The Effect of 5, 10, and 20 Repetition Maximums on the Recovery of Voluntary and Evoked Contractile Properties

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ABSTRACT
Maximal strength training has been reported to emphasize neural adaptations. The main objective of this study was to detect differences in muscle activation between 5, 10, and 20 repetition maximum (RM) sets. Fourteen subjects performed elbow flexion with 5, 10, and 20RM. Subjects were tested for maximum isometric force (maximal voluntary contraction [MVC]), twitch amplitude (peak twitch [Pt]), time to peak twitch (TPT), half relaxation time (½ RT), electromyography (EMG), and muscle activation (interpolated twitch). Subjects were tested preexercise and 30 seconds, 1, 2, and 3 minutes postexercise. There were no significant differences in MVC, muscle activation, or antagonist/agonist EMG after 5, 10, or 20RM. However, greater RM did have a greater detrimental effect on twitch properties than fewer RM. Peak twitch was significantly (p < 0.004) less (32.08%) for the 20 than for the 5RM, whereas TPT shortened (p < 0.05) by 7.3 and 11.1% with 10 and 20RM vs. 5RM, respectively. Half relaxation time at 20RM was shortened (p < 0.05) by 20.6 and 25.4% compared with that at 5 and 10RM, respectively. MVC, muscle activation, and temporal twitch properties did not recover within 3 minutes of recovery. In conclusion, whereas 5RM did not produce greater muscle inactivation, twitch contractile properties were affected to a greater degree by a higher number of RM.

Key Words: maximal strength training, neural adaptations, twitch properties


Introduction
Many resistance training programs differentiate between a training emphasis for maximal strength and muscle hypertrophy (6, 20, 30, 69). Greater volumes of training may be necessary to optimize hypertrophic adaptations (6, 60). On the other hand, maximal strength training involves fewer repetitions (<6) and higher intensity resistance (6, 20, 30). The significant strength gains with maximal strength training have been attributed to neural adaptations (6), increases in neural drive (20, 30, 69), or motoneuron excitability (25). Neural adaptations should normally occur if the nervous system has been stressed, thus forcing the system to adapt to these unaccustomed stressors.

This stress is quantifiable with evidence of fatigue-induced elbow flexor inactivation ranging from 4–14% (7, 43, 44). If maximal strength training emphasizes neural adaptations more than traditional bodybuilding methods, then the greater neural deficits should be measurable. However, there have been no studies documenting the widely accepted notion (6, 20, 25, 30, 69) that maximal strength training methods involve greater neural adaptations.

Indeed, there is research that opposes this hypothesis. Vollestad et al. (66) could not find evidence of quadriceps or adductor pollicis inactivation during their intermittent submaximal fatigue protocol. Their research corresponds to Merton’s (45) classic study, which reported no muscle inactivation after a sustained maximal fatigue protocol of the adductor pollicis. On the other hand, Behm and St-Pierre (8) reported that muscle inactivation, as measured by the interpolated twitch technique (ITT), was duration dependent. Subjects performing long duration, submaximal, isometric, intermittent contractions experienced significantly greater muscle inactivation than with a higher intensity, shorter duration fatigue protocol. However, these studies differ from maximal strength training because they either used submaximal or maximal fatigue protocols with isometric contractions, which alone or in combination may have different effects on muscle inactivation than near-maximal dy-
dynamic contractions. Therefore, the lack of direct evidence and the conflicting indirect evidence behoove an investigation of the popular perception (6, 20, 25, 30, 69) that maximal strength training methods emphasize neural adaptations.

If neural deficits are associated with dynamic resistance training, then the restoration of full activation might be important before proceeding with another set or bout of maximal or near-maximal resistance exercise. The restoration of muscle activation to preexercise levels has been reported to occur within 5–10 minutes after a prolonged submaximal, intermittent, isometric fatigue protocol (8). Rodriguez and Agre (53) indicated that neuromuscular efficiency ([torque or force]/integrated EMG activity) was restored within 3 minutes after a submaximal fatiguing workload at 40% maximal voluntary contraction (MVC) for the quadriceps. Conversely, neuromuscular efficiency has been reported to not recover within 15 minutes after 4 minutes of adductor pollicis MVCs (47). An instructional resistance training video by the Canadian Coaching Association of Canada (20) indicated that neural deficits take 2–5 times longer to recover than skeletal muscle decrements. Nevertheless, there have been no studies using the ITT to document the recovery of muscle activation after a single set of dynamic resistance exercises.

The recovery of muscle activation should directly affect the restoration of voluntary force. Typically, recovery or rest periods between sets of repetitions are between 30 seconds and 3 minutes (30, 61). Fleck and Kraemer (31) recommended 2- to 3-minute rest periods between sets to increase maximal strength. Although a review by Sahlin et al. (56) indicated that force is normally recovered within 2 minutes, there are also reports of more prolonged force recovery after fatiguing MVCs (64, 5 minutes; 47, 20 minutes) and electrical stimulation (52, >4 minutes). Zatsiorsky (69) reported that even 4–5 minutes of rest does not provide full recovery from lifting maximum training weight, and thus the individual should schedule 10- to 15-minute recovery periods between sets. Pincivero et al. (50) reported that a rest/exercise ratio of 2:1 might not be long enough to allow full recovery of isokinetic force between exercise bouts. With conflicting reports in the literature, more research is needed to investigate the recovery period of voluntary force for specific training regimens.

Finally, the force of a voluntary contraction is dependent upon both central (neural) and peripheral (muscular) factors. Duchateau and Hainaut (24) examined the effects of 60 seconds of sustained and intermittent evoked contractions (30 Hz) on the recovery of contractile properties. They found that surface action potential areas, tetanic, and twitch forces returned to control levels within 3–4 minutes of recovery. Behm and St-Pierre (8) reported that neither peak twitch forces nor muscle action potentials were recovered within 10 minutes of the cessation of a submaximal, intermittent, isometric fatigue protocol. Incomplete force recovery caused by impaired excitation-contraction (E-C) coupling has been reported to exceed 30 minutes in some studies (26, 46). Once again, the literature is divided regarding the recovery of evoked properties because of the effects of potentiation and the variety of protocols utilized. By examining both central and peripheral responses during the recovery of different sets of repetition maxima (RM), insights into the mechanisms underlying the restoration of force may be obtained.

Thus, the objectives of this paper were to: (a) determine whether muscle inactivation was more prevalent with 5, 10, or 20RM, and (b) investigate whether typical rest periods were adequate for full recovery of voluntary and evoked contractile properties after different sets of RM resistance training.

**Methods**

**Subjects**

Fourteen subjects (university students) participated in the study (Table 1). All subjects had participated in resistance training on a regular basis (minimum 3 times per week) over the past year. Training experience was documented to ensure that the testing session would not result in delayed onset muscle soreness and thus detrimentally affect the subsequent testing sessions. Research from our laboratory has demonstrated decrements in strength and muscle inactivation 24 hours after an unaccustomed resistance exercise bout, whereas evoked contractile property deficits lingered for a week (7). Subjects were verbally informed of the procedures, and they read and signed a consent form before participation. The study was approved by the School of Physical Education, Recreation, and Athletics, Memorial University of Newfoundland Ethics Committee.

**Exercise Protocol**

Subjects performed an elbow flexion task with a resistance (dumbbell) that would result in the inability to exceed the required number of repetitions (RM). The performance of a single set of 5, 10, or 20RM was randomly allocated in each testing session. The lower arm and dumbbell hung freely, with the upper arm braced against the inner thigh. The contraction speed was

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<th>Table 1. Subject characteristics.</th>
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monitored to ensure that the concentric and eccentric phases of the contraction were each performed over a 3-second duration (3:1:3 = concentric:isometric:eccentric).

**Testing**

Subjects were tested for voluntary and evoked contractile properties before and after the RM protocols at recovery intervals of 30 seconds, 1, 2, and 3 minutes. Twelve testing sessions were randomly allocated for the RM exercise sessions (5, 10, and 20) and recovery periods (30 seconds, 1, 2, and 3 minutes). Separate testing sessions were used for each recovery period because the multiple contractions utilized to measure muscle inactivation could have resulted in some fatigue, affecting later recovery measures. A minimum of 48 hours recovery was permitted between testing sessions, and each subject was tested at approximately the same time of the day.

Subjects sat in a chair, their upper arm supported in a wooden apparatus, with the shoulder extended and elbow flexed at 90°. The wrist was inserted into a padded strap attached by a high tension wire to a Wheatstone bridge configuration strain gauge (Omega Engineering Inc., LCCA 250, Don Mills, Ontario, Canada). The lower arms, upper arms, and shoulder were restricted with velcro straps, and the subject was strapped into the chair to minimize extraneous movements. All voluntary and evoked torques were detected by the strain gauge, amplified (Biopac Systems Inc. DA 100 and analog to digital converter MP100WSW, Holliston, MA), and monitored on a computer (Sona DVA 100 and analog to digital converter MP100WSW, Welwyn Garden City, Hertfordshire, UK). The amperage (10 mA–1 A) and duration (50–100 microseconds) of a 100-V rectangular pulse were progressively increased until a maximum twitch torque was achieved. Supramaximal currents were utilized to elicit the maximal response from the muscle. The average of 3 trials was used to measure peak twitch amplitude (Pt), time to peak twitch (TPT) torque, and peak twitch half relaxation time (½ RT).

The ITT was administered during an MVC. Torque signals were sent through a high gain amplifier (Biopac Systems Inc. DA100 and MP100WSW), with the superimposed force isolated and further amplified by the software computer program AcqKnowledge III (Aquasonic, Fairfield, NJ). A doublet (2 twitches delivered at a frequency of 100 Hz) rather than a single twitch was utilized for the interpolated evoked stimulation because it provided a higher signal-to-noise ratio. An interpolated twitch (IT) ratio was calculated comparing the amplitudes of the superimposed stimulation with the postcontraction stimulation to estimate the extent of inactivation during a voluntary contraction (10). Because the postcontraction stimulation represents full muscle activation, the superimposed torque using the same intensity of stimulation would activate those fibers left inactivated by the voluntary contraction. All maximal and submaximal (100, 75, 50, and 25% of MVC) forces were correlated with their respective IT ratios in order to generate a second-order polynomial equation for all subjects. Second-order polynomials using both maximal and submaximal contractions (IT ratios) have been shown to be valid and reliable, providing a more accurate estimation of muscle activation than a single IT ratio (10).

**Statistical Analyses**

Data were analyzed using a 2-way analysis of variance (ANOVA) with repeated measures ($3 \times 8$). The 2 ANOVA levels included RM (5, 10, and 20) and the comparison of pretest and recovery periods (pretest, 30 seconds; pretest, 1 minute; pretest, 2 minutes; pretest, 3 minutes). F ratios were considered significant at $p \leq 0.05$. If significant interactions were present, post hoc tests were conducted with adjustments for multiple tests using the Bonferroni Inequality to control for the level of type-I error.
Results

The major finding of this study was a lack of main effects for RM on voluntary contractile properties, contrasting with the significant RM main effects on evoked contractile properties. In other words, the effect of a 5, 10, or 20RM exercise bout did not result in significantly greater differences in muscle inactivation (ITT or IEMG), strength loss, or antagonist/agonist EMG activity. However, in general, greater RM exercise bouts did have a greater detrimental effect on twitch contractile properties than exercise bouts with a fewer number of RM. Furthermore, all voluntary and evoked measurements experienced reductions after the exercise bout.

Voluntary Contractile Properties

As previously mentioned, there were no main effects for RM on muscle inactivation (ITT or agonist IEMG), antagonist/agonist EMG activity, or MVC. However, with data collapsed over RM, MVC was depressed over the entire recovery period, ranging from a 21.4% drop at 30 seconds to a 12.3% deficit at 3 minutes of recovery (Figure 1a). Similarly, with RM data combined, muscle inactivation as measured by the ITT was significantly ($p < 0.05$) lower at 30 seconds, 2, and 3 minutes of recovery, ranging from an initial decrease of 3.2% at 30 seconds to 1.4% at 3 minutes recovery (Figure 1b). IEMG activity was depressed for 2 minutes after the exercise bout, with decreases of 20.5, 26.9, and 30.4% at 30 seconds, 1, and 2 minutes, respectively (Figure 2a). In contrast, the antagonist/agonist EMG ratio recovered after 1 minute of recovery (Figure 2b). Compared with preexercise values, relatively greater antagonist activity was experienced at 30 seconds (11.6%) and 1 minute (12.4%) of recovery ($p < 0.05$).

Evoked Contractile Properties

All evoked contractile properties experienced significant main effects for RM (Figure 3). Peak twitch force was significantly ($p = 0.004$) less when the recovery data were combined for the 20RM than the 5RM (32.1%). Although not statistically significant, Figure 3 shows that the 10RM twitch force also tends to be reduced in comparison with the 5RM. TPT showed significant ($p < 0.05$) differences between all 3 RMs, with a 7.3 and 11.1% shortening of TPT with 10 and 20RM, respectively, when compared with 5RM. Half relaxation time at 20RM was significantly shorter ($p < 0.05$) than at 5 and 10RM by 20.6 and 25.4%, respectively.

With data combined over the RMs, the temporal twitch characteristics were not restored within the 3-minute recovery period (Figure 4). TPT was shortened by 22.9% at 30 seconds and was still less by 17.3% at 3 minutes of recovery ($p < 0.0001$). Half relaxation time similarly was shortened by 42.1 and 21.7% at 30 seconds and 3 minutes of recovery, respectively ($p < 0.0001$). Peak twitch recovered to preexercise values after 1 minute of recovery (Figure 4a). Reductions in Pt were in the magnitude of 46.8 and 44.4% at 30 seconds and 1 minute of recovery, respectively ($p < 0.0001$).

Discussion

The most important findings of this study were: (a) a single set of 5RM did not produce greater muscle inactivation than 10 or 20RM, (b) twitch contractile prop-
Recovery from 5, 10, and 20 Repetition Maximums

Figure 2. Biceps brachii integrated electromyography (IEMG) activity (a: upper graph) and antagonist/agonist EMG ratio (b: lower graph) changes during the recovery period, with data collapsed over repetition maximum groups. Asterisks represent significant differences from the respective preexercise values. Vertical bars indicate ±SD.

Figure 3. Peak twitch force (a: upper graph), time to peak twitch force (b: middle graph), and half relaxation time (c: lower graph) differences between repetition maximum (RM) groups, with data collapsed over recovery periods. Asterisks represent significant differences from other RM groups. Vertical bars indicate ±SD.

Properties are affected to a greater degree by a higher number of RM, and (c) MVC, muscle activation as measured by the ITT, and temporal twitch contractile properties were not recovered within 3 minutes of recovery.

The commonly repeated suggestion that maximal strength methods produce greater neural adaptations (6, 57) or increases in neural drive (20, 30, 69) was not substantiated in this study. Muscle inactivation (ITT) after the exercise bout was detected in all sets of RM. With data collapsed over recovery periods, muscle inactivation was 4.5% (±2.3), 6.5% (±4.3), and 4.9%...
Figure 4. Peak twitch force (a: upper graph), time to peak twitch force (b: middle graph), and half relaxation time (c: lower graph) changes during the recovery period, with data collapsed over repetition maximum (RM) groups. Asterisks represent significant differences from the respective preexercise values. Vertical bars indicate ±SD.
inactivation may be related to the inadequacies of the experimental setup. Although the subjects were strapped at the forearm and shoulder and secured to the chair, it was impossible to restrict all movement. Subjects when attempting an MVC could minimally retract their scapula to slightly alter the elbow angle, resulting in a lengthening of the elbow flexors. Loring and Hershenson (42) indicated that superimposed twitches were smaller with a compliant loading device, which allowed the muscle to lengthen. Allen et al. (2) experienced similar difficulties, and reported that a lengthening of the elbow flexors during an MVC because of slight shoulder movements would allow force to increase independently of an increase in voluntary drive. Because a similar situation occurred in the present study, there could be an underestimation of the extent of muscle inactivation.

Perhaps greater muscle inactivation was present, but not in the specific muscle tested. Allen et al. (2) demonstrated that the inactivation of the brachioradialis (91.5%) was lower than that for the biceps brachii (99.1%) during an MVC. A number of authors have reported full activation of the dorsiflexors (11), quadriceps (13, 19), and elbow flexors (21). Although the prime mover may be fully activated, possible increases in neural drive may occur with the synergistic and stabilizer muscles to optimize the force output. Further research is necessary to ascertain whether fatigue-induced decreases in synergistic or stabilizer muscles are more prevalent with maximal strength training methods.

Likewise, Rutherford and Jones (54) stated that a large part of the improvement in the ability to lift weights was because of an increased coordination of other muscle groups involved in the movement, such as those used to stabilize the body. Similarly, Schmidtbleicher (58) commented that concentric-eccentric maximal contractions help to develop intermuscular coordination. Changes in intermuscular coordination or motor control could include improved contributions from synergists or stabilizers or reduced interference from antagonists. Training studies have shown both decreased (9, 17) and increased (5, 48) antagonist activity with training. The present study did not reveal significantly different antagonist activity with 5, 10, or 20RM sets. This might suggest that maximal strength training methods (5RM) do not place a greater stress on intermuscular coordination or motor control than higher volume exercise bouts (10, 20RM). However, testing conducted under isometric conditions may not parallel responses under dynamic conditions.

Twitch contractile properties were discovered to be RM-dependent, with the greatest changes occurring with the 20RM. Peak twitch force after the 20RM was 32% lower than with the 5RM. Depression of twitch torque has been demonstrated with sustained (35, 38, 44) and intermittent maximal (15) as well as sustained (51) and intermittent submaximal contractions (66). This might suggest that a portion of the fatigue-induced force loss may be attributed to decrements associated with E-C coupling.

Decreases in force production and diminished rates of force development and relaxation have been attributed to a depression in the sarcoplasmic reticulum (SR) ATPase Ca$^{2+}$ pump with exhausting exercise in rat fast twitch muscle (12). A review by Williams and Klug (68) indicated that reductions in Ca$^{2+}$ release could account for much of the tension reduction during fatigue. Possible mechanisms of fatigue-induced SR dysfunction included structural disruption of the SR, uncoupling of Ca$^{2+}$ ATPase activity with increased temperature, as well as a decrease in the number of Ca$^{2+}$ release channels (68). Impairments in Ca$^{2+}$ release channels can be twofold because of the effect of membrane or t-tubular inexcitability on voltage sensitive dihydropyridine channels, which then restrict the quantity of Ca$^{2+}$ released for Ca$^{2+}$-regulated channels (29).

Pt depression may signify impairments of E-C coupling in this study because the MVC was not as severely affected by the fatiguing sets. Whereas Pt decreased 44.4–46.8% in the first minute of recovery, MVC was only depressed by 18.1–21.4%. A greater depression of the Pt would indicate that saturation of the myofilaments with Ca$^{2+}$ by the higher frequency stimulation of an MVC restored more force. Thus, dysfunction of force production did not occur just at the level of the cross-bridges. Both McKenzie et al. (43), using intermittent MVCs, and Bigland-Ritchie et al. (14), using intermittent submaximal contractions, reported that twitch amplitudes declined more rapidly than MVC. Similar results were found by McKenzie and Gandevia (44), with intermittent (10 seconds contraction) MVCs with variable duty cycles. E-C coupling was proposed as the failure mechanism by Edwards et al. (26), who found that the decline in twitch amplitude could be overcome with high-frequency tetanic stimulation. Thus, E-C coupling impairment was more prevalent with 20RM than 5RM in the present study.

Both TPT and $\frac{1}{2}$ RT in the present study demonstrated greater decreases in duration or increased rates of force production and relaxation after a set of 20RM than with 10 or 5RM. Some researchers have reported a prolongation of TPT after fatigue in humans (24) and cats (49). Conversely, TPT has also been reported to decrease with submaximal contractions (22). McKenzie and Gandevia (44) found differing effects upon twitch contraction time, depending on the work-rest relationship. They reported prolongation of twitch contraction time with duty cycles of 20 and 50%, whereas a reduction in twitch contraction time was detected with duty cycles of 5 and 10%. They surmised that
there must be a crucial aerobic rest interval that determines prolongation or shortening of the twitch time.

Similarly, conflicting results have been found with twitch relaxation time. Prolongation of relaxation time has been reported with fatiguing MVCs (24) and electrical stimulation (39). Conversely, decreases in relaxation time have been found after intermittent fatiguing MVCs (3), submaximal voluntary contractions (22), and electrical stimulation (55). Because Ca\(^{2+}\) ATPase and the SR Ca\(^{2+}\) pumps are stimulated by increases in cytoplasmic Ca\(^{2+}\) concentration but inhibited by various metabolic products, different durations and types of fatigue could result in either a prolongation or reduction in \(\frac{1}{2}\) RT (67). Prolongation of force production and relaxation would logically be an effective mechanism to combat the effects of fatigue by increasing the time for Ca\(^{2+}\) release to optimize cross-bridge kinetics. A greater protraction rather than shortening of twitch temporal properties might be expected with 20RM as a result of a greater metabolic disturbance.

Twitch properties have been reported to be affected by H\(^+\) accumulation, increases in ADP (16) and lactate concentration (23, 28). Although these factors would be expected to be more prevalent with a 20RM rather than a 5RM set, the shortening of twitch contractile properties in this study could be attributed to changes in muscle stiffness.

Increases in muscle stiffness after fatigue can arise from changes in the viscosity of the tissues, agonist and antagonist interactions, and reflex activity (40, 59, 65). Sinkjaer et al. (59) suggested that increased stiffness after fatigue might operate as a safety factor to compensate for a reduced reflex-induced stiffness. Whereas the 20RM might have caused a greater disturbance of reflex-induced stiffness, there would also be a greater burden placed on Ca\(^{2+}\) sequestration. Residual sarcoplasmic Ca\(^{2+}\) would maintain a greater number of cross-bridges, increasing the series elasticity of active muscle tension. Thus, increases in muscle stiffness, accelerating rates of twitch tension and relaxation may have predominated over metabolic disturbances that contribute to the prolongation of twitch properties.

MVC, muscle activation (ITT), TPT, or \(\frac{1}{2}\) RT was not fully recovered within 3 minutes of recovery. Typical recovery periods of 2–3 minutes between sets of repetitions (30, 61) have been recommended to increase maximal strength (31). Because the production of force or tension on the muscle is one of the most potent signals for strength and hypertrophic adaptations (27, 34, 63), a recovery period designed to permit high force output would seem appropriate. However, in the present study, 3 minutes of recovery were not sufficient to restore all neuromuscular characteristics, adversely affecting the ability to exert maximum tension.

The 2- to 3-minute recovery period between sets may be based on a number of factors. It has been reported that a 3-minute recovery period should provide full recovery of phosphocreatine stores because initial levels recover within 60 seconds after 21 seconds of MVCs (16). Harris et al. (36) reported that phosphocreatine levels approached control levels within 2–4 minutes of recovery and were completely recovered within 20 minutes. Casey et al. (18) exhibited differential responses for type-I and -II fibers, with incomplete restoration of phosphocreatine after 4 minutes in type-II but complete recovery in type-I. Miller et al. (47) had subjects maximally abduct the thumb isometrically for 4 minutes and reported phosphocreatine stores returning to 50% of the control value after 3 minutes of recovery. Although phosphocreatine stores may or may not be nearly restored within 3 minutes, the depletion of this energy source is not the only site of fatigue. Takata et al. (62) have demonstrated that even with the restoration of phosphocreatine, force levels did not return to normal. They ascribed the mechanism of fatigue during their 2-minute tetanic contraction protocol to dysfunctions in E-C coupling.

E-C coupling disruptions may not always recover within a 3-minute recovery period. Fuglevand et al. (32) reported incomplete recovery of twitch force after 10 minutes of recovery following fatiguing protocols of 25 and 35% of MVC. A fatigue protocol at 65% MVC showed complete twitch force restoration within 10 minutes (32). Twitch forces recovered within 3–4 minutes after 60 seconds of sustained and intermittent evoked contractions (24). Another study (4) found restoration of twitch forces within 5 minutes after a fatigue protocol involving 10 minutes of intermittent 10-second MVCs with 5-second rest periods. Incomplete force recovery because of impaired E-C coupling has been reported to exceed 30 minutes in some studies (26, 46).

Twitch contractile responses to fatigue are not only affected by the fatigue protocol but also by the trained state of the individual and the type of muscle. Whereas trained subjects experienced potentiation and untrained subjects showed twitch force depression, both groups returned to prefatigue values within 1 minute after a submaximal, intermittent, isometric fatigue protocol (9). With a similar fatigue protocol, the recovery of twitch contractile properties has been reported to be muscle-specific, with potentiation of plantar flexors and depression of quadriceps (8). Thus, the recovery of twitch force is dependent on a myriad of factors. There is a paucity of studies examining the recovery of twitch properties after high intensity, short duration, dynamic fatigue. Although most other studies document longer twitch force recovery periods, the short duration of the protocol in the present study may explain the 1-minute recovery of the Pt force.

In conclusion, maximal strength training methods (5RM) did not cause significantly greater muscle inactivation than 10 or 20RM. This might be attributed
to the short duration of the exercise bouts or the lack of testing specificity (isometric testing vs. dynamic exercise). Possible RM differences may exist in the activation of nonprime movers or the coordination of muscle with dynamic contractions. A greater number of RM did have significantly greater detrimental effects on twitch contractile properties, suggesting E-C coupling dysfunction with greater volumes of work. Shortening of TPT and ½ RT were most probably related to increases in muscle stiffness. The lack of recovery of MVC, muscle activation (ITT), TPT, or ½ RT within 3 minutes postexercise suggests that longer rest periods are necessary between sets to optimize force outputs.

**Practical Applications**

The findings of the present study may force researchers to find alternative or more specific neuromuscular mechanisms underlying the increases in strength with maximal strength training methods. Decreases in muscle activation, as measured by EMG activity or ITT, with isometric contractions did not correlate with the supposition of greater neural drive deficits with high intensity, low volume training (5RM). Perhaps, dynamic neuromuscular testing will illustrate differences in muscle activation or motor control (synergistic, stabilizer, and antagonist contributions).

The present study illustrates that a 3-minute rest period between sets does not provide sufficient recovery time for muscle activation and E-C coupling decrements. If maximum force output is desired with every set, then extended rest periods may be necessary.

**References**


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