

# THE EFFECT OF LUMBOSACRAL MANIPULATION ON CORTICOSPINAL AND SPINAL REFLEX EXCITABILITY ON ASYMPTOMATIC PARTICIPANTS

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## ABSTRACT

**Objective:** The aim of the study was to examine the effects of a high-velocity, low-amplitude (HVLA) manipulation to the lumbosacral joint on corticospinal excitability, as measured by motor evoked potentials (MEPs) using transcranial magnetic stimulation, and spinal reflex excitability, as measured by the Hoffman reflex (H-reflex).

**Methods:** In a randomized, controlled, crossover design, 14 asymptomatic volunteers (mean age, 23 ± 5.4 years; 10 men; 4 women) were measured for MEPs and H-reflexes immediately before and after a randomly allocated intervention. The interventions consisted of HVLA applied bilaterally to the lumbosacral joint and a control intervention. Participants returned a week later, and the same procedures were performed using the other intervention. Data for H-reflex and MEP amplitudes were normalized to the M-wave maximum amplitude and analyzed using 2-way analysis of variance with repeated measures.

**Results:** A significant interaction of treatment by time was found for MEP ( $F_{1,13} = 4.87$ ,  $P = .04$ ), and post hoc analyses showed that the MEP/M-wave maximum ratio decreased significantly in the HVLA treatment ( $P = .02$ ; effect size, 0.68). For H-reflex, there was a significant effect of time ( $F_{1,13} = 8.186$ ,  $P = .01$ ) and treatment and time interaction ( $F_{1,13} = 9.05$ ,  $P = .01$ ), with post hoc analyses showing that H-reflexes were significantly reduced after the HVLA manipulation ( $P = .004$ ; effect size, 0.94). There were no significant changes in MEP latency or silent period duration.

**Conclusion:** An HVLA manipulation applied to the lumbosacral joint produced a significant decrease in corticospinal and spinal reflex excitability, and no significant change occurred after the control intervention. The changes in H-reflexes were larger than those in MEPs, suggesting a greater degree of inhibition at the level of the spinal cord. (*J Manipulative Physiol Ther* 2012;35:86-93)

**Key Indexing Terms:** Manipulation; Spinal; H-Reflex; Motor Evoked Potentials

**H**igh-velocity, low-amplitude (HVLA) thrust manipulation is a manual technique commonly used for the treatment of back and neck pain by manual therapy practitioners, such as chiropractors and osteopaths.<sup>1-3</sup>

In addition to reducing pain, HVLA has been proposed to increase range of motion and produce neurologic changes to influence muscle relaxation, proprioception, and motor control.<sup>4</sup> Spinal manipulation is often accompanied with an audible pop or cavitation, but the importance of this noise to clinical effects has been disputed.<sup>5-7</sup>

The effect of HVLA techniques on muscle relaxation and motor control is still unclear. Some authors have reported reductions in lumbar paraspinal muscle electromyography (EMG) during the flexion-relaxation phase of trunk flexion,<sup>8</sup> whereas others have reported greater contraction amplitudes after HVLA manipulation.<sup>9-11</sup> A few studies have shown transient (100-400 milliseconds) reflex EMG responses from paraspinal muscles after lumbar HVLA manipulation,<sup>12,13</sup> but the clinical relevance of these responses is doubtful. Several studies have reported reduction of "abnormal" muscle activity after HVLA, but many of these studies have had shortcomings such as lack of controls and blinding and poorly described methods, results, and EMG data.<sup>14</sup> It is possible that HVLA may affect motor recruitment in different ways depending on the symptoms and functional impairment of the

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participants, but the effects of HVLA on EMG and motor control require further research.

Transcranial magnetic stimulation (TMS) and the Hoffman reflex (H-reflex) are well-established, noninvasive, painless, and safe techniques to assess the central nervous system. Transcranial magnetic stimulation measures the physiology of the nervous system pathway between the brain (motor cortex) and targeted muscle by creating a motor evoked potential (MEP) that is measured from the target muscle,<sup>15</sup> whereas H-reflexes measure the reflex pathways in the spinal cord projecting to the target muscle.<sup>16</sup> Thus, MEPs may reveal changes to the excitability of the motor cortex after HVLA, whereas H-reflexes reveal changes to the excitability of corticospinal and spinal cord neurons.

Dishman et al<sup>17,18</sup> have previously reported that HVLA applied at the lumbar spine produces a significant decrease in alpha-motor neuron excitability as measured by the H-reflex. The attenuation of the reflex was reported to be transient, with values returning to baseline measures 30 seconds after the intervention. However, these studies did not use a no-treatment control group, and the findings have been disputed by Suter et al,<sup>19</sup> who attributed changes to this reflex to movement artifact during the treatment procedure.

Very few researchers have examined the changes in motor cortex excitability after HVLA manipulation. In 1 of 2 studies, Dishman et al<sup>20</sup> examined the effect of a lumbosacral HVLA technique on MEPs measured in the gastrocnemius muscle in 24 healthy subjects. They reported a significant facilitation of MEPs compared with baseline for the HVLA group but not the control; however, the facilitation returned to baseline levels 5 minutes after the intervention. In a later study, Dishman et al<sup>21</sup> examined MEPs from the lumbar erector spinae muscles of asymptomatic participants after lumbar HVLA and also reported a transient increase in MEPs. It is not clear how transient facilitation of the motor cortex may play a role in a therapeutic effect attributable to HVLA, although Dishman et al<sup>20</sup> speculated that HVLA may provide proprioceptive feedback signal to the central nervous system to stabilize the gain of the motoneuron pool.

Only a few researchers have examined the excitability of corticospinal and spinal cord neurons in response to HVLA manipulation, and none have examined the effects of using MEPs and H-reflexes in the same subjects. The present study aimed to examine both central motor and alpha-motor excitability in response to HVLA manipulation using the measurement of both MEPs and H-reflexes compared with a control intervention.

## METHODS

### Participants

Participants were recruited from the student population at Victoria University using posters placed around the

university campuses. Healthy participants of either sex without current low back pain, aged between 18 and 50 years old, were included. Participants were excluded from this study if they were currently having lower back pain or exhibited signs of radiculopathy or peripheral neuropathy during a screening examination. Calculation of sample size was conducted using an a priori power analysis that suggested that a within-participant research design sample size of 14 participants would result in statistical power greater than 0.80 at an  $\alpha$  level of .05 and an effect size of 0.6.<sup>22</sup> The procedures for this study were approved by the University Human Research Ethics Committee. Volunteers discussed the project with the researchers and signed a consent form before participating.

### Study Design

The study was a controlled crossover design where participants underwent both the experimental and control interventions, tested 1 week apart. The order of the treatment intervention was randomized.

### Measures

**Electromyography Recordings.** Surface EMG activity was recorded from the lateral head of the right gastrocnemius muscle using bipolar Ag-AgCl electrodes. The electrodes were placed 2 cm apart over the belly of the muscle, with the third reference electrode (ground electrode) placed over the bony prominence at the fibula head. The area of electrode placement was prepared by shaving (if required) and cleaned with 70% isopropyl alcohol. Electromyography signals were amplified (2-10 kHz), bandpass filtered (13-1000 kHz), digitized (2 kHz), recorded for 500 milliseconds, and analyzed off-line (Nihon-Koden, Tokyo, Japan).

**M-Wave and H-reflex Recordings.** M-wave and H-reflex testing followed the methodological considerations discussed by Palmieri et al.<sup>16</sup> A 2-cm bipolar stimulating electrode (Nihon Koden) with the cathode and anode was placed over the posterior tibial nerve in the popliteal fossa while the participant remained relaxed (Fig 1). Hoffman reflex/M-wave recruitment curves were completed to identify stimulation intensities to illicit maximal amplitude H-reflex and M-wave.<sup>16</sup> Stimuli of 1-millisecond pulse width were delivered at a frequency of 0.1 Hz, starting at 0.2 mA. Once maximal amplitude H-reflex was identified, stimulation intensity increased until M-wave saturation. After a brief pause, 10 stimuli were delivered at the identified amplitude for maximal H-reflex, and a further 10 stimuli were delivered at 20% above the stimulation intensity identified for maximal M-wave amplitude to ensure that M-wave maximum (M-max) was achieved.<sup>16</sup>

**Transcranial Magnetic Stimulation.** Maximal voluntary contraction (MVC) EMG was recorded to determine background muscle activity during the TMS protocol. The participant



**Fig 1.** Measurement of H-reflex.



**Fig 2.** Measurement of MEP.

was instructed to dorsiflex the ball of the foot, with the investigator providing resistance, as forcefully as possible for 3 seconds. The standard criteria for measurement of MVCs were fulfilled and included a period of familiarization and verbal encouragement provided by the investigators and the rejection of the trial in case the participant felt that it was not a maximal effort.<sup>23</sup>

Transcranial magnetic stimulation testing followed previously established protocols.<sup>24-27</sup> Motor evoked potentials were evoked by TMS of the contralateral motor cortical area projecting to the right gastrocnemius using a Magstim 2002 stimulator (Magstim Co, Whitland, Wales, UK), with a 90-mm circular coil placed tangential to the skull in an anteroposterior direction (Fig 2). For reliability of coil placement, participants wore a snugly fitting cap, positioned with reference to the nasion-inion and interaural lines.<sup>24,25</sup> The cap was marked with sites at 1-cm spacing in a latitude-longitude matrix to ensure reliable coil position throughout the testing protocol and for repeated testing sessions over the period of the study. The cap was checked



**Fig 3.** High-velocity, low-amplitude thrust technique directed to the lumbosacral joint.

constantly to ensure that no changes in cap position occurred. Sites near the estimated center of the right gastrocnemius area (2-4 cm anterior to the vertex) were explored to determine the site at which the largest MEP amplitude was observed. This site was defined as the “optimal” site. At the optimal site, active motor threshold (AMT) was determined by delivering 1 set of 4 stimuli at intensities (5% of stimulator output steps) from a level below the participant’s AMT until the MEP amplitude became saturated (ie, until the amplitude did not increase with increased stimulation). *Active motor threshold* was defined as the intensity at which an MEP could be obtained in 50% of MEP sweeps during 10% of MVC EMG.<sup>4</sup> For main studies, 10 stimuli were delivered during a controlled, low-level voluntary contraction of the gastrocnemius muscle at 10% ( $\pm 3\%$ ) of MVC root mean square EMG.<sup>24,25</sup> Each stimulus was delivered in random intervals between 3 and 5 seconds to avoid stimulus anticipation, and 30-second rest was provided between each set of 5 stimuli to reduce the possibility of muscular fatigue.

### Procedures

Participants were randomly allocated an HVLA or control intervention by lottery draw. Preintervention MEPs and H-reflexes were measured as described above. Participants then received an application of the appropriate treatment intervention, performed in the lateral recumbent position, and then returned to the prone position for a postintervention measure of H-reflex and MEP. Participants then returned 1 week later, and the same procedures were performed using the other treatment intervention. The treatment interventions were performed by an experienced osteopath (GF) who was blinded to the MEP and H-reflex measures. The interventions consisted of the following:

**High-Velocity, Low-Amplitude Manipulation.** An HVLA manipulation directed at the L5/S1 was administered bilaterally



**Table 1.** Evoked potentials data (mean ± SD) from control and HVLA conditions, before and after time points

	MEPs				M-wave/H-reflex					
	MEP Latency (ms)		MEP/M-max ratio (%)		SP duration (ms)		M-wave Latency (ms)		H-reflex/M-max ratio (%)	
	Before	After	Before	After	Before	After	Before	After	Before	After
Control	29.94 ± 1.32	29.29 ± 1.15	8.94 ± 10.58	19.88 ± 11.57	91.66 ± 14.81	9.62 ± 17.28	7.70 ± 2.35	7.80 ± 2.42	30.34 ± 19.77	29.42 ± 24.30
HVLA	29.03 ± 1.35	29.21 ± 1.67	23.90 ± 11.62	21.15* ± 10.03	98.21 ± 19.57	99.20 ± 15.92	8.48 ± 1.59	8.60 ± 1.65	27.86 ± 18.00	17.32* ± 9.92

\* Significant difference ( $P < .05$ ).

with the participant in a lateral recumbent position (Fig 3).<sup>28,29</sup> The participant's upper hip and knee were slightly flexed, and the lower left leg was straight. The upper body was rotated to the right until localization of motion was palpated at the level of L5/S1. The osteopath then manually contacted the tissues overlying the zygapophyseal joint, reinforcing both the lower and upper body rotations. Once tissue tension was maximized, a rotational HVLA thrust was applied. If an audible cavitation (pop) was not produced, an additional attempt was made.

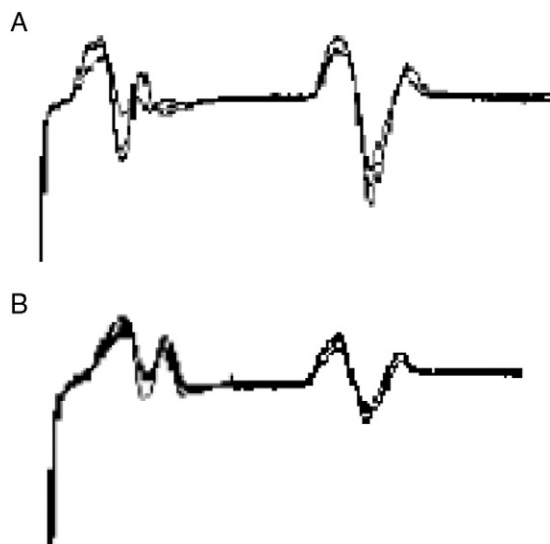
**Control Group.** The osteopath assisted the participant into a lateral recumbent position; however, no truncal torque or manual contact with the spine was applied. The participant remained in this position for approximately 45 seconds each side, the same duration as for the application of HVLA.

### Statistical Analyses

All waveforms collected (H-reflex, M-wave, and MEPs;  $n = 10$  each) were displayed and averaged online for visual inspection as well as stored off-line for further analysis. Hoffman reflex, M-max, and MEP waveforms were quantified by measuring the peak-to-trough amplitude on the EMG. Motor evoked potential latency and silent period (SP) duration were quantified by measuring the stimulus artifact to onset of MEP and from the onset of MEP to the return of EMG, respectively.<sup>30,31</sup> Data were analyzed using SPSS version 18 (SPSS, Chicago, IL). To allow for intraparticipant group comparisons, data for H-reflex and MEP amplitudes were normalized by expressing the H-reflex and MEP as a percentage of the M-max amplitude.

All data were first screened to ensure that they were normally distributed. No variable's  $z$  score of skew or kurtosis was excessive. Furthermore, Shapiro-Wilk (SW) tests suggested that all variables' latency, MEP/M-max percentage, and SP duration for pre- and postmanipulation and control conditions were normally distributed. Hoffman reflex/M-max percentage for precontrol and postmanipulation was also normally distributed; however, H-reflex/M-max for postcontrol (SW = 0.84,  $df = 14$ ,  $P = .02$ ) and premanipulation (SW = 0.80,  $df = 14$ ,  $P = .06$ ) was apparently nonnormal. After visual examination of frequency histograms and detrended Q-Q plots, for these 2 conditions that did not show excessive skewness, it was not considered sufficient to warrant a more conservative analytic strategy. Consequently, it was decided to treat all data as essentially normal in distribution.

To test the hypothesis that HVLA manipulation alters corticospinal measures, data were analyzed using 2-way analysis of variance with repeated measures with  $\alpha$  set at  $P < .05$ . A significant effect for treatment was followed by multiple pairwise comparisons of estimated marginal mean values with Bonferroni adjustments, and effect size calculations using Cohen's  $d$  were performed, where  $d$  is



**Fig 4.** Overlaid M-wave and H-reflex traces from 1 participant pre-HVLA manipulation (A) and post-HVLA manipulation (B).

interpreted as trivial ( $<0.2$ ), small ( $0.21-0.5$ ), medium ( $0.51-0.79$ ), and large ( $>0.8$ ).<sup>32</sup> Data are presented as mean values ( $\pm$ SD).

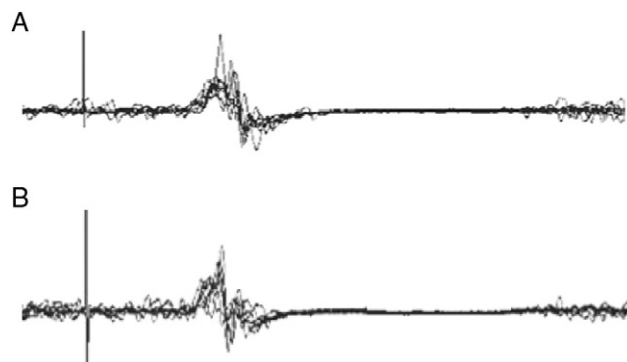
## RESULTS

Fourteen volunteers (mean age,  $23 \pm 5.4$  years; 10 men; 4 women) participated in this study. An audible cavitation was noted in every participant who received the HVLA manipulation. No adverse events were reported by any of the participants.

Data are presented in Table 1 with examples in Figures 4 and 5. Comparisons of MEP latency showed no main (treatment:  $F_{1,13} = 0.33$ ,  $P = .58$ ; time:  $F_{1,13} = 0.02$ ,  $P = .77$ ) or interaction effect ( $F_{1,13} = 2.57$ ,  $P = .14$ ). M-wave latency showed no main (treatment:  $F_{1,13} = 2.79$ ,  $P = .12$ ; time:  $F_{1,13} = 1.41$ ,  $P = .26$ ) or interaction effect ( $F_{1,13} = 0.11$ ,  $P = .92$ ). SP duration showed no difference for main effects of treatment ( $F_{1,13} = 1.89$ ,  $P = .02$ ) or time ( $F_{1,13} = 0.13$ ,  $P = .73$ ) or interaction ( $F_{1,13} = 0.15$ ,  $P = .71$ ).

Comparisons of MEP/M-max ratio and the main effects for treatment and time were nonsignificant (treatment:  $F_{1,13} = 0.62$ ;  $P = .45$ ; time:  $F_{1,13} = 2.09$ ;  $P = .17$ ). The interaction effect of treatment by time was significant ( $F_{1,13} = 4.87$ ,  $P = .04$ ) (Fig 6). Post hoc analyses, with Bonferroni-adjusted  $\alpha$  levels of .025, showed that the MEP/M-max ratio decreased significantly in the HVLA treatment ( $P = .02$ ; effect size, 0.68).

Hoffman reflex/M-max ratio showed no significant main effect for treatment ( $F_{1,13} = 1.95$ ,  $P = .19$ ). The main effect for time was significant ( $F_{1,13} = 8.186$ ,  $P = .01$ ), as was the interaction effect ( $F_{1,13} = 9.05$ ,  $P = .01$ ), with post hoc analyses showing that H-reflex/M-wave ratio significantly reduced after the HVLA manipulation ( $P = .004$ ; effect size, 0.94) (Fig 7).



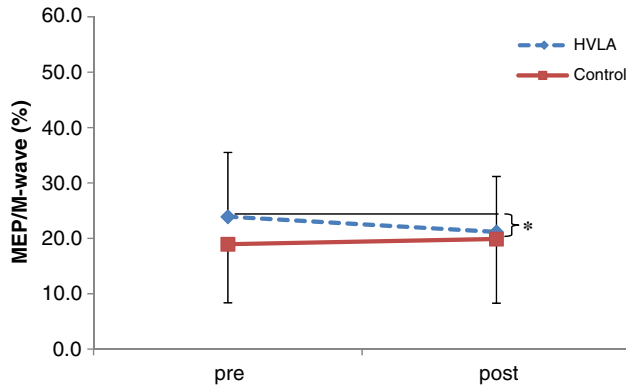
**Fig 5.** Overlaid MEP and SP duration waveform sweeps, from 1 participant, pre-HVLA manipulation (A) and post-HVLA manipulation (B).

## DISCUSSION

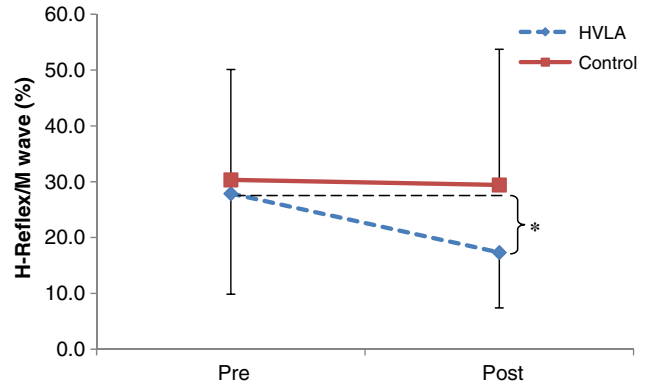
An HVLA technique, applied bilaterally to the L5/S1 segment on asymptomatic participants, was found to produce significant reductions in motor neuron excitability. The reduction in excitability of the motor cortex, as measured by MEPs, was modest and produced medium effect sizes. However, the reduction of activity at the spinal cord level, as measured by H-reflexes, appeared to be substantial, as evidenced by large effect sizes. In contrast, the changes produced in the control group were small and nonsignificant, and the effect sizes calculated were small. These findings suggest that HVLA manipulation produces an immediate inhibition of motor neuron excitability, which is more profound at the level of the spinal cord.

The finding of decreased H-reflexes in the present study was in agreement with the findings of several other studies. In 2 separate studies, Dishman et al<sup>17,18</sup> reported that H-reflex amplitudes were suppressed immediately after a bilateral HVLA manipulation to the L5/S1 segment. In both studies, the authors reported that the inhibition was transient and that amplitudes returned to pre-HVLA values 30 to 60 seconds after treatment. The present study examined H-reflexes using a standard protocol<sup>16</sup> and did not report on the longevity of any changes. Ten stimuli were delivered, and these values were averaged for analysis. Given that participants moved from the lateral recumbent position after HVLA and that intensities for optimal H-reflexes were determined before the stimuli were delivered, it is likely that H-reflexes were recorded approximately 5 minutes after delivery of the HVLA manipulation. Nonetheless, a significant decrease in H-reflex was found in the present study, despite the previous reports that these reflexes return to baseline by 60 seconds.<sup>16,17</sup>

Neither of the studies by Dishman et al<sup>16,17</sup> that investigated H-reflexes used a no-treatment control group, and Suter et al<sup>19</sup> suggested that the reported inhibition of H-reflexes in these studies could be explained by movement artifact, given that the subjects moved from a



**Fig 6.** Motor evoked potential/M-wave ratio pre- and post-intervention for both groups. After the HVLA intervention, there was a significant decrease in MEP ( $P = .02$ ). Asterisk indicates significance.



**Fig 7.** Hoffman reflex/M-wave ratio pre- and postintervention for both groups. After the HVLA intervention, there was a significant decrease in H-reflex ( $P = .004$ ). Asterisk indicates significance.

lateral recumbent position for the manipulation to a prone position for H-reflex measurement. In another study, Dishman et al<sup>33</sup> used 3 groups, an HVLA manipulation, massage, and no-treatment control, and found significant decreases in H-reflex for the HVLA group, but only the HVLA group changed position for measurement to a lateral recumbent position for treatment. In support of the movement artifact explanation, Suter et al<sup>19</sup> reported no change in H-reflexes in a subgroup of 12 participants who remained in a lateral recumbent position for both H-reflex measurement and HVLA manipulation. They did report decreases in the H-reflex in a group of 15 participants with low back pain who remained in the same position as well as subgroup of 5 asymptomatic participants who did change position. The criticisms by Suter et al<sup>19</sup> of the studies by Dishman et al<sup>16,17</sup> cannot be applied to the present study because although participants moved from lateral recumbent to prone positions, the control group underwent the same repositioning and showed no significant changes.

The present study found that MEPs were significantly decreased after the HVLA manipulation. From calculation of effect sizes, the effect of HVLA on MEPs was medium, whereas the effect on H-reflexes was large. Our findings that MEPs were inhibited contrast with the results from 2 studies by Dishman et al.<sup>20,21</sup> Dishman et al examined MEPs from the gastrocnemius<sup>20</sup> and paraspinal muscles<sup>21</sup> and reported transient increases in MEP amplitudes after a bilateral HVLA to the L5/S1 segment. The present study used an accepted protocol to measure and analyze MEPs, whereas the data collection and normalization methods for MEP used by Dishman et al<sup>20</sup> were dissimilar to previously established protocols.<sup>24-27</sup> Dishman et al<sup>20</sup> measured MEPs at 20-second intervals for 120 seconds immediately after manipulation and then at 5 and 10 minutes after manipulation and reported a significant increase immediately after intervention (20-120 seconds) but not at 5 or 10 minutes. In the currently study, MEP was

measured after the measurement of H-reflexes; therefore, MEPs were recorded approximately 10 minutes after the HVLA manipulation. However, even without the measurement of H-reflexes, it would not be possible to measure MEPs within 2 minutes of HVLA intervention using the current protocol. This protocol involved checking that the cap was correctly positioned and determining AMTs and MEP amplitude saturation before MEP measurement. Nonetheless, significant decreased MEPs were found at this period. It may have been possible that an undetected transient increase in MEPs occurred. Transient increases were reported by Dishman et al,<sup>20</sup> but these researchers reported only MEP amplitudes rather than MEP normalized to M-waves, and the increases in MEP amplitude were only in the order of 0.2 mV. In contrast to the Dishman et al study,<sup>20</sup> the present study used previously established protocols<sup>24-27</sup> for the measurement and report of MEPs, and the decreases are consistent with the findings of an unpublished data by the authors of the present study that examined the effect of HVLA of the cervical spine.

As in the studies by Dishman et al<sup>17,18,20,33</sup> and Suter et al,<sup>19</sup> a bilateral HVLA manipulation was performed to the L5/S1 segment in the present study. Unfortunately, there is no objective measure for the success or accuracy of a HVLA manipulation. An audible cavitation was noted for every manipulation in the present study, whereas this outcome was not reported in previous studies, but the audible cavitation may not be a good indicator of clinical benefit.<sup>5-7</sup> In addition, lumbar manipulation may not always be accurate for the target joint, although multiple cavitations typically occur with this procedure, making cavitation likely to occur at the target joint.<sup>34</sup> Although cavitation of the target joint cannot be confirmed, the study tested an HVLA manipulation procedure that aimed to cavitate the L5/S1 joints, and it was likely that this joint cavitated in most participants. Some of the participants

were students who were familiar with the HVLA technique, and this could be seen as a potential source of bias. However, the neurophysiologic measures used in this study are not under voluntary control and could not be influenced by the participants, so naivety to treatment was not required.

The clinical implications of decreases in H-reflexes and MEPs are speculative but may be related to decreased motor output to the involved muscles. Various authors in manual therapy have proposed that muscles associated with a painful or dysfunctional spinal region have abnormally increased motor tone<sup>35,36</sup> and that manual interventions such as HVLA may reduce this tone.<sup>4,14,37</sup> However, evidence of either increased muscle activity in individuals with spinal dysfunction<sup>35,38</sup> or decreased activity in muscles after HVLA manipulation<sup>14</sup> is inconsistent. Spinal HVLA techniques have been found to improve motor recruitment<sup>10,39</sup> and proprioceptive tasks such as joint repositioning,<sup>40,41</sup> and it is possible that the significant changes in corticospinal excitability represent changes to motor recruitment strategies. Future studies could examine whether changes in MEP after treatment are associated with other measures of motor control, such as electromyographic activation and timing.

### Limitations

The present study only investigated the immediate change in H-reflexes and MEPs, and investigation of the longevity of these changes is warranted. In addition, the subjects in the current study and studies by Dishman et al<sup>20,21</sup> were asymptomatic, so examination of changes in both H-reflexes and MEPs in participants with low back pain is also warranted. These future studies may determine if similar, or possibly greater, changes occur in a symptomatic population. Finally, the current study only investigated the effect of a single HVLA manipulation, which does not represent the eclectic approaches of practitioners in many disciplines of manual therapy disciplines.<sup>1-3</sup> Future research could examine the effect on neurophysiologic measures and clinical outcomes in a pragmatic study, where clinicians apply a variety of techniques based on their clinical judgment for the treatment of patients with spinal pain. The current study was an explanatory trial that suggests that HVLA manipulation produces immediate decreases in motor excitability both centrally and at the spinal cord level. A future pragmatic trial would be more generalizable to clinical practice and may determine whether these neurophysiologic changes are clinically relevant.

### CONCLUSION

An HVLA manipulation applied to the L5/S1 joint produced a significant decrease in central motor and alpha-motor neuron excitability, as measured by MEPs and H-

reflexes. The changes in H-reflexes were larger than those in MEPs, suggesting a greater degree of inhibition at the level of the spinal cord. No significant changes occurred after the control intervention. These changes were in contrast with reports from 2 previous studies, and further investigation of the effect of HVLA on these parameters is warranted.

### FUNDING SOURCES AND POTENTIAL CONFLICTS OF INTEREST

No funding sources or conflicts of interest were reported for this study.

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### REFERENCES

1. Fryer G, Johnson JC, Fossum C. The use of spinal and sacroiliac joint procedures within the British osteopathic profession. Part 2: treatment. *Int J Osteopath Med* 2010;13: 152-9.
2. Walker BF, French SD, Grant W, Green S. Combined chiropractic interventions for low-back pain. *Cochrane Database Syst Rev* 2010:CD005427.
3. Rubinstein SM, van Middelkoop M, Assendelft WJ, de Boer MR, van Tulder MW. Spinal manipulative therapy for chronic low-back pain: an update of a Cochrane review. *Spine* 2011; 36:E825-46.
4. Evans DW. Mechanisms and effects of spinal high-velocity, low-amplitude thrust manipulation: previous theories. *J Manipulative Physiol Ther* 2002;25:251-62.
5. Bialosky JE, Bishop MD, Robinson ME, George SZ. The relationship of the audible pop to hypoalgesia associated with high-velocity, low-amplitude thrust manipulation: a secondary analysis of an experimental study in pain-free participants. *J Manipulative Physiol Ther* Feb 2010;33:117-24.
6. Sillevs R, Cleland J. Immediate effects of the audible pop from a thoracic spine thrust manipulation on the autonomic nervous system and pain: a secondary analysis of a randomized clinical trial. *J Manipulative Physiol Ther* 2011; 34:37-45.
7. Evans DW, Lucas N. What is 'manipulation'? A reappraisal. *Man Ther* 2010;15:286-91.
8. Bicalho E, Setti JA, Macagnan J, Cano JL, Manfira EF. Immediate effects of a high-velocity spine manipulation in paraspinal muscles activity of nonspecific chronic low-back pain subjects. *Man Ther* 2010;15:469-75.
9. Suter E, McMorland G, Herzog W, Bray R. Decrease in quadriceps inhibition after sacroiliac joint manipulation in patients with anterior knee pain. *J Manipulative Physiol Ther* 1999;22:149-53.
10. Grindstaff TL, Hertel J, Beazell JR, Magrum EM, Ingersoll CD. Effects of lumbopelvic joint manipulation on quadriceps activation and strength in healthy individuals. *Man Ther* Aug 2009;14:415-20.



11. Suter E, McMorland G, Herzog W, Bray R. Conservative lower back treatment reduces inhibition in knee-extensor muscles: a randomized controlled trial. *J Manipulative Physiol Ther* 2000;23:76-80.
12. Herzog W, Conway PJ, Zhang YT, Gal J, Guimaraes ACS. Reflex responses associated with manipulative treatments on the thoracic spine: a pilot study. *J Manipulative Physiol Ther* 1995;18:233-6.
13. Herzog W, Scheele D, Conway PJ. Electromyographic responses of back and limb muscles associated with spinal manipulative therapy. *J Manipulative Physiol Ther* 1999;24:146-53.
14. Fryer G, Morris T, Gibbons P. Paraspinal muscles and intervertebral dysfunction. Part 2. *J Manipulative Physiol Ther* 2004;27:348-57.
15. Hallett M. Transcranial magnetic stimulation and the human brain. *Nature* 2000;406:147-50.
16. Palmieri RM, Ingersoll CD, Hoffman MA. The Hoffmann reflex: methodologic considerations and applications for use in sports medicine and athletic training research. *J Athl Train* 2004;39:268-77.
17. Dishman JD, Bulbulian R. Spinal reflex attenuation associated with spinal manipulation. *Spine* 2000;25:2519-25.
18. Dishman JD, Cunningham BM, Burke J. Comparison of tibial nerve H-reflex excitability after cervical and lumbar spine manipulation. *J Manipulative Physiol Ther* 2002;25:318-25.
19. Suter E, McMorland G, Herzog W. Short-term effects of spinal manipulation on H-reflex amplitude in healthy and symptomatic subjects. *J Manipulative Physiol Ther* 2005;28:667-72.
20. Dishman JD, Ball KA, Burke J. Central motor excitability changes after spinal manipulation: a transcranial magnetic stimulation study. *J Manipulative Physiol Ther* 2002;25:1-9.
21. Dishman JD, Greco DS, Burke JR. Motor-evoked potentials recorded from lumbar erector spinae muscles: a study of corticospinal excitability changes associated with spinal manipulation. *J Manipulative Physiol Ther* 2008;31:258-70.
22. Faul F, Erdfelder E, Lang AG, Buchner A. G\*Power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behav Res Methods* 2007;39:175-91.
23. Gandevia SC. Spinal and supraspinal factors in human muscle fatigue. *Physiol Rev* 2001;81:1725-89.
24. Pearce AJ, Thickbroom GW, Byrnes ML, Mastaglia FL. The corticomotor representation of elite racquet sport athletes. *Exp Brain Res* 2000;130:238-43.
25. Wilson SA, Thickbroom GW, Mastaglia FL. Topography of excitatory and inhibitory muscle responses evoked by transcranial magnetic stimulation in the human motor cortex. *Neurosci Lett* 1993;154:52-6.
26. Hortobágyi T, Howatson G, Taylor M, et al. Contraction-specificity of ipsilateral motor cortical (M1) responses to transcranial magnetic brain stimulation in humans. *Brain Stimul* 2008;1:242-3.
27. Byrnes ML, Thickbroom GW, Phillips BA, Wilson SA, Mastaglia FL. Physiological studies of the corticomotor projection to the hand after subcortical stroke. *Clin Neurophysiol* 1999;110:487-98.
28. Greenman PE. Principles of manual medicine. 3rd ed. Philadelphia: Lippincott William & Wilkins; 2003.
29. Gibbons P, Tehan P. Manipulation of the spine, thorax and pelvis. An osteopathic perspective. 2nd ed. London: Churchill Livingstone; 2006.
30. Pearce AJ, Kidgell DJ. Corticomotor excitability during precision motor tasks. *J Sci Med Sport* 2009;12:280-3.
31. Wilson SA, Lockwood RJ, Thickbroom GW, Mastaglia FL. The muscle silent period following transcranial magnetic cortical stimulation. *J Neurol Sci* 1993;114:216-22.
32. Cohen J. Statistical power analysis for the behavioral sciences. Hillsdale, NJ: Erlbaum; 1988.
33. Dishman JD, Bulbulian R. Comparison of effects of spinal manipulation and massage on motoneuron excitability. *Electromyogr Clin Neurophysiol* 2001;41:97-106.
34. Ross JK, Bereznick DE, McGill SM. Determining cavitation location during lumbar and thoracic spinal manipulation: is spinal manipulation accurate and specific? *Spine* 2004;29:1452-7.
35. Fryer G, Morris T, Gibbons P. Paraspinal muscles and intervertebral dysfunction. Part 1. *J Manipulative Physiol Ther* 2004;27:267-74.
36. Gatterman MI. Foundations of chiropractic: subluxation. 2nd ed. St Louis, MO: Elsevier Mosby; 2005.
37. Pickar JG. Neurophysiological effects of spinal manipulation. *Spine J* 2002;2:357-71.
38. Fryer G, Bird M, Robbins B, Johnson J. Resting electromyographic activity of deep thoracic transversospinalis muscles identified as abnormal with palpation. *J Am Osteopath Assoc* 2010;110:61-8.
39. Nogueira de Almeida BS, Sabatino JH, Giraldo PC. Effects of high-velocity, low-amplitude spinal manipulation on strength and the basal tonus of female pelvic floor muscles. *J Manipulative Physiol Ther* 2010;33:109-16.
40. Palmgren PJ, Sandstrom PJ, Lundqvist FJ, Heikkila H. Improvement after chiropractic care in cervicocephalic kinesthetic sensibility and subjective pain intensity in patients with nontraumatic chronic neck pain. *J Manipulative Physiol Ther* 2006;29:100-6.
41. Rogers RG. The effects of spinal manipulation on cervical kinesthesia in patients with chronic neck pain: a pilot study. *J Manipulative Physiol Ther* 1997;20:80-5.