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Review

The role of spinal manipulation in addressing disordered sensorimotor integration and altered motor control

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ABSTRACT

This review provides an overview of some of the growing body of research on the effects of spinal manipulation on sensory processing, motor output, functional performance and sensorimotor integration. It describes a body of work using somatosensory evoked potentials (SEPs), transcranial magnetic nerve stimulation, and electromyographic techniques to demonstrate neurophysiological changes following spinal manipulation. This work contributes to the understanding of how an initial episode(s) of back or neck pain may lead to ongoing changes in input from the spine which over time lead to altered sensorimotor integration of input from the spine and limbs.

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1. Introduction

Over the past 15 years our research group has conducted a variety of human experiments that have added to our understanding of the central neural plastic effects of manual spinal manipulation (Haavik and Murphy, 2011; Haavik-Taylor and Murphy, 2007a,b, 2008, 2010c; Haavik-Taylor et al., 2010; Marshall and Murphy, 2006). Spinal manipulation is used therapeutically by a number of health professionals, all of whom have different terminology for the "entity" that they manipulate. This "entity" which generally describes areas of muscle tightness, tenderness and restricted movement may be called a "vertebral (spinal) lesion" by physical medicine specialists or physiotherapists, "somatic dysfunction" or "spinal lesion" by osteopaths, and "vertebral subluxation" or "spinal fixation" by chiropractors (Leach, 1986). For the purposes of this article, the "manipulable lesion" will be referred to as an area of spinal dysfunction or joint dysfunction. Joint dysfunction as discussed in the literature ranges from experimentally induced joint effusion (Shakespeare et al., 1985), pathological joint disease such as osteoarthritis (O'Connor et al., 1993) as well as the more subtle functional alterations that are commonly treated by manipulative therapists (Suter et al., 1999, 2000).

Based on our research findings we have proposed that areas of spinal dysfunction, represent a state of altered afferent input which may be responsible for ongoing central plastic changes (Haavik-Taylor et al., 2010; Haavik-Taylor and Murphy, 2007c). Furthermore we have proposed a potential mechanism which could explain how high-velocity, low-amplitude spinal manipulation, also known as spinal adjustments, improve function and reduce symptoms. We have proposed that altered afferent feedback from an area of spinal dysfunction alters the afferent "milieu" into which subsequent afferent feedback from the spine and limbs is received and processed, thus leading to altered sensorimotor integration (SMI) of the afferent input, which is then normalized by high-velocity, low-amplitude manipulation (Haavik-Taylor et al., 2010; Haavik-Taylor and Murphy, 2007c). For a pictorial depiction of this hypothesis, see Fig. 1.

This article will provide an overview of some of the growing body of research on the effects of spinal manipulation on sensory processing, motor output, functional performance and sensorimotor integration. This body of work contributes to our understanding of how an initial episode(s) of back or neck pain may lead to ongoing changes in input from the spine which over time lead to altered sensorimotor integration of input from the spine and limbs. Increasing this understanding may provide a neurophysiological explanation for some of the beneficial clinical effects reported by chiropractors and other manipulative therapists in day to day practice. Chronic musculoskeletal pain affects the lives of millions of individuals and places a great burden on health care systems in the western world. Some of the research discussed in this review has the potential to identify objective neurophysiological markers which may be able to predict which patients will respond best to spinal manipulative treatment and/or whether a patient has had a sufficient amount of treatment to normalize neurophysiological markers of disordered sensorimotor integration. The relationship

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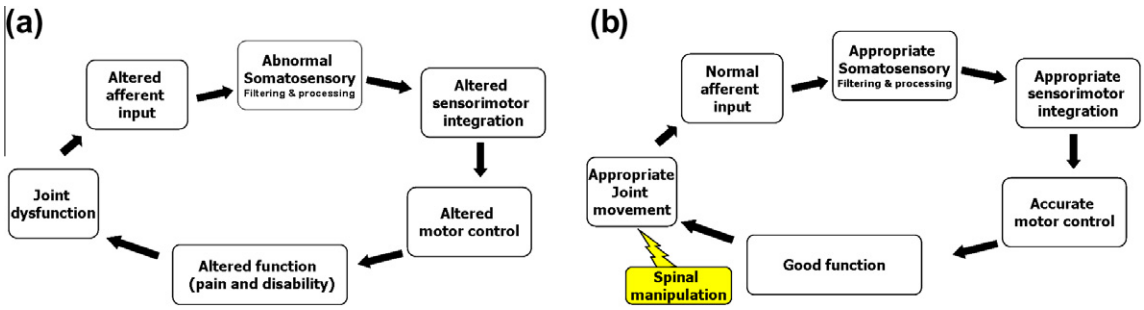

Fig. 1. (a) Diagram depicting a simplified view of the proposed effects of spinal dysfunction, leading to altered sensorimotor integration which over time in some susceptible individuals may lead to pain and gross dysfunction. (b) Schematic view of proposed effects of spinal manipulation leading to normalization of afferent input and restoration of appropriate sensorimotor integration and function.

between the development and maintenance of chronic musculoskeletal pain and neural markers of altered function is a promising area in need of further research.

1.1. Altered sensorimotor processing following spinal manipulation

Several studies utilizing somatosensory evoked potentials (SEPs) (Haavik-Taylor and Murphy, 2007c, 2010c; Haavik-Taylor et al., 2010) have shown that manipulation of areas of joint dysfunction in the cervical spine can alter somatosensory processing and early sensorimotor integration of input from the upper limb. These studies have shown alterations in the amplitude of the cortical SEP peaks N20 (Haavik-Taylor and Murphy, 2007c) and N30 (Haavik-Taylor and Murphy, 2010c) following high-velocity, low-amplitude cervical manipulation. The N20 SEP peak represents the arrival of the afferent volley at the primary somatosensory cortex (Desmedt and Cheron, 1980; Mauguiere, 1999; Nuwer et al., 1994). Later SEP peaks are thought to be generated by the processing of this somatosensory input (Cheron and Borenstein, 1991, 1992; Desmedt and Cheron, 1981; Desmedt et al., 1983; Kanovský et al., 2003; Mauguiere et al., 1983; Rossini et al., 1989, 1987; Waberski et al., 1999) and are therefore thought to reflect early sensorimotor integration (Rossi et al., 2003). One of these peaks, the N30 SEP component is thought to have multiple generators. Some authors suggest this peak is generated in the post-central cortical regions (i.e. S1) (Allison et al., 1989a,b, 1991), while evidence also suggests that this peak is related to a complex cortical and subcortical loop linking the basal ganglia, thalamus, pre-motor areas, and primary motor cortex (Cheron and Borenstein, 1991, 1992; Desmedt and Cheron, 1981; Desmedt et al., 1983; Kanovský et al., 2003; Mauguiere et al., 1983; Rossini et al., 1989, 1987; Waberski et al., 1999). Hence the N30 peak in particular is thought to reflect early sensorimotor integration (Rossi et al., 2003). More recently, Cebolla et al. have used swLORETA (standardized weighted Low Resolution Brain Electromagnetic Tomography) taking into account both phasic and oscillatory generators to determine the neural generators of the N30 (Cebolla et al., 2011). They have determined that the N30 is generated by network activity in the motor, premotor and prefrontal cortex, adding further weight to its role as a marker of neural processing relevant to sensorimotor integration.

The results of these studies (Haavik-Taylor and Murphy, 2007c, 2010c; Haavik-Taylor et al., 2010) suggest that manipulation of dysfunctional cervical segments can alter early sensory processing and SMI of information from the upper limb. SMI is the process by which the nervous system coordinates incoming sensory (afferent) information from different parts of the body and integrates with the motor system to control movement.

Over the past three decades it has become well established that the human central nervous system (CNS) retains its ability to adapt to its ever-changing environment, and that both increased (hyper-afferentation) and decreased (deafferentation) afferent input leads to changes in CNS functioning (Bertolasi et al., 1998; Brasil-Neto et al., 1993; Tinazzi et al., 1997). What has also become apparent is that these plastic changes may occur in a manner that is subjectively positive for the individual, such as with motor learning to enable complex finger movement (e.g. playing the piano). This is known as adaptive neuroplasticity. However, studies are also showing that these plastic changes may occur in a manner that has decidedly negative subjective outcomes for the individual, known as maladaptive neural plastic changes. There is a growing body of literature that demonstrates maladaptive plastic changes are present in a variety of pain conditions/syndromes and musculoskeletal dysfunction (Falla, 2004; van Vliet and Heneghan, 2006), and that such adaptive changes can occur remarkably fast following an injury (Wall et al., 2002). This has lead various authors to hypothesize that such maladaptive neuroplastic changes present in long-term pain conditions rather than the actual pain itself is responsible for the individual sufferer's symptoms and functional disturbances (Brumagne et al., 2000; Michaelson et al., 2003; Paulus and Brumagne, 2008). In particular, changes in the way the CNS processes proprioceptive information have been suggested as the most important factor responsible for the clinical presentation of neck pain sufferers (Paulus and Brumagne, 2008).

Numerous activities of daily living are dependent on appropriate SMI. Interactions between sensory and motor systems allow us to engage with our environment. It allows us to reach for and grasp objects, detect and turn towards an auditory stimuli or respond to perturbations from the environment in order to maintain postural stability, balance and locomotion (Chen et al., 2009). SMI involves strong feedback connections between different CNS structures that are associated with numerous, and perhaps all, neuroanatomical subsystems. These subsystems interconnect to form a dynamic, multimodal, sensorimotor integrative system. This system receives all afferent information from the environment. The CNS utilizes all of these peripheral signals continuously to build and maintain an internal reference frame (Lackner and DiZio, 2005; Sainsburg and Kalakanis, 1999). This information is continuously processed in relation to information it receives regarding the voluntary intent of further movements, previous known estimations about such movements and internal sensory feedback from the actual movements when they take place. Continuous comparisons and error adjustments take place, and over time so does motor learning of frequent movements or actions. A breakdown anywhere in these multimodal sensorimotor feedback loops has the potential to greatly affect other interconnected neuroanatomical subsystems, in either an adaptive or maladaptive manner.

Motor commands or motor intention (also known as "efference copies") are for example known to interact with afferent signals to generate sensation, and are known to contribute to joint position sense (Smith et al., 2009). Under normal circumstances there is an integration of intention, action and sensory feedback. Furthermore,

in a healthy state there is congruence between motor intention and sensory experience (both proprioceptive and visual) when we for example move a limb through space. Thus goal-directed action requires ongoing monitoring of sensorimotor inputs to ensure that motor outputs are congruent with current intentions as well as the proprioceptive feedback from the actual movement.

This monitoring is automatic but can become conscious if there is a mismatch between expected and realized sensorimotor states. A recent study has demonstrated that providing a sensorimotor conflict, i.e. providing unexpected visual feedback when moving a limb (via hiding a moving limb and/or distorting visual feedback of the movement of that limb) is sufficient to produce additional somaesthetic disturbances, and exacerbation of pre-existing symptoms in a group of fibromyalgia patients (McCabe et al., 2007). This suggests that a conflict between our expected and realized sensorimotor states can in some individuals produce or worsen pain sensations. It is therefore possible that a mechanism by which spinal manipulation relieves pain in patients is due to a central effect by improving somatosensory integration processes. This theory is however based on several assumptions that need to be verified in future studies, such as whether a mis-match in expected and actual sensory information can cause pain in healthy populations, whether spinal dysfunction causes somaesthetic disturbances and/or incongruence between motor intention and sensory experience (both proprioceptive and/or visual), and whether this is improved with spinal manipulation.

Discrepancies between expected and actual sensory information can cause pain in healthy populations and this has been explored in the laboratory setting (McCabe et al., 2005). Healthy pain-free volunteers were asked to perform a series of bilateral upper and lower limb movements whilst viewing these movements in a mirror that created varied degrees of sensory–motor conflict during the movements. They found that 66% of their healthy pain-free volunteers reported at least one anomalous sensory symptom at some stage in the protocol despite no peripheral nociceptive input. Several of these volunteers reported parasthesia sensations and mild aches or pain (McCabe et al., 2005), lending support to the hypothesis that motor–sensory conflict can induce pain and sensory disturbances in some normal individuals.

1.2. The importance of proprioception

Accurate proprioception is therefore an important component of sensorimotor integration in the CNS. Proprioception includes both joint position sense (JPS) and kinaesthesia (the sense of limb movement in the absence of visual cues) (for review see Gilman, 2002). The main source of afferent information for JPS arises from muscle spindles, however mechanoreceptors in joint capsules and cutaneous tactile receptors may also contribute (for review see Gilman, 2002). Joint position sense has been extensively studied in the ankle, knee and hip joints (Adachi et al., 2007; Bennell et al., 2005; Beynnon et al., 2002; Hazneci et al., 2005; Hopper et al., 2003; Ishii et al., 1997; Karanjia and Ferguson, 1983; Larsen et al., 2005; Marks, 1996; Okuda et al., 2006; Reider et al., 2003; Ribeiro et al., 2007; Tsauo and Cheng, 2008), particularly to investigate the effects of reconstructive surgery (Adachi et al., 2007; Hopper et al., 2003; Reider et al., 2003), osteoarthritis (Barrett et al., 1991; Bennell et al., 2003; Marks, 1996), joint bracing (for review see Beynnon et al., 2002), and various exercise or re-training programs (Friemert et al., 2006; Hazneci et al., 2005; Ribeiro et al., 2007; Tsauo and Cheng, 2008). Recently there has also been an increased focus in the literature on spinal JPS (Allison, 2003; Jull et al., 2007; Learman et al., 2009; Strimpakos et al., 2006; Swinkels and Dolan, 1998), however, much less research has looked at the effect of the spine on limb JPS (Knox et al., 2006a,b; Knox and Hodges, 2005).

Improved head repositioning accuracy has been demonstrated by Palmgren et al. (2006) following chiropractic care, suggesting that spinal manipulation can improve spinal proprioception. The effects of improving spinal function on upper limb proprioception has also recently been investigated in a group of 25 participants with subclinical neck pain (SCNP) (i.e. reoccurring neck dysfunction such as neck pain, ache and/or stiffness with or without a history of known neck trauma) and 18 control participants (Haavik and Murphy, 2011). This study demonstrated that the SCNP group had reduced elbow joint position sense compared with those who had no history of any neck complaints. Furthermore, the study showed that cervical spine manipulation of dysfunctional segments improved the accuracy of the SCNP groups' elbow joint position sense (Haavik and Murphy, 2011). This suggests that cervical dysfunction can impair the way that proprioceptive information from the upper limb is processed. It also suggests that improving spinal function with manipulative treatment leads to more appropriate and accurate processing and integration of such proprioceptive input. However, it is worth noting that this study does not provide conclusive evidence that improving spinal 'dysfunction' was the precise cause of these observed effects.

In all of the cited studies by our group, spinal dysfunction was 'quantified' to some degree prior to and after each spinal manipulation intervention by assessing for tenderness to palpation of the relevant joints, manually palpating for restricted intersegmental range of motion, assessing for palpable asymmetric intervertebral muscle tension, and any abnormal or blocked joint play and end-feel of a joint. All of these biomechanical characteristics are known clinical indicators of spinal dysfunction (Fryer et al., 2004; Hestboek and Leboeuf-Yde, 2000). These findings were documented pre and post each spinal manipulation intervention. Improvements in segmental function following spinal manipulation were also recorded for each subject.

An important area for future research is to document more precise biomechanical data pre and post spinal manipulation to explore whether particular biomechanical characteristics of spinal dysfunction are associated with poor proprioception. Similarly it is necessary to explore whether particular biomechanical characteristics of improved spinal function correlate with improved proprioceptive processing. Furthermore, more precise reporting of biomechanical characteristics of the manipulation may reveal correlations between certain features of the manipulation and improved proprioceptive processing. This should be explored in future research.

In light of the above findings it is possible that the changes we previously observed in the cortical N20 and N30 SEP peaks following cervical spine manipulation (Haavik-Taylor and Murphy, 2007c, 2010c; Haavik-Taylor et al., 2010) reflect changes in the way the research participant's CNS was perceiving and processing proprioceptive information from their stimulated upper limb. The low intensity stimuli applied during SEP recordings which are just above motor threshold, stimulate mainly large myelinated sensory afferents such as 1a muscle afferents (Gandevia and Burke, 1988; Gandevia et al., 1984).

There are numerous other studies that also implicate cervical spine impairment in maladaptive sensorimotor integration, for example affecting postural control and/or reduced JPS. This has been observed with chronic neck pain (Falla, 2004; Michaelson et al., 2003), neck muscle fatigue (Stapley et al., 2006), cervicobrachial pain syndrome (Karlberg et al., 1995), cervical root compression (Takayama et al., 2005a,b), and following whiplash injury (Stapley et al., 2006; Sterling et al., 2003). Therefore, there appears to be a considerable link between cervical function and accurate proprioceptive processing. Although most of these previous studies related to significant cervical injury or severe cervical symptoms, one study has demonstrated that changes in head and

neck position in a group of participants without any history of neck pain or injury led to reduced accuracy of elbow joint position sense (Knox and Hodges, 2005). The authors of this study discussed how accurate execution of movement depends on the ability of the CNS to integrate somatosensory, vestibular, and visual information regarding the position of the body (Knox and Hodges, 2005). They argued that placing their subjects' heads in full flexion and rotation could have led to an overload of the computational capacity of the CNS, thus resulting in increased JPS error (Knox and Hodges, 2005).

1.3. Altered somatosensory integration following spinal manipulation

A key component of typical SMI is early sensory integration of afferent input. The interaction and integration of afferent inputs from adjacent nerves at spinal, brainstem and cortical levels of the somatosensory system can be evaluated with a particular SEP protocol, i.e. by comparing spinal, brainstem and cortical SEP amplitudes obtained after stimulating two peripheral nerves simultaneously with the arithmetic sum of the SEP amplitudes obtained after stimulating the same nerves individually. In healthy subjects the dual input produces smaller SEP amplitudes compared with SEP amplitudes that were produced from the same nerves stimulated individually and arithmetically added together (Tinazzi et al., 2000). This type of sensory filtering is the ability of an individual's CNS to suppress or attenuate the processing of multiple afferent peripheral, mainly proprioceptive, inputs. It is thought to reflect a type of "surround-like" inhibition, which in healthy individuals, allows for the contrast between stimuli to remain high by suppressing the processing of input from surrounding areas. In the somatosensory system, such inhibition allows for the body to perceive stimuli as separate and process them accordingly (Tinazzi et al., 2000a,b).

This filtering process has been found to be altered in individuals with neck pain, after repetitive muscular activities such as typing as well as other musculoskeletal disorders such as dystonia (Haavik-Taylor and Murphy, 2007a, 2010a,b, 2007c; Tinazzi et al., 2000). Two studies, utilizing this particular dual peripheral nerve stimulation SEP protocol both demonstrated that manipulating dysfunctional cervical segments can increase the surround-like inhibition of proprioceptive afferent input (Haavik-Taylor et al., 2010; Haavik-Taylor and Murphy, 2010c). Fig. 2 is reprinted from Haavik-Taylor et al. (2010). It depicts a significant decrease in the N30 SEP peak when the median and ulnar nerve are stimulated simultaneously, as compared with the arithmetic sum of the SEP amplitudes obtained following stimulation of the two nerves individually.

Earlier work by Tinazzi et al. (2000) found that dystonia patients have a reduced ability to suppress the dual peripheral nerve input. They argued that this reduced ability to suppress the dual peripheral nerve input was evidence of inefficient integration, and that this could give rise to abnormal motor output, which might therefore contribute to the motor impairments present in dystonia. The decreased SEP ratios following cervical spine manipulation of dysfunctional spinal segments (Haavik-Taylor et al., 2010; Haavik-Taylor and Murphy, 2010c), indicative of an enhanced ability to filter sensory information, may therefore reflect an improvement in early sensory integration of afferent input. However, this assumes that a reduced ability to suppress dual peripheral nerve input is always a negative finding, and that increasing it reflects a positive or adaptive neuroplastic process, neither of which is firmly established.

The ability of the CNS to gate sensory information is thought to be important to maintain the internal representation of its current posture or activity and to avoid undesirable reactions to external or internal perturbations (Ivanenko et al., 2000; Paulus and Brumagne, 2008). As Tinazzi et al. demonstrated that gating of sensory information is distorted in patients with focal hand dystonia (Tinazzi et al., 2000). Other groups have also demonstrated that there is a shift in the gain of the sensory signals, i.e. a central re-weighting of proprioceptive input, in patients with spasmodic torticollis (Anastasopoulos et al., 2003) and low back pain patients (Brumagne et al., 2004).

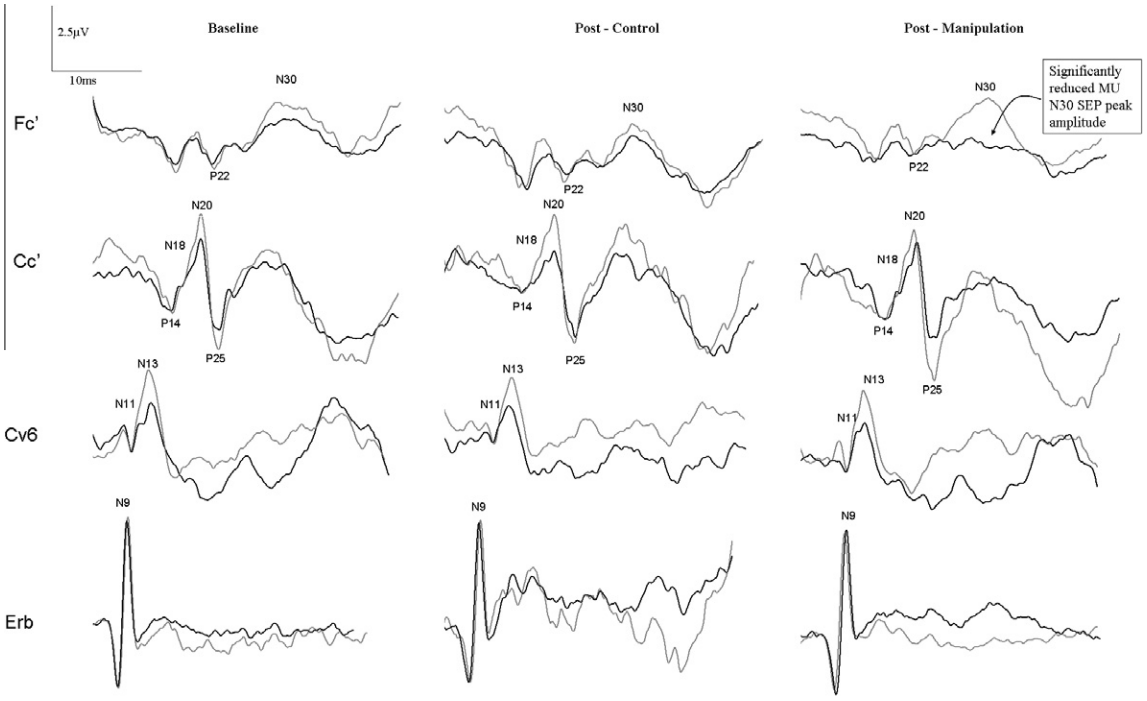

Fig. 2. Figure depicting a representative set of pre-intervention baseline somatosensory evoked potential (SEP) peaks (first set of traces), post-control SEP peaks (middle set of traces), and post-manipulation SEP peaks (third set of traces) recorded in a single subject. It shows Erb's point (Erb), cervical (Cv6), postcentral (Cc′), and precentral (Fc′) SEP peaks constructed from both the simultaneous stimulation of the median and ulnar nerves (MU) (black traces) and the arithmetic sum of the traces following stimulation of each nerve individually (M + U) (gray traces) (averages of 800 sweeps each). Reprinted with permission from Haavik-Taylor et al. (2010).

1.4. Altered motor control following spinal manipulation

Several studies utilizing transcranial magnetic stimulation (TMS) (Haavik-Taylor and Murphy, 2007c, 2008) have also shown that manipulating dysfunctional segments in the cervical spine can alter sensorimotor integration of input from the upper limb. The TMS experimental measures utilized in these studies, such as short-interval-intracortical-inhibition (SICI), short-interval-intracortical-facilitation (SICF) and the cortical silent period (CSP), are measures of sensorimotor integration that are believed to reflect processing at the level of the cortex (Cantello et al., 1992; Chen et al., 1999, 1998; Di Lazzaro et al., 1998, 1999; Fisher et al., 2002; Hanajima et al., 2002; Inghilleri et al., 1993; Kujirai et al., 1993; Kukowski and Haug, 1992; Tokimura et al., 1996; Ziemann et al., 1998). These motor control changes appear to occur in a muscle specific manner (Haavik-Taylor and Murphy, 2008). Figs. 3 and 4 are reprinted from Haavik-Taylor and Murphy (2008). Fig. 3 depicts a decrease in SICI for the abductor pollicis brevis (APB) muscle following spinal manipulation. No change was noted after the control intervention. Fig. 4 depicts an increase in SICF for the APB muscle and a decrease in SICF for the extensor indices proprios (EIP) muscle after the spinal manipulation session. Again no change was observed after the control intervention. The functional implications of these changes to upper limb function need further exploration.

1.5. Improved neuromuscular performance with spinal manipulation

When performing bodily movements, like throwing a ball for example, the central nervous system will activate a variety of postural trunk muscles prior to any movement of the arm in order to main postural stability during the throwing action. This process is known as feed-forward activation (FFA). Individuals with chronic low back pain are known to have delays in feedforward activation (Hodges and Richardson, 1999; Hodges et al., 1996), which is thought to influence postural stability. Early work by Murphy et al. (1995) had demonstrated the ability of sacroiliac joint (SIJ) manipulation to influence reflex excitability and her group sought to determine if SIJ manipulation might also influence feedforward activation. A study involving 90 healthy young males evaluated the participants for delays in FFA in the transversus abdominis muscle and internal obliques when undertaking rapid movements of the upper limb (Marshall and Murphy, 2006). Seventeen subjects had a delay in FFA which was reproducible when retested 6 months later. These subjects were examined by a chiropractor and were all found to have dysfunction of the sacroiliac joint on the side of delayed FFA. Following a single chiropractic manipulation of the dysfunctional sacroiliac joint, the feed forward activation latency was reduced by an average of 38% (Marshall and Murphy, 2006). This study demonstrated an improvement in central nervous system activation times of muscles associated with the stability of a specific joint due to spinal manipulation. What is not known is whether the improvement in FFA persisted beyond the time of treatment. However, subsequent work by Marshall and Murphy (2008) demonstrated that chronic low back pain participants treated with manipulation and/or exercise, there was a continued improvement in delayed FFA times at 1 year follow-up. Evidence for the association between delayed muscle activation times and impaired motor control is provided in a study by Radebold et al. (2001). They measured the "on" and "off" times of 12 agonist and antagonist trunk muscles during sudden trunk release movements in different directions, as well as postural sway in individuals with chronic back pain and controls. They found that chronic low back pain patients have delayed muscle "on" and "off" times and that these delays correlated significantly with impaired balance performance with eyes closed. FFA times have also been shown to correlate strongly with a patient's self-rated disability (Marshall and Murphy, 2010) indicating that these neuromuscular measures may be useful markers of both treatment effects and potential for chronicity in neck and back pain patients.

Motor impairments are present in chronic neck pain patients. Impairment of deep cervical neck flexor activation and significant postural disturbances during walking and standing have been demonstrated in both insidious-onset and trauma-induced chronic neck pain conditions (Alund et al., 1993; Branstrom et al., 2001; Jull et al., 2004; Karlberg et al., 1995; Michaelson et al., 2003; Persson et al., 1996; Rubin et al., 1995). Altered sensitivity of proprioceptors

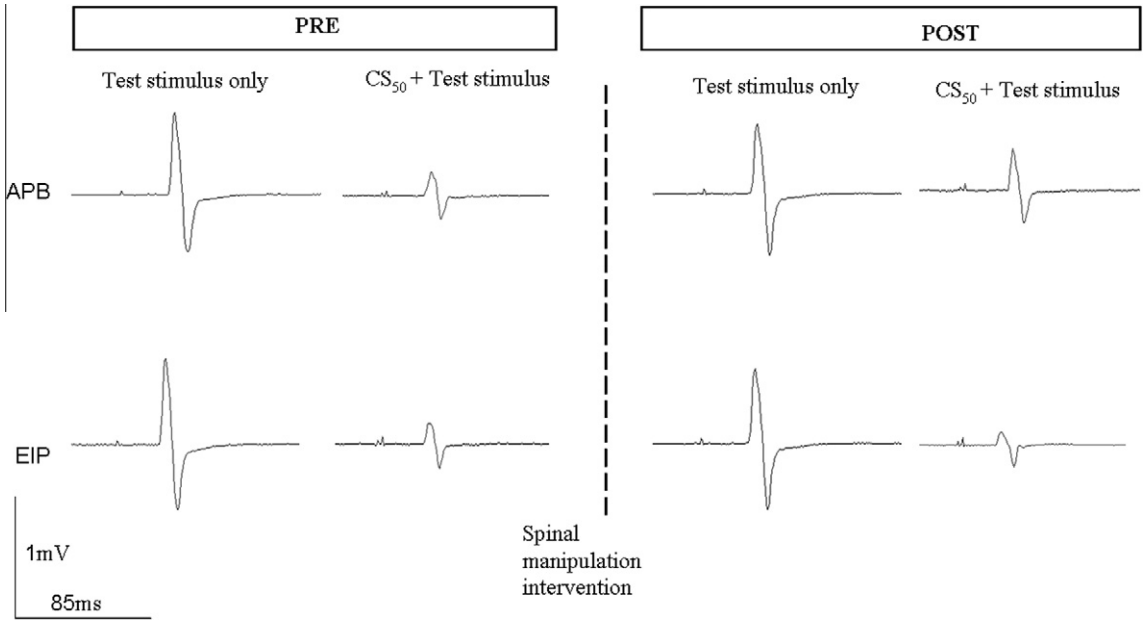

Fig. 3. Raw nonrectified electromyography traces from one representative subject showing the pre- and post-manipulation test stimulus (TS) and conditioned stimulus plus test stimulus (CS50 + TS) motor evoked potentials (MEPs) for both the abductor pollicis brevis (APB) and extensor indices proprios (EIP) muscles. Note the decrease in short interval intracortical inhibition (SICI) (i.e. less inhibition of MEP in the CS50 + TS compared with pre-manipulation MEP) for the APB muscle. Reprinted with permission, from Haavik-Taylor and Murphy (2008).

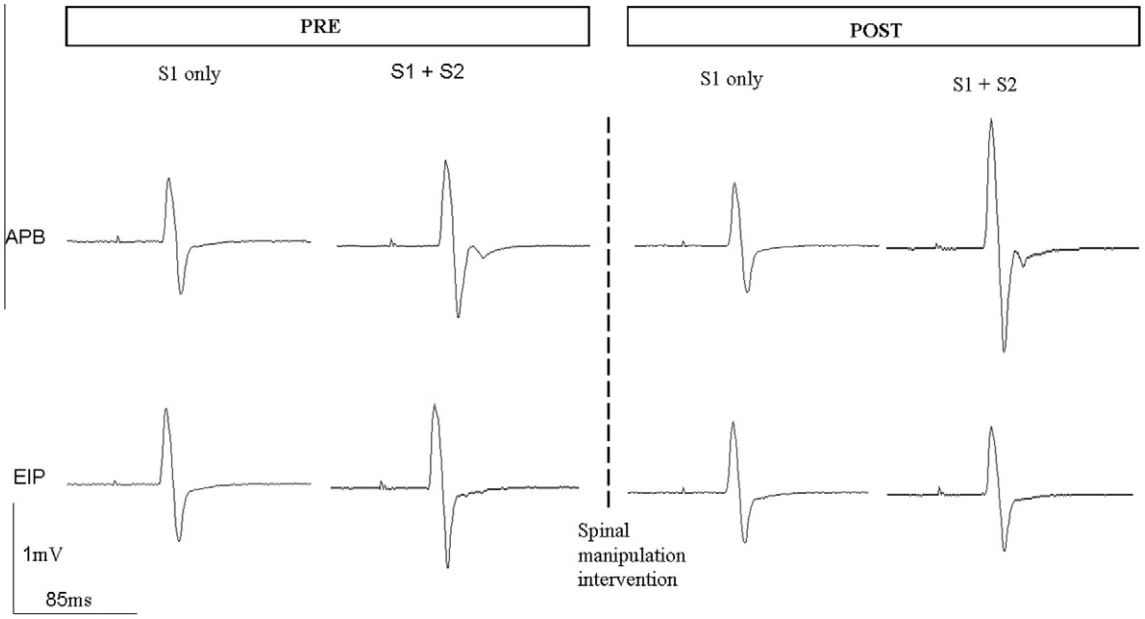

Fig. 4. Raw nonrectified electromyography traces from one representative subject showing the pre- and post-manipulation stimulus 1 (S1) and stimulus 1 plus stimulus 2 (S1 + S2) motor evoked potentials (MEPs) for both the abductor pollicis brevis (APB) and extensor indices proprios (EIP) muscles. Note the increase in short interval intracortical facilitation (SICF) for the APB muscle and the decrease in SICF for the EIP muscle after the spinal manipulation session. Reprinted with permission from Haavik-Taylor and Murphy (2008).

within the neck muscles has been suggested to be related to the postural (i.e. motor control) disturbances seen in these patients (Michaelson et al., 2003; Persson et al., 1996). It has also been argued that the degree to which proprioceptive input to the central nervous system is disturbed, and even more importantly how the CNS processes, interprets and transforms this afferent information into motor commands, that determines the degree to which subjects can successfully execute more challenging balance tasks (Michaelson et al., 2003; Paulus and Brumagne, 2008). It is therefore possible that spinal manipulation in patients with sub-clinical or more chronic neck pain is able to improve the central processing of proprioceptive information, and that this is part of the mechanism by which high-velocity, low-amplitude spinal manipulation improve function and reduce chronicity and reoccurrence in these patient populations. It is possible that the changes in cortical somatosensory processing (Haavik-Taylor and Murphy, 2007c; Zhu et al., 2000, 1993), sensorimotor integration (Haavik-Taylor and Murphy, 2007c, 2008) and motor control (Haavik-Taylor and Murphy, 2007b, 2008; Marshall and Murphy, 2006; Suter and McMorland, 2002; Suter et al., 1999) that have been previously documented following high-velocity, low-amplitude spinal manipulation reflect changes in central processing of proprioceptive afferent input.

1.6. Limitations

It is worth noting that the SEP recording protocol utilized in the cited studies (Haavik-Taylor and Murphy, 2007c, 2010c; Haavik-Taylor et al., 2010) cannot rule out spinal cord and/or brainstem or subcortical changes. For example, more than 500 stimuli need to be averaged for reliable far-field brainstem and subcortical SEP peaks. SEP recordings also took several minutes to record and the recordings did not commence until the electrode impedance was re-checked after the cervical manipulations had been performed. Short lived spinal and/or brainstem changes can therefore not be ruled out either.

It is also worth noting that the cited studies (Haavik and Murphy, 2011, 2007c, 2008, 2010a,c; Haavik-Taylor et al., 2010; Marshall and Murphy, 2006) do not provide conclusive evidence that the observed neuroplastic changes following spinal manipulation are changes due to the correction of joint dysfunction. Clinicians practicing manipulation assess, via palpation and observation, that the area of spinal dysfunction has had its appropriate range of movement restored following manipulation. It is tempting to attribute the documented neurophysiological changes to this restoration of appropriate movement (as we have proposed). However, the observed neuroplastic changes could also merely be due to the afferent barrage associated with the manipulative thrust. Future studies should endeavour to address this question. We have however cited one longer term study which showed that the FFA time continued to improve up to one year after a 3 month period of treatment with exercise and/or manipulation in a group of chronic low back pain patients (Marshall and Murphy, 2008). This suggests that some of these neurophysiological markers do indeed have the capacity to improve following a period of successful care even in a chronic back pain group.

2. Conclusion

Many of the studies discussed in this paper show that spinal manipulation results in plastic changes in sensorimotor integration within the central nervous system in human participants. Collectively these studies provide evidence to support a central mechanism of action for high-velocity, low-amplitude spinal manipulation. What is not yet clear is the degree to which these changes correlate with beneficial clinical outcomes. It is also not clear whether these changes are due to the correction of spinal dysfunction, therefore normalizing aberrant afferent input to the CNS, or whether they are merely due to an afferent barrage associated with the manipulative thrust. These questions remain to be answered and are the focus of our ongoing research efforts.

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