

Hyperthermia and central fatigue during prolonged exercise in humans

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Nybo, Lars, and Bodil Nielsen. Hyperthermia and central fatigue during prolonged exercise in humans. *J Appl Physiol* 91: 1055–1060, 2001.— The present study investigated the effects of hyperthermia on the contributions of central and peripheral factors to the development of neuromuscular fatigue. Fourteen men exercised at 60% maximal oxygen consumption on a cycle ergometer in hot (40°C; hyperthermia) and thermoneutral (18°C; control) environments. In hyperthermia, the core temperature increased throughout the exercise period and reached a peak value of $40.0 \pm 0.1^\circ\text{C}$ (mean \pm SE) at exhaustion after 50 ± 3 min of exercise. In control, core temperature stabilized at $\sim 38.0 \pm 0.1^\circ\text{C}$, and exercise was maintained for 1 h without exhausting the subjects. Immediately after the cycle trials, subjects performed 2 min of sustained maximal voluntary contraction (MVC) either with the exercised legs (knee extension) or with a “nonexercised” muscle group (handgrip). The degree of voluntary activation during sustained maximal knee extensions was assessed by superimposing electrical stimulation (EL) to nervus femoralis. Voluntary knee extensor force was similar during the first 5 s of contraction in hyperthermia and control. Thereafter, force declined in both trials, but the reduction in maximal voluntary force was more pronounced in the hyperthermic trial, and, from 30 to 120 s, the force was significantly lower in hyperthermia compared with control. Calculation of the voluntary activation percentage (MVC/MVC + EL) revealed that the degree of central activation was significantly lower in hyperthermia ($54 \pm 7\%$) compared with control ($82 \pm 6\%$). In contrast, total force of the knee extensors (MVC + force from EL) was not different in the two trials. Force development during handgrip contraction followed the same pattern of response as was observed for the knee extensors. In conclusion, these data demonstrate that the ability to generate force during a prolonged MVC is attenuated with hyperthermia, and the impaired performance is associated with a reduction in the voluntary activation percentage.

central activation; core temperature; muscle contractions

IT IS WELL DOCUMENTED that endurance can be markedly impaired in hot compared with temperate environments (12, 14, 18, 22). At high work intensities, the reduced endurance capacity seems to be closely linked to reductions in maximal oxygen uptake ($\dot{V}_{O_{2\max}}$) (20), but the mechanism(s) underlying hyperthermia-in-

duced fatigue during prolonged submaximal exercise is not well understood. Oxygen consumption, muscle blood flow, and force production in a brief maximal voluntary contraction (MVC) are similar in hyperthermic and normothermic athletes. Furthermore, neither the small increase in muscle glycogen breakdown and lactate production nor potassium release can explain the diminished endurance with hyperthermia during prolonged submaximal exercise (15, 18, 22). Alternatively, it could be that the cause of hyperthermia-induced fatigue during prolonged exercise in the heat is located in the central nervous system (CNS) (16, 18). Because hyperthermia does not affect the ability to perform a brief MVC, it has been suggested that impaired neuromuscular performance may only be observed during repeated muscle activations and that the performance limitation could be located “upstream” to the primary motor cortex (18). It has been demonstrated that goats decrease their running speed when the hypothalamic temperature is independently increased to $\sim 43^\circ\text{C}$ (11), supporting the idea that motor activity is inhibited by high temperatures in the CNS. The concept of a critical core temperature limiting exercise performance has been supported by studies in both humans and rats (13, 16, 18, 24), and several authors have suggested that “the will” or “the drive” to exercise might be diminished by hyperthermia (10, 18). However, no study has yet been able to demonstrate that “central fatigue” is actually involved in hyperthermia-induced fatigue during prolonged exercise in the heat.

Therefore, the aim of the present study was to investigate whether the development of hyperthermia during exercise would affect neuromuscular activation at the end of a prolonged exercise bout. We hypothesized that hyperthermia would result in a reduced degree of central activation during MVCs when these contractions were repeated several times or if a contraction was sustained for a prolonged duration. Different MVC protocols were therefore performed at the end of a cycle exercise bout when subjects were either normothermic ($\sim 38^\circ\text{C}$) or hyperthermic ($\sim 40^\circ\text{C}$). MVCs with the knee extensors were either repeated with short resting pe-

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riods between each MVC or a MVC was sustained for a prolonged period. In both knee extensor protocols, the capability of the CNS to activate the muscles was assessed by superimposing electrical stimulation (EL) on the voluntary contractions. The latter of the two knee extensor protocols has previously proven to be useful, when distinguishing between central and peripheral factors contributing to the development of fatigue (17). Finally, handgrip contractions were performed to see the effect on a “nonexercised” muscle group. The rationale for the different protocols was to elucidate what type (repeated or sustained) of neuromuscular activation might be affected by exercise-induced hyperthermia and, furthermore, see what influence preceding activity (either active knee extensors or relatively passive forearm muscles) had on the development of fatigue.

METHODS

Subjects. Age, body weight, height, and $\dot{V}O_{2\max}$ of the 14 healthy, endurance-trained cyclists participating in the study were 25 ± 1 yr (mean \pm SE), 71 ± 2 kg, 181 ± 2 cm, and 65 ± 2 ml \cdot kg $^{-1}\cdot$ min $^{-1}$, respectively. Subjects were informed of any risks and discomforts associated with the experiments before they gave their written consent to participate. The study was approved by the Ethics Committee of Copenhagen and Frederiksberg (KF 01-135/00). All subjects had previously participated in experiments involving cycling in hot environments, and they were familiar with the experimental setup. The subjects could not, for obvious reasons, be blinded to the treatments, but they were unaware of the researchers’ hypotheses and naive to the purpose of the study.

Experimental design. General design of the study is illustrated in Fig. 1. On two separate occasions (separated by 2–6 days), subjects cycled in a climatic chamber at $\sim 60\%$ of $\dot{V}O_{2\max}$ (power output = 188 ± 7 W; 85 rpm) in a counter-balanced order. In one trial, they exercised to exhaustion (50 ± 3 min) in a hot environment (40°C , hyperthermic trial), whereas exercise in the other trial was maintained for 1 h in a thermoneutral environment (18°C , control trial) without exhausting the subjects. Exhaustion was defined as the point at which the subject either volitionally stopped exercising or the point at which power output could no longer be main-

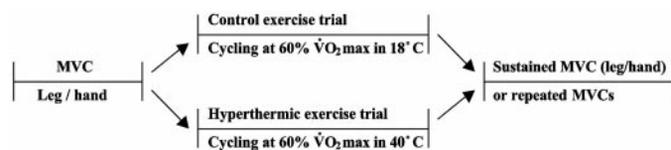


Fig. 1. Overview of the experimental protocol in which the subjects, after initial determination of their maximal voluntary contraction (MVC), either cycled at 60% maximal oxygen consumption ($\dot{V}O_{2\max}$) to exhaustion in a 40°C environment or exercised at the same intensity for 1 h in a 18°C environment without being exhausted. A total of 23 paired control and hyperthermic trials were performed. Some subjects only completed 1 control and 1 hyperthermic trial, whereas other subjects performed both knee extensor protocols (a total of 4 trials) or completed both a knee extensor and a handgrip MVC protocol. Immediately after the cycle exercise, 1 of 3 different MVC protocols were performed: 1) 40 MVCs repeated every 5 s with the knee extensors ($n = 7$), 2) 2 min of sustained MVC with the knee extensors ($n = 8$), or 3) 2 min of sustained maximal handgrip contraction ($n = 8$).

tained. The subjects arrived at the laboratory ~ 1 h before the start of the experiment and rested in a thermoneutral room while equipment was attached. The subjects then emptied their bladders, were weighed, and entered the climatic chamber. Here they were seated in a custom-made chair, and preexercise maximal voluntary isometric force and electrically stimulated force of the knee extensors were determined. Furthermore, to assess the subjects’ activation percent during a brief MVC, EL of nervus femoralis was superimposed on a maximal voluntary knee extension. Hereafter, the subjects were transferred to the cycle ergometer (Monark Ergonomic 818) and sat quietly in their racing position while resting measurements of heart rate (HR) and esophageal temperature (T_{es}) were obtained. Subjects then started to cycle, and, during the exercise period, HR, T_{es} , and rating of perceived exertion (RPE) (8) were recorded at 5, 10, 20, and 30 min and just before exhaustion (58–60 min in the control trial). Immediately (within 1 min) after termination of the cycle trial, the subjects were again seated in the custom-made chairs and performed one of three postexercise MVC protocols. The first protocol ($n = 7$) consisted of 40 maximal knee extensions repeated every 5 s (each MVC lasted ~ 2 s, and 5-s intervals made it possible to get a short resting period between each contraction). On contraction 40, EL was superimposed to assess the voluntary activation percentage. The second postexercise MVC protocol ($n = 8$) consisted of 2 min of sustained maximal isometric knee extension with EL superimposed at 30, 60, 90, and 120 s. A total of 23 paired control and hyperthermic trials were performed. Some of the 14 subjects only completed one control and one hyperthermic trial, whereas other subjects performed both knee extensor protocols (a total of 4 trials) or completed both a knee extensor and a handgrip MVC protocol. Electromyography (EMG) and force were measured continuously during both postexercise knee extensor MVC protocols, and muscle temperature (T_{mu}) was measured immediately after the contraction. The third postexercise MVC protocol ($n = 8$) consisted of 2 min of sustained maximal isometric handgrip contraction. During the 2 min of handgrip contraction, force was measured continuously, but EMG was not measured and T_{mu} was only recorded in five of the eight subjects in this protocol. The handgrip device was coated with Micropore tape, and the subject’s hand and arm were carefully dried before the contraction to avoid slipping off the grip. In two subjects, the handgrip protocol was also performed after passively induced hyperthermia. A water-perfused jacket and impermeable rain clothes were used to passively heat the subjects for ~ 2 h until T_{es} was 39.0°C .

Pre- and postexercise MVC measurements and EL. Preexercise MVC was taken as the best of three maximal voluntary isometric contractions, both in the knee extensor and handgrip protocol. Immediately after the cycle exercise, one of the three MVC protocols described above was performed, and, in all trials, subjects received equal verbal encouragement to make a maximal effort all of the time. Both before and after the cycle exercise, subjects were placed in a custom-made chair in a standardized position (knee or elbow flexed 90° , position secured with straps around the waist and thighs). The force was estimated by measuring the changes in voltage detected by a strain-gauge dynamometer placed either between the ankle and the chair or on the handgrip device. The dynamometers were calibrated with weights of known mass, and voltage changes were converted to force (expressed in N).

Superimposed EL was used to assess the degree of voluntary activation during the ongoing MVC. An anode (12×7 cm) was positioned midway between the superior aspect of the greater trochanter of the right leg and the inferior border

of the iliac crest (hip flexed 90°), whereas the ball-shaped cathode-stimulating electrode (3 cm in diameter) was positioned in the femoral triangle over the femoral nerve. A 250-ms train of stimulation (100 Hz) was delivered to the nerve at a voltage 15–20% higher than that eliciting a maximal twitch. At rest (before cycle exercise), this stimulation was able to generate a force equal to approximately one-third of MVC. The voluntary activation percentage was calculated as MVC divided by the total muscle force, where the total muscle force is the sum of MVC plus force from the superimposed EL.

EMG. Surface EMG signals were recorded from the right vastus lateralis 15 cm proximal to the superior border of the patella using a pair of EMG-recording electrodes with an interelectrode distance of 3 cm (Neuroline electrodes, Medicotest). EMG signals were amplified (gain $\times 1,000$), sampled at 1,000 Hz, and band-pass filtered [3 Hz (-3 dB) to 500 Hz (-3 dB)] using a CED 1401-plus analog-to-digital converter. Data from the isometric knee extension protocol were full-wave rectified, and integrated EMG (iEMG) was calculated from a 1,000-ms sample of data extracted every second. The changes in iEMG are expressed relative to the iEMG obtained during the preexercise MVC. Because of movement artifacts and technical problems, EMG measurements from three subjects were rejected and the EMG data, therefore, only represent five subjects.

Core and T_{mu} . T_{es} was measured in the deep esophagus with a thermocouple (model MOV-A, Ellab) inserted through the nasal passage at a distance equal to one-fourth of a subject's standing height. In the postexercise knee extensor protocols, T_{mu} was measured in musculus vastus lateralis with a needle probe (model MKA-A, Ellab) inserted 3 cm into the muscle. In the hand contraction protocol, T_{mu} was measured in caput commune flexorum at a depth of 1.5–2 cm. During cycling, HR was measured with a Polar HR recorder (Polar Electro, Kempele, Finland).

Dehydration. To avoid differences in the degree of dehydration, subjects drank 0.6 ± 0.1 liter of prewarmed water (adjusted to core temperature) in the hyperthermic trial and 0.2 ± 0.1 liter in the control trial. The degree of dehydration (estimated from the differences in body weights after exercise compared with the preexercise body weights) was thereby restricted to $0.9 \pm 0.2\%$ in the hyperthermic trial and $0.8 \pm 0.1\%$ in the control trial [$P =$ not significant (NS)]. Nude body weight was determined on a platform scale (model 1-10, Ohaus).

Statistical analysis. One- and two-way (time-by-trial) repeated-measures ANOVAs were performed to evaluate differences between and within trials. After a significant F test was performed, pairwise differences were identified using Tukey's honestly significance post hoc procedure. The significance level was set at $P < 0.05$. Data are presented as means \pm SE unless otherwise indicated.

RESULTS

T_{es} was similar at rest before the control and hyperthermic cycle trials ($36.9 \pm 0.1^\circ\text{C}$ and $37.1 \pm 0.1^\circ\text{C}$, respectively; $P =$ NS), whereas HR was slightly higher at rest before the hyperthermic trial (60 ± 2 beats/min) compared with control (55 ± 3 beats/min). During the hyperthermic trial, T_{es} increased continuously and reached a peak value of $40.0 \pm 0.1^\circ\text{C}$ at exhaustion after 50 ± 3 min of exercise. In parallel with the increase in core temperature, HR increased from 140 ± 4 beats/min at 5 min to 179 ± 3 beats/min at exhaus-

tion, and, in the same period, RPE increased from 11 ± 1 to 20 ± 0 units. In the control trial, exercise was maintained for 1 h without exhausting the subjects (RPE of 12 ± 1 at 60 min) and with T_{es} and HR stabilized at $38.0 \pm 0.1^\circ\text{C}$ and 140 ± 4 beats/min after ~ 20 min of exercise.

Repeated and sustained MVC with the knee extensors. MVC measured before the cycle exercise was similar in the two environments (537 ± 14 N before the control trial and 543 ± 12 N before hyperthermia), and, during the brief MVC, the subjects had an activation percentage of $99 \pm 1\%$. Immediately after the cycle exercise (which was exhaustive in the hyperthermic trial), brief MVC was still similar in the two experimental conditions. Furthermore, force development during the 40 MVCs repeated every 5 s was not different in the hyperthermic trial compared with the control condition (Fig. 2), and the subjects had the same voluntary activation percentage on contraction 40 in the two trials ($92 \pm 1\%$ in control and $92 \pm 2\%$ in hyperthermia). iEMG during the repeated MVCs was also similar in hyperthermia and control. T_{mu} measured after contraction 40 in the hyperthermic trial ($40.8 \pm 0.2^\circ\text{C}$) was significantly higher than that in control trial ($38.8 \pm 0.2^\circ\text{C}$; $P < 0.001$).

In the protocol with sustained maximal isometric knee extension, voluntary force was also similar during the first 5 s of contraction (454 ± 38 N in hyperthermia vs. 450 ± 48 N in control). Hereafter, force decreased in both trials, but the decline was more pronounced in the hyperthermic trial, and maximal voluntary force was therefore lower in hyperthermia compared with control from 30 to 120 s of contraction (Fig. 3A). The reduced force production with hyperthermia was associated with a significantly lower voluntary activation percentage at the end of the hyperthermic trial ($54 \pm 5\%$) compared with control ($82 \pm 5\%$; $P < 0.01$; Fig. 3B). In contrast, total force of the knee extensors (MVC + force from EL) was not different between hyperthermia and control (Fig. 3A). iEMG followed the same pattern of

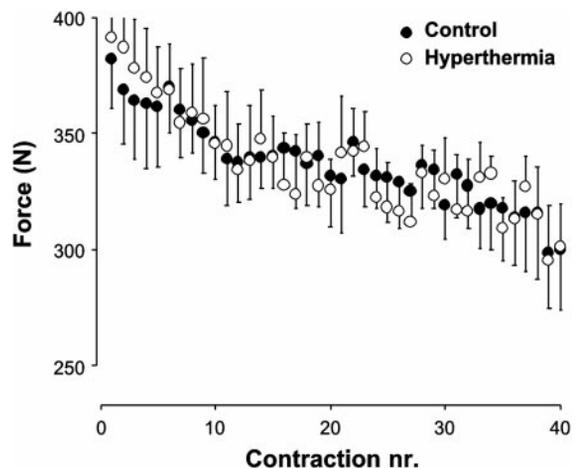


Fig. 2. Maximal force development during 40 consecutive MVCs with the knee extensors. The brief MVCs (duration of ~ 2 s) were repeated every 5 s when subjects were either normo- or hyperthermic. Data are means \pm SE for 8 subjects. nr, Number.

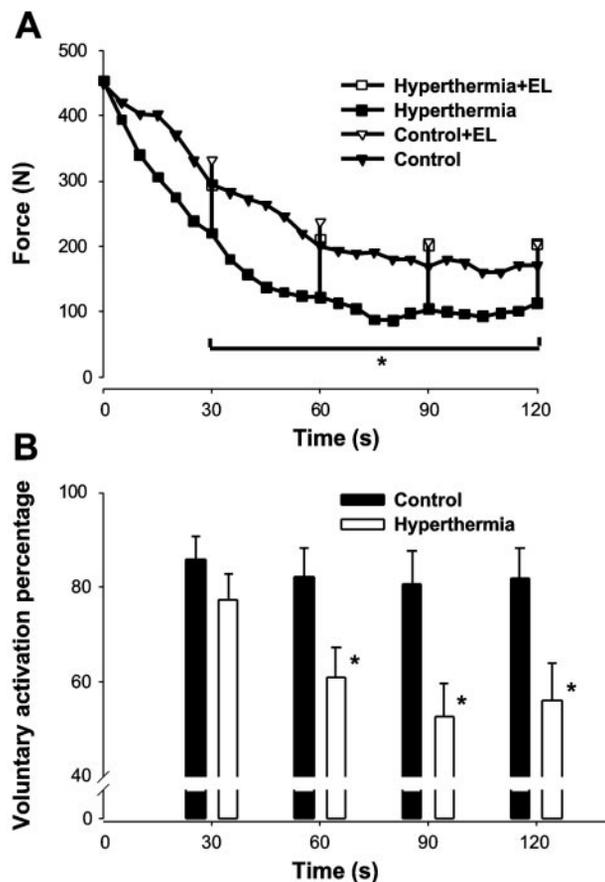


Fig. 3. The changes in force (A) and voluntary activation percentage (B) during 2 min of sustained MVC with the knee extensors during hyperthermia and control. Maximal voluntary isometric contraction was attempted by the subjects throughout the 2 min, and electrical stimulation (EL) was superimposed every 30 s to assess the degree of central activation. Data are means \pm SE for 8 subjects. A is simplified by omitting the error bars (SE were in the range of 10–20 N in both trials). *Significantly lower than control ($P < 0.05$).

response as the voluntary force production, i.e., similar magnitude at the beginning of the sustained MVC ($\sim 100\%$ of preexercise $iEMG_{max}$) but hereafter declining to $58 \pm 7\%$ during the last minute in the hyperthermic trial, which was significantly lower than the control trial value of $79 \pm 6\%$ ($P < 0.01$; Fig. 4). T_{mu} at the end of the 2 min of sustained contraction was $40.7 \pm 0.2^\circ\text{C}$ in the hyperthermic trial vs. $38.4 \pm 0.1^\circ\text{C}$ in the control, which were similar to the T_{mu} observed in the protocol with repeated MVCs.

Handgrip contractions. Maximal voluntary force development during sustained handgrip contraction (Fig. 5) followed the same pattern of response as the force during the 2 min of knee extension (Fig. 3A); similar MVC were observed before cycling and during the first 5–10 s of the postexercise protocol in both trials. Hereafter, voluntary force production declined more in the hyperthermic condition compared with control, and, during both the sustained handgrip contraction and knee extension, voluntary force development in the hyperthermic trials was reduced to $\sim 60\%$ of the control trial values during the last minute of contraction. After

30 s of recovery from the sustained handgrip contraction, MVC was again similar in the hyperthermic and normothermic conditions (Fig. 5). Forearm T_{mu} was higher after the handgrip contraction in the hyperthermic trial ($38.7 \pm 0.2^\circ\text{C}$) compared with control ($37.1 \pm 0.2^\circ\text{C}$, $P < 0.001$). However, T_{mu} in the forearm after the handgrip exercise in the hyperthermic trial was significantly lower than T_{mu} in vastus lateralis after the corresponding knee extensor protocol. Core temperatures at the end of the hyperthermic handgrip and knee extension protocols were not different ($39.8 \pm 0.2^\circ\text{C}$ and $39.7 \pm 0.1^\circ\text{C}$).

In the two subjects who were passively heated to $\sim 39^\circ\text{C}$, voluntary force production followed the same pattern of response during a sustained maximal handgrip contraction as was observed after their hyperthermic exercise trials.

DISCUSSION

The present study demonstrates that the ability to generate force during sustained MVCs is attenuated with hyperthermia and that impaired performance is highly associated with a reduction in the voluntary activation percentage. We observed that total muscle force during sustained MVCs is unaffected by hyperthermia and that brief MVCs can be repeated every 5 s without hyperthermia-induced attenuation of the force development. Thus the capacity of the skeletal muscles to generate force is not affected by hyperthermia, and impaired central activation seems to fully account for the reduction in maximal voluntary force development during prolonged isometric contractions. We speculate that the same mechanism may be involved in the fatigue that develops during prolonged dynamic exercise in the heat.

The present study clearly demonstrates that hyperthermia leads to a marked reduction in voluntary force development during a prolonged maximal isometric contraction, and the results indicate that central acti-

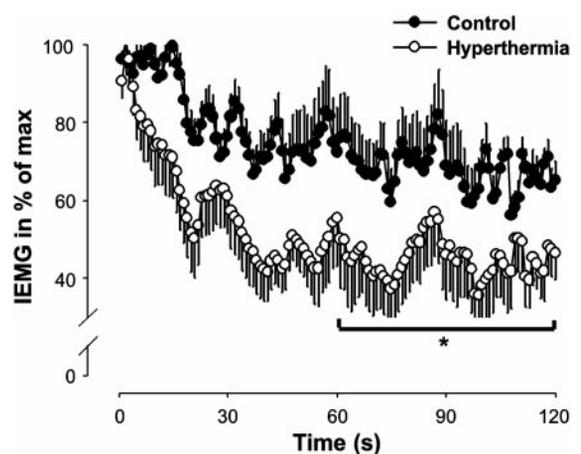


Fig. 4. Rectified integrated surface electromyography (iEMG) during 2 min of sustained MVC with the knee extensors during hyperthermia and control. Data are means \pm SE for 5 subjects. max, Maximum. *Average iEMG from 60 to 120 s in hyperthermia is significantly lower than control ($P < 0.05$).

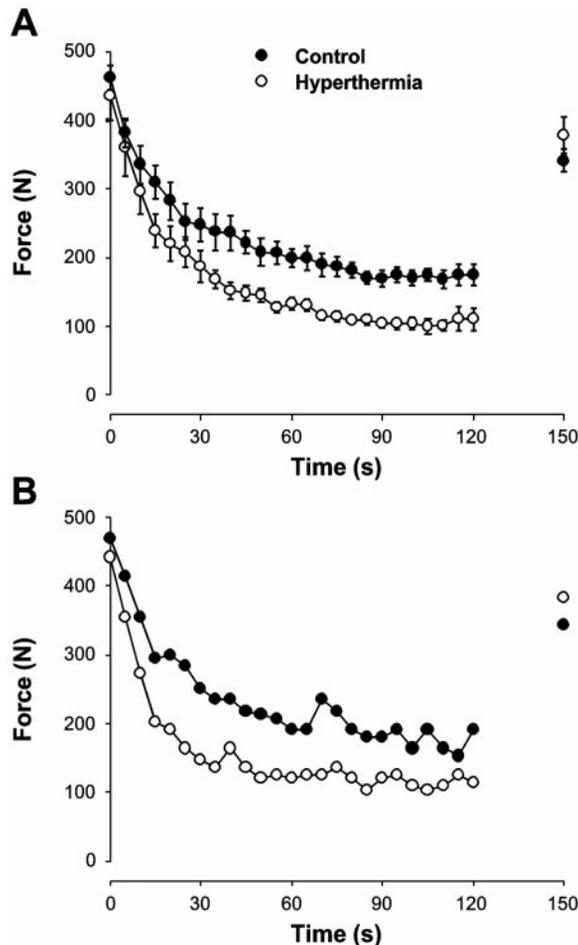


Fig. 5. A: changes in force during 2 min of sustained maximal handgrip contraction with or without exercise-induced hyperthermia. Data are means \pm SE for 8 subjects. B: changes in force during 2 min of sustained maximal handgrip contraction with or without passively induced hyperthermia (mean of 2 subjects).

vation failure plays a significant role in this reduction. In fact, total muscle force (MVC + force from EL) was unaffected by hyperthermia, and the reduced force during the last minute of the isometric contraction seems to be fully accounted for by the reduced voluntary activation of the muscles (54% in hyperthermia vs. 82% in control). The voluntary activation percentage is a measure of the degree of central activation. However, EL at rest only elicited a force equal to approximately one-third of MVC, reflecting an incomplete activation of the knee extensors during EL of the femoral nerve. It is therefore not certain whether all the α -motoneurons, which are "silent" or not firing at maximal rates during the MVC, are activated by the superimposed EL. Thus all the calculated voluntary activation percentages may be somewhat overestimated. Nevertheless, the voluntary activation percentages observed for the knee extensors in the present study during the control trial and before exercise are in accordance with the degree of voluntary activation that has been previously observed for this and other muscle groups during similar MVC protocols (1, 2, 4, 5, 17), although electrical nerve stimulation activated a greater fraction of the total

motor pool in some of the previous investigations. Furthermore, the calculated activation percentages of 82% during the last minute of the sustained knee extension in control and 54% in the hyperthermic trial are in good accordance with the EMG data. The attenuated voluntary activation with hyperthermia is thus also reflected in a reduction in the iEMG from 79% (of iEMG_{max}) in the control trial to 58% in the hyperthermic trial.

Reduced voluntary force development with hyperthermia during the prolonged MVCs was observed both for the leg and arm muscles. Therefore, the attenuated ability to voluntarily activate skeletal muscles seems to be independent of whether the muscles were active during the preceding cycle exercise in the heat. Together with the observation that, in two subjects, passively induced hyperthermia had the same effect on reducing handgrip strength during sustained MVC, the results support the idea that high body temperatures per se inhibit the ability to sustain motor activity. Brain temperature seems to be a good candidate as a major factor affecting motor activity (11), but influence from signals originating in internal organs, muscles, or other parts of the CNS cannot be ruled out. Temperature in the active muscles, on the other hand, does not seem to play a critical role because reduced maximal voluntary force during the hyperthermic trials was observed for both the isometric handgrip contraction and the prolonged knee extension despite significant differences in the active T_{mu} (38.7°C at the end of handgrip vs. 40.7°C for the knee extensors). Furthermore, muscle function and/or central activation percent was not affected in the protocol with repeated MVCs, although T_{mu} at the end of contraction 40 was close to 41°C. The detrimental effects of whole body hyperthermia and high T_{mu} on muscle metabolism are well documented (12, 23), but these factors seem to play only a minimal role in the development of fatigue during prolonged exercise in hot environments (16, 18, 22). The possibility that high temperatures have a detrimental effect on mitochondrial function is often mentioned, referring to the work by Brooks et al. (9). Furthermore, Willis et al. (25) found a 20% reduction in the ADP-to-oxygen ratio at 43°C compared with that at 37°C and suggested that high T_{mu} might compromise the properties of the inner mitochondrial membrane, resulting in increased nonspecific proton leakage. However, the transferability of the results from these in vitro measurements to in vivo situations is questionable. In humans exercising at submaximal work intensities, no differences in oxygen consumption are observed over a wide range of core temperatures and T_{mu} (~37°C to ~41°C) (16, 21), and it appears that hyperthermia-induced exhaustion in exercising humans occurs before mitochondrial respiration is perturbed and probably before other functions of the muscle cell are jeopardized. It could, however, be speculated that signals originating in the active muscles, secondary to the increased T_{mu} , are involved in some sort of feedback inhibition of the motor areas during the sustained contractions. The influence of

feedback from muscle metabolites to the CNS and subsequent alteration in central motor drive during the development of fatigue seems plausible (3, 17). Furthermore, the temperature centers in the hypothalamus integrate both efferent and afferent signals for the control of autonomic and behavioral thermoregulatory responses in animals and humans, and it seems possible that the hypothalamus influences both sensory areas responsible for the perception of effort and motor areas responsible for muscle activation (6, 7). Whether these factors interact or act independently is at present unsolved.

The observation that hyperthermia results in a reduced central activation during prolonged isometric contraction leads to the question whether "central fatigue" also is involved in the hyperthermia-induced fatigue that develops during prolonged dynamic exercise in the heat. We have recently observed that hyperthermia resulted in a marked reduction in cerebral blood-flow velocity during prolonged submaximal exercise (21), and it has also been established that alterations in the electroencephalogram are linearly related to increasing core temperature during cycle exercise in the heat (19). Both these observations and the present demonstration of increased central fatigue after exercise-induced hyperthermia point out that the cerebral function is substantially affected by hyperthermia during exercise. The present data clearly show that central fatigue can be observed after hyperthermia-induced exhaustion during exercise in the heat, but we cannot establish whether dynamic exercise (e.g., the cycle bout) is terminated because of this central fatigue. Full understanding of the functional interaction between the reduction in cerebral blood-flow velocity, the altered electrical activity, and the attenuated ability to sustain motor activation requires further investigation.

In conclusion, the present study clearly demonstrates that voluntary force development during prolonged MVCs is reduced with hyperthermia, and this reduction is highly associated with decreased central activation. Total muscle force is not different in the hyperthermic compared with the normothermic condition; therefore, hyperthermia-induced central fatigue seems to fully account for the attenuated voluntary force development.

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