The stroke volume response to passive leg raise predicts tolerance to hypovolemia induced by lower body negative pressure

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Introduction: The passive leg raise test (PLR) can create a transient increase in cardiac preload, and has been used to predict fluid responsiveness defined as a clear increase in stroke volume (SV) following a fluid bolus1,2. The lower body negative pressure (LBNP) model has been widely used to create a reversible hypovolemic state in awake volunteers by sequestering blood into the lower extremities3. In some subjects, LBNP can elicit symptoms consistent with significant hemorrhage. The present study tested the hypothesis that the SV response to PLR can predict tolerance to LBNP.

Methods: Data acquired under an IRB-approved protocol from 13 healthy volunteers aged 25 to 41 (10 male, 3 female) were used for the study. Subjects first underwent PLR at 45° angle for 2 min in the supine position prior to LBNP. After lowering the legs and allowing a period of stabilization, progressive LBNP was applied at -15, -30, -45, -60, -75, and -85 mmHg for 3 min at each stage. Subjects were noninvasively monitored for beat-to-beat arterial blood pressure (finger probe), SV at 8 second intervals (chest electrodes), and heart rate (HR). Cardiac output (CO) was calculated as HR x SV. Variables were averaged over 2 min prior to PLR for baseline (BL), during the 2 min of PLR, and with progressive LBNP. Subjects were classified as low tolerance (LT) if they experienced symptoms consistent with marked hypovolemia (lightheadedness, nausea, diaphoresis, shortness of breath), or had a measured systolic blood pressure < 90 mmHg. Subjects without symptoms or hypotension at LBNP -75 mmHg were designated as high tolerance (HT). Unpaired t-test was used to assess the difference in the mean percent change in SV (ΔSV%), HR (ΔHR%), CO (ΔCO%), Systolic blood pressure (ΔSBP%), Diastolic blood pressure (ΔDBP%) and Mean arterial pressure (ΔMAP%) in response to PLR between the HT and LT groups. To determine what, if any, level of SV change in response to PLR could predict tolerance to LBNP, a Receiver Operator Curve (ROC) was constructed. For all statistical tests, a p-value of < 0.05 regarded as significant.

Results: During LBNP, 7 of 13 subjects were designated LT, with the remaining 6 HT. There was a difference between the HT and LT groups in terms of ΔCO% and ΔSV% during PLR, but no difference in ΔHR%, ΔSBP%, ΔDBP% or ΔMAP% (table 1). ROC analysis of ΔSV% during PLR (figure 1; data with 95% confidence intervals) yielded a cut-off to predict tolerance to LBNP-induced hypovolemia of 12.8% with 100 % sensitivity (59-100%), 83% specificity (36-99%), and an AUC of 0.88 (0.66-1.1), p = 0.02.

Discussion: PLR is reported to provide roughly ~300 ml of auto transfusion4, and elicits compensatory variation in cardiac autonomic tone and biomechanics as preload acutely rises. For the current study, the auto transfusion associated with PLR produced a greater overall change in
SV among the subjects that became symptomatic during LBNP. Although the sample size is small, the data demonstrate that in this population of awake, healthy volunteers, a ΔSV% threshold of ≥12.8% predicts with high sensitivity tolerance to the central hypovolemia induced by the LBNP.

Conclusion: Study results indicate that when SV is continuously measured at 8 sec intervals, the mean ΔSV% over just 2 min of PLR can predict a subsequent symptomatic response to LBNP.