Title
Do cerebral potentials to magnetic stimulation of paraspinal muscles reflect changes in palpable muscle spasm, low back pain, and activity scores?

Journal
Journal of Manipulative and Physiological Therapeutics, 23(7)

ISSN
0161-4754

Authors
Zhu, Yu
Haldeman, Scott
Hsieh, Chang-Yu J
et al.

Publication Date
2000-09-01

DOI
10.1067/mmt.2000.108821

0161-4754/2000/$12.00 + 0 **76/1/108821** © 2000 JMPT

Do Cerebral Potentials to Magnetic Stimulation of Paraspinal Muscles Reflect Changes in Palpable Muscle Spasm, Low Back Pain, and Activity Scores?

Yu Zhu, MD,[a] Scott Haldeman, DC, MD, PhD,[a,b] Chang-Yu J. Hsieh, DC,[b] Pingjia Wu, MD,[c] and Arnold Starr, MD[a]

ABSTRACT

Objective: Previous studies have shown that cortical-evoked potentials on magnetic stimulation of muscles are influenced by muscle contraction, vibration, and muscle spasm. This study was carried out to determine whether these potentials correlate with palpatory muscle spasm, patient symptoms, and disability in patients with low back pain.

Methods: A prospective observational study was performed on 13 subjects with a history of low back pain visiting an orthopedic hospital-based clinic. Patients were screened for serious pathologic conditions by an orthopedic surgeon. The patients were then evaluated for the presence of muscle spasm by one of the investigators who was blinded to the results of the evoked potential studies. Patients were asked to complete a low back pain visual analogue scale (VAS) and a Roland-Morris Activity Scale (RMAS). Cortical-evoked potentials were recorded with a magnetic stimulator placed over the lumbar paraspinal muscles with the patient in the prone position. The palpatory examination, VAS, RMAS, and the cortical potentials were repeated after 2 weeks of therapy commonly used to reduce muscle spasm.

Results: The patients demonstrated a significant decrease in low back pain VAS and RMAS scores after treatment compared with before treatment. There was a reduction in the amount of palpatory muscle spasm in 11 of 13 cases. The cortical potentials before treatment were attenuated compared with previously reported controls and showed a significant increase before and after treatment in the amplitude of these potentials with multivariate analysis of variance. There was significant correlation between the changes in cortical potentials after treatment and the changes noted in paraspinal muscle spasm and VAS and RMAS scores.

Conclusions: This study confirms the previous report that the amplitude of cerebral-evoked potentials on magnetic stimulation of paraspinal muscles is depressed in the presence of palpable muscle spasm. The close correlation among these potentials, paraspinal muscle spasm, and clinical symptoms suggests that the measurement of muscle activity may be more important in the assessment of low back pain than is commonly accepted. (J Manipulative Physiol Ther 2000;23:458-64)

Key Indexing Terms: Chiropractic Manipulation; Evoked Potentials; Low Back Pain; Muscle Spasm

IINTRODUCTION

The role of muscle in low back pain and even the existence of muscle spasm has been the subject of controversy,[1-3] primarily because of the inadequate measurement techniques that correlate to patient symptoms and other clinical findings. Despite the skepticism, physicians commonly attribute spinal pain to muscle spasm, sprains, strains, or soft-tissue trauma.[4,5] Patients often point to muscles as the source of their pain, and many physicians describe muscle firmness or spasm as a palpable finding in the clinical examination.[6] The American Medical Association Guidelines for the Evaluation of Permanent Impairment considers vertebral muscle guarding or spasm an objective finding for differentiating categories of impairment.[7]

Muscle firmness, spasm, or so-called trigger points are perceived by many physicians as palpable and by many patients as specifically tender.[8,9] Needle electromyography of these tender muscles, however, has demonstrated that they are electrically silent.[2] As yet, there are no reliable electrodiagnostic methods to confirm the presence of clinically palpable muscle firmness and subjectively tender muscles. The ability to record cerebral potentials after magnetic stimulation of muscle has provided a new and noninvasive method for examining muscle afferent activity in human beings.[10] Cortical potentials can be evoked on magnetic stimulation of muscles in both the upper and lower extremities and the paraspinal muscles. These magnetically evoked potentials can be attenuated by vibration and voluntary muscle contraction, which suggests that they are the result of 1a-afferent fiber stimulation. The observation that these potentials are also attenuated by involuntary paraspinal muscle

[a]Department of Neurology, University of California, Irvine, Irvine, Calif.
[b]Research Division, Los Angeles College of Chiropractic, Whittier, Calif.
[c]Department of Neurology, Chang Zhen Hospital, Shanghai, China.

This project was supported by a grant from the Foundation for Chiropractic Education and Research with donations of equipment by Dantec Medical (Copenhagen) and Leander, Inc (Port Orchard, Wash).

Presented in part at the International Society for the Study of the Lumbar Spine, June 1995, Helsinki, Finland.

Submit reprint requests to: Scott Haldeman, DC, MD, PhD, FRCP(C), 1125 E 17th St, Ste W-127, Santa Ana, CA 92701; *HaldemanMD@aol.com*.

Paper submitted May 21, 1998; in revised form June 23, 1998.
doi:10.1067/mmt.2000.108821

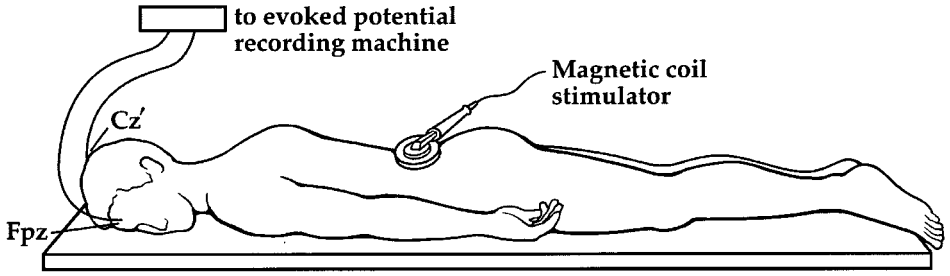

Fig 1. *The placement of the stimulator and the recording electrodes used to obtain cortical-evoked potentials on magnetic stimulation of paraspinal muscles.*

Table 1. *Demographic data of patients who completed the trial*

Patient no.	Age (y)	Diagnosis	Site of spasm	Site of pain	Duration
1	63	L5-S1 disk bulging	bilat	LLB	7 mo
2	62	Left L5 radiculopathy	left	LLB, LLE	1 mo
3	36	Lumbar strain	right	CLB	1 wk
4	35	Lumbar strain	bilat	RLB, LLB	2 wk
5	78	Spinal stenosis	bilat	LLE	2 wk
6	60	Spondylosis, right sciatica	bilat	RLE	3 mo
7	40	Lumbar strain	bilat	RLB	2 mo
8	53	L5-S1 disk bulging	bilat	RLB, LLB, LLE	2 wk
9	39	Lumbar strain	bilat	CLB	2 wk
10	43	Myofascial pain syndrome	bilat	RLB, LLB	2 y
11	38	Myofascial pain syndrome	bilat	CLB	10 y
12	45	L5-S1 disc herniation	bilat	LLE	2 mo
13	37	Lumbar spondylosis	bilat	LLB	3 wk

Bilat, Bilateral; *LLB*, left low back; *LLE*, left lower extremity; *CLB*, central low back; *RLB*, right low back; *RLE*, right lower extremity.

spasm raises the possibility that they may be of value in monitoring muscle spasm in patients with low back pain.[11]

We are interested in defining the relation between cerebral potentials evoked by magnetic stimulation of paraspinal muscles and clinical findings such as palpable muscle spasm, pain, and activity scores and determining whether evoked potentials change in concert with patient symptoms.

METHODS

This paper was designed as a prospective observational study to determine the correlation between magnetically induced, cortical-evoked responses from paraspinal muscles, clinically determined muscle spasm, Visual Analogue Scales (VAS) for pain, and Roland-Morris Activity Scales (RMAS).

Thirteen patients presenting to a hospital-based back pain clinic were screened by an orthopedic surgeon not affiliated with the project to exclude patients with serious pathologic conditions. Patients were then evaluated for inclusion criteria and clinical assessment by one of the investigators (CYH). Inclusion criteria were low back pain with or without leg pain, the presence of lumbar muscle spasm on palpation (the working definition for palpable spasm in this study was the presence of palpable hardness, bands, and localized tenderness of paraspinal muscles that could be differentiated from adjacent muscles), no prior history of lumbar surgery, and no contraindications for lumbar spinal manipulation. Table 1 presents the demographic data and working diagnoses of these patients. The patients signed a consent form to participate in the project after an explanation about the benefits and risks of the research and treatment procedures.

The patients were given a questionnaire to complete, including an RMAS and low back pain VAS. A crude assessment of the degree of muscle spasm was made from the clinical examination for each side of the spine. This ranged from no spasm (–) to marked spasm (+++), based on the clinical experience of the assessing clinician. The clinician was blinded to the results of the cortical-evoked potentials and VAS and RMAS scores but not to patient symptoms or the remainder of the clinical examination.

Evoked Potential Testing

The subjects were tested while lying on an examination table in the prone position and were awake throughout the procedure.

Magnetic stimulation was performed with a MagPro magnetic stimulator (Dantec Medical, Copenhagen, Denmark) (Fig 1). A stimulation coil with a circular outer diameter of 12.5 cm (Dantec Medical, MC-125) was placed tangentially to the skin overlying the paraspinal muscles, 2 to 3 cm lateral to the mid-line at the L2 to L5 levels. The stimulation wave form was monophasic, with a pulse width of 0.16 ms. The magnetic field at the center of the maximum stimulation output was 2.0 T, and the stimulation rate was 1 Hz. Magnetic stimulation was applied unilaterally, resulting in 2 recordings per patient. The strength of stimulus was

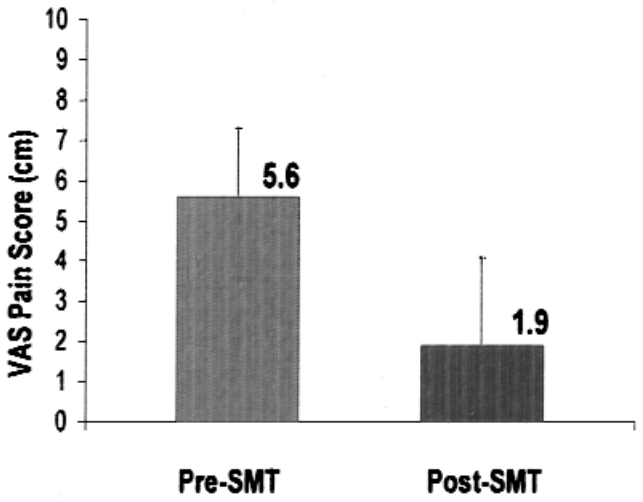

Fig 2. *Visual Analogue Scale (VAS) scores before and after treatment in 13 patients. SMT, Spinal manipulative therapy.*

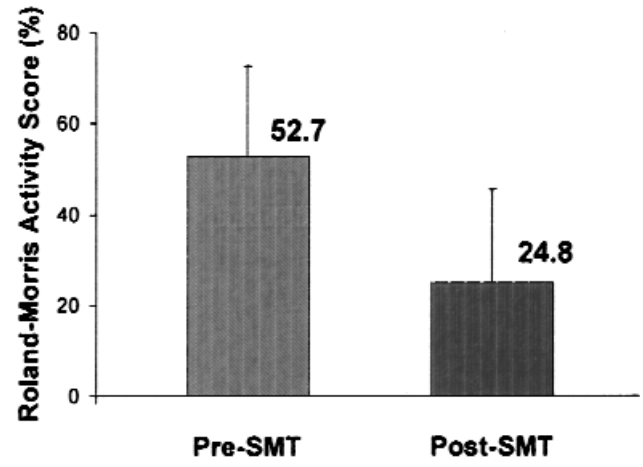

Fig 3. *Roland-Morris Activity Scale (RMAS) scores before and after treatment in 13 patients. SMT, Spinal manipulative therapy.*

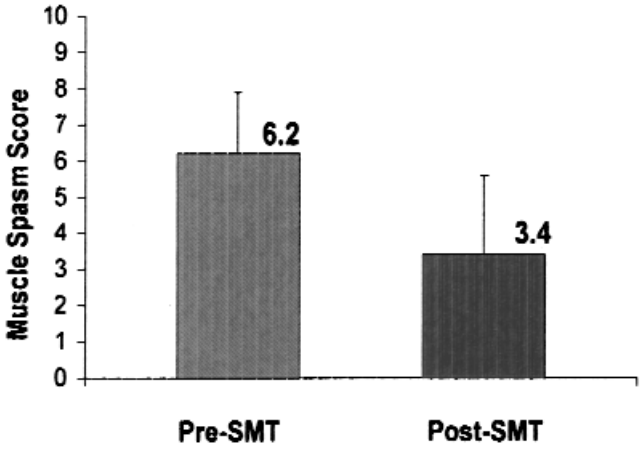

Fig 4. *Palpable muscle scales before and after treatment in 13 patients. SMT, Spinal manipulative therapy.*

30% of maximum output. No contraction of leg muscles was produced at this stimulation intensity.

Recording electrodes were 8-mm diameter silver/silver-chloride disks attached with electrode cream to the skin. Electrode impedance was maintained below 2 ko. Recording electrodes were placed on the scalp 2 cm posterior to the Cz position of the international 10-20 System referenced to Fpz. A ground electrode was placed on the scalp between the pair of recording electrodes. The bandpath filters of the amplifier were set at 5 and 1000 Hz. The analysis time was 100 ms. A total of 128 single sweeps were averaged in each trace, and the test was completed 2 times on each occasion to ensure consistency. Paraspinal, muscle-evoked cerebral potentials on magnetic stimulation were recorded at a rate of 1 Hz in each patient before and after the course of treatment. All positive and negative components within 100 ms from the time of stimulation were isolated for the measurement of latency and amplitude.

Treatment Methods

Patients were asked to attend the clinic 3 to 5 times a week for a period of 2 to 3 weeks. Treatment consisted of soft-tissue massage and manipulation. The soft-tissue treatment included ischemic compression applied to the affected muscles in the back and lower extremity, followed by distraction of the lumbar spine. The ischemic compression was delivered through a thumb-like massage hand tool for 1 to 2 minutes until the pain subsided or disappeared. Distraction was carried out with the patient lying prone on a motorized table with the ankles strapped to the table and the lumbar spine placed in flexion (Leander, Inc). Flexion occurred at 6 to 10 seconds per cycle, and the clinician further increased the stretching by spreading the spinous processes in a caudal-cephalic fashion while the table was moving downward. These distraction maneuvers were also performed in extension and lateral flexion. After this procedure, the patients received high-velocity, low-amplitude lumbar spine adjustments in the side posture position, with the affected side up and with direct contact with the mammillary process at the direction of the clinician. The patients had little or no pain during these procedures. General advice on proper lifting and simple home exercise was given to the patients. The total number of treatments ranged from 3 to 15, with a mean of 8.3 (standard deviation 2.8).

Data Analysis

Evoked potentials, clinical assessment of muscle spasm, and VAS and RMAS scores were recorded at the beginning and end of the treatment period, irrespective of the degree of recovery of the patient. These data were all sequestered until the time of analysis.

The data were entered into the SPSS-PC+ statistical program (SPSS, Chicago, Ill). Mean and standard deviations of each variable were calculated. Multivariate analysis of variance, one-way analysis of variance, and paired Student *t* tests were used for testing. The level of significance (α) was set at .05.

RESULTS

The data from the before- and after-treatment clinical evaluations for muscle spasm, paraspinal muscle cortical-evoked potentials, VAS, and RMAS are noted in Tables 2 and 3.

Fig 2 is a representation of the VAS scores before and after treatment. The VAS score before treatment (mean 5.6 ± 1.7 cm) compared with the VAS score after treatment (mean 1.9 ± 2.2 cm) showed a significant difference ($P < .001$) with a paired Student *t* test. Improvement was noted in 11 of 13 patients, with 2 patients showing no improvement. No patient showed increased symptoms. Nine of 13 patients showed >50% improvement in the VAS score.

Fig 3 is a representation of the RMAS scores before and after treatment. The RMAS was administered 2 times on the day before treatment and 1 time on completion of the treatment. The interclass correlation between the testing on the same day was 0.95. To improve the reliability of this measurement, the results from the 2 separate testings before treatment were averaged. The results before and after treatment were then analyzed for differences. The mean score before treatment was 52.7% ± 19.9%. The mean score after treatment was 24.8% ± 20.6%. A significant difference was found with the paired Student *t* tests ($P = .002$). Only 1 patient reported a decrease in activity levels after treatment. Seven of 13 patients showed a >50% improvement in their activity scores.

Fig 4 is a representation of the paraspinal muscle spasm findings before and after treatment. To enter the trial, the patients had to have been diagnosed clinically with low back pain with palpable unilateral or bilateral palpable muscle spasm in the lumbar paraspinal muscles. Eleven of 13 patients had palpable muscle spasm bilaterally; the remaining 2 patients were diagnosed with unilateral muscle spasm. After treatment, 11 patients were believed to have had a reduction in muscle spasm, with 2 patients (patients no. 2 and 13) perceived clinically as not showing any appreciable change in muscle spasm after treatment. Patients with bilateral muscle spasm who initially showed improvement tended to show improvement bilaterally.

Cerebral Evoked Potentials

These potentials consisted of both positive (P) and negative (N) components that were designated by custom according to latency in milliseconds: P30, N40, P50, N70, and P90 (Fig 5). The latency of the earliest positive potential (P30) is in the range of 26 to 36 ms when stimulation was applied to the L2 through L5 levels.

When comparing the evoked potentials before treatment with previously published normal controls,[11] 15 of 26 recordings had reduced amplitudes of the P30-N40 component; 12 of 26 recordings had reduced amplitudes of the N40-P50 component; and 8 of 26 recordings had reduced amplitude of the P40-N70 component. Three patients had components that were within the normal range in all peaks. Comparing evoked potentials after treatment with the potentials before treatment revealed that the P30-N40 component increased in amplitude in 11 of 15 recordings. A total of 4 of 15 recordings demonstrated an increase in amplitude of >50%. The N40-P50 component showed increased amplitude in 10 of 12 recordings. The P50-N70 component increased in amplitude in 8 of 8 recordings, where there was an initially attenuated response. Multivariate analysis of variance showed significant before- and after-treatment effects across the amplitudes of each of the 3 cerebral potentials as demonstrated in Fig 6 (Hotelling F = 15.88, $P = .001$). One-way analysis of variance showed a significant change in the amplitude of each of the 3 components of the cerebral potentials after treatment for both right and left lumbar paraspinal muscles ($P < .1$). Fig 7 illustrates 2 examples of the cortical-evoked responses that normalized after manipulation.

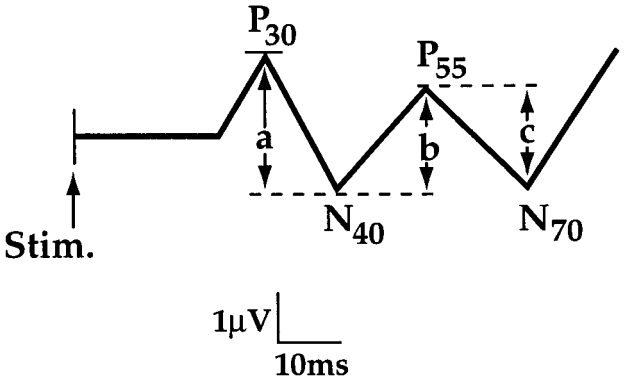

Fig 5. *Illustration of the method used to measure the amplitude of P30-N40 response* (a), *the N40-P55 response* (b), *and the P55-N70 response* (c). Stim, *stimulus;* P, *positive wave;* N, *negative wave polarity.*

Comparison of Clinical Outcomes and Cerebral-evoked Potentials

When defining improvement of cerebral-evoked potentials as showing an increase of the P30-N40 component or both the P30-N40 and N40-P50 amplitudes by >50% after treatment, we were able to identify 10 patients who showed improvement in both palpable muscle spasm and cerebral-evoked potentials. One patient had normal evoked response before and after manipulation but showed an improvement in muscle spasm. Two patients had normal evoked potentials before and after treatment and no change in palpable muscle spasm. The sensitivity of evoked potentials in predicting improvement in palpable muscle spasm was calculated to be 91% (10 of 11) and the specificity calculated to be 100% (2 of 2).

We defined an improvement in pain as a 50% reduction in the VAS score. With this definition, the sensitivity of evoked potentials in predicting improvement in pain was 89% (8 of 9) and the specificity 50% (2 of 4). By defining an improvement in function as a 50% reduction in the RMAS score, the sensitivity of evoked potentials in predicting improved activity was 71% (5 of 7) and the specificity 17% (1 of 6).

A multivariate analysis of variance with the evoked potentials before treatment as the dependent variables, out-

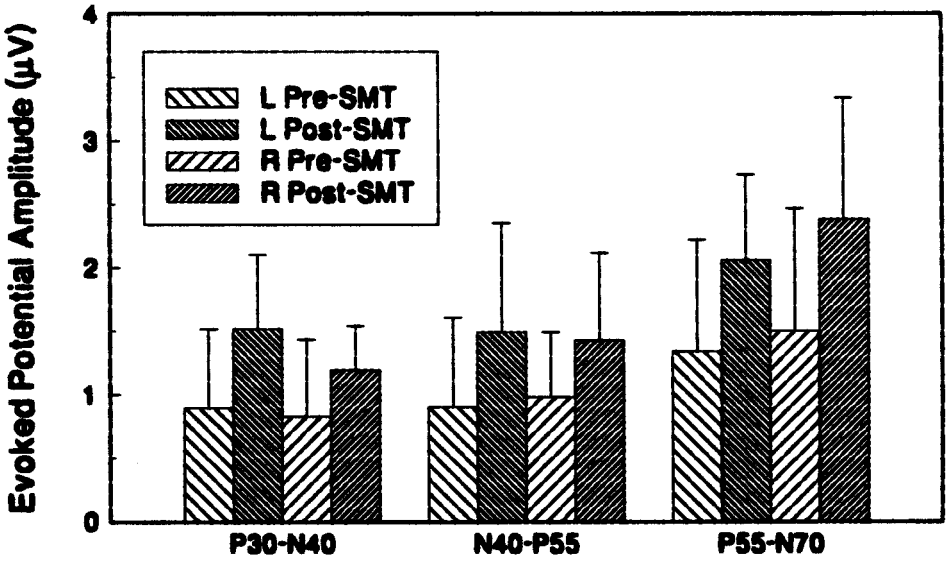

Fig 6. *Mean amplitude of cortical-evoked potential (μV) on magnetic stimulation of the left* (L) *and right* (R) *lumbar paraspinal muscles before* (pre) *and after* (post) *treatment in 13 patients.* SMT, *Spinal manipulative therapy.*

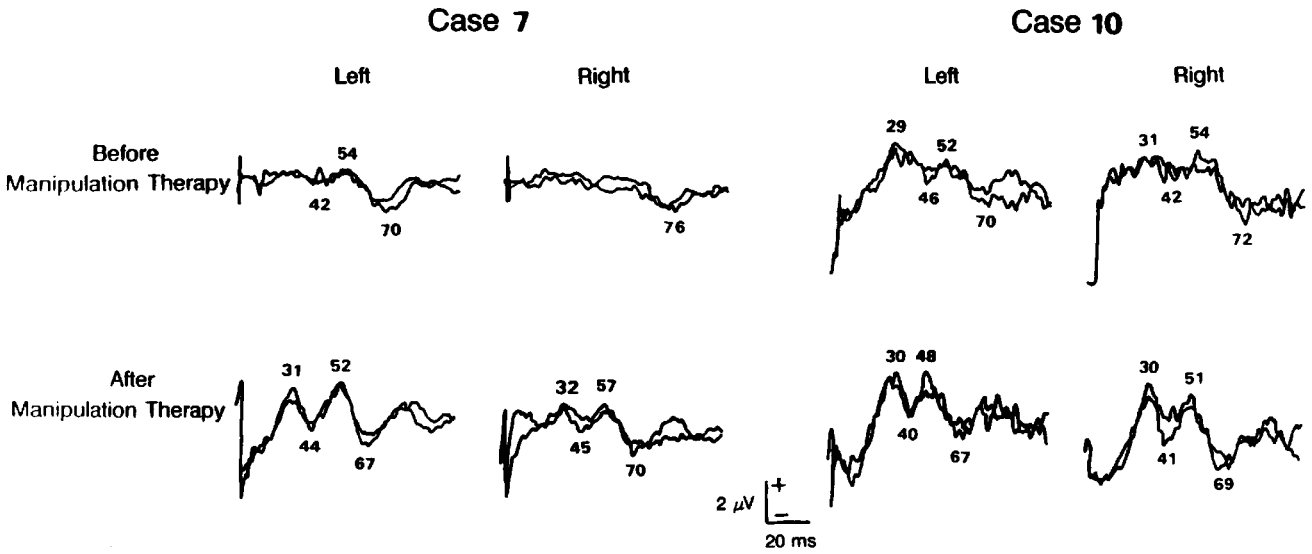

Fig 7. *Two examples of cortical-evoked potentials on magnetic stimulation of paraspinal muscles before and after treatment. Each recording represents 2 superimposed, computer-averaged potentials from 120 stimuli.*

come of the muscle spasm as the between-subject variable, and the side of the lumbar muscles as the within-subject variable resulted in a statistically significant difference of the combined evoked potentials among the 3 groups who showed different outcomes in the spasm (P = .019). The patients who showed improvement in muscle spasm (ie, total disappearance of spasm (n = 6) or less spasm (n = 5) had depressed cortical-evoked potentials before treatment. The patients (n = 2) who showed no improvement in muscle spasm had normal cortical-evoked potentials before treatment. The latter 2 patients were those who did not show improvement in pain scores after treatment.

DISCUSSION

This study confirms the previous report that the amplitude of cerebral-evoked potentials on magnetic stimulation of paraspinal muscles is depressed in the presence of palpable muscle spasm.[11] The amplitude of P30-N40, N40-P50, and P50-N70 components of cerebral-evoked potentials are noted to be lower in patients with palpable muscle spasm

Table 2. *Clinical outcome measures before and after spinal manipulative therapy*

Patient no.	No. of treatments	VAS(cm)	RMAS(%)		Spasm	
					Before	After
		B/A1	B1/B2	A	R/L	R/L
1	10	4.0/0.3	50.0/54.2	37.5	+/++	–/–
2	15	4.5/3.5	75.0/79.2	22.9	–/±	+/+
3	7	5.5/1.0	54.2/-	12.5	+/–	–/–
4	3	8.0/0	58.3/62.5	4.2	+/+	–/–
5	10	7.0/7.0	60.4/75	37.5	++/++	+/+
6	7	5.2/5.0	50/62.5	68.75	++/++	–/+
7	10	4.5/0	26.7/39.6	0	+/+	–/–
8	10	4.5/0.7	29.2/20.8	8.3	+/+	–/–
9	8	8.0/1.0	73.9/75	4.2	+/++	–/±
10	7	6.0/2.0	22.9/27.1	12.5	+/+	–/±
11	7	3.0/1.0	45.8/52.1	33.3	+/+	–/–
12	7	8.2/0	79.2/79.2	45.8	++/++	+/+
13	7	4.0/3.0	33.3/33.3	22.9	+/±	±/±

Range of motion was measured in centimeters with fingertips to the floor.
The degree of muscle spasm was measured where ++ > + > ± > –.
VAS, Visual analog scale; *RMAS*, Roland-Morris Activity Scale; *B*, before treatment; *A*, after treatment; *B1*, *B2*, first and second measurements before treatment; *R/L*, right/left low back; *A1*, *A2*, first and second measurements after treatment.

Table 3. *Peak to peak amplitudes (μV) of somatosensory evoked potentials to magnetic stimulation to the paraspinal muscles at L2 to L5 level in 13 patients with low back pain before and after spinal manipulative therapy*

Patient no.	P30-N40		N40-P50		P50-N70	
	R pre/post	L pre/post	R pre/post	L pre/post	R pre/post	L pre/post
1	0.3/0.9	0.2/0.8	0.4/1.2	0.3/0.8	1.1/2.0	1.0/1.6
2	1.8/2.0	1.5/2.2	2.0/3.0	2.5/3.2	4.0/4.5	3.5/3.5
3	2.0/1.8	1.8/2.1	2.0/2.0	2.0/1.8	1.1/1.9	0.8/1.9
4	0.3/0.8	1.0/1.5	1.1/1.8	1.5/1.3	2.0/2.0	2.1/1.2
5	0.4/1.0	0.2/1.5	0.7/2.0	0.8/2.0	0.5/2.5	0.5/2.5
6	0.5/1.0	0.9/1.0	0.8/0.5	0.4/0.8	1.0/1.5	1.2/1.3
7	0.3/1.0	0.3/1.9	0.7/0.9	0.7/1.6	0.6/1.0	0.8/2.1
8	0.3/1.2	0.5/1.2	0.8/0.5	0.6/0.8	2.5/3.4	1.2/2.0
9	1.3/1.0	1.1/1.2	0.8/1.8	0.3/0.5	1.8/3.1	1.2/1.8
10	1.0/1.2	1.3/2.0	1.0/1.2	0.4/1.5	1.8/2.2	1.1/2.2
11	1.2/1.3	0.6/2.0	1.0/1.3	0.7/2.3	1.3/3.3	2.0/2.5
12	0.3/1.0	0.2/0.3	0.3/1.0	0.3/0.3	0.5/1.5	1.0/1.2
13	1.0/1.3	2.0/2.0	1.1/1.3	1.2/2.5	1.3/2.0	2.0/2.9

than published normal values and tended to return to normal amplitudes when the muscle spasm improved with treatment. This relation suggests that the paraspinal muscle-evoked cerebral potentials may serve as an objective means to assess muscle spasm.

The usage of the amplitude of cerebral-evoked potentials to measure neuronal activity has a different connotation than the more commonly used latency measurements for neuronal deficits. Whereas the latency of an evoked potential depends on the conduction velocity, the amplitude of the response depends more on the volume of receptors that are available for stimulation. Although the amplitude of these responses varies greatly between individuals and is therefore not clinically of value in measuring conduction abnormalities, it is relatively stable in the same individual over a number of days.[10] The ability to attenuate muscle contraction-evoked cortical potentials by means of vibration and voluntary contraction of muscles has been interpreted as indicating that the muscle-evoked response is caused by direct stimulation of terminal nerve afferents in the muscle.[11] Lotz et al[12] demonstrated that magnetic stimulation of muscles induced muscle contraction by activation of terminal nerve afferents and not by activation of the muscle fibers directly. In addition, evidence favoring an indirect stimulation of muscles comes from the observation that cortical-evoked responses from muscles can be elicited in patients paralyzed by succinylcholine chloride.[13] It is assumed that paraspinal muscle spasm has an effect similar to vibration and voluntary muscle contraction and reduces the number of afferents available for magnetic stimulation. If the muscle spasm saturates the number of available afferent fibers, a diminution of the amplitude of the cortical response would be anticipated. When the spasm diminishes, thereby increasing the availability of 1a-afferents to magnetic stimulation, an increase in the amplitude of the cortical-evoked response would be anticipated.

Although the sample size was small in this study, the evoked potentials appeared to predict which patients were

most likely to improve after treatment. Those patients in whom there was distinct palpable muscle spasm and depressed evoked potentials were most likely to show a reduction of spasm and pain scores. Conversely, the 2 patients with normal evoked potentials before treatment showed no significant improvement in symptoms or in palpable muscle spasm after treatment.

The results of the RMAS scores were somewhat more difficult to evaluate. Although there was a significant improvement in scores after treatment, this did not correlate as well with the degree of pain (VAS), muscle spasm, or evoked responses. The activity scale did improve in absolute values in all except 1 patient; however, only 7 of 13 patients showed >50% improvement in the activity scores. With this value as a cutoff point, the correlation between evoked potentials and RMAS was considerably weaker than that between evoked potentials and muscle spasm or VAS, which is consistent with observations that functional activity measures do not always correlate well either with pain symptoms or physical measures of pathologic conditions.[14,15]

CONCLUSION

Although the treatment approach in this population of patients was manual therapy in the form of muscle massage, stretching, and spinal manipulation, care must be taken in evaluating the significance of this study in terms of manipulation. There were no control subjects included in this study, and the population of patients was heterogeneous. The changes noted in these measurements simply occurred in concert and could have been caused by either spontaneous improvement of patient back pain or as a result of a specific effect of the treatment.

However, it is difficult to discount the close correlation between the evoked cortical responses recorded on magnetic stimulation of paraspinal muscles, palpable muscle spasm, and the evolution of the patient's symptoms. It is this correlation that should lead to a closer examination of the role of muscles in patients with low back pain.

REFERENCES

1. Fischer AA, Chang CH. Electromyographic evidence of paraspinal muscle spasm during sleep in patients with low back pain. Clin J Pain 1985;1:147-54.
2. Johnson EW. The myth of skeletal muscle spasm [abstract]. Am J Phys Med Rehabil 1989;68:1.
3. Roland MO. A critical review of the evidence for a pain-spasm-pain cycle in spinal disorders. Clin Biomech 1986;1:102-9.
4. Lewis S, Simons DC. Myofascial pain: relief by postisometric relaxation. Arch Phys Med Rehabil 1984;65:425-56.
5. Travell JG, Simons DC. Myofascial pain and dysfunction. Baltimore (MD): Williams and Wilkins; 1983.
6. Magora A. Investigation of the relation between low back pain and occupation. Scand J Rehabil Med 1975;7:146-51.
7. Doege TC, Houston TP, American Medical Association. Guides to the evaluation of permanent impairment. 4th ed. Chicago: American Medical Association; 1993.
8. Campbell SM, Clark S, Tindell EA, Forehand ME, Bennett RM. Clinical characteristics of fibrositis. A blinded controlled study of symptoms and tender points. Ortho Rheum 1983; 26:817-26.
9. Tunks E, Crook J, Norman G, Kalaher S. Tender points in fibromyalgia. Pain 1988;34:11-9.
10. Zhu Y, Starr A. Magnetic stimulation of muscle evoked cerebral potentials. Muscle Nerve 1991;14:721-32.
11. Zhu Y, Haldeman S, Starr A, Seffinger MA, Su SH. Paraspinal muscle evoked cerebral potentials in patients with unilateral low back pain. Spine 1993;18:1092-102.
12. Lotz BP, Dunne JW, Daube JR. Preferential activation of muscle fibers with peripheral magnetic stimulation of the limb. Muscle Nerve 1989;24:568-73.
13. Zhu Y, Starr A, Haldeman S, Fu H, Liu J, Wu P. Magnetic stimulation of muscle evoked cerebral potentials by direct activation of nerve afferents: a study during muscle paralysis. Muscle Nerve 1996;19:1570-5.
14. Waddell G, Allan DB, Newton M. Clinical evaluation of disability in low back pain. In: JW Frymoyer, editor. The adult spine. 2nd ed. Philadelphia: Lippincott-Raven; 1997.
15. Haldeman S, Shouka M, Robboy S. Computed tomography, electrodiagnostic, and clinical findings in chronic workers' compensation patients with back and leg pain. Spine 1988;13:334- 50.