Effect of cycling cadence on contractile and neural properties of knee extensors

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ABSTRACT

LEPERS, R., G. Y. MILLET, and N. A. MAFFIULETTI. Effect of cycling cadence on contractile and neural properties of knee extensors. *Med. Sci. Sports Exerc.*, Vol. 33, No. 11, 2001, pp. 1882–1888. **Purpose:** This study investigated the effect of prior prolonged cycling exercise performed at different cadences on subsequent neuromuscular characteristics. **Methods:** Eight well-trained triathletes sustained 80% of their maximal aerobic power during 30 min at three cadences: the freely chosen cadence (FCC), FCC−20%, and FCC+20%. Maximal isometric and concentric ($120°·s^{-1}$ and $240°·s^{-1}$) torques were recorded before and after the exercise. Central activation, neural (M-wave), and contractile (isometric muscular twitch) parameters of quadriceps muscle were also analyzed by electrical stimulation of the femoral nerve. **Results:** Reductions in maximal isometric ($P < 0.01$) and concentric torques at $120°·s^{-1}$ ($P < 0.05$) were found after exercise. Central activation levels fell significantly ($P < 0.05$) by 13–16% depending on the pedaling rate. Although the M-wave did not significantly change after exercise, the ratio EMG RMS/M-wave amplitude decreased significantly ($P < 0.01$) on both vastus lateralis and vastus medialis muscles for FCC−20% and FCC but not for FCC+20%. Significant decreases in maximal twitch tension ($P < 0.01$), maximal rate of twitch development ($P < 0.01$), and time to half relaxation ($P < 0.01$) were observed postexercise with no effect of cadence. **Conclusions:** These findings suggest that force reduction after prolonged cycling is attributable to both central and peripheral factors but is not influenced by the pedaling rate in a range of FCC ± 20%. **Key Words:** PEDALING RATE, FATIGUE, ACTIVATION LEVEL, MUSCULAR TORQUE, ISOMETRIC TWITCH

The capacity to generate force in leg muscles decreases after prolonged exercise. Maximal voluntary isometric (MVC) and dynamic muscular contractions have been found to be reduced after prolonged running, cross-country skiing, and cycling (3,5,6,19,20,27). During cycling, power output can be sustained using different combinations of pedaling rates (and thus of forces applied to the pedals), which implies different muscular recruitment (25). Indeed, for an identical power, a low pedaling rate corresponds to low muscle shortening velocities but greater muscular tension levels, and the reverse is true for high pedaling rates. However, the relative contribution of different fiber type populations at a particular pedaling rate appears to be a complex phenomenon. The glycogen depletion method has been used in a number of studies to identify muscle fibers activated in preceding cycling exercise. Gollnick et al. (17) have reported that variations in pedaling rates had no effect on fiber-type recruitment pattern in cyclists and that the major factor affecting glycogen depletion pattern was relative work rate. In contrast, Ahlquist et al. (1) showed that cycling at the same metabolic cost for 30 min at 50 revolutions per minute (rpm) rather than 100 rpm resulted in greater Type II fiber glycogen depletion. These data were consistent with the view that in cycling force development as opposed to velocity of contraction seems to determine the degree of Type II fiber recruitment.

During training or racing, experienced cyclists or triathletes usually select relative high pedaling cadence close to 80–90 rpm. Several assumptions in relation to biomechanical, physiological, and neuromuscular parameters have previously been proposed to explain the choice of such a cadence. For example, it has been shown that pedaling rate could influence the neuromuscular fatigue in working muscles (28,29). Takaishi et al. (28) found that for noncyclists the neuromuscular fatigue estimated by the integrated electromyogram as a function of time (i.e., iEMG slope) of the vastus lateralis muscle became smaller according to the increase of pedaling rates ranging from 40 to 80 rpm. Similarly, Takaishi et al. (29) have reported that the trained cyclists pedaling rate at which minimal neuromuscular fatigue was obtained was coincident with the rate which most subjects preferred. The concept of "optimal" cadence is generally supported by experimentation where cadences varied from the lowest to the highest rates. Nevertheless, in reality, extreme cadences such as 50 or 110 rpm are very rarely used by road cyclists or triathletes, and the range of cadences adopted with common gear ratios may vary from 70 to 100 rpm (20).

Because pedaling rate may influence the fiber type recruitment pattern and the degree of neuromuscular fatigue, one can speculate that the use of a particular cadence in cycling could alter differently the capacity of force production. Beelen and Sargeant (7) have studied the effect of pedaling rates on maximal power, but after a high intensity exercise of 6-min duration. There appears therefore to be a scarcity of data concerning the effects of pedaling rate during an endurance cycling exercise upon

subsequent neuromuscular characteristics. Thus, it could be interesting to identify the mechanisms and sites of fatigue in response to particular cycling cadences, in order to more fully understand the relationships between the "freely chosen cadence" and the concept of an "optimal cadence" in cycling. Moreover, the knowledge of the effects of pedaling rates upon neuromuscular characteristics could be prove useful for multi-sports events, such as triathlon, where muscular fatigue generated during cycling could influence the athletes subsequent running performances.

Failure in force production may occur at various sites along the pathway from the central nervous system through to the peripheral contractile mechanism. It is possible to estimate central fatigue using different methods. The twitch interpolation technique consists of interjecting a maximal electrical stimulus into the contracting muscle and observing twitch that is superimposed upon recordings of voluntary force (see 16). The amplitude of the force generated by electrical stimulation of the resting muscle is then compared with the additional force generated by electrical stimulation superimposed on the voluntary contraction to estimate the voluntary activation level. Another interesting method is to compare the change in the integrated electromyographic signal (iEMG) or the root mean square (RMS) with the change in the compound muscle action potential (also named M-wave) amplitude. A reduction in the RMS without a reduction in M-wave amplitude (decrease in RMS/M-wave ratio) may be interpreted as central activation failure (10,18). Peripheral mechanisms, such as neuromuscular transmission, conduction of impulses in the muscles fibers, and excitation-contraction coupling, can also be involved in the fatigue process, singly or together. One technique of investigating neural and contractile properties of muscle consists of stimulating motor nerves and analyzing the subsequent muscle compound action potentials (M-waves) and isometric twitch force. Change in M-wave characteristics (duration, amplitude, or area) reveals alterations of the transmission of this signal across the neuromuscular junction and/or the excitability of the muscle membrane (15). Alternatively, changes in excitation-contraction coupling can be evaluated by modifications of isometric muscular twitch (14).

Therefore, the aim of this study was to investigate the changes in contractile and neural properties of knee extensors after a prolonged cycling exercise performed at different pedaling rates. It was hypothesized that different cadences and hence different muscle contraction patterns would differently alter the capacity to generate force in the quadriceps muscle. In addition to strength measurements, the changes that may occur at several sites along the pathway of force production were estimated by noninvasive measurements of central activation, M-wave amplitude and duration, and contractile properties by using electromyography and electrical stimulation techniques.

MATERIALS AND METHODS

Subjects

Eight well-trained male triathletes volunteered to participate in this study. They were informed in detail before the tests as to the nature of the experiment and the possible risks. Written consent was given by each subject and a local Ethics Committee for the protection of individuals gave prior approval to the project before its initiation.

The average age of the subjects was 28 ± 2 (SD) yr. Their body weight was 74 ± 5 kg, and their average height was 183 ± 5 cm. All subjects had regularly trained in cycling for at least 4 yr previous the study. The $\dot{V}O_{2max}$ of the triathletes averaged 64.1 ± 4.5 mL·kg^{-1}·min^{-1}.

Data Collection and Analysis

Strength measurement. Instantaneous torque of the quadriceps muscle at various preset constant angular velocities was recorded using a Biodex isokinetic dynamometer (Biodex Corporation, Shirley, NY). Subjects were placed in a 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 an abdomen belt. 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 by using a strap. During concentric testing, each subject extended his right knee as forcefully as possible throughout the full range of motion.

Muscular twitch and voluntary activation. To test the effects of the 30-min cycling exercise performed at different pedaling rates upon muscular isometric twitch, M-wave, and voluntary activation of the quadriceps muscle, the femoral nerve was electrically stimulated at rest in the seated position as previously described. Isometric tension developed by the muscular twitch was recorded using the isokinetic dynamometer. Electrical stimulations were given using a high-voltage stimulator (model DS7, Digitimer Stimulator, Hertfordshire, England). The femoral nerve was stimulated by using a monopolar cathode ball electrode (0.5 cm diameter) pressed into the femoral triangle by the experimenter. The site of stimulation was marked for postexercise stimulations. The anode was a rectangular electrode (Medicompex SA, Ecublens, Switzerland), 50 cm^2 (10 cm × 5 cm), located in the gluteal fold opposite the cathode. To find the maximal twitch, the amperage (20–100 mA) of a 400-V rectangular pulse (2 ms in duration) was progressively increased to obtain a plateau in the twitch torque. Once the plateau was achieved, five stimuli, each separated by 5 s, were given. Maximal twitch tension (Pt), twitch time to peak (CT), and time to half relaxation (HRT) were determined from the averaged mechanical response of the five stimuli. CT and HRT were measured from the force onset to peak force (Pt) and from the force peak to half the peak value, respectively. Maximal rate of tension development (RD) and relaxation (RR) were calculated as the maximum df/dt of the averaged force trace. Voluntary activation level was calculated by expressing every increment in torque

evoked during the maximal isometric contractions (twitch size when superimposed) as a fraction of the amplitude of the response evoked by the same stimulus in the relaxed muscle (twitch size evoked at rest). The activation level (AL) was then quantified using the formula (16):

$$AL(\%) = [1 - (\text{twitch size when superimposed}/\text{twitch size evoked at rest})] \times 100$$

EMG and mechanical recording. Recording of muscle electrical activity (EMG) on the vastus lateralis (VL) and the vastus medialis (VM) muscles was achieved by means of two pairs of silver-chloride surface electrodes fixed to the right leg. Low impedance at the skin-electrode surface was obtained ($Z < 5$ kΩ) by light abrasion of the skin. Electrodes were coated with electrode gel and fixed lengthwise over the middle of the muscle belly (13) with an interelectrode distance of 16 mm and with the reference electrode being fixed on the right wrist. Myoelectric signals were amplified with a bandwidth frequency ranging from 1.5 to 500 Hz (Common Mode Rejection Ratio, CMRR = 90 dB; Z input = 100 MΩ; gain = 1000). Torque and EMG signals were digitized on-line (sampling frequency 1000 Hz) using a digital computer (IPC 486). EMG signals were analyzed for VL and VM muscles to determine: duration (D) and peak-to-peak amplitude (A) of the M-wave. During isometric actions, EMG signals were quantified using the RMS, which was calculated over a 1-s period after the torque had reached a plateau. To examine separately central and peripheral activation during contraction, the changes in RMS and in M-wave amplitude (A) were compared using the ratio RMS/A for both VL and VM muscles.

Cycling test. All experiments were conducted on an electromagnetically braked cycle ergometer (Type Excalibur, Lode, Groningen, The Netherlands) where the seat and handlebars were fully adjustable both vertically and horizontally to replicate habitual positioning of subjects on their own bicycles. The ergometer was also equipped with racing pedals and toe clips, allowing subjects to wear cycling shoes. Pedaling rate was recorded instantaneously from the ergocycle by using a computer. The ergometer allowed subjects to keep power output constant independent of the pedal rate they naturally adopted.

Protocol and Experimental Procedures

During the initial session that took place at least 5 d before the experiment, each of the eight subjects performed a continuous, incremental cycling test. The test began with a warm-up at 150 W for 10 min, after which the power output was increased by increments of 25 W every 2 min until volitional exhaustion. During this incremental exercise, oxygen uptake ($\dot{V}O_2$), minute ventilation ($\dot{V}E$), and respiratory exchange ratio (RER) were continuously measured every 15 s by using a telemetric system collecting gas exchanges (Cosmed K4$_{RQ}$, Rome, Italy). The maximal aerobic power output (MAP) was the highest power completed for 2 min (384 $\pm$ 31 W). Thirty minutes after the $\dot{V}O_{2\text{max}}$ test, subjects familiarized themselves with the Biodex-iso-kinetic measurement apparatus and the transcutaneous stimulation for strength testing.

The experiment consisted of sustaining 80% of MAP for 30 min. Three different testing sessions corresponding to three different cadence tests were conducted. Each session was separated by a 72-h rest period. The first session was performed at a freely chosen cadence (FCC) that corresponded to the cadence subjects spontaneously adopted within the first 5 min. During the last 25 min of this test, the subjects were asked to keep the same cadence. For the two other tests, subjects rode in a random order at FCC−20% or FCC+20%.

A standardized warm-up period was carried out by each subject before each testing session. It consisted of 10 min cycling at 33% of MAP, followed by several submaximal concentric contractions of the quadriceps muscle in the testing position. After the warm-up period, the femoral nerve of the right leg was electrically stimulated to obtain the maximal M-wave and the associated twitch torque evoked at rest. Subjects were then asked to perform two MVC (4–5 s) at 60° knee flexion (0° = knee fully extended). During the two MVC, a superimposed twitch stimulation was given. Then, three maximal concentric knee extensions at two angular velocities (120 and 240°·s^{-1}) presented randomly (starting position corresponded to thigh/shank angle of 90°; range of motion = 90°; full extension = 0°) were executed. Verbal encouragement was given to the subjects during all maximal contractions. A rest period of only 40 s was permitted between each set of muscular actions to minimize the effects of recuperation during the post test and to avoid the effects of postactivation potentiation. Maximal torque (MT) during concentric (MT$_{120}$ and MT$_{240}$) and isometric (MT$_{iso}$) contractions were determined from the highest values of the trials. The same experimental procedure (lasting approximately 10 min) was carried out before and immediately after each of the three 30-min cycling exercises. Approximately, 1 min was necessary to replace the subject in the testing position after the cycling exercises.

Statistical Analysis

All data are expressed as mean $\pm$ SD. A two-way ANOVA [exercise (pretest, posttest) $\times$ cadence] was performed to examine the effect of exercise and cadence on strength (MT, RMS), twitch force (Pt, CT, HRT, RD, RR, AL) and M-wave (A, D) parameters. When F-ratios were significant ($P < 0.05$) from the ANOVA, post hoc multiple comparisons using Tukey's procedure were conducted.

RESULTS

The FCC averaged 86 $\pm$ 4 rpm, thus: FFC−20% and FCC+20% corresponded to 69 $\pm$ 3 and 103 $\pm$ 5 rpm, respectively.

Muscular Strength

All changes in muscular strength are presented in Table 1. After 30 min of cycling, MT values were significantly lower

TABLE 1. Peak muscular torque (MT) for isometric and concentric (120 and 240°·s^{-1}) contractions before (Pre) and after (Post) the 30-min cycling exercise at the three pedaling rates, FCC−20%, FCC, and FCC+20%. FCC: free chosen cadence; values are means ± SD.

	FCC−20%			FCC			FCC+20%		
	Pre	Post	P<	Pre	Post	P<	Pre	Post	P<
MT$_{iso}$ (Nm)	320 ± 53	281 ± 48	0.01	335 ± 30	293 ± 34	0.01	317 ± 27	287 ± 30	0.01
MT$_{120}$ (Nm)	200 ± 25	190 ± 25	0.02	209 ± 26	191 ± 18	0.02	202 ± 21	191 ± 29	0.03
MT$_{240}$ (Nm)	148 ± 20	147 ± 20	NS	153 ± 23	145 ± 18	NS	149 ± 16	144 ± 27	NS

under isometric ($P < 0.01$) and 120°·s^{-1} concentric contractions ($P < 0.03$), whereas there was no significant difference at 240°·s^{-1}. Under isometric conditions, MT$_{iso}$ decreased by 12 ± 7%, 13 ± 8%, and 9 ± 7% for FCC−20%, FCC, and FCC+20%, respectively. At 120°·s^{-1}, the decrease was lower than under isometric conditions: 5 ± 6%, 8 ± 8%, and 6 ± 7% for FCC−20%, FCC, and FCC+20%, respectively. No significant difference of MT loss was found between the three cadences regardless of the angular velocity.

Central Activation

The AL estimated by the twitch interpolation method fell postexercise by 13 ± 8% (from 84 ± 11 to 73 ± 11%, $P < 0.01$), 16 ± 14% (from 79 ± 12 to 65 ± 15%, $P < 0.05$), 15 ± 11% (from 82 ± 12 to 70 ± 12%, $P < 0.05$) at FCC−20%, FCC, and FCC+20%, respectively (Fig. 1). Analysis of variance revealed no effect of cadence.

Muscular Twitch Force and M-Wave

As shown in Table 2, significant reductions in Pt ($P < 0.01$), HRT ($P < 0.01$), and RD ($P < 0.02$) were observed after the 30-min cycling exercise, whereas CT and RR were unchanged. Again, no effect of cadence was found.

The peak-to-peak amplitude of the M-wave did not fall significantly after cycling exercise for VL or for VM muscles whatever the cadence (Table 3). Similarly, potential duration did not change postexercise.

Muscular Activity

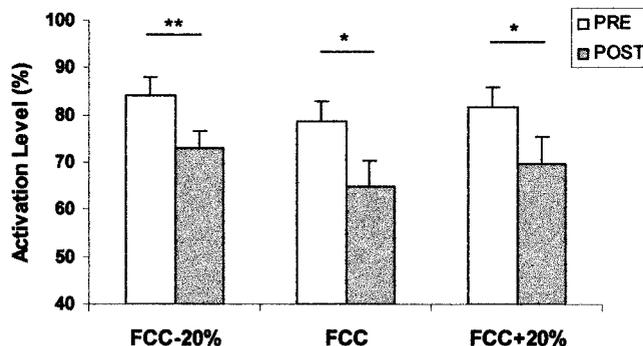Figure 2 demonstrates that after the 30-min cycling exercise, the RMS/M-wave amplitude ratio was significantly

FIGURE 1—Mean values of muscular activation levels before (PRE) and after (POST) the 30-min cycling exercise at the three pedaling rates, FCC−20%, FCC, and FCC+20%. Error bars: SD. * $P < 0.05$ and ** $P < 0.01$: statistically significant compared with the PRE-cycling exercise condition.

lower at FCC−20% and FCC but not at FCC+20%. For the VL muscle, the relative decrease was 18 ± 4% ($P < 0.05$), 30 ± 5 ($P < 0.01$), 6 ± 10% at FCC−20%, FCC, and FCC+20%, respectively. For the VM muscle, the decrease was 31 ± 5% ($P < 0.01$), 30 ± 6 ($P < 0.01$), 15 ± 7% at FCC−20%, FCC, and FCC+20%, respectively.

DISCUSSION

The present cycling exercises performed at different pedaling rates induced changes in neural and contractile properties of quadriceps muscle but no significant effect of cadence was found when considering a range of FCC ± 20%.

Strength Performances

The decreases in maximal isometric torque after 30-min cycling ranged between 9 and 13% and were very close to the 13% reported by Bentley et al. (6). On the contrary, muscular torque under concentric conditions seemed to be less altered (5–8% at 120°·s^{-1} and 1–4% at 240°·s^{-1}). The decrease in muscular strength seemed thus proportional to the level of muscular tension, i.e., the smallest torque reduction was observed at the highest angular velocity. This velocity-dependent force reduction after prolonged cycling exercise has not systematically been reported in the literature. For example, Bentley et al. (5) have found a significant reduction (12%) in isokinetic peak torque at 60°·s^{-1} but not at 120°·s^{-1} and 180°·s^{-1} after 30-min cycling. On the contrary, previous findings from our laboratory have shown that reductions in peak torque ranging from 11 to 15% between 60 and 240°·s^{-1} were quite similar after 2-h cycling (20). In the present study, the lack of torque reduction with concentric contractions at the higher velocity when compared with isometric and lower concentric contractions seems difficult to interpret.

The decrease in muscular capacities after cycling exercise was independent of pedaling rates because the different cadences induced similar force reductions. This finding seems to refute our hypothesis, which proposed a different alteration of muscle strength after the different pedaling rates that involved different fiber type recruitment patterns (1) and neuromuscular fatigue (28,29). To our knowledge, the influences of cycling cadence during exercise longer than 6 min on muscular strength have not previously been investigated. Beelen and Sargeant (7) found a 14% greater reduction in maximal peak power after a prior 6-min exercise at 120 rpm compared with 60 rpm. The hypothesis stated by these authors that a relatively greater contribution

TABLE 2. Contractile parameters measured on muscular isometric twitch of the quadriceps muscle before (Pre) and after (Post) the 30-min cycling exercise at the three pedaling rates, FCC−20%, FCC, and FCC+20%; values are means ± SD.

	FCC−20%			FCC			FCC+20%		
	Pre	Post	P<	Pre	Post	P<	Pre	Post	P<
Pt (Nm)	43.1 ± 5.2	35.8 ± 5.3	0.01	44.8 ± 6.8	37.6 ± 4.6	0.01	43.6 ± 6.5	35.1 ± 5.5	0.01
CT (ms)	67 ± 5	69 ± 9	NS	69 ± 10	65 ± 7	NS	66 ± 3	63 ± 7	NS
HRT (ms)	87 ± 7	69 ± 7	0.01	86 ± 12	67 ± 4	0.01	85 ± 13	67 ± 11	0.01
RD (Nm·ms^{-1})	1.52 ± 0.22	1.38 ± 0.21	0.02	1.55 ± 0.22	1.39 ± 0.22	0.01	1.52 ± 0.24	1.32 ± 0.25	0.01
RR (Nm·ms^{-1})	0.60 ± 0.12	0.53 ± 0.05	NS	0.55 ± 0.10	0.57 ± 0.10	NS	0.61 ± 0.13	0.58 ± 0.13	NS

Pt, maximal twitch tension; CT, contraction time; HRT, half relaxation time; RD, maximal rate of tension development; RR, maximal rate of tension relaxation.

of fast fatigue-sensitive fibers when subjects performed higher movement frequencies, and hence different muscle shortening velocities are used, could not be validated by our study. According to Ahlquist et al. (1), the recruitment of Type II fibers (demonstrated by a greater glycogen depletion) increases under high force (low cadence) conditions. Consequently, cycling at a cadence equal to 100 rpm (which corresponds approximately to FCC+20%) would not induce a significant increase in Type II fiber recruitment. Therefore, one could expect a greater force reduction after the lower cadence exercise (FCC−20%) where Type II fibers would have been preferentially recruited. Nevertheless, the absence of significant difference in force loss between the three cadences does not corroborate this hypothesis. Moreover, possible higher neuromuscular fatigue during the exercises performed at cadences different from the preferred one (28,29) did not influence the strength performances after the cycling exercise.

According to the results of the present study, there appears to be no relation between fiber type recruitment pattern and neuromuscular fatigue during a cycling exercise and the subsequent reduction of strength capacity. Even if the tasks (isometric and isokinetic contractions) used to evaluate the changes in strength characteristics differed from the task of pedaling, this method nevertheless provided interesting information concerning the changes in contractile and neural properties of leg extensors with fatigue.

Contractile and Neural Properties

In long-duration exercise, fatigue has been associated not only with metabolic alteration but also with long lasting impairment of muscular force generation, which may be attributed to excitation-contraction coupling impairment (4,14,21,24). Indeed, fatigue in skeletal muscle is generally accompanied by a decline in tension and a slowing of twitch relaxation. In agreement with previous studies where cy-

cling exercise lasted more than 1 h (12,20), changes in isometric twitch parameters were found in the present study after a 30-min exercise. Postexercise, the twitch amplitude declined without reduction in the contraction time, therefore, inducing a lower maximal rate of twitch tension development. The reduced twitch torque could be associated with several mechanisms, such as reduced Ca^{2+} release from the sarcoplasmic reticulum (30), change in metabolites (H^+, inorganic phosphate), and reduced capacity of cross bridges to form strong binding (22). The shorter twitch HRT in relation with the reduced peak twitch tension could explain the lack of slowing rate of tension relaxation.

Nonsignificant changes in the M-waves characteristics were found after the present 30-min cycling exercise, attesting a nonapparent neuromuscular transmission failure or decreased excitability of the muscle fiber sarcolemma. The alteration of the M-wave in human muscle after dynamic exercise does not appear to be a common occurrence. Reduced M-wave amplitude with lengthening of the duration has been found in the quadriceps muscle after a 5-min high-level (2) or a 2-h prolonged-cycling exercise (21). In contrast, no changes in the M-wave were found after sustained voluntary contractions (9) or marathon running (3). Differences in the fatigue protocol, exercise duration, type of contractions, and tested muscles may partially explain this discrepancy.

Central activation was investigated here by two methods. The first method was to compare the changes in AL before and after exercise by analyzing the ratio of the amplitude of the superimposed twitch over the size of the mean control twitch. The lower AL values found before exercise (range 79–84%) in comparison with other values published in the literature (6,8) can be explained by the method of AL estimation. Indeed, by calculating the AL with the ratio MVC/MVC + superimposed twitch, the AL values before exercise would be close to 98%. However, although it has

TABLE 3. M-wave characteristics for both vastus lateralis and vastus medialis muscles before (Pre) and after (Post) the 30-min cycling exercise at the three pedaling rates, FCC−20%, FCC and FCC+20%; values are means ± SD.

	FCC−20%			FCC			FCC+20%		
	Pre	Post	P<	Pre	Post	P<	Pre	Post	P<
Vastus lateralis									
D (ms)	6.4 ± 2.0	6.0 ± 2.0	NS	7.1 ± 1.6	6.9 ± 1.6	NS	6.6 ± 2.2	6.3 ± 1.9	NS
A (mV)	11.4 ± 2.1	10.7 ± 2.4	NS	9.2 ± 2.3	9.0 ± 2.7	NS	10.0 ± 2.1	9.9 ± 3.0	NS
Vastus medialis									
D (ms)	9.3 ± 1.6	9.1 ± 2.5	NS	8.7 ± 1.9	8.8 ± 2.5	NS	8.7 ± 1.9	8.6 ± 2.8	NS
A (mV)	10.3 ± 2.2	9.9 ± 2.4	NS	10.1 ± 1.3	10.7 ± 1.9	NS	10.3 ± 1.6	9.6 ± 2.9	NS

Duration (D) and peak to peak amplitude (A) of the M-wave.
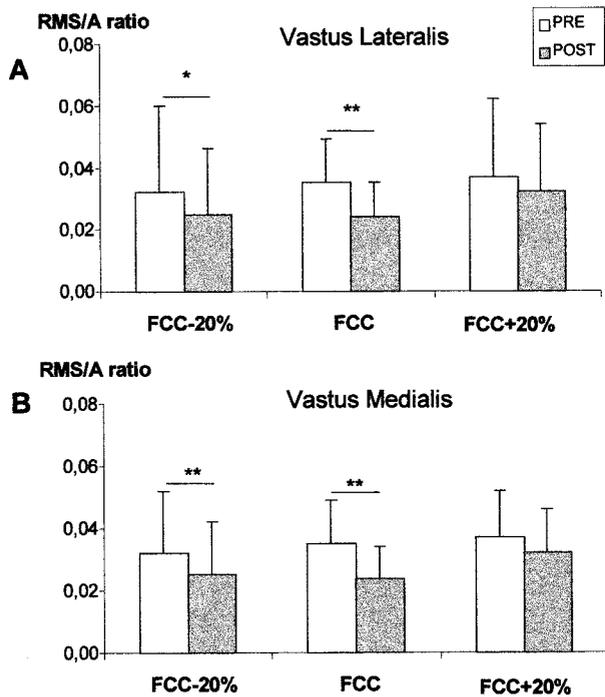
FIGURE 2—Mean values of RMS/A (M-wave amplitude) ratio before (PRE) and after (POST) the 30-min cycling exercise at the three pedaling rates, FCC−20%, FCC, and FCC+20%. Error bars: SD. A, Vastus lateralis muscle. B, Vastus medialis muscle. * $P < 0.05$ and ** $P < 0.01$: statistically significant compared with the PRE-cycling exercise condition.

previously been shown that pulse train stimulation generated a significantly larger torque increment than single impulse (23), this latter method is considered relevant to assess the level of muscular activation (11,16). Moreover, superimposed single twitch produces less discomfort than pulse train stimulation, in particular in fatigued conditions. After the cycling exercise, AL was found to decrease by 13–16%, indicating an inability to maintain the same voluntary activation of the quadriceps muscle with fatigue. Previous studies, using maximal short-term dynamic exercise, have revealed contradictory results concerning the presence or not of activation failure with fatigue (8,11,16,26). To our knowledge, Bentley et al. (6) were the sole authors that studied voluntary activation of the knee extensors after prolonged cycling exercise. Using a percutaneous electrical stimulation method (superimposed tetanus), these authors found an increased activation deficit after exercise. Their data corroborate our results and attest to the presence of a central component of fatigue in our subjects.

The second method of estimating alterations in central activation was to compare the change in EMG RMS with the change in M-wave amplitude. The reduced RMS/A ratio for the VL and VM muscles during MT_{iso} confirms that the cycling exercise significantly altered the neural drive to the muscle. Reductions in maximal iEMG have been reported earlier after running or cycling exercise (3,12,19,20) and could partly explain the weakened force output. Although EMG RMS can say something about neural drive, it cannot say alone whether the altered drive represents central fatigue. Indeed, during fatigue the neural activity estimated by EMG can be altered through changes in (i) central motoneuron excitability, (ii) transmission of the signal across the neuromuscular junction, and (iii) excitability of the muscle membrane. The absence of significant changes in M-wave post exercise suggests the existence of a central activation failure without changes in peripheral neuromuscular transmission. Nevertheless, the precise mechanisms (i.e., a decrease in motor neuron firing rates and/or in the number of motor units recruited) for a reduction in central drive cannot be ascertained using the present methodology. Although deficits of muscular activation appeared not to be significantly different between cadences, an interesting finding was that after the highest pedaling rate condition, the central neural input to VM and VL muscles remained unchanged. This result suggests that central drive is less altered when a high cadence is used, which may indicate a greater participation of peripheral mechanisms in force reduction. However, this phenomena is quite intriguing because one would expect that peripheral alterations would occur essentially at lower pedaling rates where higher muscular tension levels are required to sustain power output. Further studies using extreme cadences such as 50 and 120 rpm would be necessary to validate such a hypothesis.

In conclusion, we have shown that a prolonged cycling exercise impairs muscular strength capacity. This failure is associated with changes in contractile and neural properties of leg extensors and appears to be independent of the pedaling rate throughout a range of cadences habitually adopted by triathletes (from 69 to 103 rpm). In practical terms, these results suggest that well-trained triathletes can easily adapt to changes in cadence permitted by gear ratios classically adopted by them. Although unexpected, it is interesting to bear in mind that the freely chosen cadence does not minimize the effects of fatigue on subsequent leg extensors' strength capacities. Our findings could well lead to a reconsideration of training methods for competitive cycling using, for example, combinations of different cadences. Moreover, in multi-sports such as triathlon, changes in pedaling rates in trained athletes could have minor effects upon subsequent running performance. Such speculation needs to be confirmed, however, by specific experimentations.

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