Perceived exertion is associated with an altered brain activity during exercise with progressive hyperthermia

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Nybo, Lars, and Bodil Nielsen. Perceived exertion is associated with an altered brain activity during exercise with progressive hyperthermia. J Appl Physiol 91: 2017–2023, 2001.—The present study tested the hypothesis that perceived exertion during prolonged exercise in hot environments is associated with changes in cerebral electrical activity rather than changes in the electromyogram (EMG) of the exercising muscles. Therefore, electroencephalogram (EEG) in three positions (frontal, central, and occipital cortex), EMG, rating of perceived exertion (RPE), and core temperature were measured in 14 subjects during submaximal exercise in normal (18°C, control) and hot (40°C, hyperthermia) environments. RPE increased from 11 ± 1 units at 5 min to 20 ± 0 units at exhaustion (50 ± 3 min) in the trial with progressive hyperthermia, whereas exercise in the control trial was maintained with a stable core temperature for 1 h without exhausting the subjects. Altered EEG activity was observed in all electrode positions, and stepwise forward-regression analysis identified core temperature and a frequency index of the EEG over the frontal cortex as the best predictors of RPE. In contrast, there were no significant correlations between RPE and any of the measured EMG parameters (median spectral frequency, root mean square, or amplitude), and the EMG parameters were not different in hyperthermia compared with control. Thus hyperthermia does not seem to affect the activation pattern of the muscles. Rather, the linear correlation among core temperature, EEG frequency index, and RPE indicates that alterations in cerebral activity may be associated with the hyperthermia-induced development of fatigue during prolonged exercise in hot environments.

 electroencephalography; electromyography; core temperature

HEAT PRODUCTION DURING DYNAMIC exercise can elevate core temperature rapidly, and it seems that hyperthermia during prolonged exercise in hot environments is an independent cause of exhaustion (7, 10, 25). However, the mechanism(s) underlying hyperthermia-induced fatigue during prolonged, dynamic exercise in the heat is not well understood. Fatigue, defined as a loss of force-generating capacity or an increased difficulty in maintaining a required power output, may develop for a variety of reasons and occur at various sites along the pathway from the central nervous system to the contractile machinery of the muscles. Determinants of hyperthermia on muscle function and on metabolism have been observed, but these factors cannot explain the fatigue that develops during prolonged exercise in hot environments (4, 5, 9, 15, 20, 24). We have recently demonstrated that hyperthermia reduces voluntary force development during a sustained, maximal isometric contraction, and this impairment in performance could be explained by “central fatigue” (18). In the above-mentioned study, superimposed electrical stimulation of the femoral nerve was used to differentiate between the contribution of central and peripheral factors to the development of fatigue during sustained isometric contractions. The results revealed that hyperthermia did not affect the ability of the muscles to generate force. Instead, a markedly lower voluntary activation percentage during the hyperthermic trials indicated that the hyperthermia-induced fatigue was located within the central nervous system. Those results appear to support the idea that high core temperature may inhibit the cerebral ability to provide an adequate neural drive to the muscles, and this may be the explanation why both humans and rats fatigue when high body temperatures are reached (7, 10, 15, 25). The increasing difficulty to maintain power output during prolonged exercise with progressive hyperthermia is also reflected in the subjective rating of perceived exertion (RPE), which increases concurrently with the rise in core temperature (10, 16, 19). Furthermore, hyperthermia results in a marked reduction in cerebral blood flow velocity during prolonged, submaximal exercise (19), and alterations in the electroencephalogram (EEG) have been found to be linearly related to increasing core temperature during cycle exercise in the heat (16). Taken together, these results indicate that cerebral function is substantially affected by hyperthermia. However, the interaction among the hyperthermia-induced cerebral changes, the activation pattern of the muscles, and the development of fatigue during dynamic exercise has never been investigated. Therefore, the primary purpose of the present study

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was to elucidate whether or not hyperthermia-induced cerebral changes during dynamic exercise were related to an altered activation of the exercising muscles and, secondarily, to investigate whether the subjective RPE was correlated with changes in cerebral and/or muscular activity.

METHODS

Subjects. Age, body weight, height, and maximal oxygen consumption of the 14 healthy endurance-trained cyclists participating in the study were 25 ± 1 (SE) yr, 71 ± 2 kg, 181 ± 2 cm, and 65 ± 2 ml·kg⁻¹·min⁻¹, respectively. Maximal oxygen uptake was determined during an incremental exercise protocol on a cycle ergometer using a metabolic cart (model CPX/D, MedGraphics, St. Paul, MN). The 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 Frederiksborg (KF 01-135/00). All subjects had previously participated in experiments involving cycling in hot environments.

Experimental protocol. On two separate occasions, subjects cycled at ~60% of maximal oxygen consumption (power output 188 ± 7 W, 85 ± 1 rpm, counterbalanced order) in a climatic chamber. 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 either the point at which the subject volitionally stopped exercising or the point when power output could no longer be maintained. The subjects arrived at the laboratory ~1 h before the start of the experiment and rested in a thermoneutral room while the equipment was attached. The subjects then emptied their bladder, were weighed, and entered the climatic chamber. Here they were seated on the cycle ergometer (Monark Ergomedic 818; mounted with a triathlon handlebar to secure a steady working position) and remained in their racing position while resting measurements of heart rate (HR), esophageal temperature (Tes), and EEG were obtained. After the onset of exercise, HR, T es, EEG, electromyogram (EMG), and RPE [the subject rated his perceived effort on the Borg scale (3)] were recorded at 5, 10, 20, and 30 min and just before exhaustion or at 58–60 min in the control trial.

EMG and EEG measurements. Surface EMG signals were recorded from the right vastus lateralis 15 cm proximal to the superior border of the patella, with the use of a pair of EMG recording electrodes with an interelectrode distance of 3 cm (Neurilect electrodes, type 72001-J, Medicotest). The EMG signals were amplified (gain ×1,000), sampled at 1,000 Hz, band-pass filtered [3 Hz (−6 dB) to 500 Hz (−6 dB)] by using an IP511 alternating-current preamplifier (Astro-Med) and a CED 1401-plus analog/digital converter, and stored to a data-acquisition file. After additional high-pass filtering (at 10 Hz) to minimize movement artifacts, root mean square (RMS), median, and mean power frequency were calculated as an average of data obtained during 10 consecutive pedal cycles. Furthermore, EMG data during the same 10 pedal cycles were full-wave rectified and smoothed by using a fourth-order Butterworth low-pass filter with a cutoff frequency of 6 Hz. The amplitude of the smoothed, rectified EMG was expressed as a percentage of maximum EMG, which was measured pre- and postexercise as an average of 1 s of smoothed, rectified EMG obtained during a maximal knee extension. Because of technical problems, EMG measurements from five subjects were rejected, and the EMG data, therefore, only represent nine subjects.

EEG Beckmann Ag-AgCl electrodes were affixed to the scalp with conductive electrode paste (Bentonite paste, Dantec) and secured with Omnifix stretch tape. The electrodes were positioned 1 cm in front of Oz, at F3, and at O2 [according to the ten-twenty system (12)] chosen to represent the motor areas of the legs, the prefrontal cortex, and the visual cortex, respectively. A paired mastoid reference was used. Pilot studies showed that signals from the right and left hemisphere had similar power spectrum responses to exercise and hyperthermia, thus rendering the basis for recording EEG from just one hemisphere. Surface potentials were amplified (gain ×10,000) and sampled at 1,000 Hz in each electrode using a CED 1401-plus analog/digital converter. EEG measurements in three subjects were rejected because of movement-related artifacts. The data analysis of the EEG was similar to the method previously described by Nielsen et al. (16). In short, fast-Fourier transformation of the EEG was made with a CED software package (Spike2) using steps of 0.2 Hz for the power spectrum. The areas of the power spectrum between 8 and 13 Hz (Aα) and between 13 and 30 Hz (Aβ) were calculated as quantitative indexes of the activity in the α- and β-bands, respectively. Furthermore, an Aα/Aβ index was calculated by dividing Aα by Aβ, and the index was expressed as a percentage of the preexercise resting value obtained on the trial day, thus reducing interindividual and day-to-day variations. The described analysis allows us to focus on possible changes in relative amplitudes between α- and β-waves occurring during exercise, rather than on changes in absolute EEG power amplitudes.

Middle cerebral artery (MCA) mean blood velocity (V mean) was measured with ultrasound Doppler sonography in 8 of the 14 subjects in a parallel study, with an identical exercise protocol (see Ref. 19 for further details).

Core temperature and degree of dehydration. 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 the subject’s standing height.

HR was measured with a Polar HR recorder (Polar Electro).

To avoid differences in the degree of dehydration between trials, 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 difference between pre- and postexercise body weights) was thereby restricted to 0.9 ± 0.2% in the hyperthermic trial and 0.8 ± 0.1% in the control trial [P was not significant (NS)]. Body weight was determined on a platform scale (model 1–10 kg, Ohaus).

Statistical analysis. Two-way (time-by-trial) repeated-measures ANOVA was performed to evaluate differences between and within trials. After a significant F test, pairwise differences were identified using Tukey’s significance (honestly significant difference) post hoc procedure. Furthermore, simple linear regression was used to test the strength of the association between variables. Stepwise forward-regression analysis was also used to test the strength of the association between RPE as the dependent variable and HR, core temperature, Aα/Aβ index of the EEG, RMS, and median spectral frequency of the EMG as independent variables. All regression analyses are made on the basis of the average values from subjects when all parameters (included in the analysis) are present. Data are presented as means ± SE, unless otherwise indicated.

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RESULTS

The core temperature increased continuously during the exercise period in the uncompensable hot environment and reached a peak value of 40.0 ± 0.1°C at exhaustion after 50 ± 3 min of exercise. HR increased from 140 ± 4 beats/min at 5 min to 179 ± 3 beats/min at exhaustion, and in the same period RPE increased from 11 ± 1 to 20 ± 0 units (both P < 0.001). In the control trial, exercise was maintained for 1 h with only a modest increase in RPE from 10 ± 1 at the beginning of exercise to 12 ± 1 at 60 min and with Tes and HR stabilized at 38.0 ± 0.1°C and 140 ± 4 beats/min, respectively, after ~20 min of exercise.

EMG and EEG measurements. A representative example of raw and smoothed rectified EMG obtained at the beginning and at the end of a hyperthermic exercise trial is shown in Fig. 1. The amplitudes of the smoothed rectified EMG were in the range of 40–60% of maximum EMG in all subjects, and the average amplitude of 10 pedal cycles remained unchanged during the exercise period, both in the hyperthermic trial (45 ± 6% at 5 min vs. 48 ± 5% at 50 ± 3 min; P = NS) and in the control trial (48 ± 4% at 5 min vs. 47 ± 3% at 60 min; P = NS). RMS was also similar in the hyperthermic and normothermic condition and remained constant over time in both trials. Furthermore, neither mean nor median spectral frequency of the EMG changed significantly over time within either of the two exercise conditions, and there were no significant differences between hyperthermia and control.

In all three EEG electrode positions, the Aa/Aβ index increased significantly during the hyperthermic trial. During the control trial, Aa/Aβ indexes only increased insignificantly (P = NS), and the Aa/Aβ indexes were significantly higher in hyperthermia compared with control at the end of exercise (see Fig. 2). The increase in the Aa/Aβ indexes during the hyperthermic trial was caused by a significant reduction in the Aβ by ~50% (P < 0.05), whereas the Aa remained unchanged. All subjects had a similar pattern of response; however, large interindividual variations in the magnitude of the changes in the Aa/Aβ indexes were observed. When the Aa/Aβ indexes were plotted against Tes, it appeared that, in all three electrode positions, there was a good linear relationship between the changes in Aa/Aβ index and the changes in core temperature (r = 0.94–0.95; P < 0.001; see Fig. 3).

RPE. RPE was plotted against the changes in EEG, EMG, HR, Tes, and MCA Vmean (see Fig. 4), and stepwise forward-regression analysis identified core temperature and the Aa/Aβ index of F3 as the best predictors of RPE. Simple linear regression analysis revealed that RPE also was strongly associated with the changes in MCA Vmean (r = 0.98), HR (r = 0.97), and Aa/Aβ indexes of C2 and O2 (r = 0.95 and 0.94, respectively); however, these variables did not increase the predicting power of the F3 Aa/Aβ index and Tes, because they were all significantly correlated with Tes. A linear association between the three Aa/Aβ indexes and MCA Vmean was also observed (r = 0.93–0.97; P < 0.001; n = 7). In contrast, there were no significant correlations between RPE and any of the measured muscle parameters (see Fig. 4, E and F), and the EEG frequency changes were not correlated with any of the measured muscle parameters.

DISCUSSION

In the present study, we observed that, during prolonged exercise with progressive hyperthermia, EMG
amplitudes and frequencies of the exercising muscles remained unaltered, whereas a calculated frequency index of the EEG \( A_\alpha/A_\beta \) index in all three electrode positions) increased linearly with increasing core temperature. Stepwise forward-regression analysis indicated that the \( A_\alpha/A_\beta \) index of F3 (prefrontal cortex) and core temperature were the best predictors of the subjects' RPE, whereas there was no correlation between RPE and any of the measured EMG parameters.

The unaltered RMS of the raw EMG and amplitudes of the smoothed rectified EMG indicate that the level of muscular activation was constant throughout the exercise.
exercise periods in both the control and hyperthermic trial. Unchanged EMG amplitude and RMS during moderate-intensity cycling in a thermoneutral environment is in accordance with previous findings (11, 22), and the present results demonstrate that hyperthermia does not affect the electrical activation pattern of the active skeletal muscles. Corroborating this observation, Faiti et al. (6) recently observed that EMG amplitude measured during running was unchanged during an exercise bout with progressively developing dehydration and hyperthermia. If hyperthermia had resulted in fatigue-induced changes in motor unit recruitment and/or discharge rates, it would be expected that, similar to high-intensity cycle exercise, there would have been a progressive increase in the RMS and an increase in the amplitude of the smoothed rectified EMG (11, 22, 23), as well as a shift in the median spectral frequency (8, 14). The absence of hyperthermia-induced signs of muscular fatigue during submaximal exercise is in accordance with the results from our recent study (18), in which maximal force of the knee extensors was evaluated immediately after

Fig. 4. Rating of perceived exertion (RPE) plotted against the $A_{\alpha}/A_{\beta}$ index measured at $F_3$ ($n = 11$; $A$); core temperature ($n = 14$; $B$); heart rate ($n = 14$; $C$); middle cerebral artery (MCA) mean blood velocity ($V_{\text{mean}}$) ($n = 8$; $D$); root mean square (RMS) of the EMG from vastus lateralis as a percentage of the 5-min control trial value ($n = 9$; $E$); and median spectral frequency of the EMG from vastus lateralis ($n = 9$; $F$) measured during prolonged, submaximal exercise with hyperthermia ($\bullet$) and during control (○). Values are means for 11 subjects. Solid line, line of best fit. NS, not significant.
cycle trials with or without hyperthermia. In that study, we used superimposed electrical stimulation of the femoral nerve to differentiate between the central and peripheral factors contributing to the development of fatigue during prolonged, maximal voluntary isometric contractions. The results revealed that hyperthermia did not affect the ability of the muscles to generate force. Instead, central fatigue seemed to be the cause of the attenuated performance during the prolonged, maximal voluntary isometric contractions (18). Increased difficulty to retain power output during the hyperthermic cycle trial is reflected in the subjects' RPE, and the identification of core temperature and F3 $A_v/A_b$ index as the best predictors of RPE may support the idea that the EEG frequency shift reflects decreased arousal and impeded ability of the brain to sustain motor activity (16). However, it is also possible that the rise in the $A_v/A_b$ index simply reflects the sensation of the increasing temperature or that it responds to other signals arising secondarily to the increase in core temperature. At rest, hyperventilation-induced reductions in cerebral blood velocity are associated with a slowing of the EEG activity (13). Considering the high association between MCA $V_{mean}$ and the $A_v/A_b$ indexes in all three electrode positions, it seems plausible that, to some extent, the frequency shift during the hyperthermic trial is a consequence of the hyperventilation-induced reduction in the cerebral blood flow (see Ref. 19 for further explanation). Furthermore, this may be the reason why the frequency shift was observed in all EEG positions (i.e., the frontal, central, and occipital cortices), and it could explain why the electrical activity in all three cortical areas was similarly affected by hyperthermia. However, stepwise forward-regression analysis has limitations, and although it provides a general description of the impact that core temperature and $A_v/A_b$ indexes have on RPE during exercise in the heat, it cannot identify the source of the altered brain activity, and the causal relationship between the altered cerebral circulation, the changed electrical activity, and the development of fatigue remains at present unsolved. Considering the close association between RPE and the $F_3 A_v/A_b$ index, it is tempting to suggest that altered activity in the prefrontal cortex might be a contributing mechanism by which hyperthermia affects the ability to sustain motor activity during prolonged exercise in the heat. The prefrontal cortex is most likely involved in the initiation of volitional movements (17, 21), and we have recently demonstrated that hyperthermia reduces the voluntary activation percentage during a sustained, maximal isometric contraction (18). However, further investigations are required to elucidate the relationship among hyperthermia, altered cortical activity, and central fatigue. The mechanism by which hyperthermia might act on brain activity could be far more complex, involving afferent signals arising in the temperature centers in hypothalamus (1, 2) or signals sensing cardiovascular stressing, muscle and skin temperature, etc. However, taken together with our previous demonstration of hyperthermia-induced central fatigue, the present results appear to support the hypothesis that altered cerebral function rather than muscular changes is associated with the development of fatigue during prolonged exercise in the heat.

In conclusion, the present study demonstrates that subjectively perceived exertion is highly associated with increases in core temperature and frequency changes of the EEG obtained over the prefrontal cortex. In contrast, there were no correlations between RPE and any of the measured EMG parameters. The unaltered RMS, median frequency, and amplitude of the smoothed EMG during both the control and hyperthermic exercise trial indicate that hyperthermia did not result in fatigue-induced changes in motor unit recruitment and/or discharge rates. The results appear to support the idea that altered activity within the central nervous system rather than changed muscular activity is involved in the development of fatigue during prolonged exercise in hot environments.

REFERENCES


