ABSTRACT: Involuntary muscle contractions are common after spinal cord injury (SCI). Increased sensitivity to Ia muscle afferent input may contribute to the development of these spasms. Since tendon vibration results in a period of postactivation depression of the Ia synapse, we sought to determine whether Achilles tendon vibration (80 Hz for 2 s) altered involuntary contractions evoked by superficial peroneal nerve (SPN) stimulation (5 pulses at 300 Hz) in paralyzed leg muscles of subjects with chronic (>1 year) SCI. Responses to SPN stimulation that were conditioned by vibration were reduced in 66% of trials (by 33 ± 12% in tibialis anterior and 40 ± 16% in soleus). These reductions in electromyographic activity are unlikely to be mediated by changes at the Ia synapse or motoneuron because vibration did not alter the magnitude of the soleus H reflex. The electromyographic reductions may involve long-lasting neuromodulatory effects on spinal inhibitory interneurons or synapses involved in the flexor reflex pathway. Vibration-evoked depression of electromyographic activity may be clinically useful in controlling involuntary muscle contractions after SCI.

Muscle Nerve **33:** 637–644, 2006

DEPRESSION OF INVOLUNTARY ACTIVITY IN MUSCLES PARALYZED BY SPINAL CORD INJURY

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Accepted 21 November 2005

After chronic spinal cord injury (SCI), many people experience involuntary contractions of paralyzed muscles. A range of stimuli including non-noxious cutaneous inputs, such as cold or light touch, can evoke these involuntary muscle contractions, although these stimuli have little reflex effect in the relaxed muscles of individuals with an intact nervous system.[35,43] Both the intensity and frequency of spasms in paralyzed muscles seem to increase to a stable level over the first 2 years after SCI.[9,18] However, the mechanisms that contribute to the evolution of these spasms and their prevalence after chronic injury are poorly understood. One theory to explain clonic activity in spasms is that the rhythmic muscle contractions are sustained by the enhanced reflex effects of muscle spindle afferents on the motoneuron pools of paralyzed muscles.[17,29]

This study was designed to examine one aspect of the role of muscle spindles and their afferents in the development of involuntary muscle contractions after SCI. After muscle spindle afferents are activated by vibration, stretch, or electrical stimulation, the size of the H reflex in that muscle is reduced for up to 10 seconds by a number of presynaptic inhibitory mechanisms, including primary afferent depolarization[33] and postactivation depression of synaptic transmission.[21] In this study we assessed the role of Ia muscle spindle afferents in the development of involuntary activity in leg muscles paralyzed chronically by SCI. First, we evoked reflex or spasm-like electromyographic (EMG) activity in various leg muscles by stimulation of the superficial peroneal nerve (SPN). We then determined the extent to which this involuntary EMG activity was reduced by prior vibration of the Achilles tendon. We hypothe-

Abbreviations: EMG, electromyographic (activity); H_{max}, maximal H reflex; LG, lateral gastrocnemius; MG, medial gastrocnemius; M_{max}, maximal M wave; rms, root mean square; SCI, spinal cord injury; SOL, soleus; SPN, superficial peroneal nerve; TA, tibialis anterior

Key words: cutaneous nerve stimulation; muscle spasm; paralyzed muscle; tendon vibration

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Published online 18 January 2006 in Wiley InterScience (www.interscience.wiley.com). DOI 10.1002/mus.20500

sized that if feedback from Ia muscle spindle afferents was crucial to the development of this involuntary activity in the paralyzed muscles of SCI subjects, the involuntary muscle activity should be depressed or abolished after tendon vibration during periods of postactivation depression of the Ia afferent synapse.

METHODS

Eight people with chronic (>1 year) SCI and complete paralysis of all muscles in both legs were studied (2 women and 6 men; mean ± SEM: age, 37 ± 3 years; injury duration, 14 ± 2 years). Current injury level was at C-4 ($n = 1$), C-5 ($n = 2$), C-6 ($n = 2$), C-7 ($n = 1$), or T-6 ($n = 2$), as defined by American Spinal Cord Injury Association (ASIA) criteria.[25] The injuries were classified as ASIA A ($n = 3$) or B ($n = 5$). Two subjects took baclofen (80 mg/d). All subjects gave informed written consent to participate. All experimental procedures were approved by the University of Miami Institutional Review Board and were conducted in accordance with the Declaration of Helsinki.

Experimental Arrangement. Each subject sat in their wheelchair for the duration of the experiment. Either the left ($n = 4$) or right ($n = 4$) leg was tested with both the knee and ankle flexed at about 90°.

EMG activity was recorded using surface electrodes (1-cm diameter; Cleartrace, ConMed Andover Medical, Haverhill, Massachusetts) placed ~3–4 cm apart over the soleus (SOL), medial and lateral gastrocnemius (MG and LG), and tibialis anterior (TA) muscles (Fig. 1A). The EMG signals were amplified (Grass P511 Amplifier; Astro-Med, West Warwick, Rhode Island), filtered (30 Hz to 1 kHz), and stored on a computer (sampling rate 3200 Hz; SC/Zoom data acquisition system, Physiology Section, Umeå University, Sweden). EMG activity was also monitored on an oscilloscope throughout the experiment.

Stimulation. To evoke muscle spasms, the superficial peroneal nerve was stimulated with a train of 5 pulses at 300 Hz (pulse duration 200 μs, 25.0–67.5 mA; Digitimer DS7H, Digitimer, Ltd., Welwyn Garden City, UK) through surface electrodes placed ~4 cm apart on the dorsum of the foot on the test side (Cleartrace). The stimulator was triggered by computer-generated pulses. The pulse patterns were programmed using commercially available software (Fystat, Dataid, Umeå, Sweden).

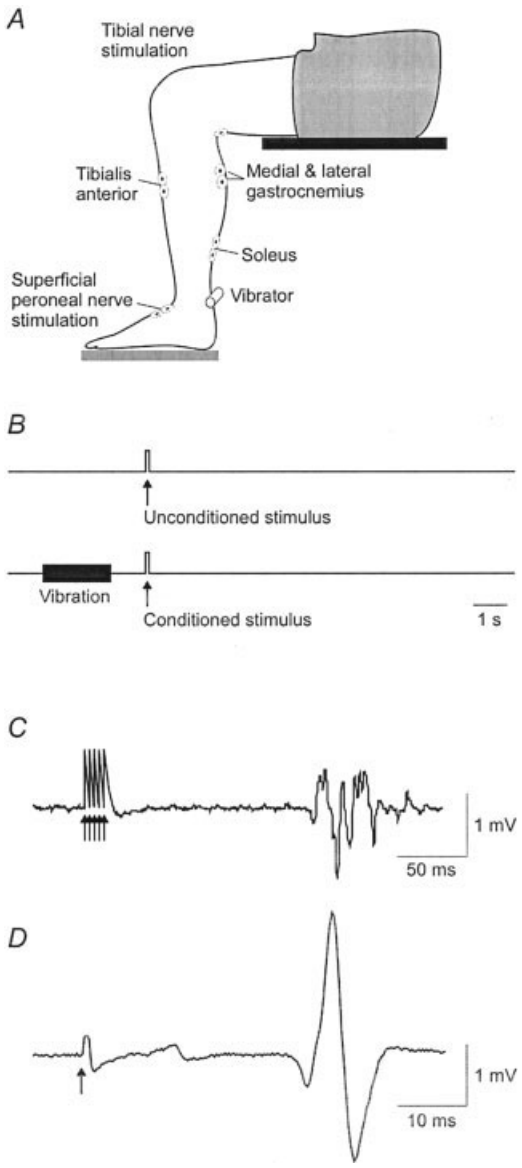

FIGURE 1. Experimental protocol. **(A)** Experimental arrangement showing placement of surface electrodes over tibialis anterior, soleus, medial gastrocnemius, and lateral gastrocnemius. Electrical stimuli were delivered to either the superficial peroneal nerve to evoke spasms or to the tibial nerve to evaluate soleus H reflexes. Vibration was delivered at 80 Hz to the Achilles tendon. **(B)** Trials were performed in pairs with one train of stimuli unconditioned, the other conditioned (i.e., without or with tendon vibration delivered prior to the electrical stimuli). **(C)** Tibialis anterior EMG activity recorded after a train of stimuli to the superficial peroneal nerve (5 pulses, 300 Hz). Note the burst of EMG at ~160 ms after the stimuli. **(D)** Soleus EMG activity recorded after a single electrical stimulus to the tibial nerve. Note the small initial M wave at ~9 ms followed by the H reflex (~30% M_{max}) at ~30 ms.

In able-bodied subjects, the radiation threshold for sensation can be used to set the intensity of stimulation.[41] However, this was not possible in our

SCI subjects. Thus, the intensity of the stimulus was increased until a reproducible spasm or involuntary burst of EMG activity was evoked in TA and SOL muscles after a delay of about 170–180 ms (Fig. 1C; the latency of the involuntary EMG responses indicated a flexor withdrawal reflex mediated by slowly conducting small-diameter afferents). Over the course of the experiment, the size of the evoked response in the unconditioned trials decreased in some subjects. In these cases, the stimulus intensity had to be increased slightly so that an unconditioned response was evoked in TA. On the basis of the radiation threshold of two able-bodied subjects, we estimate that the intensity used for the SCI subjects was between four and eight times radiation threshold. None of the SCI subjects reported any sensations of discomfort at the site of stimulation.

A conditioning stimulus, Achilles tendon vibration for 2 s at 80 Hz (Heiwa Denshi, Model TNT-18 Vibrator, Heiwa Corp., Tokyo, Japan), was delivered before half of the evoked spasms (Fig. 1B). The vibrator was either held steady against the tendon by an experimenter or strapped firmly around the ankle. The sustained vibration, and the frequency of vibration, were chosen with the intent of activating muscle spindles in the plantar flexor muscles.[4,32] To determine the effectiveness of the tendon vibration in activating Ia afferents and causing postactivation depression, the effect of the tendon vibration on H reflexes in the SOL was also assessed (Fig. 1D). H reflexes are normally depressed for at least 10 s after Ia afferent activation in able-bodied control subjects.[21] H reflexes are also depressed in subjects with SCI after vibration, although to a lesser extent than in able-bodied subjects.[6] H reflexes were evoked on the test side by single-pulse stimulation applied over the tibial nerve (1-ms pulse width, 20–67 mA; Digitimer DS7H) through surface electrodes. The cathode was located in the popliteal fossa and held in position by a firm gauze pad taped to the skin. The anode was placed on the patella. A stimulus–response curve was performed for the H reflex and M wave for each subject. The stimulus intensity chosen to evaluate the effects of vibration on the soleus H reflex was one that resulted in a small M wave (so that we could check that the intensity was comparable across trials) with a submaximal H reflex (to ensure that the H reflex could either increase or decrease when conditioned by tendon vibration).

Stimulation Protocol. For each subject, the superficial peroneal nerve on the dorsum of the foot was stimulated either with or without the conditioning vibration applied to the ankle. Ten pairs (i.e., 20 trials) of alternating unconditioned and conditioned trials were recorded for each subject. Each of the trials was separated by 30 s to allow for recovery of postactivation depression after tendon vibration. During conditioned trials, the electrical stimulation of the superficial peroneal nerve was applied 1 s after the end of the 2-s-long vibration. This conditioning–test interval was chosen to fall within the time window of postactivation depression (up to 10 s) while avoiding presynaptic inhibitory effects due to primary afferent depolarization, which lasts only up to 100 ms. Ten pairs (i.e., 20 trials) of alternating unconditioned and conditioned H reflexes were also recorded from each subject with the same protocol as just described. A 1-s conditioning–test interval was also used, a time when postactivation depression is usually most dramatic.[21]

Data Analysis. The amount of involuntary EMG activity in the muscles of the lower limb evoked by the cutaneous stimulation was measured using the calculated root mean square (rms) of each EMG signal. The onset latency, peak activity, and duration of the burst or bursts of activity in each muscle were measured. EMG activity was measured up to a cut-off point 5 s after the stimulus train to avoid the inclusion of spurious EMG activity resulting from unrelated movements (subjects sometimes adjusted their position in their wheelchair). The area of the EMG activity or total amount of evoked EMG activity was calculated using the mean rms EMG activity multiplied by the duration of EMG activity. For the H-reflex responses in soleus and the preceding small M wave, the onset latency and peak-to-peak amplitude were measured. The amplitude of each of the responses was expressed relative to the maximal M wave (M_{max}). The size of the H reflex was also expressed relative to the maximal H reflex (H_{max}). M_{max} and H_{max} amplitude and area were determined for each subject from the stimulus–response data obtained prior to the cutaneous nerve stimulation.

Statistics. Paired *t*-tests and Wilcoxon signed rank tests were used to compare the onset latency, the amount of EMG activity present in each muscle during the conditioned and unconditioned involuntary bursts of activity evoked by the cutaneous stimuli, and the relative change in evoked activity in the

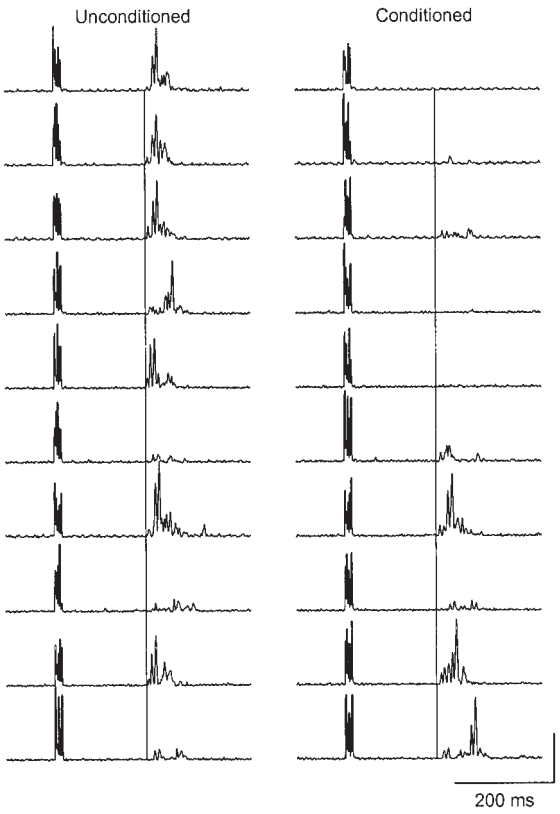

FIGURE 2. Tibialis anterior EMG activity from a single subject in unconditioned and conditioned trials. Ten pairs of unconditioned (left) and conditioned rms EMG trials (right) recorded from tibialis anterior. Stimuli were delivered to the superficial peroneal nerve at 300 Hz (5 pulses). Conditioned responses involved Achilles tendon vibration at 80 Hz prior to SPN nerve stimulation. Vertical calibration bar is 1 mV. Note that, in 7 of the 10 pairs of trials, less EMG (response area) was evoked after vibration.

conditioned compared to the unconditioned trials. Unconditioned and conditioned H reflexes were also compared using Wilcoxon signed rank tests. Statistical significance was set at $P < 0.05$.

RESULTS

Figure 2 shows that the EMG activity evoked in paralyzed leg muscles (TA) by SPN stimulation could be abolished in 3 of 10 pairs of trials by prior tendon vibration. This conditioning also reduced the evoked EMG activity in another 4 of the 10 pairs of trials. For the group data, vibration conditioning reduced the amount of evoked EMG activity across the four muscles tested in 66% of trials. In one third of these trials (22% of the total), the EMG responses were abolished. Involuntary EMG increased in only 28% of trials (significantly less than the number of decreased trials, $P < 0.05$). In 6% of trials, no responses occurred to either the unconditioned or conditioned stimuli. There was no significant difference in the incidence of reduced involuntary activity across the different muscles.

Unconditioned Involuntary EMG Responses. The unconditioned cutaneous stimuli (train of 5 pulses at 300 Hz over the superficial peroneal nerve) typically evoked a reproducible burst or bursts of EMG activity in all four of the muscles studied in every subject ($n = 8$). Unconditioned responses were evoked in TA in response to 100% of stimuli, in SOL by 89% of stimuli, in MG by 85% of stimuli, and in LG by 87% of stimuli. The mean onset latency of the unconditioned evoked EMG activity was earlier for TA and SOL than for MG and LG (Table 1).

In two subjects, multiple bursts of EMG activity occurred in a reproducible clonus-like pattern, with TA bursts occurring out of phase with bursts in SOL, MG, and LG (Fig. 3). The duration of the involuntary bursts of EMG was reproducible within subjects but variable across subjects. One subject continued to show bursts of involuntary EMG after the 5-s measurement limit and was therefore excluded in the calculations of mean burst duration. Mean burst

Table 1. Group data for unconditioned and conditioned responses.

	Unconditioned				Conditioned			
	TA	SOL	MG	LG	TA	SOL	MG	LG
Onset time (ms), $n = 8$	171 ± 13	189 ± 12	303 ± 101	350 ± 102	273 ± 105 (45 ± 41%)	242 ± 44 (30 ± 23%)	340 ± 156 (−2 ± 9%)	401 ± 210 (0 ± 12%)
Duration (ms), $n = 7$	620 ± 175	442 ± 189	287 ± 158	392 ± 115	640 ± 216 (−2 ± 9%)	336 ± 163* (−41 ± 14%)	304 ± 169 (−23 ± 32%)	174 ± 85 (−39 ± 24%)
Area (mV.ms), $n = 7$	49.8 ± 18.3	29.1 ± 15.7	15.2 ± 8.2	16.7 ± 8.2	28.8 ± 9.5* (−33 ± 12%)	16.6 ± 9.9* (−40 ± 16%)	10.9 ± 6.0 (−27 ± 31%)	9.8 ± 6.0 (−36 ± 27%)
Peak amplitude (mV), $n = 8$	0.89 ± 0.19	0.23 ± 0.10	0.15 ± 0.09	0.17 ± 0.09	0.52 ± 0.18* (−38 ± 16%)	0.12 ± 0.04* (−36 ± 13%)	0.05 ± 0.02* (−45 ± 18%)	0.06 ± 0.02* (−43 ± 19%)

Mean ± SEM data for the group from all four muscles. Percentage changes from unconditioned data are shown in parentheses.
**Significant difference from unconditioned ($P < 0.05$).*

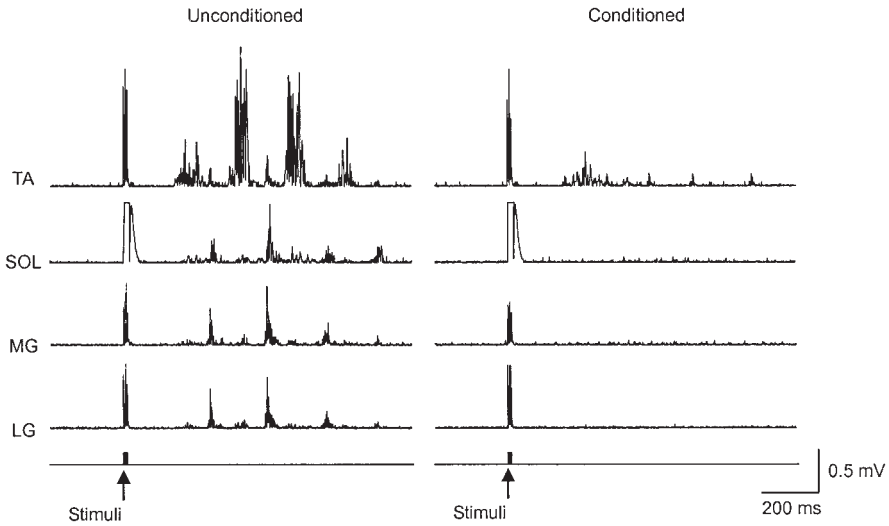

FIGURE 3. Repetitive EMG activity in a single subject in response to superficial peroneal nerve stimulation. Simultaneously recorded rms EMG from tibialis anterior (TA), soleus (SOL), and medial and lateral gastrocnemius (MG and LG) showing repetitive bursts of activity in response to stimulation at 300 Hz (5 pulses). This large amount of activity was almost abolished when the Achilles tendon was vibrated at 80 Hz for 2 s prior to nerve stimulation.

durations for the remaining 7 subjects are given in Table 1.

The amount of evoked EMG (EMG area) was determined by calculating the mean rms EMG multiplied by the duration of the EMG activity. There was no significant difference between muscles for area, although the peak EMG was significantly larger in TA ($P < 0.05$; Table 1).

Changes in Evoked EMG after Tendon Vibration. After the conditioning vibration, the stimulus to the superficial peroneal nerve on the dorsum of the foot was less effective in producing involuntary EMG activity in 7 of the 8 subjects (Table 1 and Fig. 4). Only one subject showed the opposite effect. In this subject, the vibration sometimes evoked a spasm and, in some muscles, facilitated the response to SPN stimulation. The vibration itself did not cause any detectable activity in the other 7 subjects.

Figure 4A–D shows the EMG area for each muscle and for each subject with and without conditioning vibration of the tendon. The mean EMG area for the group of subjects was significantly reduced on average by 33% for TA and by 40% for SOL ($P < 0.05$). The EMG area was also reduced for MG and LG by 27% and 37%, respectively (not significant). The group mean peak EMG was decreased significantly after tendon vibration in all of the muscles by between 36% and 45% for TA, SOL, MG, and LG (Table 1).

After the 2 s of Achilles tendon vibration, onset of evoked EMG was delayed by 40–100 ms. This delay was not significant. The duration of the bursts of involuntary EMG was decreased significantly only in SOL.

H Reflex Responses. H reflexes were evoked in SOL in 7 of the 8 subjects at rest. Only M waves were evident in the other subject. The mean (± SEM) maximal H reflex (H_{max}) across subjects was 2.5 ± 0.9 mV and the mean maximal M wave (M_{max}) was 5.8 ± 1.6 mV. We used an H reflex with submaximal amplitude to test the effects of the vibration. The mean amplitude of this test H reflex was 1.7 ± 0.6 mV (27 ± 6% M_{max} and 74 ± 10% H_{max}). The mean onset time of the H reflex was 31.3 ± 1.8 ms. The conditioning vibration did not significantly reduce the amplitude of the H reflex for the group of subjects (1.6 ± 0.7 mV; i.e., 23 ± 6% M_{max} and 65 ± 9% H_{max}). The H reflex was significantly reduced in amplitude in only 2 of the 7 subjects tested (by 37% and 44%, respectively; $P < 0.05$ for these subjects). The initial small M wave did not alter between the unconditioned and conditioned H reflex tests (3.5 ± 1.4% M_{max} and 3.4 ± 1.3% M_{max}, respectively; mean onset time 9.2 ± 0.6 ms). This suggests that the stimulus to the nerve was consistent across the pairs of trials. The degree to which the H reflex was altered after tendon vibration did not correlate with any of the changes in EMG activity evoked by the stimuli delivered to the superficial peroneal nerve (see also Fig. 4E). Thus, the behavior of the H reflex during the period of anticipated postactivation depression of the Ia afferent fibers did not

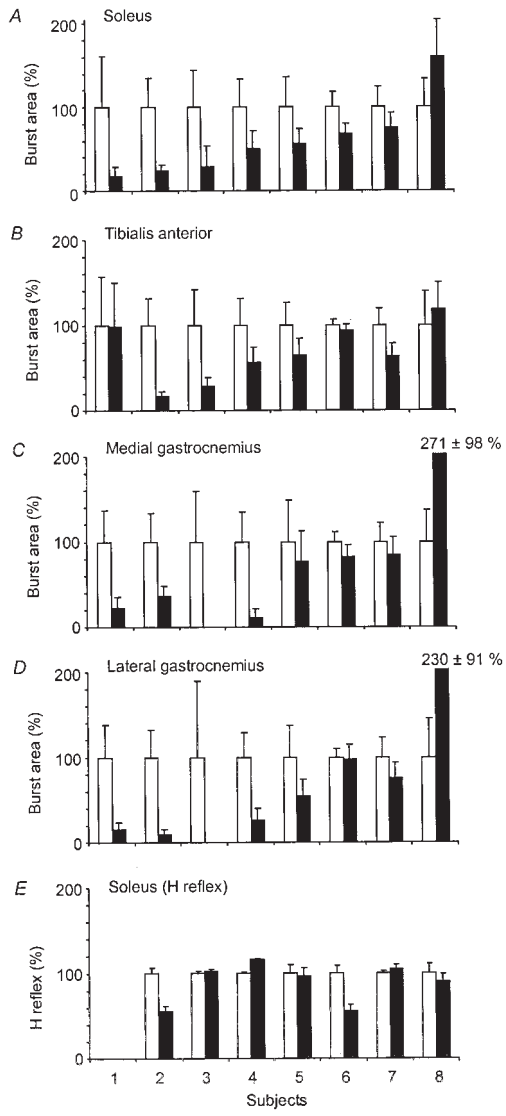

FIGURE 4. Changes in evoked EMG for individual subjects. **(A)–(D)** Mean (+ SEM) EMG area for soleus, tibialis anterior, medial gastrocnemius, and lateral gastrocnemius for each subject during unconditioned trials (open bars) and conditioned trials (filled bars). Data are normalized to the mean unconditioned value for each subject and are presented in order of the largest effect of vibration conditioning on soleus EMG area. Note that only one subject did not show a reduction in EMG activity after tendon vibration. In this subject, the tendon vibration often caused a spasm and therefore did not depress the evoked EMG. **(E)** Corresponding data for mean (+ SEM) unconditioned and conditioned H reflexes for each subject (subject 1 had no H reflex at rest).

predict the behavior of the involuntary EMG activity after vibration.

DISCUSSION

The main finding of this study is that the involuntary activity evoked in paralyzed leg muscles by SPN stimulation on the dorsum of the foot can be reduced or abolished when conditioned by prior vibration of the plantar flexor tendon at the ankle. It is possible that the depression of the evoked EMG activity relates to prolonged neuromodulatory effects that act on the flexor reflex pathway via inhibitory interneurons in the dorsal horn. The depression does not seem to involve either inhibition of presynaptic terminals of Ia afferents or a reduction in motoneuron excitability because of the lack of reduction in the soleus H reflex.

Unconditioned Responses. The SPN is a sensory nerve carrying cutaneous afferent fibers from the skin supplying the dorsum of the foot. The afferents responsible for the evoked EMG activity produced by the stimulus train are most likely nociceptive cutaneous receptors. The relatively long onset latency of the initial involuntary activity in TA and SOL (~180 ms) is consistent with a slowly conducting afferent limb and a fast-conducting efferent limb as in the flexor reflex response.[15,16,30,40] This reflex also appears with a longer latency in subjects with complete spinal cord lesions than in control subjects.[5,31] Using the average latency of the soleus H reflex (31 ms) and M wave (9 ms) evoked by tibial nerve stimulation in the SCI subjects in this study, we estimate that the efferent limb of the response (from motoneuron to soleus muscle) takes ~20 ms, because the H reflex is conducted mainly via large-diameter axons. Therefore, for the response to SPN stimulation, this leaves ~160 ms for the afferent signal to reach the spinal cord and connect to the motoneurons. Taking into account the different stimulation sites and the longer afferent pathway for the SPN stimulation, we estimate that the afferent conduction velocity is at least eight times slower than that for the large-diameter afferent fibers (50–60 $m.s^{-1}$)[24,36] and is in the range of 6–7 $m.s^{-1}$. This conduction velocity is consistent with that of small-diameter cutaneous afferents such as pain-sensitive A-delta fibers (12–30 $m.s^{-1}$), and later components of the EMG responses may involve C fibers (0.5–2 $m.s^{-1}$).[14] Alternatively, the delay in the response time may be due to complex central pathways. However, the high stimulus intensities required to produce the responses also suggest that small-diameter afferent fibers are involved.[3]

The clonus-like activity that occurred in two subjects after the initial burst of EMG may relate to muscle receptors activated by the reflex contraction of either TA or SOL muscles,[17,27,29] may include the initiation of a central oscillator,[2,10,39] or may involve both peripheral and central factors.[38]

Conditioned Responses. Conditioning vibration to the Achilles tendon caused significant depression of the involuntary response to SPN stimulation in SCI subjects. However, the same vibration did not result in significant depression of the soleus H reflex in these same subjects. In our study, only two subjects showed significant depression of the H reflex after tendon vibration, but this depression was not correlated with the reduction in the involuntary activity evoked by SPN stimulation (Fig. 4). Because both the SPN and tibial nerve stimuli were delivered 1 s after the vibration, we expected that the H reflex might be reduced by homosynaptic postactivation depression of the Ia afferent synapse, which can last up to 10 s,[21] rather than by presynaptic inhibition caused by primary afferent depolarization, which only has effects for ~100 ms.[33] In subjects with chronic SCI, postactivation depression of the H reflex is reduced less by vibration than it is in able-bodied subjects, although for uncertain reasons.[6,34] Nevertheless, the lack of depression of the H reflex in our experiment has implications for the site at which vibration depresses EMG evoked by SPN stimulation. First, the conditioning vibration did not result in any significant presynaptic inhibition of the Ia synapse in most subjects. However, we tested only at a 1-s conditioning–test interval, and thus presynaptic inhibitory effects may be more important at other intervals. Second, there were no significant changes in the excitability of the soleus motoneurons tested 1 s after the end of the vibration. Thus, the vibration-induced depression of the involuntary activity evoked by SPN stimulation is not related to reduced excitability of the motoneurons themselves or decreased efficacy of the Ia synapse.

Effect of Tendon Vibration. Vibration at 80 Hz over the Achilles tendon will predominantly activate primary muscle spindles,[4,32] and their discharge will be locked to the vibration frequency. However, vibration also activates other receptors with large-diameter afferents including group II muscle spindle endings, tendon organs, cutaneous receptors sensitive to vibration (fast-adapting type 1 and 2 mechanoreceptors), as well as some small-diameter group III and IV muscle afferents sensitive to mechanical stimuli. Activation of any of these classes of receptors could be responsible for the long-lasting inhibitory effects on the involuntary activity evoked by SPN stimulation.

Long-Lasting Reflex Effects. Only a few studies have shown long-lasting (>1 s) inhibitory effects on reflex or involuntary activity that are not ascribed to postactivation depression of the Ia synapse. Hortibagyi et al.[19] showed long-lasting (~35 s) reductions in H reflexes in flexor carpi radialis associated with contralateral voluntary contractions. However, the mechanisms for this decrease in H reflexes were not elucidated and may not apply to involuntary contractions. In individuals with complete SCI, functional electrical stimulation over rectus femoris causes a long-lasting (>1 s) reduction in the size of the H reflex in SOL that is thought to be caused by activation of cutaneous afferents.[23] Transcutaneous stimulation for 30 min over the sural nerve at 100 Hz can suppress flexor reflex responses in SCI subjects for up to 30 min.[13] In addition, activation of remote cutaneous afferents reduces the firing of spontaneously active thenar motor units after SCI.[42] Low-threshold mechanoreceptor afferents such as cutaneous afferents are known to interact with nociceptive transmission in ascending anterolateral pathways to reduce the sensation of pain and presumably also interact at a spinal level.[37] However, the mechanisms for these long-lasting effects are not known.

In animals, long-lasting postsynaptic excitatory effects have been ascribed to activation of persistent inward currents through Ca^{++} L-type channels and Na^{+}-inactivating channels in both motoneurons and interneurons.[1,8,20,28] There is some evidence for persistent inward currents operating in human motoneurons after activation of large-diameter afferents by vibration[11,22] or low-level electrical stimulation.[7] Similar effects have also been reported after SCI.[12,26,42,43] After an initial afferent input the motoneurons are induced into a state of sustained depolarization for up to 6 s.[11] It is possible that a similar mechanism can explain our data. If the conditioning vibration causes the activation of persistent inward currents operating on inhibitory interneurons (rather than motoneurons) located in the dorsal horn where the flexor reflex afferents first synapse, the result would be a long-lasting decrease in the excitability of the afferent arm of this flexor reflex pathway. This mechanism may also explain the widespread effects of the vibration on the various muscles studied here. For SCI individuals, this vibration-induced depression of involuntary activity is potentially beneficial to control unwanted muscle spasms.

This study was funded by the NHMRC Neil Hamilton Fairley Fellowship 007148, Australia (J.E.B.); USPHS grant NS-30226 (C.K.T.); and The Miami Project to Cure Paralysis.

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