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ORIGINAL ARTICLE

Effect of fluid ingestion on neuromuscular function during prolonged cycling exercise

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Objectives: To investigate the effects of fluid ingestion on neuromuscular function during prolonged cycling exercise.

Methods: Eight well trained subjects exercised for 180 minutes in a moderate environment at a workload requiring ~60% maximal oxygen uptake. Two conditions, fluid (F) and no fluid (NF) ingestion, were investigated.

Results: During maximal voluntary isometric contraction (MVC), prolonged cycling exercise reduced ($p < 0.05$) the maximal force generating capacity of quadriceps muscles (after three hours of cycling) and root mean square (RMS) values (after two hours of cycling) with no difference between the two conditions despite greater body weight loss ($p < 0.05$) in NF. The mean power frequency (MPF) for vastus lateralis muscle was reduced ($p < 0.05$) and the rate of force development (RFD) was increased ($p < 0.05$) only during NF. During cycling exercise, integrated electromyographic activity and perceived exertion were increased in both conditions ($p < 0.05$) with no significant effect of fluid ingestion.

Conclusions: The results suggest that fluid ingestion did not prevent the previously reported decrease in maximal force with exercise duration, but seems to have a positive effect on some indicators of neuromuscular fatigue such as mean power frequency and rate of force development during maximal voluntary contraction. Further investigations are needed to assess the effect of change in hydration on neural mechanisms linked to the development of muscular fatigue during prolonged exercise.

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Several factors are well known to play a role in the reduction of performance during prolonged muscle exercise. Within this framework, prevention of dehydration by adequate fluid replacement of body water losses during endurance exercise may maintain performance.¹ For example, McConnell *et al*² indicated that, when cyclists replaced their fluid losses fully during a two hour ride performed at 69% of peak oxygen consumption and 21°C, they were able to cycle 53% longer in a subsequent exercise bout at 90% of peak oxygen consumption. On the one hand, the consequences of dehydration on the physiological factors of endurance performance, including rise in core temperature and cardiac output, are well documented.²⁻⁴ On the other hand, few experiments have analysed neuromuscular changes induced by dehydration. One study⁵ indicated that sauna induced dehydration (~3% of body weight) resulted in an earlier increase in root mean square (RMS) and decrease in mean power frequency (MPF) during submaximal contraction. In contrast, Evetovich *et al*⁶ did not find any difference in muscle fatigability between the two conditions, hydrated versus dehydrated. To the best of our knowledge, the effect of dehydration induced by dynamic endurance exercise on electromyographic (EMG) signals has been investigated only by Ftaiti *et al*.⁷ They observed that the neuromuscular pattern of the knee extensor muscles assessed by mean EMG activity during a 40 minute submaximal running exercise in the heat was slightly altered despite a 2% loss of body weight. However, in that study it is difficult to discriminate the effect of dehydration on muscle activity from the confounding effects of hyperthermia.

Several studies have implicated a role for neuromuscular alterations in performance decrease during prolonged exercise. The fatigue underlying the reduction in force capacity is commonly related to either peripheral or central mechanisms.⁸⁻¹⁰ A study on the neural input and peripheral contractile mechanisms of knee extensor muscles after

two hours of cycling indicated that the inability of cyclists to generate their initial maximal force was related to a reduction in surface EMG (RMS) and to a change in muscular twitch and M-wave characteristics.¹¹

Therefore the aim of this study was to examine the effects of fluid ingestion on neuromuscular activity during and after a three hour cycling exercise performed at ~60% of maximal oxygen consumption. We hypothesised that the change in neuromuscular function observed in previous studies¹⁰⁻¹¹ may be accentuated without fluid ingestion.

METHODS

Participants

Eight trained competitive male cyclists or triathletes, accustomed to riding for prolonged periods, were well motivated to participate in this study. All subjects had carried out regular cycle training for at least six years, and their average weekly training distance during the two months before testing was 285 (85) km. All subjects were informed about the possible risks associated with the experiment and gave written consent in accordance with local ethical committee guidelines. Basic data (mean (SD)) on the subjects were as follows: age 30.8 (7.3) years; body weight 71.4 (9.4) kg; height 177.1 (10.7) cm; body fat 13.1 (3)%; maximal heart rate 182.5 (8.6) beats/min; maximal oxygen uptake ($\dot{V}O_{2\text{MAX}}$) 62.7 (6) ml/min/kg; peak power output 357 (37) W.

Abbreviations: EMG, electromyographic; HR, heart rate; iEMG, integrated EMG; MPF, mean power frequency; MVC, maximal voluntary isometric contraction; PSI, physiological strain index; RER, respiratory exchange ratio; RFD, rate of force development; RMS, root mean square; RPE, rating of perceived exertion; $\dot{V}O_{2\text{MAX}}$, maximal oxygen uptake

Table 1 Changes in physiological and metabolic variables during a 180 minute ride at ~60% of maximal oxygen uptake with (F) and without (NF) fluid ingestion

Variable	F	NF
Body mass (kg)		
Before	73.7 (10.0)	73.8 (10.2)
After	72.1 (10.1)*	70.8 (10.4)‡
Rectal temperature (°C)		
Before	37.1 (0.1)	37.3 (0.2)
After	37.6 (0.3)*	37.9 (0.3)*
Urine osmolarity (mosmol/l)		
Before	584 (225)	702 (213)
After	399 (175)	779 (117)†
Oxygen uptake (l/min)		
Beginning	2.63 (0.52)	2.66 (0.38)
End	2.76 (0.45)	2.86 (0.43)*
Respiratory exchange ratio		
Beginning	0.89 (0.04)	0.90 (0.07)
End	0.79 (0.06)*	0.80 (0.05)*
Heart rate (beats/min)		
Beginning	127 (13)	127 (10)
End	135 (15)	149 (17)*
Physiological strain index	4.2 (0.8)	5.2 (1.1)†

Values are mean (SD).

*Significantly different from values obtained before exercise: $p < 0.05$.

†Significantly different from values obtained with fluid ingestion:

$p < 0.05$.

‡Interaction effect: $p < 0.05$.

Preliminary testing

Firstly, VO_{2MAX} was determined during a continuous incremental cycling test to exhaustion on an electromagnetically braked ergometer (SRM; Schoberer Rad Messtechnik, Weldorf, Germany). Subjects were recommended to abstain from exhausting exercise during the preceding 48 hours. The test consisted of a six minute warm up at 100 W, after which the power output was increased by 30 W every two minutes until the subject could no longer maintain the required power output. The test was performed at a free pedalling rate. Subjects were asked to adopt the conventional cycling posture (upright) throughout the tests. The maximal power output corresponded to the mean of the highest power output maintained during the last two minutes. Oxygen uptake (VO_2), minute ventilation, and respiratory exchange ratio (RER) were recorded by a breath by breath exchange telemetric system and averaged every 10 seconds (Cosmed K4b², Rome, Italy). The turbine flow meter was calibrated before analysis using a 3 litre syringe and gas of known composition (4.92% CO_2 , 15.93% O_2). Heart rate (HR) was recorded using a chest belt (Polar Vantage; Polar Electro Oy, Kempele, Finland) connected to the telemetric device of the Cosmed K4b². The criteria used for determination of VO_{2MAX} were: a plateau in VO_2 despite an increase in power output; RER above 1.1.¹²

Experimental design

Each subject completed a crossover design of two experimental conditions consisting of performing a three hour cycling exercise at a freely chosen cadence corresponding to a workload estimated to require ~60% of VO_{2MAX} . The intensity was chosen in agreement with previous studies with the same population.^{10 11 13} During the test, two conditions were investigated: with fluid ingestion (F) and no fluid ingestion (NF). To ensure complete recovery, the two trials were separated by at least 72 hours. Subjects were asked to perform the same type of training during experimental sessions and to refrain from fatiguing physical exercise for 24 hours before each test session. They were also asked not to change their diet or fluid intake on the day before the experiment. Hydration was determined according to the

American College of Sports Medicine recommendations¹ and consisted of ingesting 0.4 litre of a commercial mineral water (0.5 mEq/l Na^+ , 0.17 mEq/l K^+ , 1.16 mEq/l HCO_3^-) before the start of the test and 0.2 litre at 20 minute intervals. Water was ingested at ambient temperature (~17–20°C). Environmental conditions were maintained at ~20–21°C and 50 (5)% relative humidity. Convective cooling was provided by one fan (2.5 m/s wind speed) located 100 cm away from the ergocycle to reduce sweating. All experimental sessions were conducted at the same time of day (8 am to 1 pm).

Data recording

All testing was conducted on an electromagnetically braked SRM ergometer which could be adjusted to replicate the cycle racing position of the subjects—that is, seat tube, handlebars, crank length. The cranks were equipped with personal racing pedals allowing subjects to wear their own cycling shoes. The SRM ergometer allowed the subjects to keep power output constant independent of the pedal rate that they spontaneously adopted. Power output was continuously calculated from the torque and angular velocity.¹⁴ Subjects had to adopt the same position on the ergometer during the two tests.

Pedalling rate was recorded online every 20 minutes during the 180 minute ride: T1 (17–20), T2 (37–40), T3 (57–60), T4 (77–80), T5 (97–100), T6 (117–120), T7 (137–140), T8 (157–160), T9 (177–180).

Immediately after each recording period, the subjects were asked to report their rating of perceived exertion (RPE) using the Borg 15 point scale.¹⁵

Metabolic data including VO_2 , RER, and HR were obtained during the first 20 minutes and at the end of exercise. Oxygen uptake was collected breath by breath and sampled at 10 second intervals (Cosmed K4b²). The collecting facemask (Hans-Rudolph, Kansas City, Missouri, USA) was fitted two minutes before the three minute recording period to ensure that the stress of fitting did not perturb the ventilation pattern.

In the laboratory, naked body mass of the subjects was measured on a platform scale (accuracy ± 10 g). Rectal temperature (T_{re}) was recorded using a rectal thermometer inserted 10 cm beyond the anal sphincter. Subjects were asked to provide a urine sample for determination of osmolarity as follow:

$$U_{osm} \text{ (mosmol/l)} = 2[Na^+] + [Glucose] + [Urea]$$

At the end of the exercise, the subjects removed their clothing and towelled themselves dry. Body mass and temperature were again determined and a urine sample collected. The physiological strain index (PSI), which reflects the strain imposed by the exercise on the thermoregulatory and cardiovascular systems, was evaluated using the 10 point scale proposed by Moran *et al.*¹⁶ PSI was calculated using T_{re} and HR as follow:

$$PSI = 5(T_{re-post} - T_{re-pre})(39.5 - T_{re-pre})^{-1} + 5(HR_{post} - HR_{pre})(180 - HR_{pre})^{-1}$$

where T_{re-pre} and HR_{pre} are the initial T_{re} and HR respectively, and $T_{re-post}$ and HR_{post} are measurements at the end of exercise.

Measurement of maximal muscle performance

The performance of the knee extensor muscles was tested using an isometric ergometer (Vertex II; Harvard Sports Inc, Compton, California, USA) during a maximal voluntary isometric contraction (MVC). Subjects were carefully instructed to contract “as fast and forcefully as possible” and to maintain this contraction. An isometric ergometer was placed next to the ergocycle to minimise transitions for muscle tests above one minute. During MVC, the subjects were seated in an upright position, securely strapped into the

Table 2 Changes in pedalling rate and rating of perceived exertion (RPE) during a 180 minute ride at ~60% of maximal oxygen uptake with (F) and without (NF) fluid ingestion

	T1	T2	T3	T4	T5	T6	T7	T8	T9
Pedalling rate (rpm)									
F	87 (14)	86 (14)	84 (15)	89 (14)	86 (14)	82 (15)	82 (13)	76 (11)	76 (14)
NF	86 (14)	86 (13)	85 (13)	83 (10)	82 (9)	83 (14)	83 (15)	80 (13)	80 (26)
RPE									
F	13 (1)	13 (1)	14 (2)	13 (1)	14 (2)	14 (2)	15 (2)*	17 (3)*	17 (3)*
NF	14 (1)	14 (2)	15 (1)	15 (2)	16 (2)*	17 (2)*	17 (2)*	18 (2)*	18 (2)*

Values are mean (SD).

*Significantly different from T1 values: $p < 0.05$.

muscle test chair. The angular position was fixed at 100° for the hip and 90° for the knee (0° was the full leg extension). Subjects were verbally encouraged to exert maximum effort. Force was measured using a strain gauge (F501TC; TME, Orgeval, France) and sampled at 100 Hz. After amplification, the signal was stored in a computer using a 16 bit acquisition card (DAS-1801 ST; Keithley Instruments Inc, Taunton, Massachusetts, USA) for further analysis. The periods of measurement were after the warm up and at the end of the 60th (H1), 120th (H2), and 180th (H3) minute of cycling. The alteration in the rate of force development (RFD) was determined for each MVC. Force data were linearly time aligned at 0.01 second intervals for each subject. A mathematical model was used to characterise the average response curves using a least squares non-linear regression technique, in which the best fit was defined by minimisation of the residual sum of squares. Force data from the MVC tests were fitted by a monocomponent exponential model which provides an estimation of the baseline (A_0), the asymptotic amplitude (A_1), time constant (τ_1), and time delay (TD_1)

$$f(t) = A_0 + A_1[1 - e^{-(t-TD_1)/\tau_1}]u_1$$

where $u_1 = 0$ for $t < TD_1$ and $u_1 = 1$ for $t \geq TD_1$.

RFD (milliseconds) corresponded to the time required to achieve ~63% of the total amplitude of difference from the baseline to the final plateau value.

EMG signal

The EMG signal of the vastus lateralis of the right leg was recorded over a three minute period using the timing described above (T1 to T9). The skin over the selected muscle was shaved and carefully prepared by light abrasion of the outer layer of epidermal cells; oil and dirt were removed with acetone to reduce skin impedance ($Z < 5 \text{ k}\Omega$). EMG signals were recorded using a parallel bar configuration of Ag/AgCl surface electrodes (Medicotest A/S, Rugmarken, Denmark) positioned over the distal half of the muscle belly. Electrodes were placed such that the contact surfaces were aligned longitudinally to the muscle fibres with a 20 mm interelectrode distance. A reference electrode was positioned on the front part of the right tibial tuberosity. EMG signals were digitised online (PowerLab/8SP; AD Instruments, Castle Hill, Australia), sampled at 1 kHz for the duration of the two trials, and stored on hard disk for further analysis. EMG signals were high pass filtered with a cut off frequency of 0.3 Hz, and then smoothed with a low pass filter with a cut off frequency of 500 Hz.

The power spectrum density function was calculated by a fast Fourier transformation algorithm for each MVC (H1, H2, H3). Additional high pass filtering (20 Hz) was used to eliminate movement artefacts. A mean spectrum was computed by calculation of the RMS values obtained from consecutive windows of 0.5 second duration. The resulting power spectrum was defined by 512 points in amplitude and phase. To standardise analysis, the spectrum was computed over a 1.5 second plateau after the peak force had been

reached. The final result of this signal analysis includes RMS and MPF. During cycling, the vastus lateralis EMG signal was full wave rectified and the integrated EMG (iEMG) value was calculated. To compare the two conditions, only 20 consecutive pedalling bursts were integrated within each three minute recording period. All EMG data were normalised by dividing the value at each time point during the trials by the EMG value obtained during MVC performed before the start of each ride. iEMG, RMS, and MPF data were therefore expressed as a percentage of these MVC data.¹⁷

Statistical analysis

Descriptive statistics were generated for all variables. The data for different sessions were analysed by repeated measures (time \times conditions) multivariate analysis of variance. Individual differences between means were then located using the Tukey post hoc multiple comparison procedure. A paired t test was used to determine pairwise differences in PSI values. For all the statistical analyses, the level of significance was set at $p < 0.05$. All data were expressed as mean (SD).

RESULTS

Physiological responses

Table 1 presents the mean values for physiological variables observed before and after completion of the 180 minute cycle. A significant interaction effect of exercise duration and fluid ingestion on physiological changes was found for loss of body mass, urine osmolarity, oxygen consumption, heart rate, and PSI. In the NF condition, subjects exhibited a 4.1% loss of body mass after exercise, whereas a 2.2% body mass loss was recorded in the F condition. Urine osmolarity was increased by 11.0% in NF, whereas in F it showed a 31.6% decrease. At the end of exercise, HR was increased by 16% in NF, and $\dot{V}O_2$ was increased by 8%, whereas no significant changes were reported in F. In both trials an increase in RPE was observed. However, this increase appears earlier in NF than in F (T5 ν T7; table 2). Finally no significant difference in RER changes or rectal temperature was observed between the trials. Whatever the conditions, after three hours of cycling, RER was decreased ($\Delta RER = -0.10$ in NF and F), and a slight increase in rectal temperature was recorded ($\Delta T_{re} = +0.6^\circ\text{C}$ in NF and $+0.5^\circ\text{C}$ in F).

Cycling cadence

Table 2 shows changes in freely chosen cadence. No significant effect of exercise duration or fluid ingestion was observed. Nevertheless, a non-significant decrease in pedalling rate was observed throughout the exercise from 87 (14) to 76 (14) rpm (-13%) in F and 86 (14) to 80 (26) rpm in NF (-7%) despite the power output being held constant.

Maximal voluntary contraction

Whatever the conditions, the maximal force generating capacity of the quadriceps muscle was altered with exercise

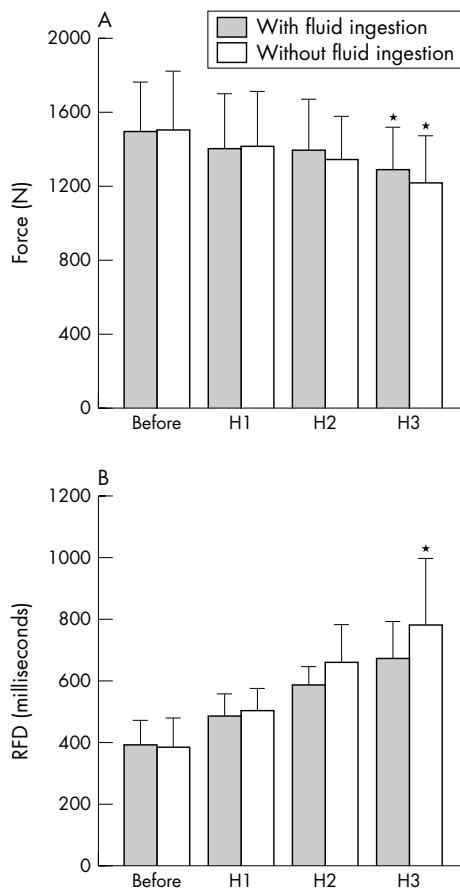


Figure 1 Changes in maximal force (A) and rate of force development (RFD) (B) of quadriceps muscles observed during maximal voluntary contraction. Tests were performed before and during (H1, 60th minute; H2, 120th minute; H3, 180th minute) a 180 minute ride at ~60% of maximal oxygen uptake with and without fluid ingestion. Values are mean (SD). *Significantly different from before exercise: $p < 0.05$.

duration. Maximal force had decreased after 180 min with no difference between F and NF (-16 (10)%; fig 1A). A significant effect of fluid ingestion was observed on the RFD (fig 1B) with a significant difference between F and NF at the end of exercise (675 (97) milliseconds in F v 785 (108) milliseconds in NF).

Figure 2A shows maximal muscle activity for vastus lateralis muscle during MVC. A significant decrease in MPF recorded during MVC was observed only during the NF trial after two hours of cycling (-19%; fig 2B). In contrast, a decrease in RMS for the vastus lateralis muscle was observed after two hours of exercise without any effect of fluid ingestion (-18% and -26% respectively for F and NF).

Muscle activity during cycling

Figure 3 shows changes in iEMG activity during the 180 minute cycle. A significant effect of exercise duration was found on vastus lateralis muscle activity with a significant increase in mean iEMG activity after two hours of cycling, without any effect of fluid ingestion.

DISCUSSION

The purpose of this investigation was to examine the effects of fluid ingestion on muscle performance during a three hour cycling exercise (60% of VO_{2MAX}). The main result is that fluid ingestion seems to play a positive role in preventing the appearance of some neuromuscular fatigue phenomena

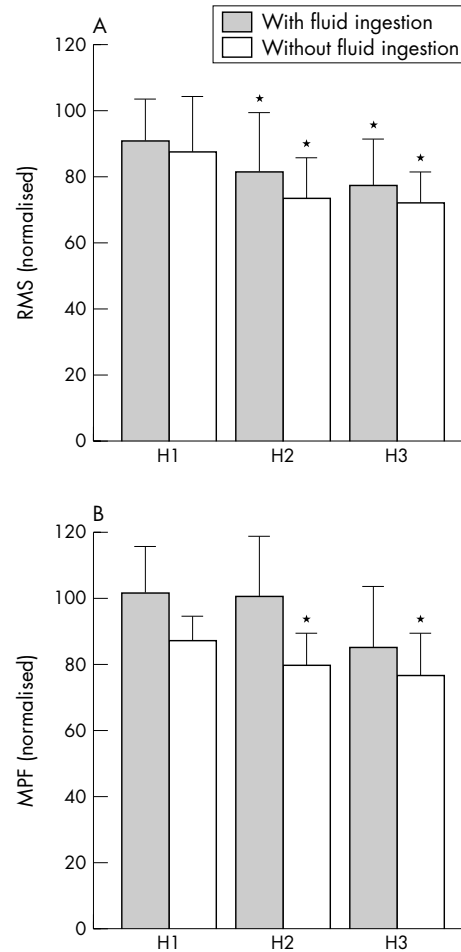


Figure 2 Root mean square (RMS) (A) and mean power frequency (MPF) (B) values measured during maximal voluntary isometric contraction (MVC) of the vastus lateralis muscle before and during (H1, 60th minute; H2, 120th minute; H3, 180th minute) a 180 minute ride at ~60% of maximal oxygen uptake with and without fluid ingestion. Values are mean (SD). For each trial, values were compared with the reference values—that is, before exercise, MVC 100%. *Significantly different from before exercise: $p < 0.05$.

indicated by a change in MPF or RFD. However, it has no significant effect on the decrease in muscular maximal force previously reported with exercise duration.^{3,4} We observed

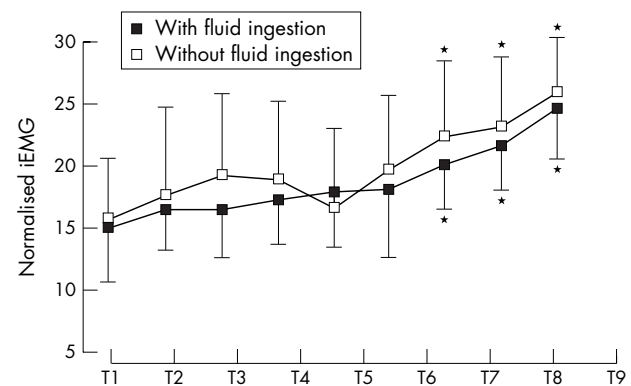


Figure 3 Integrated electromyographic (iEMG) values of the vastus lateralis recorded every 20 minutes with and without fluid ingestion. Values are mean (SD). For each trial, iEMG values were compared with the reference values—that is, before exercise, MVC, 100%. *Significantly different from before exercise: $p < 0.05$.

that maximal voluntary force was decreased after three hours of exercise, and fluid ingestion had no effect (fig 1A). The reduction in force production (-16 (10)%) after three hours of cycling is in agreement with previous studies in which a reduction in force production of knee extensor muscles was observed in well trained cyclists after a ride lasting several hours^{10 11} as well as after high intensity exercise.^{13 18 19} Within this framework, Lepers *et al*¹⁰ reported, during a five hour cycling exercise at 55% of $\dot{V}O_{2MAX}$, a decline in maximal isometric force of 10% after the third hour and 18% at the end of the exercise. The concomitant decrease in force generating capacity and muscle activity observed (fig 1A and 2A) in our study after three hours of cycling was consistent with previous investigations.^{11 18} For instance, Bentley *et al*¹⁸ showed that the decline in MVC (~12%) observed after a 30 minute cycling exercise (80% of $\dot{V}O_{2MAX}$) was accompanied by a reduction in iEMG activity of the vastus medialis and rectus femoris muscles (of 22 and 24% respectively). Considering that the level of surface EMG activity reflects the degree of neural activation, including the number of motor units recruited and the firing frequency to the muscle being investigated,²⁰ the decrease in RMS values in our study suggests that a deficit in the central drive may be responsible for the decline in force production. In our study, this decrease appears earlier than the decrease in maximal force (two hours compared with three hours). Further analysis of the origin of strength loss during prolonged exercise is necessary.

The impairment in muscular function observed during maximal isometric testing tends to be confirmed during dynamic exercise by the increase in iEMG activity observed during the last hour of the three hour cycling exercise (fig 3). Relatively few data are available on neuromuscular alterations generated by prolonged cycling (more than one hour). To the best of our knowledge, the study of St Clair Gibson *et al*²¹ is the only one to have examined the effect of prolonged cycling on muscle performance under dynamic conditions. These authors reported a progressive decline in iEMG activity of the rectus femoris muscle associated with a decrease in power output during high intensity bouts simulating a 100 km cycling time trial. The methodology of our study is different, as power output was kept constant and no significant change in pedal cadence was observed. The increase in iEMG that we observed is similar to previous results obtained during prolonged running.²² Despite methodological differences leading to different trends in the changes in iEMG activity between the study of St Clair Gibson *et al*²¹ and ours, it seems that central mechanisms of fatigue may be involved in the neuromuscular alterations reported in both studies. The rise in iEMG activity reported in the present investigation may derive from the necessity for the central command to increase the number of motor units recruited and/or their firing frequency in response to peripheral fatigue.²³ During prolonged exercise, there is a failure in the contractility of muscle fibres, which must be compensated for, with the recruitment of new fibres to maintain a constant power.

It is surprising to find that fluid ingestion had only a small effect on neuromuscular variables with no changes in maximal force, RMS, or iEMG and only a significant effect on MPF and RFD. As previously reported,^{2 3} the three hour

cycling exercise without hydration resulted in greater physiological strain, as attested by the significant differences between the F and NF conditions for the physiological variables measured ($\dot{V}O_2$, HR, RPE, PSI). Our results agree with those of a previous study, which examined the negative effect of dehydration on performance during submaximal prolonged exercise.³ The dehydration that results from an imbalance between fluids ingested and sweat loss during exercise is classically associated with weight loss, increased core temperature, increased heart rate, and impairment of performance during prolonged exercise. Indeed, we observed a beneficial effect of fluid ingestion on the variables of hydric status such as weight loss, HR, urine osmolality, and PSI. Surprisingly, despite evident signs of dehydration in the present study, core temperature remained unchanged between the F and NF trials, which could be explained by efficient thermoregulation and/or optimal environmental conditions. This stability of core temperature may explain the lack of effect of fluid ingestion on iEMG values. This result is comparable to the findings of Hunter *et al*,²⁴ who reported no changes in iEMG in cyclists performing three successive 15 minute rides (30%, 50%, and 70% of $\dot{V}O_{2MAX}$) in hot (35°C) and cold (15°C) environments (humidity 50%). These investigators suspected that the unchanged core temperature in hot and cold conditions, as the result of improved thermoregulation in the hot conditions (temperature and humidity too low, fan speed too high), may be at the root of a similar efferent neural strategy. These findings tend to confirm that improvement in thermoregulatory mechanisms and/or optimal environmental conditions may be responsible for the absence of significant differences in iEMG activity and RMS observed between our F and NF trials.

One interesting result of our study is that the significant shift in MPF in the NF condition toward lower frequencies appeared sooner than the loss of force production capability (fig 2B), whereas MPF values remained unchanged during the F condition. Changes in spectral variables are classically used to assess muscle fatigue. A shift of MPF toward lower frequencies has been shown to be mainly associated with a lower conduction velocity along the sarcolemma.²⁵ Previous studies have reported a decline in MPF during submaximal isometric contraction performed after a prolonged run.^{22 26} In these studies, the ultrastructural damage resulting from the eccentric contractions induced by running²⁷ may account for the reduction in muscle excitability. Recent work on the muscle membrane fibre as a site of fatigue has relied on the properties of the M-wave as an indirect measure of sarcolemmal excitability. Observing the changes in muscle excitability during a five hour cycling exercise, Lepers *et al*¹⁰ reported an increased M-wave duration for vastus medialis muscle after the 4th hour, which confirmed preliminary results showing that M-wave duration was increased for vastus medialis and vastus lateralis muscles after two hours of cycling.¹¹ It has been proposed that the rundown of gradients for Na^+ and K^+ during exercise can reduce the transmembrane ionic potential and thereby alter M-wave characteristics and force production.²⁸ Two recent studies

What is already known on this topic

The consequences of dehydration on the physiological factors of endurance performance, including rise in core temperature and cardiac output, are well documented, but the effects on neuromuscular activity are not known.

What this study adds

The results indicate that fluid ingestion can prevent changes in some indicators of neuromuscular fatigue, such as the decrease in MPF or RFD. However, neither the recruitment of new fibres during isometric or dynamic exercise after two hours of cycling nor the decline in the maximal voluntary force after three hours of exercise were affected.

have led to contradictory results concerning this hypothesis. On the one hand, Fowles *et al*²⁹ reported, during submaximal isometric exercise, a reduction in Na⁺-K⁺-ATPase activity associated with loss of excitability, as indicated by M-wave alterations, whereas Sandiford *et al*³⁰ failed to observe a change in M-wave response despite a decrease in Na⁺-K⁺-ATPase activity. In our study, it can be hypothesised that the fluid ingestion prevented ionic change. Further studies examining the relation between Na⁺-K⁺ pump activity and dehydration during prolonged endurance exercise are needed to better understand the changes in muscle excitability observed with fatigue. Another interesting result of our study is the positive effect of fluid ingestion on the time necessary to achieve ~63% of the maximal level of knee extensor force—that is, RFD (fig 1B). In our study, this increase observed only in the NF condition after three hours of cycling may also indicate an alteration in the neural drive.

In summary, our results indicate that fluid ingestion can prevent changes in some indicators of neuromuscular fatigue such as the decrease in MPF or RFD. On the other hand, it affects neither the recruitment of new fibres (assessed by RMS or iEMG) during isometric or dynamic exercise after two hours of cycling nor the decline in the maximal voluntary force after three hours of exercise. Further investigations are needed to assess the affect of change in hydration on neural mechanisms responsible for the development of fatigue during prolonged exercise.

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