Posttetanic potentiation of human dorsiflexors

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O’Leary, Deborah D., Karen Hope, and Digby G. Sale. Posttetanic potentiation of human dorsiflexors. J. Appl. Physiol. 83(6): 2131–2138, 1997.—Twitch contractions of the ankle dorsiflexors were evoked before and after applied 7-s tetanic stimulation at 100 Hz in 20 young adults. Torque decreased 15% during the tetanus. At 5 s after tetanus, twitch peak torque had potentiated 45%. Potentiation declined to 28% after 1 min, rose slightly to 33% at 2 min, and declined slowly with potentiation still 25% after 5 min. There was large intersubject variation in the amount of potentiation (5–140%) and its persistence (5 to 10 min). The muscle compound action potential (M wave) did not change significantly (from pretetanic value) at 5 s after tetanus but increased sharply (26%) at 2 min and then subsided. Twitch half relaxation time (23%) decreased significantly more than twitch rise time (13%) 5 s after tetanus and recovered more slowly. Twitch rates of torque development (75%) and relaxation (71%) increased similarly 5 s after tetanus and were still elevated (~25%) at 5 min. The extent of twitch torque potentiation was significantly inversely correlated with pretetanic twitch rise time \( r = -0.69 \) and twitch-to-tetanus ratio \( r = -0.66 \). The data indicate that posttetanic potentiation has a greater effect on twitch half relaxation time than on time to peak torque and is more prominent in muscles with a short twitch time course and small twitch-to-tetanus ratio.

The force of a twitch is greater after than before a brief tetanus. This enhancement is called posttetanic potentiation (PTP) (4). PTP is greatest immediately after the tetanus and then decays rapidly but is still evident for \( \approx 10 \) min (4, 10, 11, 29). PTP is often associated with a shortening of twitch contraction and half relaxation time (3, 4, 7, 14, 20, 21) and increased rate of force development (6, 15, 20, 21, 26). The mechanism of PTP may be phosphorylation of myosin light chains during tetanus, which renders actin–myosin more sensitive to Ca\(^{2+}\) in a subsequent twitch (6, 20).

PTP is present in amphibian (22) and mammalian (5) skeletal muscle. In humans, PTP can be induced by voluntary contractions (3, 7, 10, 21, 28, 29) as well as tetanic electrical stimulation (13, 14, 28). When twitch potentiation is induced by voluntary contractions, the term postactivation potentiation (PAP) is sometimes used instead of PTP (3, 28, 29). PTP or PAP has been shown in a variety of human muscles, including a facial muscle (13), small hand muscles (11, 26), elbow flexors (14), knee extensors (7, 10, 25), and ankle dorsiflexors and plantar flexors (3, 21, 28, 29).

The magnitude of PTP is influenced by the methods and conditions under which it is evoked. PTP is affected by the intensity, frequency, and duration of the conditioning tetanus (6); maximal stimulation of the whole muscle at high frequency for \( \sim 5–10 \) s causes the greatest immediate PTP (4). Maximal voluntary contractions (MVCs) lasting \( \sim 10 \) s cause the greatest PAP (29). PTP and PAP are greater at short than at long muscle lengths (25, 29). At lower muscle temperatures, pre- and posttetanic twitch force are increased and unaffected, respectively, causing reduced PTP (5).

The extent of PTP is also affected by the characteristics of the muscles tested, the most important being the duration of the twitch contraction; muscles with the shortest twitch contraction times exhibit the greatest PTP (4, 5, 13, 18, 29). For example, human gastrocnemius shows greater PTP than soleus (27), ankle dorsiflexors have greater PTP than plantar flexors (3, 29), and plantymas has greater PTP than adductor pollicis (13). Within a muscle, motor units with a shorter contraction and relaxation time show greater PTP (1, 12). The greater PTP in fast-twitch muscles (fibers) may be related to their greater capacity for myosin phosphorylation in response to a tetanus (6). Finally, muscles and muscle fibers with the lowest pretetanic twitch-to-tetanic force ratio exhibit the greatest PTP (1, 19, 22).

In the present study we examined PTP in human dorsiflexors. We monitored changes in twitch torque and time-related contractile properties immediately and for several minutes after a conditioning tetanic contraction. Concurrent changes in the muscle compound action potential (M wave) were also monitored. Given that PTP is greater in faster contracting muscles (e.g., gastrocnemius vs. soleus), we determined whether there was a correlation between pretetanic twitch contraction and relaxation time and PTP in one muscle group (dorsiflexors), i.e., whether subjects with a shorter pretetanic twitch contraction and relaxation time tended to show greater PTP. On the basis of previous reports of PTP being greater in muscle fibers and motor units with a smaller pretetanic twitch-to-tetanus ratio, we tested for a correlation between the pretetanic twitch-to-tetanus ratio and the magnitude of PTP.

METHODS

Subjects

Ten women and 10 men participated. Their average age was 20.6 ± 1.0 (SD) (range 19–23) yr. They were healthy kinesiology students with no history of neuromuscular disorders but were not participating in strength and endurance training programs. They refrained from consuming caffeine for the 24 h before the experiment. The subjects participated with informed, written consent, and the study was approved by McMaster University’s Human Ethics Committee.

Apparatus

The right ankle dorsiflexors were tested in a custom-made apparatus that fixed the seated subject’s knee joint at 90° (1.57 rad) but allowed the ankle joint to be fixed and tested at various joint angles (16). For this study the ankle was
plantar flexed 20° from the neutral position (90° angle formed by tibia and sole of foot). This joint position, by putting the dorsiflexors on stretch, ensured a maximal twitch response (16, 30). Braces and Velcro straps secured the lower leg and foot to the apparatus. The foot was strapped to a thin aluminum plate instrumented with a strain gauge, which sensed torque created by contraction of the dorsiflexors. The signal from the strain gauge (sample rate = 3 kHz) was amplified and filtered, converted from analog to digital (model DI420, Dataq Instruments), and analyzed with customized and ACODAS software on an IBM-compatible personal computer.

Stimulation and Electromyogram Recording

The common peroneal nerve was stimulated to activate the ankle dorsiflexors, which include tibialis anterior, extensor hallucis longus, extensor digitorum longus, and peroneus tertius. The principal dorsiflexor is tibialis anterior, which is also an inverter. The common peroneal nerve also supplies the peroneus longus and brevis, the action of which is ankle joint eversion. Peroneus longus is also a weak plantar flexor. The opposing (to dorsiflexion) action of the peronei was minimalized by testing with the ankle joint plantar flexed 20°, which placed these muscles at a shortened length, where their force output was small (23), while at the same time placing the dorsiflexors at a relatively long muscle length, where their force output was at or near maximal (16). The common peroneal nerve was stimulated by lead plate electrodes covered by saline-imregnated cloth placed on the skin overlying the head of the fibula (3 × 3 cm) and the proximal portion of tibialis anterior (3.5 × 5 cm). The skin was shaved and sanded, and isopropyl alcohol was applied. The stimuli were rectangular voltage pulses of 100-µs duration delivered from a Grass S11 stimulator. Ag-AgCl electromyogram disposable recording electrodes (3.8 mm diameter) were applied to the skin over the belly of the tibialis anterior (stigmatic), the inferior extensor retinaculum (reference), and the calf (ground). Electromyogram signals (sample rate = 3 kHz) were amplified (>1,000) and filtered (10 Hz–2 kHz). Analog-to-digital conversion and analysis were the same as for twitch torque (see above).

Protocol

On reporting to the laboratory, the subject sat resting for ~30 min before the right leg was placed in the apparatus. A maximum pretetanic twitch response was elicited by delivering a series of single stimuli of increasing intensity until a plateau of twitch torque and muscle compound action potential (M wave) amplitude was obtained. The same stimulus intensity was used for tetanic stimulation and the posttetanic twitch responses. Two minutes after the pretetanic twitch had been established, tetanic stimulation was applied for 7 s at a frequency of 100 Hz. A frequency of 100 Hz was chosen because this frequency is necessary to produce a maximal tetanic contraction of the dorsiflexors (16). The 7-s duration was within the range shown to produce maximal PTP (4, 29). Posttetanic twitches were elicited (P after the tetanus) at 0.5-min intervals (e.g., 0.5 and 1.0 min) for the first 8 min after tetanus, and at 1-min intervals thereafter until 20 min had elapsed. In a given subject, twitches were not evoked beyond the time when the twitch torque had returned within 5% of the pretetanic value. In some subjects, potentiation was still >5% when recordings were terminated at 20 min. Two minutes after the last posttetanic twitch was recorded, subjects performed three isometric MVCs of the dorsiflexor muscles. The joint position was the same as for previous tetanus and twitch measurements. Each MVC lasted for 5 s, and 2-min rest periods were allowed between trials. The best of the three trials (greatest peak torque) was taken as the MVC. The MVCs were not performed before the pretetanic twitch measurements to prevent the potentiating effects of the former from affecting the latter. The posttetanic twitch was recorded within the range shown to produce maximal PTP (4, 29). The MVCs would have been little influenced by the previous 7-s tetanic contraction and series of subsequent twitch contractions.

Measurements

Twitch-response measurements included peak torque, rise time (time from 10 to 90% peak torque), and half relaxation time. Time to peak torque (~3–100% peak torque), rather than rise time, is often used to measure the duration of the rising phase of the twitch. Although there is a high correlation between these two measures, we found in the present study that the time to peak torque was more variable. Therefore, we elected to use the rise time. Peak rates of twitch torque development (RTD) and relaxation (RTR) were also measured as the greatest change in torque over a 3-ms period on the rising and falling phases of the twitch contraction, respectively. Peak torque of the tetanic contraction and the decrease in torque during the 7-s tetanus were determined. The peak-to-peak amplitude of the muscle compound action potential (M wave) associated with each twitch response was measured.

Statistics

A repeated-measures analysis of variance (ANOVA) was used to test whether the tetanus changed the twitch contractile properties at the various time points after tetanus. To determine whether significant differences from pretetanic values occurred, ANOVA was done on the measures expressed in the units of measurement (e.g., torque in N·m). To determine significant differences between measures (e.g., torque vs. M-wave amplitude), ANOVA was done on the posttetanic values normalized as a percentage of the pretetanic value (pretetanic values = 100). When significant main effects or interactions were found, Tukey’s post hoc procedure tested for differences among mean values. Some measures were correlated with others by using the Pearson correlation coefficient (r). Statistical significance was set at P < 0.05. Descriptive statistics include means ± SD and SE.

RESULTS

MVC and Tetanic and Pretetanic Twitch Contractile Properties

Peak torque of MVC was 38.1 ± 9.5 (SD) N·m, which exceeded peak tetanic torque (29.7 ± 8.1 N·m) by 30 ± 14% (P < 0.001). During the 7-s tetanus at 100 Hz, peak torque decreased 15.2 ± 5.4% (P < 0.001). Pretetanic twitch peak torque was 3.6 ± 1.8 N·m, giving a twitch-to-tetanus ratio of 0.11 ± 0.03. Twitch rise time and half relaxation time were 49.2 ± 6.6 and 82.4 ± 11.1 ms, respectively. Peak RTD and RTR were 88.9 ± 36.3 and 38.5 ± 14.5 N·m·s⁻¹, respectively. The peak-to-peak amplitude of the M wave was 9.3 ± 3.3 mV.

Posttetanic Twitch Contractile Properties

Peak twitch torque and M wave. Figure 1 shows a typical test sequence of pretetanic twitch, 100-Hz tetanic stimulation for 7 s followed after 5 s by the first
posttetanic twitch. In this example, tetanic torque decreased 13% during the 7-s tetanus. Peak torque potentiated by 50% immediately after tetanus, with little change (−4%) in M-wave amplitude. Figure 2 shows twitch responses evoked before tetanus and over a 5-min period after tetanus. PTP was maximal (48%) in the first response after tetanus, then decayed, such that PTP was reduced to 8% after 5 min. M-wave amplitude increased only 5% immediately after tetanus and was still elevated by 3% 5 min after tetanus. Figure 3 shows, for all subjects combined, the changes in peak torque and M wave over the first 5 min after tetanus. Immediate (5-s) PTP averaged 44.6%, decreased to 28% at 1 min after tetanus, increased to 33% at 2 min, then decreased to 25% at 5 min. The potentiation was significant (time main effect, $P < 0.0001$) throughout the 5-min posttetanic period. A second apparent increase in potentiation at 2 min after tetanus, more easily seen in Fig. 4, was not significantly greater than the 1-min value. In comparison to twitch peak torque, there was little change in M-wave amplitude during the posttetanic period; however, M-wave amplitude was significantly (time main effect, $P < 0.02$) elevated (26%) above the pretetanic value at 2 min. Peak torque potentiation significantly exceeded M-wave potentiation at all time points except 2 and 5 min (measure main effect, $P < 0.005$). The pattern of relative change differed significantly (time × measure interaction, $P < 0.005$) between peak torque and M wave, the former and latter showing greatest potentiation at 5 s and 2 min, respectively. However, the significant elevation of M-wave amplitude at 2 min (26%) corresponded to the apparent second increase of torque potentiation at 2 min (33%).

Figure 3 is restricted to the first 5 min after the tetanus, because this was the last time point at which measurements were made in all subjects (making ANOVA on all subjects possible). At time points beyond 5 min, measurements in some subjects were no longer made, because their twitch peak torque had declined to within 5% of the pretetanic value. In Fig. 4 the subjects were divided into three groups on the basis of the time to termination of measurements. For each group, values are shown up to the time that all subjects were included (lower limit of group range). ANOVA up to the 5-min point in these three groups indicated no significant difference in peak torque potentiation, although Fig. 4 shows a trend ($P = 0.094$) of greater potentiation being associated with a longer persistence of potentiation. On the other hand, the correlation between initial potentiation and time to return within 5% of the pretetanic value was not significant ($r = 0.25$, $P = 0.30$).

Twitch rise time and half relaxation time. Figure 5 shows an example of pretetanic and immediate posttetanic twitches superimposed. A shortened time course...
of the posttetanic twitch is evident, along with increased RTD and RTR. In the subjects collectively, twitch rise time and half relaxation time decreased significantly after tetanus (Fig. 6; time main effect, \( P < 0.0001 \)). Except for the 4.5-min value, half-relaxation time remained significantly reduced throughout the first 5 min after tetanus. In contrast, rise time was no longer significantly shortened after 1 min. The immediate relative decrease in half relaxation time (23%) was significantly greater than rise time (13%), and this was also true for most additional time points during the 5-min posttetanic period (measure main effect, \( P < 0.006; \) time \( \times \) measure interaction, \( P < 0.0001 \)). Subjects with longer pretetanic rise times tended to show a greater absolute (ms) shortening of rise time after tetanus (\( r = 0.48, P < 0.05 \)). On the other hand, there was no correlation between pretetanic rise time and the relative (%) decrease in rise time (\( r = 0.28, P = \) not significant [NS]). Similarly, a longer pretetanic half relaxation time was associated with a greater absolute (\( r = 0.52, P < 0.02 \)), but not relative (\( r = 0.33, P = \) NS), decrease in half relaxation time with potentiation.

RTD and RTR. RTD and RTR increased sharply immediately after tetanus and then decayed rapidly for 30 s and more slowly thereafter (Fig. 7). RTD was significantly (time main effect, \( P < 0.0001 \)) increased at all time points up to 5 min, whereas RTR was greater at all time points up to 5 min, whereas RTR was greater at
5 s and 0.5, 2, and 3 min (time main effect, $P < 0.0001$). The relative (%) changes in RTD and RTR were similar (no measure main effect or measure × time interaction). This contrasted with the comparison of rise time and half relaxation time, in which the latter showed a greater percent decrease than the former (Fig. 6).

The changes in RTD and RTR more likely reflected changes in peak torque than rise time and half relaxation time, in which the latter showed a greater percent decrease than the former (Fig. 6).

The changes in RTD and RTR more likely reflected changes in peak torque than rise time and half relaxation time, respectively, because the correlations in pretetanic twitches between RTD ($r = 0.94$, $P < 0.001$) and peak torque and between RTR ($r = 0.78$, $P < 0.001$) and peak torque were high. In contrast, the correlations between RTD and rise time ($r = 0.2$, $P = NS$) and between RTR and half relaxation time ($r = 0.01$, $P = NS$) were low and nonsignificant. To eliminate the influence of peak torque on RTD and RTR, the latter were expressed in relation to the former by dividing RTD and RTR values by peak torque (PT) values.

RTD/PT increased 21% 5 s after tetanus and remained significantly (time main effect, $P < 0.0001$) elevated at 0.5 and 1 min. There was no significant change in RTR/PT. The changes in RTD/PT and RTR/PT were smaller than the changes in RTD and RTR (Fig. 7). For example, the immediate posttetanic increase in RTD was 75%, whereas RTD/PT increased 21%.

Correlation between peak torque potentiation and rise time, half relaxation time, and RTD and RTR. There was a significant inverse correlation between peak torque potentiation and pretetanic twitch rise time and half relaxation time: a briefer twitch was associated with greater potentiation (Fig. 8). However, Fig. 8 reveals that two subjects were outliers, in that their PTP values were $>2$ SDs above the mean. When these subjects were removed, the correlations decreased to $r = -0.44$ ($P = NS$) and $r = -0.22$ ($P = NS$) for PTP vs. rise time and half relaxation time, respectively. In contrast, there was no correlation between peak torque potentiation and RTD ($r = -0.36$, $P = NS$) and a negative correlation between peak torque potentiation and RTR ($r = -0.53$, $P < 0.02$). Thus there was a paradox in which greater peak torque potentiation was associated with pretetanic twitches with shorter half relaxation times but a slower RTR. The paradox is resolved with the observation, noted earlier, that RTR is correlated more with peak torque ($r = 0.78$, $P < 0.001$) than with half relaxation time ($r = 0.01$, $P = NS$); similarly, RTD is correlated more with peak torque ($r = 0.94$, $P < 0.001$) than with rise time ($r = 0.20$, $P = NS$). If RTD and RTR are normalized to peak torque (RTD/PT and RTR/PT), there is a positive correlation between pretetanic RTD/PT and peak torque potentiation ($r = 0.55$, $P < 0.02$). There is no correlation between RTR/PT and peak torque potentiation ($r = 0.002$, $P = NS$).

Correlation between peak torque potentiation and the twitch-to-tetanus ratio. A smaller twitch-to-tetanus ratio was associated with greater peak torque potentiation (Fig. 9). In contrast to the effect of removing the two PTP outliers in the correlation between PTP and rise time and half relaxation time (Fig. 8), which weakened the correlation, removing the (same) outliers in Fig. 9 increased the correlation to $r = 0.73$ ($P < 0.001$). Because rise time and half relaxation time and the twitch-to-tetanus ratio were correlated with the amount of peak torque potentiation, we tested whether there was a correlation between twitch-to-tetanus ratio and rise time or half relaxation time. There was a

![Fig. 7. Changes in twitch rates of torque development and relaxation over first 5 min after tetanus. Dashed line, pretetanic values. Values are means ± SE. *Significantly different from pretetanic value, $P < 0.01$. At no time point was there significant difference between rate of torque development and rate of torque relaxation.](image)

![Fig. 8. Correlation between immediate posttetanic (5-s) potentiated twitch peak torque, expressed as a percentage of pretetanic twitch torque, and pretetanic twitch rise time (A) and half relaxation time (B).](image)
positive correlation between twitch-to-tetanus ratio and rise time ($r = 0.61, P < 0.01$) but not half relaxation time ($r = 0.33, P = NS$).

**DISCUSSION**

Potentiation of twitch torque in human dorsiflexors occurs after brief MVCs (3, 28, 29). A limitation of using voluntary contractions to induce potentiation is that some subjects may not be able to fully activate their muscles (2). This would prevent maximal potentiation and cause larger intersubject variation. This limitation should not be present when, as in the present study, a maximal and constant level of electrical stimulation is used. However, the potentiation we observed in the dorsiflexors with tetanic stimulation was less and the intersubject variation no less than the potentiation induced by MVCs (28, 29).

The reason for the greater potentiation with voluntary contractions is not clear. A similar (29) or greater (28) joint angle was tested with the voluntary contractions, so the known influence of muscle length on potentiation (greater potentiation at shorter lengths) was not a factor (29). The duration of activation (3- to 10-s voluntary contraction vs. 7 s of tetanic stimulation) was also similar. Stimulation at 100 Hz is necessary to attain maximum tetanic force of the dorsiflexors (16). This frequency, if it could be achieved in voluntary contractions, could be maintained for only a few hundred milliseconds at most (8). Stimulation at 100 Hz for 7 s might tend to cause some fatigue at the neuromuscular junction (24), reducing the activation of the fibers. However, torque decreased on average by only 15% during the tetanus, and M waves were well preserved. Finally, the observed greater voluntary than evoked tetanic torque, reported elsewhere under similar conditions (30), might suggest that the stimulation failed to fully activate the dorsiflexors. However, care was taken to ensure that the stimulation was maximal; i.e., stimulus intensity was increased beyond that producing maximal twitch and M-wave responses. Stimulation of the peroneal (anterior tibial) nerve also activates peroneus longus, which is a plantar flexor rather than a dorsiflexor. Its activation may have diminished the torque generated by the dorsiflexors. However, this muscle is a weak plantar flexor, especially at an ankle joint angle of 20° plantar flexion (23), when it is at a shortened length. The most likely reason for the greater voluntary than evoked tetanic torque was the benefit of activation of synergists and fixators, which contribute to the skill of performing a voluntary contraction.

The similar intersubject variation in potentiation with voluntary and evoked contractions indicates that factors other than level of voluntary activation contribute to the large intersubject variation in potentiation (PTP). The present study identified three factors. Variation in twitch rise time, half relaxation time, and twitch-to-tetanus ratio correlated with variation in PTP. There are other possible factors. In some subjects the 30-min rest period before the experiment may have been insufficient to extinguish lingering potentiation from some activities before the subjects reported to the laboratory. Although all experiments were done at the same room temperature, muscle temperature was not monitored. It is possible that intersubject differences in muscle temperature contributed to the intersubject variation in PTP. Finally, there may have been intersubject differences in the joint angle-torque (length-tension) relation, which would have affected the amount of PTP at 20° of plantar flexion.

The time course of decay in PTP was also variable, ranging from 5 to 20 min with an average of ~10 min. The average value is similar to previous reports in dorsiflexors (29) and other muscle groups (4, 10, 11, 14, 26). In our study the decay in PTP from the immediate posttetanic value was not a simple exponential function, as sometimes observed (4, 10, 13–15). Instead, PTP declined over the 1st min after tetanus but then showed a small increase at 2 min before decreasing again (best seen in Fig. 4). This triphasic pattern of decay has been shown in the dorsiflexors after voluntary contractions (29) and in other muscle groups (7, 11). Previous studies did not test whether the second increase was significantly greater than the preceding decrease. In the present study, the second increase was not found to be significant compared with the initial decrease. Nevertheless, some investigators have suggested that the initial decay in potentiation is caused partially by fatigue; as fatigue wanes, the level of potentiation increases, again before falling away (49). Fatigue may have been a factor in our study, since tetanic torque decreased 15% during the 7-s tetanus.

There was a small and nonsignificant immediate (5-s) posttetanic change in M-wave amplitude, at a time when PTP was maximal. This common observation (3, 4, 13, 14, 27) indicates that the mechanism of twitch-torque potentiation involves excitation-contraction coupling and/or myosin-actin interaction, rather than amplified excitation of muscle fibers (i.e., enlarged muscle action potentials). A surprising finding was the significant increase in M-wave amplitude at 2 min after tetanus, although the M wave may enlarge during or after low-frequency tetanic stimulation (17) and immediately after a single (9) or a series of brief voluntary
contractions (17). The mechanism of M-wave potentiation is likely stimulation of the fiber membrane’s Na\(^+\)-K\(^+\) active transport mechanism (17). The increase in M wave occurred simultaneously (2 min) with the second increase in torque, suggesting that the former might have contributed to the latter. A larger muscle action potential might increase Ca\(^{2+}\) release from the sarcoplasmic reticulum, thereby increasing force.

The potentiated twitch had decreased rise time and half relaxation time, with a greater relative decrease in the latter. This is in general agreement with previous studies of dorsiflexors (3, 29), other human muscles (14, 29), and animal muscles (4, 15, 20). Previous studies had not tested whether the relative (%) change was significantly greater in half relaxation time than in rise time. We found that the half relaxation time decreased significantly more than the rise time, and the former’s decrease persisted longer into the posttetanic period. Twitch rise time and half relaxation time are related to Ca\(^{2+}\) release from and reuptake by the sarcoplasmic reticulum, respectively. It appears that the potentiation mechanism has a greater effect on the latter.

In contrast to potentiation’s greater effect on half relaxation time than on rise time, the relative changes in RTD and RTR were similar. When these latter measures were normalized to twitch torque, the effect was greater in RTD. Thus rise time and half relaxation time, on the one hand, and maximum RTD and RTR, on the other, cannot be used interchangeably as measures of the time rate of activation and deactivation of the twitch contractile process when correlated with PTP. We found that the maximum RTD and RTR, representing the steepest slopes (over 3 ms) of the rising and falling phases of the twitch contraction, were correlated more with twitch peak torque than with rise time or half relaxation time.

We found an inverse correlation between PTP and pretetanic twitch rise time and half relaxation time: subjects with a shorter twitch rise time and half-relaxation time tended to have greater PTP. This correlation within one muscle, not previously tested in humans, is in agreement with previous research indicating greater PTP in fast-contracting muscles consisting of a high percentage of fast-twitch (type II) fibers (1, 3–5, 12–14, 18, 27, 29).

There was also an inverse correlation between pretetanic twitch-to-tetanus ratio and PTP: subjects with a smaller twitch-to-tetanus ratio tended to have greater PTP. Our results confirm previous observations (1, 19, 22). The explanation for the correlation may be that a small twitch-to-tetanus ratio, caused by a depressed twitch, gives more scope for potentiation by a tetanus. A small twitch-to-tetanus ratio (i.e., depressed twitch) could be the result of a high proportion of fast-twitch fibers, which would produce a twitch with a very brief “active state” (and short contraction time). It would be difficult to stretch the series elastic component in such a short time, and the twitch-to-tetanus ratio would be depressed. We did find a positive correlation between twitch rise time and twitch-to-tetanus ratio, although it should be acknowledged that some research indicates similar twitch-to-tetanus ratios in fast- and slow-twitch muscles, despite the expected differences in PTP (5, 13, 26, 30). Alternatively, a low twitch-to-tetanus ratio might be related to a high muscle compliance at a given muscle length; in a compliant muscle, it would be difficult for the brief active state to fully stretch the series elastic component. In the ankle dorsiflexors, contraction time and twitch-to-tetanus ratio were smaller (30) and PTP was greater (29) at shorter muscle lengths, where compliance is high.

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