**ESSR Journal Club**

**Covered Article:** “Effect of Thermal Stress on Cardiac Function” by Thad E. Wilson and Craig G. Crandall  
**Journal Club Author:** Scott Montain

1. To study the effect of heat stress on cardiac function, the authors impose passive heat stress via a water perfused suit to manipulate body temperature. In their experimental paradigm, heat stress produces a large increase in cardiac output and heart rate, and no change or a modest increase in stroke volume. How do these physiological responses compare to the imposition of heat stress on an individual performing upright exercise?

2. The authors have found that passive heat stress produces a leftward shift in the Frank-Starling relation. How might this be advantageous for withstanding an orthostatic challenge?

3. By what signaling mechanism(s) do you think heat stress is altering cardiac function? What is the sensed variable?

4. The authors propose that changes in cardiac output and function mechanistically contribute to decreases in orthostatic tolerance during heat stress and to improvements in orthostatic tolerance during cold stress. How might this idea be experimentally tested?

5. Aerobic exercise training increases in blood volume, end-diastolic volume, and stroke volume. Would this be of advantage for withstanding heat stress and orthostatic challenge? Why?

6. Dehydration affects blood volume and cardiac filling. What would you predict would happen in regards to the Frank-Starling relation in response to heat stress and diuretic administration?

7. The authors used a supine passive heat stress model to study the effects of heat stress on cardiac function. What would you predict would happen if the experiments had been performed on individuals in an upright seated posture?