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■ Paraspinal Muscle Evoked Cerebral Potentials in Patients with Unilateral Low Back Pain

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Cerebral somatosensory evoked potentials (SEPs) were elicited by magnetic stimulation of paraspinal muscles unilaterally at the L2 and L5 levels in 20 healthy subjects and 16 patients with low back pain and unilateral muscle spasm. A magnetic coil with a mean diameter of 4.7 cm was placed tangentially to the skin. The stimulus strength was sufficient to induce a visible muscle twitch without producing muscle contraction in the legs. The potentials recorded over the scalp consisted of several components (P30, N40, P55, N70, and P90) and were elicited in all subjects. In both healthy and patient subjects, paraspinal muscle evoked potentials were readily elicited. Vibration applied to paraspinal muscles, as well as voluntary contraction of paraspinal muscles, was associated with attenuation of the evoked potentials. This finding suggests that muscle spindle receptors provide the afferent input responsible for the early components of the magnetically evoked cerebral potentials. In patients with unilateral muscle spasm, the amplitudes of P30-N40, N40-P50, and P50-N70 were decreased significantly on the affected side when compared with values on stimulation of the unaffected side, as well as those obtained from control subjects. The cerebral evoked potentials returned to normal amplitude when the muscle spasm subsided following a period of time and after the application of spinal manipulative therapy. The technique has potential for quantitative evaluation of muscle spasm in low back pain. (Key words: paraspinal muscle SEPs, muscle spasm, low back pain)

Muscle pain, tenderness, trigger point localization, and muscle spasm are commonly reported clinical findings in patients with low back pain. Muscle spasm has been defined as the sustained involuntary contraction of the entire muscle that cannot be relieved completely by voluntary effort.²⁶ Muscle spasm may occur in reaction to painful irritative dis-

orders, such as local inflammation, osteoarthritis, and nerve root or peripheral nerve irritation. In certain patients with low back pain, asymmetrical muscle spasm has been reported to produce antalgic scoliosis.⁸ The occurrence and significance of muscle spasm have been poorly quantified due to difficulties in both definition and objective documentation.²³ It has been reported that 13% of patients with low back pain have "tight or contracted" muscles.¹⁷ In other studies, however, significant interobserver errors have been reported, making the diagnoses unreliable.^{1,18} Recognizing these controversies, we have used the term "muscle spasm" in this paper to refer to the palpable changes (hardness, bands, antalgic posturing, trigger points, and localized tenderness) determined on clinical examination. This muscle spasm usually was found on the side of clinical symptoms in the patients studied.

The ability to record cerebral potentials following a brief muscle contraction induced by magnetic stimulation provides a relatively simple method for quantifying muscle afferents originating from a wide variety of skeletal muscles.²⁸ Previous studies from limb muscles have demonstrated that attenuation of cerebral potentials evoked by muscle contraction can be seen during stretching, vibration, or voluntary contraction of a muscle. The possibility that recording of muscle contraction evoked cerebral potentials using magnetic stimulation might provide a method of objective documentation of muscle function in specific clinical conditions is intriguing.

This report examines the brain potentials accompanying magnetic stimulation of the paraspinal muscles and defines how these potentials are modified in patient with unilateral low back pain and palpable muscle spasm.

■ Methods

Twenty healthy volunteers (10 men and 10 women, aged 20 to 55 years) and sixteen patients (9 men and 7 women, aged 23 to 48) with unilateral low back pain and palpable paraspinal muscle spasm were studied. The patients with

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low back pain and palpable paraspinal muscle spasm had received diagnoses of "myofascial pain syndrome" (9 patients) and "low back sprain" (7 patients) (Table 1). The presence of palpable muscle hardness, tenderness, and/or trigger points was confirmed in each patient by at least two of the authors. None of the patients had undergone spinal surgery. The duration of back pain ranged from 1 week to 3 years.

Stimulation. Subjects were tested while lying prone in a bed. They remained awake throughout the procedure. Magnetic stimulation was performed using a Cadwell MES-10 magnetic stimulator (Cadwell Laboratories, Inc., Kennewick, WA). A coil with a mean diameter of 4.7 cm was placed tangentially to the skin overlying the paraspinal muscles, 2–3 cm lateral to the midline. A brief pulse, 0.07 milliseconds in duration, up to 3,000 V at maximal output, was passed through the coil by the discharge of capacitors. The changing magnetic field, which approached 2.0 T, induced electrical currents within the tissue. Stimulus rate was 0.7 Hz. The transformer in the stimulator becomes warm during repetitive stimulation at this rate, which required that it be switched off after every 200 to 250 stimulations.

Magnetic stimulation was applied at the L2 and L5 levels in normal subjects. In the patients with unilateral low back pain, magnetic stimulation was applied to the site where muscle changes were palpated, and to the corresponding site on the spasm-free side. The motor threshold (MT) of magnetic stimulation was defined as the intensity needed to produce just-palpable contraction of the muscles beneath the magnetic coil. Cerebral evoked potentials were usually recorded at 40–50% of maximum output (approximately 1.5 to 2.0 MT). No contraction of leg muscles was produced on stimulation of paraspinal muscles at this stimulus intensity. At the site of stimulation, the subjects experienced a minor, brief contraction that was not associated with pain.

In four patients, paraspinal muscle stimulation was repeated at different times and over several days to determine

the reproducibility of latency and amplitude measurements.

In three normal subjects, percutaneous electrical stimulation of the L2 and L5 paraspinal regions was also performed. A 0.2 millisecond² pulse of constant current was delivered at a rate of two per second. The intensity was adjusted to be three times sensory threshold (ST).

Recording. Ag/AgCl disks, 8 mm in diameter, were attached with electrode cream to the skin of the scalp. Electrode impedance in all electrodes was maintained at similar levels and measured below 2 Kohms. Recording electrodes were placed 2 cm posterior to the Cz position of the international 10–20 system and referenced to Fpz. To reduce the amplitude of stimulus artifact, a ground electrode was placed on the scalp between the pair of recording electrodes, and the wires of the three electrodes were twisted together.

The discharge of the electrical current through the magnetic coil is accompanied by a clicking sound. The cerebral evoked potentials accompanying magnetic stimulation of the paraspinal muscles was found to be unaffected by the application of a masking noise through earphones (two subjects), as was previously noted on magnetic stimulation of the gastrocnemius muscle.²⁸ For this reason all studies were performed without noise-masking. The cerebral potentials were amplified with a gain of 500,000, using a band pass of 5 Hz to 500 Hz, and averaged (usually 100 trials) with a time-base of 120 milliseconds, including a 12 millisecond prestimulus baseline, and filtered between 5 Hz and 1000 Hz. Averages were repeated at each stimulus site. A potential of positive polarity at grid 1 of the amplifier was reflected by a up-going deflection on the trace. Peak latency and peak-to-peak amplitude of each evoked potential component were measured using a cursor on a computer monitor. The components of the evoked potential were designated by their polarity (P or N for positive or negative) and their approximate peak latencies in milliseconds. The T-test for related measures between the means was performed to evaluate the significance of differences.

Table 1. Patients With Low Back Pain

Patient	Age	Diagnosis	Site of Pain and Spasm	Duration of pain
1	35	Myofascial pain syndrome	Rt. L2,L3	1 year
2	28	Myofascial pain syndrome	Lt. L2,L3	6 months
3	33	Low back sprains	Lt. L5,S1	3 weeks
4	24	Myofascial pain syndrome	Lt. L4,L5	10 months
5	27	Low back sprains	Lt. L4,L5	1 week
6	25	Myofascial pain syndrome	Rt. L1,L2	5 weeks
7	36	Myofascial pain syndrome	Rt. L4,L5	2 months
8	44	Low back sprains	Rt. L5,S1	5 months
9	41	Low back sprains	Lt. L4,L5	6 months
10	23	Myofascial pain syndrome	Lt. L2,L3	2 years
11	48	Low back sprains	Rt. L3,L4	3 years
12	33	Low back sprains	Lt. L4,L5	2 weeks
13	27	Myofascial pain syndrome	Rt. L5,S1	3 years
14	39	Myofascial pain syndrome	Rt. L4,L5	4 months
15	33	Myofascial pain syndrome	Lt. L4,L5	3 months
16	35	Low back sprains	Lt. L4,L5	11 months

In four normal subjects, repeat paraspinal muscle stimulation on subsequent days was performed to determine the reproducibility of latency and amplitude measurements.

Two procedures were used to distinguish the mechanisms by which paraspinal muscle evoked cerebral potentials were modified by muscle activation: the isometric voluntary contraction of paraspinal muscles (3 subjects) and the application of vibration to paraspinal muscles (5 subjects). Isometric active contraction of the paraspinal muscle was performed with the subject lying prone. During this procedure, the legs of the subjects were fixed to the bed manually by the examiner while the shoulders were raised actively from the bed to a height of approximately 15 cm. Vibration was produced by activating a rod that had a 4 cm diameter ring at its tip and which vibrated at a sinusoidal frequency of 60 Hz. The ring was applied to the skin overlying the paraspinal muscles 2 cm rostral to the site of application of the magnetic coil. The displacement of the vibrating rod was 5 mm.

■ Results

Normal Subjects

Magnetic stimulation applied to paraspinal muscles evoked reproducible somatosensory evoked potentials (SEPs) recorded from the scalp in all normal subjects. SEPs of stimulation at the L2 or L5 level consist of several components: P30, N40, P55, N70, and P90 (Figure 1). The latency of the earliest cortical positivity, P30, decreased by approximately 3 milliseconds as the stimulated site moved rostrally from the L5 to L2 level (Table 1). The amplitudes of the early P30/N40 and N40/P55 wave forms were at the range of 1.5 to 3.0 μV . There was no significant difference ($P > 0.05$) between the potentials elicited on stimulation of the two sides of the spine.

SEPs evoked by magnetic stimulation of paraspinal muscles were able to be replicated with regard to form and latency at the same levels. Repeat examination in four healthy subjects tested several times on one day and over several different days resulted in a

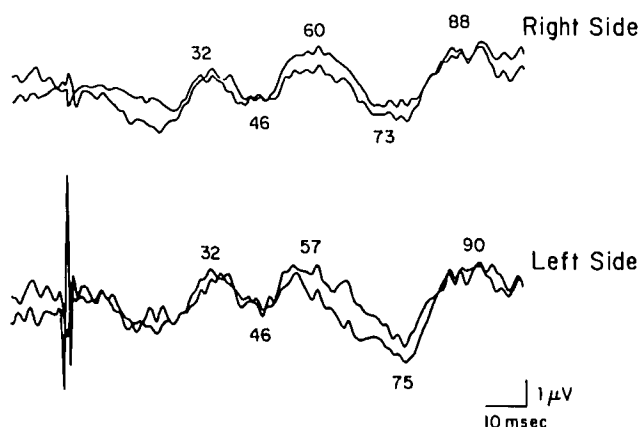


Figure 1. Examples of cerebral evoked potentials of a healthy subject after magnetic stimulation of paraspinal muscle. Each of the duplicate averages consists of 100 sweeps. Positivity upward in this and all subsequent figures.

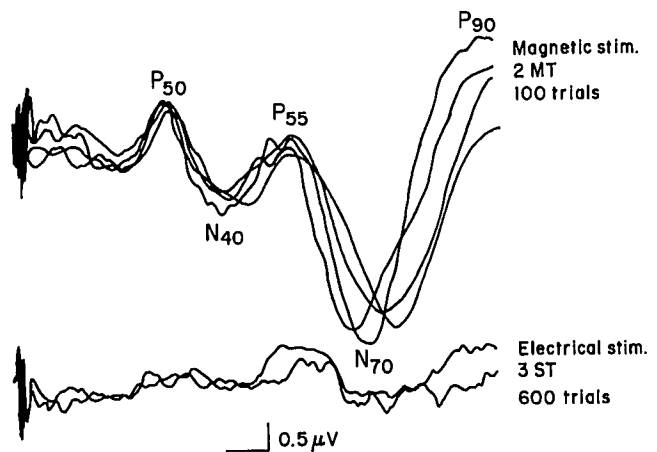


Figure 2. Comparison of a number of tests of cerebral evoked potentials after magnetic stimulation and electrical stimulation to the L2 paraspinal region at the same site in the same subject. Note that no recognizable early components of the evoked potentials could be defined from the background noise after electrical stimulation.

maximum amplitude variation of less than 20%, while latency varied by less than 1.0 ms for the early components (P30, N40, P55) (Figure 2, upper trace).

Percutaneous electrical stimulation of paraspinal tissues elicited very poorly defined cerebral potentials in three normal subjects (Figure 2, lower trace). No consistent early components could be defined.

Effects of Muscle Contraction

Isometric contraction of paraspinal muscles exerted a significant attenuation of the P30–N40 component of the cerebral potentials evoked by magnetic stimulation of the paraspinal muscle (to 60%; $P < 0.01$, five subjects) (Figure 3).

Effects of Vibration

Sustained vibration at 60 Hz applied to the paraspinal muscles significantly diminished the P30–N40 and N40–P55 components of the cerebral potentials

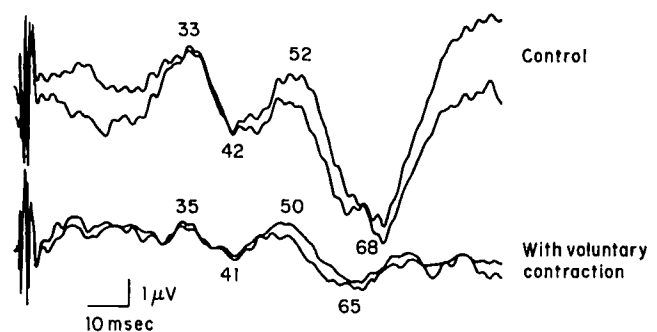


Figure 3. The effects of voluntary contraction of paraspinal muscle on cerebral potentials evoked by magnetic stimulation. Note the significant decrease of the amplitude of each component. Each average consists of 100 sweeps.

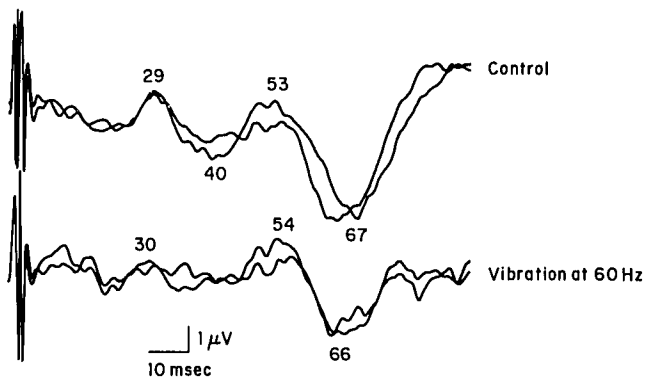


Figure 4. The attenuation exerted by vibration on cerebral potentials evoked after magnetic stimulation of paraspinal muscle in a healthy subject.

evoked by magnetic stimulation of the ipsilateral paraspinal muscle ($P < 0.01$, five subjects) (Figure 4).

Patients With Low Back Pain

Figure 5 shows that in all sixteen patients, the amplitudes of P30–N40, N40–P50, and P50–N60 components of the cerebral potentials obtained on stimulation of the side with palpable muscle spasm were significantly decreased in comparison to those obtained from the spasm free side ($P < 0.05$). The response was also significantly reduced from that obtained from healthy subjects ($P < 0.01$) (Figure 5). The difference in the amplitude of P30–N40, N40–P50 components on the spasm free side of patients and normal subjects was not significant ($P > 0.05$).

Three patients were tested after treatment using spinal manipulation when their muscle spasm and symptoms had subsided. The cerebral potentials evoked by magnetic stimulation to the paraspinal muscles on the side of previous spasm increased to amplitudes similar to that noted on the spasm free side (Figure 6).

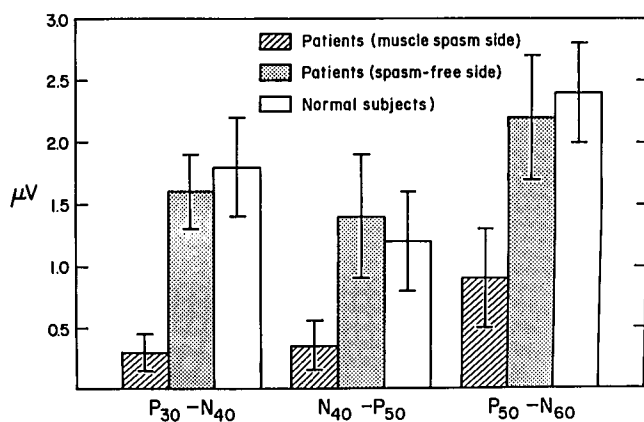


Figure 5. Comparison of peak-to-peak amplitudes of paraspinal muscle SEPs of normal subjects ($n = 20$) and patients with unilateral low back pain ($n = 16$).

Discussion

Our study suggests the existence of unilateral muscle physiological changes in a selected group of low back pain patients by demonstrating that paraspinal muscle SEPs elicited on stimulation of the symptomatic side is significantly different from that recorded on stimulation of the unaffected side. The association of muscle spasm with low back pain was reviewed by Lewis as early as 1920.¹⁵ Numerous references to contracted, hard or tight paraspinal muscles can be seen throughout the literature on back pain. Despite the almost universal opinion of clinicians that muscle abnormalities can be detected in patients with back pain, the scientific evidence for muscle spasm has been scant and its very existence as a clinical finding, disputed.¹⁴ Although standard needle electromyography (EMG) has shown no significant abnormalities associated with clinical muscle findings, certain specialized EMG studies have been reported to show changes in the muscles. Fisher and Chang⁸ reported abnormally increased EMG activity during sleep in paraspinal muscles on the side of palpable muscle spasm in patients with low back pain. Furthermore, increased EMG activity associated with but not necessarily at trigger points in acute conditions may be eliminated with local anesthetics or spinal anesthesia, which obliterate the trigger point.⁷

There are studies devoted to recording somatosensory evoked potentials after bilateral electrical stimulation to the paraspinal region¹² or magnetic stimulation of high intensity to the spinal nerve root.²⁷ Electrical stimulation to paraspinal tissues is limited by the small amplitude of the response. Furthermore, the use of bilateral stimulation trades the increase in size of the response for the loss of ability to detect unilateral lesions. The use of magnetic stimulation to elicit somatosensory evoked potentials of the spinal nerve roots requires repetitive stimulation with extremely high intensity which produces vigorous contraction of both spinal and lower limb muscles. Neither of these techniques have been widely accepted as useful clinical tests. On the other hand, the use of magnetic stimulation as demonstrated in this paper has the advantage of being easily elicited from multiple different muscles, especially the trunk muscles, which are usually difficult to test.²⁸

The mechanism by which the paraspinal muscle SEPs decrease in amplitude on the side of spinal muscle spasm has not been elucidated fully. Paraspinal muscle SEPs are attenuated by vibration, which suggests that Ia muscle afferent fibers are responsible for the generation of these cerebral potentials. Presynaptic inhibition of Ia input in the spinal cord and muscle spindle receptor occupancy are probably the mechanisms involved, since both lumbar cord- and cerebral-evoked potentials are inhibited during vibration.²

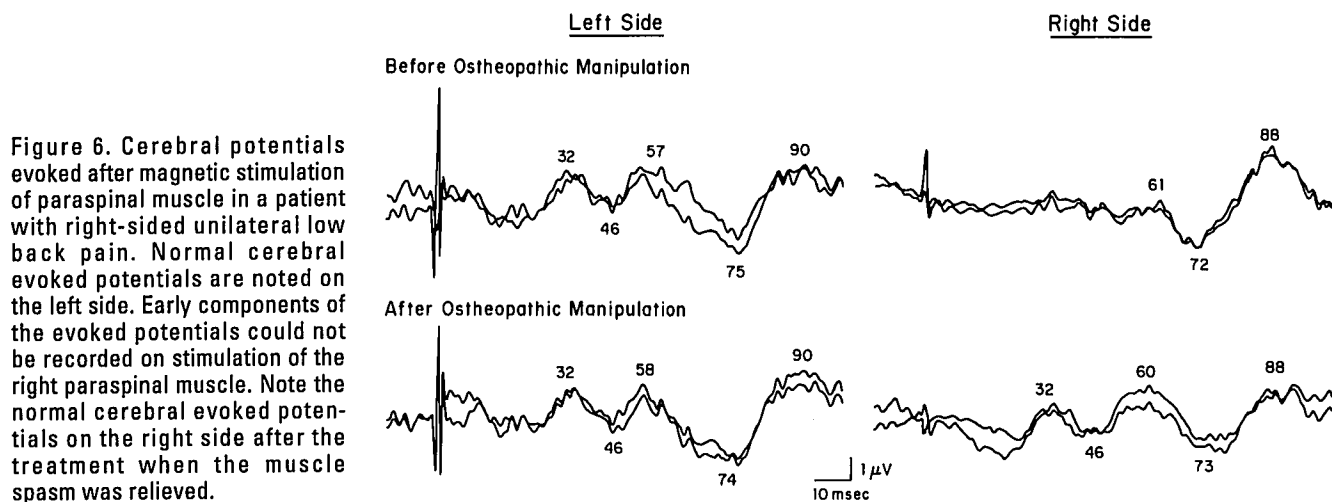


Figure 6. Cerebral potentials evoked after magnetic stimulation of paraspinal muscle in a patient with right-sided unilateral low back pain. Normal cerebral evoked potentials are noted on the left side. Early components of the evoked potentials could not be recorded on stimulation of the right paraspinal muscle. Note the normal cerebral evoked potentials on the right side after the treatment when the muscle spasm was relieved.

One possible mechanism for the attenuation of the magnetically evoked potentials from painful paraspinal muscles is through some form of "gating" mechanism.²⁵ In mammals, the execution of movement (active muscle contraction) is mediated by activity in alpha-motor neurons that serve as the final common pathway to muscles. For any movement, the number of alpha-MNS involved is likely to be outweighed significantly by the number of muscle proprioceptive afferent and fusimotor efferent fibers, which are also activated and whose discharge may contribute to the control of the movement.²² During movement, there is also an attenuation of the transmission of somatosensory afferent information in the nervous system.^{3,4,10} The site of this inhibitory process is rostral to the lumbar cord, since cortical but not lumbar evoked potentials were affected. Thus, in humans, central mechanisms modulating neurons in the dorsal columns nuclei, thalamus, or cerebral cortex are probably responsible for the observed inhibition.^{24,2} The present study shows that cerebral potentials are inhibited during paraspinal muscle spasm. Unfortunately, because of the difficulty in recording lumbar spinal evoked potentials of magnetic stimulation of these muscles, no data is available that defines the site of the inhibitory process.

Another intriguing possibility relates to the observation that muscle spindle activity is enhanced in areas of muscle tenderness. Friction et al⁹ recorded electromyographic changes from trigger points after stimulation of these muscle fibers, indicating the possibility that the muscle spindles may be sensitized in these areas. Hubbard¹³ claims to have recorded increased activity directly from muscle spindles at the trigger points in patients with myofascial pain. As magnetically-induced muscle contraction evoked cerebral potentials appear to originate from the stimulation of muscle spindle afferents, the excessive activity in these spindles would be expected to attenuate the response in the manner observed. Because magnetically-induced muscle contraction evoked cerebral potentials can be influenced by vibration and stretching of muscle, it is possible that the effect of unilateral paraspinal muscle abnormalities on these responses is not the result of active muscle contraction, but of variation in the activity of the muscle spindles. The pathophysiological basis of muscle pain is not yet well defined. Animal experimentation has shown that muscle nociceptors respond to intense mechanical or thermal stimuli, and to excessive muscle contraction.^{19,20} This finding has been the proposed basis of several different treatment

Table 2. Peak latencies and peak to peak amplitudes of SEPs to magnetic stimulation to the paraspinal muscles at L2 and L5 level in 20 healthy subjects.

A. Peak latency (mean ± SD, in msec)	P30	N40	P55	N70	P90
L2	29.5±1.7	41.2±4.4	52.0±4.1	67.7±6.2	88.9±5.9
L5	32.6±1.8	45.4±3.9	57.1±3.7	73.2±7.2	90.5±5.6
B. Peak-peak amplitude (mean ± SD, in uv)	P30-N40	N40-P55	P55-N70	N70-P90	
L2	2.13±0.45	1.55±0.58	2.37±0.51	3.55±0.78	
L5	2.25±0.49	1.67±0.31	2.23±0.44	3.34±0.69	

methods for low back pain, including the use of muscle-relaxing drugs, biofeedback, physical therapy, and even analgesics. Mooney and Robertson²¹ found that muscle spasm in hamstring muscles decreased when local anesthetics were injected into facet joints; however, clinical trials using muscle relaxants have provided conflicting results regarding their therapeutic efficacy and mechanism of action,⁵ and there is considerable debate as to whether the effects of these drugs are on muscle activity. Physical therapy, particularly stretching, has been suggested to reduce pain associated with muscle spasm,¹⁶ a claim that has yet to be supported. Brain potentials evoked by induced paraspinal muscle contractions might provide objective measures of changes in muscle receptor activation that accompany muscle spasm and provide information on the mechanisms of action of these treatment protocols.

The normalization of magnetically-induced muscle contraction cerebral responses following manipulation must be interpreted with caution. One common theoretical explanation for the effectiveness of manipulation as a treatment method for patients with low back pain has been its effect on muscles. The two most frequently quoted trials suggesting a reduction in muscle spasm following manipulation are extremely crude and of questionable validity.^{6,11} It has also been proposed that manipulation may cause a rapid stretching of spinal muscles, resulting in relaxation. The rapid stretching of muscles in experimental animals has been found to be followed by relaxation not only of motor activity in muscle, but also of muscle spindle activity. These observations would fit into the muscle spindle theory of muscle pain. On the other hand, the observation noted here may simply reflect the natural history of back pain as there are no controls and the number of patients studied is small. At this time, it is not possible to draw any conclusions regarding the physiological mechanism of spinal manipulation. Any process associated with low back pain and that normalized on reduction of the pain could explain our observed changes in muscle physiology. There does appear to be a relationship between unilateral back pain and palpable muscle findings, however, which holds promise for advancing our understanding of the physiologic processes involved in the genesis of back pain.

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