H-reflexes contribute to electrically-evoked contractions of the plantarflexors in individuals who have had a spinal cord injury

Clair JM, Lagerquist O & Collins DF

Human Neurophysiology Laboratory, Faculty of Physical Education and Recreation

Centre for Neuroscience, University of Alberta, Edmonton, Alberta, CANADA.

Introduction

Neuromuscular electrical stimulation delivered using wide (1ms) pulse widths (WP-NMES) enhances the activation of sensory axons and can generate contractions via reflex pathways through the spinal cord in able-bodied subjects.[1,2]

In able-bodied subjects; a) during WP-NMES at 20Hz the first H-reflex is often large and the second reflex is depressed by up to 90% (post-activation depression) and then reflex amplitudes "recover" but remain depressed by ~70%[2] and b) after a 2s "burst" of 100 Hz stimulation, this recovery is increased by a further ~ 20%[3].

There is evidence that the same occurs in people who have had a spinal cord injury (SCI)[4], although this remains to be confirmed by recording electromyographic (EMG) activity during WP-NMES in this group of individuals.

Purpose

Determine the extent to which WP-NMES generates contractions via reflex pathways after SCI.

Quantify the depression and recovery of H-reflexes during continuous constant-frequency and "burst-like" patterns of WP-NMES after SCI.

Hypothesis

After the first H-reflex, reflex amplitudes will be depressed but will recover and this recovery will be enhanced after a burst of 100Hz WP-NMES.

Methods

9 participants with SCI (4 complete, 5 incomplete; 10 months – 10 yrs post-injury).

Seated, leg supported at the ankle (90°) and the knee (110°).

Tibial nerve stimulated in the popliteal fossa (1 ms pulse width), EMG recorded from soleus, participants remained relaxed.

Patterns: constant frequency (15 or 20Hz for 12s), burst-like (15/20-100-15/20Hz for 4, 4, 4s). Participants received 3 trains of each stimulation pattern.

Stimulation intensity: M-wave = 10-15% of the maximal M-wave (M_{max}).

M-waves and H-reflexes were measured peak-to-peak and normalised to $\%M_{max}$.

Data were "binned" separately for each stimulation pattern according to the amplitude of the first response, the second response and then over 0.5 s bins for the remainder of the stimulation train (see Figure 1). Four measures were made from group data; response depression and 3 measures of recovery (Figure 1).

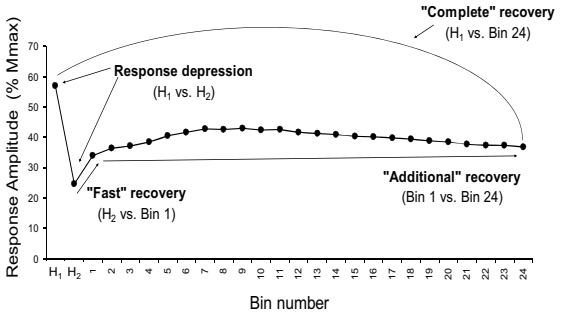

Figure 1. Schematic illustrating the method used to quantify reflex depression and recovery during 12 s trains of stimulation. The initial depression was assessed by comparing the first (H_1) and second (H_2) reflexes. "Fast" recovery was assessed by comparing the second reflex and Bin 1 (responses averaged over the first 0.5 s). "Additional" recovery, after the first 0.5 s of stimulation, was assessed by comparing Bin 24 (responses averaged over the last 0.5 s) to Bin 1. "Complete" recovery was assessed by comparing Bin 24 to the first reflex.

Results

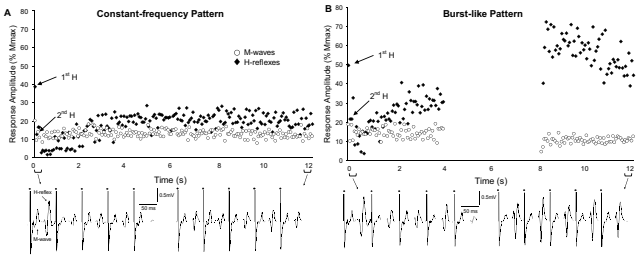

Figure 2. M-waves and H-reflexes from a single participant with incomplete SCI (C_5-C_6). Panel A: 15 Hz for 12 s. Panel B: 15-100-15 Hz for 4 s each phase. The amplitudes of each soleus M-wave and H-reflex generated during NMES at 15 Hz are shown by the open circles and filled diamonds, respectively. Soleus EMG recorded at the beginning and end of each stimulation train is shown at the bottom of each panel (filled circles denote stimulation artefacts).

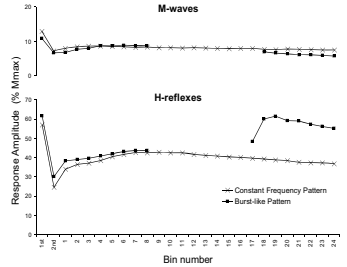

Figure 3. Group average M-wave (upper panel) and H-reflex amplitudes (lower panel) throughout the entire stimulation period for the constant-frequency and burst-like patterns. The mean of the first and second responses are shown, followed by the mean of responses averaged over 0.5 s bins. Error bars have been omitted for clarity.

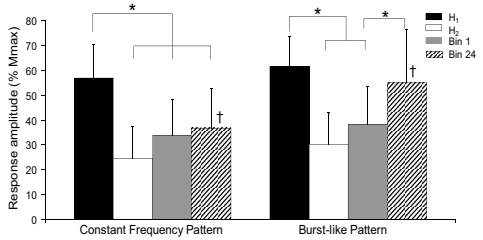

Figure 4. The effect of stimulation pattern and time on H-reflex amplitudes across the group. Four time points are shown for each stimulation pattern (H_1, H_2, Bin 1, and Bin 24). Error bars denote 1 SE. Columns marked by asterisks were significantly different from each other. Columns marked by crosses were significantly different from each other.

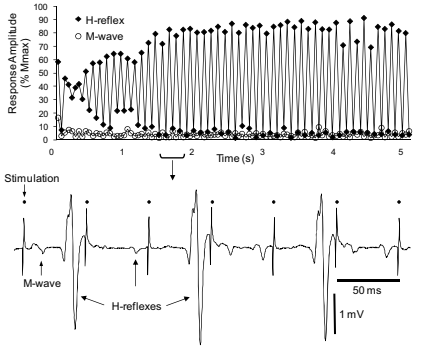

Figure 5. Data from a single participant with an incomplete SCI (C_4-C_5) recorded during a 12 s train of 20 Hz stimulation. For clarity only the first 5 s of data are shown. These data illustrate the strong alternation of reflex amplitudes that was observed in some participants. The upper panel shows M-waves and H-reflexes evoked by each stimulus pulse and the lower panel shows the EMG responses to the 30th to 35th stimulation pulses (filled circles denote stimulation artefacts).

Conclusions

Contractions were not generated solely by the activation of motor axons, instead, H-reflexes were large (~30-60% M_{max}) and contributed to contractions evoked by WP-NMES after SCI.

Reflexes were initially depressed and did not recover significantly during constant frequency stimulation but recovered completely following a burst of 100 Hz stimulation, effectively reversing post-activation depression.

These results provide insight into how WP-NMES generates contractions after an SCI. Using relatively wide pulse widths and incorporating periods of stimulation at 100 Hz may enhance the synaptic recruitment of low threshold motor units, this may help reduce disuse atrophy and generate more fatigue resistant contractions for rehabilitation (Figure 6) .

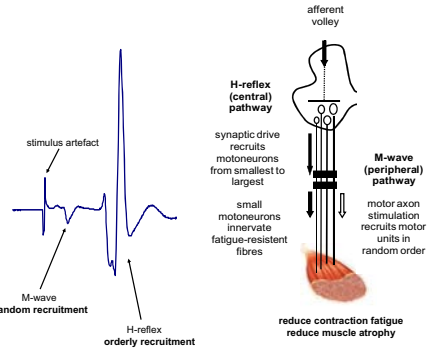

Figure 6. Schematic outlining the potential benefits of driving contractions through reflex pathways for rehabilitation after an SCI.

References

1. Bergquist et al. *Eur J Appl Physiol* 2011; 111:2409-2426.
2. Clair JM et al. *J. Neurophysiol.* 2011; 106: 184-192.
3. Nickolls P, Collins DF, Gorman RB, Burke D & Gandevia SC *Brain.* 2004; 127: 660-670.
4. Klackowicz PM, Baldwin ERL & Collins DF *J. Neurophysiol.* 2006; 96: 1293-1302.

Acknowledgements

Alejandro Ley for technical support.