ARTICLE

Central Contributions to Contractions Evoked by Tetanic Neuromuscular Electrical Stimulation

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COLLINS, D.F. Central contributions to contractions evoked by tetanic neuromuscular electrical stimulation. *Exerc. Sport Sci. Rev.*, Vol. 35, No. 3, pp. 102–109, 2007. *Tetanic electrical stimulation applied over human muscle or peripheral nerve generates contractions by depolarizing motor axons beneath the stimulating electrodes. However, the simultaneous depolarization of sensory axons can also contribute to the contractions by the synaptic recruitment of spinal motoneurons. Maximizing this central contribution may be beneficial for reducing muscle atrophy or restoring movement for persons with movement disorders.* **Key Words:** functional electrical stimulation, muscle contraction, motor unit, recruitment, rehabilitation, persistent inward current, H-reflex

INTRODUCTION

Neuromuscular electrical stimulation (NMES) of human muscles or peripheral nerves is commonly used to generate muscle contractions for rehabilitation. The stimulation is delivered at frequencies that generate fused or "tetanic" contractions to reduce muscle atrophy or restore lost function for people with movement disorders such as stroke or spinal cord injury (SCI). Conventional NMES is typically delivered using narrow stimulus pulses (50–400 μs) delivered at constant frequencies (15–40 Hz). The resultant contractions are believed to arise primarily through a peripheral mechanism, whereby motor units are recruited by depolarizing motor axons beneath the stimulating electrodes (20,25). Motor axon recruitment activates motor units in a non-physiological manner that may limit the value of NMES for rehabilitation (15). Muscle fibers are not activated directly because they have a much higher (10×) threshold for stimulation (20,25). This article describes recent evidence showing that when NMES is delivered using wider pulse widths (1 ms) and higher frequencies (up to 100 Hz; wide pulse stimulation (WPS)) than conventional NMES, a portion of the evoked contraction arises from a central mechanism, whereby spinal motoneurons are recruited by the electrically evoked sensory volley (8,9). The resulting contraction is not transient, as during a flexion reflex, but rather is sustained throughout the stimulation and can generate up to 40% of the torque produced by a maximal voluntary contraction (MVC) (9). The reflexive recruitment of motoneurons by large-diameter afferents activates motor units in the normal physiological recruitment order (5,18) and hence the central contribution to contractions evoked by WPS may recruit preferentially the fatigue-resistant motor units that are particularly vulnerable after a stroke or SCI. Delivering WPS to generate contractions through a combination of peripheral and central mechanisms may be beneficial for rehabilitation.

CENTRAL TORQUE DURING NMES

Surface NMES can be applied through electrodes placed on the skin over the muscle belly (muscle stimulation) as shown for the triceps surae (TS) in Figure 1A, or over the nerve proximal to the muscle (nerve stimulation) at a site where it runs close to the skin surface (*i.e.*, the tibial nerve in the popliteal fossa for TS). Examples of isometric torque generated by contractions evoked by muscle stimulation applied over the TS for 1 min using conventional NMES (50 μs, 25 Hz) and WPS (1 ms, 100 Hz) are shown in Figure 1B. These data were recorded from a relaxed, able-bodied subject, and the stimulus intensity was adjusted so both types of stimulation generated similar torque during the first 2 s of stimulation. Conventional stimulation is thought to generate contractions primarily through motor axon depolarization. The torque evoked by this peripheral mechanism (hereafter referred to as peripheral torque) in Figure 1B declined throughout the stimulation as would be expected if

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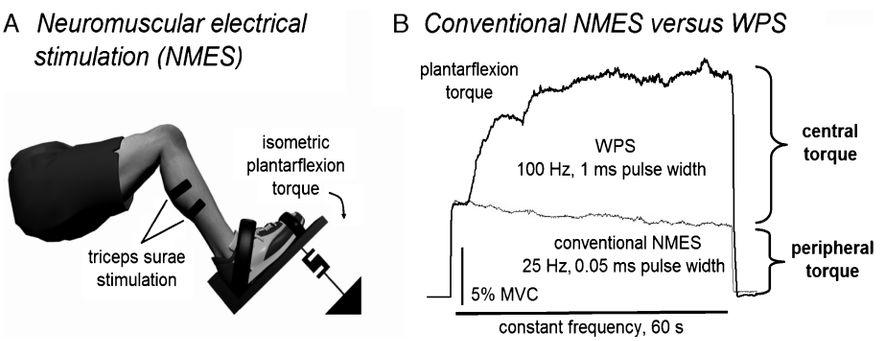

Figure 1. NMES in humans. Brief pulses of electrical stimulation are applied over peripheral muscle or nerve to generate contractions for rehabilitation. Panel A shows one method used in the study of NMES in our laboratory. Panel B shows torque recorded when NMES is delivered using conventional (*thin line) and WPS (thick line).* MVC indicates maximal voluntary contraction; NMES, neuromuscular electrical stimulation; WPS, wide pulse stimulation.

the contraction arose primarily from the recruitment of fast-fatigable muscle fibers (see IMPLICATIONS, later). However, conventional NMES may not recruit motor units in a completely reverse physiological order (which would recruit fast-fatigable fibers first) as had been traditionally proposed (4), and other mechanisms may also contribute to the gradual decline in torque including activity-dependent hyperpolarization of the stimulated motor axons. During WPS, torque increased dramatically beyond that evoked by conventional NMES generating approximately three times more torque by the end of the stimulus train. This additional torque (hereafter referred to as central torque, see Fig. 1B) represents the central contribution from the recruitment of spinal motoneurons by the evoked afferent volley (see later). WPS can trigger the development of central torque in able-bodied (2,8,9,11,22) and SCI subjects (8,26).

The central origin for this augmented torque has been demonstrated by experiments in which the stimulation was applied before and during a complete anesthetic block of the tibial and common peroneal nerves in the popliteal fossa (Figs. 2, 3). Blocking neural transmission proximal to the stimulation site transiently "disconnects" the muscle from the central nervous system, and hence only peripheral mechanisms can contribute to evoked torque during the nerve block. Augmented torque did not develop during the nerve block, confirming that it is caused by a central mechanism and not potentiation distal to the stimulation site, such as at the neuromuscular junction or within the muscle (8,9). However, in these experiments, the stimulation was applied at relatively low intensities and some peripheral potentiation may occur at higher intensities (22). Evidence that WPS can generate contractions solely from the central mechanism with little or no contribution from the peripheral mechanism is provided by the observation that contractions can develop during stimulation below motor threshold (9). Figure 4 shows plantarflexion torque recorded during stimulation delivered at an intensity that evoked an H-reflex but no M-wave. H-reflex is caused by the activation of large-diameter sensory axons resulting in the reflexive recruitment of motor units primarily through a monosynaptic pathway. M-wave arises from the activation of motor axons, and its absence is an indication that no motor axons were recruited directly. Thus, the torques depicted in Figure 4 (>40% MVC in Fig. 4A) were generated by the central mechanism alone.

We have identified several patterns of stimulation that effectively generate central torque (9,11). These are shown in Figure 3 and include constant frequency stimulation at 100 Hz (Fig. 3A), triangular patterns with the frequency increasing linearly from approximately 4 to 100 Hz and back to 4 Hz over 6 s (Fig. 3B) and burstlike patterns where one (Fig. 3C) or more (Fig. 3D) 2-s bursts of stimulation at 100 Hz are delivered during stimulation at a lower frequency (20–25 Hz). The burstlike patterns reveal that torque can remain elevated after a burst, despite the similar stimulation frequency and intensity before and after each burst (Figs. 3C, D). Central torque often increases with successive

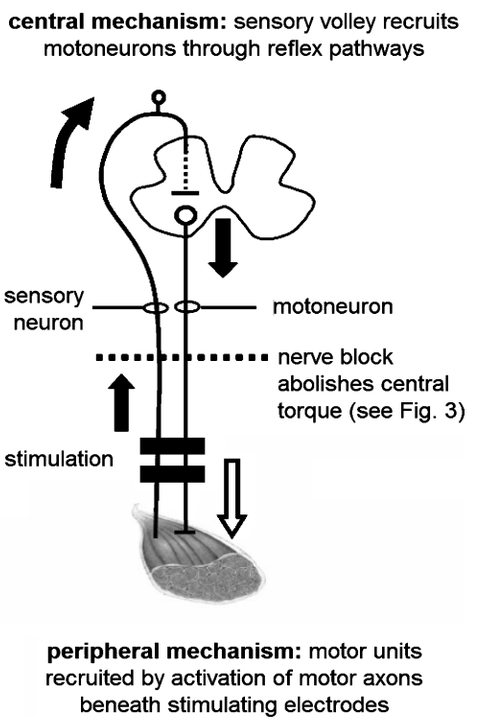

Figure 2. Central and peripheral mechanisms contribute to contractions during wide pulse stimulation (WPS). Motor units are recruited by the evoked sensory volley (central mechanism) and by the depolarization of motor axons beneath the stimulating electrodes (peripheral mechanism).

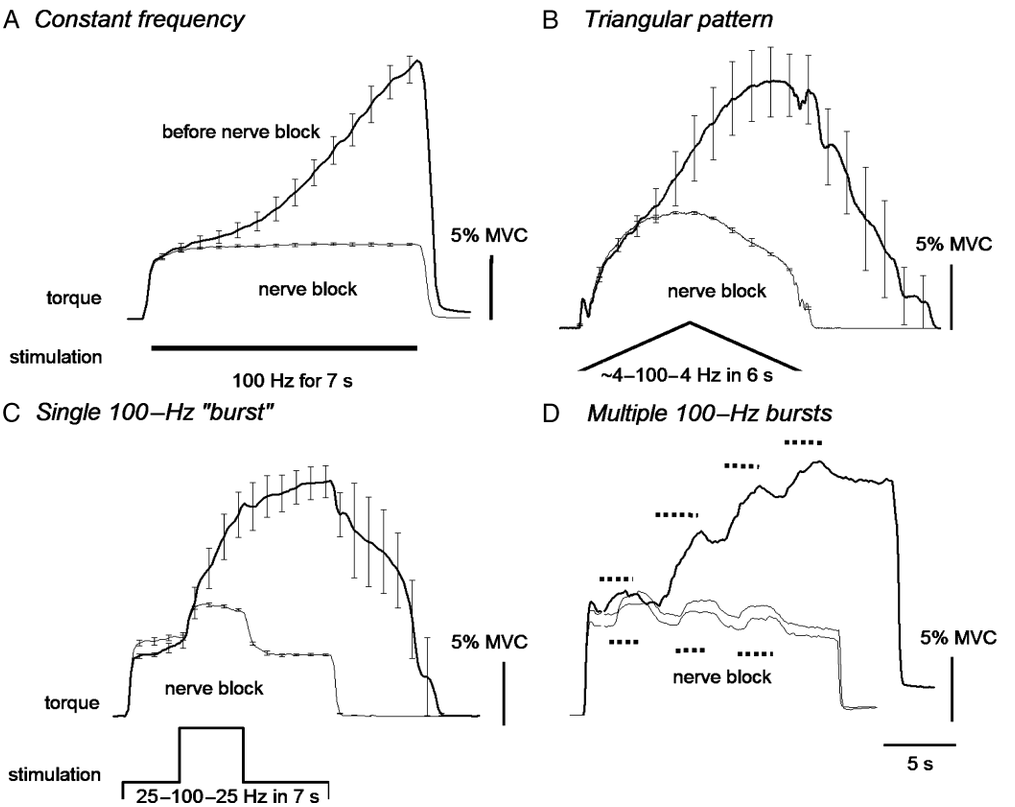

Figure 3. Central torque is abolished during a peripheral nerve block. Torques evoked in single subjects by stimulation over triceps surae (A) and tibialis anterior (B–D). The stimulation was delivered before (*thick lines*) and during (*thin lines*) a complete nerve block. Each trace in panels A–C show the mean and SEM of the torque evoked by five stimulus trains. (Reprinted from Collins, D.F., M. Gorassini, D.J. Bennett, D. Burke, and S.C. Gandevia. Recent evidence for plateau potentials in human motoneurones. In: *Sensorimotor Control of Movement and Posture*, S.C. Gandevia, U. Proske, D.G. Stuart (Eds.). New York, N.Y.: Plenum. *Adv. Exp. Med. Biol.* 508:227–235, 2002a. Copyright © 2002 Springer Science and Business Media. Used with permission.) [Using data adapted from Collins, D.F., D. Burke, and S.C. Gandevia. Sustained contractions produced by plateau-like behaviour in human motoneurones. *J. Physiol.* 538:289–301, 2002. Copyright © 2002 Blackwell Publishing. Used with permission.]

bursts (Figs. 3D, 4B), although each burst recruits progressively more motoneurons that continue to fire after the high frequency stimulation has ended. Furthermore, a weak involuntary contraction (2%–7% MVC) often persists even after the stimulation is turned off. When this occurs, subjects are typically unaware of this residual activity but can stop the contraction with a brief voluntary contraction of the antagonist muscle (27) or a focused effort to "relax completely" that is not associated with voluntary activity in the antagonist muscle (9).

Typically, central torque is maximal when the stimulation is delivered using 1-ms pulses (9) delivered at stimulation frequencies greater than 80 Hz (11). Using these parameters, torque progressively increases on average for approximately 11 s (11). In some subjects, however, central torque is readily evoked using frequencies as low as 20 Hz (11,22) and relatively narrow pulses (unpublished data, Lagerquist, 2007), raising the possibility that there may be a central contribution to contractions evoked by conventional NMES. In contrast, central torque does not develop during WPS in other subjects (11,22).

In able-bodied subjects, central torque can develop in all muscles we have tested thus far, including the ankle plantar (2,8,9,11,22)- and dorsiflexors (9,22) and the wrist flexors (2). On average, a single 100-Hz burst of stimulation (see Figs. 3C, 5) increased torque 1.9 (9)-, 1.6 (9)-, and 1.6 (2)-fold, for the plantarflexors, dorsiflexors, and wrist flexors, respectively, when comparing the torque evoked by the 20-Hz stimulation before with that of after the 100-Hz stimulation. This increase in torque after the 100-Hz burst is used as a measure of the magnitude of the central contribution as it is absent during the nerve block (Fig. 3C) (8,9). Interestingly, muscle stimulation produced more torque through the central mechanism than did nerve stimulation for the ankle plantarflexors but not the wrist flexors (2).

ELECTROMYOGRAPHIC ACTIVITY DURING NMES

Analysis of electromyographic (EMG) activity recorded during NMES can provide insights into motor unit recruitment during the stimulation. Unfortunately, an impediment to obtaining useful EMG during tetanic stimulation is the contamination of the signal by the artifact associated with each stimulus pulse. This is particularly true for muscle stimulation, when recording and when stimulation sites are in close proximity, and at higher stimulation frequencies, when successive artifacts are close together. However, by recording during nerve stimulation at frequencies no greater than 20 Hz (50 ms interstimulus interval) and using care to

reduce the size of the stimulus artifact, we have obtained valuable information about motor unit recruitment using surface (2,22) and single motor unit (8) EMG recordings. These data identify that motor units are recruited in three distinct ways during WPS.

By definition, when stimulating at intensities greater than motor threshold, some motor units are recruited by the depolarization of motor axons beneath the stimulating electrodes. These units appear in the EMG as an M-wave, and their discharge is time-locked to each stimulus pulse at a short latency (for soleus ~5 ms) consistent with orthodromic transmission along motor axons. The synchronous discharge of this population of motor units is responsible for the generation of peripheral torque (Fig. 1B). The contribution of M-waves to torque generated during NMES is not the focus of this review and will not be discussed further here. Unexpectedly, we found that H-reflexes are also present in the EMG signal during WPS (see Fig. 5) (22). Similar to motor units recruited as an M-wave, the discharge of units recruited as an H-reflex is also time-locked to each stimulus pulse, albeit at a longer latency (for soleus ~30–35 ms), given the longer pathway involving transmission to and from the spinal cord. Previously, a contribution from H-reflexes to contractions evoked by NMES stimulation had not been considered because it was thought that H-reflexes are depressed during tetanic stimulation because of a postactivation depression of reflex transmission (also called low-frequency or homosynaptic depression) (19,28). The amplitude of the H-reflex changed dramatically during WPS, and this could account for some of the changes in torque that were observed. H-reflexes recorded from soleus during periods of 20-Hz stimulation delivered over the tibial nerve are shown in Figures 4B and 5. In both examples, the first stimulus pulse of the train evoked a large H-reflex, but reflexes immediately thereafter were markedly depressed, consistent with postactivation depression of reflex transmission. The large first reflex was accompanied by an initial peak in the torque profile. During burstlike patterns of stimulation, reflex amplitude typically is low during the initial 20-Hz stimulation but after a single (Fig. 5) or multiple (Fig. 4B) bursts of 100-Hz stimulation, H-reflex amplitude recovers with a similar time course as the development of central torque. In a group of nine subjects, we found that compared with the first H-reflex in the stimulation train, reflexes in soleus were depressed by approximately 85% after 0.5 s of stimulation at 20 Hz and recovered back to approximately 40% of initial reflex amplitude during 20 Hz of stimulation after a single 100-Hz burst (22). These changes in reflex amplitude occurred with no significant change in M-wave amplitude, suggesting that they were not caused by changes in stimulation current delivered to the nerve. However, the extent to which transmission through the H-reflex pathway contributes to central torque may be dependent on which muscle is being stimulated and how. H-reflexes are predominated during nerve, but not muscle, stimulation of the ankle plantarflexors (2) and were small during WPS of tibialis anterior (22) and the wrist flexors (2). There is also a third type of recruitment during WPS, whereby motor unit discharge is not time-locked to each stimulus pulse (8,23). Such recruitment can be seen in Figure 6, where the data show a unit that is initially recruited at a latency consistent with an H-reflex (*arrow* in Fig. 6B, line 3) that then becomes dissociated from the stimulation and continues to fire even after the stimulation is turned off.

Thus, when WPS is delivered greater than motor threshold, motor units are recruited in three distinct ways; via the peripheral mechanism with a discharge time-locked to each stimulus pulse as an M-wave or via the central mechanism with a discharge that is either time-locked to each stimulus pulse at an H-reflex latency or is temporally unrelated to the stimulation.

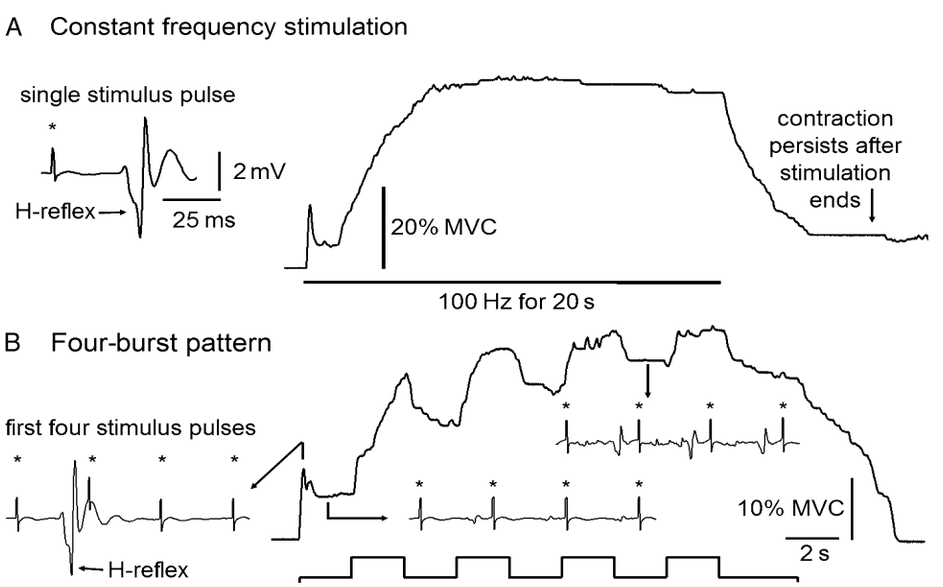

Figure 4. Contractions develop at stimulation intensities below motor threshold. Shown are plantarflexion torque and surface electromyogram (EMG) recorded during wide pulse stimulation (WPS) over the tibial nerve below motor (M-wave) threshold. Asterisks denote stimulus artifacts. In panel B, the stimulus artifacts are 1 mV in amplitude, and the interstimulus interval is 50 ms.

MECHANISMS OF MOTOR UNIT RECRUITMENT

Peripheral Mechanism

For isolated axons in a reduced animal preparation, electrical stimulation activates the largest axons first, with progressively smaller axons recruited as stimulation intensity increases (4). The largest axons innervate muscle fibers that fatigue most rapidly (7), and this reversal of recruitment order from that seen during voluntary contractions (18) is often cited as the reason that electrically evoked contractions fatigue rapidly (15). However, most studies show that this strict recruitment reversal does not hold true when NMES is delivered over human peripheral nerve, with the general conclusion being that motor axon recruitment activates motor units in a random order with no particular relationship to axon diameter (see Fig. 7) (15). Hence, the evidence suggests that the peripheral mechanism recruits motor units synchronously and with no clear relationship to muscle fiber type.

Central Mechanisms

Enhanced Afferent Volley

During NMES, changing the pulse width alters the relative recruitment of motor and sensory axons (30). Conventional NMES uses narrow pulses and evokes contractions primarily by activating motor axons. Sensory axons have a longer strength-duration time constant and lower rheobase than motor axons and are more effectively depolarized by wider pulses (30). This has lead to the recommendation that wide pulse widths (0.5–1 ms) should be used for H-reflex studies. Accordingly, wider pulse widths (0.5–1 ms) evoke larger H-reflexes at lower stimulus intensities (relative to the M-wave), consistent with the larger sensory volley, and tetanic stimulation using wide pulse widths generates more central torque than does stimulation using narrow pulses (9). By combining wide pulses (to preferentially recruit sensory axons) with higher frequencies (to further enhance the effective current delivered to spinal motoneurons), WPS represents a novel way of delivering NMES to maximize the sensory volley to the spinal cord and therefore the synaptic recruitment of motoneurons.

Motoneuron Recruitment

Upon entering the spinal cord, the sensory volley recruits spinal motoneurons, leading to the development of central torque. This recruitment is consistent with the development of persistent inward currents (PIC) in spinal motoneurons or interneurons (Fig. 8) (8–10). PIC lead to sustained depolarizations (plateau potentials), and it is becoming increasingly clear that they play an important role in regulating cell firing in normal (10,13,17) and pathophysiological contractions (3,14,16). In animals, PIC can be triggered by large-diameter afferent input, can develop

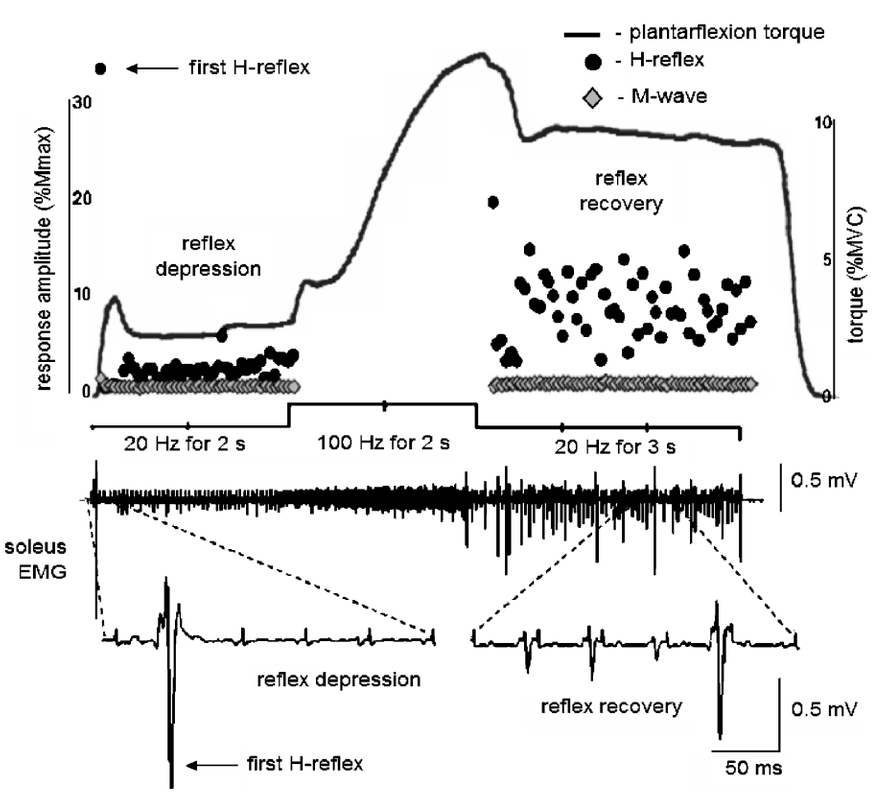

Figure 5. H-reflexes recover in amplitude during the development of central torque. Upper panel shows torque, M-wave, and H-reflex amplitude averaged over five successive stimulus trains in a single subject. The M-wave and H-reflex amplitudes are only shown for the 20-Hz stimulation. Lower panel shows soleus EMG recorded during the delivery of a single stimulation pattern. [Adapted from Klakowicz, P.M., E.R. Baldwin, and D.F. Collins. Contribution of M-waves and H-reflexes to contractions evoked by tetanic nerve stimulation in humans. *J. Neurophysiol*. 96:1293–1302, 2006. Copyright © APS. Used with permission.]

(warm-up) after repeated stimuli, and lead to prolonged discharge in the absence of synaptic drive (17). Similarly, delivering WPS over human muscle generates contractions that can develop at intensities below motor threshold (Fig. 4), increase with repeated stimuli, and persist after the stimulation ends (Figs. 3, 4, 6). PIC are also thought to underlie the sustained contractions that develop during and after periods of tendon vibration (10,12), known as the tonic vibration reflex (6). When motor units are recruited by vibration or low-intensity NMES, their discharge becomes dissociated from the sensory volley (6,23), much like the asynchronous discharge we have observed during WPS (Fig. 6) (8). Interestingly, variability in PIC prevalence in animals has been attributed to different levels of monoamines present in the spinal cord ("monoamine tone") (16). Differences in monoamine tone between our subjects could account for the intersubject variability in the magnitude of central torque evoked by WPS.

The finding that central torque is associated with increased transmission through the H-reflex pathway (Fig. 5) (22) could be accounted for by an increased excitability of the motoneurons due to PIC. However, mechanisms presynaptic to the motoneuronal membrane could also contribute (Fig. 8). The reflex depression immediately after the first H-reflex is consistent with a postactivation depression of neurotransmitter release from Ia afferents to motoneurons (19,28). The partial recovery that follows a burst of 100 Hz stimulation may arise from a posttetanic potentiation of neurotransmitter release associated with the higher frequency stimulation (24). Differences in the strength of postactivation depression and posttetanic potentiation between subjects may be another factor that contributes to the variability in the magnitude of central torque between subjects. Regardless, the central mechanism may involve potentiation on both sides of the Ia-motoneuron synapse; postsynaptically from PICS in spinal neurons and presynaptically from potentiated neurotransmitter release from Ia afferents. Importantly, antidromic transmission of action potentials along motor axons activated beneath the stimulating electrodes will block orthodromic transmission of H-reflexes in the same axon (Fig. 7). Hence, the same motor unit will not contribute to both the M-wave (peripheral torque) and the H-reflex (central torque). Similarly, it is unlikely that units activated centrally that have an asynchronous discharge will reactivate a unit that also contributed to the M-wave. Thus, it is unlikely that the same unit will be activated by both central and peripheral mechanisms.

A possible role for the cortex in the development of central torque should also be considered (Fig. 8). NMES can induce long-lasting increases in cortical excitability (21), thus, enhanced transmission through long-loop pathways may contribute to central torque. However, Nozaki *et al.* (27) suggested that cortical excitability was not altered immediately after stimulation that was associated with enhanced H-reflexes. The changes in cortical excitability that have been reported may depend on a combination of NMES and voluntary commands to motoneurons (21). Our finding that central torque can develop after a complete SCI (26) suggests that intact pathways to and from the brain are not required for central torque to develop. Although not tested directly, the magnitude of the central torque tended to be smaller after SCI than in the able-bodied population, and the importance of intact pathways to and from the cortex to the development of central torque remains to be confirmed.

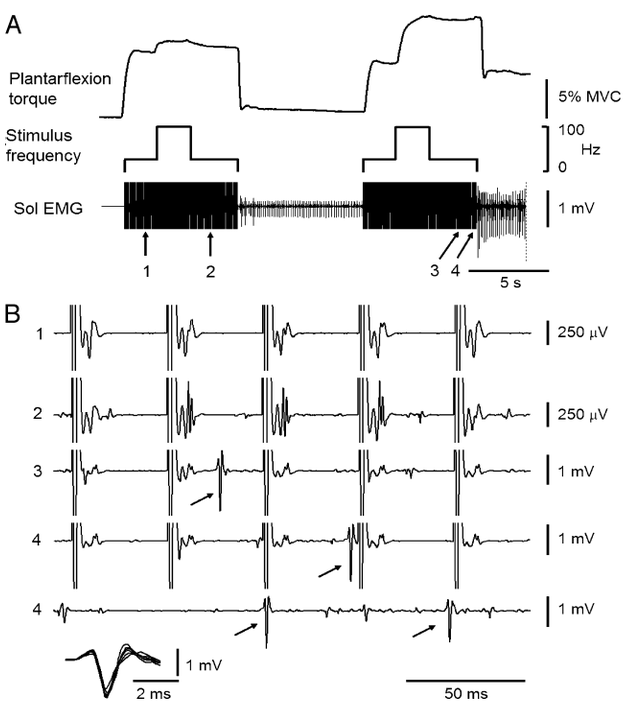

Figure 6. Motor unit activity during and after WPS. Motor unit fires asynchronously during the stimulation and continues to discharge after the stimulation ended. (Reprinted from Collins, D.F., D. Burke, and S.C. Gandevia. Large involuntary forces consistent with plateau-like behavior of human motoneurons. *J Neurosci.* 21:4059–4065, 2001. Copyright © 2001 *Journal of Neuroscience*. Used with permission.)

IMPLICATIONS OF CENTRAL RECRUITMENT

Sensorimotor Integration

The transformation of sensory input into motor output through reflex pathways is an integral part of the control of human movement. Processes such as PIC, postactivation depression and posttetanic potentiation help regulate this transformation. WPS provides a unique way to probe this input-output relationship and assess the effects of repetitive inputs to motoneurons. The finding that NMES (8,9) and vibration (12) can lead to motor unit discharge that outlasts the stimulation is consistent with the activation of PIC and suggests that PIC may be prevalent in human motoneurons. The fact that central torque was triggered when a brief voluntary contraction introduced in the middle of WPS at 20 Hz suggests PIC may be activated by voluntary commands to human motoneurons (9, see also 13). In addition, PIC have been implicated in the generation of pathological contractions such as cramps (3,17) and spasticity (14,16,17). Recent evidence suggests that spasticity may develop, at least in part, from enhanced PIC arising from a supersensitivity of motoneurons to residual serotonin in the spinal cord (16). The precise mechanism for the increased sensitivity is not clear, but it does not arise from an increase in the number of

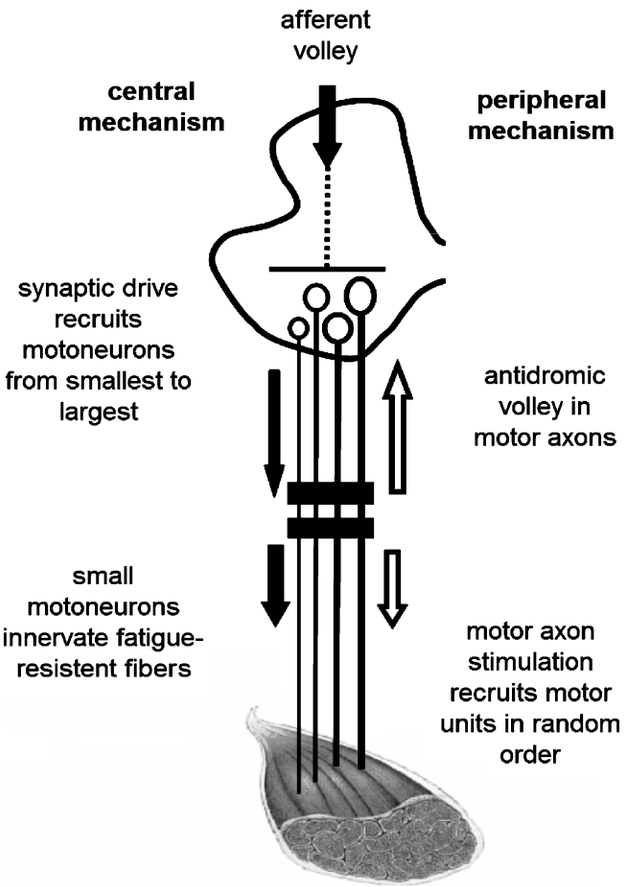

Figure 7. Proposed mechanisms for motor unit recruitment during WPS. The order in which motor units are recruited may be different for units recruited by the central and peripheral mechanisms.

receptor binding sites or from the reduction in serotonin in the spinal cord (16). It is difficult to predict how daily use of WPS might influence PIC activation, although WPS did not trigger pronounced spasms in 14 SCI patients tested previously (26). Interestingly, in the turtle, afferent drive activates motoneurons through ionotropic and metabotropic mechanisms (1). If the same is true in humans, both processes may also contribute to PIC activation during WPS.

REHABILITATION

After an SCI, skeletal muscles below the lesion undergo a loss of mass (atrophy) and muscle fibers convert to those that fatigue most rapidly (20,29). Associated complications are numerous and include reduced circulatory capacity, cardiovascular disease, spasticity, type II diabetes, and osteoporosis. Many of these deleterious sequelae of SCI stem from the inactivity and disuse imposed by the injury. Presently, NMES is the most common technique used to generate muscle contractions to maintain muscle quality (therapeutic electrical stimulation (TES)) and produce purposeful movement (functional electrical stimulation (FES)) after SCI. However, the deterioration of muscle quality means that evoked contractions generate little force and fatigue rapidly (20,29). WPS delivered to recruit motor units in their normal recruitment order may be advantageous for both TES and FES. This would be appropriate after SCI or any situation in which there is disuse atrophy (with intact peripheral nerves) such as stroke, multiple sclerosis, or the immobilization and unloading that occurs during prolonged bed rest, wearing a cast, or periods of spaceflight.

It is presently not clear whether WPS will activate a sufficiently large proportion of motor units to be useful forrehabilitation. The synchronous activation of all motor units in the pool can be achieved via the peripheral mechanism by delivering the stimulation at intensities that depolarize all motor axons beneath the stimulating electrodes. Such high-stimulation intensities can be problematic, however, for patients with residual or heightened sensation. It may be possible to deliver WPS at lower, more comfortable intensities, to recruit motor units via peripheral and central mechanisms.

Maintaining Muscle Quality (TES)

The SCI population is among the most sedentary in the world, and a leading cause of death is cardiovascular disease. FES-assisted exercise programs are one way to combat this problem, but poor muscle quality is a major barrier (20). Thus, before participation in such programs, patients undergo several weeks of TES, but the results of such programs on muscle quality are variable and often fall short of expectations (20,29). By recruiting motoneurons in the normal recruitment order, WPS may reduce, reverse, or even prevent (if applied soon enough after the injury) the atrophy and fiber-type transitions that stem from disuse. Improved muscle quality would mean patients could begin exercise programs sooner and perform at higher intensities. Even if not involved in exercise programs, improved muscle quality should reduce many of the secondary complications related to disuse.

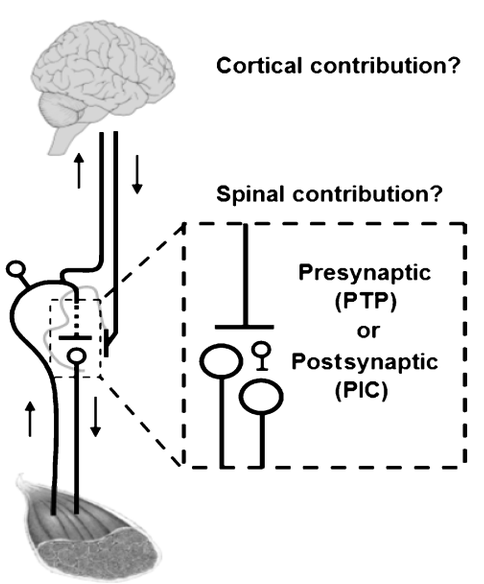

Figure 8. Proposed mechanisms for central recruitment. The portion of the contraction that develops due to the central mechanism may be caused by pre- and postsynaptic mechanisms.

Restoring Function (FES)

FES is becoming increasingly popular as a means to restore movement for activities of daily living as well as for the many benefits of FES-assisted exercise programs, including improvements in cardiovascular fitness and muscle quality (20,29). Theoretically, during WPS, contractions driven by the central mechanism should be more fatigue resistant than those activated by the peripheral mechanism because of the recruitment of fatigue-resistant muscle fibers at low-stimulation intensities and the asynchronous motor unit discharge. This should be advantageous for FES, where rapid muscle fatigue is an ongoing problem. We have established effective ways to trigger centrally driven contractions. However, tasks such as walking and reaching require a level of control (for balance and target acquisition) that we have yet to achieve using central torque (2). The FES-assisted exercise, for example, on a stationary bicycle or indoor rowing machine, does not require such a high degree of control, and this is our initial application for restoring function using WPS.

CONCLUSIONS

WPS generates contractions by depolarizing motor axons (peripheral torque), but strong contractions can also develop from the activation of spinal neurons via reflex inputs to the spinal cord (central torque). The central contribution to the evoked contraction may arise from potentiated neurotransmitter release from large diameter afferents and/or the development of PIC in spinal neurons. Regardless, different populations of motor units contribute to the peripheral and central torque, with the most fatigue resistant likely to be preferentially recruited by the central mechanism. Using WPS to generate contractions that arise from a combination of peripheral and central mechanisms may be useful to reduce muscle atrophy and restore function for people with movement disorders.

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