ORIGINAL ARTICLE

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Lowering of skin temperature decreases isokinetic maximal force production independent of core temperature

Abstract Based on studies using maximal isometric contractions, it is suggested that fatigue may be elicited due to hyperthermia-induced impairments in central neuromuscular activation. We investigated the effects of passive hyperthermia on isokinetic maximal force production. Twenty young healthy males [peak oxygen uptake 52.9 (6.8) ml kg^{-1} min^{-1}, body fat 11 (5)%] were passively warmed in a hot (42°C) water bath to rectal temperature (T_{re}) of 39.5°C and then cooled back to 38.0°C. At 0.5°C intervals, they performed two maximal voluntary knee extensions each at 60, 120, and 240 s^{-1}. Peak torques at 37.5°C were 168 (34), 145 (29), and 112 (17) N m for 60, 120, and 240 s^{-1} contraction speeds, respectively, and 166 (38), 150 (31), and 119 (17) N m at T_{re} of 39.5°C. No significant differences in peak torque at any of the three contraction speeds were observed over the range 37.5–39.5°C. Skin cooling, even with a warm core of 39.5°C, immediately decreased peak torque [159 (42), 133 (34), 107 (22) N m at 60, 120, and 240 s^{-1}, respectively). We conclude that lowered skin temperature can impair isokinetic force production independent of core temperature.

Keywords Isokinetic contraction · Maximal voluntary contraction · Passive heating · Central versus peripheral temperature

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Introduction

Hyperthermia, or elevated body temperature, may arise through a combination of endogenous heat generated through exercise and/or exogenous heat through exposure to hot environments. Hyperthermia increases the physiological strain on the body, and recent research has demonstrated a consistent core temperature at the point of voluntary fatigue during whole-body exercise in animals (Walters et al. 2000) and both fit (Gonzalez-Alonso et al. 1999) and non-fit (Cheung and McLellan 1998) humans, leading to the proposal of elevated central temperatures per se as a mechanism for eliciting fatigue (Nielsen and Nybo 2003). One potential contributor to hyperthermia-induced fatigue may be an impaired central neuromuscular activation, based on decreased maximal isometric voluntary contractions of both exercised (leg) and non-exercised (hand) muscles at esophageal temperatures of ∼40°C following exercise-induced hyperthermia in a hot environment (Nybo and Nielsen 2001a) and also a gradual reduction in motor unit activation with passive hyperthermia (Morrison et al. 2004). The idea of decreased central activation at hyperthermic fatigue is further supported by evidence of decreased mental arousal (Nielsen et al. 2001) along with decreased cerebral blood flow (Nybo and Nielsen 2001b) during hyperthermia.

Recent work on isolating the mechanisms of hyperthermia-induced fatigue has focused on peak torque and motor unit activation during brief or sustained maximal isometric contractions (Morrison et al. 2004; Nybo and Nielsen 2001a). However, the effect of core and skin temperature on dynamic muscle contractions and different speeds of contraction remain less explored. With brief bursts of high intensity exercise, elevated muscle temperature appear to result in increases in both peak and mean power outputs during cycling sprints in both free-cadence (Ball et al. 1999; Falk et al. 1998) and isokinetic cycling modes (Sargeant 1987), while cooling of the musculature results in impaired high intensity

performance (Comeau et al. 2003; Sleivert et al. 2001). The potentiation in power with muscle warming is greater at higher isokinetic pedaling cadences (Sargeant 1987) suggesting a velocity dependence with muscle warming. This is supported by higher power outputs during cycling sprinting following 30 min passive exposure to a warm (30°C) environment due to a higher pedaling cadence (Ball et al. 1999).

The relative contribution of elevations in core versus skin temperatures on isokinetic force production and hyperthermia-induced fatigue remains unclear. Force production may be impaired by changes in local arm muscle temperature without any concomitant changes in core temperature (Giesbrecht et al. 1995), and may explain why individuals suddenly exposed to cold environments, such as shipwreck survivors, are unable to perform survival tasks and greatly increase their risk of drowning and death well before the onset of core temperature cooling and hypothermia (Giesbrecht 2000). The use of active exercise in a hot environment in most protocols to elicit hyperthermia raises skin temperature at the same time, making it difficult to control one variable while manipulating the other. Convertino et al. (1980) attempted to isolate the effects of exercise and thermal factors on the hypervolemic response by matching heat stress from exercise and passive heating, and the latter method, when combined with the use of a cooling garment to alter skin temperature, may present a better modality for understanding any direct effect of hyperthermia on neuromuscular function.

The purpose of the present study was to investigate the effects of passive hyperthermia and also to isolate the effects of core versus skin temperature on force production with isokinetic maximal voluntary contractions of the quadriceps. At 0.5°C core temperature intervals during heating from 37.5 to 39.5°C and then during cooling from 39.5°C back down to 38.0°C, subjects performed maximal knee extensions at three contractions speeds on an isokinetic dynamometer. We hypothesized the following: (1) that peak torques at the highest core temperatures would be lower than at cooler temperatures, and (2) that peak torques would be lower with cool skin than with warm skin.

Methods

Subjects

Twenty healthy male subjects between the ages of 18 and 34 years [74.7 (9.0) kg, 52.9 (6.8) ml kg^{-1} min^{-1} peak oxygen uptake ($\dot{V}O_{2peak}$), 11 (5)% body fat; mean (SD)] were recruited from the university population. Subjects were screened using the Physical Activity Readiness Questionnaire developed by the Canadian Society for Exercise Physiology (2002). They were then informed of all the experimental procedures and the associated risks and discomforts before providing written consent for their participation. The Health Sciences Research Ethics Board of Dalhousie University approved the experimental protocol and instrumentation used in the present study.

Research design

An initial control condition consisting of six sets of isokinetic maximal knee extensions over 1 h was included to account for changes in performance due to learning, boredom, or fatigue from repeated exercise. Passive heating was employed to minimize the possible confounding effects of exercise in comparing heating and cooling phases. Both heating and subsequent cooling back to baseline were employed to isolate the effects of central core temperature versus skin temperature (i.e., same core temperatures but hot skin during heating phase versus cool skin during cooling phase) on force production.

Control protocol

In session 1, basic anthropometric measures were obtained, consisting of height, body mass, and body fatness estimation using skinfold measurements and calculated using the Jackson and Pollack method (1978). Subjects then performed a graded maximal exercise test on a cycle ergometer (Monark 818, Sweden) beginning at 60 W and increasing at 30 W each minute until exhaustion for the determination of $\dot{V}O_{2peak}$ from measurement of expired gases (Aerosport VO$_{2000}$, Aerosport, Ann Arbour, Mich.).

Following aerobic testing, subjects rested for 30 min and then performed a session on the isokinetic dynamometer (Kin-Com, Chattanooga Group, Hixson, Tenn.) to familiarize themselves with performing isokinetic contractions and also to determine the possible effects of boredom or fatigue on performance. At 15 min intervals, subjects performed two maximal voluntary contractions (MVCs) consisting of knee extensions of the right leg at speeds of 60, 120, and 240 s^{-1}. The order of presentation of the speeds was always 120 s^{-1} first, followed by 60 or 240 s^{-1} in a counter-balanced pattern. Recovery time was approximately 10 s between MVCs at each speed and 1 min between speeds.

Experimental protocol

The experimental trial consisted of one single session where subjects performed maximal isokinetic contractions while first being passively heated from resting temperature of 37.5°C up to 39.5°C and then passively cooled back to 38.0°C. Upon arrival at the laboratory, subjects changed into a bathing suit and inserted the rectal thermocouple (Mon-A-Therm Core, Mallinkrodt Medical, St. Louis, Mo.) 15 cm beyond the anal sphincter for measurement of rectal temperature (T_{re}). T_{re} is a valid and reliable index of body heat storage during hyperthermia that tracks ∼0.3–0.4°C below esophageal temperature (Moran and Mendal 2002), which in turn tracks ∼0.2°C below predicted brain temperature (Nybo et al. 2002). They were then instrumented for heart rate (Polar Vantage XL, Polar Electro Oy, Finland) and skin temperature (T_{sk}: Mon-A-Therm Skin, Mallinkrodt Medical) at four sites (chest, upper arm, thigh, calf) for calculation of mean skin temperature (T_{sk}) (Ramanathan 1964). Mean body temperature (T_{body}) was calculated using a core and T_{sk} weighting value of $T_{body} = 0.65$ (T_{re}) + 0.35(T_{sk}%) (Ramanathan 1964). T_{re} and T_{sk} were recorded every 30 s on an IBM-compatible laptop using a customized data acquisition system software written with Labview (Labview 5.1, National Instruments, Austin, Tex.) software.

Following instrumentation, subjects performed two MVCs at speeds of 60, 120, and 240 s^{-1}. Recovery time was approximately 10 s between MVCs at each speed and 1 min between speeds. The order of presentation of the speeds was always 120 s^{-1}, followed by 60 or 240 s^{-1} in a counter-balanced pattern. Subjects were then immersed to the neck in a hot (42°C) water bath and warmed passively. At 37.5, 38.0, 38.5, 39.0, and 39.5°C T_{re}, subjects were removed from the tub and performed the isokinetic test battery. Heart rate (HR), blood pressure, and subjective thermal sensation using a 21-point thermal comfort vote (TCV) scale ranging from +10 being unbearably hot, 0 being neutral, and −10 being

unbearably cold (Mekjavic et al. 1994) were recorded once the subjects were seated on the Kin-Com device. Total out-of-water time for each test battery was approximately 5 min. Upon finishing the test battery at 39.5°C or upon voluntary exhaustion, subjects were dressed in a liquid conditioning garment (LCG) (Core-Tech Tubesuit, Delta-Temax, Pembroke, Canada) consisting of 3.2 mm diameter Tygon tubing closely sewn over a tightly fitting hood, pants, and shirt covering the torso and arms. The face, hands, and feet were left uncovered. Cold (8°C) fluid was circulated through the suit at approximately 1 l min^{-1}, and subjects were cooled until T_{re} returned to 38.0°C, performing the isokinetic test battery at 39.5, 39.0, 38.5, and 38.0°C.

Hydration

Subjects were encouraged to drink a carbohydrate–electrolyte drink (Gatorade) throughout the heating and cooling protocol. Body weight was measured at each testing point using a digital strain gauge scale (HL120, Avery Berkel, LaPorte, Ind.), and subjects were requested to consume an amount of fluid equal to any weight lost during that interval.

Data analysis

Peak torque (newton meters) for the MVC at each contraction speed was recorded on the Kin-Com, and the higher of the two values were plotted at each time point for the control session and temperature point for the testing session. Peak torque was compared during the heating phase (T_{re} from 37.5°C up to 39.5°C) and cooling phase (T_{re} from 39.5°C down to 38.0°C) using a three-factor (phase × temperature × speed) repeated measures analysis of variance (ANOVA) with Bonferroni adjustment for multiple comparisons. Statistical significance was set at $P < 0.05$ unless otherwise stated.

Results

Temperature

The heating and cooling protocol was able to successfully elevate core body temperature from normothermic resting values up to 39.5°C and then return subjects to 38.0°C, with the total experimental session, including isokinetic testing, requiring 183 (38) min. Fluid consumption was ~1.5 l over the ~90 min of the heating phase. The hydration protocol was able to maintain euhydration in subjects throughout both the heating and cooling phases. Body mass was 76.7 (8.8) kg at the start of the experimental session at $T_{re} = 37.5°C$, and was similar at 77.0 (8.8) kg upon completion of the heating phase at $T_{re} = 39.5°C$.

Three subjects reached 39.0°C T_{re} but were unable to voluntarily reach the initial target of 39.5°C. These subjects were however able to perform the cooling phase back down to 38.0°C. These three have been included in the mean data, but have been dropped (listwise comparison) in the ANOVA analyses, which include the highest core temperature (39.5°C). All subjects reported being at or very near the point of hyperthermia-induced exhaustion, supported by near-maximal subjective scores on the TCV at the end of the heating phase (Fig. 1). The T_{sk} and TCV gradually increased over the course of the heating phase, from 32.5 (1.9)°C and 1 (1), respectively, at the starting T_{re} of 37.5°C, to 35.6 (1.5)°C and 8 (2), respectively, at the final heating T_{re} of 39.5°C (Fig. 1). Immediately upon donning of the LCG and the initiation of the cooling phase, T_{sk} and TCV rapidly decreased to 31.8 (1.9)°C and −2 (2) while T_{re} remained at 39.5°C. T_{sk} and TCV then gradually decreased to 27.6 (1.8)°C and −6 (2) when T_{re} dropped to 38.0°C.

The passive immersion and warming of the skin resulted in an elevation in HR from an initial value of 83 (20) beats min^{-1} at T_{re} of 37.5°C to 125 (17) beats min^{-1} at 39.5°C ($P < 0.001$) (Fig. 2). Mean arterial pressure (MAP) also decreased significantly from initial values of 86 (8) mmHg to 68 (6) mmHg at 39.5°C T_{re} ($P < 0.001$) (Fig. 2). Immediately upon cooling, MAP returned to near initial values [89 (6) mmHg], remaining at a plateau for the remainder of the cooling phase. HR also

Fig. 1 Mean skin temperature (T_{sk} ■), mean body temperature (T_{body}), and subjective thermal comfort vote (TCV ○) during passive heating from rectal temperature 37.5°C to 39.5°C and then passive cooling from 39.5°C to 38.0°C. Values are given in means (SD). Matching letters indicate significant differences for T_{sk}, T_{body}, and TCV ($P < 0.001$)
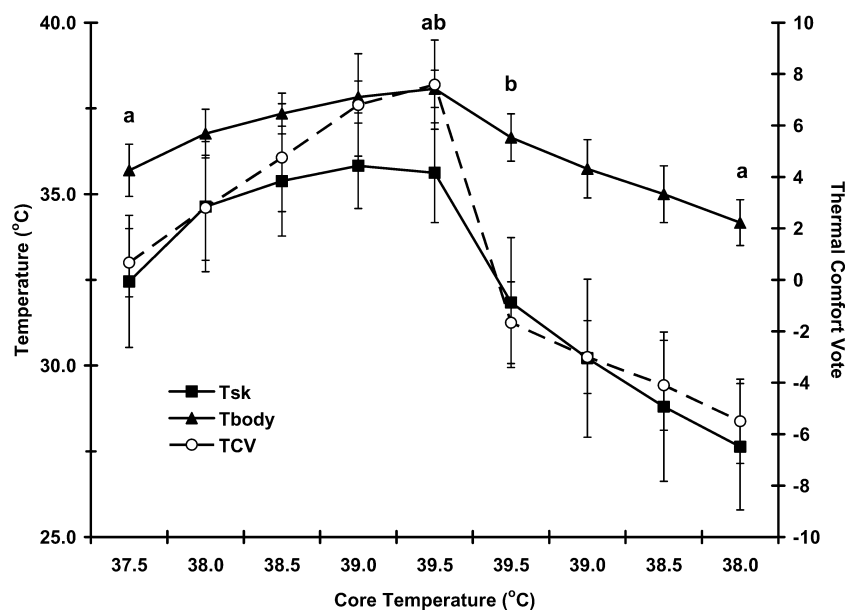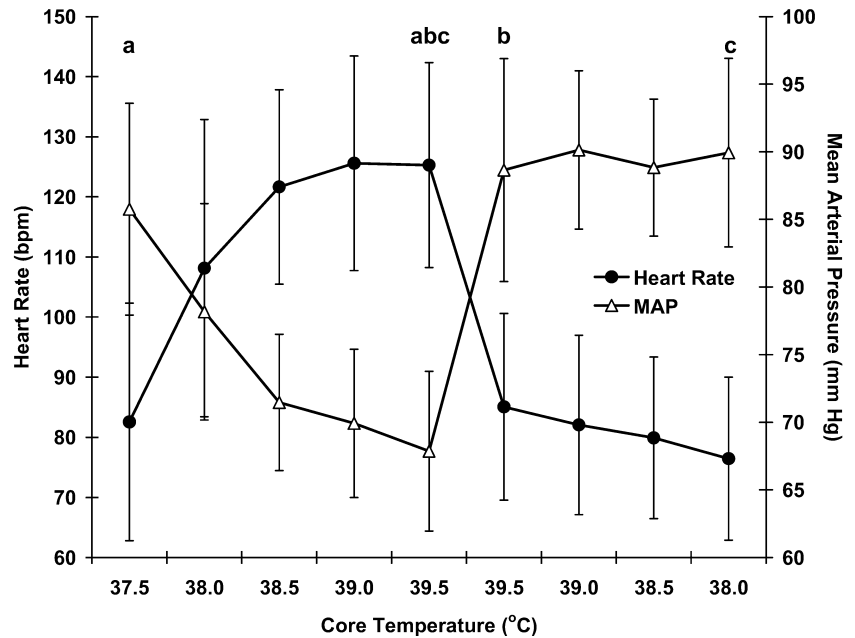
Fig. 2 Heart rate (●) and mean arterial pressure (*MAP* Δ) response during passive heating from rectal temperature 37.5°C to 39.5°C and then passive cooling from 39.5°C to 38.0°C. Values are given in means (SD). Matching letters indicate significant differences for heart rate and MAP ($P < 0.001$)

decreased back to initial values immediately upon the initiation of cooling, dropping to 85 (16) beats min^{-1} at the start of cooling ($T_{re} = 39.5$°C, $P < 0.001$) and remaining stable for the remainder of the cooling phase.

Isokinetic strength

During the control session consisting of six sets of isokinetic maximal voluntary knee extensions over 1 h (data not shown), peak torques were inversely related to the contraction speed, with significant differences ($P < 0.001$) in peak torques between 60, 120, and 240 s^{-1} contraction speeds. No differences were observed in peak torques over the course of the six control tests at any of the contraction speeds.

Peak torques at 60, 120, and 240 s^{-1} contraction speeds did not change over the heating phase from T_{re} 37.5–39.5°C ($P = 0.47$, Fig. 3). Peak torque at 60 s^{-1} was 167 (34) N m at 37.5°C and 166 (38) N m at 39.5°C. Peak torque was again inversely related to contraction speed, with significantly lower peak torque values at the 120 and 240 s^{-1} speeds ($P < 0.001$). Peak torque at 240 s^{-1} was 111 (17) N m at 37.5°C and 119 (17) N m at 39.5°C. Immediately upon skin cooling and with T_{re} remaining elevated at 39.5°C, peak torque at all contraction speeds decreased significantly, with peak torque at 60 and 240 s^{-1} speeds of 159 (42) and 107 (22) N m

Fig. 3 Peak torques generated with maximal 60 (■), 120 (○), and 240 () s^{-1} isokinetic knee extensions during passive heating from rectal temperature 37.5°C to 39.5°C and then passive cooling from 39.5°C to 38.0°C. Values are given in means (SD). No significant changes in peak torque were observed at any of the contractions speeds during the heating phase (37.5–39.5°C) or the cooling phase (39.5–38.0°C) separately. *a* A significant main effect of heating versus cooling phases on peak torque. *b* A significant main effect of contraction speed on peak torque across all temperature conditions ($P < 0.001$)
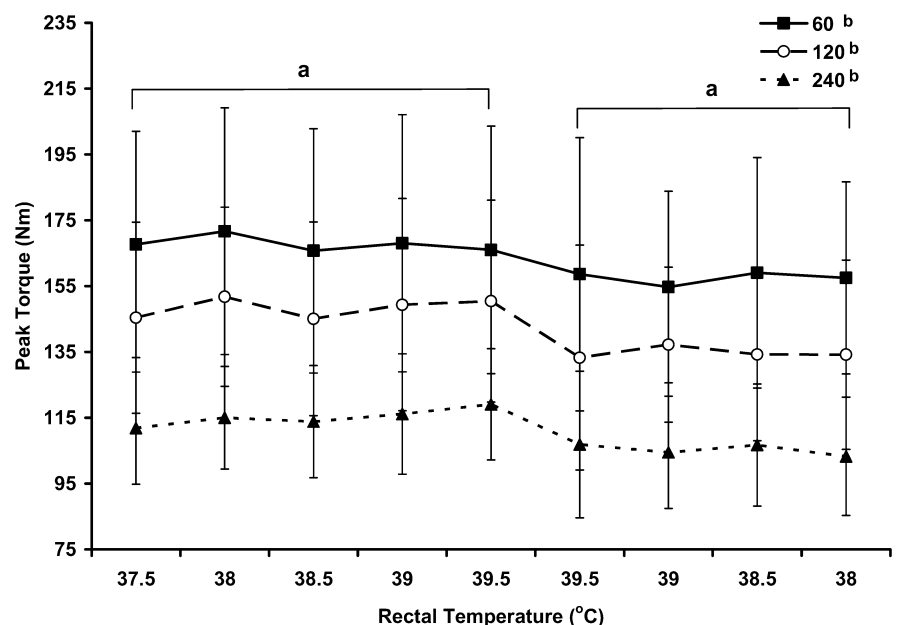
respectively ($P < 0.001$). Peak torques during the rest of the whole-body cooling phase remained stable and lower than those observed during whole-body heating, with the same pattern of higher peak torques with lower contraction speeds.

Discussion

The primary finding of the present study was that passive heating from 37.5°C up to 39.5°C T_{re} did not significantly impair torque outputs during isokinetic maximal voluntary knee extensions at three different contraction speeds. This is in contrast to other reports that isokinetic strength or dynamic force output were increased with heat exposure or local muscle heating (Ball et al. 1999; Falk et al. 1998; Sargeant 1987). In addition, we observed that decreases in local T_{sk} during the cooling phase, despite an elevated core temperature, elicited a lower peak torque at all contraction speeds and core temperatures than the peak torques generated at the equivalent core temperature during the heating phase (warm skin).

By performing passive heating over the course of a single exposure while maintaining euhydration, we were able to provide a profile of isokinetic strength over the course of heat exposure from normothermia to very high levels of hyperthermia near or at the point of voluntary exhaustion previously reported for prolonged submaximal exercise (Gonzalez-Alonso et al. 1999). Our observations of no potentiation of peak isokinetic torque over the course of whole-body hyperthermia supports a previous report of no potentiation with local leg heating (Stanley et al. 1994). This differs from other reports of enhancement with heat exposure (Ball et al. 1999; Falk et al. 1998; Sargeant 1987), possibly due to the different protocols employed. These protocols employed a single and relatively brief hot air exposure or partial water immersion at different temperatures to warm up the body. However, core temperatures were either not recorded (Ball et al. 1999; Sargeant 1987) or else the heat exposure resulted in minimal changes in body temperature (Falk et al. 1998), making the level of thermal strain either unknown or likely much lower than with the present protocol. Other studies using exercise-induced hyperthermia have also not accounted for the effects of dehydration or exercise-induced fatigue (Ftaiti et al. 2001).

The effects of hyperthermia on force production may be dependent on the mode of exercise. Major decrements in force production were reported with sustained isometric contraction of both leg and arm muscles following high levels of exercise-induced hyperthermia (Nybo and Nielsen 2001a) and also in the legs following passive hyperthermia (Morrison et al. 2004). However, an alternate explanation is that hyperthermia may only attenuate the ability to sustain maximal muscle activation rather than the ability to mobilize maximal muscle force for a short period of time (Thornley et al. 2003), as

the maximal duration of the isokinetic contractions (2–3 s for 60 s^{-1}) is much shorter than the typical use of 10–30 s isometric contractions. Ftaiti et al. (2001) found that exercise-induced hyperthermia resulted in decrements in isometric knee extension and at isokinetic speeds of 60 s^{-1} but not at the faster isokinetic speed of 240 s^{-1}. They also reported less electromyographic activity at these speeds and suggested activation deficits were specific to isometric or low velocity isokinetic contractions. We observed neither a potentiation of isokinetic peak torque with mild hyperthermia nor the sudden decrease in isometric peak force observed at high (39.5–40°C) core temperatures. This is in contrast to recent research suggesting a critical internal temperature at which fatigue is elicited through changes in cerebral activity or central neuromuscular activation (Nielsen et al. 2001; Nybo and Nielsen 2001a). The exercise protocol of Ftaiti et al. (2001) also elicited high levels of cardiovascular and physiological exhaustion, and this physiological strain may have contributed to the difference in impairment with hyperthermia at slow isokinetic speeds between their study and the present one.

While core temperature had no impact on isokinetic strength, a rapid decrease in skin temperature with the initiation of cooling significantly decreased peak torque throughout the entire cooling phase, despite elevated core temperatures, suggesting a significant contribution of local skin temperature on isokinetic strength. Handgrip strength decreased significantly with only brief (2 min) immersion of the hand or forearm in cold (5°C) water (Vincent and Tipton 1988), while impairments in manual dexterity and grip strength were primarily driven by local arm temperature rather than core temperatures (Giesbrecht et al. 1995). The second isokinetic test at 39.5°C T_{re} occurred approximately 10–15 min after the initial test at 39.5°C, coming after the donning of the cooling garment and the peaking of T_{re} following immersion. The rapid and significant drop in skin temperature upon the initiation of cooling suggests that changes in muscle temperature were likely to be negligible. In addition, over the course of the cooling phase, our use of a cooling garment likely decreased skin temperature without significant muscular effects, as direct ice-pack application for 30 min resulted in no changes in thigh muscle temperature below 2 cm (Enwemeka et al. 2002). The decrease in peak torque with skin cooling supports the decrease reported in isokinetic peak torque following 40 min of passive exposure to 10°C and 5°C air (Comeau et al. 2003) and also the decrease in peak force observed following leg immersion in cold water (Sargeant 1987). Overall, the present results support the idea that muscle function and force production may be rapidly impaired by skin cooling even with an elevated core temperature, emphasizing the importance of thermal protection of both the core and the peripheries during both sudden and sustained exposure to cold environments.

In conclusion, no effects on the peak torque generated during isokinetic maximal voluntary knee extensions

were observed during passive elevation of core body temperature from normothermia to 39.5°C, suggesting that whole-body hyperthermia both does not facilitate high-intensity exercise and also that no critical central body temperature threshold for impairment of isokinetic force production exists over the range of core temperature tested. However, local skin cooling rapidly decreased peak torque compared to equivalent core temperatures, suggesting a strong role of skin temperature in force production and supporting observations of rapid impairment of muscular function upon exposure to cold environments. Future research should focus upon isolating the respective roles of central versus peripheral temperatures on force production during dynamic exercise and also on clarifying the mechanisms and linkages between thermal manipulations, cerebral responses, and neuromuscular function.

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