Fatigue and recovery of power and isometric torque following isotonic knee extensions

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Submitted 21 April 2005; accepted in final form 21 June 2005

Cheng, Arthur J., and Charles L. Rice. Fatigue and recovery of power and isometric torque following isotonic knee extensions. J Appl Physiol 99: 1446–1452, 2005. First published June 23, 2005; doi:10.1152/japplphysiol.00452.2005.—The purpose of this study was to assess fatigue and recovery of isotonic power and isometric contractile properties after a series of maximal isotonic contractions. Using a Biodex dynamometer, 13 men [26 yr (SD 3)] performed isotonic [50% of isometric maximal voluntary contraction (MVC) every 1.2 s through 75° range of motion] single-limb knee extensions at the fastest velocity they could achieve until velocity was reduced by 35%. Time to task failure was 38 s, and, compared with baseline, power declined by ∼42% [741.0 (SD 106.0) vs. 426.5 W (SD 60.3) at task failure], and MVC declined by ∼26% [267.3 (SD 42.5) vs. 198.4 N·m (SD 45.7) at task failure]. Power recovered by 5 min, whereas MVC did not recover, and at 10 min was only ∼85% of baseline. Isometric MVC motor unit activation was ∼95% at rest and was unchanged at task failure (∼96%), but a small amount of failure was apparent between 1.5 and 10 min of recovery (∼87 to ∼91%). Half relaxation time measured from a 50-Hz isometric tetanus was significantly prolonged by ∼33% immediately after task failure but recovered by 1.5 min. A decline in the 10- to 50-Hz ratio of the evoked isometric contractions was observed at 5 and 10 min of recovery, which suggests excitation-contraction coupling impairment. Changes in velocity and half relaxation time during the protocol were strongly and negatively correlated (r = −0.85). Thus mainly peripheral mechanisms were implicated in the substantial depression but relatively fast recovery of isotonic power. Furthermore, isometric muscle contractile properties were related to some, but not all, changes in isotonic function.

Assess dynamic function (e.g., Refs. 18, 20, 23, 24), whereas isotonic contractions (in which the load is held constant but the velocity can vary) have not been extensively studied. Torque- or force-generating capacity is diminished following fatigue isokinetic and isometric contractions; however, the loss of power during isotonic contractions is related mainly to a reduction in shortening velocity (34). Furthermore, isotonic contractions are more relevant to normal voluntary activity because everyday movements are characterized by ballistic sinusoidal changes in velocity with constant loads (9) rather than movements of constant velocity (isokinetic).

From the limited number of studies on animals (3) and humans (20, 21) that have compared isokinetic dynamic function (power) and isometric force following a dynamic fatigue task, it has been shown that peak isokinetic power declines more than peak isometric maximal voluntary contraction (MVC) force. Because isometric contractions are dependent only on force-generating capacity, it was assumed that the greater power loss from isokinetic contractions was due to a decrease in shortening velocity in addition to a decline in torque- or force-generating capacity (21). To test the contribution of velocity on loss of power, isotonic contractions are required, but few studies have made these comparisons.

One study that investigated both isotonic power and isometric MVC torque reported that, after 120 low-load (25% MVC), maximal effort (i.e., as fast as possible) isotonic knee extensions, isotonic power decreased more than isometric MVC, but both power and MVC recovered by 2.5 min (33). Because the task duration (i.e., 120 contractions) was the same for all subjects, the amount of fatigue (loss of power) seemed to vary greatly among the subjects (∼20–80% power loss). This difference in the magnitude of fatigue among subjects confounded their ability to make comparisons of various potential mechanisms during recovery. In a study by Klass et al. (22), higher load (50% MVC) isotonic contractions of the triceps surae, but at a predetermined submaximal rate (i.e., set to a metronome), were performed until the ROM decreased to 50% of resting values. Despite the higher load, isometric force loss was smaller than in the first study (33) and did not recover in 5 min. Additional measures derived from isometric contractions were evaluated in the study by Klass et al. (22) to assess the contributions of central and peripheral factors to the loss of force after dynamic fatiguing contractions. They reported that central activation, determined by twitch interpolation of an isotonic contraction, was near maximal (∼98%) and was unchanged following the isotonic exercise. However, electric...
cally evoked isometric twitch properties [peak twitch torque, contraction time, half relaxation time (HRT)] were depressed immediately after task failure and did not recover within 5 min. This suggested an impairment in excitation-contraction coupling, but no measures of isotonic power or velocity were assessed because maximal effort isotonic contractions were not employed. Finally, other studies indicated that there was an association between loss of power and slowing of isometric tetanic contractile speed during dynamic fatigue in humans (21) and animals (12), and it was suggested that the strength of this association required further testing (20).

Therefore, the aim of this study was to assess both the muscle contractile properties from isometric contractions and dynamic contractile performance (power and velocity) following high-load, isotonic maximal concentric knee extensions in young men. We incorporated measures to assess the central and peripheral contributions to the expected impairments in power and isometric function. We expected that maximal effort high-load isotonic contractions would 1) result in a greater depression of isotonic power compared with isometric MVC, 2) alter excitation-contraction coupling processes to partly explain the decrease and delayed recovery of isometric force-generating capacity and isotonic power, and 3) demonstrate a relationship between the slowing in relaxation from an isometric tetanus with the reduction in isotonic shortening velocity.

METHODS

Subjects. Thirteen healthy male university students were recruited for this study. All subjects were recreationally active (did not participate in any systematic physical training) and were free of neuromuscular or musculoskeletal disorders. Subjects were instructed to refrain from caffeine consumption and physical activity for at least 24 h before testing. Informed, written consent was received from all subjects, and the local ethics committee on human research approved the study. To familiarize the subjects, two to three voluntary submaximal contractions were performed before obtaining definitive measurements. Due to technical problems, two subjects were excluded from data analysis of the percentage of motor unit activation in the knee extensors due to the possibility of electrode displacement over the quadriceps muscle. One custom-made aluminum electrode pads, one 3 cm proximal to the patella and the other 7 cm distal from the greater trochanter of the femur, were chosen to ensure stimulation of the largest motor unit activation in the knee extensors due to the possibility of electrode displacement over the quadriceps muscle. Two diagonal straps secured across the chest and a seatbelt applied over the hips minimized extraneous movements during the contractions. Proper alignment of the right knee was visually maintained by modifying the chair position, such that the knee’s axis of rotation (tibio-femoral joint) was aligned with the axis of rotation of the dynamometer’s attachment arm. The leg was secured to the attachment arm (lower edge 1 in. above lateral malleolus of the ankle) using wide Velcro straps. ROM for all subjects was maintained from 90–15° knee flexion (75° from start to end of knee extension). Complete knee extension is in anatomical reference to 0° of knee flexion.

Surface rather than nerve stimulation was used in this study, because surface stimulation is a more reliable technique to assess motor unit activation in the knee extensors due to the possibility of electrode displacement over the femoral nerve in the inguinal space during an MVC (4). Two custom-made aluminum electrode pads wrapped in a thin conductive gel-soaked cloth were bandaged transversely over the quadriceps muscle, one 3 cm proximal to the patella and the other 7 cm distal from the greater trochanter of the femur. Depending on thigh size, appropriately sized electrode pads were chosen to ensure stimulation of the largest mass of quadriceps without interference from the antagonist muscles.

Palpation and visual inspection of the hamstring muscles ensured that only the knee extensors were activated. A silver-silver chloride disk was placed over the knee and served as a protective ground electrode.

Using the Biodex System 3 computer software (revised 3.27), the dynamometer’s isometric mode was selected to test muscle power, velocity, and ROM. The isometric mode was utilized during the assessment of isometric MVC torque, % motor unit (central) activation (%MUA), HRT from an electrically evoked 50-Hz contraction, and low-frequency fatigue. To provide visual feedback to the subjects, velocity output from the Biodex was displayed in real time on an oscilloscope, whereas isometric torque was displayed on a computer monitor.

Baseline measurements. After 5 min of rest after the familiarization contractions, the testing session began in isometric mode with the subject producing three, 5-s sustained MVCs at 90° knee flexion with 2 min of rest between contractions (Fig. 1). During pilot testing, it was determined that the angle of 90° knee flexion was optimal to evaluate maximum isometric strength, because it resulted in the greatest isometric MVC torque compared with MVCs performed between 60 and 90° knee flexion. Lanza et al. (24) found a similar result using an identical apparatus and knee-extension setup. In addition to the real-time display of torque output, strong verbal encouragement was...
provided during each trial. The highest MVC value was recorded as the subject’s baseline muscle strength.

In previous studies (15, 22, 23), central activation of dynamic contractions has been quantified from isometric contractions performed before and after dynamic fatiguing protocols. In this study, peak %MUA was assessed during the MVC using the modified twitch interpolation technique (17), with a doublet (2 pulses at 100 Hz) evoked during the MVC and immediately at rest post-MVC. Although a single twitch has been shown to be sensitive for assessing %MUA at rest (1, 4), the doublet technique minimizes the low-frequency fatigue effect on the twitch usually found during high-intensity fatigue, while avoiding the problems associated with tetanic (train) stimulation of several pulses (14). To determine the stimulation intensity of the superimposed doublets used to assess motor unit activation, resting doublets were evoked using computer-controlled customized Spike 2 software several minutes after the first three MVCs were completed. Using a Digitimer stimulator (model 3072-134, Digitimer, Welwyn Garden City, Hertfordshire, UK), the intensity of the resting doublet was increased progressively in a stepwise method until a plateau in doublet torque was achieved. To assess low-frequency fatigue and HRT, 50-Hz stimulation bursts of 940-ms duration were gradually increased via a second computer-controlled Digitimer stimulator until the 50-Hz torque reached 40% MVC. This intensity of 50-Hz electrical stimulation is tolerable and is sufficient to assess the whole muscle contractile properties of the quadriceps (13). The same amount of current was used for 10- and 50-Hz stimulation (see below).

After 2 min of rest, an isometric testing sequence was performed twice with 2 min of rest between each sequence. Two isometric testing sequences were performed to obtain baseline measurements and to ensure that subjects were familiar with the sequence of voluntary and electrically evoked contractions. Definitive baseline measurements were taken from the testing sequence with the highest MVC. The isometric testing sequence consisted of a 3- to 5-s MVC with an interpolated and resting doublet followed by a low-frequency 10-Hz (1,000 ms) and a high-frequency 50-Hz train (Fig. 1).

Fatigue and recovery protocol. The isotonic contraction load during fatigue and recovery was normalized to 50% of each subject’s MVC, and 10 min of rest were provided before the start of the fatiguing protocol (Fig. 1). Both isotonic and isometric programmable protocols of the Biodex were linked to enable the assessment of static and dynamic contractile function immediately at task failure and during the recovery periods.

For the fatiguing protocol, the subject performed maximal-effort isotonic contractions at 50% MVC force at the fastest velocity he could achieve. Pilot testing indicated that ROM began to fail substantially when velocity declined below 65% of the velocity relative to baseline. Thus, to induce a similar degree of power loss among the subjects while maintaining equitable amounts of work [torque × distance (ROM)], the criterion for task failure was determined as the point at which the maximal velocity for two consecutive contractions declined to <65% of the prefatigue value. Two to three contractions were performed before the fatiguing contractions (∼15-s rest between contractions) and were used to determine maximum velocity at baseline and task failure velocity (65% velocity relative to baseline). Both velocities were marked on the oscilloscope to help motivate the subject to kick as hard and as fast as possible throughout the test. The contract-relax pace of the fatiguing protocol was 1.2 s in duration, and visual feedback for the time to initiate contraction was also provided on the oscilloscope. Strong verbal encouragement was given during the fatigue and recovery protocol. After each isotonic contraction of the fatiguing protocol, the subject was told to relax and to allow his leg to return to the starting position. Immediately after task failure from the isotonic contractions, the subject performed the isometric testing sequence (Fig. 1). In addition, at 0.5, 1.5, 3, 5, and 10 min (the recovery period) after task failure, the subject performed two consecutive isometric knee extensions at the fatiguing workload (50% MVC) with 2 s of rest between each contraction followed by the isometric testing sequence. This allowed for the assessment of both static and dynamic contractile function during the recovery phase.

Data and statistical analyses. The raw dynamic contraction data (torque, velocity, ROM) were obtained from the Biodex at a sampling frequency of 100 Hz, while raw isometric contraction data (MVC, doublet, 10 and 50 Hz) were converted from analog to digital format by a 12-bit analog-to-digital converter (CED model 1401 Plus, Science Park, Cambridge, UK). The Spike 2 computer program (Spike 2, CED) permitted real-time display of all data channels on the computer screen. After acquisition, isometric contraction data were analyzed offline with Spike 2 software.

Isometric MVCs were analyzed for peak values. Isotonic power was calculated from the maximum product of torque (N·m) and velocity (rad/s) (32). The knee flexion angle at optimal power was determined once the value for power at 50% MVC was known. Subtracting start position from end position determined ROM. %MUA was calculated as \[1 - \frac{a}{b}\] × 100%, where \(a\) is the torque of the interpolated response of the doublet at peak torque and \(b\) is the torque of the post-MVC doublet at rest. A comparison of peak torque for 10 and 50 Hz (10- to 50-Hz ratio = 10 Hz/50 Hz) was performed to assess low-frequency fatigability. HRT was calculated as the time to reach a 50% decline in 50-Hz torque after the final evoked pulse of the 50-Hz stimulation train. To examine the extent of change in velocity, power, MVC, and HRT, data for the fatigue and recovery protocol were normalized relative to baseline.

Using SPSS software (version 10), a one-factor (time) within-subjects ANOVA was used to assess the change in the obtained measures (velocity, power, MVC, ROM, angle at optimal power, 10- to 50-Hz ratio, and HRT) following the fatigue protocol and during the recovery protocol. Motor unit activation data, which was not normally distributed, was compared before and after the isotonic fatiguing task using the Mann-Whitney U-test (nonparametric). To compare the amount of fatigue and recovery between power and MVC, two-factor (measure × time), repeated-measures, within-subject ANOVAs were performed. If significance at \(P < 0.05\) was achieved, Tukey’s honestly significant difference post hoc analysis was performed. To assess the association between HRT and velocity over the course of the fatiguing and recovery protocol, the level of significance was set at \(P < 0.05\), and all data are presented as means (SD).

RESULTS

The young men in this experiment were 25.6 yr (SD 2.5) of age with an average height of 179.3 cm (SD 8.1) and a mass of 77.0 kg (SD 9.0). Baseline peak isometric MVC was 267.4 N·m (SD 42.5), whereas contractile function assessed from isometric contractions showed that the baseline value for %MUA was 95.0% (SD 3.7), 50-Hz HRT was 142.4 ms (SD 12.5), and the 10- to 50-Hz ratio was 0.41 (SD 0.05). Baseline dynamic contractile measurements revealed that isotonic power was 741.0 W (SD 106.0), and total ROM was 68.1° (SD 2.4).

Velocity declined to 62.4% (SD 3.6) of the baseline value at task failure [324.7% (SD 25.0) at baseline vs. 202.7% (SD 19.7) at task failure] and recovered by 3 min [317.2% (SD 21.2)]. The task duration was 38.0 s (SD 11.1). At the 50% MVC workload, power decreased to 57.6% (SD 3.5) of baseline by the end of the fatiguing protocol [741.0 W (SD 106.0) at baseline vs. 426.5 W (SD 60.3) at task failure]. After 30 s of rest, power output improved by 21% [78.7% (SD 6.1) of baseline] and recovered at both 5 and 10 min after task failure [106.3 (SD 6.2) and 95.7% (SD 6.7) of baseline, respectively] (Fig. 2). It is important to note at 5 min that power was
significantly greater than baseline values by 6.3% (SD 6.2). Isometric MVC decreased significantly to 74.1% (SD 5.1) [267.4 N·m (SD 42.5) at baseline vs. 198.8 N·m (SD 38.2) following task failure] and did not recover over the 10 min period [85.1% (SD 10.6) of baseline] (Fig. 2).

The incomplete recovery of isometric MVC appeared to be partially due to a small degree of central activation failure. Immediately after task failure, activation was 96.3% (SD 2.7), and after 0.5 min of recovery, activation remained high [91.8% (SD 4.8)]. These values did not differ significantly from the baseline value of 95.0% (SD 3.7). However, at 1.5–10 min of recovery, %MUA was reduced significantly relative to baseline [from 88.6% (SD 5.2) at 1.5 min to 91.3% (SD 4.0) at 10 min]. In comparing the fatigue and recovery profiles of power and isometric MVC, it was found that, immediately after task failure, power decreased more than MVC (Fig. 2). At 0.5 min of recovery, however, power and MVC were depressed equally and power recovered at 5 min, significantly earlier than MVC, which did not recover by 10 min.

ROM significantly decreased at task failure [from 68.1° (SD 2.4) at baseline to 55.6° (SD 6.2)] but recovered to 67.3° (SD 2.4) by 0.5 min. The decrease in ROM at task failure was due to a decrease in end knee-extension position [20.8° (SD 2.0) knee flexion at baseline vs. 33.8° (SD 5.5) at task failure]. The knee flexion angle at optimal power significantly changed [63.9° (SD 4.3) of knee flexion at baseline vs. 55.8° (SD 5.5) after task failure] but recovered after 0.5 min of recovery [59.5° (SD 3.7) knee flexion].

HRT of the 50-Hz isometric tetanus immediately after task failure was significantly prolonged by 33.3% (SD 12.6) compared with baseline [142.4 ms (SD 12.5) at baseline vs. 189.5 ms (SD 21.4) at task failure] and the slowing of relaxation continued through to 0.5 min recovery [114.0% (SD 8.0) of baseline] but recovered by 1.5 min [107.7% (SD 10.4) of baseline] (Fig. 3A). The 10- to 50-Hz ratio was reduced at 5 and 10 min of recovery [0.41 (SD 0.05) at baseline vs. 0.38 (SD 0.04) at 5 min and 0.33 (SD 0.06) at 10 min, respectively], which was due to the greater loss in torque at the lower (10 Hz) compared with the higher frequency (50 Hz) electrically evoked contractions. Lastly, there was a strong negative correlation between velocity and HRT over the course of the fatiguing and recovery protocol (r = −0.85, P < 0.05) (Fig. 3B).

DISCUSSION

As expected, reductions in peak knee extension velocity resulted in an ~42% loss of isotonic power, which was greater than the ~26% MVC force loss after isotonic fatiguing contractions. One of the novel findings was that power recovered by 5 min [106.3% (SD 6.2) of baseline], whereas MVC did not recover even by 10 min [85.1% (SD 10.6) of baseline]. These results indicated that impairments in maximum isometric strength did not delay the recovery of isotonic power. Although isometric central activation was slightly depressed during the recovery phase, it was likely that other factors played a more substantial role to account for the incomplete recovery of MVC force. Additional measures indicated that peripheral mechanisms were responsible for the decrements in power, velocity, and isometric MVC. Thus high-load maximal isometric contractions induced differential fatigue and recovery responses between isometric MVC and isotonic power.

Task-dependent factors. Comparisons of our results with other studies are difficult because few studies have used iso-

Fig. 3. A: fatigue-induced changes in velocity and 50-Hz half relaxation time (HRT) during exercise and recovery. Normalized fatigue protocol (dotted line) is represented as pre and post, and the time course of the recovery protocol (solid line) is represented by absolute times as R0.5 to R10 (min). *Significantly different from baseline, P < 0.05. B: relationship between changes in isotonic velocity and 50-Hz HRT relative to baseline values after task failure and during the 10-min recovery period for each of the 13 subjects.
tonic contractions as a method of examining neuromuscular fatigue and recovery; however, as noted in the Introduction, isometric contractions are representative of everyday contractions. In the study (33) that is most comparable to this study, a greater loss in isometric power (58 vs. 39% in this present study), at a lower fatiguing workload (25 vs. 50% MVC), but more rapid and complete recovery of isometric power (2.5 vs. 5 min) was reported. The greater number of isometric contractions (120 vs. ~32 in the present study) likely explained the greater loss in power, but because their subjects performed the same number of contractions to task failure, they observed a decline in power that varied greatly among subjects from ~20 to 80%, whereas in this study the variability in power loss was only ~10%. Normalizing for power loss during fatigue therefore allows for the ability to make appropriate comparisons of potential mechanisms during recovery.

A change in ROM is another factor that is important when power is assessed, but it usually is not evaluated during isotonic fatigue studies (29, 33). In the present study, we observed an 18.4% decrease in ROM at task failure that corresponded to a 12.7% decrease (from 63.9 to 55.8°) in the angle at which optimal power was measured. One reason that we observed a decrease in ROM and of the angle at optimal power is likely due to the fatiguing high-intensity contractions (25), which impaired force-generating capacity during terminal knee-extension ROM. However, decreased ROM and the angle at optimal power did not completely account for the decrease in contractile speed, because isotonic power recovered much later (5 min) than ROM and the angle at optimal power (0.5 min). In the one study with relevance to these results (34), it was reported that at rest peak velocity, and therefore it is assumed the knee-extension angle at optimal power, was achieved at 60° of knee flexion compared with 63.9° in the present study. Although not quantified, that study (34) noted simply that ROM decreased after an absolute number (45 repetitions) of low-load (33.9 N·m) isotonic knee extensions in women but surprisingly without a change in the angle of optimal power (60°). Differences between these studies are likely due to dissimilar fatiguing tasks in both the load and task failure criteria.

Central factors of fatigue. In this study, central activation failure appeared to play a secondary role in the impairment and recovery of isometric MVC torque. Despite a marginal decrease (~5%) in motor unit activation from 1.5 to 10 min of the recovery period, central activation for the isometric MVC was maintained at a high level before, immediately after, and at 0.5 min after task failure (~95 vs. 96 vs. 92%). Similar to the findings of other studies, central activation assessed from isometric contractions was found to be near maximal (≥98%) both before and after fatiguing dynamic isometric contractions of the triceps surae muscle (22) and isokinetic contractions of the quadriceps (23).

Although the use of static contractions is not ideal to detect changes in central activation from dynamic task failure, it seems to provide reasonable evidence of voluntary activation. In support of this, the superimposed interpolated twitch technique was used to assess central activation during fast dynamic contractions (concentric elbow flexion up to 300°/s), and it was found that the high level of voluntary activation during the nonfatigued maximal dynamic contractions was not different from the central activation measures obtained from the maximal isometric contractions (15). However, the limitation of not assessing activation during dynamic contractions must be recognized, and allows alternative explanations to be considered. For example, because the isometric contractions in this study were performed at a submaximal load, albeit at maximal velocity efforts, the task could be performed without activating all motor units. This suggests that some motor units during fatigue were not activated or had time to recover, which could affect the faster recovery of power compared with isometric MVC, when presumably all motor units are activated. Without an assessment of activation during the dynamic contractions, this possibility cannot be excluded, although alterations in the muscle properties per se suggest central activation is unlikely to be the only mechanism responsible for the differences. For example, in one study in which the quadriceps muscle was fatigued electrically with isotonic intermittent contractions, power declined, and the loss in power was greatest for higher contraction loads compared with lower loads (25). These results support peripheral impairments as the primary factor involved in isotonic task failure and also that isotonic power production is sensitive to the contraction load.

Peripheral factors of fatigue. Previous studies suggested an association between loss of power and slowing of isotonic tetanic contractile speed during dynamic fatigue in humans (21) and animals (12), but this association has not been explored extensively (20). In this study, we chose a high workload to induce substantial contractile slowing (25, 36) and compared the time course of isometric contractile slowing to changes in isotonic velocity following task failure and during recovery. Isotonic contractions allow for the assessment of velocity, and this study’s criterion for task failure was a 35% reduction in velocity. At task failure, 50-Hz HRT was prolonged by 33% from baseline values but recovered rapidly by 1.5 min. Thus, after fatigue and during recovery, we found a strong correlation between changes in isotonic velocity and the HRT (r = −0.85). These findings of a quick recovery of 50-Hz HRT and a strong association between HRT and velocity suggest that the slowing in relaxation is influenced by metabolic factors, which consequently reduces isotonic shortening velocity. In support of this, Vollestad and colleagues (36) suggested that the fatigue-induced slowing and relatively rapid 3-min recovery of 50-Hz HRT of the quadriceps observed only in the highest intensity fatigue task (60% vs. 30 and 45% MVC) was due to the greater accumulation of anaerobic metabolites associated with the highest load. Contractile slowing is a characteristic of muscle fatigue induced by high-intensity contractions in which metabolic factors such as ADP and H+ may be largely responsible for impairments in the slowing of the actin-myosin interaction (5, 8, 11, 27, 37). Furthermore, in a study performed on the fatigued rat diaphragm muscle, impaired maximum shortening velocity was closely related to the rate of cross-bridge cycling and, in particular, the rate of cross-bridge dissociation (10). Therefore, isotonic shortening velocity is likely also strongly influenced by the accumulation of anaerobic metabolites that impair cross-bridge dissociation.

Peak sustained isometric force production is based on the force-generating capacity of the muscle, which is dependent on the number of active cross bridges and force per cross bridge, but not the kinetics of the cross bridge (3, 10). Therefore, the main factors impairing the ability to sustain isometric force are...
likely different from those of isotonic contractions, because isotonic power is velocity dependent. In this study, a loss in force-generating capacity was apparent due to the incomplete acute recovery of peak isometric MVC. Others who have examined isometric strength after dynamic fatiguing tasks (18, 22, 26) have found similar results of an incomplete recovery of isometric strength. The impairment in maximum isometric force production has been known to persist for several days (16), which is much longer than the 10-min recovery phase in this study. This failure in maximum isometric force production has been related to impairments in excitation-contraction coupling (2, 6, 22, 38). It cannot be ruled out, however, that the depressed MVC may have been due to some muscle damage induced by the high-load, isotonic concentric contractions. It has been observed that, after performing high-load (80% of 1-repetition maximum) slow-velocity concentric elbow flexions (8 sets × 8 repetitions), extreme myofibrillar disruption in the biceps brachii was significantly correlated to the amount of decrease in isometric MVC strength ($r^2 = 0.75$) at 48 h postexercise (16).

A close relationship was observed between the depressions in Ca$^{2+}$ release with the extent of low-frequency fatigue examined after fatiguing dynamic contractions of the quadriceps (18). After task failure in our study, low-frequency fatigue was identified as a 19.4% decrease in the 10- to 50-Hz ratio at 10 min. Evidence from others indicated that low-frequency fatigue, denoted by an ~16% decrease in the 20- to 50-Hz ratio, was observed in the quadriceps muscle for as long as 3.5 h after 7 min of dynamic fatiguing isokinetic knee extensions (18). The significance of low-frequency fatigue and impairments in excitation-contraction coupling on isotonic contractions is not clear, because we found that power recoveries observed and significantly exceeded the baseline values by ~6% at 5 min. This was at the same recovery time point in which we observed a 9% decrease in the 10- to 50-Hz ratio. Therefore, excitation-contraction coupling impairments have been observed after dynamic contractions, but their contribution to the fatigue and recovery of isotonic power needs further investigation.

Fatigue and potentiation are known to coexist in the muscle, and the effects of fatigue can be masked by potentiation due to prior exercise (30, 31). It is known that postactivation potentiation (PAP) does not increase MVC (35) or maximum shortening velocity (7, 28), but its effect is greatest at submaximal loads (19, 31). For this reason, PAP is a possible explanation for the quick recovery of power but the continued depression of MVC torque. Klass and colleagues (22) found that, immediately after task failure induced by isotonic contractions, there was an 11% decrease in PAP that returned to baseline values at 5 min of recovery. Even though potentiation was not directly examined in this study, we observed an ~6% increase in power at 5 min of recovery, suggesting that it is possible that PAP improved the recovery of knee extension power.

In summary, although MVC torque declined less than isotonic power, MVC did not recover after 10 min, whereas power recovered fully within 5 min. With the limitations of assessing motor unit activation, the observed small loss of activation likely explains some of the depressed MVC force during recovery. Low-frequency fatigue was observed late in the 10-min recovery period, which provides evidence for the prevalence of an excitation-contraction coupling impairment after isotonic contractions. In addition, the strong negative correlation ($r = -0.85$) between velocity and HRT suggests that peripheral mechanisms, such as metabolic factors, may be responsible for the substantial depression but relatively fast recovery of isotonic power. It appeared that muscle contractile properties measured from isometric contractions were useful in elucidating some but not all changes in isotonic contractile performance and that the mechanisms of fatigue are very task specific.

GRANTS

This research was funded by the Natural Sciences and Engineering Research Council of Canada.

REFERENCES


