Effects of Active Hyperthermia on Upper- and Lower-Extremity Anaerobic Muscular Power

Sean E. Garvey, Michelle A. Cleary, Lindsey E. Eberman, and Zevon Stubblefield
Florida International University, Miami, FL

Objective: We aimed to identify negative implications for performance of anaerobic muscular power elicited by active hyperthermia. **Design and Setting:** The independent variable was thermal condition (normothermic and hyperthermic) elicited by both upper-extremity (UE) and lower-extremity (LE) heat stress trials (HST) designed to elicit a core body temperature (T_b) exceeding 38.0 ^{o}C (mean ambient temperature=34.3 ± 1.4 °C; mean relative humidity=51.5 ± 7.6 %; mean wind speed=1.94 ± 1.11 mph). The dependent variables were UE and LE mean and peak power. **Participants:** Eight adult males (age=24.9 ± 3.2 yr; height=123.0 ± 46.8 cm; body mass=89.9 ± 10.5 kg) participated in a familiarization session and a LE-HST. Only five participants completed the UE-HST. **Measurements:** Upper- body Wingate test was performed prior to and following the LE-HST and a lower body Wingate test was performed prior to and following the UE-HST. **Results:** During the UE-HST, T_b was significantly (t_4=-7.846, p=0.001) increased 2.4% (T_b=$38.2\pm.1$°C) and 5.3% during the LE-HST (T_b=$39.3\pm.3$°C) (t_7=-11.755, $p\leq0.001$). Following the LE-HST, upper-body mean power was significantly (t_7=2.892, p=.023) decreased by 12.22% from the normothermic (4081.13 ± 1175.94 W·kg^{-1}) compared to the hyperthermic (3582.38 ± 863.64 W·kg^{-1}) condition. **Conclusions:** Moderate hyperthermia elicited by LE exercise significantly affected mean power of the upper-body whereas mild hyperthermia elicited by UE exercise demonstrated a trend toward decreased lower-body mean and peak power. LE exercise used larger muscle groups and yielded higher T_b resulting in decreased UE mean power. We attribute our findings to varying levels of hyperthermia, the effects of actively elevating T_b on blood distribution and muscle metabolism, and the differences in the muscle masses of the UE and LE. **Key Words:** heat stress, Wingate, peak power

Hyperthermia is any elevation in core body temperature (T_b). Hyperthermia occurs when the body is unable to dissipate heat quickly enough to maintain the homeostatic temperature of 37 °C.[1] Increases in T_b greater than 40°C can cause damage within the internal organs, most notably the brain and liver potentially leading to death.[1,2] More common are the hyperthermic side effects that disrupt normal function and impact performance. Explosive power is the ability to make a forceful contraction of muscles to produce a powerful movement over a short duration of time and is necessary for successful athletic performance.[3,4] Investigators have suggested that hyperthermia adversely affects muscular performance[5,6] but few investigators have scrutinized the effects of hyperthermia on explosive power.

Explosive power is the ability to produce a maximal muscular contraction over a short period of time and is a measured by the amount of work produced in a certain amount of time. As explosive power is the combination of strength and quickness, it is required in athletic performance. Recent literature established a tendency of researchers to demonstrate the effects of passively induced hyperthermia on explosive power,[5] but have yet to establish the impact of active hyperthermia. A hyperthermic condition maybe created in the laboratory setting either

passively or actively. Passive hyperthermia involves individuals sitting passively in a sauna for a determined amount of time until the T_b increases to a hypertherimic level.[5] Research has demonstrated that muscular endurance and strength has decreased with passive hyperthermia while explosive power increases.[5] Passive localized hyperthermia is achieved through submerging a limb or limbs in hot water to increase the temperature of the tissues only within that limb of limbs, therefore preventing a rise in temperature of the torso and brain. Although passive hyperthermia, both localized and whole body are beneficial in increasing core body temperature, the methods do not simulate sport specific activity and may lack generalizability within athletics. We therefore suggest that inducing hyperthermia actively is the most appropriate method for generalizing laboratory findings with athletics and as such, the purpose of this investigation was to reveal the effects of active hyperthermia on muscular power.

Methods

Research Design

A test-retest design with two experimental conditions and two within-subjects variables were utilized for this investigation. The independent variables were thermal condition (normothermic and hyperthermic) and extremity exercised (upper-extremity (UE) and lower-extremity (LE)) during a heat stress trial (HST) (mean ambient temperature=34.3 ± 1.4 °C; mean relative humidity=$51.5 \pm 7.6\%$; mean wind speed=1.94 ± 1.11 mph) designed to elicit a T_b exceeding 39.5 °C. The dependent variables were UE and LE mean and peak power. The upper-body Wingate test was performed prior to and following the LE-HST and a lower body Wingate test was performed prior to and following the UE-HST.

Participants

Eight college aged (age=24.9 ± 3.2 yr; height=123.0 ± 46.8 cm; body mass=89.9 ± 10.5 kg) healthy males participated in the study. Participants were aerobically/anaerobically trained with no history of heat-induced illness, chronic health problems, orthopedic limitations, musculoskeletal injuries, cardiovascular disease, metabolic disease, or respiratory disease within the last year. This study was approved by the Florida International University's Institutional Review Board and informed consent was obtained prior to participation.

Instrumentation

Heat stress trial. Participants completed randomly assigned UE-HST and LE-HST designed to increase T_b while wearing an American football uniform in a hot, humid environment. For each HST, participants warmed up with a 15 min run at 50% of their maximal heart rate followed by three shuttle runs of 10 meters or upper body ergometer exercise at a heart rate of 70-80% of the age-predicted heart rate range. Participants had free access to cool water throughout the HST. Participants continued the sets of 10 repetitions until a core body temperature of 39.5 °C or the T_b plateaued for three consecutive sets.

Wingate test. The 30-sec protocol was performed on a cycle ergometer following a standard protocol.[7] Peak power was determined by number of pedal revolutions in the first 5 sec of frictional load. Mean power output was determined by averaging the six, 5-sec mean power output values. Resistance for the Wingate test was set at 7.5% of the participant's weight and for the lower body and at 5% of the participant's weight for the upper body. The peak power of the Wingate anaerobic test has been shown to have high test-re-test correlation with a strong inter-tester reliability ($r = 0.99$).[8] The mean power of the Wingate anaerobic test also has a high test-re-test reliability ($r = 0.89$).[8]

Experimental Protocol

     Familiarization session. Participants were familiarized with the parameters of the investigation and completed a health history questionnaire prior to participation. We then gathered and recorded baseline measures and participants were randomly assigned to either HST. After familiarizing the participants with the testing protocols, participants were instructed on how to properly ingest the CorTemp™ pill the night before the testing session. Participants were instructed to abstain from the use of alcohol and caffeine 48 hours prior to the testing date to negate any adverse effects on hydration or metabolism.

     Data Collection. For data collection (4-8 days after the familiarization session), the participants reported to the FIU Sport Science Research Laboratory. Participants voided urine which was measured for volume, color, and specific gravity and then followed by a body mass measurement. Participants' resting heart rate, resting blood pressure and core body temperature were recorded. After pre-exercise measurements were established, participants performed either a UE or LE Wingate test followed by either the UE or LE-HST. Participants continued the HST until they reached a core T_b of 39.5ºC or until they reached a plateau in three consecutive sets of exercise at which time exercise was terminated. Two minute rest breaks occurred between sets of exercise and the researchers gathered T_b, heart rate, blood pressure, RPE, and environmental conditions were recorded. Upon completion of the HST participants removed the football uniform and performed either the UE or LE Wingate test. Participants were monitored until heart rate, blood pressure, hydrations status, and T_b return to baseline levels and then participants were discharged from the lab.

Statistical Analyses

     Dependent t-tests were used to analyze T_b and anaerobic mean and peak power for each HST. Descriptive statistics were performed for the anthropometric, thermoregulatory response, cardiovascular response, and environmental conditions measures. Data was analyzed using the SPSS 13.0 for Windows Statistical Package (SPSS, Chicago, IL). Significance was set at $P \leq .05$ for all statistical analyses.

Results

     During the UE-HST, T_b was significantly (t_4=-7.846, p=0.001) increased 2.4% (T_b=38.2$\pm$.1°C) and 5.3% during the LE-HST (T_b=39.3$\pm$.3°C) (t_7=-11.755, $p \leq$0.001). Following the LE-HST, upper-body mean power was significantly (t_7=2.892, p=.023) decreased by 12.22% from the normothermic (4081.13$\pm$1175.94 W·kg^{-1}) compared to the hyperthermic (3582.38$\pm$863.64 W·kg^{-1}) condition. Following the LE-HST, upper-body peak power was not significantly (t_7=1.638, p=.146) changed, but decreased 13.39% from the normothermic (868.13$\pm$269.51 W·kg^{-1}) compared to the hyperthermic (751.88 W·kg^{-1}) condition. Following the UE-HST, lower-body mean power was not significantly (t_4=1.437, p=.224) changed, but decreased 9.45% from the normothermic (6191.40$\pm$975.01 W·kg^{-1}) to the hyperthermic (5606.40$\pm$647.86 W·kg^{-1}) condition. Following the UE-HST, lower-body peak power was not significantly (t_4=.454, p=.673) changed, but decreased 4.7% from the normothermic (1249.20$\pm$266.95 W·kg^{-1}) to the hyperthermic (1190.40$\pm$226.49 W·kg^{-1}) condition.

Discussion

     We investigated the effect of hyperthermia induced at the LE on the anaerobic power of the UE, as well as hyperthermia induced at the UE on the anaerobic power of the LE. Our work contradicts previous research demonstrating upper body strength was unaffected by LE cycling. Participants cycled to exhaustion reaching a mean temperature of 38.8˚C, and the researchers suggested their findings indicated that the central nervous system is capable of distinguishing

between the muscles that work during exercise and the muscles that do not work during exercise.[9] Our results challenge this concept of central nervous system control or identification of working muscles, but we do suggest there is a central nervous system effect occurring during hyperthermia. Changes in the levels of neurotransmitters, acetylcholine, dopamine and serotonin, coincide with prolonged exercise and a consequential rise in T_b. Most specifically, increases in serotonin have been associated with loss of motor drive and lethargy. Conversely the increase in dopamine during exercise directly activates motor pathways and may delay the fatiguing effect by inhibiting the synthesis of serotonin. During exercise the combined effects of altered neurotransmitter levels changes are not thoroughly understood but may have a detrimental consequence on central fatigue and muscular performance.

Hyperthermia alters blood chemistry by lowering crucial energy substances for muscles. Studies found an inverse reaction of blood glucose to blood lactate levels when hyperthermia was achieved actively in controlled environments; as core body temperatures rise blood glucose decreases and blood lactate increases.[2,10] Glucose is an essential element for ATP production, the major element in energy production . The rate at which blood circulates suggests a decrease in blood glucose should affect muscle production in both the upper and lower body Wingate tests equally. Glucose and lactate levels have also shown this inverse reaction in active muscles explaining the findings of previous studies where UE was unaffected by LE exercise with an increased T_b but LE muscle function was negatively affected. It is obvious that changes in muscle metabolism will have an adverse effect on local muscle output but it should not affect muscles in the opposite extremity. In the current study we eliminated the local fatiguing effect of exercise by testing the opposite extremity but still found deficits in muscle production. We suggest that the chemical changes that occur in the blood stream resulting from hyperthermia following the HST could be associated with decreases in anaerobic power.

As previously mentioned, levels of glucose, glycogen and ATP fall more severely during active hyperthermia possibly contributing to the decrease in muscle power. Active hyperthermia has more devastating effects on the body as compared to passive hyperthermia. During active hyperthermia the body not only has to combat the effects of the environment on the body but also the depletion of blood and muscle substances along with the stress of metabolic heat produced by the muscle.

Limitations

Limitations to the study include the relatively small sample size; thus future replications of this study should incorporate a larger sample size to increase validity of the results. Another limitation of our study was the UE-HST (T_b=38.2$\pm$.1°C) did not produce hyperthermic levels consistent with the LE-HST (T_b=39.3+.3°C). We believe a major contributor to this fact was the size of the muscles producing the work to achieve hyperthermia. This suggests that the larger leg muscles produce far more metabolic heat than the smaller arm muscle counterparts. Further research is warranted.

Clinical Implications

The negative affect of hyperthermia on anaerobic power has important clinical implications. Muscular power and performance are most crucial to individuals in athletic competition and our findings are of great importance. As athletes near hyptherthermic levels, muscular power will deteriorate and performance will suffer. This deterioration in muscular performance combined with the fatiguing effects of competition will not only lead to a decrease in athletic performance but can place the individual at an inherent risk for injury.

Conclusions

Our findings suggest that an increase in T_b can have negative performance implications. Coaches, athletes, parents and athletic trainers should be wary of extended periods of exercise in hot, humid conditions. A hyperthermic athlete's performance will suffer. More extreme levels of hyperthermia severely affect anaerobic power and can lead to poor performance, as well as dangerous participant conditions.

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