CARDIAC OUTPUT LIMITATIONS DURING EXTREME HEAT AND EXERCISE STRESS IN TRAINED MALES.
D.J. McCann, D.L. Grimes*, and W.C. Adams, Department of Physical Education, Gonzaga University, Department of Exercise Science, University of California, Davis.

This study was designed to determine if a combination of extreme thermal and exercise stress result in an inability to increase cardiac output (CO) in aerobically fit males. Six males served as subjects (average VO$_2$max of 4.10 l/min). Subjects cycled in an upright position at 40, 60 and 80% VO$_2$max for 24 minutes at 36 °C with zero air flow. VO$_2$, cardiac output (CO), heart rate (HR), and mean arterial blood pressure (MAP) were monitored during each bout. CO was determined at four minute intervals utilizing CO$_2$ rebreathing. Time dependent changes (drift) were assessed by linear regression on each subject's response and testing group mean slopes for significance differences from zero using a one way t-test (P<.05). VO$_2$, CO, and MAP were constant over time for all work intensities indicating steady state was achieved. Subsequently, mean values for each subject, as well as a group mean for VO$_2$, CO, and MAP, were tested for significant work rate effects by utilizing repeated measures ANOVA and Scheffe' post hoc tests (P<.05). Group mean steady state MAP was significantly higher at each increased work intensity (118 ± 1.48, 127 ± 2.74, and 133 ± 1.66 mmHg, respectively), while CO was higher at 60 than at 40 but similar at the 60 and 80% work rates (14.1 ± 0.89, 17.3 ± 0.35, and 18.4 ± 0.56 L/min for the 40, 60 and 80% VO$_2$max, respectively). HR increased significantly during each exercise bout while stroke volume decreased significantly during the 60 and 80% work rates. In conclusion, the lack of an increase in CO at the 80% work rate is hypothesized to be due to insufficient venous return from the peripheral circulation. However, the increase in MAP at the 80% work rate suggests a blood volume shift from the venous to the arterial circulation may have preceeded the insufficient venous return. Thus, venous return may have been compromised by an inability to decrease venous compliance in response to decreased venous volume rather than by a thermoregulatory induced increase in venous compliance commonly referred to as peripheral blood pooling.