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First Prize

Central Motor Excitability Changes After Spinal Manipulation: A Transcranial Magnetic Stimulation Study

J. Donald Dishman, DC,[a] Kevin A. Ball, PhD,[b] and Jeanmarie Burke, PhD[b]

ABSTRACT

Background: The physiologic mechanism by which spinal manipulation may reduce pain and muscular spasm is not fully understood. One such mechanistic theory proposed is that spinal manipulation may intervene in the cycle of pain and spasm by affecting the resting excitability of the motoneuron pool in the spinal cord. Previous data from our laboratory indicate that spinal manipulation leads to attenuation of the excitability of the motor neuron pool when assessed by means of peripheral nerve Ia-afferent stimulation (Hoffmann reflex).

Objective: The purpose of this study was to determine the effects of lumbar spinal manipulation on the excitability of the motor neuron pool as assessed by means of transcranial magnetic stimulation.

Methods: Motor-evoked potentials were recorded subsequent to transcranial magnetic stimulation. The motor-evoked potential peak-to-peak amplitudes in the right gastrocnemius muscle of healthy voulunteers (n = 24) were measured before and after homolateral L5-S1 spinal manipulation (experimental group) or side-posture positioning with no manipulative thrust applied (control group). Immediately after the group-specific procedure, and again at 5 and 10 minutes after the procedure, 10 motor-evoked potential responses were measured at a rate of 0.05 Hz. An optical tracking system (OptoTRAK, Northern Digital Inc, Waterloo, Canada [<0.10 mm root-mean-square]) was used to monitor the 3-dimensional (3-D) position and orientation of the transcranial magnetic stimulation coil, in real time, for each trial.

Results: The amplitudes of the motor-evoked potentials were significantly facilitated from 20 to 60 seconds relative to the prebaseline value after L5-S1 spinal manipulation, without a concomitant change after the positioning (control) procedure.

Conclusions: When motor neuron pool excitability is measured directly by central corticospinal activation with transcranial magnetic stimulation techniques, a transient but significant facilitation occurs as a consequence of spinal manipulation. Thus, a basic neurophysiologic response to spinal manipulation is central motor facilitation. (J Manipulative Physiol Ther 2002; 25:1–9)

Key Indexing Terms: Chiropractic; Transcranial Magnetic Stimulation; Electromyography.

INTRODUCTION

The use of spinal manipulative therapy (SMT) in the management of patients with low back pain is on the increase. Despite this increase in utilization and acceptance, the mechanism(s) by which SMT may reduce pain and muscular spasms is not fully understood. SMT has been postulated to relieve mechanical nerve compression at dorsal and ventral rami.[1] SMT may produce a global inhibitory response by means of integration of afferent feedback along the entire neuraxis, leading to a hypoalgesic effect.[2] Conversely, SMT may produce an inhibitory reflex response that is segmental in origin.[3] Regardless of the exact mechanisms, the clinical efficacy of SMT may involve a decrease of motoneuron activity, which, in turn, may lead to a reduction of hypertonicity.[4]

One basic physiologic response to SMT is a transient decrease in motoneuron activity as assessed by the Hoffmann reflex (H-reflex) technique.[4-6] The H-reflex technique involves peripheral stimulation of the Ia-afferent feedback pathway to assess the excitability of the alpha motoneuron pool. However, these H-reflex findings of motoneuron inhibition appear to be in frank contrast to other investigations in which SMT has been reported to produce a reflexive activation of paraspinal musculature.[7,8] In addition, mechanical strain of the ligament-muscular system of the spine evokes reflex activation of paraspinal muscles in the feline.[9,10]

[a]Associate Professor, Department of Anatomy and Department of Research, New York Chiropractic College, Seneca Falls, NY.
[b]Department of Research, New York Chiropractic College, Seneca Falls, NY.

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Submit reprint requests to: J. Donald Dishman, DC, MSc, Associate Professor, Department of Anatomy, 2360 SR 89, Seneca Falls, NY 13148 (e-mail: ddishman@nycc.edu).

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Although these research findings appear paradoxical with respect to understanding the effects of SMT on the human motor system, reflex activation of paraspinal muscles may trigger the subsequent reduction of motoneuron activity. There is one known case report in the literature to support this hypothesis. Reflex activation of SMT-targeted thoracic musculature led to a subsequent alleviation of hypertonicity in one symptomatic patient with thoracic back spasms.[8]

Mechanistically, SMT is equivalent to rapidly applying a mechanical strain to the trunk. Depression of Ia-motoneuron synapse after a previous activation of the stretch reflex arc is a well-documented neurophysiologic phenomenon known as postactivation–depression.[11] Although postactivation–depression appears to be limited to the fibers activated by the conditioning procedure,[11] there is sufficient evidence to suggest heteronymous inhibition of motoneurons by altering Ia-afferent discharge rates from postural, synergistic, and antagonistic muscles.[12-19] Our previously reported H-reflex data suggest that a SMT procedure may produce a heteronymous inhibition of the triceps surae motoneuron by means of either presynaptic or postsynaptic mechanisms. Moreover, the transient effects of SMT on motoneuron activity occur with a similar time course as other heteronymous conditioning effects reported in the literature.[13,20]

However, the H-reflex response is highly susceptible to presynaptic inhibition of Ia afferents that mediate the reflex.[21,22] If SMT produces inhibition of Ia-afferent fibers by means of stimulation of presynaptic inhibitory interneurons, the decrease in the amplitude of the H-reflex response may be independent of changes in the excitability of the alpha motoneuron pool. In essence, GABA-ergic interneurons synapsing directly with the presynaptic terminals of Ia-afferent fibers are capable of decreasing the amplitude of the H-reflex response.[15,21]

Similar to the vibration paradox,[23,24] the recruitment gain of the motoneuron pool may, in fact, be increased as a result of SMT, even though simultaneously there is SMT-induced H-reflex inhibition. Specifically, preferential activation of the Ia afferents by muscle vibration initiates impulses concurrently in spinal interneuronal pathways to increase the excitability of the alpha motoneuron by means of a postsynaptic mechanism, referred to as the tonic vibration reflex, and inhibits the tendon and H-reflex responses by means of a presynaptic inhibitory pathway.[24-27] The modulation of these spinal interneuronal pathways are set by supraspinal influences, such as corticoreticulospinal and vestibulospinal tracts.[28-32]

To assess the recruitment gain of the alpha motoneuron pool, an electrophysiologic technique that activates only postsynaptic mechanisms is preferable, because modulation of the H-reflex response is dependent on the summation of presynaptic and postsynaptic mechanisms on the Ia-alpha motoneuron synapse. Presynaptic inhibition does not influence corticospinal inputs to the alpha motoneuron pool.[33] Transcranial magnetic stimulation (TMS) is a noninvasive research tool to study effects of corticospinal inputs on alpha motoneuron pool excitability. The compound muscle action potentials elicited by TMS are referred to as motor-evoked potentials (MEPs). The amplitude of the MEP elicited in the target muscle by TMS will reflect changes in excitability of corticomotoneuronal (CM) cells and changes in the excitability of the alpha motoneuron pool. Although magnetic brainstem stimulation[33,34] is needed to differentiate between the effects of TMS on cortical excitability and motoneuron pool excitability, an accurate assessment of overall central motor system excitability can be obtained before and after SMT with the TMS technique. To date, there are no reports regarding the effect of SMT on the excitability of the central motor system.

The purpose of this study was to determine the effects of lumbar SMT on the central motor system by using TMS to activate the gastrocnemius muscle (GM). To meet the objective of this research, MEPs from the right GM were measured before and after a homolateral L5-S1 spinal manipulation (experimental group, $n = 12$) or a side-posture positioning with no manipulative thrust applied (control group, $n = 12$).

METHODS

Participants

The subjects were 24 healthy volunteers recruited from a college student population. Subjects were counterbalanced into either the L5-S1 SMT group (25.1 $\pm$ 3.96 years; 177.5 $\pm$ 8.11 cm; 79.6 $\pm$ 14.09 kg) or the control group (27.3 $\pm$ 3.80 years; 171.8 $\pm$ 6.74 cm; 78.8 $\pm$ 20.31 kg). All subjects were neurologically screened by one clinician before initiating the experiments to exclude subjects with radiculopathy or peripheral neuropathy. The local ethics committee reviewed and approved all experimental procedures. All subjects signed an informed consent form.

Experimental Design

MEPs evoked by TMS were recorded from the right GM. The amplitude of the MEP is easily quantified by measuring the peak-to-peak electromyographic (EMG) response from the test muscle. MEP amplitudes in the GM were measured before and after a L5-S1 SMT procedure. In the control group, MEP amplitudes in the GM were measured before and after side-posture positioning. The SMT procedures were delivered homolateral to the recording limb (right side). One clinician, with 15 years of experience, performed the L5-S1 SMT procedure.

TMS Techniques

TMS was performed by using a Dantec MagPro magnetic stimulator with a 9-cm circular coil (Dantec Medical, Skovlunde, Denmark). The TMS coil was clamped to a rigid, bent-angle apparatus that allowed the TMS coil to be stabilized relative to the head. The TMS coil was centered over the vertex with a tangential orientation. Coil current flow direction was clockwise. A TMS pulse intensity of 100% stimulator output was used to determine the effects of SMT on MEP amplitudes. MEPs from the GM were recorded with bipolar, surface aluminum-aluminum chloride

electrodes (15 mm × 20 mm) (Medicotest, Ølstykke, Denmark). The ground electrode was 10 mm in diameter (Conmed, Inc, Utica, NY). The Braddom and Johnson[35] method of electrode configuration was used to ensure consistent placement of recording electrodes over the GM across subjects. The EMG signal was bandpass filtered (100 Hz-1 kHz) and amplified by using the Grass P511 amplifier system (Grass Instruments, W Warwick, RI).

Experimental Protocol

Subjects were positioned supine on a treatment table. The ankles were positioned at angles of 90°. At the beginning of the experimental session, the TMS pulse intensity was set to 60% of maximum output. Stimulus intensity was increased in 10% increments on successive trials until stimulus intensity reached 100% of stimulator output. At 100% of stimulator output, the TMS coil was repositioned by hand until a MEP was recorded with a maximal amplitude and a minimal latency. During this procedure to determine the optimal stimulation site, a 3-dimensional (3D) optical tracking system was used to record the precise spatial location of the TMS coil relative to the subject. The optimal stimulation site was achieved when MEPs were deemed consistent in amplitude and latency on 3 consecutive trials. Real time 3-D monitoring of the TMS coil relative to the subject ensured that the coil remained properly oriented at the optimal stimulation site on all trials during the experiment.

Motor threshold is defined as the lowest stimulus intensity required to produce a MEP of at least 50 μV amplitude in at least 5 of 10 consecutive trials.[36] Although we did not systematically determine motor thresholds in this research, a MEP of at least 50 μV amplitude was not detected until the stimulus intensity level reached 80% to 90% of maximum output in 21 of 24 subjects. The other 3 subjects had a MEP of at least 50 μV amplitude at 60% to 70% of stimulator output. We were unable to evoke a MEP of at least 50 μV amplitude in 8 recruited subjects. These subthreshold subjects were excluded from the research project. At 100% of stimulator output, the majority of subjects were being tested with an estimated stimulus intensity of 10% to 20% above motor threshold.

After determining the optimal stimulation site, 10 MEPs were recorded as baseline values. The stimulation rate for evoking baseline MEPs was 0.05 Hz. Immediately after the spinal manipulation procedure or side posture positioning maneuver was performed, MEPs were measured at 20-second intervals within the first 120 seconds to determine the acute time course of postmanipulation effects on central motor pathways. Ten MEPs were also recorded at 5 and 10 minutes after manipulation at a stimulation rate of 0.05 Hz.

MEPs were recorded by using an analog-to-digital converter (12-bit resolution) interfaced to a computer. The sampling rate was 5 kHz per channel. MEP amplitudes were measured on-line and stored in a data output file for statistical analyses. LabView software (National Instruments Corp, Austin, Tex) was used for data acquisition and data analysis.

L5-S1 SMT Procedures

After collection of the 10 baseline MEPs, a homolateral (right side) L5-S1 side-posture SMT procedure was administered. The L5-S1 SMT procedure was a high-velocity, low-amplitude (HVLA) manipulation, which is commonly performed by practitioners of chiropractic and osteopathy. The force applied to the spine in these procedures is reported to be delivered in approximately 200 ms,[37] with linear vertebral displacements of less than 10 mm.[38] The manual force, or thrusts, to the zygapophyseal joint are applied at the end of physiologic range of joint motion and extend into the so-called "paraphysiologic zone" of joint motion.[39] The paraphysiologic zone is defined as the endpoint range of motion to which a joint can be passively forced without any deleterious effects.[39]

In the L5-S1 SMT procedure, the clinician provided a manual contact on the tissues overlying the zygapophyseal joint. By using the right-handed Cartesian orthogonal coordinate system of movement as a reference,[40] manual tension was slightly increased by providing $+\theta$Y-axis translation (axial distraction) to the spine, coupled with a $\pm\theta$Y-axis rotation force, thereby increasing the mechanical load on the soft tissues. Once tissue tension was maximized, a high-velocity (typically less than 200 ms[37]), low-amplitude impulsive force was applied. The primary force vector applied to the zygapophyseal joint was $+\theta$Z-axis translation, (posterior-anterior) with a secondary vector consisting of $\pm\theta$Y-axis rotation (right or left axial rotation). After the L5-S1 SMT procedure, the subject was immediately returned to the supine testing position to record the MEPs.

Side-Posture Positioning Procedure (Control Group)

The operator assisted the subject into a side posture; however, no lower limb flexion or truncal torque was applied. In an effort to eliminate the effects of the manual application of force and velocity to the zygapophyseal joints, no manual contact was made with the spine. In effect, the positioning procedure was nothing more than modest hand contact to assist the subject into a side posture. The subject was simply returned to the supine testing position immediately after this side-posture, control procedure. Minimal manual handling and positioning of the subject allowed for us to account for nonspecific effects of SMT and mobilization procedures on central motor pathways.[2]

Optical Tracking System

An optical tracking system (OptoTRAK, Northern Digital Inc, Waterloo, Canada [<0.15 mm root-mean-square in x, y, z]) was used to monitor the 3-D position and orientation of the TMS coil, in real time, for each trial.[41,42] Clusters of 4 infrared light-emitting diodes (LEDs) were affixed to both the subject's face (on frontal and maxilla regions) and to the TMS coil to provide these measurements. Procedures of rigid body modeling[43] were used to analyze 6–*df* changes in the position and orientation of each marker cluster.[44] The data were computed in real time, such that the operator could monitor the 6–*df* location of the TMS coil relative to

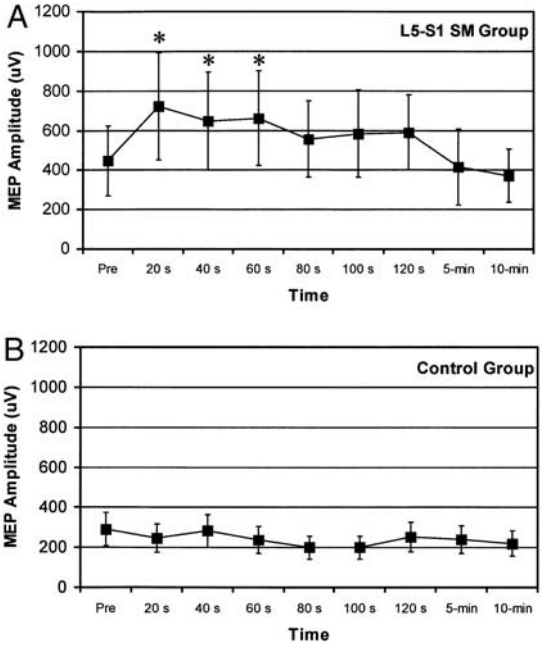

Fig 1. *Mean MEP Amplitudes (± SE) over time. MEP amplitudes at 20- second intervals from 20 to 120 seconds, and at 5 and 10 minutes after either an L5-S1 SMT procedure, **A**, or a side-posture positioning procedure (control), **B**, as compared with baseline values before procedure. Data values at prebaseline and at 5 and 10 minutes after procedure are mean of 10 MEP responses per subject.*

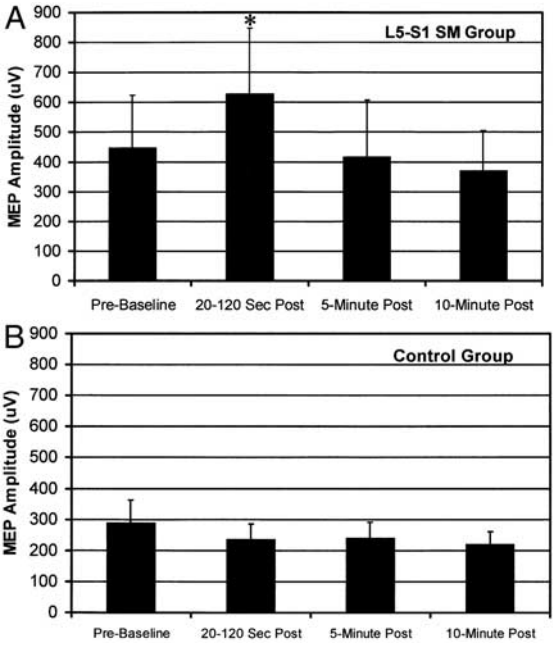

Fig 2. *Mean MEP amplitudes (± SE), collapsed from 20 to 120 seconds after procedure compared with baseline values before procedure and 5 and 10 minutes after procedure in the L5-S1 SM Group, **A**, and control group, **B**.*

the subject. Visual feedback displayed on a computer monitor allowed the operator to manually adjust and hold the TMS coil over the optimal stimulation site on all trials.

Statistical Analysis

A split-plot analysis of covariance model (Group × Time) was performed to reveal the specific effects of the L5-S1 SMT procedure on MEP amplitudes. The covariate was the baseline MEP amplitude. The Dunnett test for a priori contrasts was used to detect any differences in MEP amplitudes between baseline values and postmanipulation time points within each group. The Newman Keuls test for a posteriori pairwise comparisons was used to compare the mean MEP amplitudes among the measurement time periods within each group. The level of significance was .05 for all statistical procedures.

RESULTS

MEP amplitudes were significantly facilitated from 20 to 60 seconds after the L5-S1 SMT procedure as compared with the prebaseline value (Fig 1, *A*; $F\ [8,88] = 3.62$; $P < .05$). The mean of MEP amplitudes evoked immediately after the L5-S1 SMT procedure was significantly greater than the mean MEP amplitudes at 5 and 10 minutes after manipulation as well as being greater than the prebaseline mean (Fig 2, *A*; $F\ [3,33] = 4.44$; $P < .05$). MEP amplitudes did not change after the side-posture positioning procedure (Figures 1, *B* and 2, *B*).

The significant Group × Time interaction terms from the analysis of covariance analyses indicate that the effects of the L5-S1 SMT procedure on MEP responses were independent of positioning the individual into side-posture (Fig 1, $F\ [7,154] = 2.59$; Fig 2, $F\ [2,44] = 4.54$; $P < .05$). The amount of postmanipulation facilitation was not correlated with the amplitude of the prebaseline MEP response ($r = -.20$). Thus, it may be concluded that the experimental treatment effect was independent of the covariate. In summary, the MEP amplitudes measured from 20 to 120 seconds after the L5-S1 SMT procedure were significantly greater than the MEP amplitudes measured in the control group ($F\ [1,21] = 9.83$; $P < .05$).

The 1-way analysis of variance intraclass correlation procedure was used to determine the reliability of the MEP amplitudes during each of the measurement periods for both subject groups. The intraclass reliability coefficients ranged from .89 to .98 to indicate adequate trial-to-trial consistency for MEP amplitudes (Table 1). The 2-dimensional analysis of variance intraclass correlation procedure indicated that the reliability of measuring MEP amplitudes by time periods and trials was .98 and .95 for subjects in the SMT and control groups, respectively. Coefficients of variation were also calculated to determine the variability of MEP amplitudes during each of the measurement periods for both subject groups. The coefficients of variation reported in Table 1 for this research are in agreement with the data on the variability of MEP amplitudes as a function of stimulus intensity.[45]

The 3-D location of the TMS coil over the vertex was optimal for activation of the GM at all measurement time points. Decreases in MEP amplitudes and increases in co-

Table 1. *Intraclass reliability coefficients and coefficients of variation for MEP amplitudes*

	Intraclass reliability coefficients		Coefficients of variation*	
	L5-S1 SMT subjects	Control subjects	L5-S1 SMT subjects	Control subjects
Prebaseline	.97	.94	.38 ± .155	.28 ± .126
Immediately after	.98	.89	.39 ± .325	.42 ± .156
5 minutes after	.99	.90	.38 ± .149	.41 ± .282
10 minutes after	.96	.95	.40 ± .147	.33 ± .192

*In majority of subjects, MEPs were recorded after an estimated TMS pulse intensity of 10% to 20% above motor threshold (see Methods). Coefficients of variation are in agreement with literature for this TMS intensity.[45]

Table 2. *Positional errors (mm) incurred while attempting to maintain desired location of coil relative to each subject*

	Up-down (mm)		Left-right (mm)		In-out (mm)	
Direction*	L5-S1 SMT subjects	Control subjects	L5-S1 SMT subjects	Control subjects	L5-S1 SMT subjects	Control subjects
Prebaseline	0.4 ± 1.5	−0.2 ± 1.0	0.7 ± 1.0	−0.0 ± 0.9	0.5 ± 1.2	0.3 ± 1.4
Immediately after	−0.1 ± 1.8	−0.1 ± 1.7	0.2 ± 2.4	0.9 ± 2.0	0.5 ± 1.6	0.5 ± 1.7
5 minutes after	0.1 ± 2.3	0.1 ± 2.1	−0.5 ± 1.6	1.1 ± 1.8	0.9 ± 1.8	0.5 ± 1.7
10 minutes after	0.1 ± 1.5	0.4 ± 1.4	0.4 ± 1.4	0.5 ± 2.3	0.1 ± 1.5	0.7 ± 1.2

*Directions described with respect to experimenter, who was seated above head of supine subject.

Table 3. *Orientation errors incurred while attempting to maintain desired location of coil relative to each subject*

	Up-down (degrees)		Left-right (degrees)	
Orientation*	L5-S1 SMT subjects	Control subjects	L5-S1 SMT subjects	Control subjects
Prebaseline	−0.1 ± 2.0	0.8 ± 2.2	−0.6 ± 2.7	−0.7 ± 3.3
Immediately after	0.9 ± 3.7	1.7 ± 4.5	0.1 ± 4.8	−0.5 ± 5.5
5 minutes after	−0.4 ± 3.4	−0.8 ± 3.5	−0.6 ± 4.4	0.1 ± 2.6
10 minutes after	−0.6 ± 3.3	−2.4 ± 4.1	0.2 ± 4.0	−1.2 ± 3.3

*Directions are described with respect to experimenter, who was seated above head of supine subject.

efficients of variation are minimal when the TMS coil is within 5 mm of the optimal stimulation site.[46,47] Plots with polar coordinates used to describe the relationship between MEP amplitudes and TMS coil orientation indicate that decreases in MEP amplitudes are minimal within an effective stimulating arc of 45°.[48,49] Although these accuracy data are reported for focal TMS pulses with the figure-of-8–shaped coil, the margins of error for nonfocal TMS pulses with the tangential orientation of the circular coil over the vertex are greater, based on differences in the geometries of coil designs.[50,51]

In the current study, Tables 2 and 3 present the optical monitoring results for coil positioning and orientation, respectively. The results are described from the point of view of the operator, who was seated at the head of the supine subject. Tables 2 and 3 summarize all component errors as means and SDs, for which the former represents measurements of accuracy and the latter represents precision estimates. Table 2 presents the independent 3-D positioning errors that were determined over the 4 measurement periods for both the control and SMT groups. In contrast, Table 3 summarizes angular measurements in only 2 orientations (up-down, left-right). This is the case because the magnetic output of the coil is actually symmetrical about the third (perpendicular) axis. All component errors described in Tables 2 and 3 were normally distributed (Figs 3 and 4).

The TMS coil was positioned over the optimal stimulation site with a worst-case precision of ±2.4 mm (Table 2 [SMT, left-right, immediately after]). Coil orientation was maintained with a precision of no less than ±5.5° (Table 3 [Control, left-right, immediately after]). The mean accuracy in positioning the coil was 1.1 mm or less (Table 2 [Control, left-right, 5 minutes after]) and −2.4° in orientation or less (Table 3 [Control, up-down, 10 minutes after]). For the coil positioning data, there were no significant differences among the measurement periods and between the L5-S1 and control subjects ($P > .05$). In all subjects, there were significantly larger up-down orientation errors at the mea-

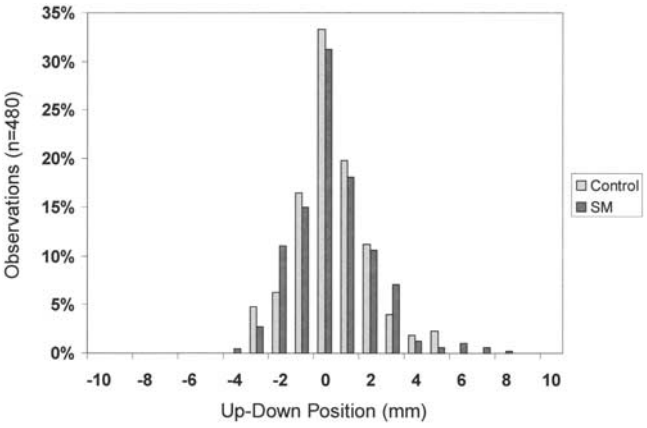

Fig 3. *Histogram demonstrating normative distribution of up-down position data (mm) recorded while maintaining desired location of TMS coil for both control and SM data sets. Figure contains summary of all values measured for all time conditions (N = 480). Similar normative characteristics were returned for left-right and in-out component measures.*

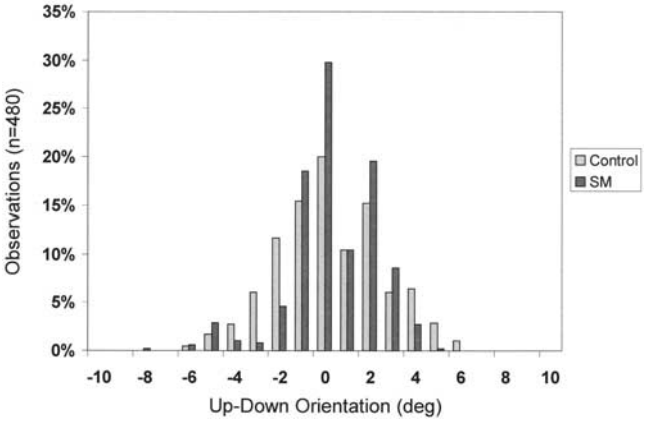

Fig 4. *Histogram demonstrating normative distribution of up-down orientation data (degrees) recorded while maintaining desired location of TMS coil for both control and SM data sets. Figure contains summary of all values measured for all time conditions (N = 480). Similar normative characteristics were returned for left-right component measures.*

surement periods immediately after and at 10 minutes after ($P < .05$). However, the magnitudes of these up-down orientation errors were minimal with respect to confounding the recordings of MEP amplitudes. Thus, it may be concluded that the 3-D location of the TMS coil was optimal for activating the GM throughout the experimental session.

DISCUSSION

The results of this study indicate that lumbar spinal manipulation leads to transient facilitation of MEPs evoked from the GM muscle. The subjects in side-posture exhibited no such facilitation of MEP amplitudes. These data suggest that there is a postsynaptic facilitation of alpha motoneuron, corticomotoneurons, or both that may be specific to the HVLA thrust. This increased excitability of the central motor system may result from a summation of sensory afferent discharges evoked from various spinal and paraspinal tissues as a consequence of HVLA SMT procedures. The current data offer further elucidation of the previously described "paradox" of motor facilitation versus sensory inhibition.

In the current investigation, TMS techniques were used to more directly assess the central motor system rather than relying on the H-reflex technique that uses peripheral nerve stimulation of the Ia afferents. In all of the H-reflex research to date, methodological procedures were used to ensure the consistency of the stimulating and recording environments, before and after the SMT procedure.[4-6] When the stimulating and recording environments are the same before and after a perturbation, the H/M_{Max} ratio is a valid index of changes in motoneuron activity.[52] However, the H-reflex response is highly susceptible to presynaptic inhibition of Ia afferents that mediate the reflex.[21,22] As such, changes in motoneuron activity detected with the H-reflex technique may be from changes in presynaptic mechanisms, changes in postsynaptic mechanisms, or changes in the relative strengths of presynaptic and postsynaptic mechanisms.

The MEP amplitude data in the current investigation clearly indicate that the central motor system is facilitated after SMT. It may be more appropriate, then, to conclude that decreases in H-reflex amplitude after SMT are a result of presynaptic inhibition of peripheral *sensory* fibers, as opposed to attenuation of motoneuron activity. However, it is important to note some physiologic differences between H-reflexes and MEPs to qualify this conclusion. Comparison of H-reflex and MEP responses evoked in limb muscles may be used to delineate differences between presynaptic and postsynaptic mechanisms.[53-55] This presynaptic/postsynaptic comparison assumes that the H-reflex and MEP responses activate the same subpopulations of motoneuron.[55] There is evidence to suggest that the same motoneurons are not recruited in MEP and H-reflex responses even when the sizes of these 2 evoked responses are standardized to the maximum M-wave amplitude.[33,56] There are substantial differences in recruitment thresholds and discharge rates of single motor units participating in MEP and H-reflex responses.[56]

The amplitude of the MEP elicited in the target muscle by TMS will reflect changes in excitability of corticomotoneuronal cells and/or changes in the excitability of the alpha motoneuron pool. Another assumption for delineating the contributions of presynaptic and postsynaptic mechanisms to the physiologic effects of SMT on motoneuron activity is the following: SMT procedures will bias alpha motoneuron pool excitability through spinal interneuronal mechanisms, and the stimulation of proprioceptive afferents by SMT procedures will not bias the excitability of corticomotoneuronal cells through ascending pathways. The discharge probability of single motor units evoked by TMS indicate that activation of excitatory spinal interneuronal pathways contribute to MEP amplitudes.[33,57] However, TMS-evoked facilitation of EMG responses, congruent in time with the long-latency component of the physiologic stretch reflex,

provides evidence that activation of muscle afferents bias motor cortical excitability.[58,59] Future research on the physiologic effects of SMT on the human motor system should address the potential changes in cortical excitability by using magnetic brainstem stimulation.[33,34]

CONCLUSION

In summary, differences in motoneuron recruitment, the activation of interneuronal pathways, temporal summation, and the contamination of EMG activity from other muscles are potential limitations to consider when comparing MEP and H-reflex responses with respect to physiologic mechanisms.[33] Within these physiologic limitations, the paradoxical effects of SMT on H-reflex and MEP responses are best explained by presynaptic gating of peripheral feedback mechanisms that may hinder the stability of the recruitment gain of the motoneuron pool. In this way, central mechanisms are predominately modulating the gain of the motoneuron pool by means of temporal and spatial summation of inhibitory and excitatory postsynaptic potentials from various spinal, supraspinal, and corticospinal pathways, thereby maintaining the physiologic stability of the motoneuron pool. The functional significance of presynaptic inhibition of peripheral afferents and postsynaptic regulation of the gain of the motoneuron pool is evident in numerous motor tasks.[53,60-63]

Clinical conditions involving spasticity and hypertonicity have been attributed to pathophysiologic abnormalities in the modulation of motoneuron activity by presynaptic and postsynaptic interneurons.[64] Similar to pharmacologic interventions, an understanding of the influence of SMT on presynaptic and postsynaptic processes is important to identify pathophysiologic abnormalities that may be corrected by SMT. There are numerous conditioned-reflex protocols that may be used to study presynaptic and postsynaptic responses. Conditioning paradigms that use H-reflex and TMS paired-stimuli techniques are available to study specific segmental responses such as reciprocal inhibition, recurrent inhibition, and presynaptic inhibition, as well as various supraspinal influences on motoneuron activity.[65-68]

This proposed SMT mechanism, involving parallel contributions from presynaptic and postsynaptic mechanisms, still allows for the summation of sensory afferent discharges evoked from various spinal and paraspinal tissues as a consequence of HVLA SMT procedures at second-order synapses throughout the central nervous system. In other words, proprioceptive feedback is still processed by the central nervous system but not at the expense of motoneuron pool stability. It may be speculated that the barrage of sensory discharges evoked by a SMT procedure provides the appropriate proprioceptive feedback signal to the central nervous system, which, in turn, stabilizes the gain of the motoneuron pool. In patients with palpatory muscle spasm and low back pain, 2 weeks of SMT alleviated their clinical symptoms and increased the synaptic efficacy of Ia afferent activation to the central nervous system.[69] Cerebral somatosensory potentials, evoked by magnetic stimulation to the paraspinal muscles, were used as an index of Ia afferent activation.[70] In the clinical study by Zhu et al,[69] the correlation among changes in cerebral potentials and improvements in motor symptoms may suggest a relationship between improved sensory feedback and motoneuron pool stability.

The current data indicate that the central motor system, in total, is facilitated. Conventional TMS techniques alone cannot assist in the determination of the exact level of the facilitation. Does SMT excite the motor cortex, which, in turn, increases the excitability level of the motoneuron pool? Or does SMT perhaps activate spinal interneuron pathways, which then increases the excitability level of the motoneuron pool? Future studies are needed in which MEPs are elicited by magnetic stimulation at various sites along the corticospinal pathway. In this manner, the etiology of the reported facilitation after SMT can be more clearly delineated. By stimulating the motor cortex, brainstem, and spinal cord, localization of the basic physiologic responses to SMT may be obtained. Regardless of the actual site of origin of the central motor facilitation, this study is the first known report of central excitability changes occurring as a consequence of spinal manipulation. In future studies, these basic physiologic responses to SMT will be extended to include the back pain population.

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