Increased H-reflexes boost muscle contractions during tetanic stimulation of tibial nerves in neurologically-intact humans

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Abstract

Tetanic neuromuscular electrical stimulation (at 100 Hz) over ankle dorsiflexors and plantarflexors in persons with and without spinal cord injury can evoke contractions, which are considered spinal of origin, extra to those due to stimulating motor axons directly. Presently, we determined whether H-reflexes contribute to "extra" plantarflexion contractions, evoked in relaxed neurologically-intact persons by stimulating the tibial nerve at just above motor threshold: at 20 Hz for 2 s – 100 Hz (burst) for 2 s – 20 Hz for 3 s (1-ms pulses). Extra contractions were generated if isometric ankle torque was larger after the 100-Hz burst (at 6 s) than before it (at 1 s). The peak-to-peak amplitude of each soleus H-reflex and M-wave was quantified during 20-Hz stimulation, and mean amplitudes were also compared at 1 s and 6 s (average of responses within 400 ms of selected time). Overall (N=6), subsequent to the first H-reflex (~50% M_{max}), reflexes were typically depressed during the initial 20-Hz stimulation. From before to after the 100-Hz burst, the mean H-reflex increased from 12 to 21 % M_{max} ($P<0.05$) and the corresponding mean torque increased from 4 to 10 % MVC ($P<0.01$), while the mean M-wave of 2 % M_{max} was unchanged. We conclude that increased spinal reflex responses contribute to the extra plantarflexion. Since motoneurons are reflexively recruited, presumably in a normal physiological order, the implication is that such contractions may be more fatigue resistant than those due to directly stimulating motor axons.

1. INTRODUCTION

Tetanic neuromuscular electrical stimulation (at 100 Hz) over ankle dorsiflexors and plantarflexors can induce atypically strong contractions that are considered "extra" to those due to directly stimulating motor axons [1-3]. Extra contractions are characterized by larger torque generation after a 100-Hz stimulation burst even as the stimulation returns to lower frequency (see torque in Figure 1). Such contractions are likely spinal of origin: these can be evoked at intensities below motor threshold [1], and often continue after stimulation ceases by self-sustained motoneuron discharge [1-3] that is unaccounted by changes in excitability at cortical and muscular levels [4]. Extra contractions are absent during anaesthetic nerve block proximal to the stimulation site [1,2], yet are present in persons with spinal cord injury (SCI) [4]. Since extra contractions involve spinal motoneurons, which are synaptically recruited probably in a normal physiological order, the implication is such contractions may be more fatigue resistant than those due to stimulating motor axons directly [1-3].

Although considered spinal of origin, extra contractions were presumed not to be due to conventional spinal reflex responses [1-3]. H-reflexes are well known to be mostly depressed during tetanic stimulation [4-7], probably due to reduced neurotransmitter release from previously activated Ia afferents [7]. During stimulation at 10 Hz, for instance, the amplitude of repetitive H-reflexes is typically depressed to less than 10% of the initial response [6]; at 25-100 Hz H-reflexes are absent [5]. Instead of H-reflexes, extra contractions were proposed to be due to asynchronous motoneuron discharge (i.e., not time-locked to the stimulus) [1-3], similar to that demonstrated during low-intensity tetanic nerve stimulation [5]. However, a recovery of H-reflexes, the like of which has been recently demonstrated during tetanic stimulation of tibial nerve afferents [4], could potentially account for extra contractions.

Our present aim was to induce extra plantarflexion contractions by stimulating the tibial nerve to determine whether –after a 100-Hz stimulation burst– the amplitude of repetitively evoked H-reflexes will increase, independent of M-waves, and thereby contribute to such extra contractions.

2. METHODS

Six neurologically-intact persons participated with consent of the Health Research Ethics Board at the University of Alberta.

Each subject was seated with the right foot strapped to a footplate configured to record isometric ankle plantarflexion torque. Initially, torque was measured during a maximal voluntary contraction (MVC) and a maximal peak-to-peak M-wave (M_{max}) was obtained.

The right tibial nerve was stimulated in the popliteal fossa via bipolar surface electrodes connected to 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 10-1,000 Hz.

Extra contractions were evoked by stimulation trains of 20 Hz for 2 s – 100 Hz (burst) for 2 s – 20 Hz for 3 s (1-ms pulses). Five such 20-100-20-Hz stimulation trains, 10 s apart, were included in a trial. Subjects were instructed to relax during the stimulation period. Stimulation intensity was set at motor threshold. Extra contractions were determined to occur if the mean torque, normalized to MVC value, was significantly larger after the 100-Hz burst (at 6 s) than before it (at 1 s) [1].

The amplitude of each M-wave and H-reflex was measured peak-to-peak and normalized to M_{max} (during 20-Hz only, not 100-Hz because of the stimulus artifact). Responses within 400-ms of the selected times (1 s and 6 s) were averaged for comparison. Data are reported as mean (SD). Group data are plotted as mean ± SEM. Paired t-tests were used to determine significant differences for group data (α=0.05).

3. RESULTS

Plantarflexion torque, soleus H-reflexes and M-waves are shown in Figure 1 during 20-100-20-Hz stimulation of the tibial nerve for an individual subject (average of 5 trains). EMG activity is shown at onset of 20-Hz stimulation in panel B and after the 100-Hz burst in panel C. The first H-reflex was ~40% M_{max} and subsequent responses were immediately depressed by ~95% during the initial 20-Hz stimulation. From before to after the 100-Hz burst, the mean H-reflex increased from 2 (1) to 29 (5) % M_{max} while the mean M-wave decreased from 4 (0.4) to 1 (0.3) % M_{max}. The corresponding mean plantarflexion torque increased from 2 (0.1) to 13 (1) % MVC.

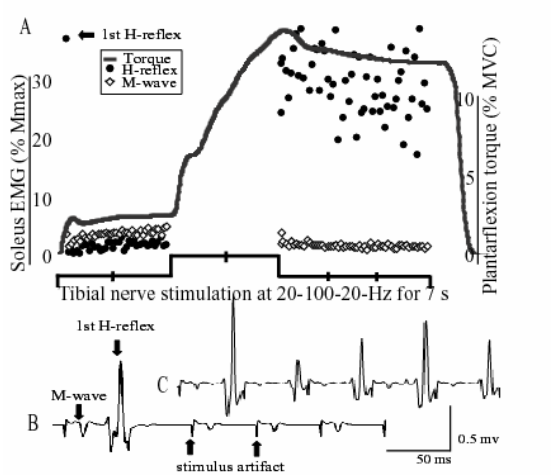

Figure 1. Results for an individual subject. EMG activity during initial 20-Hz (C) and after a 100-Hz burst (D).

In all 6 subjects, mean torque increased ($P<0.001$) after the 100-Hz burst, as did the mean H-reflex ($P<0.05$) in 5 of 6 subjects ($P=0.07$ in the other). Group data are shown in Figure 2: the mean first H-reflex was ~50% M_{max} (range 34-78%) and consecutive responses were immediately depressed by 85% during the first half second. From before to after the 100-Hz burst (from 1 s to 6 s) the mean H-reflex increased 74% from 12 (12) to 21 (12) % M_{max} ($P<0.05$), while the mean M-wave of 2 (1) % M_{max} was unchanged throughout the stimulation ($P=0.76$). The corresponding mean torque increased by 147% from 4 (2) to 10 (3) % MVC ($P<0.01$). Although the mean M-wave was significantly increased ($P<0.01$) after the 100-Hz burst in 4 of 6 subjects, the amplitude remained relatively small (across subjects 6-40 times smaller than the H-reflex after the 100-Hz burst).

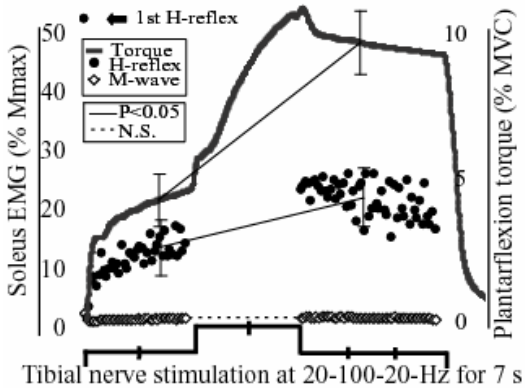

Figure 2. Group (N=6) results. Vertical lines indicate mean ± SEM; solid sloping lines indicate significant changes from before to after the 100-Hz stimulation burst.

4. DISCUSSION AND CONCLUSIONS

Extra plantarflexion contractions were presently evoked by tetanic stimulation of the tibial nerve, similar to those evoked by neuromuscular stimulation through electrodes over lower-limb muscles [1-3]. Our findings support the proposal that such contractions are of spinal origin because the size of the evoked spinal reflex responses increased, in the absence of consistent peripheral changes as indicated by M-waves, and the size of the mean H-reflex was an order of magnitude larger than the M-wave (across subjects 6-40 times larger). The hypothesis that extra contractions are solely due to asynchronous motoneuron discharge [1,2], however, is not supported by the findings of EMG activity mostly synchronized to each stimulus pulse. An intact H-reflex pathway is probably necessary for this plantarflexion to develop since extra contractions were previously demonstrated to be absent during anaesthetic block of the tibial nerve proximal to the stimulation site [1,2].

Although the spinal reflex likely contributed to the extra contractions (since more spinal motoneurons are reflexively recruited) other factors may have additionally contributed. M-waves increased in some subjects, but these changes were inconsistent and the responses were relatively small. Also, as often demonstrated in earlier studies [1-4], self-sustained EMG activity after the stimulation ceases was occasionally evident (not shown), and may have elevated the background EMG during the present stimulation period. Indeed, on one particular occasion with much self-sustained activity, changes in either M-waves or H-reflexes were observed not to account for small yet significant elevation in torque after the 100-Hz burst (unpublished observation). More typically, however, as presently demonstrated extra plantarflexion contractions were accompanied by increased amplitude of H-reflexes, while the M-waves remained small.

At least three mechanisms can underlie the increased size of spinal reflex responses: (1) Post-tetanic potentiation (PTP), in which case larger single H-reflex responses are due to increased neurotransmitter release [8], yet the presently used 2-s 100-Hz burst is shorter than the typical stimulation periods for PTP to develop (tens of second to minutes); (2) Reduction of presynaptic inhibition is well known to affect the size of the H-reflex [9] and may have had presently contributed; (3) Activation of plateau potentials in spinal motoneurons (intrinsically sustained membrane depolarization) has been proposed to underlie the extra contractions [1-3], a mechanism that may potentially amplify the afferent input.

Mechanism(s) notwithstanding, repetitively evoked spinal reflex responses are larger after a 100-Hz burst, and thereby contribute to plantarflexion contractions. Since reflexive activation of motoneurons probably proceeds in a physiological recruitment order that preferentially involves low-threshold fatigue-resistant motor units [4,9], such afferent stimulation may have implications for reducing fatigability of electrically-evoked contractions, given the tendency for recruitment order reversal occurring with stimulating motor axons. Such an intermittent high-frequency stimulation paradigm can potentially harness the persistently large H-reflexes during tetanic stimulation in persons with chronic SCI [6].

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