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Spinal Reflex Attenuation Associated With Spinal Manipulation

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Study Design. This study evaluated the effect of lumbosacral spinal manipulation with thrust and spinal mobilization without thrust on the excitability of the alpha motoneuronal pool in human subjects without low back pain.

Objectives. To investigate the effect of high velocity, low amplitude thrust, or mobilization without thrust on the excitability of the alpha motoneuron pool, and to elucidate potential mechanisms in which manual procedures may affect back muscle activity.

Summary of Background Data. The physiologic mechanisms of spinal manipulation are largely unknown. It has been proposed that spinal manipulation may reduce back muscle electromyographic activity in patients with low back pain. Although positive outcomes of spinal manipulation intervention for low back pain have been reported in clinical trials, the mechanisms involved in the amelioration of symptoms are unknown.

Methods. In this study, 17 nonpatient human subjects were used to investigate the effect of spinal manipulation and mobilization on the amplitude of the tibial nerve Hoffmann reflex recorded from the gastrocnemius muscle. Reflexes were recorded before and after manual spinal procedures.

Results. Both spinal manipulation with thrust and mobilization without thrust significantly attenuated alpha motoneuronal activity, as measured by the amplitude of the gastrocnemius Hoffmann reflex. This suppression of motoneuronal activity was significant ($P < 0.05$) but transient, with a return to baseline values exhibited 30 seconds after intervention.

Conclusions. Both spinal manipulation with thrust and mobilization without thrust procedures produce a profound but transient attenuation of alpha motoneuronal excitability. These findings substantiate the theory that manual spinal therapy procedures may lead to short-term inhibitory effects on the human motor system. [Key words: back pain, electromyography, H-reflex, mobilization, spinal manipulation] **Spine 2000;25:2519–2525**

The exact mechanism underlying the neurophysiologic effects of spinal manipulation has yet to be determined. Mechanically, spinal manipulation has been postulated to relieve mechanical nerve compression at dorsal and ventral rami.[12] Results of recent investigations suggest that spinal manipulation may produce hypoalgesia by activation of a central control mechanism.[35] Other investigators have postulated that spinal manipulation may produce a stretch reflex from joint capsules that may lead to inhibition of muscle spasm.[22] Thus, although spinal manipulation may lead to a reduction in pain for many patients with low back pain and muscle spasm, the specific mechanism is unknown. Spinal mobilization without a manipulative thrust also is a commonly used conservative technique in the management of patients with low back pain. As with manipulation, the clinical efficacy of mobilization procedures are reported in the literature. However, the physiologic mechanisms are largely unknown.[22,33]

A recently proposed mechanism for the attenuation of pain after spinal manipulation is that the procedure elicits an inhibitory stretch reflex response generated from the capsules of the zygapophysial joints.[22] In support of this neurophysiologic mechanism, Indahl et al,[22] using a porcine model, reported that distension of the zygapophysial joint by injection of physiologic saline reduced the amplitudes of motor unit action potentials recorded from the paraspinal musculature. Specifically, mechanical perturbation, such as spinal manipulation, may initiate afferent discharges from cutaneous receptors, muscle spindles, mechanoreceptors, and free nerve endings in the zygapophysial joint capsule and the ligaments of the spine.[22,34] These afferent discharges may synapse on inhibitory interneurons to inhibit alpha motoneuron pools of the paraspinal musculature.[22]

The data in support of the inhibitory stretch reflex response are equivocal. Recent reports indicate that an excitatory reflexive discharge of paraspinal muscles occurs as a consequence of spinal manipulative therapy.[19] This result has been attributed to a reflexive primary afferent discharge of various receptors, such as joint mechanoreceptors and muscle spindles.[19] Some investigators have reported inhibitory effects on the motoneuron pool as a consequence of spinal manipulation,[9,10,25] whereas others report excitatory effects on the human motor system.[19,31,32] Thus, a paradox has developed in the investigation of the mechanism that spinal manipulation may exert on the excitability of the motoneuron pool. This apparent paradox is further promoted by the fact that most of the individual mechanoreceptors in spinal and paraspinal structures[3,13] produce excitatory discharges when stimulated.[30]

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Currently, no clear consensus exists on whether spinal manipulative therapy evokes an overall excitatory or inhibitory response from motoneurons. At this writing, few reported studies evaluate the effect of lumbar spine manipulative procedures on the excitability of alpha motoneurons.

In the current study, the effect of lumbar spine manipulation on the excitability of the motoneuronal pool was examined in human subjects using the tibial nerve Hoffmann reflex (H-reflex) technique.[20] The tibial nerve H-reflex response provides a neurophysiologic index of alpha motoneuron pool excitability as a consequence of lumbosacral spinal manipulation, as Ia afferents from the triceps surae muscle activate the alpha motoneuron pool of the lumbosacral spine. Thus, the amplitude of the tibial nerve H-reflex response is reduced or enhanced if activation of proprioceptive afferents after lumbosacral spinal manipulation inhibits or excites the alpha motoneuron pool, respectively. Monitoring the amplitude of the tibial nerve H-reflex at various time points after lumbosacral spinal manipulation may provide additional insight concerning the neurophysiologic mechanisms underlying spinal manipulation.

The purpose of this study was to determine the effect of lumbosacral spinal manipulation on the excitability of the motoneuron pool. This information was ascertained by comparing changes in the amplitude of the tibial nerve H-reflex after both spinal manipulation procedures using manual thrusts and spinal mobilization without manual thrusts.

■ Methods

Participants. All experimental procedures described were reviewed and approved by the Institutional Review Board. For this study, 17 volunteers (10 men and 7 women) were recruited from a college student population and assigned to one of two experimental groups: 1) spinal manipulation with thrust (n = 10) or 2) spinal mobilization without thrust (n = 7). Participants ranged in age from 20 to 43 years (average, 28 years) and had no history of peripheral neuropathy or radiculopathy. All the participants were screened neurologically by one clinician before the initiation of the experiments.

Experimental Design. All manipulation and mobilization procedures were performed by one clinician. Group 1 participants received lumbar spinal manipulation with thrust. Group 2 participants were subjected first to spinal mobilization without thrust, and then in a subsequent session (within 1 hour) to lumbar spine manipulation. The general method for data collection in both studies was the same. M-wave and H-reflex responses were recorded from the gastrocnemius muscle. For both procedures, the H/M recruitment curve was performed according to the method of Hugon and determined at the beginning of the experimental session.[20] Before spinal manipulation or mobilization was performed, the amplitude of 10 maximum H-reflexes were recorded as prebaseline values. After the group-specific treatment, maximal H-reflexes were evoked immediately after intervention, then at 5, 10, 15, and 20 minutes after treatment. At the completion of H-reflex testing, the post-treatment maximal M-wave response was recorded.

H-Reflex Methods. The tibial nerve H-reflex general method as described by Hugon was used.[20] The participants were placed prone on a treatment table with the right foot secured lightly to a plate to maintain a 90° angle of the foot to the tibia. The tibial nerve was stimulated in the popliteal fossa using a 0.5 msec square wave pulse delivered by a constant voltage stimulator (Grass S88; Grass Instruments, W. Warwick, RI). The cathode-stimulating electrode (10-mm self-adhesive, pregelled, Ag-AgCl) was positioned in the popliteal fossa at the optimal location for evoking an H-reflex in the gastrocnemius muscle. The optimal location for the cathode was defined as the site in the popliteal fossa at which a slightly suprathreshold stimulus for evoking an H-reflex did not evoke an M-wave response.

The anode-stimulating electrode was placed 10 cm proximally to the cathode on the posterior thigh. The electromyographic (EMG) response of the gastrocnemius muscle was recorded using 10-mm bipolar self-adhesive, pregelled, surface disposable Ag-AgCl electrodes. The Braddom and Johnson method[7] of electrode configuration was used to ensure consistent placement of recording electrodes over the gastrocnemius muscle across subjects. The EMG signal was bandpass filtered (10 Hz to 1 kHz) and amplified using an EMG amplifier system (Grass P511; Grass Instruments). The peak-to-peak EMG values (EMG amplitudes) of M-wave and H-reflex responses evoked in the gastrocnemius muscle were recorded with a digital oscilloscope (Tektronix TDS 420; Tektronix Inc., Beaverton, OR).

Maximal M-Wave and H-Reflex Responses. To assess alpha motoneuron pool excitability of the lumbosacral spine, this study used the tibial nerve H-reflex technique. Electrical stimulation of the tibial nerve at low stimulus intensities preferentially recruits Ia afferents and evokes a monosynaptic reflex response with a latency of 30 msec on the average (H-reflex response). With increasing stimulation intensity, the alpha motoneuron axons are activated, producing a direct muscle response with a latency of 5 ms on the average (M-response). The H/M recruitment curve describes the activation of the Ia afferents (H-reflex amplitude) and alpha motoneuron axons (M-wave amplitude) as a function of stimulus intensity. The maximal M-wave amplitude represents activation of the entire alpha motoneuron pool. The H/M max ratio reflects the proportion of the alpha motoneuron pool recruited by Ia afferents and is used as a functional index of alpha motoneuron pool excitability.[20]

The H/M recruitment curve was generated by increasing stimulus intensity from subthreshold to the maximum in 5-V increments.[20] The maximal M-wave was defined as the plateau in EMG amplitude that occurred in response to three successive 5-V increments of stimulus intensity. To determine the stimulus intensity for evoking maximal H-reflexes, stimulus intensity was increased in 2-V increments within the range of ±5 V from the apex of the H-reflex recruitment curve. This optimal stimulus intensity for evoking maximal H-reflexes was not adjusted for the remainder of the experimental session. The mean EMG amplitudes of 10 maximal H-reflexes were recorded as a pre-intervention baseline.

Spinal Manipulation With Thrust Procedures (Group 1). After collection of 10 prebaseline H-reflexes, participants in group 1 were administered a bilateral L5–S1 spinal manipulation procedure. The spinal manipulative procedures performed consisted of high-velocity, low-amplitude manipulation, as commonly performed by practitioners of chiropractic and osteopathy.[14,18,19] The force applied to the spine in these procedures is reported to be delivered in approximately 200 msec,[18] with linear vertebral displacements less than 10 mm.[14] These procedures consist of "side-posture" lumbar spine manipulation. The manual force, or thrusts, to the zygapophysial joint are applied at the end of the physiologic range of joint motion and extend into the so-called "paraphysiologic zone" of joint motion.[28] The paraphysiologic zone is defined as the end point range of motion, in which a joint can be forced passively without any deleterious effects.[28]

In these procedures, the clinician provided a manual contact on the tissues overlying the lumbar zygapophysial joint. With the right-hand Cartesian orthogonal coordinate system of movement used as a reference,[36] manual tension was increased slightly 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 msec),[18] low-amplitude impulsive force was applied. The primary force vector applied to the zygapophysial joint was $+\theta$Z-axis translation, (posteroanterior) with a secondary vector consisting of $\pm\theta$Y-axis rotation (right or left axial rotation).

On completion of the procedure, the participants were returned immediately to the prone relaxed position for data acquisition. Maximal H-reflexes were evoked 0 to 100 seconds after manipulation in 10-second intervals. The EMG amplitudes of 10 maximal H-reflexes were recorded at 5, 10, 15, and 20 minutes after treatment. The H-reflexes were evoked at a rate of 0.1 Hz.

Mobilization Without Thrust Followed by Spinal Manipulation With Thrust Procedures (Group 2). In the mobilization procedure, used with participants in group 2, manual contact was applied to the joints of the lumbosacral spine. However, no thrust was applied. Except for the manipulative thrust, the mobilization procedures did not differ from the manipulation procedures. Nonforceful manual excursion of the zygapophysial joint to the passive end point range of motion is considered an aspect of therapeutic mobilization procedures. The intention of the mobilization posture was to replicate the manipulation posture accurately except for the application of the high-velocity thrust. On completion of the mobilization procedure, the participants were returned immediately to the prone relaxed position for data acquisition of maximal H-reflexes in accordance with the experimental design.

At the completion of a 20-minute period after the mobilization procedure, the EMG amplitudes of 10 maximal H-reflexes were recorded in the same way as for the spinal manipulation procedures. Within an additional 1 hour after mobilization without thrust, the group 2 participants were administered a bilateral L5–S1 spinal manipulation procedure as performed on group 1 participants to obtain a repeated measures within-group analysis trial. This combination of procedures in group 2 provided for an internal control by which the effects of the two different procedures could be compared in one particular participant.

On completion of the spinal manipulation procedure, the participants were returned immediately to the prone relaxed position for maximal H-reflex recording in accordance with the experimental design. At the completion of a 20-minute period after the spinal manipulation procedure, the EMG amplitude of the maximal M-wave response was recorded.

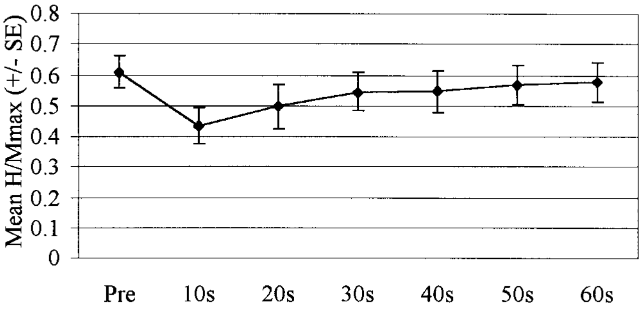

Figure 1. Mean H/M_{max} ± SE over time (n = 10) for group 1. *A priori* contrasts comparing spinal manipulation to all times are shown. Postmanipulation showed significant decreases in H/M_{max} at 10 and 20 seconds after manipulation.

Statistical Analysis. The ratio of H_{max}/M_{max} was used as the dependent variable for both studies, because this ratio allows for a comparison of compound muscle action potentials derived from direct nerve stimulation (M) and those resulting from alpha motoneuronal excitation.[20] Data from the participants in group 1 (n = 10) were submitted to a one-way repeated measures analysis of variance (ANOVA), with *a priori* contrasts that compared all postmanipulation time points to the premanipulation baseline. The H/M_{max} data from group 2 (n = 7) were submitted to a 2 (condition: manipulation/mobilization) × 7 (time) completely within-group ANOVA. A split-plot ANOVA model (treatment × time) for maximal M-wave responses was performed to determine any possible changes in the EMG recording environment from the beginning to end of the experimental session.

■ Results

Group 1

The attenuation of the H-reflex was transient, with a return to baseline values by 30 seconds after manipulation (Figure 1). There was a significant decrease in the H/M_{max} ratio measured 10 seconds after the spinal manipulation ($F[1,9] = 23.51$; $P < 0.01$). The *a priori* contrast comparing 20-second postmanipulation and premanipulation values also was significant ($F[1,9] = 5.71$; $P < 0.05$). Although H/M max values at 30 seconds was depressed with respect to premanipulation values, this decrease failed to achieve traditional levels of significance ($F[1,9] = 4.04$; $P < 0.08$). The H-reflex amplitude remained normal at 5, 10, 15, and 20 minutes after spinal manipulation.

Group 2

There was a significant time main effect ($F[6,36] = 10.92$; $P < 0.01$). The condition main effect and time by condition interaction ($F[1,6] = 1.18$; $P < 0.33$ and $F[6,36] = 1$; $P < 0.443$, respectively) were not significant. These results indicate that although the general time course of H/M_{max} of group 2 is similar to that of group 1, data from the mobilization condition were not appreciably different from that from the spinal manipulation treatment (Figure 2).

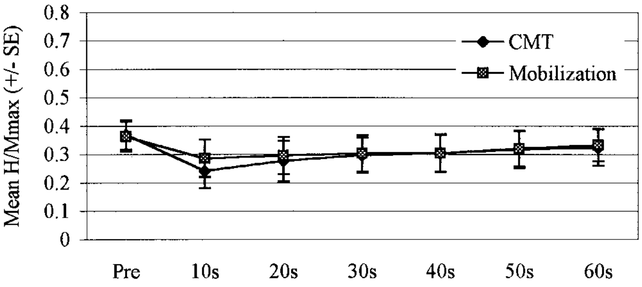

Figure 2. Mean H/M_{max} ± SE over time ($n = 7$) by condition for group 2. The analysis of variance (ANOVA) showed no effects involving condition. However, the time effects were similar to those reported for group 1. *A priori* contrasts showed significant premanipulation *versus* all times postmanipulation (chiropractic manipulative therapy) except at 60 seconds ($P < 0.06$).

In an attempt to replicate the findings of group 1, group 2 data were collapsed across conditions and submitted to a one-way repeated measures ANOVA, with *a priori* contrasts comparing the pre-H/M_{max} data with that from all time points afterward. As with group 1, the analysis showed a significant drop in H/M_{max} immediately (10 seconds) after mobilization/manipulation ($F[1,6] = 23.78$; $P < 0.01$). Furthermore, the effect remained significant at 20, 30, 40, and 50 seconds ($P <$ 0.01, 0.02, 0.03, and 0.05, respectively), and the effect at 60 seconds compared with the precontrast value approached traditional levels of significance ($P < 0.06$). Although a direct comparison cannot be drawn between this data and that from group 1 because the data generated reflect a combination of mobilization and manipulation effects, which may be additive, it is encouraging to note that the overall time course effect can be replicated. Again, the H-reflex amplitude remained normal at 5, 10, 15, and 20 minutes after spinal manipulation.

For both groups, the maximal M-wave responses recorded at the beginning and end of the experimental session were not significantly different. These data indicate that the EMG recording environment was maintained throughout the experimental session. Therefore, the significant decreases in H/M_{max} values observed in the study reflected decreases in alpha motoneuron pool excitability.

■ Discussion

The results of this study indicate that high-velocity, low-amplitude lumbosacral spinal manipulative procedures lead to short-term attenuation of alpha motoneuronal activity, as quantified by H-reflex amplitude changes. Additionally, the reported data suggest that the relative contribution that receptors stimulated by participant body positioning (mobilization) may exert on the excitability of the motoneuron pool is of significance. These data indicate that the combination of body positioning and dynamic thrust application appears to exert a greater inhibitory influence on alpha motoneuronal activity collectively than the procedures performed singularly. The authors therefore suggest that cutaneous receptors, muscle spindles, and joint mechanoreceptors all may contribute individually or in concert to attenuation of alpha motoneurons as a consequence of spinal manipulation and mobilization.

The similarity in magnitude between alpha motoneuron excitability attenuation resulting from manipulation and mobilization was unexpected. This finding suggests that cutaneous receptors, muscle spindles, and Golgi tendon organs rather than velocity-dependent joint mechanoreceptors may contribute to most of the afferent discharge to the inhibitory interneuron pool. As such, the current findings suggest that the velocity and force of the manipulative thrust may be of little significance with respect to reflexive inhibition of the motoneuron pool.

The data from this study appear to be consistent with the findings of previous investigators, who have reported that reflex activation of paraspinal muscles as a sequel to spinal manipulation did not differ when thrusts produced an “audible” response to the manipulative force.[17] The manipulative procedure often yields an “audible” release from the joint. This audible response may represent a release of synovial gases from the zygapophysial joint. However, this phenomenon is of equivocal significance to the effectiveness of the procedure.[8,17] In effect, the current findings support the concept that the force and velocity of the therapeutic thrust may not be the most critical factor in altering motoneuron pool excitability.

The major hypothesis of this investigation is that spinal manipulation reduces the excitability of spinal cord motoneurons. The clinical consequence of motoneuron attenuation is unknown. It is reasonable to propose that a secondary consequence of this reduced excitability is that the so-called “pain–spasm–pain” cycle may be disrupted. The results of this investigation indicate that inhibition of motor neurons is a short-term consequence of spinal manipulative procedures, lasting a maximum of 20 to 30 seconds.

The current findings are in general agreement with those previously reported after sacroiliac joint manipulation.[25] Murphy et al[25] reported findings of motoneuron suppression after sacroiliac manipulation, but their data indicates a longer duration of inhibition than reported in the current study. This difference in duration may be attributable to the discharge properties, quantity, and distribution of mechanoreceptors of the relatively large, less mobile, sacroiliac joint as compared with those of the smaller, more mobile lumbar zygapophysial joints.

It has been reported in the literature that individuals who experience low back pain are likely to have an abnormal increase in EMG activity of the erector spinae muscles.[2,4,5,26,27,29] It has been hypothesized that nociceptive afferent stimuli generated from various spinal and extraspinal tissues produce a facilitation of alpha motoneurons.[26] This facilitation is thought to lead subsequently to a state of hypertonicity or spasm of the innervated back muscles. Thus, the patient with low back pain may have a perpetuation of pain by the so-called "pain–spasm–pain" cycle.[26] Although this concept is controversial and its existence perhaps equivocal, it is plausible that chronic muscle activation or spasm caused by pain predisposes individuals to a perpetuation of cyclic reflex activation.[22]

Recent investigators have reported that various types of stimuli, such as electrical stimulation and distention of the zygapophysial joint capsules with physiologic saline, produce a reduction in the motor unit action potential amplitude of the multifidus and longissimus muscles.[22] In the current investigation, spinal manipulation–induced activation of primary afferent discharges resulted in a similar action (*i.e.,* elicitation of short-term reflex inhibition of alpha motoneuronal excitability).

Although most of the primary afferent discharge generators (mechanoreceptors) are thought to be excitatory, the overall effect produced by the manual therapy procedures performed in this study was inhibitory. These data appear to be somewhat paradoxical because a summated discharge of excitatory receptors would be expected to produce excitatory effects. One plausible explanation of the paradox may lie in the time course, or latency, of the evoked responses. Herzog[18] reported the observation of one subject with back pain who was deemed to have baseline (premanipulation) hypertonicity of paraspinal musculature, as evidenced by elevated surface electromyographic amplitudes. In that subject, it was observed that during the manipulative procedure, a reflex activation of increased EMG activity was recorded, with a subsequent near quiescence of baseline EMG activity within 1 second.[18] This latency of postmanipulation inhibitory effects is consistent with the current findings of short-term reflex inhibition after spinal manipulative procedures. Although the primary afferent discharges evoked by spinal manipulation and mobilization may be excitatory initially (less than 500 msec after manipulation),[19] the overall response appears to be one of reflex inhibition.

The current data indicating reflex inhibition were recorded in nearly the same time course as the inhibition of EMG in the hypertonic subject reported by Herzog,[18] in which it was noted that the reflex inhibition, or near quiescence of EMG activity, was recorded approximately 1.5 to 2 seconds after manipulation. The current H-reflex data were recorded within a 10-second period after spinal manipulation procedures. For this reason, the data likely show a late, but pronounced, sequel to excitatory primary afferent discharge to the internuncial pool of the spinal cord. Therefore, it is suggested on the basis of the current data and the findings of Herzog et al[19] that spinal manipulative procedures exert a profound influence on motoneuron excitability, with consequences that lead to reflex inhibition.

As a possible physiologic explanation of the current findings, spinal manipulative procedures may produce "aftereffects," or changes in sensory discharge rates, predominantly in Ia afferents, that occur in response to an alteration in a muscle's history of activation and length changes.[15] The time course of aftereffects ranges from 2 to 400 seconds, with maximum effects typically encountered at 50 seconds.[11,15] Because the extremity and paraspinal muscles[3,13] maintain a relatively high density of spindles, the potential muscle stretch stimulus imposed by spinal manipulation and mobilization procedures may alter the mechanical state of muscle spindle receptors, leading to reflex inhibition of motoneurons.

Postactivation depression is another mechanism that may be involved in postmanipulation inhibition. Depression of the Ia–motoneuron synapse after a previous activation of a stretch reflex arc is a well-documented neurophysiologic response known as postactivation depression.[1,6,21]

Regardless of the exact mechanism, the available reports concerning the effects of spinal manipulative procedures on motoneuron pool excitability clearly show inhibition of motoneurons that is persistent in nature.[9,10,25] Moreover, the inhibitory effects of manipulative procedures occur with a time course similar to that of other aftereffects phenomena reported in the literature.[16] In the case of spinal manipulation, the impulsive thrust likely leads to profound, but short-term, inhibition of alpha motoneurons. However, the contribution of mechanoreceptors discharged by the manipulative thrust appears minimal.

A limitation of the current study stems from the use of the gastrocnemius muscle H-reflex effects as an index of motoneuronal excitability relative to lumbar spine musculature. The technique was used because the tibial nerve motoneuronal pool originates from the same spinal level as that of the segmental paralumbar musculature.

It should be noted also that these data were collected from healthy participants with no low back pain, and as such, should be interpreted with caution relative to the possible effects in patients with back pain. The response in those patients should be studied in the future to determine whether similar attenuation of motoneurons is obtained. Additionally, the possible secondary effects that reduction of motoneuron excitability may have on the muscles of the lumbar spine also should be investigated in the future.

■ Key Points

- The physiologic mechanisms that spinal manipulation exerts on the human motor system are largely unknown.
- Spinal manipulation and mobilization therapy has been postulated to intervene in the "pain–spasm–pain" cycle exhibited by many patients with low back pain.
- Spinal manipulation and mobilization produces a profound but transient attenuation of alpha motoneuronal excitability.
- The findings from this study suggest that manual spinal therapies may lead to short-term inhibition of human motoneurons.

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Point of View

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Several types of manipulations are long used as therapeutic methods for treatment of various spinal disorders, with various rates of success. The rationale and justification for the utilization of any therapy should be solidly founded in the known infrastructure of human anatomy and physiology. The authors describe a neurophysiologic study in which the H-reflex was used to evaluate the effectiveness of lumbosacral spinal manipulation with thrust and mobilization without thrust on healthy human subjects. They found that such therapy results in extremely short period of motor units inhibition, *e.g.*, muscle relaxation.

With this initial data, one must forge forward to answer the next set of questions to formulate the scientific justification for manipulation/mobilization therapy. Does such therapy also inhibit motor units in patients with muscle spasms secondary to disorders? What kind of disorders are responsive to such therapies, and what disorders are not? Is the therapy more effective and longer lasting in patients with muscle spasms? And, if so, how often should it be applied? Would such therapies solve the problem or only provide a temporary but drug-free relief from muscle spasms?

Without the answers to the above questions, manipulation/mobilization therapy will continue to have highly variable rates of success, similar to other methods that did not receive full scientific evaluation to pinpoint their operating principles, applicability, effectiveness, and possible risk.