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M-wave and H-reflex amplitude increases during tetanic stimulation over triceps surae muscles

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Abstract

High-frequency stimulation (100 Hz, 1 ms pulse width) delivered over triceps surae and tibialis anterior muscles (muscle stimulation) can evoke contractions "extra" to those due to stimulating motor axons directly. These extra contractions are thought to arise from central mechanisms (Collins et al., 2001). Stimulation of the tibial nerve in the popliteal fossa (nerve stimulation) produces similar contractions and these are associated with increased amplitudes of H-reflexes, but not of M-waves. The present aim was to deliver muscle stimulation to evoke extra contractions and assess changes in M-wave and H-reflex amplitudes. Muscle stimulation was applied over the right triceps surae muscles (20-100-20 Hz for 2-3-2 s, respectively) while recording isometric plantarflexion torque and surface soleus EMG. In 4/10 subjects the stimulus artifact was sufficiently small to eanlabe an accurate measurement of changes in amplitudes of soleus M-waves, H-reflexes, and torque during 20-Hz stimulation. Data were compared between 1 (1s) and 6 (6s) seconds after the onset of stimulation (i.e.1 s before to 2 s after the 100 Hz burst). For these four subjects, torque increased by 213% (p = 0.01)between 1 and 6s. Amplitudes of M-waves increased by 63% (p = 0.03) and H-reflexes by 249% (p = 0.05) over the same interval. After the 100 Hz burst M-waves were 5-20 times larger than the concurrent H-reflexes. Contrary to tibial nerve stimulation, during muscle stimulation not only H-reflex but also M-wave amplitude increased after the 100 Hz stimulation. The data suggest that different mechanisms contribute to the extra contractions evoked during nerve versus muscle stimulation.

1. INTRODUCTION

High-frequency stimulation (100 Hz) delivered using 1 ms pulse widths over the triceps surae and tibialis anterior muscles (muscle stimulation) can evoke contractions "extra" to those due to stimulating motor axons directly. These extra contractions are thought to arise from mechanisms within the spinal cord(1,2). Such contractions are characterized by a sustained increase in torque after a high-frequency burst when stimulation returns to a lower frequency (see Figure 1). Persons with complete spinal cord injury also exhibit extra contractions, thus these contractions are unlikely to be due to voluntary or descending drive (2,3). Also, contractions sometimes persist after stimulation, and increased cortical excitability does not account for the accompanying motoneuron discharge (4). Moreover, extra contractions are abolished when the tibial nerve is anaesthetically blocked proximal to the stimulation site nerve (2). The results from participants with spinal cord injury, cortical excitability testing, and stimulation during nerve block indicate that the increased torque after high-frequency stimulation is of central origin and at the level of the spinal cord.

High frequency stimulation over the tibial nerve in the popliteal fossa can also generate these extra contractions. We have found that the H-reflex is elevated during the extra force, while the M-wave is unchanged (5). Our goal was to determine whether a similar mechanism accounts for contractions during stimulation through electrodes placed over the muscle. We hypothesized that high frequency muscle stimulation would be associated with an increased H-reflex but not M-wave amplitude during the sustained contractions.

2. METHODS

Ten neurologically-intact persons participated with consent from the Health Research Ethics Board at the University of Alberta. The study was conducted in accordance with the Declaration of Helsinki.

Each subject was seated with the right foot strapped to a footplate configured to record isometric ankle torque. Initially, torque was measured during a maximal voluntary contraction (MVC) of plantarflexors, which was used to normalize torque.

Stimulation was applied over the triceps surae via two flexible electrodes (10- to 18-cm-long × 3.5-cm-wide) positioned ~10 and 20 cm distal to the popliteal fossa. Pulses of 1-ms duration were delivered, via electrodes positioned such that the stimuli produced minimal local discomfort, from a Grass S88 stimulator and a CCU1 constant current unit. Soleus EMG was recorded at 2 kHz with surface electrodes, and signals were amplified ×200-500 and filtered 10-1000 Hz.

Subjects were instructed to relax while extra contractions were evoked by a single train consisting of stimulation at 20-100-20 Hz for 2-3-2 s, respectively. Five stimulation trains, 10 s apart, were included in one trial. The intensity was set to generate torque levels of ~10% MVC after the 100 Hz burst. Extra contractions were said to occur when the torque increased during the 100-Hz burst and remained elevated after returning to the initial stimulation frequency (1-3).

M-wave and H-reflex amplitudes were measured peak-to-peak at set latency-windows after each stimulus, inspected to contain the appropriate responses, and normalized to the first M-wave during the stimulation trial. In 4 subjects, in whom stimulus artifact was sufficiently small to identify EMG responses (in 6 other subjects artifact contaminated M-waves), changes in amplitudes of soleus M-waves, H-reflexes, and torque level during 20-Hz stimulation were compared before to after the 100-Hz burst (1 s vs. 6 s). During the two seconds of 100 Hz stimulation, the rapid artifact contaminated the EMG, therefore H-reflexes or M-waves are not presented during this time. Mean values represent respective responses within 400-ms of time selected for comparison (1 s vs. 6 s). Group data are presented as mean ± SEM. Paired t-tests were used to measure significant differences (α was set at 0.05).

3. RESULTS

In all subjects, torque, H-reflexes and M-waves increased significantly after the 100-Hz burst.

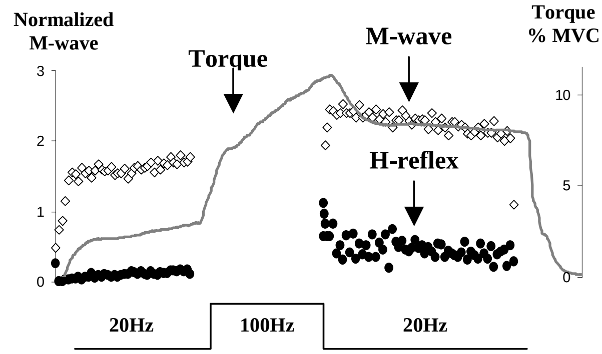

Figure 1. Response of a single subject to a stepwise increase in stimulation frequency.

Torque, H-reflex and M-wave data are shown for a single subject in Figure 1. Torque can be seen to increase sharply when the frequency of stimulation increases to from 20 to 100 Hz. This larger torque persists during the subsequent 20-Hz stimulation and is remains greater than prior to the 100-Hz stimulation burst. Amplitudes of H-reflexes and M-waves both increased after the 100-Hz burst, however, the M-wave was consistently larger than the H-reflex.

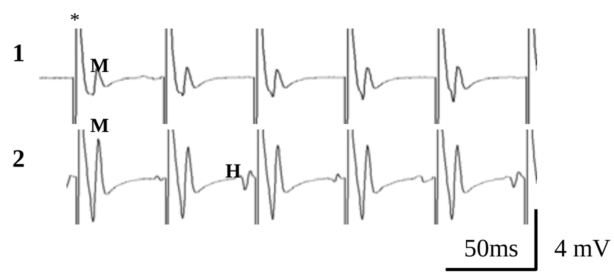

Figure 2. Single subject EMG trace showing artifact (*), M-waves (M) and H-reflex responses (H), before (trace 1) and after (trace 2) the 100 Hz stimulation. EMG data is recorded during 20 Hz muscle stimulation.

Figure 2 shows a single subject EMG trace displaying stimulus artifact, M-waves and H-reflexes during the 20 Hz stimulation, before the 100-Hz burst (trace 1) and after (trace 2). Before the 100-Hz burst there was a small M-wave and no H-reflex. Trace 2, recorded during 20-Hz stimulation *after* the 100-Hz burst, shows an increased M-wave amplitudes and the appearance of small H-reflexes.

Similar results were obtained in all 4 subjects. Group data are shown in Figure 3. When comparing responses during 20-Hz stimulation before to after the 100-Hz burst; M-wave amplitude increased 63% (p=0.03), H-reflex amplitude increased 249% (p=0.05), and torque increased 213% (p=0.01). Across subjects, the increased M-waves were 5-20 times larger than the concurrent H-reflexes.

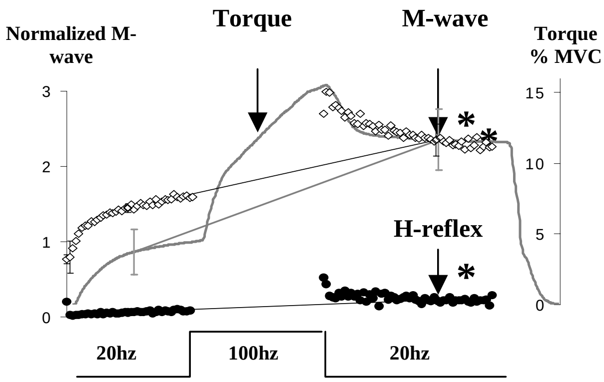

Figure 3. Mean data from four subjects during stepwise increase in stimulation frequency. Asterisks (*) denote $p \leq 0.05$.

4. DISCUSSION AND CONCLUSIONS

Stimulation over the triceps surae muscles generated extra contractions in all 10 subjects. In 4 subjects we were able to discern M-waves and H-reflexes from the stimulus artifact to make accurate measurements of peak to peak amplitudes. In accordance with our hypothesis we observed increased H-reflex amplitude during periods of increased torque after the 100-Hz stimulation. However, contrary to our hypothesis, this was also associated with an increased M-wave amplitude. These results suggest that different mechanisms may contribute to extra muscle contractions evoked during stimulation over the muscle as compared to stimulating the nerve more proximally.

It has previously been hypothesized that these extra contractions are due to central mechanisms, specifically, asynchronous motor units discharging under synaptic recruitment (1,2) These extra contractions have also been demonstrated in subjects with complete spinal cord injury (2,3), and are thus not due to voluntary drive or cortical projections. Furthermore, extra contractions induced by 100 Hz muscle stimulation over triceps surae and tibialis anterior were abolished by blocking the tibial and common peroneal nerve respectively (1), further suggesting that central and not peripheral mechanisms underlie these 'extra' contractions.

Across subjects, the increased M-waves were 5-20 times larger than the concurrent H-reflexes. This is contrary to previously observed outcomes of tibial nerve stimulation where H-reflexes predominate with concomitant small M-waves.

Increased H-reflex values with a stable small M-wave are an indication of the excitability of the spinal motoneuron pool (6). However, since both M wave and H-reflex values increased for our subjects, it is not possible to exclude the possibility of peripheral mechanisms.

The increased M-wave amplitude suggests that peripheral changes may account for a portion of the "extra" contraction during stimulation through electrodes over the muscle. However, this is at odds with previous experiments showing the absence of extra contractions during nerve block proximal to the stimulation site. This discrepancy remains to be resolved. However, the present experiments show that during muscle stimulation M-waves tend to be are much larger than H reflexes. In contrast, during nerve stimulation H-reflexes predominate. Thus, different mechanisms may mediate contractions evoked by these two types of stimulation.

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