

INFLUENCE OF STIMULUS PULSE WIDTH ON M-WAVES, H-REFLEXES, AND TORQUE DURING TETANIC LOW-INTENSITY NEUROMUSCULAR STIMULATION

OLLE LAGERQUIST, PhD, and DAVID F. COLLINS, PhD

Human Neurophysiology Laboratory, Centre for Neuroscience, University of Alberta, Edmonton, Alberta T6G 2H9, Canada

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ABSTRACT: Neuromuscular electrical stimulation (NMES) has been shown to generate contractions that include a central recruitment of motoneurons; however, the effect of pulse width on electromyographic (EMG) and torque responses during NMES are not well documented. Soleus EMG and isometric plantarflexion torque were recorded from 14 subjects with NMES delivered to the tibial nerve using 50, 200, 500, and 1000 μ s pulse widths. M-waves were significantly smaller during 20 Hz NMES compared with responses evoked by single pulses of 200, 500, and 1000 μ s, but not 50 μ s pulse widths. At all pulse widths, stimulation at 20 Hz depressed soleus H-reflexes compared with single pulses. Two seconds of 100 Hz NMES significantly increased H-reflexes and torque during the subsequent 20 Hz NMES with 200, 500, and 1000 μ s, but not 50 μ s, pulse widths. NMES delivered using wide pulses generated larger contractions with a relatively greater central contribution than narrow pulses. This may help reduce atrophy and produce fatigue-resistant contractions for rehabilitation.

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Neuromuscular electrical stimulation (NMES) is often used to enhance function and reduce muscle atrophy for people living with paralysis after a central nervous system (CNS) injury or disease. NMES for rehabilitation is typically delivered using pulse widths of 200–400 μ s at frequencies between 20 and 50 Hz.¹ This method of stimulation recruits motor units by depolarizing motor axons beneath the stimulating electrodes,^{2–4} and thus generates contractions at least in part through the summation of twitches associated with successive motor waves (M-waves). However, NMES also depolarizes sensory axons and generates an afferent volley that can recruit motor units reflexively to contribute to the evoked contraction.^{5–13} This central contribution to electrically evoked contractions has been confirmed by experiments involving an anesthetic nerve block. In those experiments, the same intensity and pattern of NMES generated significantly more torque before the nerve block when the CNS could contribute than during the nerve block, when only the activation of motor axons could contribute.^{6–8,13} These experiments were designed

to identify the influence of stimulus pulse width on the recruitment of motor axons (M-waves), the reflexive recruitment of motoneurons (H-reflexes), and isometric torque during NMES.

When NMES is delivered to generate tetanic contractions suitable for rehabilitation, both post-activation depression of neurotransmitter release from afferent terminals^{14–16} and antidromic transmission along motor axons^{16–18} (particularly at high stimulus intensities) reduce the likelihood that transmission along reflex pathways can make a significant contribution to the evoked contractions. However, H-reflexes *can* contribute to contractions during NMES^{11,19} and are augmented following a brief period of NMES at 100 Hz.¹¹ Theoretically, this H-reflex contribution should be greater when NMES is delivered using wider pulse widths, as wide pulses depolarize sensory axons more effectively than narrow pulses.^{20–22} It has previously been shown that wide pulses generate significantly more torque than narrow pulses during NMES even when stimulus intensity was adjusted to account for differences in charge.⁸ In that study, the increased torque evoked using wide pulses was attributed to a greater reflexive recruitment of motor units due to the larger afferent volley, but M-waves and H-reflexes were not recorded.⁸ We have recently shown that larger H-reflexes were evoked for a given sized M-wave with wider pulses (200–1000 μ s) compared with narrow pulses (50 μ s) when single pulses were delivered to construct H-reflex vs. M-wave recruitment curves.²¹ It has not been tested whether the same relationship exists between pulse width and H-reflex recruitment when NMES is delivered at frequencies suitable for rehabilitation. Similarly, the relationship between pulse width and M-wave amplitude during NMES has not been explored.

The order in which motor units are recruited during NMES is still unclear. Experiments have shown a reversed,^{23,24} random,^{25–28} or near-normal²⁹ recruitment order. Regardless, it is generally agreed that a non-physiological recruitment order during contractions thought to be driven primarily by M-waves accounts for the rapid fatigue associated with NMES.^{23,24} Our working hypothesis is that NMES delivered using wide pulse widths generates contractions with a greater central contribution than those evoked using narrow pulses.

Abbreviations: ANOVA, analysis of variance; CNS, central nervous system; EMG, electromyogram; H_{max} , maximal Hoffman reflex; H-reflex, Hoffmann reflex; M_{max} , maximal M-wave; M-wave, motor wave; MVC, maximum voluntary contraction; NMES, neuromuscular electrical stimulation

Key words: H-reflex, M-wave, neuromuscular electrical stimulation, pulse width, torque

Correspondence to: D.F. Collins; e-mail: dave.collins@ualberta.ca

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Because synaptic drive recruits motor units from smallest to largest according to Henneman's size principle^{30,31} we have proposed that using wide pulses, relatively high frequencies, and low intensities may be advantageous for rehabilitation, because it should maximize the central contribution to contractions evoked by NMES.⁵⁻¹³ The experiments reported herein extend our previous work on stimulus pulse width and H-reflex recruitment²¹ and investigate the recruitment of motor axons (M-waves), the reflexive recruitment of motoneurons (H-reflex), and the development of torque during tetanic NMES.

In these experiments we delivered NMES for 7 s in a pattern (2 s at 20 Hz, 3 s at 100 Hz, 3 s at 20 Hz) that we have previously used to study contractions evoked by NMES. This stimulation pattern allowed us to investigate the influence of pulse width on M-waves, H-reflexes, and torque during 20 Hz NMES (a typical frequency for NMES) as well as the effects of delivering 2 s of 100 Hz stimulation at different pulse widths. Using these stimulation patterns we have shown that 2 s of 100 Hz NMES using 1000 μ s pulse widths leads to a sustained increase in torque⁵⁻¹³ and H-reflex amplitude¹¹ that persists during the subsequent return to 20 Hz stimulation. We tested three hypotheses: (1) M-wave amplitudes during 20 Hz NMES will be depressed compared with those evoked by single pulses, but this depression will be unaffected by pulse width; (2) H-reflexes will be depressed by NMES at all pulse widths compared with H-reflexes evoked by single pulses; and (3) wider pulse widths (200, 500, and 1000 μ s) will increase H-reflex amplitude and torque after the delivery of 2 s of 100 Hz stimulation (*post-100 Hz*) compared with before (*pre-100 Hz*), whereas the narrowest pulse width (50 μ s) will not. Stimulus intensity was adjusted to evoke M-waves of similar amplitude using single pulses across pulse widths, thus recruiting a comparable proportion of motor axons. Low stimulus intensities were used so that we could record H-reflexes with minimal obstruction by block along motor axons.¹⁶⁻¹⁸ The results of these experiments provide further insight into the central and peripheral recruitment of motor units during NMES.

METHODS

Subjects. Eighteen individuals with no known neurological impairment participated after providing informed consent. Four participants withdrew from the study due to discomfort during NMES, and thus analyses were conducted on data from 14 participants, comprised of 12 males and 2 females (19-43 years of age). Data from 12 participants were collected during the same experimental ses-

sions as data that were part of a companion study.²¹ The only data common to the present study and the companion study²¹ were maximal M-wave (M_{\max}) values that were used to normalize each subject's electromyographic (EMG) data. This study was approved by the Human Research Ethics Board at the University of Alberta.

Protocol. All experimental procedures were performed on the right leg. Subjects were seated with the right hip, knee, and ankle at 90°, 110°, and 90°, respectively. Both feet were supported, and the isometric torque generated by the right plantar flexors was transduced (System 3 Dynamometer; Biodex Medical Systems, Shirley, New York). Each experimental session lasted \sim 3 h.

Electromyography. Surface EMG was recorded from the right soleus and tibialis anterior muscles with bipolar (2.25-cm²) surface electrodes (Vermed Medical, Bellows Falls, Vermont). EMG signals were pre-amplified 500-1000 \times and band-pass filtered at 10-3000 Hz (NeuroLog System; Digitimer, Welwyn Garden City, Hertfordshire, UK).

Maximal Voluntary Isometric Contractions. At the beginning of each experiment, subjects performed between three and seven maximal voluntary isometric contractions (MVCs) of the plantar flexors until three consistent maximal contractions with no more than 5% variability were achieved. The average torque produced in the 0.5-s period centered on the point of maximal torque during the largest of the three contractions was used to establish individual MVC values. This MVC value was used to normalize each subject's torque during the tetanic stimulation trials. Subjects were provided with visual feedback of their torque production and received verbal encouragement to perform maximally during each MVC.

Electrical Stimulation. The right tibial nerve was stimulated using bipolar surface (2.25-cm²) electrodes (Vermed Medical) placed over the popliteal fossa at the site that evoked a soleus response (M-wave or H-reflex) at the lowest stimulation intensity. Rectangular pulses of 50, 200, 500, and 1000 μ s were delivered from a constant-current stimulator (DS7A; Digitimer). Stimulus intensity was adjusted based on the amplitude of the M-wave evoked by single pulses and ranged between \sim 5 and 15 mA. Two stimulus intensities were used for each pulse width: (1) motor threshold (an M-wave of \sim 1-2% M_{\max}); and (2) an intensity that evoked an M-wave of 5% M_{\max} . Subjects were instructed to relax and not contribute to the evoked contractions. In each trial, a single pulse width was used in which three single pulses were delivered 5 s apart \sim 10 s before five trains of NMES were

applied. The five trains of NMES were delivered in each trial to generate a representative mean value at each pulse width and intensity for all subjects. The NMES pattern used in this study was 20 Hz for 2 s to 100 Hz for 2 s to 20 Hz for 3 s; that is, 20–100–20 Hz for 7 s (Fig. 1). The five stimulation trains, 45 s apart, were delivered using each of the four pulse widths (50, 200, 500, and 1000 μ s) and two intensities (motor threshold and 5% M_{\max}). The order of testing was randomly selected by drawing lots.

Data Analysis. The amplitudes of M-waves and H-reflexes evoked by single pulses and during periods of 20 Hz NMES were measured peak-to-peak and normalized to M_{\max} . M_{\max} was taken to be the single largest M-wave evoked by single pulses delivered at supramaximal intensities for each pulse width, as described in the companion study.²¹ Torque recorded during the 7-s NMES trains was normalized to each subject's MVC torque. The amplitudes of M-waves, H-reflexes, and torque were calculated for each NMES train and averaged over the five trains in each trial. M-wave, H-reflex and torque values were calculated during the period 1.25–1.75 s into the initial 20 Hz stimulation (*pre-100 Hz*; see Fig. 1). These M-waves and H-reflex amplitudes were compared with those obtained with single pulses to assess the influence of pulse width on EMG responses during NMES of a typical frequency. In addition, the *pre-100 Hz* values were compared with values obtained 1.25–1.75 s after the 100 Hz stimulation (*post-100 Hz*) to assess the influence of 2 s of 100 Hz NMES on torque and EMG responses. Last, *post-100 Hz* EMG responses were compared with single-pulse values. Group data were obtained by pooling mean data from each subject. EMG responses were not quantified during the 100 Hz stimulation due to interference from overlapping of M-waves, H-reflexes, and stimulus artifacts. Data were sampled at a minimum of 5 kHz using a custom-written program (LabVIEW; National Instruments, Austin, Texas) and stored on a computer for analysis.

Statistics. To assess differences in torque between data collected *pre-100 Hz* and *post-100 Hz*, we performed 2×4 repeated-measures analyses of variance (ANOVAs) with “time” having two levels (*pre-100 Hz* and *post-100 Hz*) and “pulse” having four levels (50, 200, 500, and 1000 μ s). To assess M-waves and H-reflexes obtained between single pulses, *pre-100 Hz*, and *post-100 Hz* at the four pulse widths, two separate 3×4 repeated-measures ANOVAs with an additional level of Time (single pulse) were performed. Tests for normality using Shapiro–Wilk tests showed that H-reflex data were not normally distributed; therefore, we performed

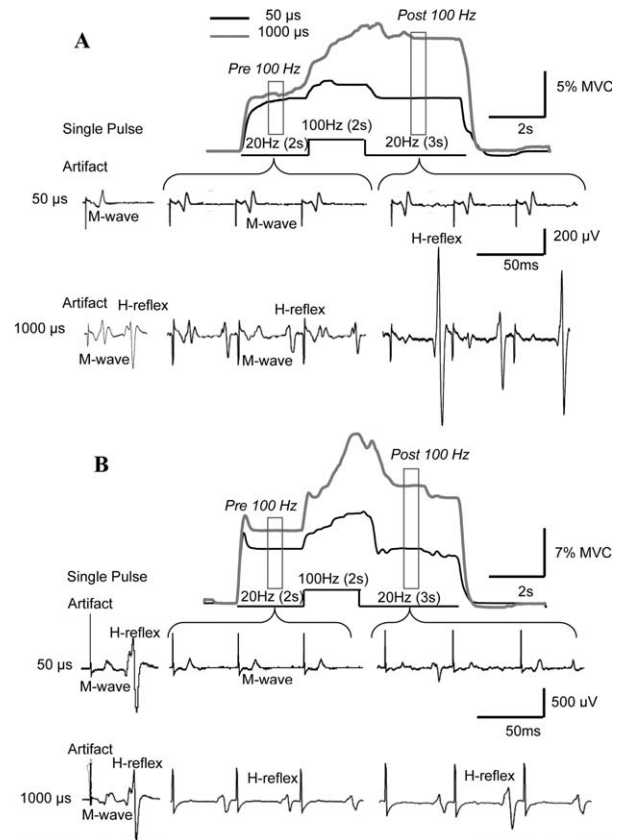


FIGURE 1. Single-subject data showing plantarflexion torque and soleus EMG responses recorded during the 20/100/20 Hz stimulus pattern using 50 μ s (black) and 1000 μ s (gray) pulse widths. **(A, B)** Data collected while stimulating at motor threshold and 5% M_{\max} , respectively. Vertical rectangles indicate the intervals over which data were quantified before (*pre-100 Hz*) and after (*post-100 Hz*) the 100 Hz stimulation. A sample of soleus EMG from the *pre-* and *post-100 Hz* intervals for each pulse width is displayed.

a \log_{10} transform on H-reflex data prior to the ANOVA of H-reflexes. We were specifically interested in Time \times Pulse interactions, and thus significant main effects are only reported when no significant interaction was present. The α level was set at $P \leq 0.05$. When appropriate, post hoc analyses were performed using Tukey's honestly significant differences test. Data are reported as mean \pm standard deviation.

RESULTS

Data recorded from a single subject during NMES delivered at motor threshold and 5% M_{\max} are shown in Figure 1A and B, respectively. For this subject, the 50 μ s stimulation (black traces) did not generate more torque *post-100 Hz* compared with *pre-100 Hz*. In addition, the EMG from the 50 μ s trials showed relatively stable M-waves with little or no H-reflex present in either *pre-100 Hz* or *post-100 Hz* windows. In contrast, the same subject showed elevated torque *post-100 Hz* compared with *pre-100 Hz* when 1000 μ s pulse widths were used

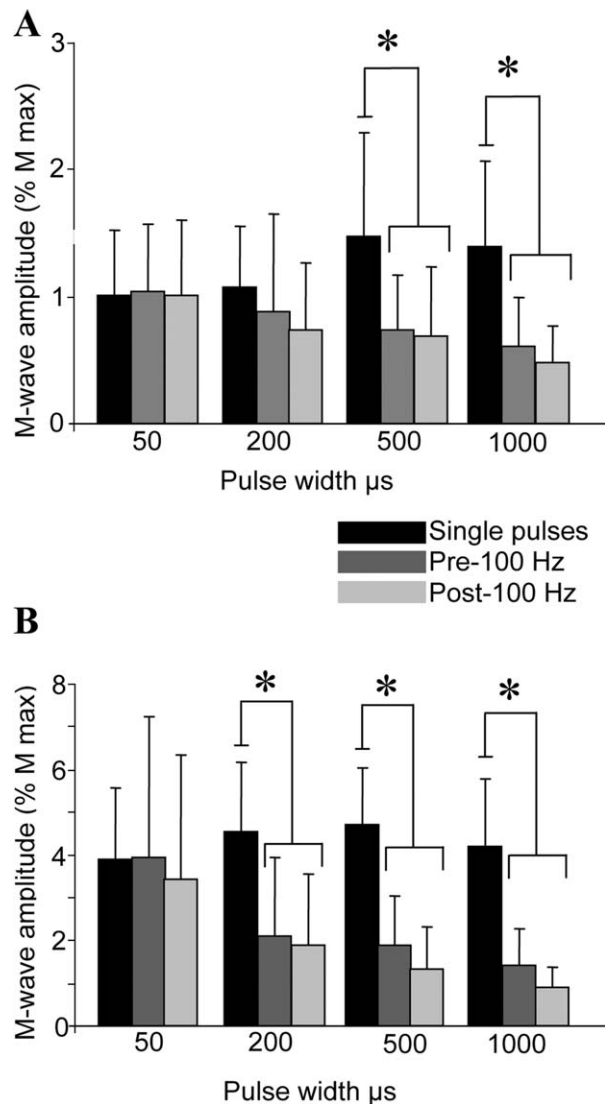


FIGURE 2. Mean group M-waves at motor threshold (**A**) and 5% M_{\max} intensity (**B**) using different pulse widths. Asterisks (*) represent significant differences between single-pulse data and *pre/post-100 Hz* data at each respective pulse width.

(gray traces). During these 1000 μs trials, both M-waves and H-reflexes were smaller during 20 Hz stimulation compared with responses evoked by single pulses; however, H-reflex amplitude recovered from the initial depression during the *post-100 Hz* stimulation.

M-Waves. Stimulus intensity was adjusted so that the amplitudes of M-waves evoked by single pulses were similar. Statistical analysis confirmed that there were no significant differences in M-wave amplitude evoked by single pulses between pulse widths for either stimulus intensity (see “single pulses”; Fig. 2). During 20 Hz NMES, however, M-wave amplitude was influenced by pulse width.

M-waves collected with stimulation at motor threshold showed a significant Time \times Pulse interaction [$F_{(6,72)} = 8.4$; $P \leq 0.05$]. Post hoc analysis

showed that M-waves were not significantly different during 20 Hz NMES (*pre-100 Hz* or *post-100 Hz*) compared with single pulses obtained with 50 and 200 μs pulse widths. However, increasing the pulse width to 500 and 1000 μs caused M-waves to depress on average 53% compared with those evoked by single pulses ($P \leq 0.05$; Fig. 2A).

M-waves at the higher intensity of 5% M_{\max} also revealed a significant Time \times Pulse interaction [$F_{(6,48)} = 10.8$; $P \leq 0.05$]. Post hoc analysis revealed that 50 μs was the only pulse width to not show significant depression of the M-wave during 20 Hz NMES compared with single-pulse values (“50” in Fig. 2B). In contrast, significant M-wave depression (63% on average) occurred during 20 Hz NMES when 200, 500, and 1000 μs pulses were used ($P \leq 0.05$) (see Fig. 2B). At both stimulus intensities and at all pulse widths, M-wave amplitude during NMES was unaffected by the 2 s of 100 Hz stimulation, as there were no significant differences between M-wave amplitudes *pre-100 Hz* to *post-100 Hz*.

H-Reflexes. H-reflexes at motor threshold showed a significant Pulse \times Time interaction [$F_{(6,72)} = 7.8$; $P \leq 0.05$]. Post hoc analysis revealed that H-reflexes evoked by single pulses were significantly larger than both their respective *pre-100 Hz* and *post-100 Hz* values when using 200, 500 and 1000 μs pulse widths ($P \leq 0.05$), but not 50 μs (see Fig. 3A). In addition, H-reflexes evoked by single pulses were on average 82% smaller ($P \leq 0.05$) with 50 μs pulses than H-reflexes generated by single pulses with 200, 500, and 1000 μs pulse widths (see “50” and “single pulses” in Fig. 3A). At motor threshold, H-reflexes showed a significant increase from *pre-* to *post-100 Hz* (194% on average) when 200, 500, and 1000 μs pulse widths were used ($P \leq 0.05$); however, H-reflex amplitude did not increase significantly from *pre-* to *post-100 Hz* when 50 μs pulse widths were used (see Fig. 3A).

H-reflexes obtained during the higher intensity of 5% M_{\max} showed a significant Time \times Pulse interaction [$F_{(6,66)} = 13.5$; $P \leq 0.05$]. Post hoc analysis showed that H-reflexes evoked by single pulses were larger than both their respective *pre-100 Hz* and *post-100 Hz* values at all pulse widths ($P \leq 0.05$) (see Fig. 3B). H-reflexes evoked by single pulses were on average 45% smaller ($P \leq 0.05$) with 50 μs pulses than H-reflexes generated by single pulses with 200, 500, and 1000 μs pulse widths (see “50” and “single pulses” in Fig. 3B). H-reflexes increased on average 225% from *pre-* to *post-100 Hz* with 200, 500, and 1000 μs pulse widths ($P \leq 0.05$); however, H-reflexes did not increase significantly with 50 μs pulse widths (see Fig. 3B).

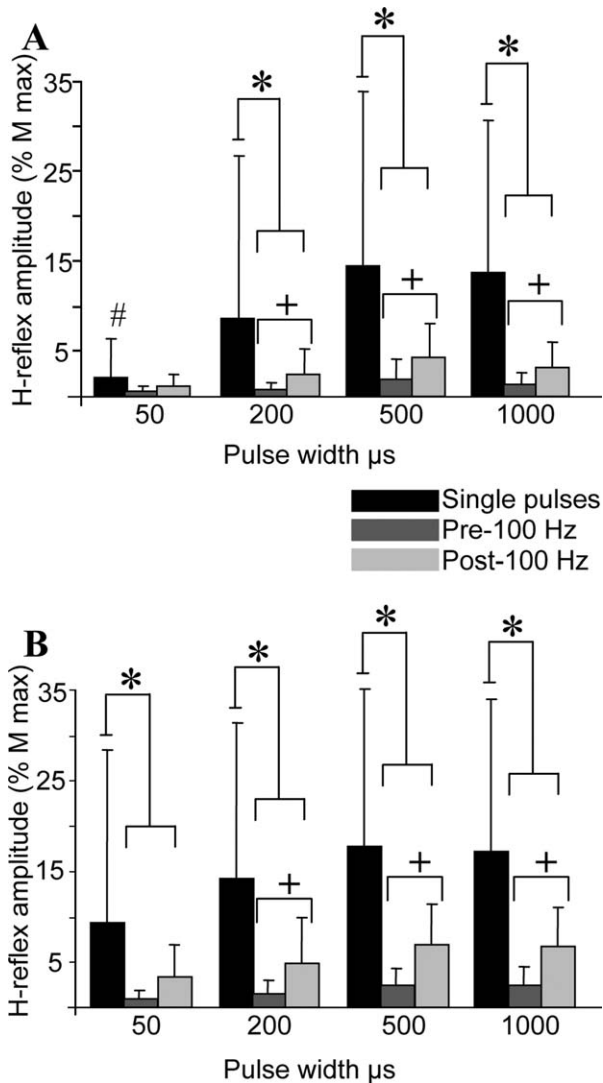


FIGURE 3. Mean group H-reflex amplitudes at motor threshold (**A**) and 5% M_{max} intensity (**B**) using different pulse widths. Asterisks (*) represent significant differences between single pulses and *pre/post-100 Hz* data at each respective width. Plus signs (+) indicate significant differences between *pre-100 Hz* and *post-100 Hz* values. The number sign (#) in (**A**) indicates significant differences between H-reflexes obtained with single pulses relative to single pulses collected with 50 and 100 μ s.

Torque. Torque data at motor threshold showed a significant Time \times Pulse interaction [$F_{(3,39)} = 5.5$; $P \leq 0.05$]. Post hoc analysis revealed that torque increased significantly *post-100 Hz* (on average 55%; Fig. 4A) when pulse widths of 200, 500, and 1000 μ s were used ($P < 0.05$), but 50 μ s pulses did not significantly increase torque. Similarly, with stimulation at the higher intensity of 5% M_{max} , a significant Time \times Pulse interaction was found [$F_{(3,39)} = 6.2$; $P \leq 0.05$]. Again, torque increased significantly *post-100 Hz* (38% on average; Fig. 4B) when pulse widths of 200, 500, and 1000 μ s were used ($P \leq 0.05$), but 50 μ s pulses did not significantly increase torque.

DISCUSSION

These experiments have demonstrated that 20 Hz NMES resulted in depression of M-wave amplitudes compared with M-waves evoked by single pulses of 200–1000 μ s but not 50 μ s pulse widths. Also, compared with single pulses, H-reflexes were initially depressed during 20 Hz NMES (*pre-100 Hz*) at all pulse widths and partially recovered following 2 s of NMES at 100 Hz (*post-100 Hz*) with 200, 500, and 1000 μ s pulse widths, but not 50 μ s pulse widths. In conjunction with increased H-reflexes, torque was significantly greater *post-100 Hz* vs. *pre-100 Hz* NMES at all pulse widths except 50 μ s. Thus, increased torque *post-100 Hz* was associated with decreased M-wave and increased H-reflex amplitudes. These findings support our working hypothesis that NMES delivered with 200–1000 μ s pulse widths generates contractions that have a greater central contribution than those evoked with narrower pulses.

M-Waves during NMES. M-wave amplitude is used as an indicator of changes in muscle fiber excitation during investigations of human muscle fatigue.³² A reduction in M-wave amplitude during NMES has been demonstrated at a number of

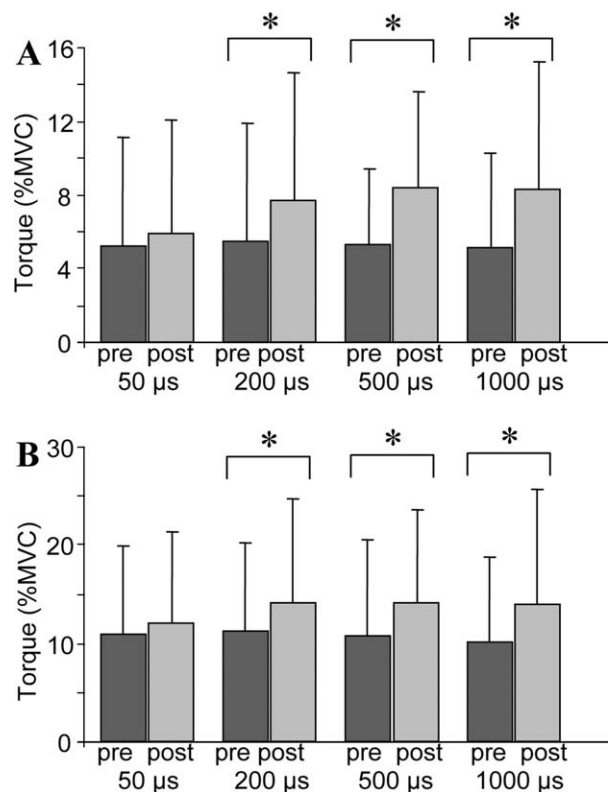


FIGURE 4. Mean group torque values from *pre-100 Hz* (dark gray) and *post-100 Hz* (light gray) intervals when using pulse width stimulation of 50, 100, 500, and 1000 μ s at motor threshold (**A**) and 5% M_{max} intensity (**B**). Asterisks (*) indicate a significant increase in torque from *pre-100 Hz* to *post-100 Hz* values within each respective pulse width.

frequencies when supramaximal stimulation is used (such as 20–70 Hz,³³ 50–80 Hz,³⁴ and 40 Hz³⁵); however, potentiation of M-waves has also been found to occur at supramaximal intensities with 10 and 20 Hz stimulation,^{36,37} as well as during submaximal 20 Hz stimulation.¹¹ Typical pulse widths for clinical use of NMES range from 200 to 400 μ s.¹ Experimentally, pulse widths vary from 50 μ s^{34,36} to 600 μ s³⁸ and 1000 μ s.⁹

Contrary to our hypothesis we found a pulse width- and intensity-dependent effect on the amplitude of M-waves during NMES. During NMES at motor threshold our two narrowest pulse widths (50 and 200 μ s) did not depress M-wave amplitudes compared with M-waves evoked by single pulses, whereas wider pulse widths (500 and 1000 μ s) did. When the stimulus intensity was increased to evoke an M-wave of 5% M_{\max} , 50 μ s was the only pulse width that did not depress M-wave amplitudes. A previous study found that 50 μ s pulse widths delivered at 20 Hz depressed M-wave amplitude,³⁶ although the intensity of stimulation was supramaximal and thus much higher than in our study. Therefore, had we increased our stimulus intensity, it is possible that we too would have observed a depression of M-waves during NMES when using 50 μ s.

Mechanisms that affect the size of the M-wave may be at the level of the axon, the neuromuscular junction, or the muscle fibers. However, because NMES generates action potentials in motor axons beneath the stimulating electrodes in an “all-or-none” manner, the mechanism underlying the depression of M-wave amplitudes must be related to a differential ability of pulse widths to depolarize motor axons repetitively. Increasing the stimulus pulse width used for NMES caused a greater depression of the M-wave relative to single stimuli, thus fewer motor axons must have been recruited. Because Na^+ channels are the major determinant of threshold in axons,³⁹ the depression of M-waves during NMES is likely related to the inactivation of voltage-gated Na^+ channels. The wider pulse widths may have increased the inactivation of voltage-gated Na^+ channels in motor axons, as the inactivation time constant of classic fast voltage-gated Na^+ channels is 500–1000 μ s.^{40,41} Thus, it is likely that the wider pulse widths used in our study (500–1000 μ s) inactivated a greater number of Na^+ channels and led to decreased Na^+ influx, fewer motor axons reaching threshold, and smaller M-wave amplitudes.

H-Reflexes and Torque during NMES. H-reflex amplitude is attenuated during repetitive stimulation at rates above 0.1 Hz due to post-activation depression.¹⁶ This depression is believed to be caused by

reduced transmitter release from previously activated afferent fibers.^{14,15} Some of this reduced transmitter release may reflect an inability to repetitively activate sensory axons with electrical stimulation (as described earlier for motor axons), but post-activation depression also affects stretch reflexes,⁴² suggesting that much of the reflex depression arises centrally. During several seconds of NMES, recovery of the soleus H-reflex can follow the initial depression,^{11,19} and it has been hypothesized that this increased reflex recruitment of motoneurons contributes to an increase in torque.^{9,11} Collins et al.⁸ examined the effect of pulse width on plantarflexion and dorsiflexion torque during NMES by matching the initial electrically evoked forces at each pulse width. They showed that 1000 μ s pulses evoked significantly more plantarflexion torque at the end of a 7-s train of NMES than 50 μ s pulses, but in that study EMG responses were not analyzed. We found that with stimulation at motor threshold and at 5% M_{\max} , pulse widths of 200–1000 μ s resulted in significantly larger soleus H-reflexes *post-100 Hz*, and this was associated with increased torque. Thus, these experiments support our hypothesis that wider pulse widths (200, 500, 1000 μ s) increase H-reflex amplitude and torque *post-100 Hz* vs. *pre-100 Hz*, whereas narrower pulse widths (50 μ s) do not.

Several mechanisms could account for the H-reflex recovery during the *post-100 Hz* period of NMES. These most likely include: voluntary activation, reduced presynaptic inhibition, posttetanic potentiation, and the activation of plateau potentials in spinal neurons. Voluntary activation of the plantar flexors⁴³ as well as general muscle activation during tensing of the body, such as in a Jendrassik maneuver,⁴⁴ will increase the amplitude of the soleus H-reflex. In this study, however, all subjects included in the analysis reported the stimulation to be comfortable and they remained relaxed throughout. In addition, previous experiments have documented an increase in torque using similar stimulation protocols in sleeping⁸ and complete spinal cord-injured subjects.¹² We therefore do not believe that the increased H-reflexes and torque during NMES in our experiments were due to voluntary activation of the plantar flexors or any other muscle group.

Reduced presynaptic inhibition at Ia terminals could increase H-reflex amplitude,^{44–47} thereby generating more torque from reflex recruitment of spinal motoneurons. In addition, posttetanic potentiation may also increase the amplitude of H-reflexes by increasing neurotransmitter release from Ia afferent terminals.^{48–50} Another possibility is that NMES may activate persistent inward currents in spinal motoneurons to cause a sustained

discharge and make them more responsive to sensory input.⁵⁻¹³ It has been suggested that, although persistent inward currents are active, motoneuron discharge may frequently become “time-locked” to each stimulus pulse as H-reflexes.^{11,19} Once persistent inward currents are activated in motoneurons, discharge may also continue in a self-sustained manner, contributing to the generation of torque via activity that is asynchronous from the stimulus pulses.⁷ All of the aforementioned mechanisms could modify the amplitude of the H-reflex, and further experiments are required to differentiate the relative contribution made by each one. The increased torque and H-reflex amplitude *post-100 Hz* with wider pulse widths, however, suggests that very narrow pulses are not as effective for recruiting motoneurons synaptically.

Relevance for NMES. Currently, there is controversy regarding motor unit recruitment order during NMES, as reports have ranged from reversed,^{23,24} random,²⁵⁻²⁸ or near normal,²⁹ compared with synaptic activation. These discrepancies may reflect differences in the relative contribution made by motor axon recruitment (peripheral mechanism) and synaptic recruitment of motoneurons (central mechanism) to the contractions between different studies; however, the extent to which the CNS contributes to contractions evoked by NMES is rarely considered. We have proposed that increasing the central contribution to contractions evoked by NMES by using wide pulses delivered at relatively high frequencies and low intensities may be advantageous for rehabilitation,⁵⁻¹³ because synaptic drive from Ia afferents recruits motoneurons beginning with the smallest, according to Henneman’s size principle.^{30,31} These small, low-threshold motoneurons innervate muscle fibers that are the most fatigue-resistant.⁵¹ Therefore, during synaptic recruitment, slow, fatigue-resistant motor units will be recruited before the fast-fatigable motor units, which may improve fatigue resistance of electrically evoked contractions. In addition, recruiting fatigue-resistant motor units reflexively that are less accessible via direct motor axon depolarization may help protect them from atrophy and the transformation to fast-twitch fiber types that occurs after periods of disuse such as occurs following spinal cord injury.^{52,53} It is important to note that, although it has been confirmed that there is a central contribution to torque produced by NMES of the triceps surae,^{7,8,13} tibialis anterior,⁸ and flexor pollicis longus⁶ muscles, the strength of this contribution may vary between muscle groups. It may be particularly strong for the triceps surae. Similarly, the strength of the central contribution to electrically evoked contractions

can vary from person to person and, although it can reach ~50% of the torque generated during a MVC,^{8,9} it can be negligible in others. On average it reached only ~5% MVC in this study. Such inter-subject variability may suggest that utilizing this central contribution for rehabilitation may be more effective for some patients than others. The present results suggest that, for NMES of the triceps surae, pulse widths of 200, 500, and 1000 μ s are equally capable of generating contractions with a central contribution during low-intensity NMES. Thus, traditional NMES protocols that utilize 200–400 μ s pulse widths at frequencies between 20 and 50 Hz¹ may also generate contractions with a contribution from the CNS provided that stimulus intensities are submaximal. However, the influence of greater stimulus intensities on the central contribution during NMES remains to be evaluated. Delivering NMES at intensities that evoke H_{max} may maximize the reflex contribution to the evoked contractions with minimal antidromic block in motor axons. In contrast, lower intensity NMES, such as that used in this study, which is capable of activating motoneurons centrally via reflex pathways, may be especially useful for patients who cannot tolerate high-intensity stimulation due to heightened cutaneous sensitivity. Furthermore, electrical stimulation of sensory fibers has been shown to enhance both spinal⁵⁴ and cortical circuits⁵⁵; thus, the ability to maximize the afferent volley by using wide-pulse-width and high-frequency NMES may prove to have beneficial effects within the CNS for the rehabilitation of individuals living with paralysis. In contrast, if stable M-waves and a minimal central contribution are preferred, narrow pulse widths, such as 50 μ s, should be used.

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