Shortening-induced depression of voluntary force in unfatigued and fatigued human adductor pollicis muscle

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De Ruiter, C. J., and A. De Haan. Shortening-induced depression of voluntary force in unfatigued and fatigued human adductor pollicis muscle. J Appl Physiol 94: 69–74, 2003. First published September 13, 2002; 10.1152/japplphysiol.00672.2002.—The goals of this study were to investigate adductor pollicis muscle (n = 7) force depression after maximal electrically stimulated and voluntarily activated isovelocity (19 and 306°/s) shortening contractions and the effects of fatigue. After shortening contractions, redeveloped isometric force was significantly (P < 0.05) depressed relative to isometric force obtained without preceding shortening. For voluntarily and electrically stimulated contractions, relative force deficits respectively were (means ± SE) 25.0 ± 3.5 and 26.6 ± 1.9% (19°/s), 7.8 ± 2.2 and 11.5 ± 0.6% (306°/s), and 23.9 ± 4.4 and 31.6 ± 4.7% (19°/s-fatigued). The relative force deficit was significantly smaller after fast compared with slow shortening contractions, whereas activation manner and fatigue did not significantly affect the deficit. It was concluded that in unfatigued and fatigued muscle the velocity-dependent relative force deficit was similar with maximal voluntary activation and electrical stimulation. These findings have important implications for experimental studies of force-velocity relationships. Moreover, if not accounted for in muscle models, they will contribute to differences observed between the predicted and the actually measured performance during in vivo locomotion.

force deficit; voluntary activation; electrical stimulation; velocity

It has been well documented for isolated skeletal muscle (fibers) that force declines during a single loaded shortening contraction and that force remains depressed during redevelopment of the isometric force immediately after shortening (1, 3, 9, 10, 13, 17, 18). This so-called shortening-induced force deficit instantaneously disappears, when muscle activation after shortening is interrupted just long enough for the muscle to fully relax (1, 9, 10, 13). The exact mechanism behind this phenomenon remains unclear, but it is probably related to a redistribution of muscle fiber sarcomere lengths during loaded shortening (9, 10, 13, 18).

De Ruiter et al. (4) were the first to demonstrate that shortening-induced force depression in the intact human muscle-tendon complex could be at least as great as in isolated animal preparations. In their study, maximal depression of the redeveloped isometric force was ~37%, but the relative force depression during the preceding shortening phase was even greater. Moreover, force depression linearly increased with the magnitude of shortening ($r^2 = 0.98$) and with the force level during shortening ($r^2 = 0.89$) (4). Clearly, these findings would have important implications for experimental studies of force-velocity relationships because they implicate that the force-velocity relationship constantly changes during a single loaded shortening contraction. However, as rightly pointed out by Lee et al. (16), in all the previous studies, muscle (fibers) were electrically activated, and consequently the force depression could have been an artifact of the “artificial activation” without any real consequences during voluntary effort in vivo. In contrast to what happens during electrical stimulation, during voluntary contractions the motor units fire asynchronously with different and varying frequencies, which are usually much lower than the frequencies imposed during maximal electrical stimulation (2). Consequently, muscle shortening during voluntary effort may lead to an additional loss of force due to “shortening-induced deactivation” (8). On the other hand, even during maximal effort, activation is almost never truly maximal during voluntary contractions. Therefore, there is also the possibility that shortening-induced force depression may, at least to some extent, be masked by an upregulation of activation after shortening (16).

To date, there have been only two studies of shortening-induced force depression during voluntary contractions. Lee et al. (16) found a significant depression of maximal voluntary isometric knee extension force after shortening compared with the maximal isometric force measured at the same knee angle but without a preceding shortening phase. However, the depression of force was not consistently observed in all subjects and was of only small magnitude (3.8 ± 6.7%), which suggests that shortening-induced force depression may

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only be of limited importance during voluntary contractions. In a later study, Lee et al. (15) found that, in contrast to studies using electrical activation, shortening-induced force depression was independent of shortening speed during voluntary contractions and that again the force depression was rather moderate (up to 8.4%). A limitation of the studies by Lee et al. was that they did not directly compare voluntary contractions with electrically activated contractions. Consequently, it is still unclear whether shortening-induced force losses are of similar magnitude during voluntary compared with electrically activated muscle contractions.

The first objective of the present study was to directly compare shortening-induced force depression during maximal voluntary effort and electrical activation at relatively slow and fast contraction speeds in the same muscle. The intact human adductor pollicis muscle operating under in vivo conditions is studied, and consequently the present study cannot provide a detailed insight into the mechanisms of shortening-induced force depression.

As indicated above, shortening-induced force depression has been shown to decrease with the force level during shortening. Reducing the force level previously has been accomplished by either increasing the velocity of shortening (10) or by reducing the stimulation frequency during the contraction (4). Another obvious and functionally relevant way of reducing muscle force is by fatiguing the muscle. To date, there have been no studies of shortening-induced force depression in fatigued muscle, but the reduction of force caused by fatigue may attenuate the force depression.

The second objective of the present study was, therefore, to study the effects of muscle fatigue on shortening-induced force depression during maximal voluntary effort and electrical activation.

METHODS

Subjects. Seven healthy students, three men and four women, 21–24 yr of age, participated in this study. The study was approved by the local ethics committee, and all subjects took part after giving their informed consent.

Force recording and stimulation. Methods for stimulating the adductor pollicis and force recording are given in detail elsewhere (4). Briefly, the subject sat in an adjustable chair with the left forearm supinated, and the hand was held horizontally and securely fixed with the thumb abducted and in contact with a vertical pin. The pin was attached to a strain gauge mounted to the support below the plane of the hand. The forces reported in the present study are those applied by the thumb at the vertical pin. When the thumb was fully adducted, its length axis was parallel with the length axis of the index finger, and this position was defined as 0° thumb angle. Because the vertical pin of the force transducer was placed between the thumb and the index finger, the smallest thumb angle at which forces could be measured was 36°. It was possible to increase thumb angle up to 74° (maximal abduction) before anatomic limits were approached. Timing and duration of stimulation, onset and speed of motor movement, and data sampling frequency (1,000 Hz) of the force and length signal were computer controlled.

The adductor pollicis muscle was activated by percutaneous electrical stimulation of the ulnar nerve at the wrist with constant-current unidirectional square-wave pulses of 100-μs duration (model DS7, Digitimer, Welwyn Garden City, UK). The current was set 30% above the stimulus that produced maximal isometric tetanic force with 50-Hz stimulation. To maintain a constant muscle temperature, the subject's hand and forearm were immersed in a water bath at 45.0°C for 20 min before the test, and during the experiment a lamp was used to warm the hand. This procedure will lead to a muscle temperature of ~37°C (6).

Experimental protocol. The first contraction was either a maximal voluntarily or an electrically activated isometric contraction at the 74° thumb angle (applied in random order) to determine baseline forces at the longer muscle length. A short tetanic burst (80 ms, 200 Hz) was applied at the end of the voluntary contraction and 2 s later on the fully relaxed muscle. Voluntary activation was calculated as follows. The extra, stimulation-induced, force on top of the maximal voluntary contraction was expressed as a percentage of the force response of the burst applied on the relaxed muscle (e.g., Ref. 15). In this manner, the extra force responses were normalized to the maximal force response of the same active muscle mass. Expression of the extra force as a percentage of the maximal voluntary force would be less accurate, because during maximal voluntary contractions additional muscles, which were not activated with the electrical stimulation, contributed to the force production (e.g., Fig. 1). Subsequently, this percentage of additional force was subtracted from 100% to obtain voluntary activation (7). Voluntary activation had to be at least 90% otherwise the voluntary contraction was repeated.

Subsequently, a series of alternating isometric and isovelocity shortening, electrically stimulated (50 Hz), and voluntarily activated contractions was done (see below), with always 3 min of rest between contractions.

The shortening contractions started with the adductor pollicis muscle being passively stretched from a 36° thumb angle to a thumb angle of 74°. The muscle was then either electrically stimulated or maximally voluntarily activated, and after 700 ms (or 1,000 ms during fatigue) of isometric contraction, the muscle complex was allowed to shorten (thumb abduction) either at a slow (19°/s) or fast (306°/s) velocity, after which force redeveloped for 1,500 ms at the shorter muscle length (36° thumb angle). Total durations of the slow and fast contractions were 4,300 and 2,425 ms, respectively. The force deficit was defined as the difference between the redeveloped isometric force after an isovelocity shortening contraction and the force of an isometric contraction during which the thumb remained at 36° throughout the activation and stimulation. For electrically stimulated and voluntarily activated contractions, the force deficit was subsequently expressed as a percentage of respectively the maximal tetanic and voluntary isometric force at the 36° thumb angle. Relative force deficits were calculated 1,000 and 1,500 ms after the end of the shortening phase, because there is recovery of the deficit during isometric force redevelopment (4), and therefore the time of measurement may affect the results.

To determine voluntary activation, a short tetanic burst (80 ms, 200 Hz) was always applied at the end of each voluntary contraction and 2 s later on the fully relaxed muscle. Examples of the types of contractions used are presented in Fig. 1. Please note that the optimum for force production is at 51°, but the angle force relationship is very flat over the range (74–36°) of applied thumb angles (4).
Fig. 1. Examples of force traces obtained from the same subject during maximal electrical stimulation (B), maximal voluntary thumb adduction (C), and fatigue (D). Changes in thumb angle are shown in A for isometric (dotted lines), slow shortening at 19°/s (solid lines), and fast shortening at 306°/s (dashed lines). Vertical dotted lines between the redeveloped isometric force after shortening and the isometric force without preceding shortening mark the force deficits. Fifty-hertz stimulation is indicated by the solid bars above the time axis in B and D. Short, 80-ms (200 Hz) bursts are indicated by the arrows on the time axis. Superimposed (arrows on the left in C and D) burst stimulation only slightly increased voluntary force output, illustrative of near maximal voluntary activation. For reasons of clarity, the burst response after fast shortening in C has been shifted from 4,300 to 6,300 ms. Please note that during fatigue when the rate of force rise was slower (D), the isometric phase before shortening was elongated by 300 ms to begin the shortening from a force plateau. During fatigue, electrically stimulated and voluntary shortening contractions were only done at the slow speed (D).

In total, four sets of three contractions were made. The first set consisted of three electrically stimulated or voluntary (random order) contractions: isometric, slow shortening, and fast shortening (applied in random order). When the first set consisted of electrically stimulated contractions, the second set consisted of voluntary contractions. The contractions of the first and second set were respectively repeated in a third and fourth set. Thus two data sets were obtained for every condition.

If either voluntary activation before shortening or at the end of the isometric phase after shortening was <90%, the contraction was repeated. The latter occurred one to three times in five of the seven subjects.

Muscle fatigue. After the measurements in the unfatigued state had been completed, a cuff around the subject’s upper arm was inflated to 250 mmHg and the muscle was then fatigued. The adductor pollicis performed 60 electrically stimulated (50 Hz) isometric contractions of 240-ms duration, one contraction each second, at the 36° thumb angle. The 60 fatiguing contractions were immediately followed by 2 (isometric and slow shortening) voluntary and 2 (isometric and slow shortening) electrically stimulated contractions applied in random order. Subsequently, the cuff was deflated. After 5 min of rest, electrically stimulated isometric force at the 36° thumb angle had recovered to 96 ± 1.5% (mean ± SE) of the prefatigue value.

Data analysis and statistics. For the unfatigued muscle, two complete data sets were obtained for each subject. For each subject, the mean values of the two data sets were taken and subsequently entered into the statistics. The results are presented as means ± SE. Repeated-measures ANOVA with three within-subject factors was used for determination of statistical significance (P < 0.05). The within-subject factors were “activation” (voluntary or electrical), “time of assessment during isometric force redevelopment” (1,000 or 1,500 ms after shortening), and either “speed” (19 or 306°/s) or “state” (unfatigued or fatigued).

RESULTS

Voluntary isometric muscle forces were significantly (~20%) higher compared with electrically activated forces; the respective values were 84.9 ± 8.2 vs. 69.8 ± 5.9 N at the 74° thumb angle and 57.2 ± 4.0 vs. 48.0 ± 4.1 N at the 36° thumb angle. This indicates that more muscle in addition to the adductor pollicis contributed to the thumb adduction during voluntary contractions. Please note that the forces at the 74° thumb angle include ~15 N of passive force, whereas passive force was zero at the 36° thumb angle.

Voluntary muscle activation during purely isometric contractions was similar between thumb angles: 92.8 ± 0.8% at 74° and 93.7 ± 0.6% at 36°. Moreover, voluntary activation was not significantly different during isometric force redevelopment after slow (94.9 ± 0.9%) and fast (94.7 ± 0.3%) shortening contractions.

During all contractions, force continued to decline during the shortening phase, without any tendency to level off (Fig. 1), illustrating that the force exerted by the muscle during shortening at a certain constant velocity depends on the amount of preceding shortening.

After muscle shortening, redeveloped isometric force was significantly lower than isometric force obtained without preceding shortening at the same (36°) thumb angle (Fig. 1, Table 1). Relative force deficits were slightly (but significantly) lower at 1,500 ms compared with 1,000 ms after shortening (Table 1). Force deficit was significantly greater after slow compared with fast shortening contractions (Table 1), regardless whether the muscle was electrically stimulated (Fig. 1B) or voluntarily activated (Fig. 1C). An important finding of
the present study was that the force deficit after shortening at both speeds was not significantly different between the electrically induced and the voluntary contractions (Table 1), which is also clearly illustrated by Fig. 1, B and C.

Sixty repetitive ischaemic contractions significantly reduced isometric force at the 36° thumb angle, to 76.0 ± 4.3 and 78.9 ± 3.4% for the voluntarily and electrically activated contractions, respectively. Voluntary muscle activation was well maintained during fatigue, values obtained during purely isometric and during isometric force redevelopment after slow shortening contractions, respectively, were 94.7 ± 1.4% and 96.4 ± 1.1%.

Similar to the prefatigue situation, the force deficits of the fatigued muscle did not significantly differ between electrically stimulated and voluntary contractions. Moreover, the relative force deficits after slow shortening contractions during fatigue were not significantly different from those obtained before fatigue (Table 1).

### DISCUSSION

The present study is the first to demonstrate that the immediate reduction of force after a single loaded shortening contraction is independent of whether the contraction is performed with voluntary activation or electrical stimulation. Another new (unexpected) finding was that the relative shortening-induced force deficit was unchanged during muscle fatigue.

Although it has repeatedly been shown that muscle loses part of its force-generating capacity during a single loaded shortening contraction, the existence of such a shortening-induced force deficit does not seem to be a generally accepted, well-known phenomenon. In studies on shortening-induced force depression, the loss of force is usually quantified by measuring the reduction of the redeveloped isometric force after shortening. The shortening-induced force deficit has a positive linear relationship with displacement (1, 17, 18) and the force level during shortening (10). The rapid recovery of the force deficit immediately after muscle relaxation (1, 9, 10, 13) suggests that probably a mechanical factor plays an important role. Indeed, there are strong indications from the work on isolated fibers that the shortening-induced force deficit is caused by an increase of sarcomere heterogeneity along the fiber length (9, 10, 13, 18). It is important to distinguish shortening-induced force depression from shortening-induced deactivation, which is probably caused by a transitory decrease in the calcium affinity of the troponin binding sites during stimulation at low frequencies (8). Compared with electrical stimulation at 50 Hz, adductor pollicis motor units on average fire at relatively low frequencies during maximal voluntary effort (27 Hz, range: 10–50 Hz), and motor unit firing frequencies even further decline during fatigue (2). Therefore, especially during the voluntary contractions, in addition to the shortening-induced force depression, some degree of shortening-induced deactivation may occur. However, there are several reasons why we believe that shortening-induced deactivation did not play an important role in the present study (see also discussion in Ref. 9). First, force was depressed during and after maximal electrically activated and consequently fused tetanic contractions, whereas deactivation is only present during unfused tetani. Second, unlike what is seen with deactivation, the depression was velocity dependent, and, third, it was present for as long as the muscle was activated. Because the deficit and the velocity dependency of the deficit were similar between voluntary and electrically stimulated contractions, we conclude that also during voluntary contractions shortening-induced force depression and not deactivation was the major cause of the force depression.

De Ruiter et al. (4) were the first to demonstrate that shortening-induced force deficit also occurred in (electrically stimulated) human muscle, and they also showed that these deficits were even greater (up to 37%) than those obtained in isolated animal preparations. The latter was suggested to be related to more series elastic structures with the muscle in vivo compared with the isolated preparations studied by others. However, recently Lee et al. suggested that “changes in activation” during and after a maximal voluntary knee extension might explain the absence of a deficit in some of their subjects (16) and account for the absence of a velocity effect in another study (15). However, in the present study the shortening-induced force depression was velocity dependent and, as stated before, of similar size during voluntary contractions. Therefore our results do not indicate that shortening-induced force losses were (partly) masked by an upregulation of muscle activation during voluntary effort as suggested by Lee et al. (16). This is supported by the fact that, in the present study, voluntary activation during the isometric phases before and after shortening was at least 90% (on average 95%) of the maximum in all contractions, which leaves little room for an upregulation of activation. Moreover, during pilot experiments, superimposed electrical stimulation during the shortening phase did not cause any detectable force increases.

### Table 1. Relative force deficits after isovelocity shortening contractions

<table>
<thead>
<tr>
<th>Thumb Angular Velocity, °/s</th>
<th>Voluntary Activation at 1,000 ms</th>
<th>Voluntary Activation at 1,500 ms</th>
<th>Electrical Stimulation at 1,000 ms</th>
<th>Electrical Stimulation at 1,500 ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>306</td>
<td>10.4 ± 3.1*</td>
<td>7.8 ± 2.2*</td>
<td>12.0 ± 0.8*</td>
<td>11.5 ± 0.6*</td>
</tr>
<tr>
<td>19</td>
<td>26.1 ± 3.4</td>
<td>25.0 ± 3.5</td>
<td>28.2 ± 2.0</td>
<td>26.6 ± 1.9</td>
</tr>
<tr>
<td>19 Fatigue</td>
<td>27.0 ± 5.2</td>
<td>23.9 ± 4.4</td>
<td>34.5 ± 4.7</td>
<td>31.6 ± 4.7</td>
</tr>
</tbody>
</table>

Values are means ± SE. Force deficits (% maximal voluntary or tetanic isometric force at the 36° thumb angle) were obtained during isometric force redevelopment 1,000 and 1,500 ms after the end of an isovelocity shortening contraction at 306 and 19°/s, performed with voluntary activation or electrical stimulation. Fatigue, values obtained after slow shortening of the fatigued muscle, *Significant difference between velocities, P < 0.05. There also was a significant main effect (not denoted) of the time of measurement indicating that the force deficits obtained at 1,500 ms were (slightly) smaller compared with the force deficits obtained at 1,000 ms. There were no significant differences between voluntary activated and electrically stimulated muscles.
Therefore, we are positive that our results are unaffected by any changes in voluntary muscle activation during the contractions. The present results clearly show that, in human adductor pollicis muscle, shortening-induced force depression is an important phenomenon, which is seen not only with electrical stimulation but also during maximal voluntary activation.

The presented force deficits of ~26 and 10% after shortening at 19 and 306/s, respectively, are quite substantial but somewhat smaller than the values previously obtained after shortening at the same speeds in other subjects (4). It should be kept in mind that the exact magnitude of the calculated force deficit depends on the time of force measurement after the end of shortening. Redeveloping isometric force only slowly approached a plateau value (Fig. 1), which can also be seen in other preparations (4). In the present study, the shortening deficits obtained after 1,500 ms of isometric force redevelopment were, at least in the unfatigued muscle, only marginally smaller than at 1,000 ms, indicating that the force deficits in the present study were obtained when the deficit had almost stabilized. Nevertheless, pilot experiments showed that, in the most extreme case, electrical stimulation had to be continued for 6,500 ms (instead of 1,500 ms) after shortening before the force deficit had completely stabilized. Such long stimulation times (over 9 s in total) are not practical because they are unpleasant, fatigue inducing, and almost impossible to produce repetitively during voluntary contractions. Moreover, even after complete redevelopment of isometric force in the pilot experiments, deficits were still 85–90% of those obtained at 1,500 ms. A slow recovery of redeveloping isometric force after shortening would be in agreement with an ongoing readjustment of sarcomere lengths within the muscle after shortening.

Previous studies have shown that the relative shortening-induced depression of force was positively (linearly) related with the force level during shortening (1, 17, 18). In earlier work, our laboratory reduced absolute force levels to ~73% by stimulating the muscle with 20 Hz instead of 50 Hz; this also reduced the relative force deficit to ~73% (4). In the present study, contractile force was reduced to about a similar (~77%) extent by fatigue inducing contractions. However, and rather unexpectedly, the relative force deficit was unaffected by fatigue. The present data cannot be compared with those of others because shortening-induced force depression during fatigue has not been studied before. However, the present results indicate that the way by which force is reduced has important consequences for the extent of the shortening-induced force deficit. To account for our results, we propose the following explanation. By reducing the stimulation frequency, force is reduced by a reduction of the proportion of attached cross bridges at any moment, leading to a force reduction in each muscle fiber. Less force per active fiber could reduce the force deficit of the muscle by decreasing the degree of sarcomere inhomogeneity along the length of all the muscle fibers. In contrast, repetitive ischemic contractions may reduce the number of active fibers, because the fastest and greatest metabolic changes will occur in the fast fibers, which could make those fibers unresponsive to the activation (14). Therefore, the force deficit would be expected to decline in proportion to the fatigue-related force loss, as in fact was the case in the present study. Clearly, with the present setup, any explanation for (changes of) the shortening-induced force depression is speculative. Nevertheless, and whatever the exact underlying mechanism may be, the present results demonstrate that the relative depression of force output is of similar magnitude in fatigued and unfatigued muscle, regardless whether the activation is electrical or voluntary.

Several decades ago, Joyce et al. (11, 12) already showed in cat soleus muscle that the force, or shortening velocity, obtained at a certain muscle length depends on the amount of preceding shortening and that, consequently, for any given muscle, a family of force-velocity curves could be constructed. More recently, it was shown that the same is true in electrically stimulated human muscle (4, 5). With the present work, we demonstrate that, also during a single voluntary contraction, both in unfatigued and fatigued muscle, a decline of force persists during shortening although the imposed velocity is constant. As indicated before, there is a significant degree of recovery of the force deficit during the isometric phase after shortening (4). Therefore, the force deficits obtained after 1,000–1,500 ms of isometric force redevelopment, although they were still quite substantial (~25% at 19%), must be a vast underestimation of the relative force losses immediately at the end of the shortening phase. However, because the degree of force depression during shortening is dependent on many (history dependent) factors, it is difficult to incorporate this phenomenon in models used to predict the contribution of individual muscles during in vivo movements. Nevertheless, one must realize that the use of just a “standard” force-velocity relationship of each muscle in such models will lead to large deviations between the predicted and the actually measured performance during in vivo locomotion.

In conclusion, the velocity-dependent reduction of force after and during a single loaded isovelocity shortening contraction is of similar relative size with maximal voluntary activation and electrical stimulation, both in unfatigued and fatigued human adductor pollicis muscle. These findings have important implications for experimental studies of force-velocity relationships. Moreover, if not accounted for in muscle models, they will contribute to differences observed between the predicted and the actually measured performance during in vivo locomotion.

REFERENCES


