Where applicable, the authors confirm that the experiments described here conform with The Physiological Society ethical requirements.

PCA267

Cardiovascular responsiveness to sympathoexcitatory stress in mild-hypertensive and normotensive participants

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Autonomic dysfunction is implicated in cardiovascular disease, with studies reporting an exaggerated blood pressure response to sympathoexcitatory stress in individuals with resting hypertension (1-3). It is not clear whether this response is associated with changes in cardiac autonomic modulation or altered peripheral cardiovascular dynamics. The aim of this study was to compare blood pressure (BP), heart rate variability (HRV) and limb blood flow between normotensive (N: mean±SD; BP 116.9±1.4/73.4±0.9mmHg; age 49.4±10.4years, BMI 26.4±4.5kg.m-2; n=49) and mildly hypertensive participants (H: BP 141.3±2.0/88.8±2.2mmHg; age 51.7±11.4years, BMI 30.5±5.0kg.m-2; n=17) at rest and in response to sympathoexcitatory stress. Participants performed a cold pressor test (CPT) and an ischaemic handgrip test (IHGT) and were assessed for BP, forearm blood flow and HRV at rest and in response to the tests. Data are expressed as mean±SD and comparisons were made using ANOVA (SPSS Version 19, IBM Statistics, SPSS Inc., USA). The CPT evoked greater increases in systolic blood pressure (SBP; H: 18.13±13.01mmHg; N: 8.84±12.38mmHg; p=0.011) and mean arterial pressure (MAP; H: 10.13±9.55mmHg; N 4.65±8.16mmHg; p=0.026) in hypertensive compared with normotensive participants. The IHGT evoked greater increases in diastolic (DBP; H: 6.88±5.48mmHg; N: 0.32±7.20mmHg; p=0.001) and MAP (H: 9.24±4.85mmHg, N: 3.26±6.24mmHg; p=0.001) in hypertensive compared with normotensive participants. Hypertensive participants had significantly lower levels of resting cardiac parasympathetic modulations measured as the high frequency power of HRV (H: 31.7±4.1nu; N: 42.1±2.2nu; p=0.026). There were no significant differences in the HRV or blood flow responses to the CPT or IHGT between hypertensive and normotensive participants. This study demonstrated that sympathoexcitatory stress triggered an augmented blood pressure response in hypertensive participants, in line with previous studies (1-3). However, we found no evidence to support stress-activated hyper-reactivity in HRV or forearm blood flow in hypertensive participants. These findings lend support to the notion that sympathetic dominance contributes to the pathogenesis of hypertension. From the findings, we propose that more direct markers of autonomic function be investigated to better understand the role of the autonomic nervous system on the stress induced blood pressure response.

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This project was funded through a University of the Sunshine Coast research development grant.

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PCA268

A novel integrated tilt table-lower body negative pressure box to investigate differential arterial baroreflex responses with tilt in humans

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The arterial baroreflex responds to acute reductions in mean arterial pressure (MAP) by eliciting a heart rate response, maintaining MAP at normal levels. In the case of traumatic blood loss, Trendelenburg position (i.e., head-down tilt) is often used clinically to help compensate for reductions in MAP through gravity-dependent shifts in blood volume distribution toward the central cavity, but its use is controversial. A lower-body negative pressure (LBNP) box can be used to study baroreflexes by eliciting acute reductions in MAP through shifts in blood volume distribution toward the lower extremities, reducing venous return. We developed a novel integrated tilt table-LBNP box to investigate the cardiovascular and cerebrovascular effects of head-up and head-down tilt (HUT, HDT) on LBNP-induced hypotension. We hypothesized that 45° HUT would elicit a larger arterial baroreflex heart rate response to acute -50 mm Hg LBNP than supine or 45° HDT. Additionally, we hypothesized that there would be differential cerebral autoregulation (CA) between the middle cerebral artery (MCA) and posterior cerebral artery (PCA) during acute LBNP. 13 male volunteers (BMI 26.4±3 kg/m2; 24.2±4.8 yrs) were recruited. Following familiarization and consent, subjects were instrumented for measurement of instantaneous heart rate (IHR; ECG), MAP (mm Hg; finometer), end-tidal (PET)CO2 (Torr) and MCA and PCA cerebral blood velocity (CBV; cm/s; transcranial Doppler ultrasound). Subjects were placed in the tilt-table-LBNP box in supine position, underwent a 10-min baseline period, and exposed to -50 mmHg of acute LBNP for a maximum of ten minutes. The protocol was then repeated in 45° HUT and HDT in randomized order. Subjects were coached to maintain PETCO2 to resting values (±2 Torr). LBNP was terminated if systolic blood pressure was reduced 30% from resting values (pre-syncope; PS). We found that the arterial baroreflex-mediated IHR responses were linear and tilt-dependent. The slopes of the IHR responses were largest in 45° HUT, but less effective at protecting MAP than in supine and 45° HDT during acute LBNP. 10/13 subjects in 45° HUT reached PS in 427.7s, 3/13 subjects in supine position reached PS in 450.3s and no subjects reached PS in 45° HDT. PETCO2 was unchanged during LBNP in all three positions with coached breathing, eliminating the confounding effects of cerebrovascular CO2 reactivity on CBV. Although MAP was unchanged during LBNP prior to PS in all positions, there were differences between baseline and LBNP MCA in 45° HDT and 45° HUT, but there were no differences in the PCA between baseline and LBNP in any body position. Our findings suggest (a) Trendelenburg may be a useful application to prevent hypovolemic shock, and (b) CA