4. CAFARELLI, E., AND B. BIGLAND-RITCHIE. Sensation of static force in muscles of different length. *Exper. Neurol.* 65:511-525. 1972.
5. FELTS, W.M., S. CROUSE, AND M. BRUNETZ. Influence of aerobic fitness on ratings of perceived exertion during light to moderate exercise. *Percept. Mot. Skills* 67:671-676. 1988.
6. GARBUTT, G., M.G. BOOCOCK, T. REILLY, AND J.D.G. TROUP. Physiological and spinal responses to circuit weight training. *Ergonomics* 37:117-125. 1994.
7. GARCIN, M., J.-F. VAUTIER, H. VANDEWALLE, AND H. MONOD. Ratings of perceived exertion as an index of aerobic endurance during local and general exercises. *Ergonomics* 41:1105-1114. 1998.
8. GEARHART, R.F., F.L. GOSS, K.M. LAGALLY, J.M. JAKICIC, J. GALLAGHER, AND R.J. ROBERTSON. Standardized scaling procedures for rating perceived exertion during resistance exercise. *J. Strength Cond. Res.* 15:320-325. 2001.
9. HASSON, S.M., J.F. SIGNORILE, AND J.H. WILLIAMS. Fatigue-induced changes in myoelectric signal characteristics and perceived exertion. *Can. J. Sport Sci.* 14:99-102. 1989.
10. KRAEMER, W.J., B.J. NOBLE, M.J. CLARK, AND B.W. CULVER. Physiologic responses to heavy-resistance exercise with very short rest periods. *Int. J. Sports Med.* 8:247-252. 1987.
11. LAGALLY, K.M., R.J. ROBERTSON, K.I. GALLAGHER, R. GEARHART, AND F.L. GOSS. Ratings of perceived exertion during low- and high-intensity resistance exercise by young adults. *Percept. Mot. Skills* 94:723-731. 2002.
12. LAGALLY, K.M., R.J. ROBERTSON, K.I. GALLAGHER, F.L. GOSS, J.M. JAKICIC, S.M. LEPHART, S.T. MCCAW, AND B. GOODPASTER. Perceived exertion, electromyography, and blood lactate during acute bouts of resistance exercise. *Med. Sci. Sports Exerc.* 34:552-559. 2002.
13. MAYHEW, J.L., J.L. PRINSTER, J.S. WARE, D.L. ZIMMER, J.C. ARABAS, AND M.G. BEMBEN. Muscular endurance repetitions to predict bench press strength in men of different training levels. *J. Sports Med. Phys. Fitness* 35:108-113. 1995.
14. MCCLOSKEY, D.E., S. GANDEVIA, E.K. PORTER, AND J.G. COLBATCH. Muscle sense and effort: Motor commands and judgments about muscular contractions. *Adv. Neurol.* 39:151-167. 1983.
15. NOBLE, B.J., AND R.J. ROBERTSON. *Perceived Exertion*. Champaign, IL: Human Kinetics, 1996.
16. PIERCE, K., R. ROZENEK, AND M.H. STONE. Effects of high volume weight training on lactate, heart rate, and perceived exertion. *J. Strength Cond. Res.* 7:211-215. 1993.
17. PINCIVERO, D.M., AND W.S. GEAR. Quadriceps activation and perceived exertion during a high intensity, steady state contraction to failure. *Muscle Nerve* 23:514-520. 2000.
18. PINCIVERO, D.M., S.M. LEPHART, N.M. MOYNA, R.G. KARUNARA, AND R.J. ROBERTSON. Neuromuscular activation and RPE in the quadriceps at low and high isometric intensities. *Electromyogr. Clin. Neurophysiol.* 39:43-48. 1999.
19. ROBERTSON, R.J. Exercise testing and prescription using RPE as a criterion variable. *Int. J. Sport Psychol.* 32:177-188. 2001.
20. SUMINSKI, R.R., R.J. ROBERTSON, S. ARSLANIAN, J. KANG, A.C. UTTER, S.G. DASILVA, F.L. GOSS, AND K.F. METZ. Perception of effort during resistance exercise. *J. Strength Cond. Res.* 11:261-265. 1997.
21. WINTERS, D.A. *Biomechanics and Motor Control of Human Movement*. (2nd ed.). New York: Wiley, 1990.

Address correspondence to Kristen M. Lagally, kmlagal@ilstu.edu.

CONFLICTING EFFECTS OF FATIGUE AND POTENTIATION ON VOLUNTARY FORCE

DAVID G. BEHM,¹ DUANE C. BUTTON,¹ GLEN BARBOUR,¹ JEREMY C. BUTT,¹ AND WARREN B. YOUNG²

¹School of Human Kinetics and Recreation, Memorial University of Newfoundland, St. John's, Newfoundland, Canada; ²School of Human Movement and Sport Sciences, University of Ballarat, Ballarat, Victoria, Australia.

ABSTRACT. Behm, D.G., D.C. Button, G. Barbour, J.C. Butt, and W.B. Young. Conflicting effects of fatigue and potentiation on voluntary force. *J. Strength Cond. Res.* 18(2):365-372. 2004.—The objective of this study was to investigate whether a warm-up consisting of a series of maximal contractions would augment the force and activation of subsequent leg extensor contractions. Both voluntary and evoked isometric contractions were tested to determine the mechanisms underlying the response. Nine subjects were tested for twitch, tetanic, submaximal (30%), and maximal voluntary contractile (MVC) properties before and after (1, 5, 10, and 15 minutes) one to three 10-second MVCs. MVC force either did not change following 1-2 MVCs or was depressed at 10 and 15 minutes after 3 MVCs. MVC activation was decreased (4.4-6.9%) throughout recovery, whereas submaximal contractions were minimally affected. Although overall, twitches were potentiated (15.5-19.8%) posttest, 3 MVCs had significantly greater twitch potentiation than 1 or 2 MVCs at 5 and 10 minutes. Results suggest that voluntary and evoked contractions respond differently to prior 10-second MVCs. In the present study, a warm-up routine of 1-3 MVCs of a 10-second duration did not enhance subsequent voluntary performance.

KEY WORDS. quadriceps, interpolated twitch technique, electromyography, rate of force development, twitch, tetanus, maximum voluntary contractions

INTRODUCTION

The implementation of high-intensity contractions as a component of the warm-up prior to an event has been suggested to improve performance. Although increases in voluntary force or performance have been documented following prior contractions, the results have been conflicting. Gullich and Schmidbleicher (18) reported increased performance with isometric, concentric, and stretch-shortening cycle contractions following 3-5 repetitions of 5-second maximal voluntary contractions (MVCs). Some studies have shown improvements in long jumps (38) and jump squats (50) following 4 sets of 4 snatches and 5 repetition maximum (5RM) squats, respectively. However, other studies have not illustrated significant improvements in dynamic contractions following three 5-second MVCs (49) or dynamic resistance exercise (10, 43). Ebben et al. (11) had subjects perform a prior set of heavy bench press and reported no change in electromyographic activity or ground reaction forces with a medicine ball power drop. Perhaps the duration or volume of the contractions was not sufficient to stimulate improvements in voluntary performance in all individuals. For example, Vandervoort et al. (48) investigated the potentiating effects of single contractions and reported that a 10-second MVC provided a greater potentiating effect than a 5-second MVC on subsequent evoked twitches. Whereas the muscle component

may become more efficient with a prior 10-second MVC, will 10-second MVCs improve voluntary performance? It has been reported that the potentiation effect of prior contractions can increase neuromuscular activity (18). Since potentiation also involves an increase in the rate constant of crossbridge attachment (34), might there be an enhancement of dynamic power or explosive strength? Furthermore, since a single 10-second MVC seems to provide immediate twitch potentiation effects with minimal fatigue (48) and a series of three to five 5-second MVCs has been shown to improve performance (18), it is important to know whether multiple repetitions of 10-second MVCs would provide an even greater improvement in evoked twitch force and muscle activation, thus contributing to an augmentation of voluntary force. There have been no studies, to our knowledge, that document possible changes in both voluntary and evoked contractions following a series of prior voluntary contractions.

Prior voluntary contractions have been reported to increase the force of subsequent submaximal evoked or voluntary contractions (18, 40, 45). If force is augmented with submaximal contractions, would the maintenance of a prescribed submaximal force involve less stress on the neuromuscular system, as evidenced by decreased muscle activation? Prolonged contractions can lead to fatigue. Since fatigue can be defined as "an increase in the perceived effort necessary to exert a desired force" (12), no change in the maintenance of a submaximal voluntary performance can still involve fatigue processes. Decreased neuromuscular activation due to the improved efficiency of the muscle might counteract some of the fatigue effects, resulting in improved athletic performances that involve prolonged submaximal forces. The effect of prior contractions on the extent of neuromuscular activation of submaximal voluntary contractions has not been thoroughly researched in the literature.

Therefore, the goal of this study was to examine the effects of one, two, and three 10-second MVCs on (a) twitch and tetanic potentiation, (b) force and activation of an MVC, and (c) activation of a submaximal (30%) voluntary contraction at 1, 5, 10, and 15 minutes of recovery. On the basis of previous findings of improved voluntary measures following prior contractions (18, 40) and evoked twitch potentiation (19, 22, 23), it was hypothesized that a greater number of 10-second MVCs would (a) increase twitch and tetanic force, (b) increase the force and activation of a subsequent MVC, (c) decrease the activation associated with a 30% MVC, and (d) increase the rate of force development (RFD) of evoked contractions.

FIGURE 1. Experimental design.

METHODS

Experimental Approach to the Problem

To investigate whether a warm-up consisting of maximal contractions would improve subsequent performance (maximal and submaximal voluntary contractions and evoked contractions), subjects were tested before and after (1, 5, 10, and 15 minutes of recovery) the intervention of one to three 10-second MVCs (1-minute rest periods between repetitions). To determine whether changes occurred at the muscular or neuromuscular levels, participants were subjected to evoked (twitch and tetanus) and voluntary (maximal and submaximal) isometric leg extension contractions (Figure 1). The mechanisms underlying the responses to prior contractions were illustrated using electrophysiologic measures (twitch, tetanus, and interpolated twitch technique [ITT]), which could be best accomplished with isometric contractions. Forces, temporal

characteristics, and activation (voluntary contractions) were monitored.

Subjects

Nine physically active male subjects from the Memorial University of Newfoundland student and staff population (179.6 ± 8.8 cm, 86.2 ± 14.5 kg, 24.2 ± 7.2 years) participated in the study. Because of the discomfort of tetanic stimulation, not all experimental subjects were willing to undergo the control testing. Thus, while all 9 subjects were involved in the experimental sessions (1–3 MVCs), only 6 of the 9 subjects acted as controls, performing the pre- and posttests without the intervention MVCs to determine whether the tests resulted in potentiation effects. Subjects were verbally informed of the procedures and provided written consent prior to participation. The study was approved by the Memorial University of Newfoundland Human Investigation Committee.

Instrumentation

Force Measurement. For all voluntary and evoked contractions, subjects were seated on a bench with their hips and knees flexed at 90° . The subject's dominant lower limb was inserted into a padded strap at the ankle and attached by a high-tension wire to a Wheatstone bridge configuration strain gauge (LCCA 250, Omega Engineering Inc., Don Mills, Ontario, Canada). All voluntary and evoked forces were detected by strain gauges, amplified (DA 100: analog-digital converter MP100WSW, Biopac Systems Inc., Holliston, MA), and monitored on a computer (Sona Phoenix, St. John's, Newfoundland, Canada). Data were stored on a computer at a sampling rate of 2,000 Hz. Data were recorded and analyzed with a commercially designed software program (AcqKnowledge III, Biopac).

Evoked Stimulation. Thorough skin preparation for the electrodes included removing dead epithelial cells with an abrasive paper around the designated areas and then cleansing with an isopropyl alcohol swab. Stimulating electrodes, 4–5 cm in width, were constructed from aluminum foil, coated with conduction gel (Signa Crema, Parker Laboratories, Fairfield, NJ), and immersed in a saline solution. Surface-stimulating electrodes were secured over the proximal portions of the vastus medialis, vastus lateralis, and rectus femoris as well as over the distal portion of the quadriceps (vastus medialis and vastus lateralis). Superficial stimulation of the femoral nerve was not achieved, since previous experience had resulted in the electrodes being displaced from the appropriate position in the inguinal space by the quadriceps' contractions.

Peak twitch torques were evoked with electrodes connected to a high-voltage stimulator (Stimulator Model DS7H+, Digitimer, Welwyn Garden City, Hertfordshire, UK). Both the amperage (10 mA–1A) and voltage (100–150 V square-wave pulse) of a 50-microsecond duration stimulation were progressively increased until a maximum twitch torque was achieved. Measurements included peak twitch force, time to peak twitch contraction (CT_{tw}), and absolute and relative twitch RFD (RFD_{tw}). Absolute RFD_{tw} was calculated by dividing the peak twitch force by the duration to achieve peak force. Relative RFD_{tw} was calculated by normalizing the peak twitch forces during recovery to the pretest value and dividing

the normalized value by the duration to achieve peak force.

Tetanic stimulation involved 100-Hz stimulation for 400 milliseconds using the same amperage and pulse duration as the maximum twitch with a maximum of 100 V. A single repetition per test (pretest and recovery tests), with voltages not exceeding 100 V, was used because of the pain tolerance of the subjects. Thus, in some subjects, tetanic forces may not have been maximal. Mean tetanic forces were 68.2% of MVC forces (MVC: 786.6 ± 19.4 N; tetanus: 536.7 ± 20.6 N). Measurements included peak tetanic force, time to peak tetanic contraction (CT_{tet}), and tetanic RFD (RFD_{tet}). RFD_{tet} was calculated by dividing the force achieved when the rise in force reached the first distinguishable peak or plateau by the duration.

Experimental Procedure

Pretest. Subjects prepared for the testing and intervention by cycling on a cycle ergometer for 5 minutes at 1 kp at a self-selected pace. Evoked twitches were elicited at the beginning of the pretest. Tetanic, maximal, and submaximal (30%) voluntary contractions were randomly allocated following the initial twitch. The voluntary and evoked contractions involved with testing could possibly potentiate the posttest twitch. Thus, a second pretest twitch was elicited after the voluntary and tetanic contractions and immediately before the intervention MVCs. Whereas there were significant differences between the first and second pretest twitch forces, all posttest twitch comparisons were made with the second twitch to nullify the pretest potentiation effect.

Prior to attempting MVCs, subjects would perform approximately 3–5 submaximal isometric leg extension contractions. During the pretest, subjects were requested to perform at least 2 isometric ($90 \pm 1^\circ$) MVCs to determine their maximum isometric force output. To ensure a consistent maximal effort, the subjects proceeded with the ITT if there was less than a 5% difference between the 2 MVCs (2).

The ITT, which has been extensively described previously (3, 7, 8), was performed with 2 evoked doublets (at 100 Hz using maximal twitch stimulation parameters) superimposed at 1.5-second intervals on a 3-second duration MVC to estimate an average superimposed signal (Figure 2). Doublets rather than single stimuli were used to increase the signal-to-noise ratio. Furthermore, a potentiated doublet was recorded 1.5 seconds after the voluntary contractions. An interpolated doublet (IT) ratio was calculated, comparing the amplitudes of the superimposed stimulation with the postcontraction stimulation to estimate the extent of inactivation (percentage of muscle fibers not voluntarily activated) during a voluntary contraction (IT amplitude/potentiated doublet amplitude $\times 100$ = percentage of muscle inactivation) (7). Although polynomial and exponential equations derived from multiple contractions have been reported to provide the most accurate estimation of muscle activation, the use of single contractions with the ITT have still been shown to be a reliable and valid method for demonstrating changes in muscle activation (7).

The smallest recorded superimposed evoked force was 1.5% of the voluntary force. The voluntary contractions of 1-second duration included a MVC and a submaximal contraction at 30% of MVC (Figure 3). The 30% MVC would illustrate whether prior 10-second contractions

FIGURE 2. The interpolated twitch technique (ITT) was used with a maximal voluntary contraction (MVC) to estimate the extent of inactivation during a voluntary contraction by comparing the amplitudes of evoked doublets superimposed on MVC with the postcontraction potentiated doublet (interpolated doublet amplitude/potentiated doublet amplitude $\times 100$ = percentage of muscle inactivation).

would improve the performance of activities using prolonged submaximal contractions. Grid lines were provided on the computer monitor at 30% MVC to guide the force output of the subjects. Rest periods of 3 minutes were provided between all maximal and submaximal contractions during the pretest. A 5-minute rest period was allowed between the pretest and the intervention MVCs.

Intervention MVCs

After the pretest, experimental sessions involved either 1, 2, or 3 leg extensor MVCs for a duration of 10 seconds on separate days. The control session did not involve any intervention MVCs. Ten-second MVCs have been reported to cause the greatest twitch potentiation (48). The number of intervention MVCs was randomized, with at least 48 hours of rest between experimental sessions. Subjects were asked to achieve and maintain maximum force but were not given specific instructions regarding the rate of force production (i.e., "push as hard and as fast as possible"); however, all subjects did increase force rapidly. When more than 1 MVC was performed in a session, rest periods of 1 minute were allowed.

Posttest

All posttest procedures were conducted 1, 5, 10, and 15 minutes following the MVCs. Only 1 MVC with IT was performed at each posttest interval to attenuate fatigue effects. Whereas the MVC and 30% MVC with the IT and tetanic contractions were randomized, the twitch was always elicited first to minimize twitch potentiation result-

FIGURE 3. The interpolated twitch technique (ITT) was used with 30% of a maximal voluntary contraction (MVC) to estimate the extent of inactivation.

ing from the testing procedure. One-minute rest periods were permitted between test contractions.

Statistical Analyses

Data were analyzed with a 2-way analysis of variance (4×5) with repeated measures to determine whether there were significant main effects or interactions for the number of MVCs (control; 1, 2, and 3 MVCs) and the testing interval (pretest; 1, 5, 10, and 15 minutes of recovery). F ratios were considered significant at $p \leq 0.05$. If significant main effects or interactions were present, a Bonferroni (Dunn) procedure was conducted. Mean $\pm$ SE are illustrated in the text and figures.

RESULTS

All recovery changes are reported in comparison to pretest values. The control group did not experience statistically significant changes in voluntary or evoked force, RFD, or voluntary activation because of the testing procedure.

Test-retest reliability of twitch, tetanic, MVC forces, and inactivation as measured by the ITT achieved reliability coefficients of $r = 0.89, 0.86, 0.80$, and 0.79 , respectively.

Whether data were collapsed over recovery periods or number of intervention MVCs, there were no significant changes in MVC force during the recovery periods or between the number of intervention MVCs. However, with 3 MVCs, there were significant ($p = 0.03$) decreases in MVC force of 8.9 and 7.5% at 10 and 15 minutes of recovery (Figure 4).

There were no significant main effects detected for te-

FIGURE 4. Lines represent differences in maximal leg extensor force (newtons) during pretest and recovery from the intervention of 1 (diamonds), 2 (squares), and 3 (triangles) maximal voluntary contractions (MVCs). An inverted "v" represents a significant difference from the pretest with the intervention of 3 MVCs. Vertical bars represent SEs.

tanic force with the intervention of 1 (536.7 ± 20.6 N), 2 (545.5 ± 13.3 N), or 3 (508.2 ± 17.9 N) MVCs during the recovery period. Tetanic force demonstrated a significant ($p = 0.04$) potentiation of 3.6 and 3.7% at 5 and 10 minutes of recovery following 1 MVC.

The voluntary and evoked contractions involved with pretesting had a significant ($p = 0.002$) potentiation effect on the second pretest twitch (first to second twitch duration was approximately 5 minutes and was evoked prior to the intervention MVCs). The second pretest twitch (133.6 ± 7.1 N) exceeded the first twitch (121.5 ± 6.6 N) by 9.1%. Since twitch force did potentiate because of the pretesting procedure, all of the following percent changes are in comparison to a potentiated twitch immediately following the pretest contractions and prior to the intervention MVCs. With twitch force data collapsed over intervention MVCs, there were significant ($p < 0.0001$) increases of 15.5, 19.8, and 16.5% at 1 (156.6 ± 9.2 N), 5 (163.1 ± 10.6 N), and 10 (157.2 ± 10.8 N) minutes of recovery, respectively. Twitch forces following the intervention of 1 MVC averaged 128.2 ± 4.9 N, while following 2 and 3 MVCs, twitch forces reached an average of 143.7 ± 3.7 N and 176.6 ± 11.1 N, respectively ($p = 0.09$). With 1 MVC, twitch forces were significantly ($p < 0.0001$) potentiated by 17.5% at 5 minutes of recovery. Two MVCs resulted in a significant ($p < 0.0001$) 21.1% increase in twitch force at 1 minute of recovery. Significant ($p < 0.0001$) potentiation of twitch forces also occurred with 3 MVCs with 55.6, 54.1, and 53.1% increases at 5, 10, and 15 minutes of recovery. The intervention of 3 MVCs resulted in significantly greater potentiation than did 1 and 2 MVCs at 5, 10, and 15 minutes of recovery (Figure 5).

With IT data collapsed over intervention MVCs, there were significant ($p < 0.0001$) increases in inactivation (or decreases in voluntary muscle activation) of 6.4, 4.4, 5.3, and 6.9% at 1, 5, 10, and 15 minutes of recovery. With data collapsed over recovery periods, there were no significant differences between the number of intervention MVCs. One MVC contributed to significant ($p = 0.005$) 6.7, 7.1, and 6.5% increases in IT inactivation at 1, 10, and 15 minutes of recovery. Two MVCs had significantly ($p = 0.03$) increased IT inactivation over pretest values

FIGURE 5. Lines represent differences in twitch leg extensor forces (newtons) during pretests and recovery from the intervention of 1 (diamonds), 2 (squares), and 3 (triangles) maximal voluntary contractions (MVCs). Asterisks, crosses, and inverted "v"s represent differences from the pretest(s) with the intervention of 1, 2, and 3 MVCs, respectively. Hatched lines represent significant differences between 3 MVCs and all other groups. Vertical bars represent SEs.

of 6.4, 5.4, 6.8, and 7.8% at 1, 5, 10, and 15 minutes of recovery. An intervention of 3 MVCs resulted in significantly ($p = 0.02$) greater IT inactivation of 7.1 and 6.5% over pretest values at 1 and 5 minutes of recovery (Figure 6a).

With IT data collapsed over intervention MVCs, there was a strong tendency ($p = 0.06$) for IT inactivation to decrease (or muscle activation to increase) at 15 minutes of recovery. Inactivation at 15 minutes of recovery was 3.4% greater than the pretest. With data collapsed over recovery periods, there were no significant differences between the number of intervention MVCs. Similarly, 1 MVC demonstrated a tendency ($p = 0.06$) to decrease inactivation by 3.3% at 15 minutes of recovery. Both 2 and 3 MVCs showed significant ($p = 0.03$) decreases in inactivation (increased activation) at 15 minutes recovery of 4.7 and 5.6%, respectively (Figure 6b).

There were no significant main effects or interactions detected with tetanic CT_{tet} or RFD_{tet} with the intervention of 1, 2, or 3 MVCs. With data collapsed over intervention MVCs, the posttest CT_{tw} decreased compared to the pretest CT_{tw} by 9.1 and 6.3% ($p < 0.03$) at 1 and 10 minutes of recovery, respectively (Figure 7a). With data collapsed over recovery periods, 3 MVCs CT_{tw} was prolonged by 20.1 and 26.2% ($p < 0.0002$) when compared to 1 and 2 MVCs, respectively (Figure 7b). With twitch absolute RFD_{tw} data collapsed over intervention MVCs, there was a significant ($p < 0.0001$) increase of 16.1% at 5 minutes of recovery over the second pretest twitch.

One MVC caused a significant ($p < 0.0001$) 19.6% increase in absolute RFD_{tw} at 5 minutes of recovery over the second pretest twitch. There was a significant ($p < 0.0001$) 27.1% increase in absolute RFD_{tw} with 2 MVCs at 1 minute of recovery. The increase in absolute RFD_{tw} with 2 MVCs was significantly greater than 1 or 3 MVCs at 1 minute of recovery (Figure 8). Whereas Figure 8 illustrates substantial increases in absolute RFD_{tw} with 3 MVCs from 5 to 15 minutes of recovery, the recovery values in comparison to the second pretwitch did not reach statistical significance.

FIGURE 6. Lines represent differences in the percentage of muscle inactivation as measured by the interpolated twitch technique (ITT) with a maximal voluntary contraction (MVC) (a: upper graph) and a 30% MVC (b: lower graph) during pretest and recovery. Interventions included 1 (diamonds), 2 (squares), and 3 (triangles) MVCs. Asterisks, crosses, and inverted "v"s represent differences from the pretest with the intervention of 1, 2, and 3 MVCs, respectively. Vertical bars represent SEs.

DISCUSSION

The major finding of this study was the conflicting effects of prior MVC(s) on subsequent voluntary and evoked contractions. While a greater number of prior MVCs did result in greater twitch potentiation as stated in the hypothesis, other measures, particularly voluntary force, did not benefit from the prior 10-second contractions.

In contrast to evoked twitch potentiation, there was no evidence of performance enhancement with a warm-up of 10-second MVCs. MVC force was significantly decreased at 10 and 15 minutes of recovery with 3 MVCs. Furthermore, with data collapsed over intervention MVCs, there were significant decreases in MVC activation (increases in IT inactivation) throughout the recovery period. Unlike the findings of Gullich and Schmidbleicher (18) of enhanced performance following 5-second MVCs, 10-second MVCs did not enhance (1–2 MVCs) or reduce (3 MVCs) voluntary performance. Similarly, other studies have not illustrated significant improvements in voluntary performance following three 5-second MVCs (49) or dynamic resistance exercise (10, 43). A 1-minute recovery period between the 10-second contractions may have been too brief to fully restore creatine phosphate, as nearly full restoration has been reported to occur at approximately 4 minutes (9, 21, 35). Perhaps longer recovery periods between the 10-second contractions would minimize fatigue effects and provide potential benefits to

FIGURE 7. Bars represent differences in time to peak twitch (milliseconds) during pretest and recovery with data collapsed over intervention maximal voluntary contractions (MVCs) (a: upper graph) and with data collapsed over testing periods (b: lower graph). Asterisks represent significant differences at $p < 0.05$. Vertical bars represent SEs.

voluntary performance. Nonetheless, three 10-second MVCs with 1-minute rest periods provided conflicting neuromuscular fatigue (no change or decreases in voluntary force and activation) and potentiation (evoked force augmentation) effects. Although a more efficient (potentiated) muscle might be expected to improve performance, the potentiation effects were offset by voluntary deficits.

Fatigue-induced decreases in muscle activation are prevalent in the literature (4–6, 24, 25, 29, 30, 36). The lack of significant inactivation differences between 1–3 MVCs concurs with Behm et al. (3), who reported no significant inactivation differences between 5, 10, and 20RM. Conversely, the finding by Gullich and Schmidtbleicher (18) of increased afferent excitability (increased H-reflex) rather than inhibition of motoneurons might be explained by the lesser fatigue associated with a 5-second rather than a 10-second MVC.

Muscle inactivation could originate from the inhibition of spinal motoneurons. Motoneuron inhibition may

FIGURE 8. Lines represent differences in twitch rate of force development (RFD) during pretests and recovery from the intervention of 1 (diamonds), 2 (squares), and 3 (triangles) maximal voluntary contractions (MVCs). Asterisks and crosses represent differences from the pretests with the intervention of 1 and 2 MVCs, respectively. Hatched lines represent significant differences between 2 MVCs and all other groups. Vertical bars represent SEs.

arise from a variety of peripheral sources. Chemical stimuli may excite chemosensitive afferents (groups III and IV), which are known to be nociceptive (14). Group III afferents have been shown to be stimulated by metabolic products such as bradykinin (31, 33), arachidonic acid and prostaglandin E_2 (32), potassium (26), and lactic acid (39, 42, 46). Even 30 seconds of total maximal contractions would be expected to accumulate extracellular metabolites such as potassium and lactic acid. Metabolites reduce the mechanical thresholds of group III and IV afferents (28). Since they have little background discharge and their presence is both widespread and dense, group III and IV afferents can have massive increases in their input to the central nervous system (13). Thus, the perception by the central nervous system of fatigue-induced metabolic disruptions can be effectively transmitted to the motoneuron. Moreover, indirect evidence indicates that Ia afferents from intrafusal stretch receptors can contribute up to 30% of the motoneuron excitation with sustained fatiguing isometric contractions (13). Yet if the contraction is sustained for more than 1–2 seconds with the development of fatigue, discharge frequency diminishes (13). The resulting disfacilitation of motoneurons, while not providing direct inhibition, may decrease motoneuron excitability. Hence, it is possible to have muscle potentiating effects and voluntary force decrements concurrently.

The greater twitch potentiation with 10-second MVCs might positively affect activities involving repeated or sustained voluntary contractions (suboptimal Ca^{++} release). It could be hypothesized that potentiation would increase performance in submaximal activity. There was no change in IT activation during the 30% MVC following 1 MVC. The intervention of 2 and 3 MVCs exhibited no change in submaximal activation until a significant increase in muscle activation was detected at 15 minutes of recovery. While potentiation may have occurred with the fast twitch fibers, metabolic-induced decrements in slow twitch fiber force during submaximal contractions may

have necessitated greater activation of already activated or higher-threshold slow twitch fibers. While the testing alone (control) or with 1 MVC did not increase activation, the interventions of 2–3 MVCs suggest a fatigue-induced increase in neuromuscular activation (decreased inactivation) at 15 minutes of recovery.

There are many studies describing the potentiation effects of voluntary (19, 22, 23, 48) (PAP) and evoked tetanic (posttetanic potentiation) (17, 27, 37) contractions on subsequent twitch force. Twitch potentiation has been attributed to regulatory light chain phosphorylation (16, 22, 23, 45), which increases the number of force producing crossbridges under conditions of suboptimal Ca^{++} activation (45). Suboptimal Ca^{++} activation may be present with lower frequency stimulation such as a twitch, unfused tetanus, or fatigue. Since potentiation also involves an increase in the rate constant of crossbridge attachment (34), the overall increase in absolute RFD_{tw} at 5 minutes of recovery (34) as well as a significantly shorter CT_{tw} at 1 and 10 minutes of recovery would be expected. In accordance with other studies, twitch potentiation was present at 1, 5, and 10 minutes of recovery. The lack of twitch potentiation with 1 and 2 MVCs at 15 minutes of recovery agrees with Houston and Grange (22), who suggested that phosphorylation does not persist for more than 10 minutes. Conversely, the greater number of repetitions associated with 3 MVCs prolonged the potentiation to 15 minutes of recovery. If phosphorylation is not extended for more than 10 minutes (22), other factors such as changes in muscle stiffness due to residual cross-bridge attachments (41) might contribute to the potentiation of multiple contractions. Changes in muscle compliance have been shown to persist for 90 minutes following 5 repetitions of 8-second contract-relax actions (47). However, a definitive explanation is not possible with the present study, since there was no direct investigation of the mechanisms underlying potentiation.

Vandervoort et al. (48) followed twitch potentiation after single MVCs lasting 1, 3, 10, 30, and 60 seconds and reported that a 10-second MVC provided the greatest potentiating effect. Others have used single MVCs lasting 5 (1, 44), 10 (19, 20, 22), and 60 seconds (15). In the present study, a greater number of prior 10-second contractions (3 MVCs) provided a significantly greater twitch potentiation than 1 or 2 MVCs. There was no significant difference in twitch potentiation between 1 or 2 MVCs. Thus, 3 MVCs with 1-minute rest periods seemed to have a significantly greater effect on twitch but not on tetanic potentiation or voluntary performance. Since PAP enhances muscle performance under conditions of suboptimal Ca^{++} concentration (45), the lack of a substantial effect with fused 100-Hz tetanic contractions might be expected.

PRACTICAL APPLICATIONS

In the present study, three 10-second MVCs with 1-minute rest periods provided significantly greater twitch potentiation than 1 or 2 prior MVCs. Concurrently, these parameters also induced a greater degree of muscle inactivation contributing to no significant change or decreases in MVC. Whereas there is some evidence for the increase in voluntary performance following prior contractions, the most appropriate type, intensity, and duration of contraction have yet to be established. Although 10-second MVCs have been documented to provide the

greatest muscle potentiation, the 1-minute rest periods in the present study may have contributed to neuromuscular fatigue effects. Further research should investigate the specificity of prior contractions (type, intensity, duration, and rest intervals) on subsequent performances.

REFERENCES

1. ALWAY, S.E., R.L. HUGHSON, H.J. GREEN, A.E. PATLA, AND J.S. FRANK. Twitch potentiation after fatiguing exercise in man. *Eur. J. Appl. Physiol.* 56:461–466. 1987.
2. AROKOSKI, J.P., T. VALTA, O. AIRAKSINEN, AND M. KANKAANPAA. Back and abdominal muscle function during stabilization exercises. *Arch. Phys. Med. Rehabil.* 82:1089–1098. 2001.
3. BEHM, D.G., G. REARDON, J. FITZGERALD, AND E. DRINKWATER. The effect of 5, 10, and 20 repetition maximum on the recovery of voluntary and evoked contractile properties. *J. Strength Cond. Res.* 16:209–218. 2002.
4. BEHM, D.G., AND D.M.M. ST-PIERRE. Fatigue characteristics following ankle fractures. *Med. Sci. Sports Exerc.* 29:1115–1123. 1997.
5. BEHM, D.G., AND D.M.M. ST-PIERRE. Effects of fatigue duration and muscle type on voluntary and evoked contractile properties. *Eur. J. Appl. Physiol.* 82:1654–1661. 1997.
6. BEHM, D.G., AND D.M.M. ST-PIERRE. Fatigue mechanisms in trained and untrained plantar flexors. *J. Strength Cond. Res.* 12:166–172. 1998.
7. BEHM, D.G., D.M.M. ST-PIERRE, AND D. PEREZ. Muscle inactivation: Assessment of interpolated twitch technique. *J. Appl. Physiol.* 81:2267–2273. 1996.
8. BELANGER, A.Y., AND A.J. MCCOMAS. Extent of motor unit activation during effort. *J. Appl. Physiol.* 51:1131–1135. 1981.
9. CASEY, A., D. CONSTANTIN-TEODOSIU, S. HOWELL, E. HULTMAN, AND P.L. GREENHAFF. Metabolic response of type I and II muscle fibers during repeated bouts of maximal exercise in humans. *Am. J. Physiol.* 271:E38–E43. 1996.
10. DUTHIE, G.M., W.B. YOUNG, AND D.A. ATKEN. The acute effects of heavy loads on jump squat performance: An evaluation of the complex and contrast methods of power development. *J. Strength Cond. Res.* 16:530–538. 2002.
11. EBBEN, W.P., R.L. JENSEN, AND D.O. BLACKARD. Electromyographic and kinetic analysis of complex training variables. *J. Strength Cond. Res.* 14:451–456. 2000.
12. ENOKA, R.M., AND D.G. STUART. Neurobiology of muscle fatigue. *J. Appl. Physiol.* 72:1631–1648. 1992.
13. GANDEVIA, S.C. Neural control in human muscle fatigue: Changes in muscle afferents, moto neurones and moto cortical drive. *Acta Physiol. Scand.* 162:275–283. 1998.
14. GARLAND, S.J., AND M.P. KAUFMAN. Role of muscle afferents in the inhibition of motoneurons during fatigue. In: *Fatigue*. New York: Plenum Press, 1995. pp. 271–278.
15. GRANGE, R.W., AND M.E. HOUSTON. Simultaneous potentiation and fatigue in quadriceps after a 60-second maximal voluntary isometric contraction. *J. Appl. Physiol.* 70:726–731. 1991.
16. GRANGE, R.W., R. VANDENBOOM, AND M.E. HOUSTON. Physiological significance of myosin phosphorylation in skeletal muscle. *Can. J. Appl. Physiol.* 18:229–242. 1993.
17. GRANGE, R.W., R. VANDENBOOM, J. XENI, AND M.E. HOUSTON. Potentiation of in vitro concentric work in mouse fast muscle. *J. Appl. Physiol.* 84:236–243. 1998.
18. GULLICH, A., AND D. SCHMIDTBLEICHER. MVC-induced short-term potentiation of explosive force. *New Stud. Athlet.* 11:67–81. 1996.
19. HAMADA, T., D.G. SALE, D. MACDOUGALL, AND M.A. TARNOPOLSKY. Postactivation potentiation, fiber type, and twitch contraction time in human knee extensor muscles. *J. Appl. Physiol.* 88:2131–2137. 2000.
20. HAMADA, T., D.G. SALE, AND J.D. MACDOUGALL. Postactivation potentiation in endurance trained male athletes. *Med. Sci. Sport Exerc.* 32:403–411. 2000.
21. HARRIS, R.C., R.H.T. EDWARDS, E. HULTMAN, L.-O. NORDESJÖ, B. NYLIND, AND K. SAHLIN. The time course of phosphorylcreatine

- tine resynthesis during recovery of the quadriceps muscle in man. *Pflügers Arch. Eur. J. Physiol.* 367:137-142. 1976.
22. HOUSTON, M.E., AND R.W. GRANGE. Myosin phosphorylation, twitch potentiation, and fatigue in human skeletal muscle. *Can. J. Physiol. Pharmacol.* 68:908-913. 1990.
 23. HOUSTON, M.E., H.J. GREEN, AND J.T. STULL. Myosin light chain phosphorylation and isometric twitch potentiation in intact human muscle. *Pflügers Arch. Eur. J. Physiol.* 403:348-352. 1985.
 24. KAWAKAMI, Y., K. AMEMIYA, H. KANEHISA, S. IKEGAWA, AND T. FUKUNAGA. Fatigue responses of human triceps surae muscles during repetitive maximal isometric contractions. *J. Appl. Physiol.* 88:1969-1975. 2000.
 25. KENT-BRAUM, J.A. Central and peripheral contributions to muscle fatigue in humans during sustained maximal effort. *Eur. J. Appl. Physiol.* 80:57-63. 1999.
 26. KNIFFKI, K.D., S. MENSE, AND R.F. SCHMIDT. Response of group IV afferent units from skeletal muscle to stretch, contraction and chemical stimuli. *Exp. Brain Res.* 31:511-522. 1978.
 27. KUMAR, S., Y. NARAYAN, AND D. GARAND. Isometric axial rotation of the trunk in the neutral posture. *Eur. J. Appl. Physiol.* 86:53-61. 2001.
 28. LORING, S.H., AND M.B. HERSHENSON. Effects of series compliance on twitches superimposed on voluntary contractions. *J. Appl. Physiol.* 73:516-521. 1992.
 29. MCKENZIE, D.K., B. BIGLAND-RITCHIE, R.B. GORMAN, AND S.C. GANDEVIA. Central and peripheral fatigue of human diaphragm and limb muscles assessed by twitch interpolation. *J. Physiol.* 454:643-656. 1992.
 30. MCKENZIE, D.K., AND S.C. GANDEVIA. Recovery from fatigue of human diaphragm and limb muscles. *Respir. Physiol.* 84:49-60. 1991.
 31. MENSE, S. Nervous outflow from skeletal muscle following chemical noxious stimulation. *J. Physiol. (Lond.)* 267:75-88. 1977.
 32. MENSE, S. Sensitization of group IV muscle receptors to bradykinin by 5-hydroxytryptamine and prostaglandin E-2. *Brain Res.* 225:95-105. 1981.
 33. MENSE, S., AND R.F. SCHMIDT. Activation of group IV afferent units from muscle by algescic agents. *Brain Res.* 72:305-310. 1974.
 34. METZGER, J.M., M. GREASER, AND R.L. MOSS. Variations in cross-bridge attachment rate and tension with phosphorylation of myosin in mammalian skinned skeletal muscle fibres. *J. Gen. Physiol.* 93:855-883. 1989.
 35. NEWHAM, D.J., AND E.B. CADY. A ³¹P study of fatigue and metabolism in human skeletal muscle with voluntary, intermittent contractions at different forces. *NMR Biomed.* 3:211-219. 1990.
 36. NEWHAM, D.J., T. MCCARTHY, AND J. TURNER. Voluntary activation of human quadriceps during and after isokinetic exercise. *J. Appl. Physiol.* 71:2122-2126. 1991.
 37. PALMER, B.M., AND R.L. MOORE. Myosin light chain phosphorylation and tension potentiation in mouse skeletal muscle. *Am. J. Physiol.* 257:C1012-C1019. 1989.
 38. RADCLIFFE, J.C., AND J.L. RADCLIFFE. Effects of different warm-up protocols on peak power output during a single response jump task. *Med. Sci. Sport Exerc.* 28:S189. 1996.
 39. ROTTO, D.M., AND M.P. KAUFMAN. Effects of metabolic products of muscular contraction on the discharge of group III and IV afferents. *J. Appl. Physiol.* 64:2306-2313. 1988.
 40. SALE, D.G. Influence of exercise and training on motor unit activation. In: *Strength and Power in Sport*. Boston, MA: Blackwell Publishers, 1987. pp. 95-151.
 41. SHORTEN, M.R. Muscle elasticity and human performance. *Med. Sport Sci.* 25:1-18. 1987.
 42. SINOWAY, L.I., J.M. HILL, J.G. PICKAR, AND M.P. KAUFMAN. Effects of contraction and lactic acid on the discharge of group III afferents in cats. *J. Neurophysiol.* 69:1053-1059. 1993.
 43. SLJIPER, H., M.L. LATASH, AND J.T. MORDKOFF. Anticipatory postural adjustments under simple and choice reaction time conditions. *Brain Res.* 924:184-197. 2002.
 44. SMALL, S.C., AND M.J. STOKES. Stimulation frequency and force potentiation in the human adductor pollicis muscle. *Eur. J. Appl. Physiol.* 65:229-233. 1992.
 45. SWEENEY, H.L., B.F. BOWMAN, AND J.T. STULL. Myosin light chain phosphorylation in vertebrate striated muscle: Regulation and function. *Am. J. Physiol. (Cell Physiol.)* 264:c1085-c1095. 1993.
 46. THIMM, F., AND K. BAUM. Response of chemosensitive nerve fibres of group III and IV to metabolic changes in rat muscles. *Pflügers Arch. Eur. J. Physiol.* 410:143-152. 2003.
 47. TOFT, E., G.T. ESPERSEN, S. KÅLUND, T. SINKJÆR, AND B.C. HORNEMANN. Passive tension of the ankle before and after stretching. *Am. J. Sports Med.* 17:489-494. 1989.
 48. VANDERVOORT, A.A., J. QUINLIN, AND A.J. MCCOMAS. Twitch potentiation after voluntary contraction. *Exp. Neurol.* 81:141-152. 1983.
 49. YOUNG, W., AND S. ELLIOT. Acute effects of static stretching, proprioceptive neuromuscular facilitation stretching, and maximum voluntary contractions on explosive force production and jumping performance. *Res. Q. Exerc. Sport* 72:273-279. 2001.
 50. YOUNG, W., A. JENNER, AND K. GRIFFITHS. Acute enhancement of power performance from heavy load squats. *J. Strength Cond. Res.* 12:82-84. 1998.

Acknowledgments

This research was supported by the Special Initiatives Opportunity Fund as well as a Research Infrastructure Grant through the Memorial University of Newfoundland.

Address correspondence to Dr. David G. Behm, dbehm@mun.ca.