ORIGINAL ARTICLE

Neuromuscular fatigue is greater following highly variable versus constant intensity endurance cycling

Jean Theurel · Romuald Lepers

Abstract The present study compared neuromuscular fatigue of the knee extensor muscles following highly variable versus constant power output cycling. Ten subjects performed two 33-min cycling trials of the same average power output, in a random order. Cycling exercise was performed either at constant (CST) power output, corresponding to 70% of the maximal aerobic power (MAP), or at variable (VAR) power output with alternating high (200, 150 and 100% of MAP during 10, 15 and 20 s, respectively) and moderate (50% of MAP) power output periods. Neuromuscular tests were performed before and immediately after the two trials. Heart rate (HR) was measured during exercise and blood lactate concentration ([La]) at the end of both trials. Reductions in maximal voluntary contraction torque, voluntary activation level and peak doublet were significantly greater after VAR than after CST. HR and [La] were significantly higher during VAR than during CST. Cycling at a varying power output in comparison to constant power resulted in additional muscular fatigue that may be explained by greater anaerobic contribution and muscle solicitation during the highly variable power output protocol.

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Introduction

Previous studies have demonstrated significant knee extensor muscle fatigue following prolonged (>30 min) cycling exercise (Lepers et al. 2000, 2002). Muscular fatigue, which refers to a reduction in maximal voluntary contraction (MVC) force or power production, can originate from sites located proximal (central fatigue) or distal (peripheral fatigue) to the neuromuscular junction (Gandevia 1998). After prolonged cycling, the reduction in MVC torque of the quadriceps muscle resulted from changes in both central and peripheral processes (Lepers et al. 2000, 2002, 2001a; Kay et al. 2001). Muscular fatigue has often been examined during/following cycling exercise at a constant power output. However, with the exception of time trials, cycling competitions are rarely performed in these conditions (Martin et al. 2001; Ebert et al. 2005). Vogt et al. (2007) evidenced that an elite cycling race is characterized by medium power output (300 W) for prolonged periods (≈30 min) and by very high peak power (>800 W) bursts for short time periods (15–30 s).

The response to variable versus constant intensity cycling exercise has been recently examined. For example, Palmer et al. (1997) showed that cycling time trial performance using variable intensity (±12% of the mean power output) was significantly impeded compared to constant load cycling at the same absolute intensity and duration. In contrast, other researchers suggested that slight power variations (±5%) may result in improved performance during cycling time trialing (Aktinson and Brunskill 2000; Vogt et al. 2007), without additional physiological stress compared to a constant power trial (Liedl et al. 1999). Interestingly, we recently demonstrated that varying power output from 68 to 92% of maximal aerobic power (MAP) during 30 min induced similar alterations of central and peripheral

components of neuromuscular fatigue compared to those observed after constant power output cycling (Lepers et al. 2008). In this previous study, the lack of difference in muscle fatigue between variable and constant power output protocols could be explained by small power output variations. A greater variation of power output profile, including efforts beyond the MAP, may exacerbate neuromuscular fatigue compared to constant power output cycling exercise. Indeed, an increased contribution of the anaerobic system may induce a greater production and accumulation of H^+ protons that could in turn impair neuromuscular propagation and excitation–contraction coupling processes at the peripheral level (Metzger and Moss 1990). In addition, high muscle tension at high cycling power output could increase the group III and IV afferents discharge at the spinal and/or supraspinal level, thus resulting in considerable muscle activation deficit (Gandevia 1998).

The purpose of the present study was to examine peripheral and central components of quadriceps muscle fatigue following cycling with high variable versus constant intensity performed at the same average power. It was hypothesized that cycling with high power output changes could result in greater alterations at both peripheral and central levels that would enhance fatigue in comparison with a constant intensity exercise.

Methods and procedures

Subjects

Ten well-trained male cyclists (age: 29 ± 7 (SD) years, body mass: 71 ± 4 kg, height: 179 ± 5 cm) volunteered to participate in this study after they were informed in detail about the nature of the experiment and possible risks. Written informed consent was given by each subject and the study was conducted according to the Declaration of Helsinki. A local ethics committee for the protection of individuals gave approval concerning the project before its initiation. Subjects had regularly trained in cycling for 10.5 ± 4.6 years prior to the study. During the 2 months preceding the study, they completed 340 ± 95 km of cycling training/week on average, corresponding approximately to 11.7 ± 3.2 h/week.

Maximal cycling test

During the initial session, which took place at least 5 days before the experiment, each subject performed a continuous, incremental test on a bicycle ergometer. The test began at 150 W for 2 min, after which the power output was increased by 25 W every 2 min until volitional exhaustion. The MAP (395 ± 25 W) was determined using the following formula:

$MAP = P + (t/120) \times 25$, where P is the last completed stage and t is the time in the final stage. During this incremental exercise, heart rate (HR) values were monitored on-line using a Polar unit (S725x, Polar Electro, Kempele, Finland). Thirty minutes after the maximal test, subjects familiarised themselves with the isokinetic measurements and apparatus as well as with the transcutaneous stimulation.

Experimental protocol

Each subject performed two 33 min cycling trials at the same average power output, at a freely chosen cadence: one at a constant power output (CST) and one at a variable power output (VAR). The two sessions, performed in a random order, were separated by 1 week. In the VAR condition, the power output was alternated during 10 identical periods each lasting 3 min 20 s (T1-T10). These periods consisted of power peaks of 200, 150, and 100% of MAP during 10, 15, and 20 s, respectively, separated by moderate power output periods at 50% of the MAP (Fig. 1). The CST condition consisted of 70% of MAP (277 ± 17 W), corresponding exactly to the mean power output produced during the VAR condition.

Neuromuscular test

A standardized warm-up was carried out by each subject before each testing session. It consisted of 5-min cycling at 33% of MAP followed by 5 min at 50% of MAP. After the warm-up period, the right knee extensor muscles were tested using a series of neuromuscular assessments.

Neuromuscular testing protocol consisted of: three electrically evoked twitches separated by 4 s and two or three (if the difference between the two first trials was greater than 5%) knee extensors MVC with doublets delivered (1) 2 s before the MVC, (2) over the MVC isometric plateau (superimposed doublet) and 1.5 s after the MVC (potentiated doublet), to assess voluntary activation level according
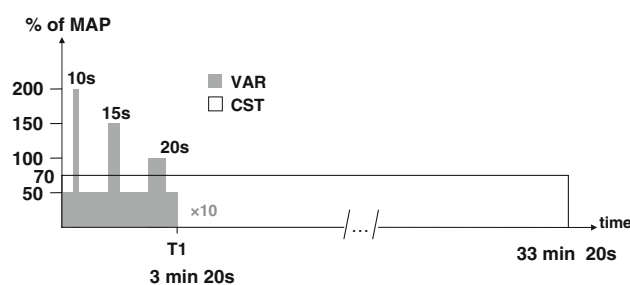
Fig. 1 Representation of the two cycling conditions: Variable (*VAR*) and constant (*CST*) power output protocols. VAR protocol was composed by 10 identical periods alternating power output peaks of 200, 150 and 100% of the maximal aerobic power (*MAP*) and moderate power output periods (50% of the MAP). During CST, power output corresponded to 70% of MAP

to the twitch interpolation technique (Merton 1954). The use of superimposed doublets at 100 Hz has been shown to be as sensitive as quintuplets for detecting muscle inactivation (Behm et al. 1996) but with reduced pain compared to the latter condition. Each MVC was approximately 4 s in duration and 60 s of rest was interspersed between trials. Strong verbal encouragement was given to the subjects during each MVC. The same experimental procedure (lasting approximately 5 min) was performed before and immediately after both cycling sessions. Approximately 1 min was needed to place the subject in the testing position following cycling exercise.

Data collection

Evoked contractions

Electrically evoked contractions were induced using a high-voltage (maximal voltage 400 V) constant-current stimulator (model DS7, Digitimer, Hertfordshire, UK). The femoral nerve was stimulated using a monopolar cathode ball electrode (0.5 cm diameter) pressed into the femoral triangle by the experimenter. The site of stimulation was marked on the skin so that it could be repeated after the cycling exercise. The anode was a 50 cm^2 (10 × 5 cm) rectangular electrode (Compex SA, Ecublens, Switzerland) located in the gluteal fold opposite to the cathode. The optimal intensity of stimulation was considered to be reached when an increase in the stimulation intensity did not induce a further increase in the amplitude of the twitch force and of the peak-to-peak amplitude of the vastus lateralis (VL) M wave (see Electrical recordings). The stimulus duration was 1 ms and the interval between paired stimuli was 10 ms. Once the optimal intensity was found, it was kept constant throughout the session for each subject.

Torque recordings

Isometric knee extension torque was recorded using a Biodex isokinetic dynamometer (Biodex Shirley Corporation, NY, USA). Subjects were placed in the seated position, and were securely strapped into the test chair. Extraneous movements of the upper body were limited by two crossover shoulder harnesses and a belt across the abdomen. The trunk/thigh angle was 90°. The axis of the dynamometer was aligned with the knee flexion-extension axis and the lever arm was attached to the shank using a strap. The knee angle was fixed at 90° of flexion (0° = knee fully extended).

EMG recordings

Surface EMG activity was recorded with pairs of silver chloride circular (recording diameter of 10 mm) electrodes

(Contrôle Graphique Médical, Brie-Comte-Robert, France) positioned lengthwise over the middle of the VL muscle belly with an interelectrode (center-to-center) distance of 20 mm. The reference electrode was attached to the left wrist. Low resistance between the two electrodes (<5 kΩ) was obtained by abrading and cleaning the skin using alcohol. EMG signals were amplified with a bandwidth frequency ranging from 15 Hz to 2 kHz (common mode rejection ratio = 90 dB; impedance input = 100 MΩ; gain = 1,000), digitized on-line at a sampling frequency 2 kHz using a computer (IPC 486) and stored for analysis with commercially available software (Tida, Heka electronic, Lambrecht/Pfalz, Germany).

Physiological data recordings

HR values were recorded continuously during cycling exercise and averaged over each of the 10 standardized periods (T1–T10) for VAR condition and at the same corresponding times for the CST condition. Lactate concentration ([La]) was measured during the last minute of each protocol, using a commercially available analyzer (Lactate Pro, Arkray, Inc. Kyoto, Japan). Blood samples were collected from the fingertip, using a lancing device.

Ratings of perceived exertion

At beginning of each test, subjects were provided with a typewritten set of standardized directions for the use of a scale (6–20) of the rating of perceived exertion (RPE) (Borg 1970). They were instructed to judge their global effort at the end of T1–T10 periods for VAR condition and at the same corresponding times for the CST condition.

Data analysis

Torque and EMG data

Mechanical parameters for single and paired stimuli were analyzed and the average of three trials was retained. Peak twitch torque (Pt) and peak doublet torque (Dt) were calculated from the single and potentiated doublet torque trace, respectively. M-wave peak-to-peak amplitude and duration were analyzed for the VL muscle (average of three trials). MVC torque was considered as the peak torque attained during the contraction and maximal voluntary activation level (VAL) was estimated according to the following formula: VAL = [1 − (superimposed doublet amplitude/potentiated doublet amplitude)] × 100 (Behm et al. 1996). In a few cases, in which the superimposed doublet was applied when the torque level was slightly below the maximal voluntary torque, a correction was applied in the original equation, as suggested by

Strojnik and Komi (1998). During MVC, EMG signals were quantified using the root mean square (RMS), which was calculated over a 0.5 s period after the torque had reached a plateau. The EMG ratio between RMS and M-wave amplitude (RMS/M), was also calculated for the VL muscle.

Cycling exercise

All experiments were conducted on an electromagnetically braked cycle ergometer (Type Excalibur, Lode, Groningen, Netherlands) where the seat and handlebars were fully adjustable both vertically and horizontally in order to replicate habitual positioning of subjects on their own bicycle. The ergometer was also equipped with racing pedals (toe clips), which allowed subjects to wear their own cycling shoes. Pedaling cadence was recorded instantaneously from the ergometer using a computer. The ergometer allowed subjects to keep power output constant independently of the cadence naturally adopted. No feedback was given to the subjects concerning their self-selected cadence during the entire experiment. The experiments were performed in the laboratory at a temperature of $21 \pm 2°C$ and relative humidity of $50 \pm 5\%$. At the start of exercise, two fans were placed in front of the bicycle ergometer in order to increase cooling of the subject during cycling.

Statistical analysis

Kolmogorov–Smirnov tests confirmed that all data were normally distributed. Subsequently, a two-way (time × condition) ANOVA with repeated measures on both factors was performed on variables obtained during the cycling exercise (HR, cadence and RPE). One-way ANOVA was used to compare pre- to post-exercise changes in neuromuscular variables as well as average [La] between the two cycling conditions. Post hoc analyses (Newman–Keuls) were used to test differences among pairs of means when appropriate. A level of $P < 0.05$ was used to identify statistical significance. Statistical analyses were performed using Statistica software for Windows (Statsoft, version 6.1, Statistica, Tulsa, OK, USA). Data were presented as the mean ± standard deviation in the text and as the mean ± standard error in the figures.

Results

Cardiovascular responses

Figure 2 shows the changes in HR, RPE and cadence during the two cycling exercises. Mean HR was significantly
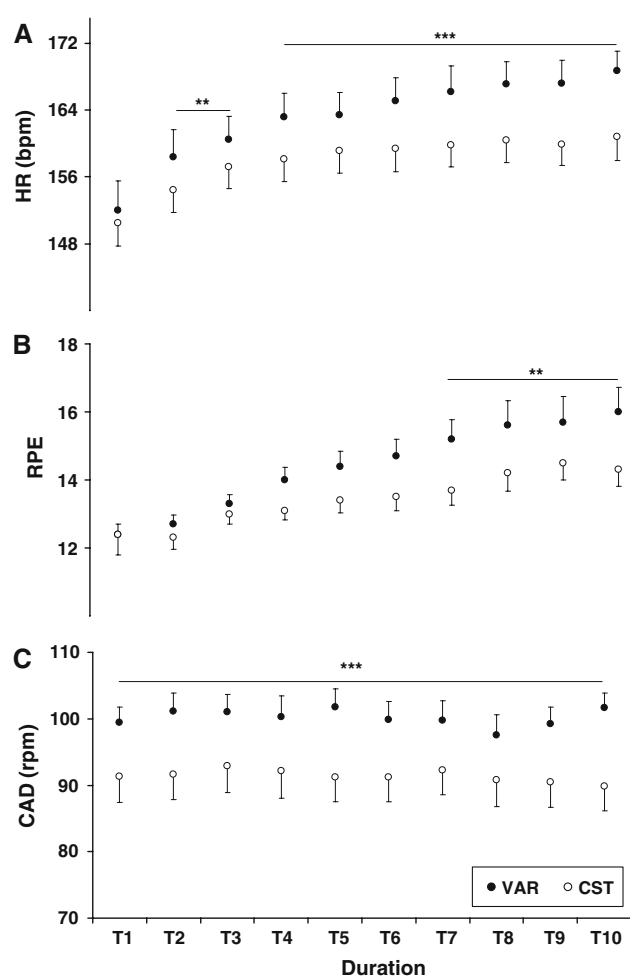
Fig. 2 Variations in heart rate (*HR*), rate of perceived exertion (*RPE*) and cadence during the two cycling conditions. CST: constant power output condition (*white*), VAR: Variable power output condition (*black*). These variables were recorded after each of the ten periods (T1–T10) corresponding to 3 min 20 s for both conditions. Mean ± SE. Significantly different from the CST condition at the same time: ***P < 0.01, ***P < 0.001

($P < 0.05$) greater during VAR (162 ± 9 bpm) than during CST condition (157 ± 8 bpm). In the VAR condition, HR and RPE values from T2 to T10 were significantly different ($P < 0.01$) from those at T1. In the CST condition, HR and RPE increased significantly ($P < 0.01$) during cycling exercise, from T2 and T7, respectively. HR values were significantly different ($P < 0.01$) between the two cycling conditions at the same time interval, from T2 to the end of exercise. RPE values were significantly different between the two conditions from T7 to T10 (see Fig. 2). Average cadence did not change during the VAR cycling and was significantly greater ($P < 0.001$) for VAR (99 ± 2 rpm) than for CST condition (90 ± 1 rpm).

At the end of exercise, [La] was significantly ($P < 0.001$) greater for VAR (11.2 ± 4.1 mmol L^{-1}) than for CST protocol (6.1 ± 2.7 mmol L^{-1}).

Neuromuscular fatigue

The MVC torque measured before exercise was similar in the two conditions (CST: 229 ± 53 N m; VAR: 214 ± 34 N m). MVC torque was significantly ($P < 0.01$) reduced after exercise, -16 ± 13 N m for CST, and -26 ± 15 N m for VAR. MVC torque reduction was significantly ($P < 0.01$) greater after VAR compared to CST (Fig. 3a). Pre-exercise VAL was similar for the CST ($98.1 \pm 2.0\%$) and VAR condition ($98.0 \pm 1.4\%$). VAL was significantly ($P < 0.05$) reduced after both cycling trials, to $97.4 \pm 1.6\%$ for CST and to $96.4 \pm 2.2\%$ for VAR.
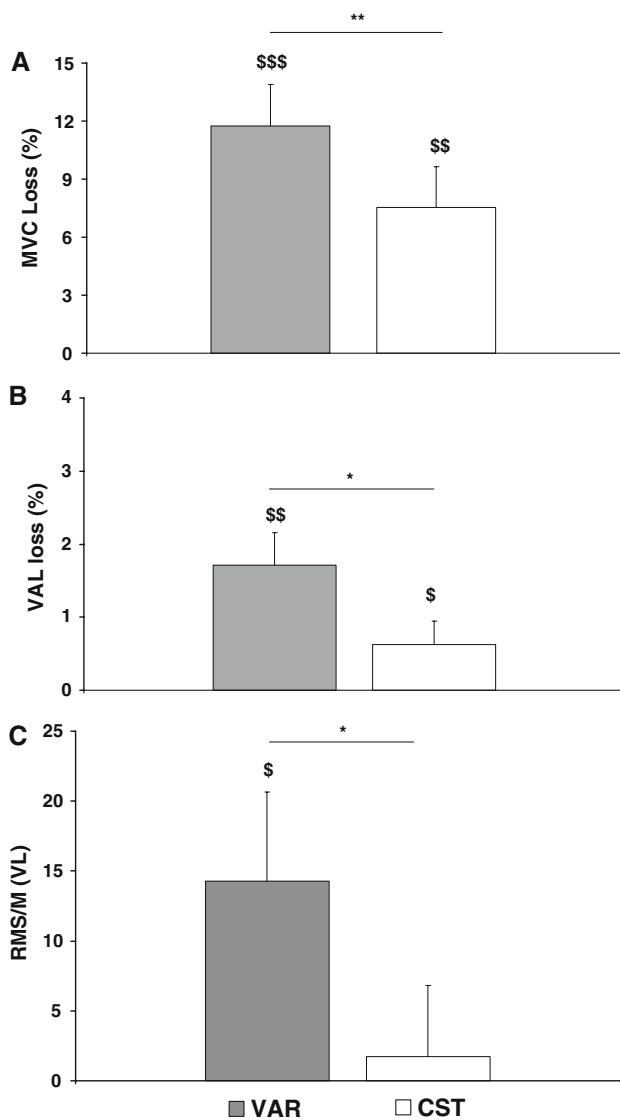
The decrease of VAL after VAR was significantly ($P < 0.05$) greater compared to CST (Fig. 3b). The RMS/M ratio before exercise was similar in the two conditions ($P = 0.28$). Exercise-induced reduction in this RMS/M ratio was significantly ($P < 0.05$) greater following VAR compared to CST (Fig. 3c).

Electrically evoked torque amplitudes before CST (Pt: 33.2 ± 6.4 N m; Dt: 73.6 ± 13.9 N m) were similar to pre-exercise VAR values (Pt: 32.6 ± 4.4 N m; Dt: 78.9 ± 15.9 N m). The reduction in Dt amplitude ($P < 0.01$) was significantly greater after VAR (-7.6 ± 7.1 N m) than after CST (-4.0 ± 3.0 N m) (Fig. 4b). The exercise-induced reduction ($P < 0.01$) in Pt amplitude (-3.0 ± 2.5 N m for CST, -3.9 ± 3.7 N m for VAR) was not significantly different between the two conditions (Fig. 4b).

The M-wave amplitude of VL muscle was significantly reduced to the same extent after the two protocols ($\approx -6.5\%$).

Discussion

The aim of the present study was to compare neuromuscular fatigue of the quadriceps muscle following constant versus highly variable power output cycling exercise. In agreement with previous studies (Lepers et al. 2000, 2002; St Clair Gibson et al. 2001; Bentley et al. 2000), our
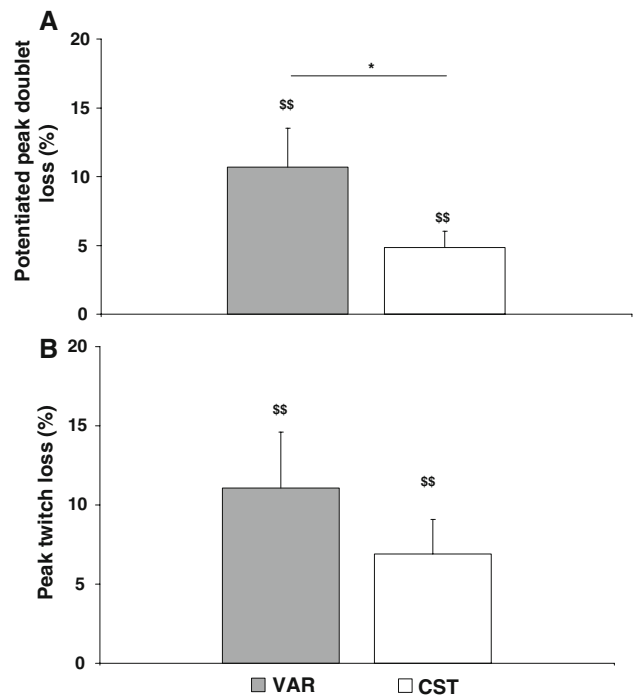
Fig. 3 **a** Reductions in maximal voluntary contraction (*MVC*) torque immediately after constant power output condition (*CST*) versus variable power output condition (*VAR*) for each subject. **b** Reductions in voluntary activation level (*VAL*) after CST versus VAR for each subject. **c** Reductions in the RMS/M ratio for VL muscle. Mean ± SE. Significant loss: $P < 0.05$, $\$\$P < 0.01$, $\$\$\$P < 0.001$; VAR > CST: $*P < 0.05$, $**P < 0.01$

Fig. 4 Peak torque changes for potentiated doublet (**a**) and twitch (**b**) immediately after the cycling exercise. *CST* Constant power output condition (*white*), *VAR* variable power output condition (*grey*). Mean ± SE. $\$\$$ Significant loss ($P < 0.01$). $*$VAR > CST ($P < 0.05$)

findings demonstrated a reduction of MVC following both protocols. However, the present study is the first that evidenced a greater reduction of maximal strength capacity of the knee extensor muscles after a variable compared to a constant power output condition. Furthermore, the greater MVC torque reduction following the variable protocol resulted from greater alterations of both central and peripheral fatigue mechanisms.

The method of superimposed stimulation during MVC used here had been successfully included to investigate the voluntary activation level of the quadriceps muscle (Miller et al. 1999). A reduction in VAL estimated by this technique suggested that a reduced number of motor units was voluntarily recruited during MVC at the end of exercise. In this experiment, the decrease in VAL was significantly greater after VAR than after CST protocol, suggesting that central fatigue was greater after VAR. Other methods used for estimating alterations to central activation compare the changes in maximal EMG with the changes in M wave. RMS/M ratio for VL muscle was significantly reduced following VAR while it was unchanged after CST protocol. In this experiment, both methods used for estimating central activation attested that central fatigue contributed to the reduction of MVC, and to a greater extent after the variable power output exercise. Nevertheless, by measuring an activation deficit with these techniques, it was impossible to determine if the central fatigue originated from supraspinal and/or spinal mechanisms (Gandevia 2001).

Central fatigue can occur at levels upstream of corticospinal neurons, which could result in an impaired efficiency in generating the central command. Indeed, during prolonged exercise, the increase of lypolisis due to glycogen depletion is known to change the concentration of free tryptophan (the serotonin precursor) in the brain (Wilson and Maughan 1992; Pitsiladis et al. 2002). Increased brain serotonin has been suspected to lead to central fatigue (Davis and Bailey 1997). Since variable intensity exercise could induce greater glucose oxidation compared to constant-load cycling (Palmer et al. 1999), the serotoninergic hypothesis might be responsible to the greater central fatigue observed in the former condition. Nevertheless, in the present study, it is suspected that exercise duration ($\approx$30 min) was not sufficient to involve significantly different glycogen depletion between the two conditions (Ball-Burnett et al. 1991).

Central fatigue could also originate from spinal mechanisms, e.g., from peripheral reflex inhibition of the α-motoneuron pool (Garland and McComas 1990) and/or disfacilitation of the α-motoneuron by muscle spindle afferents (Bongiovanni and Hagbarth 1990). Therefore, it is possible that the higher activation deficit after VAR cycling was in part due to greater muscle afferent inhibition inputs. It is known that metabolite accumulation and ischemia could cause an increase of the group III and IV afferents

discharge. The production of lactic acid results in a lowering of both muscle and blood pH, due to the dissociation of lactic acid into lactate and H^+ ions (Juel 1998). Indeed, greater [La] observed at the end of VAR exercise corroborates the findings of Palmer et al. (1999) who demonstrated higher lactate concentrations during stochastic exercise than during constant-load riding.

Muscle fatigue is also generally accompanied by a decline in electrically evoked torque. In agreement with previous works examining prolonged cycling exercise (Booth et al. 1997; Lepers et al. 2000), changes in isometric twitch tension were found in the present study. However, the reduction of peak twitch torque was not significantly different between protocols. This corroborates recent observations that challenged the use of peak twitch to estimate peripheral fatigue of the quadriceps muscle after prolonged exercise (Millet et al. 2002). The net twitch tension depends on both potentiation and fatigue-associated effects (Rassier and Macintosh 2000). Recently, Place et al. (2007) suggested that the potentiated peak doublet should be adopted to characterise contractile impairment of the quadriceps muscle. In our experiment, potentiated peak doublet reduction was greater after the variable intensity exercise compared to the constant power output protocol. Even if the precise mechanisms underlying the electrically evoked torque reduction are not known, several processes might be impaired, including reduced Ca^{2+} release from the sarcoplasmic reticulum (Westerblad et al. 1993) and reduced myofibrillar cross-bridge interaction (Davies and White 1982). These alterations in turn may be due to metabolic changes induced by exercise such as accumulation of H^+ and inorganic phosphate.

Moreover, high-muscle tension associated to high power output (>MAP) during variable intensity protocol might influence motor unit recruitment in comparison with constant intensity cycling. For example, Palmer et al. (1999) showed that the number of depleted type II fibres was relatively important ($\approx$10%) after variable intensity cycling while it was negligible after steady state cycling. A greater glycogen depletion of type II fibres during VAR exercise might also explain the greater reduction of MVC torque compared to constant intensity cycling.

The difference in muscle fatigue between VAR and CST conditions was corroborated by the feeling of exertion since mean RPE reached 16 at the end of variable intensity cycling versus 14 after the constant protocol. In the same way, heart rate was significantly higher during VAR compared to CST. These results are not in agreement with recent studies having used similar (Bernard et al. 2007; Lepers et al. 2008) or longer (Liedl et al. 1999; Palmer et al. 1999) exercise durations, where no additional physiological stress was observed by varying power compared to constant power effort. The main difference between the

present study protocol and previous works is represented by the amplitude of power output variation. In the present experiments, the variation of intensity reached 150 and 200% of MAP. Although respiratory exchange ratio was not quantified in this study, we can consider that the anaerobic metabolism significantly contributed to the power production during VAR protocol. Previous studies, comparing equal-work submaximal (<MAP) and supramaximal (>MAP) exercise, demonstrated that HR increase during recovery was significantly greater following supramaximal compared to submaximal exercise (Laforgia et al. 1997). This was attributed to greater postexercise oxygen consumption. According to Laforgia et al. (1997), lactate metabolism could explain some of the differences in oxygen consumption following supra versus submaximal exercise. In our study, the greater anaerobic demand during the variable high-intensity cycling could be responsible for the greater HR responses during exercise.

Freely chosen cycling cadence was higher (≈10%) during variable power output exercise than during constant-load exercise. It has been previously demonstrated that, in a range of 20% around freely chosen cadence, imposed cycling cadence did not influence cardiovascular drift or muscle fatigue during a 30-min cycling exercise (Lepers et al. 2001a, b). Therefore, in the present study, the higher cycling cadence during VAR probably did not influence the cardiovascular responses, or the muscle fatigue.

In conclusion, in contrast to Lepers et al. (2008) study, in which a relatively low variation of power output (70% of MAP ± 15%) was used, the present results support the hypothesis that high variations in power output, including efforts beyond the MAP, exacerbated neuromuscular fatigue and cardiovascular responses compared to constant power cycling exercise. In practical terms, although small variations of power output (<MAP) seem to be effective for improving time trial performance (Swain 1997; Liedl et al. 1999), these results suggest that large variations of power output (>MAP) could impair time trial performance. It seems thus that the effect of power output variation on time trial performance depended on the range of these variations. Concerning cycling training methods, these findings could well support the interest of including supramaximal intensity exercises in training programs for cycling competition. Such speculations need however to be confirmed by analysing the chronic (training) effect of variable versus constant power output cycling training.

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