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Independent Effect of Heat Stress During Exercise on Arterial Stiffness

Cash Arcement
University of Arkansas

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Cash H. Arcement

University of Arkansas

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ABSTRACT

Context: Cardiovascular disease (CVD) is one of the leading causes of mortality in the United States, accounting for about 1 in every 3 deaths annually. While studies have shown that arterial stiffness, a leading precursor to CVD, improves with passive heat stress not much is known about the independent effect of heat stress during exercise on arterial stiffness. **Objective:** The objective of this study was to examine the independent effect of heat stress during exercise on arterial stiffness. **Design:** Participants visited the lab three times; one familiarization and two experimental trials. Experimental trials were randomized and counter-balanced. **Setting:** All trials occurred in the Human Performance Laboratory at the University of Arkansas, Fayetteville. The experimental trials consisted of subjects cycling at ~50% of their maximum aerobic capacity in an environment of 40°C / 40% relative humidity (Heat Cycle) or 15°C / 30% relative humidity (Cool Cycle). **Participants:** Participants included five male subjects and four female subjects. Subjects were older individuals (Age = 49 ± 12 y, Body Mass = 66.71 ± 12.64 kg) with stiffer arteries at baseline (> 6 m/s) identified in the familiarization trial. **Interventions:** The intervention was environmental condition; one trial occurred at 40°C / 40% relative humidity (Heat Cycle) and the other at 15°C / 30% relative humidity (Cool Cycle). Since the participants are exercising in both trials at the same aerobic capacity, we can elucidate the independent effect that mean body temperature (i.e., heat stress) had on arterial stiffness during exercise. **Main Outcome Measures:** Before and after cycling, pulse wave velocity (PWV measures occurred via ultrasound at the tibial, radial, femoral and carotid artery sites) were used to assess arterial stiffness. Specifically, central arterial stiffness was assessed by using measures between the carotid and femoral artery sites, while peripheral stiffness was assessed using the radial and tibial artery sites. At the same time, mean body temperature (T_{body}) was measured via skin and rectal thermistors. **Results:** T_{body} at the end of exercise showed significant differences between the heat cycle and cool cycle trials respectively (36.47 ± 0.23 vs. 34.84 ± 0.40°C). There were no interactions between time and condition for central PWV for Heat Cycle and Cool Cycle respectively (100.02 ± 138.64 vs. 24.19 ± 82.40 cm/s, $p = 0.38$), upper peripheral PWV (47.76 ± 131.35 vs. 64.41 ± 109.99 cm/s, $p = 0.56$) and lower peripheral PWV (40.77 ± 142.96 vs. 3.77 ± 167.67 cm/s, $p = 0.47$). **Conclusions:** The findings of this study suggest that differences in mean body temperature do not result in significant differences in arterial stiffness following exercise.

INTRODUCTION

Cardiovascular (CV) disease is one of the leading causes of mortality in the United States. With roughly 600,000 deaths from CV disease annually, about 1 in every 3 deaths in the United States is the result of poor cardiovascular health (Go, et al., 2013). The most predominant and costly form of cardiovascular disease is hypertension, which affects 78 million adults in the United States. Hypertension is often preceded by increases in arterial stiffness (O'Rourke, 1990). Arterial stiffness has been shown to be a strong indicator of arterial health that is stronger than looking at blood pressure alone (Laurent, et al. 2006; Duprez & Cohn, 2007). Poor arterial health and thus stiffer arteries are often observed in middle-to-older-aged individuals. It is pertinent to develop ways to combat arterial stiffness so that we may slow down the effects of cardiovascular disease. One well-known way to decrease arterial stiffness, and thus improve arterial health, is to exercise.

The mechanism by which exercise improves arterial health is complicated. Briefly, exercise increases blood flow throughout your peripheral arteries to ensure that nutrients reach the exercising muscles. When more blood is moving through blood vessels it increases shear stress on those arteries. Shear stress is created by the flow of blood on the arterial wall. This stimulates the release of nitric oxide into the bloodstream. Nitric oxide is a free radical that stimulates vasodilation, thus furthering the blood flow throughout the body. This increased vasodilation and blood flow helps promote arterial health as the artery "adapts" in a healthy way.

Acute exercise brings about a host of physiological changes, so it is difficult to know which changes are affecting arterial stiffness. For example, it is well known that moderate-intensity exercise can increase body temperature as much as 1.0°C in as little as 30 minutes (Saltin & Hermansen, 1966; Gregson, Drust, Batterham, & Cable, 2002). The body's metabolic rate (i.e., heat production) increases with exercise, which in turn causes an increase in core body

temperature. It has been shown that independent increases in the body's core temperature (from passive heat stress) causes acute decreases in arterial stiffness (Ganio, Brothers, Shibata, Hastings, & Crandall, 2011). Therefore, it is possible that part of the acute improvements in arterial stiffness (increased arterial stiffness observed with exercise are due to the elevated internal body temperature.

While it has been shown that exercise and the heat stress that comes from exercise brings about a decrease in arterial stiffness, the independent effect of heat stress on changes on arterial stiffness during exercise is still relatively unknown. If heat stress decreases arterial stiffness with no exercise (Ganio et al., 2011), it is possible that when you combine the two (Heat + Exercise) that there will be a cumulative effect on arterial stiffness, thus decreasing arterial stiffness further than with either exercise or heat alone. This project was designed to shed light on whether there is a combined effect (exercise + heat = combined reductions in arterial stiffness) so that we can elucidate how to combat arterial stiffness clinically in the most effective way. In order to control for the independent effect of heat stress, we had two trials, one in which core temperature does not go up ("Cool Cycle" trial) and one in which core temperature goes up ("Heat Cycle" trial). Therefore, the purpose of this study was to examine if there is an additive reduction in arterial stiffness from the combined effects of exercise and an elevated core body temperature versus exercise alone. We hypothesized that if the body is subjected to both heat and exercise stress, then there would be greater reductions (i.e., improvements) in arterial stiffness compared to the cool condition. The measurement of the dependent variables before and after each perturbation tested our hypothesis that heat stress has an independent effect on arterial stiffness during acute activity (i.e., exercise).

METHODS

Participants in this study reported to the Human Performance Laboratory for a total of three visits:

1. A familiarization and arterial stiffness screening

2. Exercise + Heat trial (Heat Cycle)

3. Exercise + No Heat trial (Cool Cycle)

Participants of this study were 5 healthy men and 4 healthy women (Age = 49 ± 12 y, height = 168.22 ± 8.79 cm, body mass = 66.71 ± 12.64 kg). Participants of this study were excluded if they were on anti-hypertensive medication, had any signs or symptoms of cardiovascular disease, or if they had a baseline pulse wave velocity measure below 6 m/s. By setting a cutoff for arterial stiffness, we targeted a population with stiffer arteries at baseline. The study was designed this way using information gained by (Ganio et al., 2011) that indicated changes in stiffness were dependent on how stiff the participant's arteries were at baseline. This study was approved by the University of Arkansas's Institutional Review Board.

Upon arrival at the familiarization trial, participants were walked through what was expected from them in each trial. An informed consent form was thoroughly explained to each participant before any measurements were taken. After the informed consent were signed by the participants, a medical history and physical activity questionnaire were completed to ensure that the trial could be safely completed with each participant.

Height was obtained by a stadiometer and a nude body mass was obtained. Body composition was assessed by dual x-ray absorptiometry (DXA). Each participant was placed in a supine position for 15 minutes before pulse wave velocity measures. All pulse wave velocity measures were taken using a Doppler ultrasound (see below). Baseline arterial stiffness measures

were taken and only participants who met our cutoff (greater than 6 m/s) were included in the study. Each participant then completed a cycle protocol to elucidate their maximum aerobic capacity (VO$_2$ Max). The cycle protocol was completed using an electronically braked cycle ergometer (Racemate Veletron, Seattle, Washington) with nose clips attached while breathing in room air and exhaling into a mouthpiece connected to a metabolic cart (Parvo Medics' TrueOne® 2400, Sandy, Utah). Exercise started at ~ 50 Watts (W) and increased 25W every 2 minutes until exhaustion. Heart rate and ratings of perceived exertion (RPE) were taken every 2 minutes and at exhaustion.

Upon arrival and instrumentation at the experimental trials, subjects laid quietly in a supine position for 15 minutes. After this rest, baseline pulse wave velocity measures were obtained. After baseline measures were taken, subjects entered the environmental chamber for their perturbation. The environmental chamber was set to 40°C, 40% relative humidity for the Heat Cycle trial, and 15°C, 30% relative humidity for the Cool Cycle trial. After entering the chamber for each exercise trial, the subject exercised at a moderate intensity by cycling for 30 minutes (≈ 50% VO$_2$ Max). During each trial, VO$_2$ was assessed during the first 5 minutes of exercise and during the 25th minute of exercise. Because posture is important for measures of arterial stiffness and limb movement precludes measurements during exercise, all measures occurred with subjects lying down. All post-exercise measures occurred in a thermo-neutral site (i.e., outside of the environmental chamber). Measurements were taken in this environment to ensure that the associated changes in pulse wave velocity were a result of the exercise bout and not changes that could occur during recovery in a specific environmental condition.

Recognizing that changes in cardiovascular measures (e.g., heart rate, blood pressure) may be changing after exercise, HR and blood pressure measures were also obtained 15, 30, 45, and 60

minutes after the end of each experimental perturbation, when stabilization has occurred (McEniery, Wilkinsons, & Avolio, 2007; Pickering, 2005).

Core temperature was measured from a rectal thermistor. Before exercising, each participant was asked to insert the rectal thermistor ~16cm beyond their anal sphincter. This method is the safest, most valid way to measure core body temperature (Saltin & Hermansen, 1966). Mean skin temperature was measured via the weighted average of four thermocouples attached to the subject's lateral calf, thigh, chest and deltoid. Mean body temperature (T_{body}) was calculated by using the Burton Formula (0.64*rectal temperature + 0.36*mean skin temperature) (Lenhardt & Sessler, 2006).

Heart rate was measured with a Polar Heart Rate Monitor and 3-lead ECG. Mean arterial blood pressure was measured by auscultation of the brachial artery via R-wave gated K sound electrosphygmomanometry (SunTech, Raleigh, NC).

Perceptual ratings were taken throughout each exercise trial. Levels of RPE, thirst, pain in the thighs, and thermal sensation were all taken before exercise and at every 5 minutes during their exercise bout. Thermal sensation was also taken every 15 minutes throughout the post exercise rest.

Arterial stiffness measurement and data analysis occurred using standard procedures as done previously (Ganio et al., 2011). Briefly, arterial stiffness was measured by pulse wave velocity (PWV). Although there are several methods to measure arterial stiffness, PWV is the preferred method (Kukkonen-Harjula & Kauppinen, 2006; Rowell, Brenglmann, & Murray, 1969). PWV is the measure of the speed with which the pressure waveform propagates along a segment of the arterial tree; thus the stiffer the vessel, the faster the wave travels. Pulse wave velocity was measured with Doppler ultrasound (GE GoldSeal LOGIQ e BT08) and calculated as

the distance between measurement sites divided by the time delay between the two waveforms (Kukkonen-Harjula & Kauppinen, 2006).

We took pulse wave velocity measures at four locations at the body: radial, carotid, femoral, and tibialis anterior. Central pulse wave velocity was calculated from analyzing data from the carotid and femoral sites, while peripheral pulse wave velocity was calculated using data from carotid, radial, and tibialis anterior arteries using the foot-to-foot method (Kukkonen-Harjula & Kauppinen, 2006). Measures of central pulse wave velocity were calculated using the femoral and carotid arterial sites. Measure of upper peripheral pulse wave velocity were calculated using measures from the carotid and radial arterial sites, while lower peripheral pulse wave velocity measures were calculated by using the femoral and tibialis anterior sites. All pulse wave velocity measures were performed on the left side of the body with consistent probe location being assured by marking the skin with a surgical marker. Distance between arterial measurement sites was calculated by subtracting the distance from the carotid location to the sternal notch from the distance between the sternal notch and the femoral with the radial, and tibialis anterior site (Kukkonen-Harjula & Kauppinen, 2006). Our coefficients of variation for measures of PWV are ≤ 3 % (Ganio et al., 2011).

RESULTS

There was an interaction between time and condition for mean body temperature ($p < 0.05$) Pairwise comparisons revealed significant differences between the Heat Cycle and Cool Cycle trials at immediate post, 15 minutes after exercise, 30 minutes after exercise, 45 minutes after exercise, and 60 minutes after exercise. No significant differences were found at baseline. These results can be seen below (Figure 1).

There was an interaction between time and condition for rectal temperature ($p < 0.05$). Pairwise comparisons revealed significant differences between the Heat Cycle and Cool Cycle trials at 15 minutes after exercise, 30 minutes after exercise , 45 minutes after exercise and 60 minutes after exercise. No significant differences were present at baseline or immediately post exercise. These results can be seen below (Figure 2).

There was an interaction between time and condition for mean skin temperature ($p < 0.05$). Pairwise comparisons revealed significant differences between the Heat Cycle and Cool Cycle trials at the following time points: baseline, immediate post exercise, 15 minutes post exercise, 30 minutes post exercise, 45 minutes post exercise, and 60 minutes post exercise. Differences between the baseline mean skin temperatures were minimal (~0.30°C). These values can be seen below (Figure 3).

There was no interaction or main effect between time and condition for mean arterial blood pressure ($p > 0.05$). There was also no interaction between time and condition for heart rate ($p > 0.05$). There was, however, a main effect of both time and condition for heart rate ($p < 0.05$). These conditions are listed below in Figure 4 and Figure 5, respectively.

There was no interaction between time and condition for VO_2 during the trials ($p > 0.05$). There was, however, a main effect of time on VO_2 ($p < 0.05$). The average 25th minute VO_2 during the Heat Cycle and Cool Cycle trials were 18.34 and 18.59 mL/kg/min, respectively ($p > 0.05$).

Further, there was no interaction between time and condition for central pulse wave velocity ($p = 0.38$). There was also no main effect of time or condition on central pulse wave velocity ($p > 0.05$).

There was no interaction between time and condition for upper peripheral pulse wave velocity ($p = 0.56$). There was also no main effect of time or condition on upper peripheral pulse wave velocity (*p > 0.05*).

There was no interaction between time and condition for lower peripheral pulse wave velocity ($p = 0.47$). There was also no main effect of time or condition on lower peripheral pulse wave velocity (*p > 0.05*). Measurements of for central, upper peripheral, and lower peripheral are presented in Figures 6, 7, and 8 respectively.

DISCUSSION

The purpose of this study was to determine the independent effect of heat stress during an acute bout of exercise on arterial stiffness. This study was also unique in that it targeted a population that has stiffer arteries at baseline. This is due to the notion that changes in arterial stiffness with passive heat stress seem to be correlated with having stiffer arteries at baseline (Ganio et. al., 2011). The main finding of this study was that heat stress did not have a significant effect on reducing arterial stiffness in this population. Further, in both the Heat Cycle and Cool Cycle trials, pulse wave velocity did not change in central and peripheral measures immediately following exercise. Physiological levels of arterial stiffness vary from person to person. For the population our study observed, normal physiological levels of pulse wave velocity could range from 6.5 m/s – 10.9 m/s (Reference Values. 2010).

As designed, subjects displayed significantly different mean body temperatures between the two trials, with the Heat Cycle trial having a higher mean body temperature. Rectal temperatures were the same immediate post. However, it was lower in the Cool Cycle trial starting at 15 minutes post exercise. Despite these differences (or lack thereof), there were no differences

in PWV between immediate post and the time points. This was important to the overall study so that while holding exercise constant, we could see the effect that mean body temperature changes (i.e., heat stress) had on arterial stiffness during exercise.

After increasing slightly immediately post exercise, measures of central, upper peripheral, and lower peripheral (PWV) showed values that returned to baseline after 1 hour of supine rest following the acute exercise bout. This could be due to different physiological mechanisms that help the body maintain homeostasis. While pulse wave velocities returned to baseline, mean body temperature did not return to baseline in the Heat Cycle trial. This seems to indicate that an elevated mean body temperature during rest after exercise does not have a significant effect on arterial stiffness.

Acute exercise bouts can have varying effects on arterial stiffness. In a study conducted by (Heffernan, Collier, Kelly, Jae, & Fernhall, 2007), decreases in pulse wave velocity (and thus arterial stiffness) were seen in measures taken 20 minutes after an acute bout of aerobic exercise. In the same study, increases in pulse wave velocity were associated with acute bouts of resistance training. These results indicate that different mechanisms are activated by different modes of exercise (i.e., aerobic vs. resistance). Decreases in arterial stiffness have also been seen with other acute bouts of aerobic exercise. In one particular study, decreases were seen with 30 minutes of cycling at 65% maximum aerobic capacity. (Kingwell, Berry, Cameron, Jennings, & Dart, 1997). This indicates that the mechanisms behind altering arterial stiffness may be altered by varying intensities of aerobic stress.

Further research should continue to investigate how arterial stiffness is affected by physiological responses to temperature stress and exercise. Exercise and heat stress bring about a host of physiological changes, so it is difficult to know which changes are affecting arterial

stiffness. Cardiac output increases with exercise. With this increase in blood flow, shear stress is increased. This increased shear stress can in turn increase the release of the vasodilator nitric oxide. During exercise, levels of NO released could be different between central and peripheral arteries. Sympathetic nervous system activity can also effect arterial tone. It is unclear if during different modes of heat stress or exercise can produce different nervous system responses. That is, we are unsure how the neurons that can effect arterial tone fire in response to different types of stress. It is still unclear, however, what role these mechanisms and others play in the complexity that is arterial stiffness. While our study did not see any significant differences, this alludes to the complexity of arterial stiffness and how it is a multi-faceted question that should be researched further to help prevent patients from developing cardiovascular disease.

LIMITATIONS

One possible limitation of this study was that the foot of the pulse wave was identified visually (versus computer aided) when doing analysis. To account for this, analysis of pulse wave information was done by only the three primary researchers. Once analysis of an individual trial was started it was finished by the same researcher to maintain consistency. Further, in this study an ultrasound was used to measure pulse wave velocity whereas previous research has utilized tonometry. Taking pulse wave measures from ultrasound have been found to be comparable to those taken from tonometry (Jiang, Liu, McNeill, & Chowjenczyk, 2008). Using other methods, such as tonometry by the PulsePen device, may provide researchers a different viewpoint on what is occurring physiologically. The PulsePen is different in that it allows simultaneous measuring at two arterial sites, rather than the one site allowed by the single probed ultrasound (Salvi et al., 2004).

CONCLUSION

The purpose of this study was to determine if heat stress during exercise has an independent effect on arterial stiffness. We found that heat stress during exercise had no significant effect on arterial stiffness. It is known that aerobic exercise improves arterial stiffness, but the exact mechanism behind this is unknown. This study helped to determine what effect the increase in core temperature plays that is associated with an acute bout of exercise. The fluctuations in arterial stiffness are likely due to a complex combination of exercise released metabolites, increase in heart rate during exercise, and the increase in shear stress associated with an increase in cardiac output during exercise.

ACKNOWLEDGEMENTS

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non-invasive portable tonometer for determining arterial pressure wave and pulse wave

velocity: the PulsePen device. Journal of hypertension, 22(12), 2285-2293.
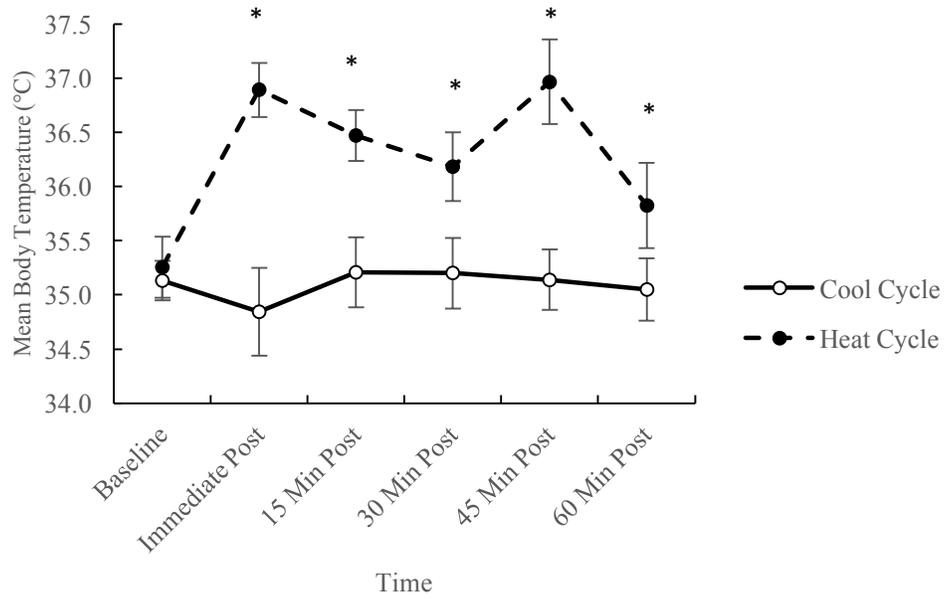
FIGURES

Figure 1. Effect of heat stress during exercise on mean body temperature. Significance, between conditions, are denoted by (*).
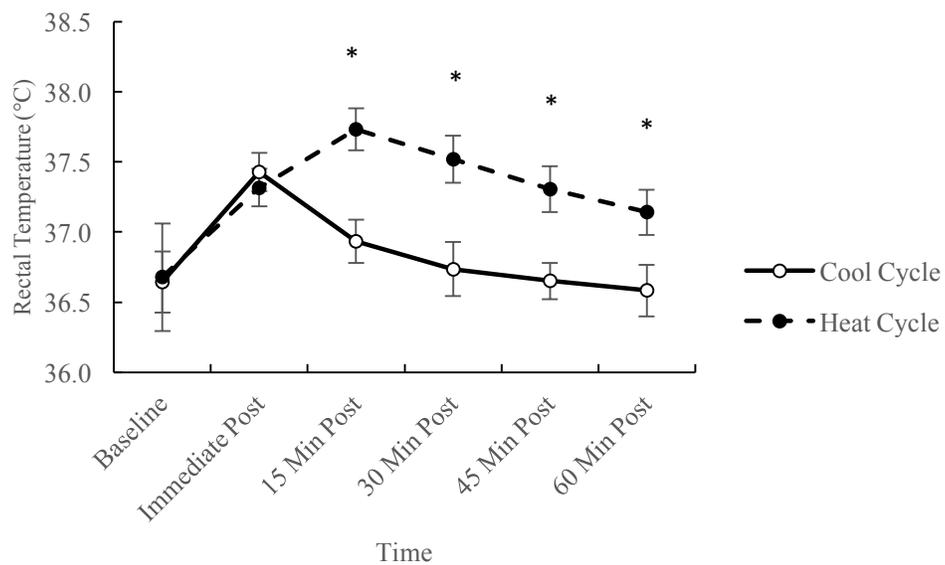
Figure 2. Effect of heat stress during exercise on rectal temperature. Significance, between conditions, are denoted by (*).
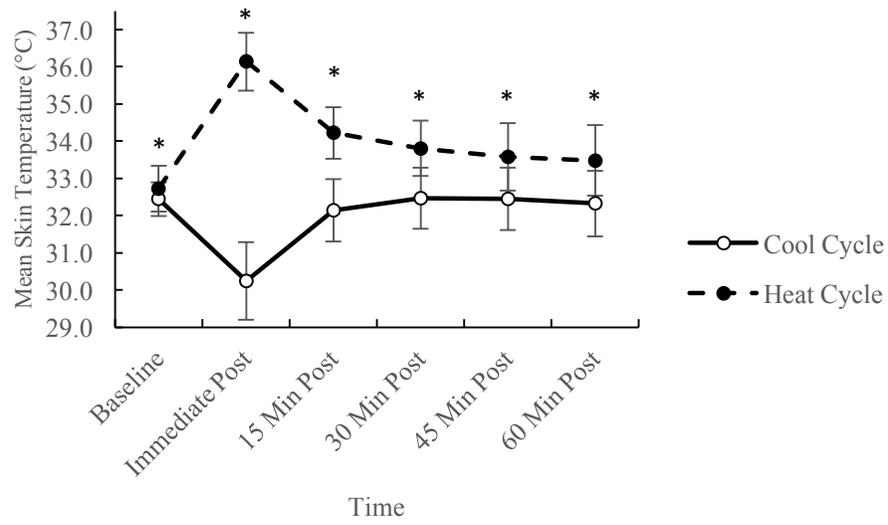
Figure 3. Effect of heat stress during exercise on mean skin temperature. Significance, between conditions, are denoted by (*).
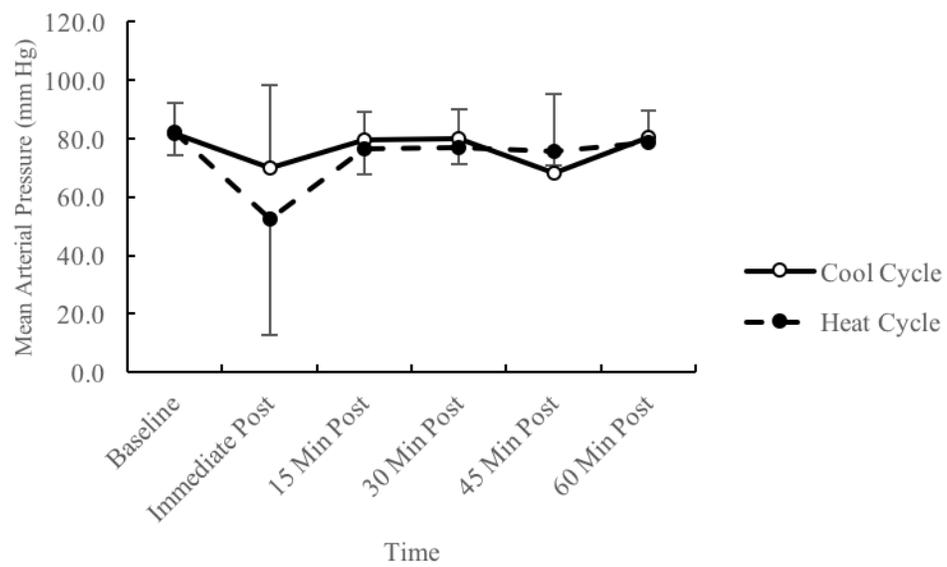
Figure 4. Effect of heat stress during exercise on mean arterial blood pressure.
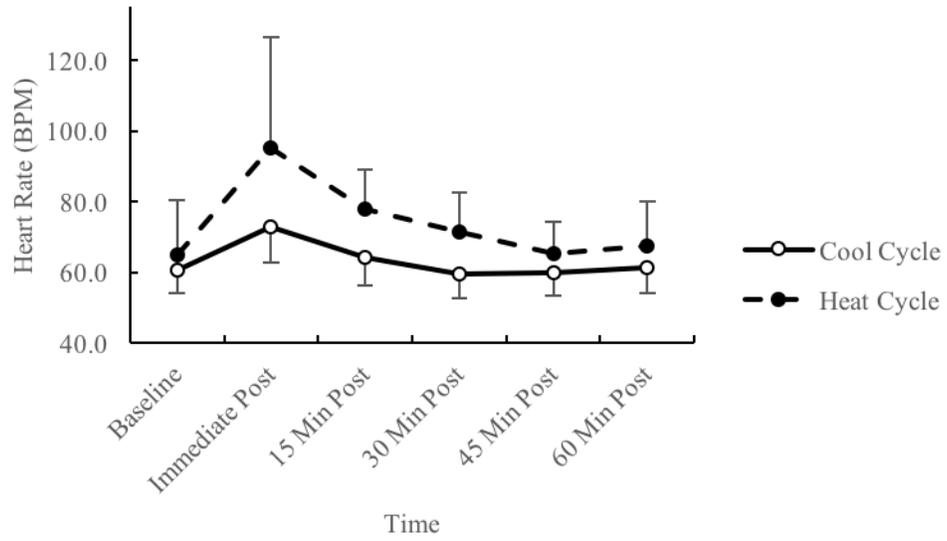
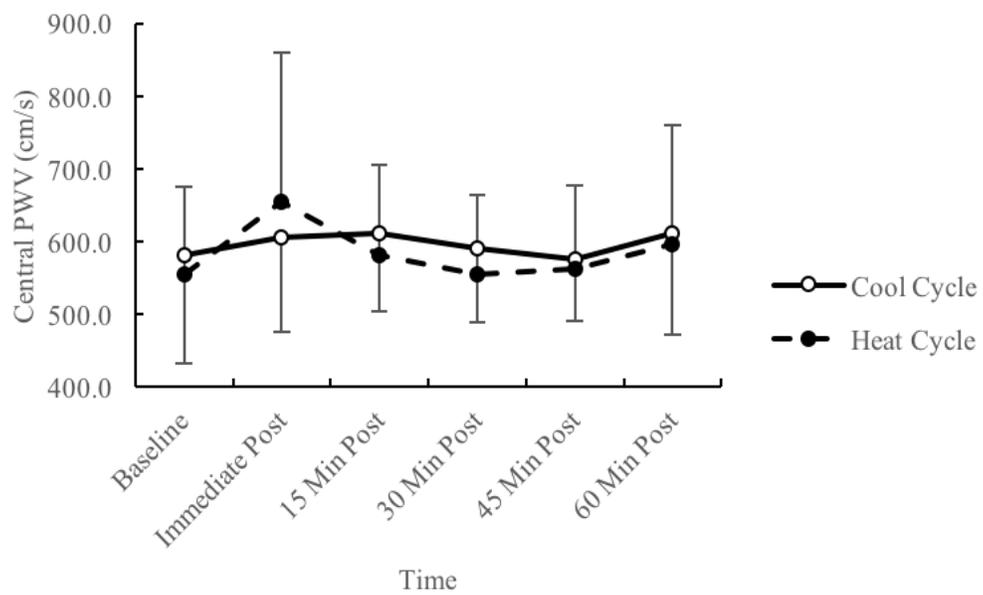Figure 5. Effect of heat stress during exercise on heart rate.

Figure 6. Effect of heat stress during exercise on central pulse wave velocity.

Figure 7. Effect of heat stress during exercise on upper peripheral pulse wave velocity.

Figure 8. Effect of heat stress during exercise on lower peripheral pulse wave velocity.

Figure 1. Effect of heat stress during exercise on mean body temperature. Significance, between conditions, are denoted by (*).

Figure 2. Effect of heat stress during exercise on rectal temperature. Significance, between conditions, are denoted by (*).

Figure 3. Effect of heat stress during exercise on mean skin temperature. Significance, between conditions, are denoted by (*).

Figure 4. Effect of heat stress during exercise on mean arterial blood pressure.

Figure 5. Effect of heat stress during exercise on heart rate.

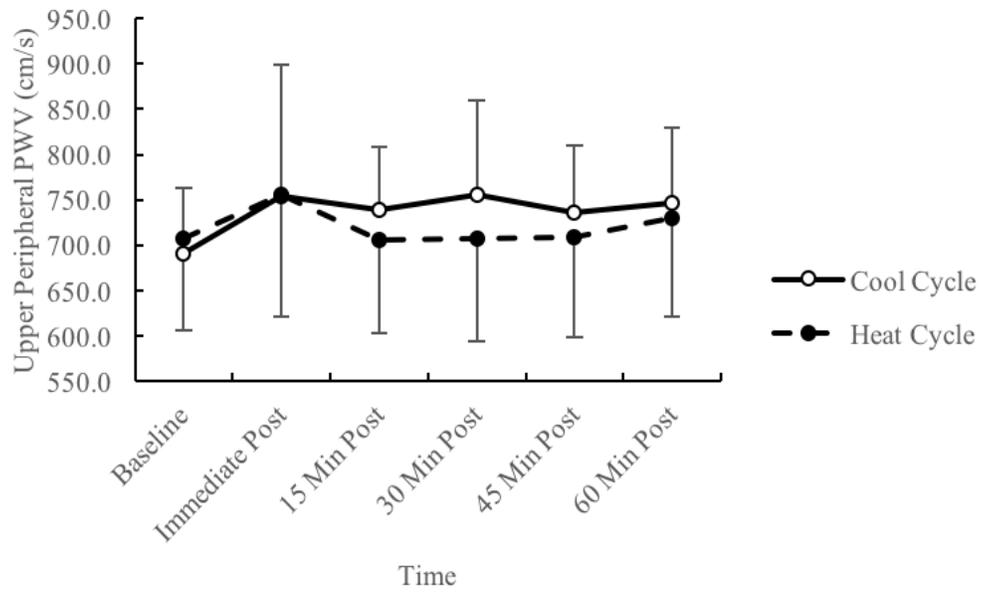*Figure 6. Effect of heat stress during exercise on central pulse wave velocity.*

Figure 7. Effect of heat stress during exercise on upper peripheral pulse wave velocity.
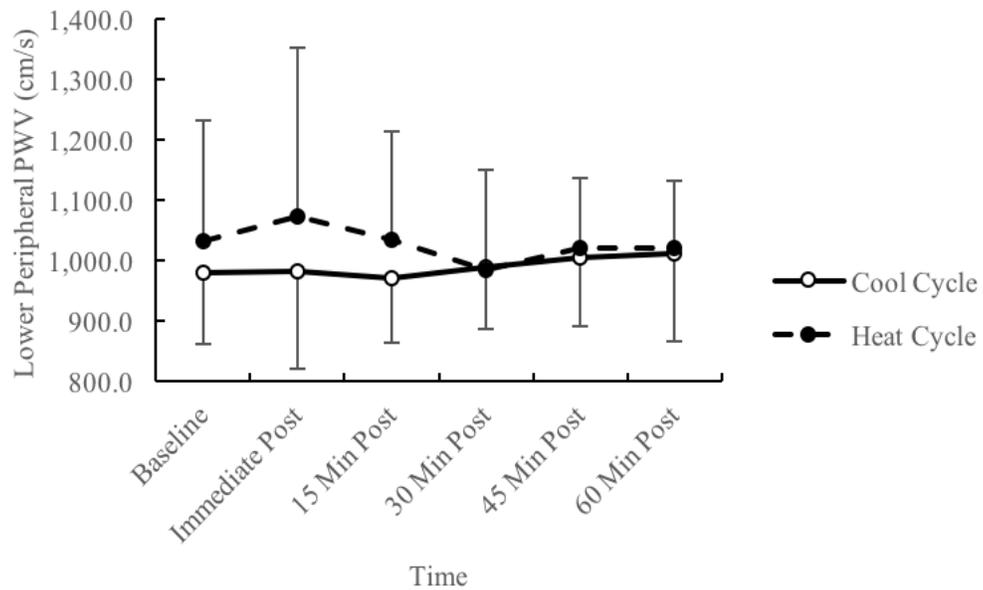
Figure 8. Effect of heat stress during exercise on lower peripheral pulse wave velocity.