HIGHLIGHTED TOPIC | A Physiological Systems Approach to Human and Mammalian Thermoregulation

Voluntary muscle activation is impaired by core temperature rather than local muscle temperature

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Voluntary muscle activation is impaired by core temperature rather than local muscle temperature. J Appl Physiol 100: 1361–1369, 2006. First published December 8, 2005; doi:10.1152/japplphysiol.00945.2005.—Fatigue during hyperthermia may be due in part to a failure of the central nervous system to fully activate the working muscles. We investigated the effects of passive hyperthermia on maximal plantar flexor isometric torque (maximal isometric voluntary contraction) and voluntary activation to determine the roles of local skin temperature, core temperature, and peripheral muscle temperature in fatigue. Nine healthy subjects were passively heated from 37.2 to 39.5°C (core temperature) and then cooled back down to 37.9°C using a liquid-conditioning garment, with the right leg kept at a thermoneutral temperature throughout the protocol, whereas the left leg was allowed to heat and cool. Passive heating resulted in significant decreases in torque from [mean (SD)] 172 N·m (SD 39) to 160 N·m (SD 44) and in voluntary activation from 96% (SD 2) to 91% (SD 5) in the heated leg, and maximal isometric voluntary contraction decreased similarly from 178 N·m (SD 37) to 165 N·m (SD 38) and voluntary activation from 97% (SD 2) to 94% (SD 5) in the thermoneutral leg. The initiation of cooling, which produced a rapid decrease in skin temperature and cardiovascular strain [heart rate reserve decreased from 58% (SD 12) to 31% (SD 12)], did not immediately restore either torque or voluntary activation. However, when core temperature was lowered back to normal, torque and voluntary activation were restored to baseline values. It was concluded that an increase in core temperature is a factor responsible for reducing voluntary activation during brief voluntary isometric contractions and that temperature-induced changes in the contractile properties of muscle and local thermal afferent input from the skin do not contribute significantly to the decrement in torque.

maximal voluntary contraction; hyperthermia; fatigue; neuromuscular activation; triceps surae

IN BOTH ANIMAL (39) AND HUMAN (10) studies, voluntary termination of exercise in the heat occurred at a consistent core temperature (~40°C) despite alterations in either starting core temperature or the rate of heat storage. This suggests that a critical internal temperature, rather than circulatory failure, may directly elicit exhaustion (5, 24, 39). Recent research has focused on the direct effects of hyperthermia on the central nervous system, including alterations in brain arousal (25), cerebral blood flow (27, 29), brain heat storage (31), perceived exertion (30), and muscular activation (23, 28, 35, 37).

Both central and peripheral mechanisms can contribute to neuromuscular fatigue (for review see Ref. 8) (14, 16, 26), and the extent of central vs. peripheral mechanisms that influence neuromuscular impairment during hyperthermia remains unclear. Nybo and Nielsen (28) demonstrated a decrease in voluntary isometric force in both the exercised leg and nonexercised arm muscles after exercise-induced hyperthermia, which they attributed to a lower central voluntary activation. However, a similar study by Saboisky et al. (35) reported a reduction in the central activation ratio, the ratio of maximal voluntary contraction force to superimposed force, of the exercised leg muscles but no decrease in the central activation ratio of the nonexercised arm muscles after exhaustive exercise in the heat. However, the use of exercise-induced hyperthermia, although of practical relevance, typically leads to high cardiovascular strain and dehydration (7), which, coupled with metabolic changes from exercise (33), can confound direct interpretation of the role of hyperthermia per se.

To target the central effects of hyperthermia, Morrison et al. (23) passively heated and then cooled subjects using a liquid-conditioning garment while maintaining euhydration and relatively low (~55% heart rate reserve) cardiovascular strain, permitting the comparison of the same core temperatures with both hot and cool skin. Both maximal voluntary force and central voluntary activation during brief (10 s) maximal isometric knee extension gradually decreased with an increase in core temperature. Both subsequently returned to baseline values when cooled to normothermia, thus suggesting a centrally mediated impairment of neuromuscular activation (23). Using transcranial magnetic stimulation, Todd et al. (37) reported impairment of voluntary torque during brief and sustained maximal isometric elbow flexion during passive hyperthermia. Coupled with a decrease in half relaxation time while hyperthermic, this suggests that descending voluntary drive was not able to compensate for local muscular changes. However, the whole body passive heating model used in these studies resulted in increases in both core and muscle temperature.
Consequently, it was not possible to isolate whether the effects were due to central and/or peripheral mechanisms.

To further isolate the role of hyperthermia on central impairment of neuromuscular activation, the present experiment passively raised core temperature while maintaining thermoneutrality in one leg and allowing the other leg to change temperature with core body temperature. Therefore, the purpose of the study was to test the role of local skin and muscle temperature during hyperthermia on the ability to perform maximal isometric voluntary contractions. The hypothesis was that hyperthermia would reduce voluntary drive to the muscle, resulting in equivalent impairment of force production and voluntary activation in both the experimental and thermoneutral leg during brief maximal isometric plantar flexion.

METHODS

Subjects. This research was approved by the Health Sciences Research Ethics Board of Dalhousie University. Subjects were screened for cardiovascular and neuromuscular health problems before providing written, informed consent. Nine nonspecifically trained subjects were recruited from the university and community population. Seven men of age 25.3 yr (SD 5), height 177.7 cm (SD 6), weight 72.8 kg (SD 8), and body fat 11.2% (SD 2) participated. Two women of age 19.5 yr (SD 1), height 170.2 cm (SD 3), weight 76.1 kg (SD 2), and body fat 28.0% (SD 1) participated. Women were tested during the late follicular phase of their menstrual cycle for the experimental trial. Subjects refrained from alcohol and heavy exercise the day before testing and caffeine on the day of testing.

Subject characterization. In an initial familiarization session, both height (cm) and weight (kg) were taken, and body fatness was calculated using the seven-site skinfold method (13). Subjects then performed a maximal aerobic power test using an incremental cycle ergometer (model 824E, Monark, Varberg, Sweden) protocol. Subjects warmed up for 4 min at a power output of 60 W followed by 30-W increases each minute until they could no longer maintain the work rate. The subjects breathed into an oronasal mask during the maximal aerobic power test to obtain respiratory measures through open-circuit spirometry. Gas samples were collected at 30-s intervals using a metabolic gas analyzer (model VO2000, Aerosport, Ann Arbor, MI) Before the exercise test, the gas analyzers and the volume transducers were calibrated. Verbal encouragement was provided during the later stages of the test. Heart rate was recorded during each stage of the test using a transmitter-receiver telemetry unit (Polar Electro, Kempele, Finland). Subjects were also introduced to a plantar flexor torque-measuring apparatus and practiced performing maximal isometric voluntary contractions.

Research design. Subjects were passively heated from a resting core temperature of ~37.0–39.5°C. On reaching 39.5°C, subjects were cooled back down to 38.0°C. Local muscle and skin temperature of the right calf (thermoneutral leg) was kept constant throughout the protocol by wearing a sleeve with ice packs in it, while the left calf (heated leg) was allowed to heat up and cool down during the protocol. At multiple core temperature increments, subjects performed neuromuscular tests and cardiovascular and psychophysical measures were collected. Muscle temperature of the soleus was also recorded before maximal voluntary contractions. See Fig. 1 for time line.

Control protocol. Five subjects [3 men mean (SD): age 28 yr (SD 7) height, 180 cm (SD 6), weight 67.0 kg (SD 8), body fat 10.5% (SD 1); and 2 women: age 29 yr (SD 5), height 170 cm (SD 7), weight 62.1 kg (SD 5), body fat 19.3% (SD 1)] completed a control trial during which they performed the neuromuscular function tests (supramaximal twitch, maximal voluntary contraction, interpolated twitch technique) without any thermal manipulations. The subjects were seated in the same specially designed chair used for the experimental trial, which has been described in detail previously (40). The control trial lasted 1.5 h, and the subject performed neuromuscular tests every 15 min. This trial was completed to determine the effect of time, boredom, and possible fatigue on neuromuscular function. Subjects showed no difference in maximal voluntary contraction torque, percent voluntary activation, or peak twitch tension (P > 0.05) during the 90-min-duration control trial.

Core body temperature control. The subject was seated in an environmental chamber kept at 32°C (SD 1.1) and 15% (SD 1.0) humidity. Subjects wore a liquid-conditioning garment (Med-Eng, Pembroke, ON, Canada) that consisted of close-fitting stretchable material with Tygon tubing sewn throughout the suit. The liquid-conditioning garment covered the arms, upper legs, lower left leg, head, neck, and torso. Subjects wore shorts, and women also wore a bra under the liquid-conditioning garment to promote maximum heat transfer between the skin and the suit. Water at 50°C was pumped through the suit tubing at ~1 l/min to heat the subject from resting core temperature up to 39.5°C. On reaching core temperature of 39.5°C, 10°C water was then pumped through the liquid-conditioning garment tubing to cool the core temperature down to 38.0°C. The subjects were removed from the environmental chamber during cooling and were seated in a temperate (21°C, 17% humidity) environment. Local muscle and skin temperature of the right calf was kept constant throughout both the heating and cooling phases using a custom-made sleeve filled with ice packs.

Thermal measurements. Core temperature was measured using both rectal and esophageal thermistors (Mon-A-Therm Core, Mallinckrodt Medical, St. Louis, MO). The rectal thermistor was inserted 15 cm beyond the anal sphincter when the subject first arrived in the laboratory. The esophageal probe was inserted to the depth of the xiphoid process based on Mekjavic and Rempel’s regression equations (20). Subjects were then instrumented with skin thermistors to monitor skin temperature (MA-100, Thermometrics, Edison, NJ). Skin temperature was recorded at four sites on the left side of the body, and mean skin temperature was calculated using an area-weighted equation (0.3 chest, 0.3 upper arm, 0.2 thigh, 0.2 calf) (34). Skin temperature was also recorded on the right calf. Mean body temperature was calculated using a core and skin temperature weighting of 0.65 and 0.35, respectively (34). Core temperature and skin temperature were recorded every 8 s using a portable data unit and stored on a computer with accompanying software (SmartReader 8 Plus, ACR Systems, Surrey, BC, Canada).

A needle microprobe (model MT-26/2, Physiemp, Clifton, NJ) was used to measure intramuscular temperature of the soleus in both
legs. The thermocouple was connected to a portable thermometer (Thermalert TH-5, Physitemp), which digitally displayed temperature. The intramuscular temperature probe was inserted 2 cm into the soleus on the lateral side of the leg, posterior to the fibula and 2 cm below the base of the gastrocnemius. At the posterior aspect of the calf, subcutaneous fat thickness averaged 4.2 mm for healthy subjects (22). The probe was inserted into the same site each time and remained in the muscle until the temperature reached a stable value (~5 s) and then was immediately removed. The intramuscular temperature of the soleus was recorded before the neuromuscular tests were performed.

Neuromuscular function. Strength of the plantar flexors was evaluated using a custom-made apparatus described below (Fig. 2). The neuromuscular testing protocol consisted of measuring two supramaximal single twitches, followed by two maximum isometric voluntary contractions of 3- to 5-s duration to determine their maximum isometric voluntary contraction. Subjects then performed two additional isometric maximal voluntary contractions with the interpolated twitch technique to assess voluntary activation. Subjects received visual feedback during each trial, and they were provided with a target torque that was based on prior measurement during the familiarization session. Moderate encouragement was given to the subject during each maximum isometric voluntary contraction attempt.

Evoked force of the plantar flexors was determined using percutaneous nerve stimulation of the posterior tibial nerve. Two-lead stimulating electrodes covered with wet gauze and electrode gel were used to activate the plantar flexors. Subjects were seated in a custom-designed apparatus with their knee bent at 90°. The foot being tested was secured to a footplate by Velcro straps, and the plantar flexors were stretched to their optimal length for maximal strength by setting the ankle angle at 20° dorsiflexion (40). The axis of rotation of the ankle joint was estimated by taking a central point between the medial and lateral malleoli in both the vertical and horizontal planes. This point was then aligned with the axis of rotation of the footplate. An adjustable knee clamp applied sufficient pressure on the test leg to prevent lateral movement of the lower leg and the heel from rising during plantar flexion. Strain gauges were mounted to a steel beam attached to the shaft of the footplate. Force applied to the footplate resulted in a change in voltage. Torque was calculated as the product of the force and the distance between the ankle joint and the end of the footplate. The strain gauges were calibrated before experimentation by suspending known weights from the end of the footplate. A custom-designed amplifier was used to amplify the output of the strain gauges, which was then passed to an analog-to-digital board (model DAQPAD-6020E, National Instruments, Austin, TX) that sampled the signal at 6 kHz. The signal was displayed on two oscilloscopes and was simultaneously recorded on a personal computer using custom-designed software (LabView, National Instruments).

Electrically evoked muscle twitches were first recorded while the subject sat in position. The posterior tibial nerve of both legs was stimulated in the popliteal fossa with a 3.5 × 2.5-cm cathode lead electrode with a 11.5 × 16.5-cm anode placed under the posterior surface of the thigh. The position on the anode was adjusted to ensure it was only activating the tibial nerve and not the common peroneal nerve. A supramaximal 1-ms-duration square-wave pulse was delivered to the tibial nerve using a constant voltage stimulator (Grass S48 stimulator and Grass SIU5A stimulus isolation unit, West Warwick, RI). The intensity of the supramaximal pulse was determined at the start of the experiment by increasing the voltage in 5- and 10-V increments (beginning at 50 V) until no further increase in twitch torque was observed. The twitch duration was checked to ensure it was within normal range of 110–130 ms (6). Two supramaximal twitches delivered 5 s apart were recorded at each time point. Contractile characteristics of the single twitches, which included peak twitch torque, contraction time, and half relaxation time, were averaged at each test point. A rest period of 1 min was given between the twitches and in between the maximal voluntary contractions.

Maximal voluntary plantar flexor activation was assessed using the interpolated twitch technique (21). Two supramaximal (~150% of control twitch stimulus intensity) square-wave 1-ms pulses were delivered at a frequency of 200 Hz at the point where subjects achieved maximal torque. The experimenter watched the torque signal on two oscilloscopes, one set at a low and the other at a high sensitivity. The nerve was stimulated when no further increment in torque was observed on the oscilloscope set at a higher sensitivity. If the stimulus was not given at the point of maximal torque, the trial was rejected, and the subject was asked to perform an additional trial. A second stimulus was given 2 s after the subject completed the contraction for comparison with superimposed stimulus. The trial with the highest activation was used to prevent underestimation of voluntary activation. Percent voluntary activation (%VA) was calculated using the following formula:

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%VA = (1 - \text{superimposed doublet/potentiated doublet}) \times 100 \ (3)
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Cardiovascular response. Heart rate and blood pressure were recorded before recording of the intramuscular temperature of the soleus. Heart rate was recorded using a Polar heart rate monitor (Polar Vantage XL, Polar Electro). Heart rate is expressed as heart rate reserve, a percentage of the difference between maximal heart rate and resting heart rate. Blood pressure was recorded manually using a sphygmomanometer on the arm.

Hydration. To prevent dehydration subjects were encouraged to drink lukewarm Gatorade throughout the protocol. Subjects’ nude body weight was measured before beginning the session and before each neuromuscular function test. If there was a decrease in body weight, subjects were encouraged to drink more fluid.

Data analysis. For all descriptive variables (e.g., maximal isometric voluntary contraction, heart rate, blood pressure), mean and standard deviation values are reported. Repeated-measures analysis of variance (core temperature) was performed to determine the effect of core temperature on all repeated variables. Normality of the data was confirmed using Mauchly’s test sphericity, if the assumption of sphericity was not met the P value was Huynh-Feldt corrected. If significant main effects were found for maximal isometric voluntary contraction or voluntary activation, post hoc trend analysis of within-subject contrasts was performed to determine the best fit of the data. Trend analysis was done rather than pairwise comparisons to encompass the changes seen over the entire range of core temperatures. For
all other data, significant main effects were further investigated using Bonferroni post hoc tests. Because of the possible number of pairwise comparisons, only values at initial, peak core temperatures, and final time points were compared. A t-test was performed to test whether there were any differences between relative changes in maximal isometric voluntary contraction and voluntary activation from respective baseline values to values at peak core temperatures for respective legs. This comparison was done twice, once using the peak values with warm skin and another using the peak values with cool skin. Significance was accepted at $P < 0.05$ level. Statistical analysis was performed using SPSS 11.5.1 software (SPSS, Chicago, IL).

RESULTS

Thermal strain. Data are presented as means (SD) in the text and figures. The total time spent in the heating and cooling protocol was 129 min (SD 25), with 108 min (SD 22) for heating and 20 min (SD 10) for cooling. During the heating protocol, rectal core temperature increased from 37.2°C (SD 0.2) to 39.5°C (SD 0.1) ($P < 0.001$) and returned to 37.9°C (SD 0.1) with cooling. Esophageal temperature followed a similar pattern to rectal temperature during heating; however, it was affected by the cooler room air and ventilation during cooling and did not provide an accurate measurement of core temperature. Therefore, all measures are reported relative to rectal temperature throughout the study. Mean skin temperature increased with heating and then dropped quickly with the initiation of cooling. Body temperature followed a similar pattern to that of core and skin temperature (Fig. 3). The skin temperature of the thermoneutral leg was maintained at a lower temperature than the heated leg during passive heating ($P < 0.001$), beginning from a resting value of 30.6 (2) and rising to 37.8 (1)°C at peak core temperature, with the heated leg moving from 31.5°C (SD 1) and rising to 41.1°C (SD 2). With initiation of the cooling, both the thermoneutral and heated leg temperatures dropped to similar values and remained similar for the remainder of the protocol. Muscle temperature of the thermoneural leg did not change throughout the protocol ($P > 0.05$). The temperature of the heated soleus muscle was significantly increased from 34.5°C (SD 0.7) to 38.7°C (SD 0.4) ($P < 0.001$) and returned to resting levels at the end of the cooling protocol (Fig. 3).

Cardiovascular strain. Heart rate reserve significantly increased at the maximum core temperature during passive heating and immediately decreased ($P < 0.001$) with the initiation of cooling and decrease in skin temperature, and returned to baseline values by completion of the cooling period (Fig. 4). Systolic blood pressure was significantly increased by the heating and cooling protocols ($P = 0.005$), whereas diastolic blood pressure was not affected ($P > 0.05$). Mean arterial pressure was significantly increased by the heating and cooling protocols ($P = 0.04$; Fig. 4).

Hydration status. Sweat was not directly measured; rather, body mass was used to indicate hydration status. Mean subject body mass did not change pre- and postexperiment [74.7 kg (SD 6) and 74.0 kg (SD 5), respectively ($P > 0.05$)]. The subjects drank 1.45 liters (SD 0.6) of Gatorade during the heating and cooling protocols.

![Fig. 3. Mean skin temperature (●), body temperature (■), and soleus muscle temperature of H (●) and TN (○) legs during passive heating from rectal temperature of 37.2–39.5°C and then passive cooling from 39.4–37.9°C. Values are means (SD). Matching letters indicate significant differences for mean skin and body temperature ($P < 0.001$). *Muscle temperature different than all other time points ($P < 0.001$).](image-url)
Neuromuscular function. There was no significant difference found in change in maximal isometric voluntary contraction or voluntary activation between thermoneutral and heated ($P > 0.05$). Maximal isometric voluntary contraction of the thermoneutral leg was significantly affected by the heating and cooling protocols ($P = 0.04$). Post hoc trend analysis revealed a significant quadratic trend ($P = 0.03$) with torque initially at $177.8 \text{ N} \cdot \text{m}$ (SD 37) and then decreasing to $165.4 \text{ N} \cdot \text{m}$ (SD 38) at the end of passive heating. There was no significant change in torque [161.3 N·m (SD 38)] with the initiation of cooling; however, at the completion of the cooling phase, torque had returned to baseline values [171.6 N·m (SD 39)] (Fig. 5). Voluntary activation of the thermoneutral leg was also significantly affected by the heating and cooling protocols ($P = 0.004$) beginning at 97.0% (SD 2) and decreasing to 94.0% (SD 5) at peak core temperature. With initiation of cooling, voluntary activation remained depressed at 93.8% (SD 5), but it returned to baseline [95.2% (SD 3)] on completion of the protocol (Fig. 5).

Maximal isometric voluntary contraction of the heated leg, which was allowed to heat up and cool down, was significantly reduced at increased core temperatures ($P < 0.001$), and remained depressed during cooling until core temperature decreased (Fig. 5). Voluntary activation was also affected by the protocol ($P = 0.002$) beginning at 95.8% (SD 2) and decreasing to 91.2% (SD 5) at peak core temperature. Voluntary activation remained depressed with the initiation of cooling [91.2% (SD 5)], and it returned to baseline values [95.2% (SD 3)] at the end of the protocol to (Fig. 5).

DISCUSSION

Recent research into the mechanisms underlying voluntary fatigue during exercise in the heat has reported decrements in neuromuscular activation during exercise-induced hyperthermia (28, 35). The primary aim of the present study was to isolate the direct effects of elevated core temperature on neuromuscular performance by using a passive heating and cooling model. Core and peripheral temperature contributions were determined by allowing one leg to heat and cool while maintaining thermoneutrality throughout in the contralateral leg. Despite differences in local soleus temperature over the course of the thermal manipulation, the pattern and magnitude of maximal isometric voluntary contraction and voluntary activation impairment during hyperthermia, and the subsequent return to baseline values with cooling back to resting core temperatures, were similar in both legs. This observation supports previous reports that hyperthermia directly impaired
neuromuscular activation (23, 37). The present results also identified that the impairment in neuromuscular activation was centrally mediated independent of changes in local muscle temperature.

This study found small but significant changes in voluntary activation as determined by the interpolated twitch technique. Although the interpolated twitch technique is the most commonly used method to assess voluntary activation, the sensitivity, especially at high contraction levels, has been questioned (2). During maximal isometric voluntary contractions, there is increased fluctuation of voluntary torque, which can affect discrimination of the interpolated twitch. We collected torque data at a high-resolution level in an attempt to maximize the resolution of the twitch. A double pulse was also used to increase the sensitivity of the interpolated twitch technique (9). Use of a double pulse to compare the interpolated twitch has been suggested to minimize any potentiation effects on variability in voluntary activation (32). Allen et al. (1) found that there was considerable variability in voluntary activation between subjects, however, within-subject variability was consistent and reproducible.

Neuromuscular fatigue during exercise can be caused by either a central failure to maximally activate muscles, alterations in local muscle characteristics, or a combination of both (16, 26). Other studies investigating fatigue of the plantar flexors in a thermoneutral environment used repeated and prolonged maximal contractions and found contributions to fatigue from both central and peripheral mechanisms (14, 26). The strong correlation between peripheral fatigue and decreases in plantar flexor strength (14, 26) suggests that the cumulative fatigue from repeated muscle contractions, such as changes in the intramuscular metabolic environment, can contribute significantly to fatigue beyond a failure of neuromuscular transmission (16). By removing sustained exercise and choosing a brief 5-s isometric plantar flexion that did not alter force production or voluntary activation when repeated periodically over 90 min of thermoneutral exposure in our protocol, the impairment with our heating protocol would not be due to peripheral fatigue or boredom. Rather, our finding of equivalent magnitude and similar patterns of decrease in torque and voluntary activation in both the thermoneutral and heated leg during passive heating to 39.5°C, despite differences in soleus temperature, supports the earlier finding that both exercised (leg) and nonexercised (arm) muscle exhibited a decrease in maximal isometric voluntary contraction and voluntary activation after exercise-induced hyperthermia even with differences in arm and leg muscle temperatures (28). Together, these studies suggest that elevated core temperatures directly affect the ability to maximally activate a muscle, and changes in muscle contractile characteristics due to peripheral muscle temperature alterations minimally influenced voluntary activation. Although none of our subjects were initially able to achieve full activation of the plantar flexors, the voluntary activation levels are in agreement with other studies (14). It is interesting to note that the change in muscle temperature in our heated leg was less than the thigh muscle temperatures at the point of volitional exhaustion during exercise in the heat (10, 28). Therefore, it remains possible that a higher soleus temperature in our heated leg may ultimately have resulted in a greater impairment in torque than observed in the thermoneutral leg.

A central dominance of muscle activation failure during hyperthermia supports the primary role of core temperature afferents in mediating responses to the muscle from a point at

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Fig. 5. Resting maximal voluntary contraction (MVC) torque and voluntary activation (VA) for H (●) and TN (○) legs during passive heating from rectal temperature of 37.2–39.5°C and then passive cooling from 39.4–37.9°C. Values are means (SD). Significant quadratic trends for H (solid lines) and TN (dashed lines) legs for torque and VA (P < 0.05).
or above the level of the motor axons. In a review by Hensel (12), it was established that sweating, metabolic, and vascular responses in humans are controlled by both central and peripheral thermal factors. However, at core temperatures above 40.3°C panting as a means of cooling in dogs could not be prevented by reductions in skin temperature, whereas at lower core temperatures skin cooling had an effect (12), suggesting an increased importance of core temperature vs. peripheral temperature in thermoregulation at high core temperatures. By both heating and cooling the subjects, we were able to differentiate the effects of skin temperature on maximal isometric voluntary contraction and voluntary activation. At the onset of the cooling process, mean skin temperature immediately decreased, whereas core temperature remained elevated. Despite this change in skin temperature, there was no increase in plantar flexor torque or voluntary activation, in agreement with the findings of Morrison et al. (23). Skin temperature also does not appear to be a significant factor in voluntary exhaustion during exercise in the heat. When the rate of heat storage and trunk skin temperature was manipulated by wearing a water-perfused jacket while cycling, highly trained subjects still fatigued at the same core and thigh temperature (~40°C) despite a difference of ~3°C in skin temperature (10).

Increases in muscle temperature have been shown to increase the speed of contraction and relaxation twitch times of the triceps surae while having no effect on peak twitch tension or maximal isometric voluntary contraction force (6). The changes in twitch characteristics noted in this study are consistent with previous findings during passive hyperthermia studies (23, 37), with changes seen only in speed of the twitch and not the peak tension. Surprisingly, both the heated and the thermoneutral leg exhibited changes in twitch characteristics, despite the soleus temperature of the thermoneutral leg remaining similar to resting values throughout the heating and cooling protocol. The soleus muscle was selected for muscle temperature measurement because in a seated flexed knee position the contribution of gastrocnemius is reduced (36), and plantar flexor torque would have been primarily determined by the soleus in our protocol. During the evoked twitches, however, both the gastrocnemius and the soleus were stimulated, and it is possible that the gastrocnemius temperature was increased enough to affect the overall twitch properties of the calf during

Fig. 6. Twitch characteristics of TN leg (○) and H leg (■), contraction time, half relaxation time, and peak torque. Values are means (SD). Matching letters indicate significant differences (a and b, for TN leg; c and d, for H leg; P < 0.001).
plantar flexion. Hansen et al. (11) have shown that increases in catecholamines can prevent force loss due to high levels of K+ and activation of glycogen phosphorylase (17). Passive heating has been shown to produce small increases in catecholamines, which may have played a role in altered twitch characteristics in the thermoneutral leg.

Care must be taken in extrapolating results from isolated muscle movements to exhaustion during whole body exercise in the heat, because discrepancies exist in neuromuscular responses to hyperthermia depending on duration and type of contraction. Although this study found significant decreases in maximal isometric voluntary contraction and voluntary activation using a relatively brief isometric contraction of 3–5 s, others only found impairment with longer duration contractions (28, 37). Nybo and Nielson (28) observed that at voluntary exhaustion after active hyperthermia, maximal isometric voluntary contraction did not become significantly less than the thermoneutral level until after 30 s of maximal contraction. These differences may have been confounded by effects of exercise itself on the metabolic state of exercising and nonexercising muscles. Todd et al. (37) used passive methods to induce hyperthermia and found that only maximal isometric voluntary contraction was decreased during brief (2 s) contractions and not voluntary activation; however, subjects were only heated to 38.5°C. They suggested that, during hyperthermia, motor unit firing rate can be increased to overcome the faster contractile speeds of muscle and maintain voluntary activation in contractions up to 2–3 s (37). Isokinetic contractions after hyperthermia have also shown a similar time-influenced response pattern. Cheung and Sleivert (4) found that isokinetic torque was not affected by passive hyperthermia during two brief contractions. After exercise-induced hyperthermia, no changes were seen in torque or voluntary activation during initial contractions, although a decrement in both torque and voluntary activation occurred with multiple contractions (19).

Thus it is possible that the body is able to overcome the effects of increased core temperature for very brief contractions but that the body may respond differently to prolonged contractions.

Although a critical core temperature of ~40°C has been hypothesized to directly elicit voluntary exhaustion (7, 10, 28, 30), an alternative model has also emerged suggesting that a central thermal anticipatory mechanism inhibits exercise capacity before the attainment of a critical temperature (18). Neuromuscular support for this model can be found during whole body self-paced exercise, with decreases in power output and/or electromyogram before significant increases in core temperature, heart rate, or perception of effort occurred (15, 38). The significant quadratic trend shown by the gradual decrease in torque seen with the progressive increase in core temperature in our study, combined with similar findings by Morrison et al. (23), also suggests that there is no critical internal temperature at which force and voluntary activation suddenly drop off. In addition, decreases in force and voluntary activation during isometric contractions of both arm and leg muscle after either passive (37) or active (35) heating was evident even at relatively low core temperature of 38.5–38.8°C, respectively. Clearly, further work is required to directly determine whether the presence and nature of a central anticipatory process regulating exercise drive involve thermal or other inputs.

In summary, the present study demonstrates that voluntary isometric force production and activation is reduced with hyperthermia, occurring independently of peripheral muscle and skin temperature. Furthermore, this reduction is progressive with increases in core temperature, gradually decreasing with rising core temperature and progressively increasing back to baseline levels as core temperature returns to thermoneutral levels. Therefore, we conclude that 1) neuromuscular impairment during hyperthermia is primarily due to a central failure to fully activate the muscle and can occur independent of local thermal alterations within the muscle, and 2) neuromuscular function does not become critically impaired on attainment of one particular temperature.

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