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Reduced neuromuscular activity and force generation during prolonged cycling

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St Clair Gibson, A., E. J. Schabort, and T. D. Noakes. Reduced neuromuscular activity and force generation during prolonged cycling. *Am J Physiol Regulatory Integrative Comp Physiol* 281: R187–R196, 2001.—We examined neuromuscular activity during stochastic (variable intensity) 100-km cycling time trials (TT) and the effect of dietary carbohydrate manipulation. Seven endurance-trained cyclists performed two 100-km TT that included five 1-km and four 4-km high-intensity epochs (HIE) during which power output, electromyogram (EMG), and muscle glycogen data were analyzed. The mean power output of the 4-km HIE decreased significantly throughout the trial from 319 ± 48 W for the first 4-km HIE to 278 ± 39 W for the last 4-km HIE ($P < 0.01$). The mean integrated EMG (IEMG) activity during the first 4-km HIE was 16.4 ± 9.8% of the value attained during the pretrial maximal voluntary contraction (MVC). IEMG decreased significantly throughout the trial, reaching 11.1 ± 5.6% during the last 4-km HIE ($P < 0.01$). The study establishes that neuromuscular activity in peripheral skeletal muscle falls parallel with reduction in power output during bouts of high-intensity exercise. These changes occurred when <20% of available muscle was recruited and suggest the presence of a central neural governor that reduces the active muscle recruited during prolonged exercise.

electromyogram; muscle; power output; carbohydrate; glycogen

PREVIOUS STUDIES OF THE FATIGUE process during endurance cycling exercise have examined the ability of fuel substrates such as carbohydrate (CHO) or fat to attenuate these fatigue processes (5, 11). These studies have used either an open-loop design in which there is no fixed endpoint so that subjects terminate exercise of their own volition or a closed-loop design in which either the distance or duration of exercise is predetermined. They have shown that the ingestion of CHO before or during the trial increases the time to fatigue during the open-loop design or decreases the time required to complete the trial during closed-loop design experiments (7, 8, 23, 24, 48).

The mechanisms for this enhancement are unknown but are generally assumed to result from changes in skeletal muscle metabolism (30), which delay the onset of muscle glycogen depletion, or as a result of the prevention or reversal of hypoglycemia (10). But it remains unclear why altered skeletal muscle metabolism induced by muscle glycogen depletion should induce fatigue. Thus Fitts (15) concludes that muscle glycogen depletion has not been identified as the exclusive cause of fatigue during prolonged exercise. Similarly, using an intermittent closed-loop sprint model, Bangsbo et al. (2) showed that decrements in skeletal muscle power output were not tightly correlated with any measured metabolic changes. They suggested that other factors, in particular neural control mechanisms, could cause fatigue during prolonged closed-loop exercise.

Yet there are relatively few studies of the contribution of neural factors to fatigue during prolonged, submaximal exercise. Rather, research investigating any possible neural basis for fatigue has used surface or invasive electromyographic (EMG) techniques during submaximal or maximal isometric or isokinetic contractions.

It is usually found that during submaximal isometric or isokinetic contractions, the subject is able to increase the motor command to counteract the reduction of force output thought to be due to metabolic changes in the recruited but fatiguing muscle fibers (12, 19, 21). Peripheral fatigue of this type is thus defined as a decrease in the force-generating capacity of the skeletal muscle due to altered crossbridge cycle activity, to excitation/contraction coupling failure, or to failure of the action potential propagation in the presence of unchanged or increasing neural drive (43).

Thus, if exercise-induced metabolic factors, in particular muscle glycogen depletion, cause peripheral fatigue during prolonged exercise, there may be a progressive increase in neural efferent drive to the active skeletal muscles. Increased neural efferent drive would initiate the recruitment of additional muscle fibers to offset any decrement in force production by the substrate-depleted, fatiguing muscle fibers.

In contrast, central fatigue is defined as a reduction in efferent motor command to the active muscles resulting in a decline in force or tension development (13). Although some studies have shown that maximal

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voluntary contraction (MVC) and integrated EMG (IEMG) are decreased by ~30% after a 42-km marathon foot race (33), no study has examined the changes in central neural efferent drive during exhaustive endurance exercise or the effect of CHO manipulation on this central command.

The aim of this study was therefore to examine neuromuscular activity and force output in the active skeletal muscle and the effect of CHO manipulation on this neuromuscular activity during prolonged cycling involving a stochastic closed-loop exercise design that mimicked normal racing conditions. In addition, the study was adequately placebo controlled and evaluated endurance-trained individuals performing at their own volition and subjective maximal capacity.

METHODS

Aspects of this study have been published in a companion paper that evaluated metabolic changes and performance during these trials (9).

Subjects. Seven endurance-trained male cyclists, accustomed to riding for prolonged periods (3–4 h), participated in this study. At the time of the investigation, each subject was riding between 250 and 500 km/wk. Before commencement of the trial, all subjects were informed that the purpose of the investigation was to test two different sports supplements designed for race preparation. The study was approved by the Ethics and Research Committee of the University of Cape Town Faculty of Health Sciences. Subjects gave written informed consent in accordance with the guidelines outlined by the American College of Sports Medicine (1). The subject characteristics are shown in Table 1.

Preliminary testing. On their first visit to the laboratory, subjects were tested for peak oxygen uptake ($V_{O_{2peak}}$) and peak sustained power output (PPO) on their own bicycles, which were mounted on the Kingcycle ergometer (described below). After a 5- to 10-min warm-up at a self-selected intensity, the test commenced at a workload of 200 W; the load was then increased by 20 W/min until the subject could no longer maintain the required power output. The subject's PPO was taken as the highest average power produced during any 60-s period of the exercise test. During these incremental tests to exhaustion, subjects were requested to remain in a seated position to prevent whole limb recruitment pattern changes initiated by changing from a seated to a standing cycling position.

Throughout the maximal test, subjects wore a face mask attached to an Oxygen Alpha automated gas analyzer (Jaguar, The Netherlands). Before each test, the gas analyzer was calibrated using a Hans Rudolf 5530 3-liter syringe and a 5% $C0_2$-95% N_2 gas mixture. Analyzer outputs were processed by an IBM computer that calculated minute ventilation, oxygen consumption, and rates of carbon dioxide production using conventional equations. Each subject's $V_{O_{2peak}}$ was taken as the highest oxygen uptake measured during any 60-s period of the test.

Table 1. *Subject characteristics*

	Mean ± SD	Range
Age, yr	28 ± 4.5	22–37
Mass, kg	72.1 ± 6.7	62–81
$V_{O_{2peak}}$		
ml·kg^{-1}·min^{-1}	63.9 ± 4.7	58.0–70.2
l/min	4.6 ± 0.6	3.5–5.2
Peak power output, W	411 ± 52	313–464
P:W, W/kg	5.7 ± 0.5	5.1–6.2

$V_{O_{2peak}}$, peak oxygen uptake; P:W, power-to-weight ratio (n = 7).

After completing the maximal test, subjects performed a familiarization ride on the Kingcycle (Kingcycle, High Wycombe, UK) ergometry system, which allows cyclists to ride on their own racing bicycles in the laboratory. After removing the front wheel, the subject's bicycle is attached to the ergometer by the front fork and supported by an adjustable pillar under the bottom bracket. The bottom bracket support is used to position the rolling resistance of the rear wheel correctly on the air-braked flywheel. The ergometer was calibrated as previously reported in detail (35). The familiarization ride consisted of the first 25 km of the 100-km time trial (TT; described below). Subjects were requested to "ride as fast as possible" and were not given any feedback other than their elapsed distance. Before the trial, all subjects were told that the subject with the fastest time in either trial would receive a financial reward at the end of the trial. This was done to motivate subjects to complete the trial in the fastest time possible.

Dietary intervention. Individual food plans were constructed for each subject based on body mass (BM) and food preferences. Each subject was supplied with his food intake for the 72-h period before each trial. Menu plans were provided in written form, and the food assigned to each meal was individually prepared and packaged so that the need for further preparation by subjects was minimized. Subjects were required to record their actual food and fluid intake in dietary logs to account for any portion of meals left unconsumed or any additional intake. Although some food items differed between subjects, each cyclist received identical breakfast, lunch, and dinner menus for both of his trials, with the CHO intake of these meals designed to provide 6 g·kg BM^{-1}·day^{-1}. Snacks were then provided for each day to provide the dietary source of differentiation between TT. For the CHO-loading diet, subjects received 1,200 ml of water each day and a number of sports bars (Gijima, Sasko, Paarl, South Africa) calculated to provide an additional 3 g CHO·kg^{-1}·BM^{-1}. Each sports bar had a composition of 27 g CHO, 6.5 g fat, and 2.7 g protein. For the placebo trial, daily snacks were provided in the form of 1,200 ml of an artificially sweetened, low-calorie drink that was described to the subjects as a "CHO-loading" drink. Total energy (4,149 ± 315 vs. 2,726 ± 202 kcal; $P < 0.05$), protein (110 ± 7 vs. 88 ± 6 g; $P < 0.05$), and fat (126 ± 9 vs. 79 ± 7 g; $P < 0.05$) intake were significantly higher during CHO compared with placebo trials.

Experimental trials. Each subject completed a random crossover design of two experimental TT separated by 7 days. Subjects performed their TT at the same time of day under standard laboratory conditions (~20°C, 55% relative humidity). Subjects were requested to perform the same type of training for the duration of the experimental period and to refrain from heavy physical exercise on the day preceding a TT. Training diaries were kept to assess compliance to this condition. On the morning of an experiment, subjects reported to the laboratory between 0700 and 0800, 12–14 h after an overnight fast. At this time, the Kingcycle ergometer was calibrated, after which subjects consumed a standard breakfast providing 2 g of CHO/kg BM. After resting quietly for 105 min, a preexercise muscle biopsy sample was taken from the vastus lateralis of the right leg according to the technique of Bergstrom (4) as modified by Evans et al. (14). After this procedure, the subject mounted his bicycle and began a 5-min self-paced warm-up.

Exactly 2 h after the subjects had consumed breakfast, they commenced the 100-km TT. To mimic the stochastic nature (variable nature) of cycle road races (36), the TT included a series of high-intensity epochs (HIE): five 1-km HIE after 10, 32, 52, 72, and 99 km, as well as four 4-km HIE after 20, 40, 60, and 80 km. Just before commencement of these sprints, an investigator gave a distance countdown and instructed the cyclist to complete the sprint in the fastest time possible as soon as he reached the specific distance at which the sprint started. Subjects viewed a diagram of the "course profile," which graphically illustrated where the 1- and 4-km HIE occurred before and during each ride. Instantaneous power output was recorded at each 500-m split of both the 1- and 4-km HIE to provide an estimate of the average power output for that HIE. Subjects were instructed to complete the total distance as well as the HIE, in "the fastest time possible." The only feedback given to subjects during TT was their elapsed distance and heart rate (HR). Subjects were not informed of their total time or their times for the HIE until completion of the experiment. Throughout each TT, power output, speed, and elapsed time were monitored continuously and stored for later analysis. HR was recorded using a SportTester HR monitor (Polar Electro, Kempele, Finland). We previously reported that the between-test correlation for the time taken by eight well-trained cyclists and triathletes to complete this protocol was 0.93 [95% confidence interval (CI), 0.79–0.98], and the within-cyclist coefficient of variation was 1.7% (95% CI, 1.1–2.5%) (39).

During the 100-km ride, subjects ingested a 7 g/100 ml glucose polymer solution at a rate of 15 ml·kgBM^{-1}·h^{-1}. This drinking regimen was intended to replace ~80% of sweat loss (32) while providing CHO at ~ 1 g·kg BM^{-1}·min^{-1}, the maximum rate at which muscle can oxidize exogenous glucose (22). A fan was positioned to cool the subjects during their TT. The average sweat loss was estimated as the average fluid intake plus weight change over the trial of all seven subjects. Immediately on completion of the TT, a second biopsy was taken from the same leg at a distance 4 cm distal to the first incision. No metabolic measurements were taken during the trials, as the TT were performance trials and it was felt that interfering with the athletes during the trials would impair their performance by distracting their concentration.

Isometric testing. Before the start of each cyclist's TT, his quadriceps muscle strength was tested on a Kin-Com isokinetic dynamometer (Chattanooga Group). The knee extensors were tested isometrically at a knee angle of 60°, with the reference point being full knee extension. Each subject performed four submaximal familiarization 5-s isometric knee extensions at ~50% of maximal effort, two 5-s isometric knee extensions at ~70% of maximal effort, and two 5-s isometric knee extensions at ~90% of maximal effort. Subsequent to this familiarization and warm-up protocol, subjects performed four maximal 5-s second isometric contractions. The MVC attained in these four trials was used for subsequent analysis. Subjects were directed to begin maximal effort immediately and not "save" effort for the final seconds of the test. Subjects were verbally encouraged during the test to exert maximum effort.

EMG testing. Before maximal isometric strength testing on the Kin-Com isokinetic dynamometer, EMG electrodes with a bandwidth of 20–500 Hz and sensitivity of <0.08 μV were attached to the "belly" of the rectus femoris muscle. The skin overlying the vastus lateralis muscle was carefully prepared. Hair was removed by shaving, the outer layer of epidermal cells were abraded, and oil and dirt were removed from the skin with an alcohol swab. Triode electrodes (Thought Technology Triode, MIEP01–00, Montreal, Canada) were placed on the muscle site as described above and linked via a fiber-optic cable to the Flexcomp/DSP EMG apparatus (Thought Technology) and host computer. A 50-Hz line filter was applied to the EMG data to prevent interference from electrical sources. Each test was sampled at 1,984 Hz for the duration of the two TT and isometric tests, thus yielding raw signals.

Five seconds of raw data were collected at the midpoint of each 1-km HIE (10.5, 32.5, 52.5, 72.5, and 99.5 km), 4-km HIE (22, 42, 62, and 82 km), and at three nonsprint distances (5, 55, and 95 km) during each TT.

The raw EMG signals were full-wave rectified, the movement artifact was removed using a high-pass second-order Butterworth filter with a cut-off frequency of 15 Hz, then smoothed with a low-pass second-order Butterworth filter with a cut-off frequency of 5 Hz. This was performed using MATLAB gait-analysis software. This IEMG data was used for subsequent analysis.

The frequency spectrum of each HIE of the raw EMG data was analyzed using a fast Fourier transformation algorithm. The frequency spectrum analysis was restricted to frequencies in the range 5–500 Hz, as the EMG signal content outside of this range consists mostly of noise. The frequency spectrum from each epoch of data was compared with that derived from the MVC, and the amount of spectral compression was estimated. This was performed using the technique described by Lowery et al. (28) as a modification of the work of Lo Conte and Merletti (27) and Merletti and Lo Conte (31). The spectrum of the raw signal of each epoch was obtained, and the normalized cumulative power at each frequency was calculated. The shift in each percentile frequency (i.e., at 0, 50, 100% of the total cumulative data) was examined. The percentile shift was then estimated by calculating the mean shift in all percentile frequencies throughout the midfrequency range (i.e.. 5–500 Hz). This method has been suggested as a more accurate estimate of spectral compression than median frequency analysis, which uses the value of a single (50th) percentile frequency only (27, 28, 31). This change in mean percentile frequency shift (MPFS) data was used for subsequent analysis.

All EMG data were normalized by dividing the value at each time point during the cycle trial by the EMG value obtained during the MVC performed before the start of each TT. IEMG and MPFS data were therefore expressed as a percentage of this MVC data.

Analytic techniques. Muscle samples were subsequently freeze-dried; dissected free of blood, connective tissue, and fat; and weighed. Muscle glycogen content was determined after acid hydrolysis by a hexokinase method (25). The coefficient of variation for this assay in this laboratory is <5% for duplicate glycogen assays of a single piece of muscle and <7% for assays of the glycogen content of separate pieces of the same muscle biopsy sample (22).

Statistical analyses. Data from both TT were first combined and analyzed, and, subsequent to this, data were also analyzed to assess differences between the CHO and placebo trials. The EMG and force data are also expressed as normalized data, as described above. Differences in the HIE times and HIE power outputs, IEMG and MPFS data, and glycogen concentrations were examined using repeated-measures ANOVA, whereas glycogen use and total 100-km time and total power output were compared using Student's *t*-tests. All data are reported as means ± SD, and all calculations were performed using Statistica for Windows (Version 6, Statsoft, Tulsa, OK).

RESULTS

The mean $V_{O_{2peak}}$ value for the subjects was 63.9 ± 4.7 ml·kg^{-1}·min^{-1}, and PPO was 411 ± 52 W (Table 1). Their maximal force output during the pretest MVC was 703 ± 119 N. The mean power output during the 100-km TT and the time taken for completion were 256 ± 39 W and 148.27 ± 10.11 min, respectively.

The mean power output, IEMG, time taken, and MPFS for the five 1-km HIE for the group are shown in Fig. 1. The mean power output for the first 1-km HIE decreased significantly throughout the trial from 379 ± 52 to 300 ± 63 W for the last 1-km HIE ($P < 0.01$). The mean IEMG activity during the first 1-km HIE was 19.6 ± 12.8% of the value attained during the pretrial MVC. IEMG decreased significantly from this initial value throughout the trial to 11.34 ± 5.62% attained during the last HIE ($P < 0.05$). Time taken for each 1-km HIE increased significantly from 1.22 ± 0.07 for the first HIE to 1.36 ± 0.11 min for the last HIE ($P < 0.01$). MPFS for the first 1-km HIE compared with the pretest MVC was 84.8 ± 9.3%. MPFS increased significantly throughout the test and the value for the last HIE was 98.0 ± 14.4% ($P < 0.01$).

The mean power output, IEMG, time taken, and MPFS for the four 4-km HIE for the group are shown in Fig. 2. The mean power output decreased significantly throughout the trial from 319 ± 48 W for the first 4-km HIE to 278 ± 39 W for the last HIE ($P < 0.01$). The mean IEMG activity during the first 4-km HIE was 16.4 ± 9.8% of the value attained during the pretrial MVC. IEMG decreased significantly from this initial value throughout the trial to 11.1 ± 5.6% during the last HIE ($P < 0.01$). Time taken for each 4-km HIE increased significantly from 5.37 ± 0.32 for the first 4-km HIE to 5.67 ± 0.11 min for the last HIE ($P < 0.01$). MPFS for the first 4-km HIE compared with the pretest MVC was 91.3 ± 9.3%. Although MPFS increased throughout the TT, with the value for the last HIE being 95.7 ± 14.7%, this increase was not significant.

The HR changes during the three self-paced, five 1-km HIE, and four 4-km HIE for both TT are shown in Fig. 3. The HR during self-paced cycling increased significantly from 152 ± 11 beats/min after 5 km to 164 ± 12 beats/min after 95 km ($P < 0.01$). The HR during the 4-km HIE was maintained at 175 ± 8 beats/min for the first 4-km HIE to 175 ± 9 beats/min for the last 4-km HIE. The HR during the 1-km HIE increased from 172 ± 10 beats/min after the first 1-km HIE to 175 ± 9 beats/min after the last 1-km HIE. None of these differences was statistically significant.

With regard to the effects of CHO manipulation, there were no significant differences in the force output generated during the pretest MVC between CHO (720 ± 158 N) and placebo (684 ± 72 N) trials. Nor were there significant differences in the decrease in power output and IEMG between CHO and placebo trials during either the 1-km HIE (Fig. 4) or 4-km HIE (Fig. 5). There were no significant differences in the increase in time taken and PFS between CHO and placebo trials during either the 1- or 4-km HIE. There were also no significant differences in HR changes between CHO and placebo groups during either the self-paced trial, 1-km HIE, or 4-km HIE.

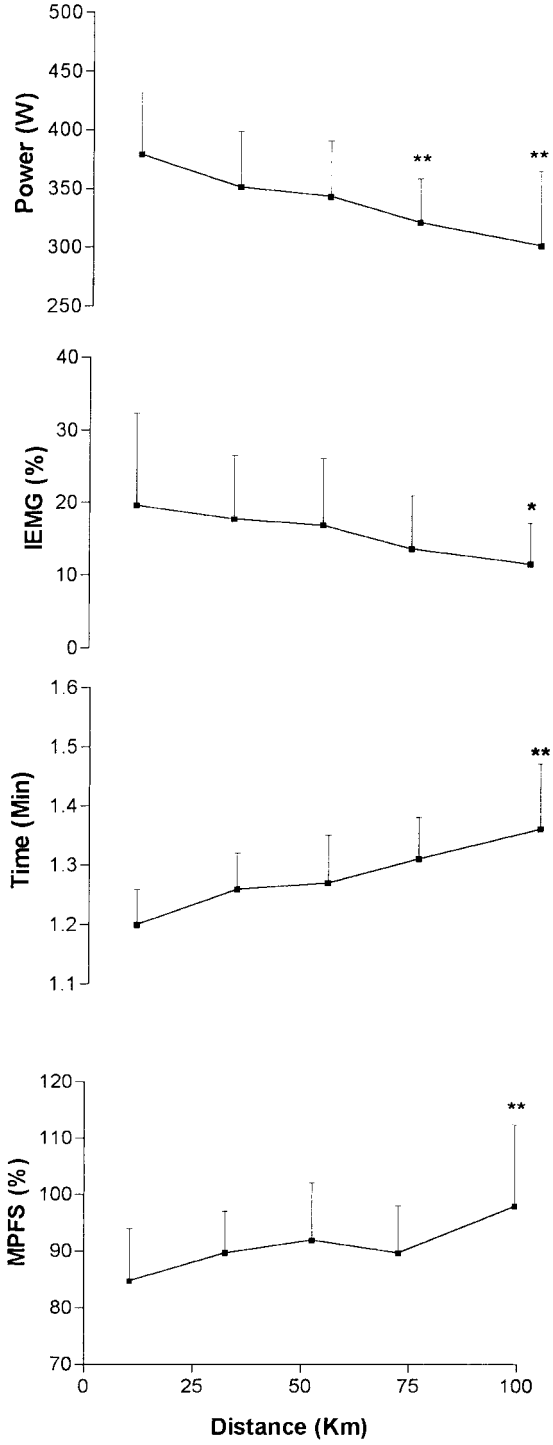

Fig. 1. Power output (W), integrated electromyogram (IEMG; %), time (min), and mean percentile frequency shift (MPFS; %) changes for the entire group during the 1-km high-intensity epoch (HIE). *$P < 0.05$, time *point 1* vs. time *point 5* IEMG; **$P < 0.01$, time *point 1* vs. time *points 4* and *5*; time, power, and MPFS.

As reported previously (8), the dietary CHO intake was significantly higher when subjects were CHO

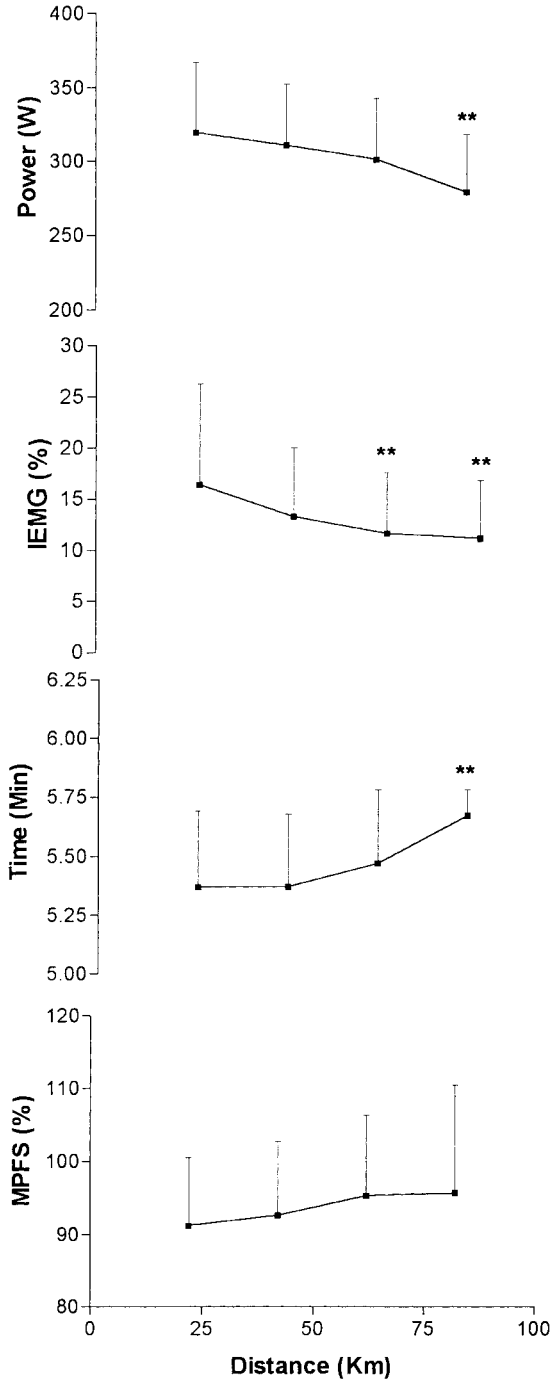

Fig. 2. Power output (W), IEMG (%), time (min), and MPFS (%) changes for the entire group during the 4-km HIE. $^{**}P < 0.01$ time *points 1* vs. *3* and *4*; IEMG, power, MPFS.

loaded (9.0 ± 0.3 g/kg BM) compared with the placebo diet (5.8 ± 0.2 g/kg BM; $P < 0.05$). The preexercise muscle glycogen concentrations were significantly higher in the CHO trial (572 ± 107 mmol/kg dry wt) compared with the placebo trial (485 ± 128 mmol/kg dry wt; $P < 0.05$). There were also no significant differences in postexercise muscle glycogen values between CHO (96 ± 63 mmol/kg dry wt) and placebo (55 ± 28 mmol/kg dry wt) trials (Fig. 6). There were no significant differences in glycogen use during the TT between CHO (476 ± 66.5 mmol/kg dry wt) and placebo (431 ± 116 mmol/kg dry wt) trials, as previously reported (8).

A representative illustration of normalized IEMG activity of rectus femoris activity during 1-km HIE for one subject is shown in Fig. 7. No obvious EMG pattern changes were detected during cycling activity during either TT as subjects fatigued.

DISCUSSION

In this study, we report two unexpected findings. First, we found that IEMG activity declined parallel with decreases in power output and increases in the time taken to complete repeat 1- and 4-km high-intensity exercise bouts during a 100-km cycling TT. Second, we found that these changes occurred although only ~20% or less of the available muscle activated during an MVC was recruited at any time during the cycling TT.

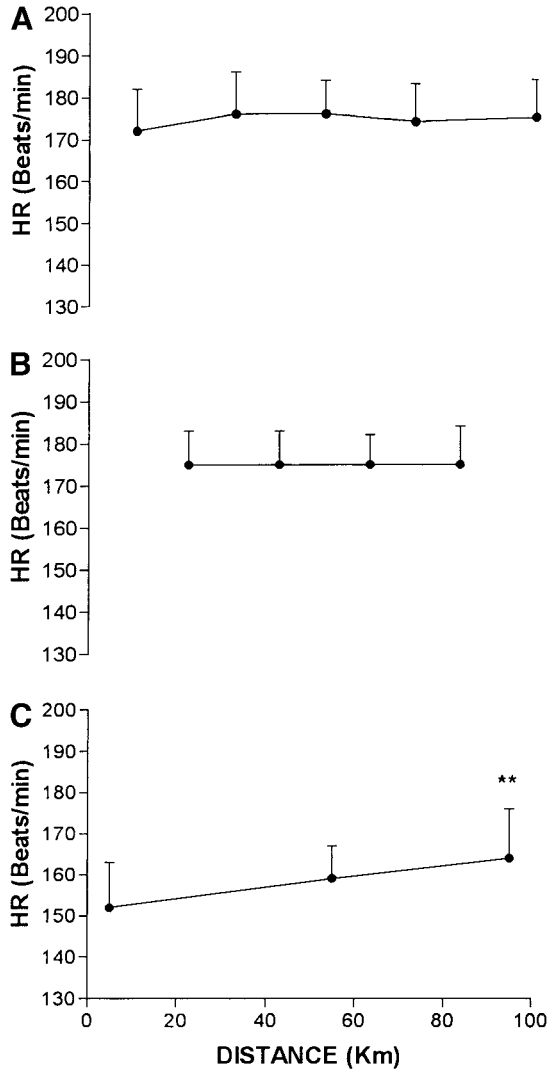

Fig. 3. Heart rate (HR) changes for the entire group for 1 (*top*)- and 4 (*middle*)-km HIE and self-paced (*bottom*) cycling during the 100-km time trial (TT; $^{**}P < 0.01$ self-paced 5 vs. 95 km).

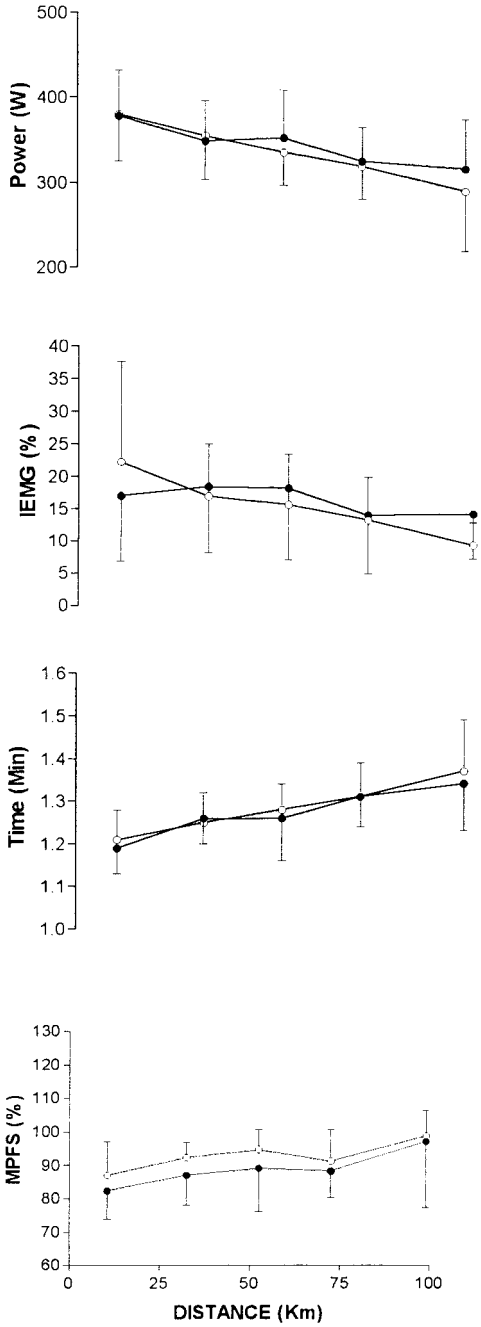

Fig. 4. Power output (W), IEMG (%), time (min), and MPFS (%) changes for the carbohydrate (CHO; ●) and placebo (○) groups during the 1-km HIE.

The first finding of decreased IEMG activity associated with decreased power output was unexpected, as previous studies have described that IEMG activity increases during prolonged submaximal exercise (12, 19, 21). As described previously, increasing IEMG activity generally indicates the recruitment of a greater number of motor units controlled by the development of a peripherally located fatigue due either to skeletal muscle mechanical damage or metabolic substrate perturbations (21). However, some other studies have reported decreased IEMG activity after prolonged submaximal exercise. For example, Nicol et al. (33) found that MVC and IEMG decreased by ~30% in subjects studied after a 42-km marathon foot race. They speculated that this was caused either by insufficient conscious effort or altered central recruitment strategies.

The findings of this study therefore show, for the first time, that neuromuscular activity starts to decline early in the exercise activity despite the conscious efforts of the subjects to maintain maximal power output. Others have shown that during isometric contractions these changes effect not only the active muscles (18) but also synergistic muscles uninvolved in the exercise (38).

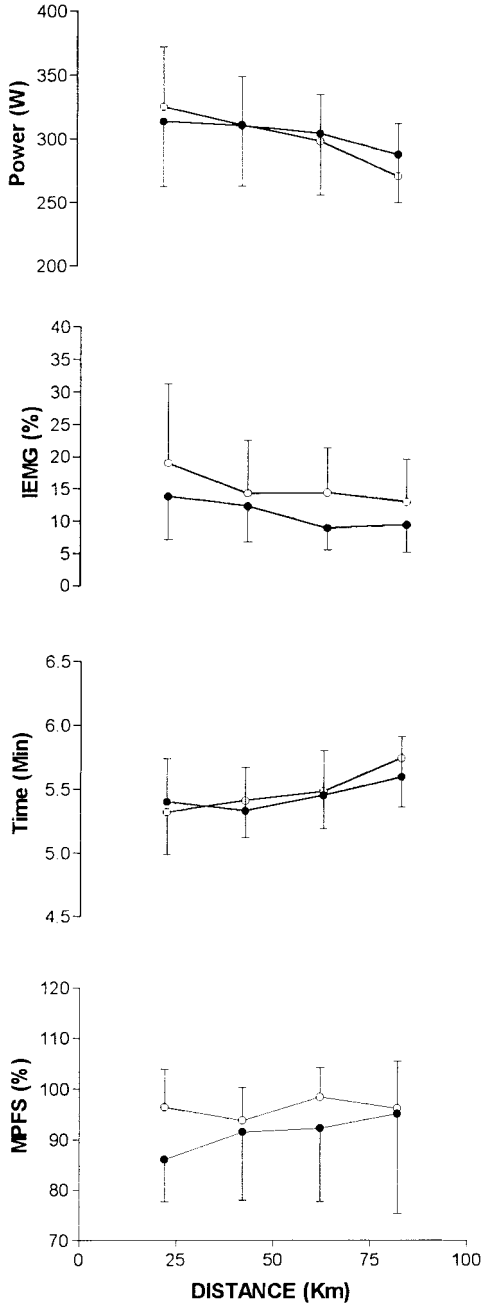

Fig. 5. Power output (W), IEMG (%), time (min), and MPFS (%) changes for the CHO (●) and placebo (○) groups during the 4-km HIE.

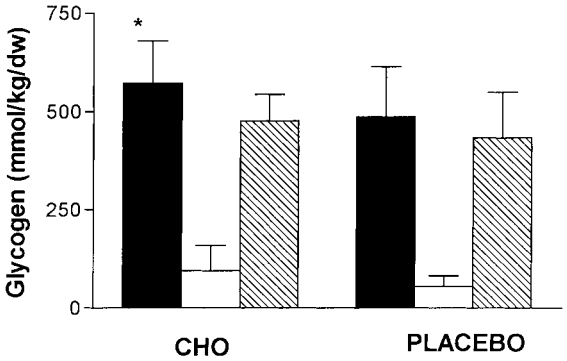

Fig. 6. Muscle glycogen content before (solid bars) and after (open bars) the 100-km TT and muscle glycogen (hatched bars) use during the 100-km TT for CHO and placebo groups (*$P < 0.05$ preexercise muscle glycogen in CHO compared with placebo trial). dw, Dry wt.

These findings could be due to fatigue originating in the motor cortex or to decreased efferent output from the motor cortex secondary to inhibitory influences arising from elsewhere in the body or to reflex muscle recruitment changes arising directly from groups III and IV metaboreceptor afferent input from the peripheral muscles at the spinal cord level (37). Previous studies that examined cortical and efferent output changes during isometric contractions (16, 17, 44) found that fatigue of the motor cortex was not responsible for decreased efferent command. Rather, the changes in motor command were regulated by changes "upstream" from the motor cortex, either excitatory or inhibitory changes in other brain structures, or as a result of failure to recruit corticospinal neurons due to decreased afferent input from group III and IV afferents in the exercising muscles. Changes in neuromuscular activity in our study could therefore be due to commands generated in the higher cortical structures or in response to afferent input from metabolic changes in the peripheral organs or both.

The protocol in this study was of a closed-loop design so that subjects knew the length of the trial and the number of sprints to be performed during the trial. Ulmer (45) speculated that efferent command signals to skeletal muscles regulate not only the spatial and temporal pattern of motion but also control the metabolic rate by adjustment of power output. He speculates that a central "programmer" would consider the time necessary to complete both the sprints and the total activity and the metabolic changes in the different body systems involved during exercise. The programmer would then regulate those systems so that the individual would complete the activity without bodily damage or premature fatigue. This regulatory system is described as teleoanticipation. In our study, the decrease in neuromuscular activity during the sprint activity may represent teleoanticipation, in which the power output was subconsciously decreased despite external encouragement and each subject's maximal conscious effort.

A limitation of our study was that the efferent neural command was measured only in the rectus femoris muscle. Other lower limb muscles might have received either excitatory or inhibitory commands different to those measured in that muscle. Other studies have shown that muscle recruitment patterns change during fatiguing activity (2, 33, 34). For example, Takaishi

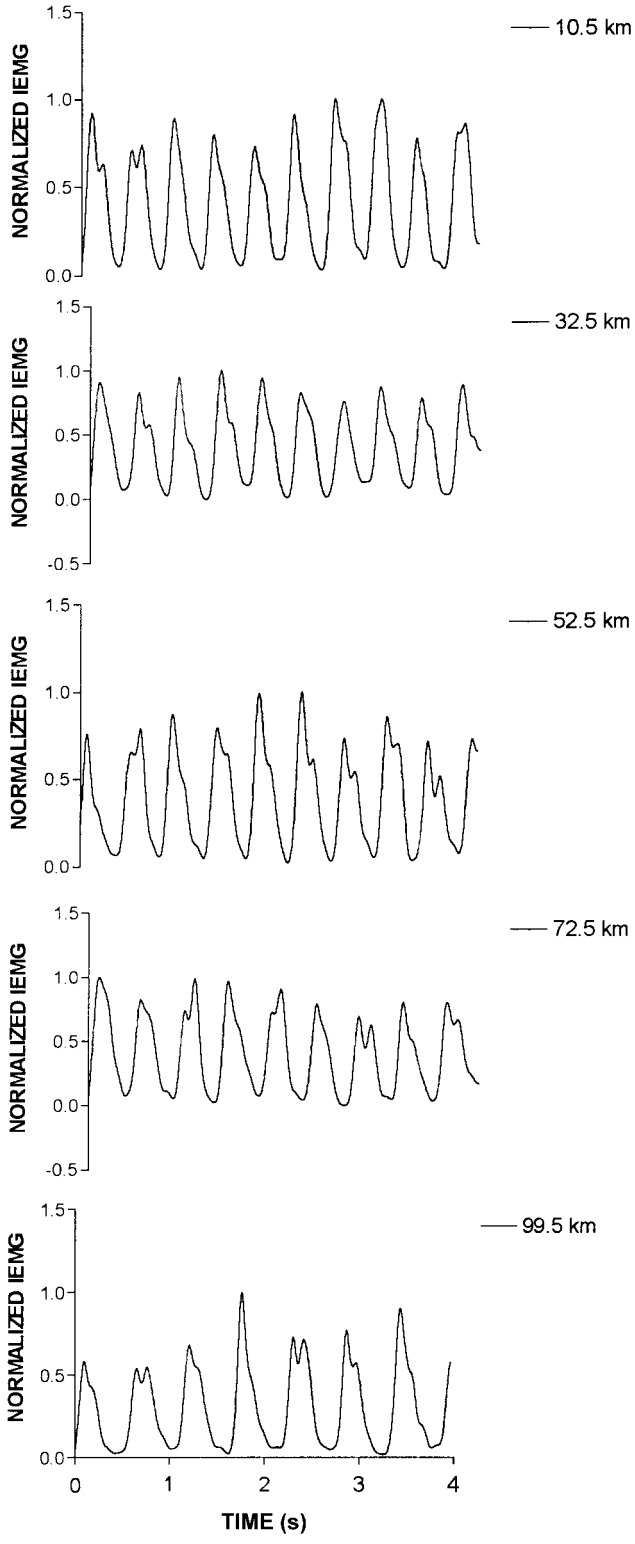

Fig. 7. Representative normalized IEMG data during the 1-km HIE in 1 subject.

et al. (42) showed that recruitment pattern changes during cycling were cadence dependent so that different muscles were used at different cadences. Thus it is possible in this study that the recruitment strategies of the lower limb were altered as the cyclists became fatigued during the TT, with other muscles being recruited to different levels compared with the changes measured in the rectus femoris muscle. The decrement in power output was ~21%, whereas the decrement in IEMG activity was ~42% during the 1-km high-intensity bouts. Similar differences were found during the 4-km high-intensity bouts. These findings suggest that recruitment strategies of different muscles involved in cycling activity must vary, as either a relative increase in force is provided by the remaining rectus femorus muscles not tested or other muscles are recruited to a greater extent compared with the rectus femoris during the high-intensity cycling activity. However, the study of Sacco et al. (38) found that reduced recruitment of one fatigued muscle was associated with reduced recruitment of synergistic muscles and there was a decline in both the overall IEMG and power output during the cycling trial.

It is also unlikely that these findings can be explained by the biomechanics of cycling preventing greater levels of muscle recruitment. Studies in our (unpublished observations) and other laboratories (29, 46) have shown that during open-loop exercise testing, including progressive exercise to exhaustion for measurement of $V_{O_{2max}}$, IEMG activity increases with increasing work rate in a repeatable manner (29). Indeed, further studies in our laboratory (26) have shown that in stochastic exercise of short duration (1 h), power output and IEMG activity decreased during the first sprints similar to the finding in this study. However, in contrast to the finding in Figs. 1 and 2, in that study both power output and IEMG activity increased in the final sprint, indicating that IEMG activity was tracking power output changes and that decrements in IEMG during the first few sprints could not be explained solely by temperature, conductivity, or electrode placement changes during the trial. Hence the decrements in IEMG activity and low percentage of muscle recruitment throughout this trial are not an artifact of the testing method used. These findings were reproduced on two separate occasions in the above study as part of a repeatability trial (26).

It was previously suggested that alterations in excitation/contraction coupling or in the contractile apparatus (2, 20) may be responsible for the decrement in power output during fatigue. Although no obvious cycling EMG pattern changes occurred (Fig. 7) during the HIE bouts, excitation/contraction coupling impairments may also be a cause of the reduced neuromuscular activity and force output decrements.

The decreases in power output during the 1 (Fig. 4)- and 4-km (Fig. 5) HIE bouts occurred similarly in both trials despite different starting muscle glycogen concentrations induced by the different dietary practices. Hence the progressive reduction in power output and neuromuscular activity was not related to differences in preexercise muscle glycogen concentrations.

The second important finding was that these decreases in power output occurred despite the recruitment of ~20% or less of the muscle mass during the cycling TTs compared with during the MVC. Previous studies have also shown the recruitment of an equally small muscle mass during cycling activity (40). Metabolic studies have shown that all available muscle fibers are never completely activated during maximal treadmill running (41), with ~41–90% of muscle fibers being recruited in different subjects during horizontal treadmill running and ~40–80% during uphill running.

Despite the low percentage of muscle fiber recruited at any instance in this study, severe muscle glycogen depletion occurred at the end of the trial coincident with marked subjective fatigue in both CHO and placebo groups. Such low muscle glycogen concentrations could not have occurred if only the same 20% or less of all muscle fibers were recruited during the entire TT.

Therefore, we conclude that the decreased neuromuscular activity was part of a strategy in which the recruitment patterns of either the entire lower limb or individual muscle fibers in the same muscle were altered during the fatiguing process. Thus different muscle fibers must have been recruited at different times so that previously quiescent muscle fibers were recruited to replace fatiguing fibers at the same time that overall efferent neural command was decreasing.

Indeed, Enoka and Stuart (13) suggested that recruitment of motor units in the same muscle alternate during fatiguing activity to attenuate fatigue during submaximal activity. Recently, Westgaard and De Luca (47) confirmed that motor unit substitution and alternation occurs during submaximal isometric contractions. They speculated that efferent neural command patterns must have knowledge of the previous activation history and temporal variation in recruitment activity and that this substitution phenomenon protected motor units from excessive fatigue during sustained submaximal isometric contractions. Further studies examining the relationship between glycogen, glycogen phosphorylase, and succinyl dehydrogenase histochemical changes in type I and II fibers during this protocol and IEMG and power output from the agonist and antagonist muscles are needed to confirm the findings of this study.

The percentile frequency shift increased significantly during the 1- and 4-km HIE exercise bouts after showing a leftward spectral compression in the first HIE relative to that during MVC. The initial leftward spectral compression may be related to the dynamic nature of cycling. A rising core temperature increases spectral density toward the higher frequencies (6). Although not measured, it is likely that rectal temperature rose progressively during this trial and might have explained this effect. It is also possible that these changes were related to a recruitment strategy in which larger, fatigue-sensitive type II fibers were selectively recruited later in the trial (3).

Surprisingly, decreases in IEMG and power output were not associated with any significant changes in HR during either the 1- or 4-km HIE (Fig. 3). Furthermore, heart rate rose significantly during the self-paced cycling (Fig. 3). These findings indicate that the subjects performed to their own maximal volitional capacity and support the conclusion that the neuromuscular activity changes were subconsciously generated. In contrast, reduced conscious effort would have been shown by reduced HR during both the HIE bouts and the steady-state exercise. Subjects also indicated verbally that they were working at maximal effort during all the HIE bouts and subjectively appeared completely exhausted at the completion of the trial. Indeed, the terminal glycogen concentrations were amongst the lowest ever recorded in this laboratory, indicating an exhaustive conscious effort.

Finally, we note that the first HIE bouts were performed at an intensity of ~90% of that achieved during their $V_{O_{2peak}}$ protocols and at ~70% of their $V_{O_{2peak}}$ intensity in the last HIE bout. Therefore, at no stage did subjects perform at power outputs higher than those reached during their $V_{O_{2max}}$ test.

In conclusion, the main findings of this study were that during 1- and 4-km HIE cycling bouts, IEMG declined parallel to decreases in power output and increases in time taken to complete these HIE. These changes occurred despite only ~20% or less of the available muscle fibers being recruited at any time during the time trial. Decreases in power output and IEMG activity were not associated with a reduction in HR during either 1- or 4-km HIE despite significant increases in HR during self-paced cycling. Although preexercise CHO loading significantly increases pretrial muscle glycogen concentrations, this did not influence any of the variables measured during the trial.

On the basis of these findings, we postulate that the progressive reduction in neuromuscular activity may be the cause of fatigue in this trial as an anticipatory mechanism occurring at a subconscious level. Further work examining multiple physiological systems, including afferent input and central command generation during dynamic endurance cycling activity, is needed to verify these findings.

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