Influence of fatigue on EMG/force ratio and cocontraction in cycling

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ABSTRACT

HAUTIER, C. A., L. M. ARSAC, K. DEGHDEGH, J. SOUQUET, A. BELLi, and J.-R. LACOUR. Influence of fatigue on EMG/force ratio and cocontraction in cycling. Med. Sci. Sports Exerc., Vol. 32, No. 4, pp. 839 – 843, 2000. Purpose: The purpose of the present study was to observe force and power losses and electromyographic manifestations of fatigue during repeated sprints performed on a friction-loaded cycle ergometer. Methods: Ten subjects performed 15 maximal 5-s sprints with 25-s rests between them. Power, velocity, and torque were measured during sprints 1 and 13 and during two submaximal constant-velocity (50 rpm) periods of cycling performed before and after the sprints. The EMG signals of five leg muscles were stored to determine the EMG/force ratio of power producer muscles and the coactivation of antagonist muscles. The power producer muscles were activated to the same level during sprints 1 and 13, despite a loss of force, whereas the vastus lateralis muscle was recruited more during the submaximal cycling period under fatigue conditions. Results: This led to an increased EMG/force ratio for the power producer muscles, indicating the peripheral fatigue status of these muscles. Antagonist muscles were less activated during the sprints after fatigue; whereas they stayed unchanged during the last submaximal cycling period. Conclusions: This suggests that there is a decrease in coactivation as agonist force is lost. This decrease in coactivation under fatigue conditions has not been previously reported and is probably due to the training status of the subjects. Subjects may have learned to better use their antagonist muscles to efficiently transfer force and power to the rotating pedal. This coordination can be adapted to cope with fatigue of the power producer muscles. Key Words: REPEATED SPRINTS, PERIPHERAL FATIGUE, COACTIVATION, MUSCLE COORDINATION

Muscle fatigue can be defined as the failure to maintain a required or expected power output (10). Because the type of effort required for many sprint sports such as soccer, basketball, and hockey is maximal-intensity intermittent exercise, many recent studies have focused on the influence of recovery time on performance during repeated sprints (2,3). They showed that a series of 6-s bouts of work separated by a 30-s rest caused muscular fatigue. Colliander et al. (8) used a similar protocol to show that subjects with a high percentage of fast twitch fibers were more sensitive to fatigue than those with more slow ones. But fatigue during repeated sprints has never been seen as a change in EMG. It is thus important to know whether a decrease in force and power that accompanies fatigue is completely attributable to contractile loss or whether muscle activation accounts for a part of this loss.

Several authors have studied the influence of fatigue on EMG parameters during isometric contractions (4,5,25), isokinetic contractions (16), and stretch-shortening cycles (11,20,26), Häkkinen et al. (14) reported a significant increase in IEMG during sustained submaximal isometric contractions; whereas Krogh-Lund and Jørgensen (21,22) and Öberg et al. (27) obtained the same result with the root mean squared (RMS) EMG signal. Whereas single muscle fatigue has been well documented, little attention has been paid to intermuscular contractions in pluriarticular movement. Lombard (23) was the first to observe antagonistic contraction during knee extension movement in cycling, which demonstrated the complexity of intermuscular coordination patterns. Others have studied intermuscular coordination patterns in complex movements such as jumping (6), running (19), or cycling (12,17,18). All of these studies indicated that monoarticular and biarticular muscles have different functions. The first can be considered to be power producers, whereas the second type are modulated to transfer power between articulations. These functions are involved in the optimization of intermuscular coordination for the best efficiency. Psek and Cafarelli (28) examined the activation of antagonist muscles under fatigue conditions and found that fatigue of the vastus lateralis muscle increased the activation of the biceps femoris muscle, which acts as an antagonist in knee extension movement. This
sprints. RMS, root mean squared.

suggests that fatigue of a muscle group decreases the global movement overall efficiency by disorganizing muscular coordination.

The present study was carried out to observe the electromyographic changes that occur during fatigue produced by repeated maximal sprints on a friction-loaded cycle ergometer. The fatigue of each muscle group was determined by EMG analysis together with the possible influence of fatigue status on antagonist muscles activation. These results will help clarify the extent to which force and power losses are the result of muscle contractile loss or changes in intermuscular coordination.

METHODS

Subjects. Ten subjects (eight men and two women) volunteered to participate in this study (mean age 20.3 ± 0.7 yr, mean height 174.6 ± 7.2 cm, and mean body mass 65.5 ± 8.6 kg). The subjects had all been trained four times a week for 9 wk and detrained for 7 wk. Each training session included two sets of 15 maximal cycling sprints (5-s sprint and 55-s rest). The recovery between the two sets was 15 min. A series was performed against the same friction load during all training (near 8% of the body mass). The other series was performed against a braking force adjusted to obtain a 100% EMG signal. The EMG voltages have been calculated and expressed as microvolts. Individual loads were determined from previous tests. The cycling performances were recorded during the first and the 13th sprints to have time for storing the data before the constant cycling period.

Material. The cycle ergometer (Monark 818E, Stockholm, Sweden) was equipped with a strain gauge (200 N, bandwidth 500 Hz. Interface Mfg., Scottsdale, AZ) for measuring friction force and an optical encoder (HengstlerRIS IP 50) fixed on a castor for measuring flywheel displacement. Force and displacement signals were sampled (200 Hz) and stored on a PC (Victor Technologies, 386sx, Rueil-Malmaison, France) via a 12-bit analog-digital converter (DAS-8, 12 bits, Keithley Metabyte Traveling Salesman, Taunton, MA). The total force produced by the subject was calculated as the sum of frictional and inertial (dependent of the acceleration) forces (1,15,24) and recalculated as moment at the crank. First and second order derivatives of the flywheel displacement were calculated to obtain flywheel velocity and acceleration (1,15). Power, force, moment, and velocity were averaged for each downstroke (i.e., from top dead center of one foot to top dead center of the other foot).

EMG signals from the gluteus maximus (GM), rectus femoris (RF), vastus lateralis (VL), gastrocnemius lateralis (GL), and biceps femoris (BF) muscles were recorded via bipolar Ag-AgCl surface electrodes (interelectrode distance 1.2 cm), including an amplifier (gain 600) and a band pass filter (6–600 Hz) (Biochip, Elmatek S.A., Crolles, France) fixed longitudinally over the muscle belly. Subjects wore a skin suit to prevent the cables from swinging and causing movement artifacts. Raw EMG readings were electronically RMS with a time averaging period of 25 ms (536AJ, Analog Device, Norwood, MA), converted from A to D, and stored on the PC at the same sampling frequency as the mechanical data (200 Hz). The activation of each muscle was determined by measuring the mean value of the RMS EMG signal between the onset of activation and the end of the burst. The EMG signal of the GM muscle was not recorded during the constant-velocity cycling bouts (50 rpm) because of a poor signal-to-noise ratio after fatigue. The EMG voltages have been calculated and expressed as microvolts. Subjects performed a maximal isometric voluntary contraction (MVC) of the five muscles before the fatigue protocol to obtain a 100% EMG signal.

The results are expressed as mean ± SD. The Wilcoxon test was used to compare paired groups. Significance was set at P < 0.05.

RESULTS

Mechanical Results

Maximal mean power decreased significantly from the first (957.1 ± 217.3 W) to the 13th sprint (849.3 ± 199.3 W) (P < 0.01). Similarly, the moment produced at P max decreased significantly from the first (65.8 ± 13.3 Nm) to the 13th sprint (61.8 ± 12 Nm) (P < 0.05), and cycling rate at P max decreased from 125 ± 1.5 rpm in the first sprint to 119 ± 1.3 rpm in the 13th sprint (P < 0.05). The moment at 50 rpm remained unchanged before and after fatigue (33.8 ± 10.5 Nm).
TABLE 1. Relative activation of muscles during sprints, compared to 100% maximal isometric voluntary contraction.

<table>
<thead>
<tr>
<th>Muscles</th>
<th>Activation Level (% MVC)</th>
<th>SD (% MVC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GM</td>
<td>86.0</td>
<td>13.3</td>
</tr>
<tr>
<td>VL</td>
<td>126.2</td>
<td>29.9</td>
</tr>
<tr>
<td>RF</td>
<td>99.9</td>
<td>30.0</td>
</tr>
<tr>
<td>BF</td>
<td>75.7</td>
<td>24.1</td>
</tr>
<tr>
<td>GL</td>
<td>80.8</td>
<td>10.1</td>
</tr>
</tbody>
</table>

MVC, maximal isometric voluntary contraction; GM, gluteus maximus; VL, vastus lateralis; RF, rectus femoris; BF, biceps femoris; GL, gastrocnemius lateralsalis.

**EMG Results**

**Maximal exercise.** Mean activation levels of all the muscles were not different from one downstroke to another during the sprints. The EMG signal was constant throughout a sprint, whatever the downstroke and whatever the external force and velocity recorded. The activation level of the muscles relative to MVC during the first sprint are shown in Table 1.

The changes in the EMG after repeated sprints are summarized in Table 2. There was significantly less RMS EMG for the knee flexor muscles in the 13th sprint than in the first (P < 0.01). But there was no difference in the activation of the other three muscles.

**Submaximal exercise.** The changes in the EMG during submaximal cycling periods are summarized in Table 3. There was significantly more RMS EMG during the last constant cycling period in the VL muscle (155 ± 88 µV vs 180 ± 101 µV) than during the first period (P < 0.05). The activation of the other muscles was not significantly altered after repeated sprints.

**DISCUSSION**

The loss of power and force observed in the 13th sprint demonstrates that maximal intermittent cycling exercise causes muscle fatigue. The present study was performed to observe the behavior of power producer muscles during fatigue caused by maximal intermittent sprint cycling exercise. The EMGs of the prime mover muscles (GM and VL) remained unchanged after fatigue during maximal cycling bouts, whereas the moment calculated at the crank decreased. Additionally, the VL EMG recorded during the constant-velocity cycling period (50 rpm) increased, whereas the moment remained unchanged. This sort of increase during submaximal contraction has been reported in several studies on muscle fatigue (4,5,9,14,25). These results are also in line with previous studies on muscle fatigue resulting in a neural compensation, which demonstrated that the EMG/torque ratio increases to compensate for the contractile loss (13,20). It is difficult to calculate an EMG/torque ratio from our data because the moment calculated at the pedal cannot be attributed to a single muscle (17–19). However, the EMG activation of antagonist muscles indicates that the decrease in force on the pedal cannot be attributed to increased coactivation. The observed increase in the global ratio EMG/external force is probably due to a loss of prime mover muscle contractile force. The stability of the EMG level during maximal sprint may account for the maximal activation required during each push-off, even in the first sprint, especially for the VL muscle (Table 1). The contractile failure during maximal sprints cannot be offset by overactivating power producer muscles. Therefore, subjects produce less force and power after fatigue (20). But this result does not agree with the results of Tesch et al. (29), who found parallel declines in IEMG and the force of knee extensor muscles during maximal exercise.

The changes in the EMG/force ratio point to two types of fatigue: an increased EMG/force ratio is classified as “peripheral” fatigue, and a constant EMG/force ratio associated with a force decrease is classified as “central” fatigue. The type of fatigue obtained in the present study for the power producer muscles is peripheral fatigue. It may result from a lack of force generation capacity by the whole muscle, involving impaired neuromuscular transmission and impaired excitation-contraction coupling. The increased EMG/force ratio of power producer muscles may be attributed to the changes in contractile process after repeated sprints, as found previously (2,3,8,10,).

We also determined the coordination between antagonist muscles during sprint fatigue. The changes in the activation of biarticular antagonist muscles (BF and GL) show that these muscles are significantly less activated at the end of the exercise. After fatigue, the EMG of BF and GL muscles are 13.3% and 17.3% lower (P < 0.05) during the sprint. Such a difference may not be attributed to force and power decreases because the EMG of BF and GL muscles are 9.4% (NS) and 22.4% (P < 0.05) lower during the submaximal cycling period at 50 rpm with a constant force and power. Of course, the activation of VL, BF, and GL cannot be totally considered as cocontraction because we averaged the EMG signals all over the push-off, whereas Unnithan et al. (30) calculated their cocontraction index by overlapping agonist and antagonist muscle signals. However, such a decrease in the activation of antagonist muscles is contrary to the findings of Psek and Cafarelli (28), who demonstrated that coactivation of the biceps femoris muscle increased with fatigue of knee extensors. However, their subjects included male students with low-to-average levels of physical

**TABLE 2. EMG activation of muscles before and after fatigue in maximal exercise (in microvolts).**

<table>
<thead>
<tr>
<th>S1</th>
<th>GM (165 ± 88)</th>
<th>VL (231 ± 78)</th>
<th>RF (275 ± 100)</th>
<th>BF (213 ± 93)</th>
<th>GL (221 ± 66)</th>
</tr>
</thead>
<tbody>
<tr>
<td>S13</td>
<td>171 ± 81 ns</td>
<td>216 ± 78 ns</td>
<td>241 ± 96 ns</td>
<td>185 ± 93*</td>
<td>183 ± 51*</td>
</tr>
</tbody>
</table>

GM, gluteus maximus; VL, vastus lateralis; RF, rectus femoris; BF, biceps femoris; GL, gastrocnemius lateralsalis; S1, sprint 1; S13, sprint 13.

Values are mean ± SD.

ns P > 0.05.

* P < 0.01.
activity and who were not trained to the specific task. In the present study, the subjects were trained to the cycling task and repeated sprints, and the same fatigue protocol, performed before training, failed to demonstrate any significant changes between the first sprint and the 13th sprint in the EMG levels of BF (171 ± 15 μV vs 186 ± 20 μV, P > 0.05) and GL muscles (189 ± 36 μV vs 245 ± 99 μV, P > 0.05). These results demonstrate that the untrained group tended to increase cocontraction of antagonist muscles after performing 13 sprints. This is in accordance with Carolan and Cafarelli (7), who demonstrated that training can reduce the coactivation of antagonist muscles during monoarticular movement. We therefore propose that the subjects involved in the present study had learned to use their biarticular muscles to modulate muscle activation patterns efficiently to cope with cycling constraints. Although the biceps femoris muscle is a real antagonist muscle in monoarticular knee extension, the function of biarticular muscles in cycling is not so clear. The activation of antagonist muscles during hip and knee extension in cycling is no longer considered to be a paradox (23). Several studies have shown that these muscles are efficiently recruited with the specific constraints of cycling: how to effectively transfer muscle power to the rotating pedal (12, 17–19). We found that the BF and GL muscles were certainly not fatigued by the sprint cycling exercise, but the force and power they were required to transfer was reduced. Thus, the EMG of antagonist muscles is adapted to the force produced by the fatigued power producer muscles to ensure the efficient transfer of this power to the pedal without braking the hip and knee extension power. This adaptation of muscle coordination to fatigue of the prime mover muscles could have been centrally evoked because our subjects were trained for cycling and the test protocol. However, a reflex compensation for knee extensor muscles fatigue has been previously reported in a very different type of movement (20). That study demonstrated that muscle fatigue can be compensated for by an increased reflex response to stretch, as indicated by an increase in EMG response. The mechanisms that might be responsible for reflex adaptation are very different in the present study, but it is reasonable to assume that antagonist activation could be reduced without any conscious effort because coactivation is facilitated by the firing of Renshaw cells (7). Thus, a decrease in force transmitted by the patella tendon may cause a decrease in the activation of antagonist muscles (BF and GL) without any central mediation.

In conclusion, the present study demonstrates that repeated sprint cycling elicits muscular fatigue in monoarticular power producer muscles (VL and GM). The main manifestations of the fatigue are a decrease in the efficiency of the EMG signal recorded on power producer muscles. These changes may be principally attributed to muscle lactate accumulation and/or depletion of high energy phosphates. We also find that intermuscle coordination in cycling can be efficiently adapted to the contractile loss of the power producer muscles. The lower activation of antagonist muscles after fatigue seems to be an efficient adaptation of the intermuscular coordination to transfer reduced force and power to the pedal. Such an adaptation could be centrally mediated patterns and/or due to reflex changes caused by Golgi tendon organs. The adaptability by our specifically-trained subjects does not seem to occur in less well-adapted subjects. Finally, the present study also demonstrates that force and power losses in repeated sprint cycling cannot be attributed to an increase in antagonist muscle coactivation.

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