Limiting mechanisms of force production after repetitive dynamic contractions in human triceps surae

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Klass, M., N. Guissard, and J. Duchateau. Limiting mechanisms of force production after repetitive dynamic contractions in human triceps surae. J Appl Physiol 96: 1516–1521, 2004. First published November 7, 2003; 10.1152/japplphysiol.01049.2003.—The influence of repetitive dynamic fatiguing contractions on the neuromuscular characteristics of the human triceps surae was investigated in 10 subjects. The load was 50% of the torque produced during a maximal voluntary contraction, and the exercise ended when the ankle range of motion declined to 50% of control. The maximal torque of the triceps surae and the electromyographic (EMG) activities of the soleus and medial gastrocnemius were studied in response to voluntary and electrically induced contractions before and after the fatiguing task and after 5 min of recovery. Reflex activities were also tested by recording the Hoffmann reflex (H reflex) and tendon reflex (T reflex) in the soleus muscle. The results indicated that whereas the maximal voluntary contraction torque, tested in isometric conditions, was reduced to a greater extent (P < 0.05) at 20° of plantar flexion (−33%) compared with the neutral position (−23%) of the ankle joint, the EMG activity of both muscles was not significantly reduced after fatigue. Muscle activation, tested by the interpolated-twitch method or the ratio of the voluntary EMG to the amplitude of the muscle action potential (M-wave), as well as the neuromuscular transmission and sarcolemmal excitation, tested by the M-wave amplitude, did not change significantly after the fatiguing exercise. Although the H and T reflexes declined slightly (10–13%; P < 0.05) after fatigue, these adjustments did not appear to have a direct deleterious effect on muscle activation. In contrast, alterations in the mechanical twitch time course and postactivation potentiation indicated that intracellular Ca2+-controlled excitation-contraction coupling processes most likely played a major role in the force decrease after dynamic fatiguing contractions performed for short duration.}

HIGHLIGHTED TOPIC | Neural Control of Movement

Since the end of the 19th Century, many studies have investigated the etiology of muscle fatigue (for reviews, see Refs. 1, 14, 16). The cause of the loss of force during muscle fatigue has been attributed to different mechanisms, ranging from the generation of the central command to the interaction between the contractile proteins (16). However, there is now convincing evidence that fatigue is not caused by a single factor, but that the mechanisms underlying the force reduction are task specific. This means that fatigue can be induced by a combination of processes that contribute in different ways to the decline in force, according to the details of the task (14, 16). Factors such as the intensity and the duration of the exercise (24, 32), the type of load (19), and the fiber-type composition of the active muscle (21, 25), as well as the type of contraction (30, 36), induce specific neural and muscular changes.

A main factor influencing task failure is blood flow perfusion. Blood occlusion occurs during static contractions when the force exceeds 20–30% of the maximal voluntary contraction (MVC; Ref. 3). As a consequence, the accumulation of chemicals and metabolites is higher during sustained contractions compared with intermittent isometric ones (5, 29). These changes and a reduced O2 availability have a direct effect on muscle contractility (10), and it is also believed that they have an indirect action through metaboreceptors (6, 17). When activated, the metaboreceptors inhibit the motoneuron pool via a reflex pathway mediated by small-diameter group III and IV muscle afferents (20, 33). Thus the metaboreceptors possibly contribute to the reduction in voluntary drive through spinal and supraspinal actions during sustained contractions (6, 12, 13, 16, 17). In contrast, intermittent isometric contractions, which are achieved by alternating muscle contractions and resting phases, maintain or even increase blood flow (38). This maintained perfusion contributes to the continuous removal of metabolic by-products and therefore should reduce the alteration of the neural drive to the motoneuron pool. This is supported by a recent study in our laboratory (13), which tested the excitability of spinal reflex loops by measuring the H reflex amplitude during sustained and intermittent submaximal isometric contractions. Under these conditions, the H reflex was reduced during sustained contractions, whereas no significant change occurred during intermittent ones.

There are numerous studies that explore muscle fatigue during both isometric and isokinetic contractions. Surprisingly, the mechanisms that limit performance of a series of movements performed against a constant load, the most frequent modality in daily life and athletic activities, have not been carefully investigated, except during repetitive dynamic actions of long duration (23, 28). Therefore, to contribute to the assessment of mechanisms that cause task failure (14, 16), we have investigated the influence of moderately loaded repetitive dynamic contractions for short duration on the human triceps surae. The neuromuscular adjustments of this muscle group, which performs a key role in locomotion, have been examined through a combination of voluntary, reflex, and electrically induced contractions.
MATERIALS AND METHODS

Ten healthy subjects (6 men and 4 women; 20–30 yr) volunteered to participate in the study. All subjects were naive to the purpose of the experiment. The local Ethics Committee approved the experimental procedures, and the experiments were performed in accordance with the Declaration of Helsinki. Before participation in the study, all subjects gave informed consent.

Experimental setup and ergometric device. During the experiments, the subjects lay prone on a table with both legs extended and the right foot strapped to a movable wooden plate. The foot was attached to the plate by two straps and held in place by an adjustable heel block. The first strap was placed over the dorsum of the foot, and the second one was attached around the ankle and the calcaneum. The angular movement of the ankle joint was monitored by a linear potentiometer mounted on the rotational axis of the foot plate. The second one was attached around the ankle and the calcaneum. The heel block. The right foot strapped to a movable wooden plate. The foot was connected or disconnected either to record the muscle contractile properties under isometric conditions or to perform ankle movements during the fatigue test.

Electromyography recordings. Voluntary and electrically induced electromyography (EMG) activities of the soleus (Sol) and the medial gastrocnemius (MG) were obtained with bipolar surface electrodes (8-mm diameter; silver electrode), which were placed 3 cm apart (center to center), one fixed over the muscle’s motor point. A ground electrode (2 × 5 cm silver plate) was placed over the tibia. The motor point was determined by electrical stimulation and was defined as the point of stimulation at which a barely perceptible muscle contraction was observed at the lowest intensity of the stimulus. The EMG signals were amplified (×1,000 or 2,000) and filtered (10 Hz–1 kHz) by a custom-made differential amplifier and displayed online on a digital oscilloscope (model 4094c; Nicolet, Madison, WI).

All signals were recorded on a personal computer (Pentium 450 MHz) at a sampling rate of 2.5 kHz, by using a data-acquisition system (AcqKnowledge, Model MP100; Biopac Systems, Santa Barbara, CA). The data were analyzed offline by using the AcqKnowledge 3.2.4 data-analysis software.

Electrical and mechanical stimulations. The contraction of the triceps surae was induced by a rectangular electrical pulse (1 ms in duration) delivered by a custom-made stimulator triggered by a digital timer (model 4030, Digitimer, Welwyn Garden City, UK). The stimulus was delivered to the tibial nerve through two electrodes: the triceps surae was induced by a rectangular electrical pulse (1 ms in duration) delivered by a custom-made stimulator triggered by a digital timer (model 4030, Digitimer, Welwyn Garden City, UK). The mechanical responses to maximal single and paired stimuli (2 responses each) were recorded. The muscle compound action potential (M-wave) was recorded from the averaged of 20 responses. All EMG (voluntary and reflex) were expressed as a percentage of the M_max amplitude. The skin temperature was continuously monitored throughout the experimental sessions.

The following tests were performed before and immediately after the fatigue task and after 5 min of recovery: 1) the electrical and mechanical responses to maximal single and paired stimuli (2 responses each) were recorded. 2) The torque during a 5-s MVC with the ankle in the neutral position and at 20° of plantar flexion was determined. Double stimuli were superimposed on the MVC (interstimulus-interval method) in neutral ankle position. Five seconds after the first MVC, electrical and mechanical responses to a single supermaximal stimulus were recorded to measure the extent of postactivation potentiation (PAP). 3) H_max and T_max were recorded in neutral ankle joint position. After the fatigue task, the stimulus intensity to induce the H reflex was slightly adapted to record the actual maximal amplitude (H_max).

The mean torque and the associated average EMG (aEMG) activity were measured during the plateau of the isometric MVCs in the two ankle joint positions. Twitch torque (T_t), contraction time (CT), time to half-relaxation (RT 1/2), maximal rate of torque development (+dP/dt), and relaxation (−dP/dt) were measured from the twitch response to single stimulation. The torque (T_P) and the maximal rate of tension development (+dP/dt) of the contractions recorded in response to the double electrical stimulation were also measured. The extent of PAP was determined by computing the ratio between the twitch size recorded before and after the isometric MVC. The size of the superimposed mechanical response to double stimuli was expressed as a percentage of the control response recorded in the resting muscle. This value was then subtracted from 100% to provide a quantitative measure of the central activation. The maximal peak-to-peak amplitude of the M-wave (M_max), H_max, and T_max were measured. For T_max, a mean value was obtained from the averaging of 20 responses. All EMG (voluntary and reflex) were expressed as a percentage of the M_max amplitude. The skin temperature was continuously monitored throughout the experimental sessions.

Statistics. Conventional statistical methods were used to calculate means, standard deviations (SD), and standard errors of the mean (SE). When not specified, data were reported as means ± SE. All data were analyzed by means of a one-way ANOVA with repeated measures. When significant main effects were observed, Dunnett’s test was used for post hoc analysis. A probability of P < 0.05 was chosen as the significant level in all analyses.

RESULTS

Isometric torque and aEMG during MVC. After the fatigue task (meas ± SD: 450 ± 191 contractions), the decline in isometric MVC torque was 23% (P < 0.01; Fig. 1). This reduction in torque was related to the ankle’s angle and was greater (33%; P < 0.001) when tested at 20° of plantar flexion. In contrast, the corresponding aEMG activity of the Sol and MG muscles did not significantly decrease regardless of ankle position (Table 1). The amplitude of the M-wave recorded in
neural position in the Sol and MG muscles showed a slight but nonsignificant ($P > 0.05$) decrease after the fatiguing task (Table 1). After 5 min of recovery, the MVC torque did not return to control values: a significant deficit of 17% in the neutral position ($P < 0.05$), and 20% in 20° of plantar flexion ($P < 0.05$), was still present (Table 1).

**Muscle activation.** The neural activation of the muscle, tested by the interpolated-twitch method, appeared to be nearly maximal in all subjects, both in control conditions (98.4 ± 0.8%) and immediately after the fatigue test (98.3 ± 1.0%; Fig. 2). The absence of change in activation is supported by the EMG results because the aEMG, normalized to the M-wave amplitude, did not change significantly during the fatiguing task (Table 1). This normalization procedure allows the exclusion of contamination of fatigue-induced adjustments of the voluntary neural activity by changes in neuromuscular propagation.

**Reflex responses.** The effects of the fatiguing task on the electrically ($H_{\text{max}}$) and mechanically ($T_{\text{max}}$) induced reflex responses are illustrated in Fig. 3. The normalized amplitudes of the two reflexes declined to a similar extent: $H_{\text{max}}$-to-$M_{\text{max}}$ ratio ($H_{\text{max}}/M_{\text{max}}$) decreased by 10% ($P < 0.01$), and $T_{\text{max}}$-to-$M_{\text{max}}$ ratio ($T_{\text{max}}/M_{\text{max}}$) decreased by 13% ($P < 0.05$) (Table 2). Although a similar deficit in $T_{\text{max}}/M_{\text{max}}$ and $H_{\text{max}}/M_{\text{max}}$ persisted after 5 min of recovery ($T_{\text{max}}/M_{\text{max}}$: 7% decrease; $H_{\text{max}}/M_{\text{max}}$: 6% decrease), the latter ratio remained significantly ($P < 0.05$) different from control (Table 2).

**Mechanical properties.** The mechanical responses to both single and double supramaximal stimulations were different after the fatigue test (Table 3). The P1 declined by 23% ($P < 0.01$). This decrease was accompanied by a shortening of its time course. In fact, $+dP/dt$ increased by 22% ($P < 0.05$), and CT and RT$_{1/2}$ were shortened by 31% ($P < 0.001$) and 21% ($P < 0.001$), respectively. The mechanical response evoked by the double stimuli displayed similar changes. P3 was reduced by 16% ($P < 0.01$), whereas $+dP/dt$ was increased by 21% ($P < 0.05$) after the fatiguing task (Table 3). After 5 min rest, neither P1 nor P3 returned to their control values, and significant deficits ($P < 0.001$) of 33 and 23%, respectively, were still present. At the same time, the twitch CT and RT$_{1/2}$ were also significantly ($P < 0.001$) shortened by 20 and 25%, respectively, compared with control values. However, $+dP/dt$ and $+dP/dt$, which were increased after the fatigue test, did not differ statistically from their control level after 5 min of recovery (Table 3).

**PAP.** The PAP was quantified by computing the ratio between the twitch size recorded 5 s after the isometric MVC and that observed in control conditions. In the unfatigued condition, PAP was 115 ± 5%. Compared with the pretest values, an 11% ($P < 0.05$) decline in PAP was observed after the fatiguing task (Table 3). After 5 min of recovery, PAP was not significantly different from control.

**Skin temperature.** A mean increase of 2.4°C (31.4 ± 0.5°C vs. 33.7 ± 0.3°C, $P < 0.01$) in skin temperature, measured in the middle part of the subjects’ calf muscles, was observed after the fatiguing task. After 5 min of recovery, skin temperature was still above prefatigue values (32.7 ± 0.4°C; $P < 0.01$).

**DISCUSSION**

Repetitive dynamic movements involving shortening and lengthening contractions against a constant load are performed frequently in daily and athletic activities. However, the investigation of these types of movements is lacking. We have studied the influence of moderately loaded repetitive dynamic contractions for short duration on the human triceps surae through a combination of voluntary, reflex, and electrically induced contractions. Our results showed that the neural drive and M-wave amplitude were not significantly affected by the fatiguing experiment, indicating that mechanisms located be-

### Table 1. MVC torque and EMG activities of the soleus and the medial gastrocnemius in control conditions, after the fatiguing task, and after 5 min of recovery

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Fatigue</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>MVC torque, Nm</td>
<td>150.3 ± 20.7</td>
<td>116.1 ± 15.4†</td>
<td>124.9 ± 16.0*</td>
</tr>
<tr>
<td>Neural position</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20° plantar flexion</td>
<td>81.0 ± 18.4</td>
<td>54.4 ± 16.1‡</td>
<td>65.0 ± 17.3*</td>
</tr>
<tr>
<td>aEMG Sol, µV</td>
<td>114 ± 21</td>
<td>105 ± 17</td>
<td>109 ± 19</td>
</tr>
<tr>
<td>aEMG MG, µV</td>
<td>115 ± 23</td>
<td>107 ± 21</td>
<td>115 ± 24</td>
</tr>
<tr>
<td>aEMG (M-wave)</td>
<td>73 ± 16</td>
<td>68 ± 16</td>
<td>71 ± 16</td>
</tr>
<tr>
<td>20° plantar flexion</td>
<td>73 ± 20</td>
<td>71 ± 17</td>
<td>78 ± 19</td>
</tr>
<tr>
<td>M-wave, mV</td>
<td>4.71 ± 0.36</td>
<td>4.47 ± 0.38</td>
<td>4.34 ± 0.36</td>
</tr>
<tr>
<td>Sol</td>
<td>3.25 ± 1.46</td>
<td>3.02 ± 1.27</td>
<td>2.94 ± 1.25</td>
</tr>
<tr>
<td>aEMG/M-wave, %</td>
<td>2.1 ± 0.4</td>
<td>2.0 ± 0.3</td>
<td>2.1 ± 0.3</td>
</tr>
<tr>
<td>MG</td>
<td>3.0 ± 0.8</td>
<td>2.8 ± 0.6</td>
<td>2.9 ± 0.5</td>
</tr>
</tbody>
</table>

Values are means ± SE. MVC, maximal voluntary contraction; Sol, soleus; MG, medial gastrocnemius; aEMG, average electromyographic activity; M-wave, muscle compound action potential. Significant difference from prefatigue values: *$P < 0.05$; †$P < 0.01$; ‡$P < 0.001$. 

*Ref: 1518 MUSCLE FATIGUE AFTER REPETITIVE DYNAMIC CONTRACTIONS*
yond the muscle cell membrane limited the force production after the fatiguing task in the present experimental conditions.

The ankle ROM was progressively reduced during the repetitive dynamic movements against a constant load. This observation can be explained by the change in the moment arm of the triceps surae throughout the ROM. Indeed, at the end of the shortening phase of the contraction, the torque required to move the load was greater because of a reduction in the moment arm of the muscle group. Consequently, the full ROM was not attained because of the fatigue-induced decline in force-generating capacity of the muscle. However, this mechanism alone cannot explain the reduced ROM: the original observation that the maximum isometric torque decline due to the fatiguing task was 10% greater ($P < 0.05$) when the triceps surae muscles were tested at 20$^\circ$ of plantar flexion compared with neutral position indicates that the force produced by the muscle was more affected in the shortened condition. Although muscle activation was not tested by the interpolated-twitch method at 20$^\circ$ plantar flexion (see below), the absence of aEMG change (Fig. 1) after the fatiguing task for the two ankle positions suggests that reduced neural drive should not have contributed to the greater loss of force in that position. Possible explanations could be change in cross-bridges mechanics (31) and reduced sensitivity of the contractile apparatus to Ca$^{2+}$ at short muscle fiber length (34).

The potential contribution of central fatigue to the decline in torque was tested by measuring the level of voluntary activation (interpolated-twitch method). Our data showed that the nonsignificant deficit in activation ($\pm 2\%$) observed after the fatiguing task was comparable to that observed in prefatigue conditions. Similarly, the aEMG recorded during MVC and the M-wave amplitude did not change after fatigue, neither in the Sol nor in the MG. Therefore, the aEMG-to-M-wave ratio, which provided an estimate of the neural adjustment, remained unchanged. Although the nonlinear summation of motor unit action potentials could affect the significance of using EMG magnitude as an index of muscle activation (8), these results indicate that the subjects’ capacity to generate maximal neural drive to the muscle was not affected in our experimental conditions. This means that, in contrast to sustained isometric contractions (16, 22, 25, 32) and long-duration repetitive dynamic actions (23, 28), central fatigue does not explain the loss of performance after moderately loaded repetitive dynamic contractions for short duration.

In agreement with previous work that compared the change in H and T reflexes during fatiguing sustained MVCs (2), the normalized reflex responses in the present experiments showed a similar reduction in amplitude. Although the mono- and oligosynaptic contributions to the spinal reflex evoked by

![Fig. 2. Comparison, in 1 subject, of the triceps surae torque (a) and raw EMG activities of the soleus (c) in response to a double electrical stimulation (10-ms interval) and during an MVC, before (A) and after (B) the fatiguing task. The arrows indicate the time at which a double stimulation was delivered to the tibial nerve during the MVC (interpolated-twitch method). Note that, in this subject, the peak torque induced by the double stimuli at rest was greater than in the other subjects and was close to the MVC torque.](image)

![Fig. 3. Histograms showing the changes in ratio between maximal H reflex and M-wave amplitudes ($H_{max}/M_{max}$; A) and maximal T reflex ($T_{max}/M_{max}$) (B) of the soleus in control conditions, after the fatiguing task and after 5 min of recovery. Data, expressed as percentage of control values, are means ± SE. Significant difference from prefatigue values: *$P < 0.05$; †$P < 0.01$.](image)

Table 2. Normalized amplitude of the soleus H and T reflex responses in control conditions, after the fatiguing task, and after 5 min of recovery

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Fatigue</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>$H_{max}/M_{max}$</td>
<td>0.51 ± 0.04</td>
<td>0.46 ± 0.05†</td>
<td>0.48 ± 0.05*</td>
</tr>
<tr>
<td>$T_{max}/M_{max}$</td>
<td>0.15 ± 0.03</td>
<td>0.13 ± 0.03*</td>
<td>0.14 ± 0.03</td>
</tr>
</tbody>
</table>

Values are means ± SE. $H_{max}/M_{max}$ ratio between maximal H reflex and M-wave amplitudes; $T_{max}/M_{max}$ ratio between maximal T reflex and M-wave amplitudes. Significant difference from prefatigue values: *$P < 0.05$; †$P < 0.01$. 

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Table 3. Characteristics of the mechanical response to a single and double electrical stimulation and PAP in control conditions, after the fatiguing task, and after 5 min of recovery

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Fatigue</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Twitch</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P0, Nm</td>
<td>33.2±3.1</td>
<td>25.5±2.1†‡</td>
<td>22.2±2.4‡</td>
</tr>
<tr>
<td>CT, ms</td>
<td>117±2</td>
<td>81±3†</td>
<td>93±3‡</td>
</tr>
<tr>
<td>RT½, ms</td>
<td>97±3</td>
<td>77±2‡</td>
<td>73±3‡</td>
</tr>
<tr>
<td>+dP/dt, N/m/s</td>
<td>516.8±41.9</td>
<td>631.4±46.1†*</td>
<td>514.7±47.9</td>
</tr>
<tr>
<td>dP/dt, N/m/s</td>
<td>190.8±26.1</td>
<td>187.3±27.8</td>
<td>180.6±28.5</td>
</tr>
<tr>
<td>Double stimulation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P0, N-m</td>
<td>63.2±6.6</td>
<td>53.1±4.8‡</td>
<td>48.8±5.3‡</td>
</tr>
<tr>
<td>+dP/dt, N-m/s</td>
<td>901.7±97.8</td>
<td>1087.5±89.5*</td>
<td>966.7±105.6*</td>
</tr>
<tr>
<td>PAP</td>
<td>1.15±0.05</td>
<td>1.02±0.02‡*</td>
<td>1.07±0.06</td>
</tr>
</tbody>
</table>

Values are means ± SE. PAP, postactivation potentiation; P0, twitch torque; CT, contraction time; RT½, half-relaxation time; +dP/dt and −dP/dt, maximal rate of twitch torque development and of relaxation, respectively; P0 and +dP/dt, torque and maximal rate of torque development of double stimuli, respectively. Significant difference from prefatigue values: *P < 0.05; †P < 0.01; ‡P < 0.001.

tendon tap (T reflex) and electrical stimulation (H reflex) may differ slightly, the main difference is that the H reflex procedure bypasses the muscle spindle. Therefore, the similar change in the two reflex responses suggests that a change in spindle sensitivity cannot explain the reduced reflex excitability. In the absence of reduced voluntary drive to the muscle (see above), and although reflex activities were tested at rest, these results suggest that presynaptic inhibition is increased after the fatiguing task (12, 13, 16).

In this study, the decrease in normalized H reflex amplitude (± 10%) is smaller than the ones (30–40%) reported after sustained contractions at different intensities (25–100% MVC) in the adductor pollicis brevis (13). It is also smaller than after electrically induced intermittent contractions (15 Hz) in the Sol muscle (47%) under ischemic conditions (17). This discrepancy can be explained by a greater degree of fatigue, as estimated by the loss of torque during an MVC, in those studies compared with the present investigation. This viewpoint is also supported by the observed lack of decline in the H reflex in conjunction with a small (nonsignificant) decrease in MVC torque during intermittent isometric contractions (13). Our results indicate that the excitatory spinal reflex mediated input to the Sol motoneuron pool declined after repeated dynamic contractions. The observed lack of a deficit in voluntary activation after the fatiguing task is consistent with earlier suggestions of increased descending supraspinal drive to compensate for the loss of excitation from the peripheral afferents onto motoneurons (12).

The amplitude of the M-wave recorded in the Sol and MG muscles did not change significantly after the fatiguing task. This observation, which is in agreement with previous studies using intermittent isometric contractions (13, 21) or isokinetic movements (30), suggests that short rest periods between each muscle activation are sufficient to maintain the neuromuscular excitability at a normal level. In contrast, during sustained isometric contractions, a progressive reduction of M-wave amplitude is usually observed, at least during long-lasting exercises (13, 15, 32). The present results indicate that a failure in the neuromuscular transmission and/or sarcolemmal excitation cannot explain the observed torque decrease, but, rather, processes located beyond the muscle cell membrane play a dominant role.

The analysis of the mechanical responses induced by single or double supramaximal electrical stimulation allows one to indirectly investigate the possible muscle intracellular changes responsible for the reduced mechanical performance. The decrease in the twitch torque observed at the end of the fatigue test, without any change in the M-wave, can be explained by an alteration of the excitation-contraction (E-C) coupling (5, 10). This reduced torque was associated with a shortening of the twitch CT and RT½ and an accelerated time course. Such acceleration during fatigue contrasts with the slowing of muscle contraction and relaxation usually reported after sustained isometric contractions (4, 10). However, other fatigue studies using intermittent contractions also showed a shortening of the twitch time course (21, 23, 28, 30, 35). Bottermann and Cope (7) claimed that, in cats, the changes in contractile speed are related to the fiber-type composition of the muscle. These authors observed that the speed of fatigue-resistant units (in contrast to the fast-fatigable ones) remained unchanged or was faster. Our observation of an accelerated twitch time course could be related, at least in part, to the fact that the Sol contains mainly fatigue-resistant fibers and plays a dominant role in the mechanical time course of the triceps surae. Thus the opposite change in contractile speed during different fatigue tasks appears to depend on the fiber-type composition of the muscle and to variable contributions of potentiation and fatigue (18).

An additional factor that could induce an acceleration of the twitch time course is the increase of the muscle temperature, which is known to intensify the contractile processes (9). Whereas in the adductor pollicis an increase in skin temperature of 5.2°C induced a 11% reduction in twitch CT and RT½ (26), the smaller increase in skin temperature (2.4°C) observed in this study should not have greatly affected the twitch time course.

Another approach to the study of the E-C coupling changes during fatigue is to analyze muscle twitch PAP. In our experimental conditions, PAP decreased by 11% after the fatiguing task. The alteration of the E-C coupling could be explained by a decrease in Ca2+ released by the sarcoplasmic reticulum and/or by a reduction in the sensitivity of contractile proteins to Ca2+ related metabolic changes that occur during fatigue (1). Because the twitch time course was shown to be controlled by phasic cytosolic Ca2+ movements during contraction (11) whereas PAP change seems to be more closely related to the sensitivity of contractile proteins to Ca2+ (37), our results suggest that these different intracellular processes may have been altered, resulting in reduced muscle force. This viewpoint is supported by the fact that the twitch torque remained depressed and its time course slower after 5 min of recovery, at a time that PAP was not significantly different from control. Therefore, a large portion of the long-lasting deficit in the twitch torque is most likely caused by impaired Ca2+ release (1). Whatever the underlying mechanisms, the alteration in the contractile properties observed after fatigue due to dynamic contractions indicates that intracellular Ca2+-controlled processes were affected.

In conclusion, the present results show that the limiting mechanisms of force production after dynamic fatigue contractions of the triceps surae muscles performed for short
duration are not related to reduced central drive to the muscle or impaired neuromuscular transmission and/or sarcoclemmal excitation. In contrast, failure of intracellular Ca\(^{2+}\)-controlled E-C coupling processes appears to play a major role in the decreased muscle performance after moderately loaded repetitive dynamic contractions for short duration.

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