Objective: The aim of the current study was to investigate changes in the intrinsic inhibitory interactions within the somatosensory system subsequent to a session of spinal manipulation of dysfunctional cervical joints.

Method: Dual peripheral nerve stimulation somatosensory evoked potential (SEP) ratio technique was used in 13 subjects with a history of reoccurring neck stiffness and/or neck pain but no acute symptoms at the time of the study. Somatosensory evoked potentials were recorded after median and ulnar nerve stimulation at the wrist (1 millisecond square wave pulse, 2.47 Hz, 1 × motor threshold). The SEP ratios were calculated for the N9, N11, N13, P14-18, N20-P25, and P22-N30 peak complexes from SEP amplitudes obtained from simultaneous median and ulnar (MU) stimulation divided by the arithmetic sum of SEPs obtained from individual stimulation of the median (M) and ulnar (U) nerves.

Results: There was a significant decrease in the MU/M + U ratio for the cortical P22-N30 SEP component after chiropractic manipulation of the cervical spine. The P22-N30 cortical ratio change appears to be due to an increased ability to suppress the dual input as there was also a significant decrease in the amplitude of the MU recordings for the same cortical SEP peak (P22-N30) after the manipulations. No changes were observed after a control intervention.

Conclusion: This study suggests that cervical spine manipulation may alter cortical integration of dual somatosensory input. These findings may help to elucidate the mechanisms responsible for the effective relief of pain and restoration of functional ability documented after spinal manipulation treatment. (J Manipulative Physiol Ther 2010;33:178-188)

Key Indexing Terms: Somatosensory Evoked Potentials; Neuronal Plasticity; Spinal Manipulation; Sensory Filtering; Sensorimotor Integration; Chiropractic
information at the level of the primary somatosensory cortex.23-25 The N30 SEP peak reflects central sensori-motor integration processing involving primary sensory cortex, primary motor cortex, premotor cortex, and deeper brain structures such as the basal ganglia.26-33

One possible mechanism responsible for altering the amplitude of the cortical N20 and N30 SEP components after spinal manipulation is altered reciprocal sensory inhibition, that is, the filtering of afferent information by the somatosensory system. Reciprocal sensory inhibition enhances the contrast between stimuli, so that information from adjacent body parts is perceived and processed separately. One method, first used in the early 1980s,34-38 to investigate reciprocal sensory inhibition is to stimulate 2 peripheral nerves simultaneously while recording SEPs. By comparing the amplitudes of SEP peaks obtained by stimulating 2 nerves simultaneously, for example, the median and ulnar nerves (MU), with the amplitude obtained from the arithmetic sum of the SEPs elicited by stimulating the same 2 nerves separately (M + U), the resulting ratio (MU/M + U) can be used as a measure of the central interaction between afferent inputs from these 2 peripheral nerves before and after an intervention, such as a 20-minute repetitive muscle contraction task.39

This study sought to investigate whether spinal manipulation alters the intrinsic inhibitory interactions within the somatosensory system by comparing the amplitudes of SEP peaks obtained by stimulating 2 nerves simultaneously with the amplitude obtained from the arithmetic sum of the SEPs elicited by stimulating the same 2 nerves separately.

METHODS

Subjects

Thirteen subjects (8 women, 5 men), aged 18 to 40 (mean age, 28.0 ± 6.3 years), with no history of neurologic disorders participated in this study. Twelve of the subjects were deemed to be right-handed (mean laterality quotient, 89.9%; range, 58.3%-100%) and one left-handed (laterality quotient, 75.0%), using the Edinburgh handedness questionnaire.40 The subjects were required to have a history of reoccurring neck problems. However, at the time of the experiment, all subjects were required to be pain-free. This was done to assess the potential effects of joint manipulation delivered to dysfunctional joints in the absence of acute pain, as acute pain on its own is known to induce a significant reduction of the postcentral N20-P25 SEP complex and a significant increase of the N18 wave.41 All subjects were also screened for evidence of vertebral artery ischemia and questioned regarding other contra-indications to cervical spine manipulation such as a history of previous fractures; high blood pressure; and metabolic, inflammatory, or neoplastic disease. Informed consent was obtained from each participant. The University of Auckland Human Participant Ethics Committee approved the study.

SEP Stimulating and Recording Parameters

The stimulating electrodes (cathode proximal) were placed over the median and ulnar nerve at the wrist of the dominant arm. Stimuli (at 1 × motor threshold) consisted of electrical square pulses of 1-millisecond duration delivered at a rate of 2.47 Hz, a rate that does not lead to SEP peak attenuation,42 through 7-mm Ag/AgCl disposable adhesive electrodes (Hydrospot from Physiometrix [Physiometrix Inc, Billerica, MA]) (impedance < 5 kΩ). At motor threshold, the sensation of transcutaneous electrical stimulation is usually described as a slight “buzzing” sensation accompanied by the sensation of the low level involuntary contractions of the muscles innervated by the stimulated nerve. Motor threshold was defined as the lowest intensity that produced a visible muscle contraction of the abductor pollicis brevis muscle for median nerve stimulation or abductor digiti minimi muscle for ulnar nerve stimulation. The stimulated arm was splinted to ensure stable stimulating conditions throughout the experiment. During the recording periods, the subjects were asked to close their eyes, sit as still and quietly as possible, and the lights in the room were turned off.

All SEP recording electrodes (7-mm Ag/AgCl disposable adhesive electrodes, Hydrospot from Physiometrix, impedance < 5 kΩ) were placed according to the International Federation of Clinical Neurophysiologists recommendations.25 Recording electrodes were placed on the ipsilateral Erb’s point, over the C6 spinous process ( Cv6), and 2 cm posterior to contralateral, central, and frontal scalp sites C3/4 and F3/4, which will be referred to as Cc’ and Fc’, respectively. Cc’ and Fc’ recording electrodes were referenced to the contralateral earlobe. The C6 spinous electrode was referenced to the anterior neck (tracheal cartilage). The Erb’s point electrode was referenced to the contralateral shoulder. Finally, the central Cc’ electrode was also referenced to the contralateral shoulder as SEP components originating from subcortical regions are best recorded with a noncephalic reference.41 A ground electrode was attached to FPz, on the forehead. All electrodes were securely fastened and held in place throughout the duration of the data recording session. During the data recording sessions, the subjects were installed in a quiet room and seated in a reclining chair. Throughout the course of the experiment, the subjects were asked to sit still and as quietly as possible. During the SEP recordings, the lights in the room were also turned off and subjects’ eyes were closed. Figure 1 shows a picture of the experimental setup with a subject positioned for a chiropractic manipulation of the cervical spine. Note the stimulating electrodes are attached to the subject’s right arm, and the recording electrodes are attached over the anterior neck and over the scalp, which are held in place with Velcro straps (Fig 1).
Experimental Protocol

The subjects were asked to attend 2 sessions. They were screened for the presence of joint dysfunction by a registered doctor of chiropractic. As the most reliable spinal dysfunction indicators relating to the cervical spine are tenderness on palpation of the dysfunctional joint and passive intervertebral and global motion of the cervical spine, spinal dysfunction was for this study defined as the presence of both restricted intersegmental range of motion and tenderness to palpation of the joint at at least one cervical spine segment. If the subjects were judged to have cervical spine dysfunction (according to the set criteria), the relevant information (including detailed health history) was then obtained from the subject. All subjects were screened for evidence of potential vertebral artery ischemia as well as other contraindications for manipulation.

After assessment of joint dysfunction, 3 baseline SEP trials were carried out in a randomized order: 1 after stimulation of the median nerve individually (M), 1 after stimulation of the ulnar nerve individually (U), and 1 trial after simultaneous stimulation of both nerves (MU). These 3 recordings were repeated immediately after either a control intervention or after the spinal manipulation intervention (high-velocity, low-amplitude). The order of the interventions was randomized. If the manipulation intervention occurred first, there was at least 4 weeks between the 2 experimental sessions. The passive head movement control experiment was not intended to act as a sham manipulation but to act as a physiologic control for possible changes occurring due to the cutaneous, muscular, or vestibular input that would occur with the type of passive head movement involved in preparing a subject/patient for a cervical manipulation. The passive head movement control intervention was carried out by the same chiropractor who had prechecked the subjects for spinal dysfunction and who performed the spinal manipulations for the spinal manipulation experiment. The passive head movement intervention involved the subject’s head being passively laterally flexed and slightly extended and rotated to a position in which the chiropractor would normally manipulate that person’s cervical spine and then returning the subject’s head back to neutral position. This was repeated to both the left and the right. However, the experimenter was particularly careful not to put pressure on any individual cervical segment. Loading a joint, as is done before spinal manipulation, has been shown to alter paraspinal proprioceptive firing in anesthetized cats and was therefore carefully avoided by ending the movement before end-range-of-motion when passively moving the subjects’ heads. No spinal manipulation was performed during any passive head movement experiment.

Data Collection and Analysis

Each M, U, and MU trial (800 sweeps at 2.47 Hz) took 5.4 minutes to record (ie, a total of 16.2 minutes for all 3 trials). In addition, the N9 peak amplitude from the Erb’s point recording site was analyzed, which took an additional 30 to 60 seconds per trial and the N9 MU/M + U ratio was calculated which took an additional 30 to 60 seconds. This meant that each condition (baseline, postmanipulation, and postcontraction) took at least 18 minutes to collect, allowing for an additional trial to be collected to satisfy the data inclusion criteria if necessary. All postintervention SEPs were recorded within 25 minutes after the intervention. This was done as previous research has shown that sensorimotor processing alterations, as measured by altered SEP peak amplitudes observed after spinal manipulation may only last about 20 to 30 minutes after the intervention before returning to baseline values.

The signals were band-passed filtered (3-1000Hz) (−6 dB octave roll-off), amplified (gain 100 000), and then passed to a National Instruments Data Acquisition Board (NI-AT-MIO-46E-3) via a specially shielded cable and National Instruments Cable box (SC2056) (LabView 7, a commercial software package controlled the NI-AT-MIO-46E-3 board). The LabView program controlled the data acquisition, signal averaging, and graphing functions for data analyses. The electroencephalography was digitized at a sample rate of 5000 samples per second and recorded with a sweep length of 55 milliseconds (5 milliseconds prestimulus and 50 milliseconds poststimulus). A total of 800 sweeps were averaged and displayed on an analysis.
panel from which the waveforms of interest were measured for amplitude and latency. Only trials with a stable peripheral nerve volley (N9 peak amplitude) were included for analysis. This was achieved by only including trials for analysis if the N9 SEP peak amplitude was within ±10% of baseline values and that the N9 MU/M + U ratio was within the range of 0.92 to 1.08. At motor threshold stimulation, the SEP ratio (MU/M + U) calculated for the N9 SEP peak amplitude, which is recorded over Erb’s point should equal one to indicate that no suppression of the electrical signal is occurring at the peripheral level (i.e., at the brachial plexus).

Before any SEP peak analysis, the data files were coded by an independent person to reduce any bias during SEP peak amplitude and latency analysis. The SEP amplitudes were measured, from the averaged (800 sweeps) nonrectified traces, from the peak of interest to the preceding or succeeding peak of opposite deflection, according to international recommendations, and past studies in this field. The SEP latencies were measured at the peak of the waveform of interest. The amplitude and latency of the peripheral N9, the spinal N11 and N13, the far-field P14 and N18 potentials, the parietal N20 (N20-P25 complex), and the frontal N30 (P22-N30 complex) were identified and measured. This was done for the individual median (M) and ulnar (U) nerve recordings, the simultaneous median and ulnar (MU) recordings, and from the traces derived from the arithmetic sum of the individual M and U recordings. The way in which the M + U traces are created from the individual M and U recordings have been previously published.

Finally, the MU/M + U SEP peak ratios were calculated. This was achieved by dividing the amplitudes of the SEP peaks obtained by stimulating the median and ulnar nerves simultaneously (MU) by the amplitude value being obtained from the arithmetic sum of the SEPs elicited by stimulating the same nerves separately (M + U). After all the SEP peak amplitudes and latencies had been measured from the M, U, and M + U traces, the data were decoded, grouped according to intervention, and transferred into SPSS statistical software (version 11.5 for Windows; SPSS Inc, Chicago, IL) for statistical analysis.

For statistical analysis, a multifactorial repeated measures analysis of variance (ANOVA) was initially run for the averaged MU/M + U ratio data for each SEP component, with INTERVENTION (control and manipulation) and TIME (pre and post) as factors. If significance was found, a one-way repeated measures ANOVA was run separately for the control and manipulation data with TIME (pre and post intervention) as factor. Further post hoc analysis was not necessary because only 2 levels were present in this analysis. The level of significance was set at $P < .01$ to account for the multiple ANOVAs.

As the individual median and ulnar nerve SEP peak amplitudes and latencies, as well as the averaged MU and M + U data were not normally distributed, nonparametric Wilcoxon signed ranks tests were performed to compare the data obtained pre and post both the control and manipulation interventions. Each SEP component was analyzed separately. The level of significance was set at $P < .01$ to account for the multiple tests.

**RESULTS**

The averaged baseline recordings of the median and ulnar nerves elicited SEPs of which the amplitudes of N11, N13, P14-N18, N20-P25, and P22-N30 SEP peak complexes were always smaller than the amplitude of the arithmetic sum of the individual SEPs. This is in accordance to previous research. The frontal P22-N30 peak ratio is significantly reduced after the manipulation intervention and is marked with an asterisk. This decrease in SEP ratio represents an increase of inhibition of the dual input from the 2 peripheral nerves occurring at the cortical level.

![Fig 2. Bar graph of averaged normalized SEP ratios (MU/M + U) ± SE showing before and after the control intervention (top bar graphs) and before and after the cervical manipulation intervention (bottom bar graphs). The normalized values have been calculated by dividing the postintervention SEP MU/M + U ratios by the preintervention SEP MU/M + U ratio and thus reflect the degree of change for each peak ratio that occurred after the interventions. The frontal P22-N30 peak ratio is significantly reduced after the manipulation intervention and is marked with an asterisk. This decrease in SEP ratio represents an increase of inhibition of the dual input from the 2 peripheral nerves occurring at the cortical level.](image-url)
one-way repeated measure ANOVA revealed a significant effect of the factor “TIME” for the manipulation intervention data \([F_{(1,11)} = 19.42; P = .001]\). Figure 2 depicts the averaged normalized changes in the ratios of each SEP component that have occurred after the interventions. The normalized values have been calculated by dividing the postintervention SEP MU/M + U ratios by the preintervention SEP MU/M + U ratio and thus reflect the degree of change for each peak ratio that occurred after the interventions. This change represented on average a 17%
decrease for the P22-N30 SEP ratio postmanipulation (Fig 2). There were no significant changes after the passive head movement intervention. Figure 3 demonstrates in one representational subject the changes in the difference between the MU and M + U traces before and after the cervical manipulations and that no such changes occur after the control condition. Note that there is a greater difference between the MU and M + U cortical P22-N30 SEP amplitude postmanipulation. This would result in a decrease in the MU/M + U SEP ratio for the P22-N30 SEP complex postmanipulation and reflect an increase in surround-like inhibition (Figs 2 and 3).

The individual median nerve P22-N30 SEP peak amplitude also decreased significantly \((P = .005\) with Wilcoxon signed ranks test) after the cervical manipulations (see Table 1). This represented an 18% decrease in amplitude of the averaged median nerve P22-N30 SEP peak amplitude. There were no significant changes for the M + U data for any SEP peak after the spinal manipulation intervention (Fig 4). For the MU data, the P22-N30 SEP component decreased significantly \((P = .009\) with Wilcoxon signed ranks test) after the cervical spine manipulations (Figs 4 and 5). This represented a 24% decrease in amplitude of the averaged MU P22-N30 SEP peak amplitude. Figure 4 depicts the averaged normalized changes in the MU and M + U data of each SEP component that have occurred after the interventions. The bar graphs are of the averaged “normalized” SEP peak MU (top graphs) and M + U (bottom graphs) data ± SE showing pre and postcontrol (left graphs) as well as pre and postmanipulation values (right graphs). The normalized values have been calculated by dividing each individual's the postintervention SEP MU and M + U amplitudes by their preintervention SEP MU and M + U amplitudes and thus reflect the degree of change for each SEP peak MU and M + U data that occurred after the interventions. Note that the frontal P22-N30 normalized MU amplitude was significantly decreased after the manipulation intervention and is marked with an asterisk. This indicates that the changes in the P22-N30 ratio are due to a surround-like inhibition and not merely due to an increase in either the individual median and/or ulnar nerve SEP peaks.

**Fig 4.** Bar graph of averaged normalized SEP peak MU (top graphs) and M + U (bottom graphs) data ± SE showing pre and postcontrol (left graphs) as well as pre and postmanipulation values (right graphs). The normalized values have been calculated by dividing each individual's the postintervention SEP MU and M + U amplitudes by their preintervention SEP MU and M + U amplitudes and thus reflect the degree of change for each SEP peak MU and M + U data that occurred after the interventions. Note that the frontal P22-N30 normalized MU amplitude was significantly decreased after the manipulation intervention and is marked with an asterisk. This indicates that the changes in the P22-N30 ratio are due to a surround-like inhibition and not merely due to an increase in either the individual median and/or ulnar nerve SEP peaks.

**DISCUSSION**

The major finding in this study was that a single session of spinal manipulation of dysfunctional cervical joints resulted in improved suppression of SEPs, evoked by dual upper limb nerve stimulation, at the cortical level of the lemniscal pathway. More specifically, the improved...
suppression of dual input was evident for the frontal P22-N30 SEP component. This study extends previous work that has demonstrated attenuated parietal N20 and frontal N30 SEP components, reflecting altered cortical processing, for 20 to 30 minutes postmanipulation.22

Evidence for Cortical Neural Plastic Changes After Spinal Manipulation

The current study findings suggest that the initial changes that occur after spinal manipulation occur at the cortical level. This is in agreement with previous research.22 The peripheral N9 peak, representing the afferent volley in the brachial plexus,54-56 was maintained stable in this experiment. The changes observed in this study therefore most likely reflect central changes.

However, although the P14 and N18 SEP components, known to originate at the level of the brainstem,23,55,57-60 did not show any changes in this study, the design of the study limits the ability to rule out the possibility that subcortical changes did occur. It is generally agreed that although 500 sweeps (and the current study averaged 800 sweeps per trial) are sufficient to record reliable Erbs and cortical SEP potentials, far-field potentials such as subcortical P14-N18 do generally require a higher number of averaged sweeps.24,25 The possibility for subcortical SEP changes after spinal manipulation does therefore need further investigation.

The Frontal P22-N30 SEP Peak Changes

The changes observed in the current study only occurred for the frontal N30 component of the SEP peaks. Although some authors suggest this peak is generated in the postcentral cortical regions (ie, S1),26-28 most evidence suggests that this peak is related to a complex cortical and subcortical loop linking the basal ganglia, thalamus, premotor areas, and primary motor cortex.29-33 The frontal N30 peak is therefore thought to reflect sensorimotor integration.31 The decreased frontal N30 SEP peak ratio observed in the current study therefore suggests that there may be an increase in surround inhibition or filtering of sensory information from the upper limb occurring somewhere in these cortical and subcortical loops linking the basal ganglia, thalamus, premotor areas, and primary motor cortex for at least 20 minutes immediately after spinal manipulation. Impaired surround inhibition before spinal manipulation may account for this finding. The SEP ratio changes appear to be due to an increased inhibition of the dual peripheral input, as the MU data significantly decreased for this SEP component after the manipulation intervention.

The passive head movement SEP experiment demonstrated that no significant changes occurred after a simple movement of the subject’s head. The results after manipulation are therefore not simply due to altered input from vestibular, muscle, or cutaneous afferents as a result of the doctor of chiropractic’s touch or due to the actual movement of the subject’s head. This therefore strengthens the argument that the results in this study are more likely specific to the delivery of the high-velocity, low-amplitude thrust to the dysfunctional joints. The passive head movement experiment was to control for the potential neural changes due to the afferent traffic resulting from touch and head movement alone. It was not intended to be a sham manipulation.

The individual median nerve (M) frontal P22-N30 SEP peak amplitude also decreased significantly after spinal manipulation in the current study. This is in agreement with previous research.22 The individual M changes observed for this peak after spinal manipulation appears to be more robust than the changes observed previously for the parietal N20-P25 SEP component, known to originate in the primary somatosensory cortex,23-25 as no change to this peak was observed in the current study compared to what has been shown previously.22

The Parietal N20 SEP Peak

No changes were observed for the parietal N20-P25 SEP peak component after spinal manipulation in the current study. Previous research has shown that cervical spine manipulation attenuates both frontal N30 and parietal N20 SEP peak amplitudes after median nerve stimulation at the wrist.22 It was therefore surprising to find no changes to the parietal N20 SEP component. It is possible that some of
the subjects in the previous study may have experienced some discomfort after the spinal manipulations, as the presence of pain alone is known to induce a significant reduction of the postcentral N20-P25 complex.\textsuperscript{11} Although none of the subjects reported any discomfort after the manipulations, this could be a possible explanation for the significant reduction of the parietal N20-P25 in the previous experiment\textsuperscript{22} because this was not observed in the present study. However, it is also possible that cervical spine manipulation(s) alters the afferent information originating from the cervical spine (eg, from joints and muscles), which in turn can alter the way that the 3b pyramidal cells in the primary somatosensory cortex (S1) respond to any subsequent afferent input such as the median nerve stimulation. It would be reasonable to expect different degrees of such changes in different people, which could also account for altered parietal N20-P25 SEP peak amplitudes in some subjects and not in others.

The current study findings do suggest that the reduced parietal N20 changes observed previously\textsuperscript{22} are not due to enhanced sensory surround inhibition in S1, as no change in the parietal N20-P25 SEP peak ratio were observed in the current study. However, this possibility cannot be ruled out, as this may occur only when observable changes are seen in the individual median nerve SEP peak amplitudes.

**Implications for Investigations of Neural Plasticity and Spinal Manipulation**

Episodes of acute pain, such as after an injury, may initially induce plastic changes in the sensorimotor system (for a review of this topic please see reference \textsuperscript{61}). These changes could include dysfunctional motor control of spinal joint segments, that is, the manipulable lesion that chiropractic physicians and other manipulative therapists treat. Pain alone, without deafferentation, has been shown to induce increased SEP peak amplitudes\textsuperscript{62,63} and increased somatosensory evoked magnetic fields.\textsuperscript{54} Sensorimotor disturbances are known to persist beyond acute episode of pain,\textsuperscript{65,66} and these disturbances are thought to play a defining role in the clinical picture and chronicity of different chronic neck pain conditions.\textsuperscript{67} Therefore, the reduced frontal N30 SEP peak ratio observed in the current study after spinal manipulation may reflect an improvement of plastic changes induced by previous injury and may reflect one mechanism for the improvement of functional ability reported after spinal manipulation. This requires further investigation.

Abnormal central integration of a dual somatosensory input has previously been demonstrated at the cortical level after as little as 20 minutes of repetitive thumb abductions\textsuperscript{39} and throughout the somatosensory system in patients with dystonia.\textsuperscript{55} Tinazzi et al\textsuperscript{55} argued that the increased central dual SEP peak ratios represented reduced surround inhibition in the patients with dystonia and that their findings suggest that the inhibitory integration of mainly proprioceptive afferent inputs coming from adjacent body parts is abnormal in patients with dystonia.\textsuperscript{55} Furthermore, they argue that the inefficient integration of dual input was most likely due to altered surround inhibition and could in turn lead to abnormal motor output, contributing to the motor impairment present in dystonia.\textsuperscript{53} Motor impairments are also present in chronic neck pain patients. Impairment of deep cervical neck flexors and significant postural disturbances during walking and standing has been demonstrated in both insidious-onset and trauma-induced chronic neck pain conditions.\textsuperscript{65,73} Altered sensitivity of proprioceptors within the neck muscles has been suggested to be related to the postural disturbances seen in these patients.\textsuperscript{67,70} It is therefore possible that this leads to altered or inefficient integration of dual input in this patient group also, resulting in the above mentioned motor impairments. There is also evidence to suggest that muscle impairment occurs early in the history of onset of neck pain\textsuperscript{65} and that this muscle impairment does not automatically resolve even when neck pain symptoms improve.\textsuperscript{65,66} Some authors have therefore suggested that the deficits in proprioception and motor control, rather than neck pain itself, may be the main factors defining the clinical picture and chronicity of different chronic neck pain conditions.\textsuperscript{67} These deficits in proprioception and motor control may be partly due to spinal dysfunction causing either inhibition or facilitation of neural input to the muscles surrounding the spine. However, the central sensorimotor plastic changes that occur with spinal dysfunction may also lead to abnormalities in the way the CNS processes incoming information from more distal regions, such as the upper limb. The altered frontal P22-N30 SEP peak ratio after spinal manipulation may reflect an improvement of such maladaptive plastic changes in the current study population, who all had recoccurring neck problems, but were not in acute pain at the time they participated in this study.

**Limitations**

This study assessed 13 subjects under specified conditions. Therefore, findings may possibly be different in other populations and with different inclusion and exclusion criteria. Further studies should be performed using larger groups of subjects with different presentations.

**Conclusion**

The observations in the present study suggest that spinal manipulation of dysfunctional cervical joints may improve suppression of SEPs evoked by dual upper limb nerve stimulation at the level of the motor cortex, premotor areas, and/or subcortical areas such as basal ganglia and/or thalamus, lasting at least 20 minutes postmanipulation. Further studies are needed to elucidate the role and mechanisms of these cortical changes and their relationship to a patient’s clinical presentation and their ability to perform daily tasks.
Practical Applications

- Spinal manipulation of dysfunctional cervical joints can alter cortical integration of dual somatosensory input from the upper limb.
- This suggests that spinal manipulation alters sensorimotor integration at the cortical level of the CNS.
- These findings may help to elucidate the mechanisms responsible for the effective relief of pain and restoration of functional ability documented after spinal manipulation.

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