ACCRAC Award Winning Paper

The Neuromuscular Response to Spinal Manipulation in the Presence of Pain

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Abstract

Objective: The purpose of this study was to evaluate differences in muscle activity in participants with and without low back pain during a side-lying lumbar diversified spinal manipulation.

Methods: Surface and indwelling electromyography at eight muscle locations were recorded during lumbar side-lying manipulations in 20 asymptomatic participants and 20 participants with low back pain. The number of muscle responses and muscle activity onset delays in relation to the manipulation impulse were compared in the 2 pain groups using mixed linear regressions. Effect sizes for all comparisons were calculated using Cohen's *d*.

Results: Muscle responses occurred in 61.6% ± 23.6% of the EMG locations in the asymptomatic group and 52.8% ± 26.3% of the symptomatic group. The difference was not statistically significant but there was a small effect of pain ($d = 0.350$). Muscle activity onset delays were longer for the symptomatic group at every EMG location except the right side indwelling L5 electrode, and a small effect of pain was present at the left L2, quadratus lumborum and trapezius surface electrodes ($d = 0.311$, 0.278, and 0.265) respectively. The indwelling electrodes demonstrated greater muscle responses ($P \leq .01$) and shorter muscle activity onset delays ($P < .01$) than the surface electrodes.

Conclusions: The results revealed trends that indicate participants with low back pain have less muscle responses, and when muscle responses are present they occur with longer onset delays following the onset of a manipulation impulse. (J Manipulative Physiol Ther 2016;39:288-293)

Key Indexing Terms: *Manipulation; Spinal; Low Back Pain; Chiropractic; Reflex; Electromyography; Biomechanical Phenomena; Kinetics*

Spinal manipulation (SM) is a mechanical treatment that is associated with neurophysiological changes, and increasing our understanding of these mechanisms may help improve clinical delivery and patient outcomes. Clinical evidence supports the use of SM for acute low back pain[1,2]; however, the etiology of clinical improvement as it relates to the neurophysiological response remains unknown. Several neural effects of manipulation have been observed that include changes in muscle activity, central motor excitability, H-reflexes, and pain processing.[3,4] Improving our understanding of how these effects are related to clinical conditions may lead us toward additional insight into the mechanism of SM as a treatment.

Muscle activation is the primary means to assess the neuromuscular response to SM, and gain insight into the neuromuscular pathways. Two aspects of muscle activity are commonly quantified using electromyography (EMG): (1) amplitude of the signal and (2) timing of the signal. Evidence of EMG amplitude changes in response to manipulation is conflicting, and includes both increases and decreases following SM.[5,6] In addition, amplitude measures are subject to interpretive difficulties across participants and investigations because they are confounded by the normalization process, which is influenced by the type of muscle studied, training level, and participant motivation.[7]

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Paper submitted April 18, 2015; in revised form November 12, 2015; accepted November 14, 2015.
0161-4754

http://dx.doi.org/10.1016/j.jmpt.2016.02.011

The second measure of the neuromuscular response is timing, which quantifies the reflex response of the underlying muscles in the treatment area.[8] Onset delay of a muscle in response to SM is too short to be a voluntary activation,[9] and may indicate the presence of a spinal reflex. Timing of this reflex in response to manipulation has been documented in wide ranges between 2 and 200 milliseconds after force onset, and is associated with the location and type of manipulation administered.[10,11]

If a spinal reflex is present in response to manipulation, quantifying the differences in timing between participants with low back pain (symptomatic) and those without (asymptomatic) will lead to a better understanding of the benefits of spinal manipulation therapy. Presence of low back pain (LBP) alters the activity of trunk muscles during functional activities[12] and both increases and decreases in EMG amplitude have been noted in participants with LBP compared to controls.[13] In participants with frequent or constant LBP, a greater occurrence of reflex responses to manipulation has been reported in the paraspinal musculature when compared to healthy controls[14]; however, these effects have not been investigated in non-instrumented diversified side-lying style of SM commonly performed in the clinic.

The purpose of this study was to evaluate differences in muscle activity in participants with and without LBP during a side-lying lumbar diversified SM. We hypothesized differences between groups in both the number of muscle responses and muscle activity onset delay. We anticipate these results will encourage more investigations into quantifying the neuromuscular response during SM and foster a better mechanistic understanding of the effects of SM on pain.

Methods

Participant Information

Forty participants were recruited and used to compare the number of muscle responses and muscle activity onset delay during SM. Twenty participants (age: 32.6 ± 11.0 years, mass: 70.5 ± 12.1 kg, height: 172.2 ± 9.1 cm) had no history of low back pain (asymptomatic group), and twenty participants (age: 33.4 ± 9.9 years, mass: 71.3 ± 11.5 kg, height: 167.9 ± 10.2 cm) had a history of low back pain located between the lowest rib and the pelvis (symptomatic group). The symptomatic participants were experiencing pain at the time of testing (average verbal pain scale 3.3 out of 10; average Oswestry disability rating = 23.4% moderate disability).

Each participant was screened for contraindications to SM by performing an orthopedic and neurologic examination. Exclusion from the investigation occurred if: (1) current level of pain exceeded a seven out of ten on a verbal pain scale; (2) radicular pain below the knee during orthopedic testing was present; or (3) a neurologic exam revealed absent reflexes, decreased sensation, or weakness. Each participant provided written, informed consent in accordance with Colorado Multiple Institutional Review Board prior to the start of the experimental session.

Application of Spinal Manipulation

High-velocity, low-amplitude (HVLA) SMs at the L3 and Sacroiliac (SI) spinal level with a hypothenar contact in the side-lying position were applied to each participant (Figure 1A). In addition, 2 grade IV mobilizations were applied but were not analyzed in this investigation. The order of treatments was randomized, and the time between manipulations was between one and three minutes. Manipulations were performed by 2 chiropractors, each with over 10 years of clinical experience.

EMG and Manipulation Impulse Instrumentation

Bilateral surface EMG was recorded from the erector spinae (ES) at the L2 level. Bilateral indwelling EMG was recorded from the multifidus at the L2 and L5 levels[15] and unilateral surface EMG was recorded from the left quadratus lumborum (QL) and the left lower trapezius (LT) for a total of eight EMG recording locations (Figure 1B). The treatment force (manipulation impulse) between the chiropractor and the patient at the site of treatment was estimated using an optimized weighted least squares model that combines direct and indirect measurements of force with an accuracy of 3.6 N, and allows natural contact between the practitioner and the patient.[16]

EMG Signal Processing

The raw EMG signals were sampled at 2000 Hz, bandpass filtered to remove movement artifact and high frequency noise (4th order Butterworth, 15-350 Hz), and transformed using the Teager-Kaiser Energy Operator (TKEO).[17,18] Linear envelopes of the EMG signals were created by applying full-wave rectification and a low-pass filter (4th-order Butterworth, 50 Hz cutoff).

Muscle Responses and Muscle Activity Onset Delay Calculations

Whether or not a muscle responded and was considered "on" (positive muscle response) was determined using the double-threshold method with an amplitude threshold of 8 standard deviations (SD) calculated from 1 sec of baseline data, and a duration threshold of 10 milliseconds.[19] The muscle response variable was calculated by recording the number of positive responses across 8 EMG channels and dividing by the total number of channels, expressed as a percentage.

$$\text{Muscle Response} = \frac{\#\ \text{of Positive Responses}}{\#\ \text{of EMG Channels}} \times 100 \quad (1)$$

Muscle activity onset delay was quantified as the time delay between the positive slope of the manipulation impulse and the first EMG activity that met the previously reported double threshold criterion.[19]

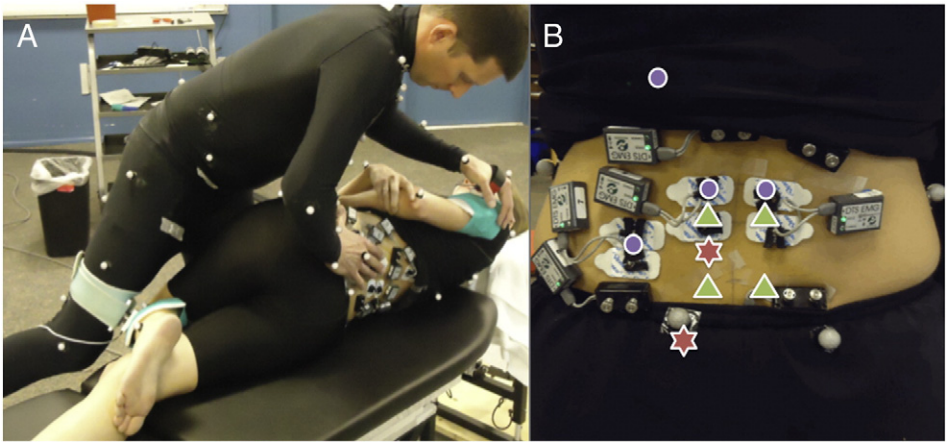

Fig 1. *A. Side posture set-up. B, Instrumentation included indwelling (triangles) and surface (circles) EMG. Stars indicate manipulation sites at the L3 and SI spinal level on the left.*

Comparison of Asymptomatic and Symptomatic Participants

Differences in the muscle response and the muscle activity onset delay between the 2 pain groups (symptomatic and asymptomatic), 2 electrode types (indwelling and surface), 2 manipulation locations (L3 and SI), and manipulation order (first or second manipulation) were compared using mixed linear regressions with a random effect for subject. Effect sizes for all comparisons were calculated using Cohen's *d*. A small effect was defined as $0.2 < d < 0.5$, a moderate effect as $0.5 \leq d < 0.79$, and a large effect as $d \geq 0.8$. Level of significance for all statistical tests was set at $\alpha = .05$.

Results

Muscle Responses

Muscle responses occurred in 57.2% of all EMG recording sites during spinal manipulation. Across participants, muscle response occurred in 61.6 ± 23.6% of the EMG locations in the asymptomatic group and 52.8 ± 26.3% of the symptomatic group (Figure 2). The effect size was small ($d = 0.350$), but the difference was not statistically significant ($\beta = -4.3$, SE = 3.9, $P = .27$) where β is the model-predicted regression offset of the symptomatic group (Figure 2).

Greater muscle response occurred in the muscles recorded with the indwelling electrodes than the surface electrodes ($\beta = 4.3$, SE = 1.9, $P \leq .01$), and greater muscle response occurred during the L3 manipulation than the SI manipulation ($\beta = 5.3$, SE = 2.2, $P = .02$). In the indwelling electrodes a small effect of pain was present during the SI manipulation ($d = 0.227$). In the surface electrodes a small effect of pain was present during the L3 and SI manipulations (0.495, and 0.384 respectively) (Figure 3).

Muscle Activity Onset Delay

Muscle activity onset delays (when activity occurred) ranged from 0 to 395 milliseconds for the asymptomatic participants and from 1 to 397 milliseconds for the symptomatic participants. The mean onset delay across all active muscles in symptomatic group was 14 milliseconds longer than the asymptomatic group (Figure 4). No effect size was detectable ($d = 0.139$) and the difference was not statistically significant ($\beta = 4.7$, SE = 10.1, $P = .64$) (Figure 4).

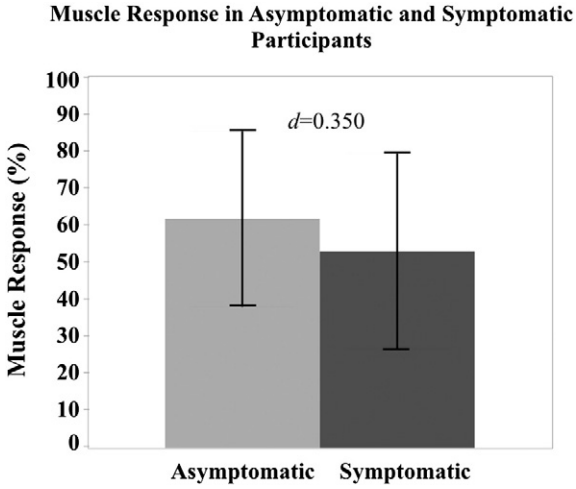

Fig 2. *Percentage of muscle responses for each pain group across all EMG recording locations. A trend of greater muscle responses in the asymptomatic group with a small effect of pain was present.*

Muscle activity onset delay was longer for the symptomatic group in every EMG location except the right side indwelling L5 electrode, and a small effect of pain was present at the left L2, quadratus lumborum and trapezius surface electrodes ($d = 0.311$, 0.278, and 0.265 respectively, Figure 5). Muscle activity onset delays demonstrated observably large variability across participants regardless of pain group.

The indwelling electrodes demonstrated shorter muscle activity onset delays than the surface electrodes ($\beta = 16.1$, SE = 4.2, $P < .01$) and the L3 manipulation demonstrated

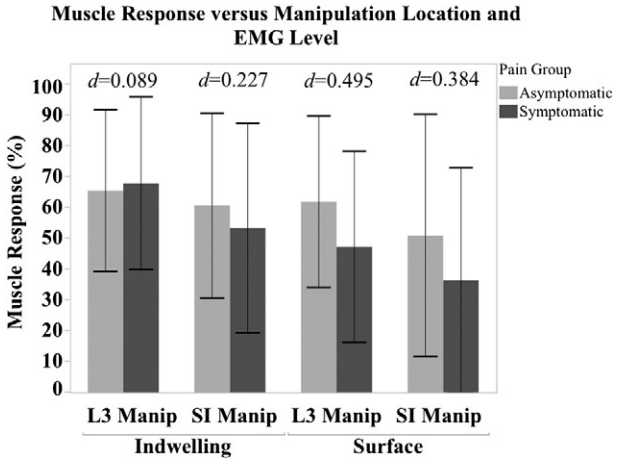

Fig 3. *Muscle response for each pain group (asymptomatic, symptomatic) for the L3 and SI manipulation locations and the indwelling and surface electrodes. A trend of greater muscle responses in the asymptomatic group with small effects of pain was present.*

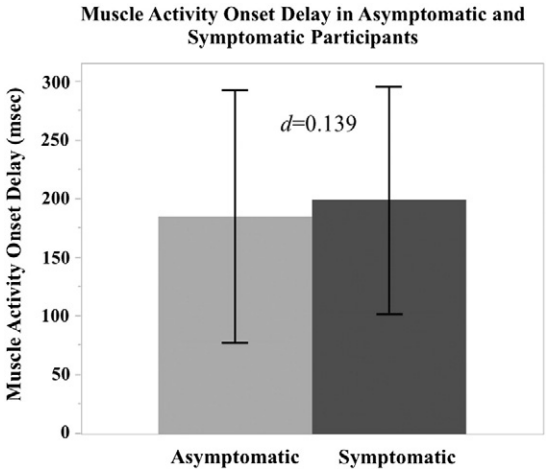

Fig 4. *Muscle activity onset delays for asymptomatic and symptomatic participants across all EMG recording sites. A trend of longer delays in the symptomatic group was present with no detectable effect of pain.*

shorter muscle activity onset delays than the SI manipulation but the differences were not statistically significant (β = 6.0 milliseconds, SE = 4.3, P = .17).

Manipulation Order

A linear mixed model regression analysis revealed no effect of manipulation order on muscle response (β = 0.3, SE = 0.86, P = .97) or onset delays (β = 6.0, SE = 8.6, P = .49).

Discussion

This investigation was the first to quantify differences in the muscle response and muscle activity onset delay

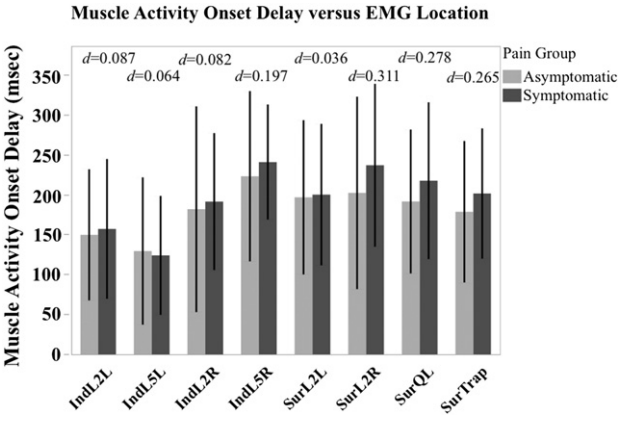

Fig 5. *Muscle activity onset delays for all 8 EMG locations for symptomatic and asymptomatic participants. A trend of longer onset delays was seen for the symptomatic group with a small effect for pain in three of the surface electrodes.*

between healthy participants (asymptomatic) and participants in pain (symptomatic) *during* a side-lying lumbar manipulation. Symptomatic participants had less muscle responses and longer muscle activity onset delays than the asymptomatic participants. These differences suggest that underlying mechanisms of SM are linked to neuromuscular responses, and create a foundation on which to improve clinical delivery of SM based on quantifiable neuromuscular response variables.

Several limitations in this investigation should be considered. First, the treatment location in the symptomatic group was not chosen based on patient-specific assessment of clinical lesion or injury. The nonspecific application of SM allows an unbiased comparison of treatment locations across groups, but may not represent the response that would occur in clinical practice. Second, the state of the muscle (ie, hypertonicity) may affect the presence or absence of muscle response,[6] and was not quantified. Therefore, the results are generalizable only to the presence or absence of low back pain, and not to a specific clinical lesion. Last, the manipulations analyzed in this investigation were part of a larger data set that included 2 grade IV mobilizations applied to the L3 and SI spinal levels. To eliminate bias, treatment order was randomized, and an effect of treatment order on these muscle activity variables was not present according to the statistical analyses.

The longer muscle activity onset delay in the symptomatic group may indicate that patients in pain experience more excitability in capsular reflex pathways than muscle spindle pathways. The difference in delay was small (5 milliseconds) but potentially relevant given that previous work demonstrated muscle activity onset delays in the range of 2.4 to 18.2 milliseconds[11] and that a spinal reflex is thought to occur within 120 milliseconds.[20] Herzog[21] proposed that 2 different pathways—muscle spindle pathway and capsule mechanoreceptor pathway—are involved in the response to SM, and are likely differentiated by time delay. In addition, Herzog[21]

anticipated that muscle spindle pathways would be activated before mechanoreceptor pathways due to the reliance of large diameter IA tracts, and may characterize the response in our symptomatic participants.

Quantifying the similarities and differences in the neuromuscular response between asymptomatic and symptomatic participants provides insight into how SM achieves a therapeutic effect. Overall, symptomatic participants demonstrated a similar neuromuscular response to asymptomatic participants, which indicates that as a population they may be studied in a similar manner to an asymptomatic population. The trends of greater muscle response and shorter muscle activity onset delays in the asymptomatic population could be used as a clinical indicator of an asymptomatic "status", and the goal of treatment for patients that visit the clinic. The most apparent effects of pain occurred in neuromuscular responses in muscles that were located farthest from the manipulation site (quadratus lumborum and lower trapezius) and recorded with surface electrodes (Figures 3 and 5). This may indicate that these muscles were primary generators of the participant's symptomatic condition. If SM can be used to influence the neuromuscular reflex pathways (e.g. muscle spindle pathway in a symptomatic patient), then the greater number of muscle activity responses and shorter delays in the symptomatic participants suggest activation criteria for an effective application of SM.

The large variability of our data supports the view that many factors other than pain influence the neuromuscular response to SM. Numerous low back pain generators and etiologies such as muscular, arthrogenic, neurological, and discogenic exist that may contribute to differences in neuromuscular responses. In addition, no single treatment method may produce consistent responses across all forms of low back pain. As a result, design of future investigations on SM on neuromuscular response should optimize the study scope with the goal of detecting small differences with highly variable responses and accounting for other factors that may contribute to the neuromuscular response of participants in pain. Biomechanical investigations such as this one are often performed on a limited number of participants due to the significant collection and processing time required for this type of data. While our investigation was ambitious in its attempts to quantify the differences between these populations (40 participants), the variability in the data prevented strong statistical conclusions.

Conclusion

This investigation is the first to quantify and compare differences in the neuromuscular response during manipulation in symptomatic and asymptomatic participants. The results revealed trends that indicate participants with low back pain have less muscle responses, and when muscle response is present they occur with longer onset delays following the onset of a treatment impulse. Differences in the presence and timing of the neuromuscular response to SM between groups suggest which muscles and spinal pathways may be affected by the presence of pain. We anticipate that future work will build on this foundation to identify specific mechanistic associations of SM with pain relief, and develop clinical indicators of effective delivery of SM.

Funding Sources and Potential Conflicts of Interest

Funding was provided by the National Institutes of Health (National Center for Complimentary and Alternative Medicine) Grant# R00AT004983 and the NCMIC Foundation No conflicts of interest were reported for this study.

Contributorship Information

Concept development (provided idea for the research): B.D., B.E.
Design (planned the methods to generate the results): B.D., B.E.
Supervision (provided oversight, responsible for organization and implementation, writing of the manuscript): B.D., B.E.
Data collection/processing (responsible for experiments, patient management, organization, or reporting data): B.D., S.C., C.M.
Analysis/interpretation (responsible for statistical analysis, evaluation, and presentation of the results): B.D., S.C., C.M., C.D.
Literature search (performed the literature search): B.D., S.C., C.M.
Writing (responsible for writing a substantive part of the manuscript): B.D., S.C., C.M.
Critical review (revised manuscript for intellectual content, this does not relate to spelling and grammar checking): B.D., B.E., S.C., C.M., C.D.

Practical Applications

- The neuromuscular responses to spinal manipulation as evaluated by the number of muscle responses and muscle activity onset delays were measured during a lumbar spinal manipulation.
- Comparisons were made between asymptomatic participants and those experiencing low back pain
- The indwelling electrodes demonstrated greater number of muscle responses and shorter muscle activity onset delays when compared to the surface electrodes.
- Small effects of pain were present in the number of muscle responses and the muscle activity onset delays.
- The results revealed trends that indicate participants with low back pain have less muscle responses, and when muscle response is present they occur with longer onset delays following the onset of a manipulation impulse.

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