, pre-heat $-4 \pm 1\%$ vs. recovery $-4 \pm 2\%$; FiO_2 12%, pre-heat $-8 \pm 3\%$ vs. recovery $-10 \pm 4\%$; $P = 0.3$ for interaction). The pressor response to a voluntary apnea was attenuated in recovery from acute heat exposure across all concentrations of FiO_2 (FiO_2 21%, pre-heat 19 ± 8 mmHg vs. recovery 16 ± 8 mmHg; FiO_2 16%, pre-heat 27 ± 8 mmHg vs. recovery 20 ± 8 mmHg; FiO_2 12%, pre-heat 33 ± 11 mmHg vs. recovery 27 ± 13 mmHg; $P = 0.02$ for main effect of time). **CONCLUSION:** These data suggest that acute heat exposure induces a cross-tolerance effect such that the pressor response to a voluntary hypoxic apnea is reduced. Acute heat exposure could improve hypertension in adults with obstructive sleep apnea, secondary to altered chemoreflex function and sympathetic neural control, and provide additional therapeutic options for this population to improve cardiovascular health.