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).

Results
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).

Conclusions

References

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