EFFECT OF MAXIMAL VOLUNTARY CONTRACTION ON THE AMPLITUDE OF THE COMPOUND MUSCLE ACTION POTENTIAL: IMPLICATIONS FOR THE INTERPOLATED TWITCH TECHNIQUE

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ABSTRACT: The compound muscle action potential (M_{MAX}) during a maximal voluntary contraction (MVC) may be measured to determine if the motor nerve has been supramaximally stimulated during the interpolated twitch technique (ITT). Ten males performed isometric knee extension MVCs. M_{MAX} for the vastus medialis was recorded during MVC and rest. To examine the effect of stimulating electrode movement, the M_{MAX} of the thenar group and antidromic sensory nerve action potentials (SNAPs) to the third digit were recorded in a separate experiment. M_{MAX} during MVC was reduced by 18% ($P < 0.0001$) and 43% ($p < 0.0001$) for the quadriceps and thenar group, respectively. The SNAP amplitude was not different between rest and MVC ($P = 0.18$). Reduction of M_{MAX} during MVC suggests that some motor axons are refractory and unable to respond to a superimposed maximal stimulus. These results have implications for the sensitivity of the interpolated twitch technique.

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Since the classic experiments by Merton,[1] the interpolated twitch technique (ITT) has been the most commonly used method to determine voluntary activation deficits in the assessment of skeletal muscle weakness or fatigue. The basic premise underlying this technique is that a supramaximal electrical stimulus delivered to the motor nerve to a muscle group during a maximal voluntary contraction (MVC) will result in additional recruitment and/or maximal rate coding of the highest threshold motor units, if central drive is inadequate. The size of the resultant interpolated twitch (IT) provides an index of voluntary activation when normalized to a maximal, potentiated control twitch.[2,3] Although conceptually simple, the validity of an ITT protocol can be compromised by a number of methodological issues including the number of stimuli used (single pulse vs. stimulus train),[4–8] slight changes in muscle length during isometric contraction,[4,9] the contribution of synergists to torque production,[4] the site of stimulation (intramuscular nerve fibers vs. peripheral nerve trunk),[6,8,10,11] stimulus intensity (supramaximal vs. submaximal),[12] the type of muscle studied,[13] and the timing of the control twitch (before vs. after MVC).[7,12] Many of these factors are subject to manipulation on the part of the experimenter, and research regarding the optimal ITT protocol is ongoing. Rarely has it been considered that the validity of this technique may be compromised by physiological factors intrinsic to the nerve and muscle under study, which are insensitive to experimental control.

If the goal of the superimposed stimulus is to maximally activate all motor units supplying a given muscle, one must assume that the motor axons are able to respond to the stimulus. If some motor axons are in a refractory state because of prior activation from voluntary contraction, then the full effect of the supramaximal stimulation on IT torque will not be realized.[14] Underestimation of the IT amplitude would result in overestimation of voluntary activation, compromising the validity of this measure as a marker of maximal activation.[15] In order to gain insight into how motor axons respond to evoked stimulation during an MVC, the amplitude of the maximal compound muscle action potential (M_{MAX}) measured with surface recording electrodes over the motor point can be used to assess the portion of the motor axon pool that responds to the superimposed stimulus.[16]

The purpose of this study was to use the information derived from measurement of the surface-recorded M_{MAX} to gain further insight into motor axon responsiveness during an ITT protocol. The knee extensors were investigated, because voluntary activation is commonly measured in these muscles due to their importance in mobility and because they often show incomplete activation even in young, healthy subjects.[13]

SUBJECTS AND METHODS

Participants. Ten men (age 25 ± 1.7, height = 1.76 ± 0.07 m, mass = 72.7 ± 6.98 kg) volunteered to participate in the study. Participants were all university students who engaged in moderate levels of physical activity, including strength training exercise and recreational sports. All were

Abbreviations: IT, interpolated twitch; ITT, interpolated twitch technique; M_{MAX}, maximal compound muscle action potential; MNS, median nerve stimulation; MVC, maximal voluntary contraction; QS, quadriceps stimulation; SNAP, sensory nerve action potential

Key words: interpolated twitch technique; quadriceps muscle weakness; voluntary activation; compound muscle action potential; sensory nerve action potential

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healthy, with no history of musculoskeletal or neuromuscular disease. Ethical approval was obtained from the University of Western Ontario Research Ethics Board, and written consent was obtained from each participant prior to study commencement. Each participant completed two separate test protocols; percutaneous muscle stimulation of the intramuscular nerve fibers of the quadriceps (QS) and median nerve stimulation of the thenar muscle group (MNS).

Torque Measurement and Subject Preparation. For the QS protocol, subjects were seated upright in a dynamometer (Biodex System 3, Shirley, New York) to measure isometric torque during knee extension MVCs in the dominant limb. The force transducer was positioned proximal to the ankle, with the bottom edge secured two fingerbreadths proximal to the medial malleolus and the lower leg immobilized with knee and hip joint angles at 90° and 85°, respectively. The center of rotation of the knee was aligned with the axis of rotation of the dynamometer lever arm. The upper body was further immobilized by securing seatbelt straps across the waist and shoulders. During MVC, subjects were instructed to cross their arms over their chest and to avoid hip flexion or plantar flexion, so that the contribution of synergist muscles to MVC torque could be attenuated. Torque was sampled at 100 Hz, A-D converted with a 12-bit converter (CED micro1401 mk II, Cambridge Electronic Design, Cambridge, UK) and displayed in real time on an online digital system using commercially available software (Spike2 v. 5, Cambridge Electronic Design).

For the MNS condition the thenar muscle group of the nondominant hand was used to measure electrophysiological responses. This muscle group was used to control for the possibility that submaximal stimulation could occur due to shifting of the stimulating electrodes during a maximal muscle contraction. The torque produced by this muscle is relatively small, so the stimulator could be easily held in place. Additionally, the median nerve possesses both efferent motor axons to the thenar group and afferent sensory axons from the lateral 3½ digits. Antidromic sensory nerve action potentials (SNAPs) were recorded to assess whether stimulus intensity had declined during contraction, as SNAP amplitude is independent of the effects of muscle contraction.[17] Since only electrophysiological data were of interest in this condition, torque was not measured. Subjects were positioned supine on a bed, and the hand and wrist were secured in a customized brace with the thumb and wrist immobilized to facilitate isometric contraction. The subjects produced a maximal thumb abduction contraction with explicit instructions to avoid recruitment of forearm and/or finger flexors.

Electrophysiological Parameters ***Median Nerve Stimulation.*** Prior to electrode placement the skin underneath the recording area was abraded and cleansed with isopropyl rubbing alcohol. Self-adhesive silver/silver chloride surface electrodes (1 × 3 cm) were applied and secured to the skin with surgical tape. Single pulses (10–100 mA, 100–200 μs) were applied to the median nerve from the isolated stimulator of a standard clinical electromyogram (EMG) system (Advantage Medical Systems, London, Ontario). A bipolar stimulator was used to stimulate the median nerve two fingerbreadths proximal to the distal wrist crease. The stimulator was held in place manually by one of the study investigators. To record M_{MAX} from the thenar eminence a monopolar electrode configuration was used, with the active electrode placed over the motor point of the thenar muscles and the reference electrode over the metacarpophalangeal joint of the thumb. To record SNAPs an active electrode was placed midway between the metacarpophalangeal and proximal interphalangeal joints of the third digit, with the reference electrode positioned 4 cm distal to it. A saline-soaked ground electrode was secured around the wrist. EMG responses were sampled at 25 kHz, differentially amplified, filtered (5 Hz to 5 kHz for motor responses, 10 Hz to 5 kHz for sensory responses), visualized, and stored for offline analysis using a standard clinical EMG device (Advantage Medical Systems).

Quadriceps Stimulation. For the QS protocol a higher intensity stimulator with a maximal current output of 1A was used (Digitimer DS7AH, Digitimer, Hertfordshire, UK) because higher current was required to depolarize the intramuscular nerve fibers of the quadriceps muscle to achieve maximal responses. If the stimulus intensity was submaximal at a pulse width of 100 μs it was increased to 200 μs. The quadriceps muscle belly was stimulated percutaneously using aluminum foil electrodes (6 cm in width) soaked in water and conducting gel. Before application of the electrodes the patient was asked to perform a submaximal isometric knee extension contraction so that the bellies of the quadriceps could be visualized and palpated to avoid erroneous electrode placement over antagonist muscle fibers. The cathode was placed with its proximal edge at a point just distal to the inguinal crease, and the anode was positioned 6 cm distal to the cathode. Surface electrodes to record M_{MAX} were positioned in a bipolar configuration (interelectrode distance 3 cm) in line with the direction

of muscle fibers over the motor point of the vastus medialis. This configuration was used because it yielded the largest negative peak amplitude of M_{MAX} and reduced the large stimulus artifact generated from QS in pilot testing. A ground electrode was positioned over the patella. The raw EMG signal was sampled at 10 kHz, differentially amplified (Digitimer D360, Digitimer), filtered (5 Hz to 5 kHz), and A-D converted with a 12-bit converter. The signal was monitored in real time and stored for offline analysis using customized software (Spike 2, CED).

Study Protocol. Each protocol consisted of a series of five isometric MVCs, separated by 3 min rest to avoid the effects of fatigue. A familiarization period preceded each condition, which included submaximal warm-up contractions and stimuli to establish proper electrode placement and to acclimatize the participants to the sensation of stimulation. Reliability of the knee extension MVC was established with two practice MVCs performed prior to the protocol. If the torque level achieved differed by more than 10%, a third MVC was performed. Stimulus intensity was increased incrementally until no further increase in the negative peak amplitude of the response was observed (i.e., M_{MAX}). The intensity was then further increased an additional 10%–15% to ensure that the stimulus was supramaximal. A pre-MVC twitch was elicited, followed 5 s later by the MVC. For the MVCs the subjects were instructed to attain their maximal torque capacity as quickly as possible and hold the contraction steadily for ≈5 s. The interpolated stimulus was triggered manually once a plateau in the torque tracing was observed. In the case of the MNS protocol where torque was not measured, the stimulus was applied ≈3 s into the contraction. For each MVC verbal encouragement was provided, and visual feedback of performance was provided for the knee extensor contractions. A resting control twitch was elicited 5 s after each MVC.

Data Analysis and Statistics. The extent of voluntary activation was calculated according to Belanger and McComas[2] as: voluntary activation = [1 − (Superimposed Twitch / Potentiated Resting Twitch)] × 100%. Torque data were processed with a digital smoothing argument (time constant = 0.01 s) to improve the signal-to-noise ratio. Offline analysis of each trial was performed to determine its acceptability. If the interpolated stimulus was applied at a point where voluntary torque level was clearly submaximal the trial was rejected. Subjects were only included in the analysis if a minimum of three acceptable trials were recorded for each condition. Study data meeting the Komolgorov–Smirnov test of normality were analyzed with parametric statistics. All descriptive data are reported as the mean ± standard deviation (SD) of 3–5 trials.

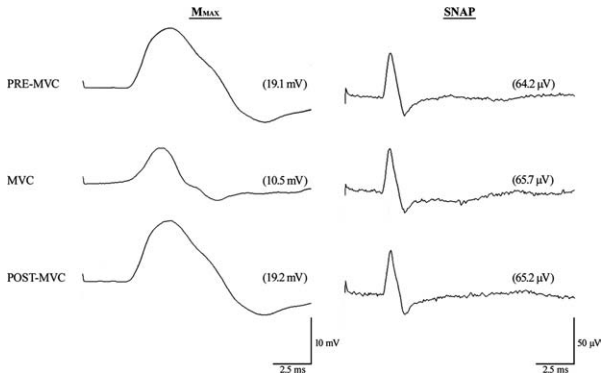

FIGURE 1. M_{MAX} of the thenar muscles (left side) and SNAPs from the third digit, recorded from a representative subject during MNS at the wrist. Note that the M_{MAX} during MVC has reduced negative-peak amplitude compared to the resting responses, but the morphology and amplitude of the SNAPs are almost identical. SNAP, sensory nerve action potential; M_{MAX}, maximal compound muscle action potential; MNS, median nerve stimulation; MVC, maximal voluntary contraction.

Differences in the negative peak amplitude of M_{MAX} and SNAPs for the MVC and rest conditions (both before and after MVC) were assessed with one-way analysis of variance (ANOVA) with repeated measures. Post-hoc testing for between-group differences was performed with Tukey's honestly significant differences test when necessary. Significance was set at $P < 0.05$. The relationship between M_{MAX} amplitude during MVC (normalized to post-MVC M_{MAX} amplitude) and IT amplitude (skewed distribution) was assessed with the Spearman rank correlation coefficient (r). All statistics were performed with GraphPad Prism v. 5.0b (La Jolla, California).

RESULTS

All 10 subjects completed the QS protocol. The data from one subject was excluded from analysis in the MNS protocol due to difficulty in stimulating the median nerve underneath the tendon of the palmaris longus muscle during maximal contractions.

Compound Muscle Action Potential Response to Maximal Voluntary Contraction. The raw EMG tracings of a representative subject are shown in Figure 1 for the MNS protocol. It can be seen that M_{MAX} amplitude was reduced during MVC compared to rest, while SNAP amplitude and morphology were similar to the resting and postcontraction conditions. The mean values for the motor and sensory nerve responses are displayed in Table 1. In both QS and MNS, significant differences were observed for the amplitude of the M_{MAX}. Upon post-hoc analysis pre-MVC M_{MAX} and post-MVC M_{MAX}

Table 1. Mean negative peak amplitude for motor and sensory nerve action potentials measured by surface EMG during the ITT protocol.

Study parameter	Pre-MVC	MVC	Post-MVC
MNS M_{MAX} (mV)	19.19 ± 4.64	13.37 ± 3.86*	20.15 ± 4.97
QS M_{MAX} (mV)	12.08 ± 2.16	9.89 ± 1.77*	12.10 ± 2.25
MNS SNAP (μV)	71.23 ± 21.33	71.89 ± 23.05	72.48 ± 24.65

**Significantly different than pre-MVC and post-MVC.*
ITT, interpolated twitch technique; MVC, maximal voluntary contraction; MNS, median nerve stimulation; QS, quadriceps stimulation; M_{MAX}, maximal compound muscle action potential; SNAP, sensory nerve action potential.

amplitudes were significantly different than MVC M_{MAX} amplitude, but they were not different from each other. In the MNS protocol no significant difference was observed for the SNAP amplitude between resting and MVC conditions ($P > 0.05$). For mean data, M_{MAX} amplitudes were reduced by 43% and 18% during MVC compared to post-MVC for the MNS and QS protocols, respectively.

Voluntary Activation of the Quadriceps. Mean quadriceps MVC for 10 subjects was 240.8 ± 66.4 N·m. Mean voluntary activation was 95.6 ± 3.7%. Only 5/10 participants were able to achieve 100% voluntary activation in at least one trial. The relationship between M_{MAX} amplitude during MVC (normalized to post-MVC M_{MAX} amplitude) and interpolated twitch amplitude is plotted in Figure 2 for a total of 46 trials. The degree of association for the relationship based on Spearman's rank correlation coefficient was $r = 0.69$ ($P < 0.0001$).

DISCUSSION

The primary finding of this study was a reduction in M_{MAX} amplitude during MVC. Conversely, the SNAP amplitude was unaltered in the MNS condition during MVC, suggesting that M_{MAX} amplitude reduction is due to physiological factors such as axon refractoriness, rather than suboptimal stimulation due to shifting of the stimulating electrodes during background contraction. Furthermore, a significant correlation was found between MVC M_{MAX} amplitude and IT torque. When IT amplitude is high (i.e., voluntary activation deficit increases), MVC M_{MAX} amplitude approaches resting M_{MAX}, suggesting that twitch amplitude and, in turn, the ability to determine the extent of voluntary activation is dependent in part on the responsiveness of the motor axons at the instant of stimulation. Therefore, based on these observations it is likely that, in many cases, the superimposed stimulus is unsuccessful in activating all of the motor axons that could contribute to IT amplitude and thus voluntary activation is overestimated.

Behm et al.[6] reported M_{MAX} amplitude reductions in the vastus lateralis during knee extension MVC compared to rest, as the shifting of stimulating electrodes made it difficult to maintain maximal stimulation intensity. In order to determine whether the reduction in M_{MAX} seen in the QS condition in our study was due to submaximal stimulation, it was necessary to perform a separate set of experiments utilizing the median nerve to collect compound muscle action potentials and antidromic SNAPs simultaneously during MVCs. In previous studies, SNAP amplitudes have been shown to be unaffected by background contraction of the hand muscles.[17] Therefore, the SNAP amplitudes could be used as a means to ensure that stimulation intensity remained supramaximal during the background MVC. There is strong evidence from the MNS condition to suggest that reduction in M_{MAX} can occur even if stimulation intensity remains supramaximal. First, there was no change in the SNAP morphology between rest and MVC in the MNS condition. If stimulus intensity was submaximal a concomitant reduction in SNAP amplitude should have been observed. Second, no change in M_{MAX} was observed between pre-MVC and post-MVC. As the stimulating electrodes were kept in place for the duration of each trial it is likely that they also remained in an optimal position during MVC. Furthermore, it has been shown that high-frequency stimulation (e.g., 30 Hz) of the median nerve in healthy subjects does not cause reduction in M_{MAX} amplitude despite the movement artifact that would occur at the site of

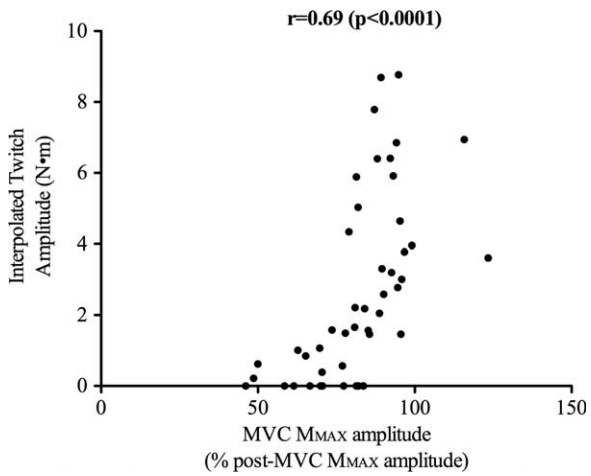

FIGURE 2. A plot of the M_{MAX} amplitude during MVC expressed as the percent of post-MVC M_{MAX} amplitude, versus the amplitude of the IT torque amplitude for 46 ITT trials in the study. This relationship reveals that when M_{MAX} is reduced during MVC (presumably due to reduced motor axon responsiveness), IT amplitude approaches zero. Conversely, when MVC M_{MAX} amplitude approaches its resting value IT amplitude increases to a varying degree. M_{MAX}, maximal compound muscle action potential; MVC, maximal voluntary contraction, IT, interpolated twitch; ITT, interpolated twitch technique.

the recording electrodes at a frequency high enough to induce tetanic muscle contraction.[19] Thus, the decrement in M_{MAX} amplitude seen in this condition was not due to shifting stimulating electrodes and is unlikely related to movement of the recording electrodes.

Contrary to our observation, others using similar paradigms have shown increases in M_{MAX} amplitude during voluntary contraction versus rest.[20–22] The mechanisms purported for increases in M_{MAX} amplitude include: (1) greater spatial summation of muscle fiber action potentials underneath the recording electrode surface due to small changes in muscle length that occur during isometric contractions[20,21]; (2) increased muscle fiber action potential conduction velocity in response to voluntary contraction[21]; and (3) greater synchronization of motor unit action potentials as a result of increased descending drive.[20,22] It is possible that methodological differences including muscle group studied, muscle length during testing, and the use of submaximal contractions in some studies are responsible for the discrepant findings.

In another study of vastus medialis M_{MAX} properties during knee extension MVC, Linnamo et al.[23] observed no change in quadriceps M_{MAX} amplitudes between rest and contraction. The authors suggested that the stimulus encountered some axons that were made absolutely refractory by the background contraction, thus negating the impact of the aforementioned potentiating mechanisms. It is possible that axon refractoriness was the mechanism responsible for the reduced M_{MAX} amplitude in our study. While it is difficult to test this hypothesis due to the inability to measure membrane potential in vivo, observations from threshold tracking studies of motor axon membrane potential provide support for this mechanism. Threshold tracking of membrane potential in axons supplying the thenar and tibialis anterior muscles have shown reduced motor axon excitability measured directly after brief, voluntary contractions.[14,25,26] Mechanisms proposed for this response include potentiation of the electrogenic sodium-potassium pump leading to membrane afterhyperpolarization and an increase in extra-axonal K^+ (presumably due to the occlusion of vasculature caused by increased intramuscular pressure during muscle contraction) causing lengthening of the relatively refractory period. It is possible that the superimposed stimulus applied during the ITT that was strong enough at rest is no longer adequate to overcome the hyperpolarization and prolonged relatively refractory period of certain axons at the instant of stimulus application during an ITT protocol.

The implication of this result is that reduced motor axon excitability may contribute to a reduction in IT amplitude. Some motor axons that may have been included in the generation of additional twitch torque may not respond to stimulation while relatively refractory. Indeed, Figure 2 illustrates the positive relationship between amplitude of the M_{MAX} during MVC and amplitude of the IT. When MVC M_{MAX} amplitude approaches the resting value it can be inferred that a greater proportion of motor axons have participated in the subsequent response. Consequently, larger IT amplitudes are observed, and apparent voluntary activation is reduced. Conversely, should an evoked stimulus occur at an instant where a large proportion of motor axons are relatively unresponsive to stimulation, M_{MAX} amplitude will be reduced, and apparent voluntary activation will approach 100%. While the correlation in Figure 2 is only moderate, other factors have been suggested to cause underestimation of twitch amplitude.[27–29] Herbert and Gandevia[28] proposed that it is difficult to determine experimentally whether the interpolated stimulus is activating dormant high-threshold motor axons while simultaneously allowing unobstructed voluntary efferent output in order to produce a twitch that is truly representative of the entire motor unit pool. An interpolated stimulus in the periphery will propagate in both the orthodromic (toward the neuromuscular junction) and antidromic (toward the spinal cord) directions, causing collisions between orthodromic voluntary output and the antidromic evoked volley, thus negating the contribution of some axons to IT production. In support of this hypothesis, this group reported a reduction in voluntary activation when the effect of collisions was removed during simulations of the ITT in a computer model of the human adductor pollicis motor unit pool (91% activation without collisions vs. 95% with collisions). Furthermore, it has been hypothesized that the interpolated stimulus may also cause recurrent inhibition via antidromic activation of the Renshaw cells and spinal reflex inhibition through muscle spindle unloading and Ib afferent stimulation, both of which may also reduce the amplitude of the IT.[27–29] These mechanisms, in conjunction with a relatively unresponsive motor axon pool, would serve to underestimate true IT amplitude and thus overestimate voluntary activation.

In conclusion, the results of our study reveal that measurement of M_{MAX} during ITT can provide useful information related to validity of measures of voluntary activation. MVC may alter motor axon excitability and render some motor axons unresponsive to supramaximal stimulation. This was manifested by a reduction in M_{MAX} during MVC versus rest. This is of significance to validity

of the ITT, as superimposed twitch amplitude may be underestimated.

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