Force depression in human quadriceps femoris following voluntary shortening contractions

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Lee, Hae-Dong, Esther Suter, and Walter Herzog. Force depression in human quadriceps femoris following voluntary shortening contractions. J. Appl. Physiol. 87(5): 1651–1655, 1999.—The purpose of this study was to investigate whether the isometric muscle force, redeveloped following maximal-effort voluntary shortening contractions in human skeletal muscle, is smaller than the purely isometric muscle force at the corresponding length. Isometric knee extensor moments, surface electromyographic (EMG) signals of quadriceps femoris, and interpolated twitch moments (ITMs) were measured while 10 subjects performed purely isometric knee extensor contractions at a 60° knee angle and isometric knee extensor contractions at a 60° knee angle preceded by maximal-effort voluntary shortening of the quadriceps muscles. It was found that the knee extensor moments were significantly decreased for the isometric-shortening-isometric contractions compared with the isometric contractions for the group as a whole, whereas the corresponding EMG and ITM values were the same. This study is the first to demonstrate force depression following muscle shortening for voluntary contractions. We concluded that force depression following muscle shortening is an actual property of skeletal muscle rather than a stimulation artifact and that force depression during voluntary contraction is not accompanied by systematic changes in muscle activation as evaluated by EMG and ITM.

skeletal muscle property; in vivo; electromyography; interpolated twitch technique

The observation that the isometric muscle force, redeveloped immediately after shortening contractions, is smaller than the muscle force during purely isometric (Iso) contractions at the corresponding length has been made in isolated skeletal muscle fibers (6, 8, 13) and in situ skeletal muscles (1, 11, 12). This phenomenon is referred to as force depression following shortening contractions (11). Its existence has been well accepted in the scientific community. Force depression is directly related to the amount of muscle shortening (11, 12) and the force during the shortening phase (1, 11) and is inversely related to the speed of shortening (1, 11, 12). Furthermore, force depression following shortening contractions is long lasting (1, 11) and can be abolished instantaneously by deactivating the muscle for a period long enough so that the force drops to zero (1, 8, 11). Despite these consistent experimental observations in muscle fibers and whole muscle, the mechanism caus-

ing force depression following muscle shortening is a matter of intense debate.

Recently, force depression following muscle shortening was demonstrated for the first time in human skeletal muscles. De Ruiter et al. (5) reproduced many of the results obtained from in situ animal skeletal muscles in human adductor pollicis. Most importantly, they found a direct linear relationship between force depression and the magnitude of shortening ($r^2 > 0.98$) as well as force depression and force during shortening ($r^2 > 0.89$). De Ruiter et al. found a maximum force depression in the human adductor pollicis of 37.2% with a shortening velocity of 6.1 °/s in thumb adduction and concluded that these results had profound practical implications for intact human skeletal muscles.

De Ruiter et al. (5) used artificial electrical stimulation of the ulnar nerve (20 and 50 Hz) to produce adductor pollicis contractions. Similarly, in all previous studies in entire muscles or muscle fibers, artificial stimulation of the preparations had been used. For entire muscles, electrical stimulation means that all (stimulated) motor units are recruited almost simultaneously at the same frequency and at a nearly constant current.

During voluntary contractions, motor units are recruited asynchronously and nonperiodically at different firing frequencies, and the magnitude of activation may change (2, 4). Therefore, it is not clear whether force depression following shortening occurs during voluntary contractions as it does during electrically stimulated contractions. The force depressions observed in the past could have been an artifact of electrical stimulation rather than represent an actual muscle property.

During voluntary contractions, force depression might be masked by an upregulation of muscle activation following muscle shortening. Such upregulation is possible during maximal voluntary contractions, because motor unit pools are (typically) not recruited to their full extent in these situations (2, 14, 15). Observations of increased muscle activation during maximal voluntary efforts have been made when the contractile conditions became unfavorable for large-force production (e.g., at very short muscle length; Ref. 9).

The purpose of this study was to investigate whether force depression was observed following maximal-effort voluntary shortening contractions in human skeletal muscles. It was hypothesized that there would be no observable force depression during maximal voluntary contractions, because any possible loss of force associated with muscle shortening would be compensated for by increased muscle activation.
METHODS

Subjects. Ten healthy volunteers (7 men and 3 women; age: 29.6 ± 7.4 yr; height: 1.79 ± 0.13 m; body wt: 77 ± 14 kg) participated in this study. The subjects were informed about all testing procedures and gave free informed consent to participate in this study. The study was approved by the Conjoint Ethics Committee of the University of Calgary.

Testing machine setup. The subjects were seated on a strength-testing machine (CYBEX NORM Testing and Rehabilitation System, Lumex), which was set up for left leg flexion/extension exercises. The subjects wore a double-shoulder seat belt to stabilize the upper body. The distal ends of the thigh and shank were strapped to the test seat and the dynamometer arm, respectively. The rotational axis of the dynamometer was aligned with the knee-joint axis (i.e., the most prominent point on the lateral epicondyle of the femur). A zero knee angle was defined as the knee fully extended.

Test contractions. Subjects performed two different sets of voluntary contractions: Iso and isometric-shortening-isometric (ISI). For the Iso contractions, the subjects produced a maximal-voluntary-effort, isometric knee extension contraction at a 60° knee angle for ~9 s. For the ISI contractions, the subjects produced maximal voluntary effortIso contractions for 2 s at a knee angle of 100°, followed immediately by a maximal-effort shortening of the knee extensors from a knee angle of 100° to a knee angle of 60° at an angular speed of 16°/s, and finally the subjects continued the maximal-effort Iso contractions for another 4–5 s at the 60° knee angle (Fig. 1). The duration of the ISI contractions was ~9 s.

Electromyography (EMG). To assess the degree of muscle activation, bipolar Ag-AgCl surface electrodes were placed on the distal one-third of the vastus lateralis (VL), vastus medialis (VM), and rectus femoris (RF) muscles. A ground electrode was placed on the patella. EMG signals for each muscle were passed through amplifiers located no further than 10 cm from the recording electrodes. Amplification was between 500 and 5,000 Hz and was kept constant for a given electrode. The signals were band-pass filtered by using cutoff frequencies of 10 Hz and 1 kHz.

Interpolated twitch technique. To evaluate the extent of motor unit recruitment, the twitch interpolation technique was used (3, 10, 14). Two carbon-impregnated rubber electrodes (4.5 × 10 cm) were thinly covered with a layer of conductive gel. One electrode was placed over the femoral nerve just distal to the inguinal ligament; the other electrode was placed at the distal end of the quadriceps muscle. During the knee extensor contractions, a doublet electrical stimulus (110 V, 8-ms interpulse interval, 0.8-ms pulse duration) was applied to the femoral nerve with the use of a Grass (Quincy, MA) 588 muscle stimulator with an isolation unit approved for human use. Resting twitch moment (RTM) was measured by applying a doublet twitch to the relaxed muscle and recording the corresponding twitch moment. The interpolated twitch moment (ITM) was determined by superimposing a doublet twitch to the voluntarily contracting muscle and recording the additional moment produced by the electrical stimulation on top of the maximal voluntary moment. The extent of motor unit recruitment during the maximal voluntary contraction was calculated as (ITM/RTM) × 100%.

RESULTS

Isometric knee extensor moments following shortening contractions. The mean isometric knee extensor moments for the entire group (n = 10) were found to be significantly smaller following shortening contractions...
than were the corresponding moments during the purely Iso contractions (Table 1, Fig. 2). In the post hoc analysis, we identified that 8 of the 10 subjects showed consistent force depressions, i.e., force depressions in 61 of 62 individual test comparisons. The mean values of force depression following shortening contractions in these eight subjects ranged from 0.7 to 11.8% (mean ± SD = 6.5 ± 3.7%; Table 1). In the remaining two subjects, the mean isometric knee extensor moments following shortening contractions were consistently higher than the corresponding isometric moments in all individual test comparisons (n = 8); the force enhancement following shortening in these two subjects ranged from 6.1 to 8.3% (mean ± SD = 7.2 ± 1.6%; Table 1).

Muscle activation (EMG and interpolated twitch technique). Muscle activation evaluated by the RMS values of the combined EMGs from all instrumented muscles was the same for the Iso and ISI contractions (Table 1). In addition, the ratio of (ITM/RTM)×100% determined by the interpolated twitch technique between the two types of contractions revealed no statistical significance (Table 1). Furthermore, for the eight subjects who showed consistent force depression, EMG and ITMs were the same for the Iso and ISI contractions (Fig. 3).

### Table 1. Summary of data analysis (1-tailed Student's t-test for paired data and post hoc comparisons for individual trials)

<table>
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<tr>
<th>Subject No.</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
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<th>9</th>
<th>10</th>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
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<td>2</td>
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<td>2</td>
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<td>2</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>24</td>
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<tr>
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<td>4</td>
<td>1</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>4</td>
<td>2</td>
<td>28</td>
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<td>2</td>
<td>8</td>
<td>3</td>
<td>2</td>
<td>6</td>
<td>9</td>
<td>12</td>
<td>4</td>
<td>70</td>
</tr>
<tr>
<td>KEM</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>16/16</td>
<td>0/2</td>
<td>8/8</td>
<td>2/3</td>
<td>2/2</td>
<td>0/6</td>
<td>9/9</td>
<td>12/12</td>
<td>4/4</td>
<td>61/70</td>
</tr>
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<td>Diff, %</td>
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<td>-4.4</td>
<td>+8.3</td>
<td>-11.8</td>
<td>-0.7</td>
<td>-7.0</td>
<td>+6.1</td>
<td>-10.8</td>
<td>-5.9</td>
<td>-7.7</td>
<td>-3.8 (6.7)*</td>
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<td>RMS of EMGs</td>
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<td></td>
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<tr>
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<td>14/16</td>
<td>0/2</td>
<td>2/8</td>
<td>0/3</td>
<td>0/2</td>
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<td>7/9</td>
<td>12/12</td>
<td>0/4</td>
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<td>3.1</td>
<td>6.6</td>
<td>10.4</td>
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<td>(ITM/RTM)×100%</td>
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<td></td>
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</tr>
<tr>
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<td>0/2</td>
<td>5/8</td>
<td>1/3</td>
<td>1/2</td>
<td>4/6</td>
<td>9/9</td>
<td>9/12</td>
<td>0/4</td>
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<tr>
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<td>79.2</td>
<td>31.2</td>
<td>-30.9 (165.2)</td>
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Iso, isometric contraction; ISI, isometric-shortening-isometric contraction; KEM, knee extensor moment; Diff (%), percent difference of ISI contractions relative to Iso contractions (values were calculated by using means of all accepted trials for each subject; - represents Iso > ISI and + represents ISI > Iso). RMS of EMGs, root mean square of electromyograms; ITM, interpolated twitch moment; RTM, resting twitch moment. *Mean knee extensor moment was significantly smaller for ISI contractions than that for Iso contractions (nos. in parentheses; 1-tailed Student's t-test for paired data with P = 0.05; n = 10 using the means of all accepted trials for each subject). Mean value for subjects who showed force depression was 6.5 ± 3.7 (SD) %, and mean value for subjects who showed force enhancement was 7.2 ± 1.6 (SD) %.

Fig. 2. Knee extensor moment-time (A) and knee angle-time histories (B) for an isometric (Iso) and an ISI contraction from 1 representative subject who showed consistent force depression (FD) following muscle shortening. Line 1, Iso contraction; line 2, ISI contraction.

Fig. 3. Mean (± SD) knee extensor moments (KEM) and corresponding mean of root-mean-square (RMS) values of knee extensor electromyogram for the 8 subjects who showed (in 61 of 62 test comparisons) FD following muscle shortening, and for the 2 subjects who showed FE following muscle shortening. Values <100% represent FD, and values >100% represent FE following muscle shortening. Similarly, RMS values <100% and >100% indicate a decrease and increase, respectively, in muscle activation in ISI compared with Iso contractions.
For the two subjects who showed consistent force enhancement following muscle shortening, the EMG values were significantly larger in the ISI compared with theIso contractions (Fig. 3).

DISCUSSION

To our knowledge, this is the first time that force depressions following shortening contractions were demonstrated during voluntary muscle contractions. Force depression was found to be statistically significant for the entire group of subjects and was consistently observed in 8 of the 10 subjects and 61 of the 62 individual test comparisons from these subjects. In these eight subjects, muscular EMG and superimposed twitch moment values were, on average, the same for the Iso and ISI tests, indicating that muscular activation was similar in both tests (Fig. 3). Under these conditions, isometric knee extensor moments following muscular shortening were smaller than the corresponding moments obtained during purely Iso contractions. These results agree with those obtained by using electrical stimulation of muscle or fiber preparations (1, 5, 6, 8, 11–13). Therefore, the results of this study lead to the conclusion that force depression following shortening is an actual property of skeletal muscle and not an artifact of artificial electrical stimulation.

The mean force depression in our study was 3.8%. The only other study in which force depressions in human skeletal muscle were investigated was the study by De Ruiter et al. (5). These researchers found maximal force depressions for electrically stimulated adductor pollicis of ~37% when shortening was over a range of 38° (of thumb adduction) at a speed of 6.1 °/s. For other contractile conditions, force depression was lower, reaching a minimum value (~4%) for the smallest shortening range (8°) and the fastest shortening speed (458.4 °/s).

There are several reasons why the maximal force depressions found by De Ruiter et al. (5) were three times the maximal values found in our study. First, it is conceivable that the supramaximal electrical stimulation and the choice of muscle might have influenced the results. Based on our results and those of De Ruiter at al., this point cannot be evaluated. Second, the contractile conditions (amount and speed of shortening) relative to the maximal range and speed of shortening might have been different for the two muscles tested. For lack of the relevant information on the adductor pollicis, this point is also hard to evaluate. Third and, in the context of this study, most importantly, the differences in force depression in the two studies might be associated with the different definitions of force depression. In our study, force depression was evaluated ~3.5 s after the end of the shortening contraction. Therefore, it might be safely assumed that the recovery of force following shortening was complete (Fig. 2). De Ruiter et al. did not define at what instant in time following the shortening contraction force depression was determined. However, from their timing of the test in which maximal force depression was measured (total test period = 8.5 s, initial Iso contraction = 1 s, period required for shortening = 38°/6.1 °/s = 6.2 s), it becomes apparent that force depressions must have been determined within 1.3 s of the end of muscle shortening. In Fig. 4 of De Ruiter et al., force depression measurements are shown after ~1.5–1.7 s. From their Fig. 4, it is obvious that the top (isometric) and bottom traces (shortening of 38° at 38.2 °/s) are not parallel (i.e., they converge), showing that force depression values would decrease if force depressions were determined at a later instant in time. For the tests in which maximal force depression was obtained, the convergence of the isometric and the shortening traces would likely be much more pronounced than those shown in Fig. 4 of De Ruiter et al., because force depression was evaluated earlier in time than is shown in their Fig. 4. Moreover, the recovery of force following 6.1 °/s shortening depends directly on the shortening speed, i.e., the slower the shortening speed, the slower the force recovery (e.g., Ref. 11). Therefore, the recovery after shortening at 6.1 °/s, which gave the maximal force depressions for De Ruiter et al., would likely be much less complete than those shown in Fig. 4 of De Ruiter et al., indicating that their maximal force depression of 37.2% is probably a vast overestimation of the actual force depression that would be measured once force had been fully recovered after the shortening contraction.

From the way the present study was performed and from the results of this study, it is not possible to elucidate a mechanism responsible for the observed force depression in voluntary human skeletal muscle contraction following shortening. However, the results of this study support the idea that force depression is an actual property of skeletal muscle rather than an artifact of artificial electrical stimulation. The most common mechanism associated with force depression following muscle shortening is the idea that, during the shortening phase, sarcomeres become nonuniform in length. Therefore, some sarcomeres are weaker than others because of their local force-length properties. These “weak” sarcomeres are thought to dictate the force capacity of the muscle, whereas the “strong” sarcomeres are somehow limited in developing their full force potential. There is no evidence that sarcomere nonuniformity is developed in the entire muscle during shortening under physiological stimulation. Furthermore, sarcomeres and fibers are connected in parallel in whole muscle; therefore, it is hard to perceive how substantial sarcomere-length nonuniformity may evolve as they have been observed to occur in single-fiber preparations (e.g., Ref. 7). Finally, large-force depressions have been observed in single-fiber preparations in which nonuniformity was prevented from occurring (8), thus indicating that mechanisms other than sarcomere nonuniformity may be responsible for the force depressions observed here and elsewhere.

In 2 of the 10 subjects, and in all eight individual comparisons between Iso and ISI contractions from these subjects, the knee extensor moments were larger in the ISI compared with the corresponding Iso contractions. This result is in contrast to all published observations in which artificial muscle stimulation was used.
it is assumed, as we have done above, that force depression following shortening is an inherent property of skeletal muscle, the results of these two subjects can only be explained by an increased muscle activation in the ISO contractions following shortening compared with the corresponding purely ISO contractions. Inspection of the EMG values of the two subjects (subjects 3 and 7, Table 1) revealed that the EMG values were always higher in the ISI than the ISO contractions, as expected (Fig. 3). The percent increase in the EMG values of these two subjects from the ISO to the ISI contractions was larger than in any of the other subjects (Table 1), suggesting that, indeed, muscular activation was increased from the ISO to the ISI contractions, thus possibly explaining the increased knee extensor moments following shortening contractions in these two subjects.

It is not clear from the results of this study why two subjects showed results opposite to those of the remaining eight subjects. These two subjects appeared to be able to compensate for the loss of muscular force potential following shortening with increased activation (Fig. 3) and thus were able to eliminate any force depression. The two subjects who were able to increase muscular activation following shortening were the only two within the subject group who lifted weights on a regular basis as part of their training. Therefore, one may speculate that it is possible to overcome the mechanical disadvantage of muscular force production following muscle shortening with systematic weight training. However, at present, the above notion remains speculation.

Summary and conclusions. To our knowledge, this is the first study in which force depressions following shortening contractions were demonstrated for voluntary contractions. From the results of this study, it can be concluded that force depression is a property of skeletal muscle contraction and not an artifact of artificial electrical stimulation. It appears that force depressions during voluntary contractions might be compensated for, partly or completely, by an increase in muscle activation following shortening beyond the levels obtained during purely ISO contractions.

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