Alterations in Cortical and Cerebellar Motor Processing in Subclinical Neck Pain Patients Following Spinal Manipulation

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Abstract

Objective: The purpose of this study was investigate whether there are alterations in cerebellar output in a subclinical neck pain (SCNP) group and whether spinal manipulation before motor sequence learning might restore the baseline functional relationship between the cerebellum and motor cortex.

Methods: Ten volunteers were tested with SCNP using transcranial magnetic stimulation before and after a combined intervention of spinal manipulation and motor sequence learning. In a separate experiment, we tested 10 healthy controls using the same measures before and after motor sequence learning. Our transcranial magnetic stimulation measurements included short-interval intracortical inhibition, long-interval intracortical inhibition, and cerebellar inhibition (CBI).

Results: The SCNP group showed a significant improvement in task performance as indicated by a 19% decrease in mean reaction time ($P < .0001$), which occurred concurrently with a decrease in CBI following the combined spinal manipulation and motor sequence learning intervention ($F_{1,6} = 7.92$, $P < .05$). The control group also showed an improvement in task performance as indicated by a 25% increase in reaction time ($P < .001$) with no changes to CBI.

Conclusions: Subclinical neck pain patients have altered CBI when compared with healthy controls, and spinal manipulation before a motor sequence learning task changes the CBI pattern to one similar to healthy controls.

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Key Indexing Terms: *Manipulation, Spinal; Transcranial Magnetic Stimulation; Cerebellum; Learning*

Spinal manipulation is one of the most common treatments for neck and back pain; however, the neurophysiological mechanism responsible for improved function and reduction of pain is not yet fully understood. Neck pain is a common and significant problem that affects about 30% to 50% of people every year and places a great burden on health care systems.[1] One category of neck pain has been described as subclinical neck pain (SCNP)[2-4] and is minor neck pain for which participants have not yet sought treatment. Interestingly this group has shown changes in range of motion, cervical kinesthesia, and muscle endurance.[2-4] There is growing interest in SCNP because individuals that fall into this category provide an opportunity to explore neurophysiologic dysfunction without the interactive effect of current pain, which is known to alter measurements of sensorimotor integration and motor control.[5-7]

Numerous studies indicate that significant cortical plastic changes are present in various musculoskeletal pain syndromes.[8,9] In particular, altered feed-forward postural adjustments have been demonstrated in a variety of musculoskeletal conditions including anterior knee pain,[10] low back pain,[11] and idiopathic neck pain.[12] Furthermore, alterations in trunk muscle recruitment patterns have been observed in patients with mechanical low back pain.[13-15]

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It has been hypothesized that these changes in muscle recruitment patterns are an adaptation to underlying spinal instability resulting from osteoligamentous laxity or damage, muscle dysfunction, or reduced neuromuscular control.[16,17] There is also evidence in the literature to suggest that muscle impairment occurs early in the history of onset of spinal complaints,[18] and that such muscle impairment does not automatically resolve even when pain symptoms improve.[18,19] This has led some authors to suggest that the deficits in proprioception and motor control, rather than the pain itself, may be the main factors defining the clinical picture and chronicity of various chronic pain conditions.[20,21]

Furthermore, recent evidence has demonstrated that spinal manipulation can alter neuromuscular and proprioceptive function in patients with neck and back pain as well as in asymptomatic participants. For instance, cervical spine manipulation has been shown to produce greater changes in pressure pain threshold in lateral epicondylalgia than thoracic manipulation[22]; and in asymptomatic patients, lumbar spine manipulation was found to significantly influence corticospinal and spinal reflex excitability.[23] Interestingly, Soon et al did not find neurophysiological changes following mobilization on motor function and pressure pain threshold in asymptomatic individuals,[24] perhaps suggesting that manipulation, as distinct from mobilization, induces unique physiological changes. There is also accumulating evidence to suggest that chiropractic manipulation can result in changes to central nervous system function including reflex excitability, cognitive processing, sensory processing, and motor output.[25-29] There is also evidence in SCNP individuals that chiropractic manipulation alters cortical somatosensory processing[29,30] and elbow joint position sense.[25] This evidence suggests that chiropractic manipulation may have a positive neuromodulatory effect on the central nervous system, and this may play a role in the effect it has in the treatment of neck pain. It is hoped that improving our understanding of the neurophysiological mechanisms that may precede the development of chronic neck pain in individuals with SCNP will help provide a neurophysiological marker of altered sensory processing that could help determine if an individual is showing evidence of disordered sensorimotor integration and thus might benefit from early intervention to prevent the progression of SCNP into more long-term pain states.

One mechanism proposed by Haavik-Taylor and Murphy[28] suggests that the presence of spinal dysfunction would alter sensory feedback from the area of joint dysfunction and could therefore be responsible for improper sensorimotor integration due to central plastic changes. They further hypothesized that the use of appropriate spinal manipulation to the areas of spinal dysfunction would normalize the afferent input, thus resulting in appropriate sensorimotor integration. Previous work using paired-pulse transcranial magnetic stimulation (TMS) of the motor cortex has indicated that cervical spine manipulation can alter sensorimotor integration of the upper limb by decreasing the amount of short-interval intracortical inhibition (SICI).[28] A recent somatosensory evoked potential (SEP) study involving dual SEPs from the median and ulnar nerves demonstrated that cervical manipulation of dysfunctional areas in patients with a history of reoccurring neck pain or stiffness was able to affect sensorimotor integration.[30] There was a significant increase in the dual SEP ratio for cortical SEP components after the 20-minute motor training task. This did not occur when the motor training task was preceded with spinal manipulation; that is, spinal manipulation altered the way the central nervous system responded to the motor training task. When spinal manipulation preceded the motor training task, there was a significant decrease in the dual SEP ratio for one of the cortical SEP components, most likely due to changes in the ability to appropriately filter and integrate the dual input.

Motor learning refers to the acquisition or improvement of a motor skill with practice.[31] Motor learning leads to changes in the primary motor cortex (M1), as seen when changes are made to direction and speed during training.[32] The cerebellum is a neural structure that is actively involved in both motor learning[31,33-35] and sensorimotor integration of afferent input from the joints of the neck and spine.[36,37] It has also been suggested that the cerebellum is a plastic structure responsible for modulation of motor circuitry.[38,39] More recently, studies have shown that the cerebellum is also involved in the modulation of motor cortex excitability due to a reduction of cerebellar inhibition in patients with migraine with aura[40] and patients with focal hand dystonia.[41]

A current theoretical model suggests that cerebellum modulates activity and aids in learning through the formation of internal schema and network connections that dictate the needed movements for executing a task.[33] There is strong support for the encoding of limb position in the spinocerebellar tract[42] and in the external cuneate nucleus projecting to the thalamus and cerebellum,[43] and this encoding likely dictates the discharge produced from Purkinje cells during movement and in the formative sensitization and learning stage of an internal schema. The increased activity of cerebellar nuclei seen in early stages of sensitization and learning[34] may arise because of prenuclei encoding and Purkinje cell activity needed for error correction. Decreases to cerebellar nuclei activity in later stages of sensitization and learning[34] reflect greater reliance on internal schema with new postnuclei encodings that reflect the sensitized and learned state.[33] The ability of the cerebellum to form these internal models (ie, body map or body schema) may be overactive or impaired in the group with SCNP as a result of poorly encoded sensory signals and increased encoding of these signals to compensate for

the maladaptive state. We have hypothesized that altered afferent input from the neck due to joint dysfunction leads to disordered sensorimotor integration within the cerebellum and a subsequent derangement in motor commands to the upper limb.[44] The cerebellum plays a fundamental role in detecting the encoded afferent signal and relaying this information as part of the body schema. When the input signal is no longer encoded as a result of joint dysfunction and altered afferent input, the cerebellum must adjust to new encodings that dictate the body schema and affect proper execution of the motor task.

The contributions of both the cerebellum and the motor cortex need to be investigated to understand the effect of spinal manipulation and its impact on forming internal schema which will affect motor sequence sensitization, learning, and behavioral performance. Therefore, the purpose of this study was to investigate if there is modulation in cerebellar output to the motor cortex in SCNP patients compared with healthy controls and to investigate whether spinal manipulation and motor sequence learning have an effect on sensorimotor integration with respect to the cerebellum and subsequently the motor cortex.

Methods

This study was performed using the cortical TMS measures of SICI and long-interval intracortical inhibition (LICI) and the paired stimulator TMS technique known as *cerebellar inhibition* (CBI)[45] following spinal manipulation and a motor learning task.

Participants

Experiments were performed on 10 volunteers (mean age, 23.8; range, 20-35; 7 men and 3 women), each of whom experienced recurring neck pain or stiffness classified as *SCNP*, which was defined as intermittent neck pain such as mild neck pain, ache, and/or stiffness experienced over at least 3 months' duration for which they have not yet sought treatment, who were not experiencing an acute exacerbation of their pain on the testing day, as acute pain is known to alter neurophysiological measurements.[5-7] An extensive exclusion criteria checklist was applied by the registered chiropractor including absolute and relative contraindications to spinal manipulation. Specifically, participants were screened for cervical spine surgery; known fractures, anatomical abnormalities, or radicular arm pain; rheumatoid arthritis or other inflammatory conditions; history of cervical spine trauma in the last 3 months or trauma with persistent symptoms beyond 3 months; bleeding disorders or being on anticoagulant therapy; history of stroke; and history of cancer in the last 5 years. Contraindications to TMS included participants taking neuroactive medications, metal fragments or implants in the upper body or head, epilepsy, heart disease, severe headaches, skull fracture or serious head injury, and pregnancy. Each participant was assessed by a registered chiropractor as having spinal joint dysfunction. The neck disability index (NDI)[46] was administered to ensure that participants were not experiencing an acute exacerbation at the time of testing, as acute pain is known to alter some of the dependent measures of this study. All participants gave their informed written consent. A group of 10 healthy volunteers was also recruited. Participants did not qualify for the healthy control group if they had any previous history of neck pain or injury. The NDI[46] was administered to this group as well to confirm the absence of neck pain. All of the participants were right-handed as assessed by the Edinburgh Handedness Inventory, and none had any history of neurological disease. The study was approved by the University of Ontario Institute of Technology Research Ethics Board and conducted in accordance with regulations laid down in the Declaration of Helsinki.

Experimental Design. This experiment looked to examine the effects of spinal manipulation and a motor sequence learning task on the cerebellar and motor cortices. The cortical measures used were SICI and LICI, whereas the cerebellar measure used was CBI. These were measured both before and after a combined intervention of the chiropractic treatment and motor sequence learning task for the SCNP group. These measures were also recorded before and after the motor sequence learning task alone in the healthy control group.

Electromyographic Recordings. Electromyographic (EMG) activity was recorded from the right first dorsal interosseus (FDI) muscle using a pair of Ag-AgCl surface electrodes in a belly-tendon arrangement. The ground electrode was placed around the wrist of the right arm, in a location that was located between the stimulating coil and the surface electrodes. The EMG signal was amplified (1000×) and band-pass filtered (20-2000 Hz) with a Cambridge Electronic Design 1902 isolated amplifier (Cambridge Electronic Design, Cambridge, UK) digitizing at a sampling rate of 1 kHz (CED 1401 laboratory interface, Cambridge Electronic Design) and received by a laboratory computer for off-line analysis. Data were analyzed using SIGNAL software version 4.08 (Cambridge Electronic Design). Participants were asked to maintain a relaxed position throughout the experiment, while their hand was placed in a pronated position. The EMG activity was monitored during the protocol to ensure that the muscle was at rest.

Motor Sequence Task. Throughout the motor sequence learning task, participants were asked to sit in a chair with their arm supported by a soft pillow with a modified numeric keypad lying on top. With their hand palm down in a relaxed position, participants were asked to place their index finger on the keypad in a comfortable position so that they could reach the 7, 8, and 9 keys, while the other 3 fingers and thumb were taped down to maintain proper

hand orientation (Fig 1). A custom program was created using E-prime 2.0 software (Psychology Software Tools, Sharpsburg, PA) that prompted the participants to enter randomized sequences of the keys 7, 8, and 9 in 6 letter blocks being displayed on the screen. This side-to-side movement of the index finger allowed optimal activation of the FDI muscle by performing its primary action of abducting the index finger. Each participant's performance was measured by accuracy and reaction time to the task. Because of the long duration of the task (~20 minutes), the task was separated into 3 parts: a presection, the complex task, and a postsection. The task was the same for each section; however, the pre- and postsections only consisted of 15 trials, whereas the complex task itself consisted of 225 trials.

Spinal Manipulation Intervention. Clinical evidence of joint dysfunction has been said to include restricted intersegmental range of motion, palpable muscle tension at the intervertebral level, and tenderness to palpation of the joint.[47,48] Manipulations were targeted to segments in the cervical spine, clinically assessed by the registered chiropractor as showing evidence of joint dysfunction. It is recognized that there are issues with the reliability of palpation methods[49,50]; and we therefore used a pragmatic approach, instructing the clinician to determine the segments to treat as he or she would clinically. The clinician that treated the SCNP group of this study chose to use a combination of static and motion palpation and confirmed with questions to the participant whether spinal segments with a "hard-end feel" and decreased intersegmental movement on palpation were also tender before manipulating those segments. The SCNP group received high-velocity, low-amplitude spinal manipulation immediately following the preintervention measures. During the manipulation intervention, the EMG electrodes were left in place on the FDI muscle and the leads were unplugged; so the participant could move to a reclining chair. Upon returning to the experimental chair, the EMG leads were replugged, allowing identical placement upon the stationary surface electrodes. Particular care was taken to ensure the participants' positioning and posture remained the same as before the manipulation intervention. The high-velocity, low-amplitude manipulation consisted of thrusts to the spine held in lateral flexion, with slight rotation and slight extension. High-velocity, low-amplitude manipulation was chosen as the intervention for this study because previous research has shown that reflex EMG responses only occur after high-velocity manipulation, rather than low-velocity manipulation; thus, it would be more capable of modulating afferent input to the central nervous system.[51]

Transcranial Magnetic Stimulation. Cortical stimulation was performed using a figure-of-8 coil (outer diameter, 10 mm) and was applied over the hand region of the left motor cortex at the optimal position to elicit a motor evoked potential (MEP) in the right FDI. This location was then marked with a felt-tip pen onto a cap that the participant was asked to wear throughout the entire procedure. Magnetic stimulation was given via the use of 2 Magstim 200 stimulator units (Magstim Co, Whitland, Dyfed, UK) connected together with a BiStim unit at a frequency of 0.2 Hz with a 20% variance to account for anticipatory effects. The coil was held with the handle pointed backwards at approximately 45° away from the midsagittal line, with the current flowing posteriorly. This coil orientation has been previously shown to allow the induced current to be perpendicular to the central sulcus, which allows for the optimal activation of corticospinal neurons transsynaptically.[52,53]

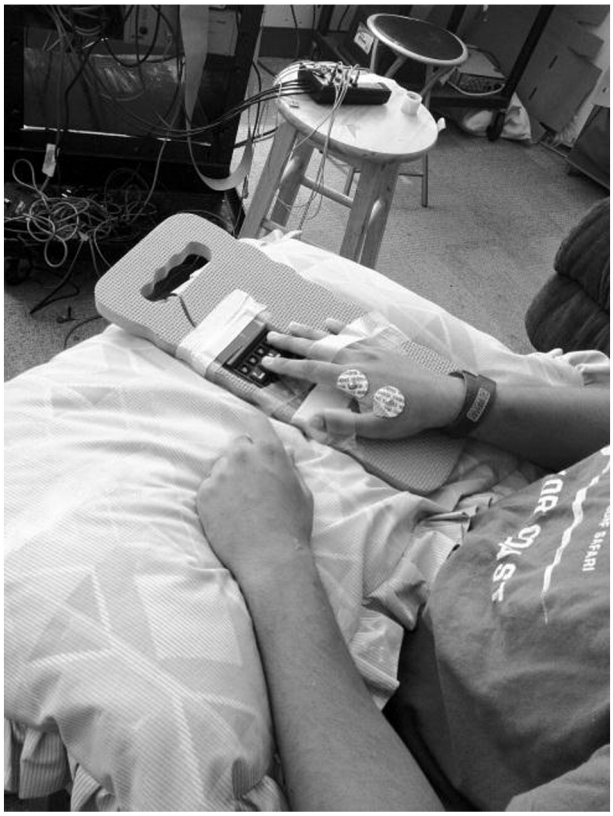

Fig 1. *A custom keyboard was developed to allow the index finger to reach the 7, 8, and 9 keys of the numeric keypad. Other digits were then taped down to allow the proper hand orientation to enable the index finger to move freely and optimally activate the FDI muscle through abduction. Recording electrodes placed over FDI are also shown.*

Paired Pulse TMS

The SICI and LICI were assessed using paired-pulse TMS paradigms. The SICI protocol consisted of a subthreshold conditioning stimulus (CS) set to 80% of the rest threshold followed by a suprathreshold test stimulus (TS) set to elicit an MEP of 1 mV with an interstimulus interval (ISI) of 2.5 milliseconds.[54] Rest threshold was

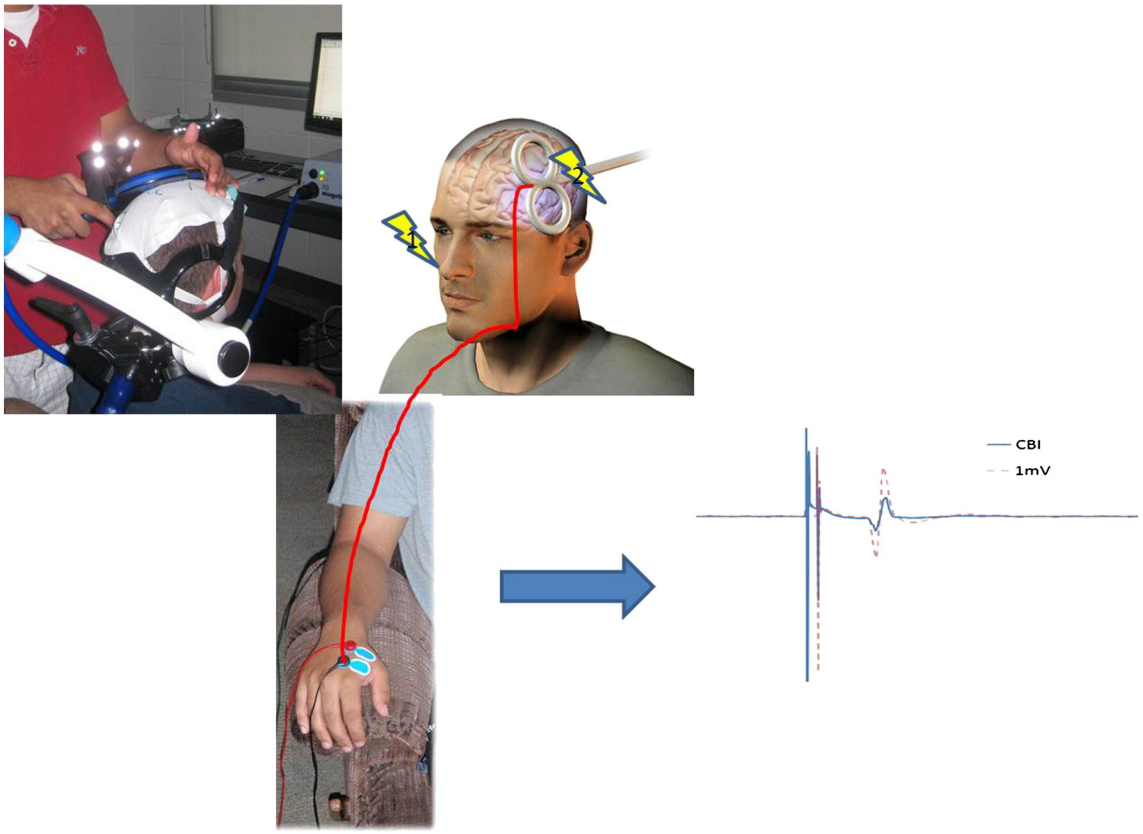

Fig 2. *A, Cerebellar coil positioned over the right hemisphere of the participant's cerebellum and strapped in place around the participant's head to maintain a close fit. The CS is applied over the cerebellum 5 milliseconds before the test MEP. B, The TS applied over the motor cortex and eliciting an MEP in the FDI muscle (electrodes shown). C, Red dotted line shows a 1-mV test MEP amplitude, and the solid blue line shows the effect of applying cerebellar conditioning 5 milliseconds before the TS on the MEP amplitude. (Color version of figure is available online.)*

determined by finding the lowest level of stimulator output that would elicit an MEP of approximately 50 μV in at least 5 of 10 trials while the participant's hand was at rest. The TS was monitored both before and after the intervention to ensure that the peak-to-peak amplitude was within 15% of each other. For 3 participants outside this 15% allowance, the stimulator intensity was raised until the TS was back within range. Each data block consisted of 16 stimuli. Thus, a total of 16 TSs and 16 combined CSs + TSs were delivered per trial. For data analysis, the conditioned MEP amplitude was expressed as a percentage of the suprathreshold 1-mV amplitude.

The LICI protocol consisted of a suprathreshold stimulus (S1) followed by another suprathreshold stimuli (S2) with an ISI of 100 milliseconds.[55,56] The 2 suprathreshold stimuli were set to the stimulator intensity that elicited the 1-mV MEP. Each LICI data block consisted of 16 stimuli. Thus, a total of 16 S1s and 16 combined S1s + S2s were delivered per trial. For data analysis, the LICI inhibition was measured as a ratio between the S2 MEP recorded when S1 and S2 were delivered within 100 milliseconds as compared with the MEP evoked from S1 alone.

Cerebellar TMS

The cerebellar conditioning stimulus (CCS) was delivered over the right cerebellar hemisphere with a double-cone coil (110-mm diameter). This coil has been previously shown to be effective to induce inhibition of the EMG response when applied 5 to 8 milliseconds in advance of stimulation over M1.[57] The coil was placed at the midpoint of a line joining the external auditory meatus to the inion, and the coil was oriented downwards to produce an upwards current within the cerebellar cortex.[40,45,57] The coil was placed in a stand and was strapped around the head of the participant to maintain a close fit and proper coil orientation (Fig 2). The intensity of the stimulator was pseudorandomized to stimulate at 70%, 80%, or 90% of the maximal stimulator output (MSO). These intensities were chosen based on pilot data that showed that an inhibitory modulation of the test MEP could be attained at these levels, without the contamination of brain stem or nerve root stimulation. The TS, which was applied over the left motor cortex, was set to a stimulus intensity that elicited an MEP of approximately 0.8 mV, as CBI has been demonstrated to be most effective when MEP amplitudes

are less than 1 mV.[45] Therefore, CBI was performed following the attainment of a 0.8-mV MEP (single Magstim setup) and was averaged over 10 stimuli. The ISI between the CCS and the TS of the motor cortex was set to 5 milliseconds, as it has been previously shown to induce CBI.[45,57] The inhibition was expressed as a percentage of the 0.8-mV TS. An additional 4 CCS stimuli on their own were given at each of the 3 intensities used to ensure that the CCS was not leading to brain stem or cervical nerve root activation.

Statistical Analysis

Once the data were acquired, the peak-to-peak amplitude for each sweep was measured off-line using a customized Signal configuration (Cambridge Electronic Design); and the average amplitude was calculated for each session using Microsoft (Redmond, WA) Excel. The SICI and CBI were measured as a ratio of test MEPs, and the LICI was measured as a ratio of the first to second MEPs. Paired *t* tests were run between pre- and postintervention to compare the mean peak-to-peak amplitudes for SICI and LICI. The CBI was analyzed using a repeated-measures analysis of variance with time (2 levels: pre- and postintervention) and between CS intensity (3 levels: 70%, 80%, and 90% MSO), with appropriate post hoc analyses as needed using IBM (Armonk, NY) SPSS Statistics (Version 19). The performance of the motor sequence learning task was analyzed based on the measures of reaction time and accuracy of the keystrokes using a paired *t* test between the pre- and postintervention trials, which was also performed in IBM SPSS Statistics.

Results

The NDI scores ranged between 5 and 12 with a mean of 7.5 ± 2.51. The SICI and LICI were performed on all SCNP participants both before and after the spinal manipulation and motor sequence learning task. However, data from only 7 participants in this group were able to be included for CBI measure analysis, as 3 of the participants had large artifacts from the high-intensity cerebellar stimulation that swamped the EMG signal, which meant the data could not be analyzed. Therefore, there were 10 data sets for SICI and LICI and 7 data sets for the CBI data analysis in the SCNP group, and 10 data sets for SICI and LICI and 8 data sets for the CBI data for the healthy control group.

In the SCNP group, the motor sequence learning task showed that, following the training intervention, the participants' reaction time improved significantly (from 451.63 to 364.14 milliseconds, $P < .0001$) (Fig 3A), whereas the participants' accuracy of the task remained unchanged ($P = .55$). In the healthy control group, the motor sequence learning task also demonstrated that, following the training intervention, the reaction time improved significantly

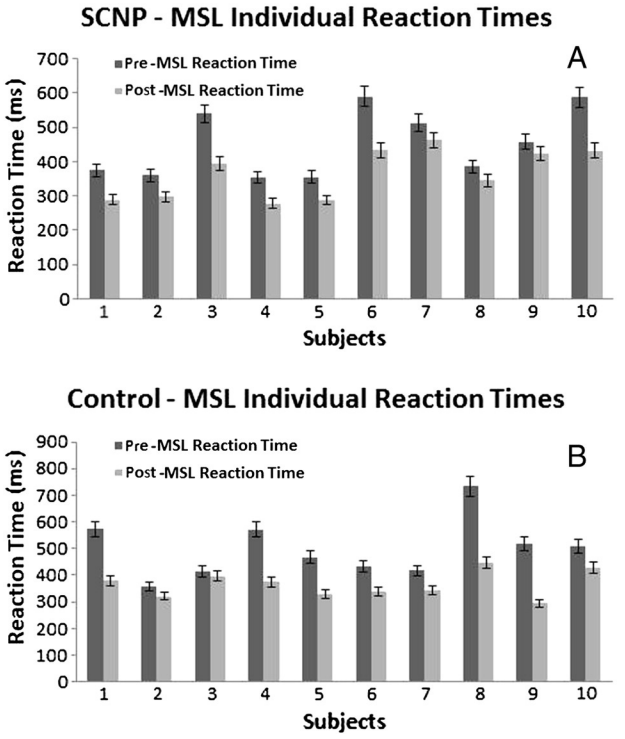

Fig 3. *Motor sequence learning reaction times for all participants in both the SCNP (subclinical neck pain) (A) and control (B) groups. The motor sequence learning task resulted in a significantly decreased reaction time to the intervention for all participants in both groups.*

(from 493.1 milliseconds to 367.29 milliseconds, $P = .001$) (Fig 3B).

For the SCNP group's CBI measure, a significant difference was seen when comparing pre- and postintervention with respect to the factor of time ($F_{1,6} = 7.92$, $P < .05$) and the factor of CS intensity ($F_{2,5} = 6.56$, $P < .05$). However, there was no reported interactive effect between the 2 factors. Post hoc paired *t* tests revealed that there was significant difference between pre- and postintervention at both 70% ($P < .05$) and 80% ($P < .05$) MSO; however, no significant difference was observed at 90% MSO (Fig 2). The healthy control group demonstrated a significant effect for the factor of stimulus intensity ($F_{2,6} = 31.64$, $P = .003$); however, the factor of time (ie, following motor training) was not significant (Fig 4). None of the additional 4 CCS stimuli on their own at each of the 3 intensities demonstrated any MEP.

In the SCNP group, both SICI and LICI remained unchanged when comparing from pre- to postintervention. The healthy control group demonstrated a significant effect when comparing pre- to postintervention results (Fig 5). The mean amplitude of the preintervention SICI measure was 0.237 ± 0.47 SE, compared with the postintervention SICI that was 0.346 ± 0.66 SE ($P < .03$). Long-interval intracortical inhibition showed no significant change from preintervention (mean ratio, 17.98 ±

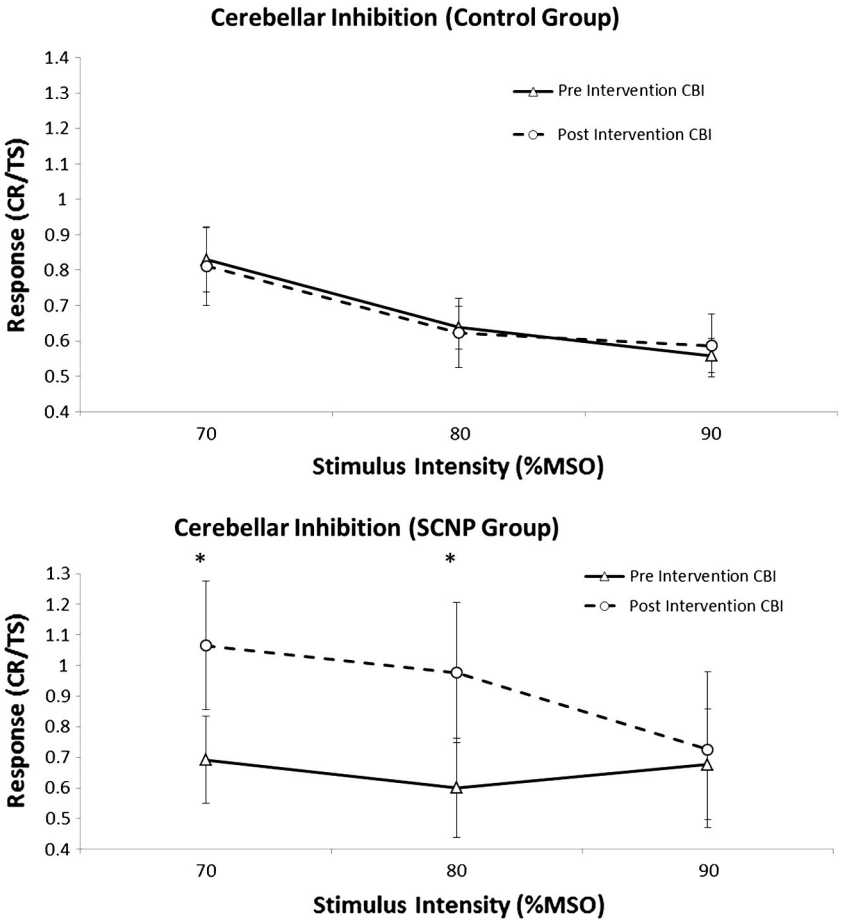

Fig 4. *Responses for CBI (error bars = SD) at all CS intensities compared pre- to postintervention for both the SCNP (subclinical neck pain) manipulation and the control group with the conditioned response being averaged to the TS (test stimulus). For both groups, there was a significant effect of cerebellar stimulation intensity. For the SCNP group, there was a significant effect of the intervention. Post hoc t tests indicated that the significant differences occurred at 70% and 80% of MSO (maximal stimulator output) following the intervention compared with baseline (indicated by the asterisk). CBI, cerebellar inhibition; CR, conditioned response.*

6.19 SE) compared with postintervention (mean ratio, 16.48 ± 5.84 SE; P = .831).

Discussion

Cortical TMS measures SICI and LICI were recorded to evaluate the level of intracortical inhibition, whereas CBI was used to measure the inhibitory effect of the cerebellum on the motor cortex. The CBI was clearly modulated by increasing cerebellar magnetic stimulation in the healthy control group preintervention, and this modulation was not changed by the motor sequence learning task. There was no modulation of CBI initially in the SCNP group (ie, increasing cerebellar magnetic stimulation had no effect on cortical stimulation), but CBI was significantly altered following the spinal manipulation plus motor sequence learning intervention. Following this spinal manipulation and motor sequence learning intervention, there was a significantly greater modulation with cerebellar stimulation at 70% and 80% of stimulator output (Fig 4). A significant decrease in SICI was found following the motor sequence learning in the healthy controls, with no change in the SCNP group. Significant improvement in reaction time occurred after the motor sequence learning segment of the intervention for both groups, indicating motor learning had occurred.

Motor sequence learning tasks have been previously shown to induce plasticity within the circuitry of both the motor cortex[58-60] and the cerebellum.[34] The decrease in mean reaction time as demonstrated in this study reflects implicit learning, which has been previously reported to induce altered representations of finger muscles in the motor cortex.[58] Neck manipulation has also been shown to provide a modulatory effect on the motor cortex by reducing the amount of intracortical inhibition.[28] However, there are no known studies that have demonstrated the effects of neck manipulation alongside motor sequence learning using TMS to measure cortical and cerebellar output.

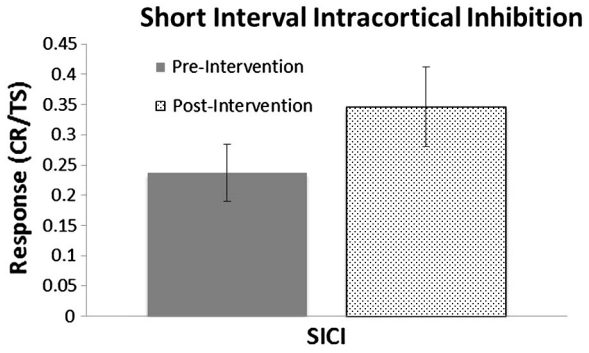

Fig 5. *Averaged SICI (±SE) results for pre– and post–motor sequence learning task for the healthy controls with the CR being averaged to the TS. The motor sequence learning intervention led to a 32% decrease in the effect of SICI. CR, conditioned response; TS, test stimulus; SICI, short-interval intracortical inhibition.*

It has been previously demonstrated that changes in the degree of cerebellar modulation of motor cortical output are present in certain patient groups including focal hand dystonia[41] and migraine with aura.[40] This study further adds to the literature by demonstrating an alteration in cerebellar modulation of motor output in SCNP patients when they received a manipulation-based chiropractic treatment before performing motor sequence learning. In the healthy control group, there was no change in CBI seen following motor sequence learning alone. It is possible that these differences in response between a healthy group and an SCNP group are due to altered sensorimotor integration as proposed by Haavik-Taylor and Murphy,[44] which was partly remedied following treatment. However, a limitation to these results is that, because of the time limit being placed on the protocol, the chiropractic treatment and motor sequence learning task had to be performed one after another. Therefore, interactive effects between manipulation and motor sequence learning cannot be fully separated with this design.

It is interesting to note that there was no significant effect on SICI following chiropractic treatment and the motor sequence learning. Referring back to the healthy control group, it was found that, after motor sequence learning alone, there was a significant decrease in the amount of intracortical inhibition as determined by SICI, whereas in another previous study by Haavik-Taylor and Murphy, there was also a decrease in SICI following spinal manipulation.[28] Therefore, the lack of a significant change in SICI can be seen as uncharacteristic compared with some previous literature.

This lack of similar results may have occurred for numerous reasons. Firstly, there may have been an interaction between the spinal manipulation and the motor sequence learning task that may have potentially cancelled out the effect observed from strictly the motor sequence learning task alone. These postintervention measurements were taken after the SCNP group had undergone manipulation and performed the motor sequence learning task to keep the experimental procedure at under 3 hours to prevent the participants from becoming tired and thus decreasing their excitability levels. It has been shown in past work that spinal manipulation altered sensorimotor integration as assessed by dual SEP ratios[30] and that this also affected the response to subsequent motor training. Secondly, the previous study by Haavik-Taylor and Murphy[28] measured changes in the abductor pollicis brevis muscle, rather than the FDI that was used in the current study. Therefore, the FDI may not be as susceptible to changes in excitability following spinal manipulation. Thirdly, an SCNP group was used in the current study; and their altered sensorimotor integration may have led to insignificant changes in cortical excitability pre- to postintervention. Finally, because we were collecting CBI and SICI data in the same experiment, there may have been an interactive effect between the 2 measures.

Daskalakis et al[45] demonstrated that there is an interaction between CBI and SICI. This study postulated that if TMS of the cerebellum activated inhibitory Purkinje cells, the output from the deep cerebellar nuclei to the motor cortex via the ventrolateral nucleus of the thalamus would be reduced. Furthermore, if the cerebellothalamocortical pathway terminated on inhibitory neurons within the motor cortex, it would be expected that the cerebellum would also have the potential to reduce local intracortical inhibition. If the motor sequence learning task had a significant effect on the cerebellum in this group of participants due to their neck pain and altered sensorimotor integration, then it is possible that a decreased level of CBI output to the motor cortex would result in an increase in SICI. However, with previous studies demonstrating that both chiropractic care and motor sequence learning tasks decrease SICI levels, the combined effects may have negated one another, resulting in the lack of change seen in this study.

The findings of this study, which suggest that normalizing afferent input from the neck may have restored a more correct internal body schema that allowed correct sensorimotor integration and normalized motor output, may have clinical applications. This is important for clinicians, as it suggests that improving neck dysfunction may improve upper limb task performance and execution.

Limitations

Because we did not include a control group with SCNP, we cannot concretely attribute the observed changes to spinal manipulation. A control condition, such as a passive head movement and/or sham manipulation group, should be included to act as a control for the nonspecific physiological effects that occur with a neck manipulation such as the application of pressure over a joint and head movements that occur during a neck manipulation. Future studies should investigate the effects of motor sequence learning and chiropractic manipulation on neck pain patients in separate experiments. Alternately, by performing CBI and

SICI protocols in separate experiments, the design could include an immediate postmanipulation measure before the motor sequence learning, which would allow us to more clearly attribute changes to either manipulation or motor sequence learning. This comparison should be performed in an age- and sex-matched SCNP group or on the same SCNP participants on a different day.

Conclusion

Cervical spine manipulation in an SCNP group leads to a pattern of cerebellar modulation more similar to a non–neck pain group. These findings suggest that normalizing afferent input from the neck may have restored a more correct internal body schema that allowed correct sensorimotor integration and normalized motor output.

Funding Sources and Conflicts of Interest

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Practical Applications

- The cerebellum is able to inhibit motor cortical output in a modulated fashion as demonstrated by the effect of graded cerebellar stimulation on corticomotor output.
- Subclinical neck pain appears to influence the capacity of the cerebellum to influence motor cortical excitability, as the cerebellar-cortical modulation was not present in the SCNP group of this study. However, this statement should be interpreted with caution because of low participant numbers.
- When a single session of chiropractic treatment is provided before a 20-minute motor training task in an SCNP group, a pattern of cerebellar modulation similar to that seen in healthy controls is observed.
- Futures studies should run separate experiments on cortical and cerebellar measures because of possible interactive effects, measure changes in SCNP groups with motor training in the absence of prior manipulation, and measure SCNP groups over longer periods.
- Neurophysiological measures should also be taken after manipulation and after motor training to clarify interactive effects between the two.
- The findings of this study suggest that improving neck dysfunction may improve upper limb task performance and execution.

Contributorship

Concept development (provided idea for the research): BM, JD, PY, HH

Design (planned the methods to generate the results): BM, JD

Supervision (provided oversight, responsible for organization and implementation, writing of the manuscript): BM, PY, HH

Data collection/processing (responsible for experiments, patient management, organization, or reporting data): JD, BM, JB

Analysis/interpretation (responsible for statistical analysis, evaluation, and presentation of the results): JD, BM, JB, PY, HH

Literature search (performed the literature search): JD

Writing (responsible for writing a substantive part of the manuscript): JD, BM

Critical review (revised manuscript for intellectual content, this does not relate to spelling and grammar checking): BM, PY, HH

Other (list other specific novel contributions)

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