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Romuald Lepers, Jean Theurel, Christophe Hausswirth, Thierry Bernard

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Neuromuscular fatigue following constant versus variable-intensity endurance cycling in triathletes

R. Lepers[a], J. Theurel[a], C. Hausswirth[b], T. Bernard[c]

[a] University of Burgundy, Faculty of Sport Sciences, INSERM, U887, BP 27877, F-21078 Dijon cedex France

[b] Laboratory of Biomechanics and Physiology, French National Institute of Sport and Physical Education (INSEP), Avenue du Tremblay, 75012 Paris, France

[c] Department of Sport Ergonomic and Performance, UFR STAPS, University of Sud Toulon-Var, BP 132, 83957 La Garde Cedex, France

Corresponding author :

R. Lepers
Université de Bourgogne
INSERM U 887
BP 27877
21078 Dijon cedex
France
Tel : +33.3.80.39.67.60
Fax : +33.3+80.39.67.02
Email : romuald.lepers@u-bourgogne.fr

Abstract

The aim of this study was to determine whether variable power cycling produced greater neuromuscular fatigue of knee extensor muscles than constant power cycling at the same mean power output. Eight male triathletes (age : 33 ± 5 yr, mass : 74 ± 4 kg, $\dot{V}O_{2max}$: 62 ± 5 ml.kg^{-1}.min^{-1}, maximal aerobic power : 392 ± 17 W) performed two 30 min trials on a cycle ergometer in a random order. Cycling exercise was performed either at a constant power output (CP) corresponding to 75% of the maximal aerobic power (MAP) or a variable power output (VP) with alternating $\pm$ 15%, $\pm$ 5%, and $\pm$ 10% of 75% MAP approximately every 5 min. Maximal voluntary contraction (MVC) torque, maximal voluntary activation level, and excitation-contraction coupling process of knee extensor muscles were evaluated before and immediately after the exercise using the technique of electrically evoked contractions (single and paired stimulations). Oxygen uptake, ventilation, and heart rate were also measured at regular intervals during the exercise. Averaged metabolic variables were not significantly different between the two conditions. Similarly, reductions in MVC torque ($\approx$ -11%, P < 0.05) after cycling were not different (P > 0.05) between CP and VP trials. The magnitude of central and peripheral fatigue was also similar at the end of the two cycling exercises. It is concluded that following 30 min of endurance cycling, semi-elite triathletes experienced no additional neuromuscular fatigue by varying power (from $\pm$ 5 to 15%) compared with a protocol that involved a constant power.

KEYWORDS : Maximal voluntary contraction, Central activation, Twitch, M-wave, Triathlon

Introduction

Neuromuscular fatigue, which is characterized by an exercise-induced reduction in maximal voluntary contraction (MVC) force, can originate from sites located proximal (central fatigue) or distal (peripheral fatigue) to the neuromuscular junction.[1] Previous studies conducted in our laboratory have demonstrated neuromuscular fatigue of the knee extensor muscles following prolonged (> 30 min) cycling exercise.[2,3,4] The exercise-induced reduction in MVC torque of the quadriceps muscle resulted from changes in central and peripheral processes such as decreased neural input and/or alterations in excitation-contraction coupling process. The twitch superposition (or interpolation) technique is used to quantify central fatigue by determining the reduction in voluntary activation. Voluntary activation is quantified by comparing the twitch amplitude superimposed during the MVC to the twitch amplitude evoked on when the muscle is at rest.[5] For example, following 30 min cycling exercise performed at an intensity corresponding to 80% of the maximal aerobic power (MAP), a 12% reduction in MVC torque of the knee extensors was accompanied by a reduction in voluntary activation of 13-16% although this depended on the rate of pedalling.[3] Peripheral mechanisms will also contribute to muscle fatigue. For example, a decrease in maximal evoked twitch tension was observed following prolonged cycling exercise.[2,3,4] In contrast, changes in compound muscle action potential (ie M-wave) that is a index of neuromuscular transmission and action potential propagation in muscle fibres is not systematically observed after cycling exercise.[6]

In all the aforementioned studies, neuromuscular fatigue was examined while subjects performed the cycling exercise at a constant power output at a percentage of the MAP. With the exception of time trials, cycling and triathlon competitions are rarely performed at a constant power output.[7,8,9] For example, the constant power output profile is not observed within the groups of triathletes who cycle in sheltered position inducing several changes of pace.[7] Variation of power output profile, including efforts close to the maximal aerobic power, may induce higher metabolic fatigue during the cycling exercise.[7] However, it is not known whether variation of power output would result also in greater neuromuscular fatigue, compared with constant power cycling.

Previous research suggested that varying power slightly (±5%) to counter external conditions may result in improved performance during cycling time trailing,[10,11] without additional physiological stress compared constant power trial.[12] In contrast, other findings supported the concept of relatively constant pacing in middle distance events with negative consequences for even small variations in this strategy.[13,14,15] For example, Palmer et al.[13] showed that cycling time trial performance following variable intensity was significantly impeded when compared with constant load work at the same absolute intensity and duration.

In triathlon performance, the exercise intensity can be relatively constant during flat cycling courses and when triathletes perform the cycle bout as an individual time trial i.e. when drafting is not allowed. In contrast, a constant power output profile is not observed when external conditions change due to hills, technical bike course, or wind variations or when triathletes cycle in a sheltered position inducing frequently changes in pace and intensity.[7,9] Interestingly, a recent study found that varying power output from 5 to 15% of mean power during 20-km cycling in triathlon resulted in decreased performance in the subsequent 5-km run in comparison with a constant power output cycling strategy.[16] These authors suggested that the alteration in running performance could be due to greater neuromuscular fatigue induced by variable power cycling.

Therefore this study tested the hypothesis that variable power cycling produced greater neuromuscular fatigue than constant power cycling. The purpose of the study was to examine peripheral and central components of fatigue associated with the reduction in MVC torque of the quadriceps muscle following cycling at a variable power compared with cycling at a constant power when the average power was similar.

Methods and procedures

Subjects

Eight well-trained males triathletes (age: 33 ± 5 (SD) years, weight : 74 ± 4 kg , height : 180 ± 4 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. The sub-elite subjects had regularly trained in cycling and triathlons for 5.6 ± 1.0 years prior to the study. During the previous two months, subjects participated in an average training each week of 10.2 ± 2.1 km of swimming, 210 ± 40 km of cycling and 39 ± 7.1 km running. This training was conducted in an average number of training session per week of 3.9 ± 0.3 for swimming, 2.9 ± 0.4 for cycling and 3.1 ± 0.4 for running.

Maximal cycling test

During the initial session which took place at least 5 days before the experiment, each of the 8 subjects performed a continuous, incremental cycling test. The test began at 100 W for 2 min, after which the power output was increased by increments of 30 W every 2 min until volitional exhaustion. During this incremental exercise, subjects breathed through a face mask and respiratory gas was monitored breath-by-breath (CPX, Medical Graphics, St Paul, MN, USA). Before each test, the system was calibrated with 2-L Rudolph syringe and gases of known concentrations. The following gas exchange variables were quantified : oxygen uptake ($\dot{V}O_2$), minute ventilation ($\dot{V}_E$), Heart rate (HR) values were monitored on-line using a Polar unit (S610i, Polar Electro, Kemple, Finland). Expired gas and HR values were averaged every 10 s. The four highest consecutive $\dot{V}O_2$ values recorded during the last minute were averaged to determine $\dot{V}O_{2max}$ (62.2 ± 5.3 mL.kg^{-1}.min^{-1} on average for our subjects). The MAP output was defined as the highest power completed for 2 min (392 ± 17 W). Thirty min after the $\dot{V}O_{2max}$ test, subjects familiarized themselves with the Biodex measurement apparatus and the transcutaneous stimulation for strength testing.

Experimental protocol

Each subject performed two 30 min cycling trials at a freely chosen cadence : one at a constant power output (CP) and one at a variable power output (VP) with alternating $\pm 15\%$, $\pm 5\%$ and $\pm 10\%$ of 75% MAP approximately every 5 min. The CP condition consisted of 75% of MAP (294 ± 13 W). In the VP condition, the power output was alternated in 6 periods at : +15%, -15%, +5%, -5%, -10%,

and +10% of 75% MAP i.e. 86.25%, 63.75%, 78.75%, 71.25%, 67.50%, and 82.50% MAP respectively. The exact duration of the 6 periods was calculated to produce the same work for each period (see Figure 1). The present cycling protocol mimicked the protocol used in a previous study that assessed the effect of power output variation on running performance.[16] During the first and the last periods of VP condition, subjects were required to sustain a high power output, to simulate the race situation. Indeed, during a sprint distance triathlon with drafting, triathletes usually perform high intensity cycling at the beginning of bike course in order to catch a cycling pack, and also at the toward the end of the cycling course, to enter with the transition area in the best position. [7, 16,17]

Before and immediately after the cycling exercise, neuromuscular tests were performed to quantify fatigue of the knee extensor muscles. The two experiments were performed for each subject at the same time of the day and were separated by at least seven days.

Neuromuscular tests

A standardized warm-up period was carried out by each subject before each testing session. It consisted of 5 min cycling at 33% of MAP, and 5 min at 50% of MAP. After the warm-up period, each subject was required to perform the same protocol with the right knee extensor muscles.

The experimental 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 extensor maximal voluntary contractions (MVC) with doublets delivered 2 s before, over the isometric plateau (superimposed doublet) and 1.5 s after each MVC (potentiated doublet), to assess voluntary activation level according to the twitch interpolation technique.[5] The use of superimposed doublet at 100Hz has been shown to be as sensitive as quintuplets but is less painful for the subjects.[18] Each MVC was approximately 4 s in duration and there was 60 s of rest between MVC 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 each of the two cycling sessions. Approximately, one minute was needed to replace the subject in the testing position following cycling.

Data collection

Evoked contractions

Electrically-evoked contractions were induced by 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 cm² (10 x 5 cm) rectangular electrode (Compex SA, Ecublens, Switzerland) located in the gluteal fold opposite the cathode. The optimal intensity of stimulation (i.e., that which recruited all knee extensor motor units) 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 the peak-to-peak amplitude of the compound muscle action potential (M-wave, see Electrical recordings). The stimulus duration was 1 ms and the interval of the stimuli in the doublet was 10 ms. Once the optimal intensity was found, it was kept constant throughout the session for each subject.

Mechanical recordings

Maximal 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 movement of the upper body was limited by two cross-over 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).

Electrical recordings

EMG activity was recorded with pairs of silver chloride circular (recording diameter of 10 mm) surface electrodes (Controle Graphique Medical, Brie-Comte-Robert, France) positioned lengthwise over the middle of the muscle belly with an interelectrode (center-to-center) distance of 20 mm. These recording sites were determined in pilot testing by eliciting the greatest M-wave amplitude

for each muscle via femoral nerve stimulation.[19] The reference electrode was attached to the left wrist. Low resistance between the two electrodes (< 5 kΩ) was obtained by abrading the skin, and oil and dirt were removed from 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 = 1000), 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).

Cardiorespiratory recordings

The $\dot{V}O_2$, $\dot{V}_E$, and HR values were recorded and averaged over the last minute of each of the 6 periods (T1, T2, T3, T4, T4, T5 and T6) for VP condition and at the same corresponding times for the CP condition.

Ratings of perceived exertion (RPE)

At beginning of each test, subjects were provided with a typewritten set of standardized directions for the use of the rating of perceived exertion (RPE).[20] Subjects were instructed to judge their global effort at the end of each of the 6 periods for VP condition and at the same corresponding times for the CP condition.

Data analysis

Mechanical and EMG data

Mechanical parameters for single stimuli were analyzed and the average of three trials used for further analysis. MVC was considered as the peak force attained during the contraction and maximal voluntary activation level (VAL) was estimated according to the following formula, i.e., VAL = [1 - (superimposed doublet amplitude / potentiated doublet amplitude)] x 100.[18] In a few cases, in which the doublet was applied when the torque level was slightly below the maximal voluntary torque obtained during the MVC, a correction was applied in the original equation, as suggested by

Strojnik and Komi.[21] Peak torque (Dt) was the only parameter analyzed from the doublet torque traces. Peak twitch torque (Pt) and time to peak twitch (TPT) were analyzed from the twitch response.

M-wave peak-to-peak amplitude and duration were analyzed for the vastus lateralis (VL), vastus medialis (VM) and rectus femoris (RF) muscles with the average of the three trials used for analysis.

Cycling exercise

All experiments were conducted on an electromagnetically braked cycle ergometer (Type Excalibur, Lode, Groningen, Nederlands) 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, with toe clips attached, which allowed subjects to wear cycling shoes. Pedaling rate was recorded instantaneously from the ergocycle using a computer. The ergometer allowed subjects to keep power output constant independent of the cadence naturally adopted. No feed-back 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 ± 2°C and relative humidity of 50 ± 5 %. Two fans were placed in front of the ergocycle in order to increase cooling of the subject during cycling.

Statistical analysis

Kolmogorov-Smirnov tests were conducted before statistical analysis and confirmed that all data were normally distributed. Subsequently, a two-factor (time x power condition) ANOVA with repeated measures on time and on power condition were performed to compare the following variables measured before and after the cycling exercise : cardiorespiratory variables and RPE during the cycling test. One way-Anova was used to compare pre-exercise to post-exercise changes in neuromuscular variables between the two cycling conditions. Post hoc analysis (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).

Results

Cardiorespiratory responses

Figure 2 shows the changes in physiological parameters during the two cycling exercises. There was a significant ($P < 0.05$) main time effect for $\dot{V}O_2$, $\dot{V}_E$, HR and RPE for the VP condition. For these four parameters, values at T2, T4 and T5 were significantly different ($P < 0.01$) from those at T1. For the CP condition, no variation in $\dot{V}O_2$ and $\dot{V}_E$, was found with exercise duration but values of HR and RPE increased significantly ($P < 0.01$). $\dot{V}O_2$ and $\dot{V}_E$ values were significantly different between the two cycling conditions at the same time interval: T1, T2, and T6. HR and RPE values were significantly different between the two conditions at T1, T2, T4 and T5 (see Fig. 2).

Table 1 shows the mean values of T1, T2, T3, T4, T5, and T6 for $\dot{V}O_2$, $\dot{V}_E$, HR, and RPE. Mean $\dot{V}O_2$, $\dot{V}_E$, HR, and RPE were not significantly different between the CP and VP conditions. Cycling cadence did not change during the power variations and was not significantly different between the two power conditions (CP : 88 ± 14 rpm ; VP : 87 ± 13 rpm).

(Insert table 1)

(Insert figure 2)

Neuromuscular fatigue

The MVC torque measured before CP exercise (253 ± 60 N.m) was similar to that performed before VP exercise (256 ± 59 N.m). Figure 3 shows the variability between subjects for MVC and VAL losses. MVC torque was significantly ($P<0.05$) reduced after the cycling by 9.0 ± 5.7% for CP, and 12.8 ± 6.1 % for VP (Fig. 3A). Reductions in MVC torque were not significantly different between the two conditions. VAL assessed during the MVC before exercise was similar for the CP and VP conditions (96.0 ± 3.0 % for CP vs 95.8 ± 3.0 % for VP). The reduction in VAL was significant

(P<0.01) after both sessions (-5.0 ± 3.2 % for CP, -5.1 ± 3.9 % for VP), and the reduction was similar in the two conditions (Fig. 3B).

(Insert figure 3)

Peak twitch and peak doublet amplitude assessed before CP (Pt : 39.7 ± 3.4 N.m ; Dt : 83.9 ± 12.5 N.m) were similar to those values obtained before VP exercise (Pt : 38.2 ± 4.0 N.m ; Dt : 81.6 ± 1.2 N.m). The reduction in peak twitch amplitude (P<0.05) after exercise was not significantly different between the two conditions (Fig. 4A). Similarly, the reduction in peak doublet (P<0.01) was not significantly different between the two conditions (Fig. 4B). Time to peak twitch were similar for CP and VP conditions and were not significantly affected by the cycling exercise (two conditions averaged : Pre : 79.8 ± 20.0 ms ; Post : 72.3 ± 18.1 ms).

(Insert figure 4)

The two protocols significantly reduced M-wave amplitude of VL and RF muscles (P<0.05) to a similar extent after cycling. M-wave duration of VL, VM and RF muscles was similar before and after cycling for both conditions. (Data not presented).

Discussion

The aim of the present study was to compare neuromuscular fatigue of the quadriceps muscle between constant versus variable power cycling exercise. Our results showed that cycling trials induced a significant reduction of maximal strength of the knee extensor muscles that was not different between the constant and variable power output conditions. Furthermore, the central and peripheral mechanisms contributing to knee extensors strength loss after cycling were similar for the

constant and the variable power output protocols. These findings suggest that variations of intensity during an endurance cycling did not influence neuromuscular fatigue of knee extensors.

The similar reduction in MVC torque for both conditions was unexpected and did not support the hypothesis that fatigue of the quadriceps muscle would be greater following variable power output compared with the constant output cycling exercise. Despites the great intersubject variability, the magnitude of MVC loss ($\approx$ 11%) is consistent with previous studies that have studied similar cycling intensity and duration.[2,22] Peripheral and central mechanisms had similar contributions to the reduction of maximal strength of knee extensors for CP and VP conditions. The peak twitch and the peak doublet torque were reduced to similar magnitudes for both protocols. The precise mechanism for the reduction in electrically evoked torque is not known but several processes might be impaired, including a reduced Ca^{2+} release from the sarcoplasmic reticulum and reduced myofibrillar cross bridges interaction.[23] These alterations in turn may be due to metabolic changes induced by exercise such as accumulation of H^+ and inorganic phosphate. Similarly, the reduction of M-wave amplitude observed for VL and RF muscles did not differ between CP and VP conditions. This decreased excitability could explain in part the reduced peak twitch and doublet amplitude. However the mechanisms for the changes in M-wave amplitude such as reduced Na^+-K^+ pump activity, impaired inactivation of Na^+ channels were not assessed in this study.

Reductions in voluntary activation of the quadriceps muscle after cycling exercise has been previously studied using the using the superimposed stimulation during an MVC.[2,3,22] This method showed that the central component of muscle fatigue that contributed to the reduction of MVC torque was similar for both power protocols. However, it was not possible to determine if the central fatigue originated from a site at a supraspinal or the spinal level.[1]

The absence of difference in muscle fatigue between VP and CP conditions was corroborated by the feeling of exertion that was similar at the end of cycling because the triathletes attained a mean RPE of 16 (hard-very hard) at the end of each protocol. Mean cardiorespiratory responses to 30 min of either CP or VP exercise at the same average power output were similar, despite differences in kinetics of variation of gas exchange parameters. These results are similar to those obtained by Liedl et al.[12] and Palmer et al.[24] for longer duration exercise, 60 and 140 min respectively ; where no additional

physiological stress were observed by varying power compared with that during a constant power effort.

Variable pacing is thought to lead to excessive glycogen depletion and premature onset of fatigue. For example, Palmer et al.[24] showed that the number of glycogen-depleted type I muscle fibers at the end of 140 min cycling exercise was greater when it was performed at steady state (98%) than when it was performed at a variable intensity (59%). Conversely, the number of depleted type II fibers was lower after steady state than after variable intensity. Despite these metabolic differences, subsequent 20-km time trial performance was similar in both conditions. Bernard et al.[16] using a similar protocol to this study but in outdoor conditions suggested that the repeated increases in cycle intensity during the VP exercise and especially during the last minute of cycling may result in additional muscular fatigue. The greater fatigue during the cycling was therefore, thought to explain the decrement in running performance after VP compared with the CP cycling. However, our findings do not support this hypothesis and show that that an impaired running performance after VP cycling can not be explained by greater neuromuscular fatigue due to the cycling. Compared with the subjects of Bernard et al.[16] study, the triathletes in the present study were of sub-elite status with similar training history and experience in triathlon. Thus differences in subjects' training level between the two studies could not explain our results. Therefore, further studies are required to examine the mechanism for better running performance after constant intensity cycling exercise in triathlon. In contrast, Suriano et al.[25] examining the effect of constant (90% of the lactate threshold) versus variable (± 20% of the constant workload alternating every 5 min) power output cycling on subsequent high-intensity running performance (16.7 km.h^{-1}), found run time to exhaustion was higher after 30 min of variable cycling (~ 15 min) compared with constant cycling (~11 min). The reduced power output in the final 5 min of variable cycling protocol may allow for recovery before the transition to running and may explain why this result differs from Bernard et al.[16] study. Therefore the order of power output levels during a variable protocol may influence the measurements (running performance or neuromuscular parameters) performed immediately after cycling.

The absence of any differences in muscle fatigue between VP and CP cycling might be explained by lower variations of power output: the two higher changes corresponding to +10% and

+15% of the mean power output i.e. 82.50% and 86.25% of the MAP respectively. Greater variations of power output during cycling, close or higher than MAP, might exacerbated muscle fatigue. Changes in energy contribution, particularly from anaerobic sources during cycling with high power output variations could increase both peripheral and central fatigue. Indeed, a greater anaerobic contribution may induce an increased lactate production and protons H^+, that could in turn impair neuromuscular propagation, excitation-contraction coupling processes and intrinsic capacity of force production at the peripheral level. [26] In addition, higher metabolite accumulation and muscle tension could increase the group III and IV afferents discharge at spinal level, resulting in higher muscle activation deficit.[27] Thus, greater power output changes could result in greater alteration at peripheral and spinal level that enhance fatigue in comparison with a constant intensity exercise.

Methodological limitations of this study are the small sample size (n = 8) of sub-elite triathletes. However, level of fitness and training history of the triathletes were similar so that the subjects were homogeneous in their training and level of performance. This is important because there is evidence that patterns of leg recruitment and neuromuscular adaptations vary with the training status.[28] Nevertheless, the present findings need to be verified for a greater sample size, and for elite-level triathletes.

Conclusion

The study indicates that 30-minutes of cycling at a variable power output (from ± 5 to 15% of mean power output) compared with a constant but similar average cycling power induced similar magnitude mechanisms (central and peripheral) of neuromuscular fatigue. These data suggest that in the field of triathlon, the improved subsequent running performance after constant compared to variable-intensity cycling[16] can therefore not be explained by a lower neuromuscular fatigue. However, these findings need to be confirmed for higher various cycling intensities or /and for longer duration exercise.

Acknowledgments

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Figure Legends

Figure 1

Representation of the two cycling conditions. CP : Constant power output, VP : Variable power output. MAP : Maximal Aerobic Power.

In the VP condition, the power output alternated for 6 periods at $\pm$ 15%, $\pm$ 5%, and $\pm$ 10% of 75% MAP. The first and the last minute that were performed at a power output corresponding to 75% MAP. The duration of the periods was calculated in order to produce the same work for each period.

In both conditions, the first and the last minute of cycling were performed at constant power output (75% MAP) with a fixed cadence (90 rpm) for EMG recording ; in the remaining the time, the subjects adopted a free chosen cadence.

Figure 2

Variations in oxygen uptake ($\dot{V}O_2$), minute ventilation ($\dot{V}_E$), heart rate (HR), rate of perceived exertion (RPE) and cadence (CAD) during the two cycling conditions. CP: Constant power output condition, VP: Variable power output condition. The $\dot{V}O_2$, $\dot{V}_E$, and HR values were recorded and averaged over the last minute of each of the six periods (T1, …,T6) corresponding at a different power outputs for VP condition and at the same corresponding time for the CP condition. Mean $\pm$ SE. Significantly different from the CP condition at the same period time : * $P<0.05$, ** $P< 0.01$

Figure 3

A : Reductions in maximal voluntary contraction (MVC) torque immediately after constant power output condition (CP) vs. variable power output condition (VP) for each subject. B: Reductions in voluntary activation level (VAL) assessed during the MVC after CP vs. VP for each subject.

Figure 4

Peak torque changes for twitch (A) and doublet (B) evoked immediately after the cycling exercise. CP

: Constant power output condition, VP : Variable power output condition. Mean ± SD.
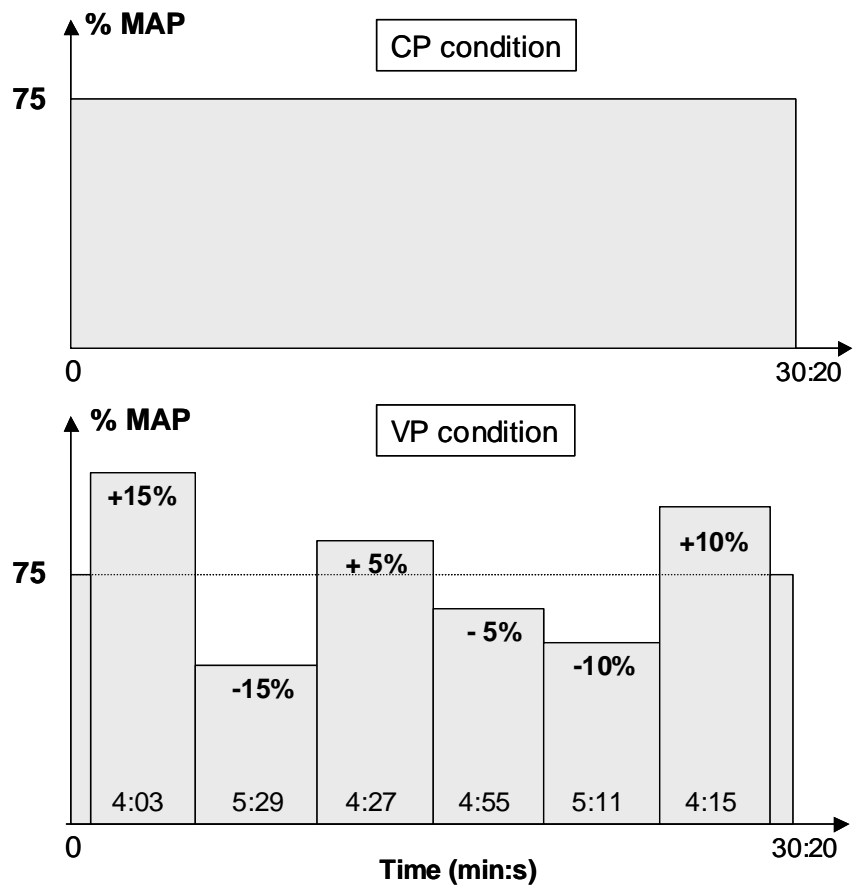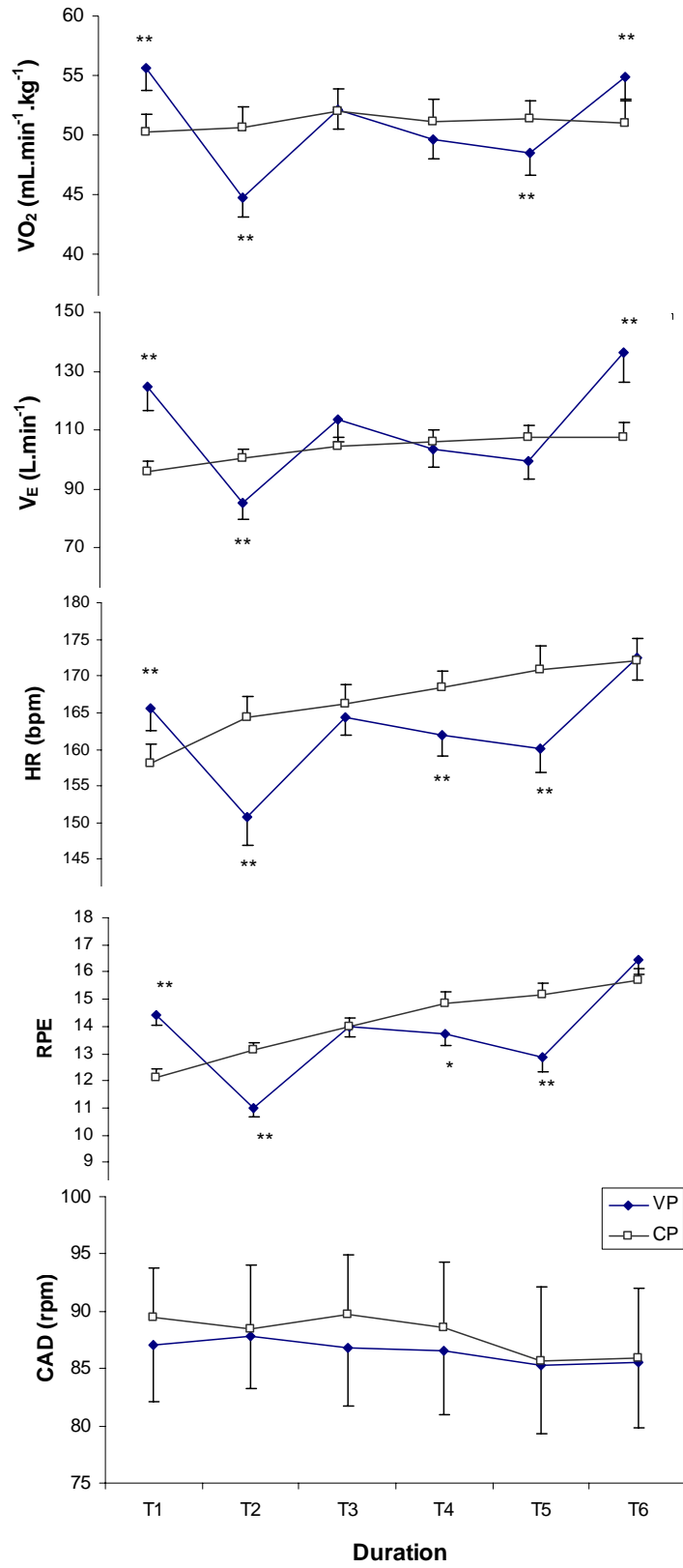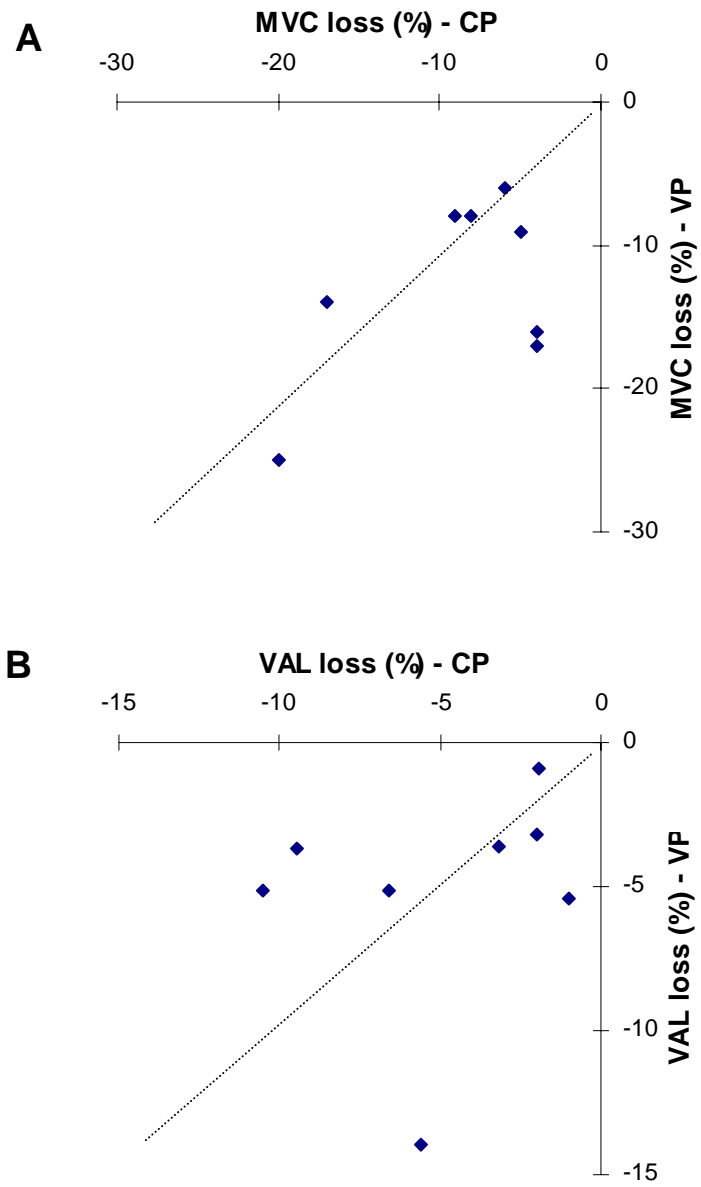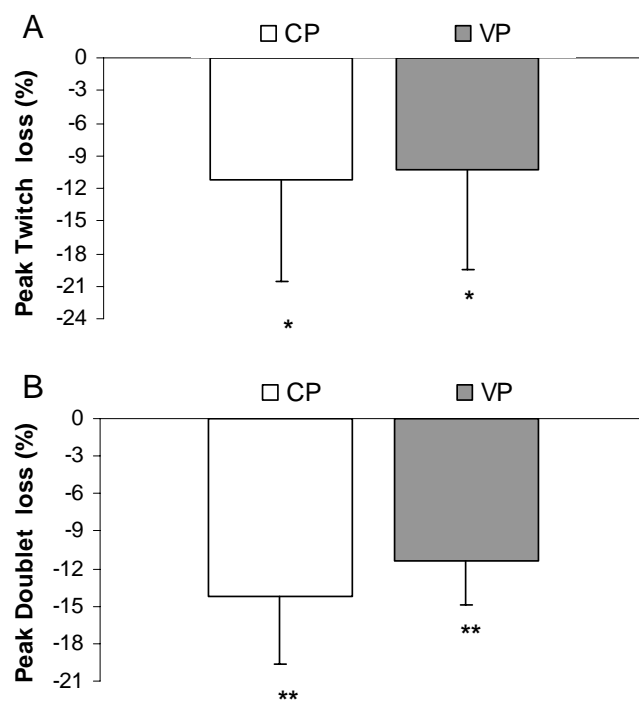
Significant loss : * P < 0.05, ** P < 0.01.

Figure 1

Figure 2

Figure 3

Figure 4

Table 1

Mean ($\pm$ SD) values of $\dot{V}O_2$ (mL.Kg^{-1}.min^{-1}), $\dot{V}_E$ (L.min^{-1}), HR (bpm), RPE, and cycling cadence CAD (rpm) for the two cycling conditions. CP : constant power output condition, VP : variable power output condition.

	$\dot{V}O_2$	$\dot{V}_E$	HR	RPE	CAD
CP	51.1 ± 4.7	103.7 ± 8.8	167 ± 8	14.1 ± 0.8	88 ± 14
VP	50.9 ± 5.0	110.5 ± 19.8	163 ± 8	13.7 ± 0.9	87 ± 13