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Cervical spine manipulation alters sensorimotor integration: A somatosensory evoked potential study

Heidi Haavik-Taylor *, Bernadette Murphy

Human Neurophysiology and Rehabilitation Laboratory, Department of Sport and Exercise Science, Tamaki Campus, University of Auckland, Private Bag 92019, 261 Morrin Road, Glen Innes, Auckland, New Zealand

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Abstract

Objective: To study the immediate sensorimotor neurophysiological effects of cervical spine manipulation using somatosensory evoked potentials (SEPs).
Methods: Twelve subjects with a history of reoccurring neck stiffness and/or neck pain, but no acute symptoms at the time of the study were invited to participate in the study. An additional twelve subjects participated in a passive head movement control experiment. Spinal (N11, N13) brainstem (P14) and cortical (N20, N30) SEPs to median nerve stimulation were recorded before and for 30 min after a single session of cervical spine manipulation, or passive head movement.
Results: There was a significant decrease in the amplitude of parietal N20 and frontal N30 SEP components following the single session of cervical spine manipulation compared to pre-manipulation baseline values. These changes lasted on average 20 min following the manipulation intervention. No changes were observed in the passive head movement control condition.
Conclusions: Spinal manipulation of dysfunctional cervical joints can lead to transient cortical plastic changes, as demonstrated by attenuation of cortical somatosensory evoked responses.
Significance: This study suggests that cervical spine manipulation may alter cortical somatosensory processing and sensorimotor integration. These findings may help to elucidate the mechanisms responsible for the effective relief of pain and restoration of functional ability documented following spinal manipulation treatment.

Keywords: Cervical spine manipulation; Human; Somatosensory evoked potentials; Brain plasticity; Somatosensory system; Sensorimotor integration

1. Introduction

Spinal manipulation is a commonly used conservative treatment for neck, back, and pelvic pain. The effectiveness of spinal manipulation in the treatment of acute and chronic low back and neck pain has been well established by outcome-based research (for review, see Hurwitz et al., 1996 and; Vernon, 1996). However, the mechanism(s) responsible for the effective relief of pain and restoration of functional ability after spinal manipulation are not well understood, as there is limited evidence to date regarding the neurophysiological effects of spinal manipulation. The evidence to date indicates that spinal manipulation can lead to alterations in reflex excitability (Herzog et al., 1999; Murphy et al., 1995; Symons et al., 2000), altered sensory processing (Zhu et al., 2000, 1993) and altered motor excitability (Herzog et al., 1999; Dishman et al., 2002; Suter et al., 2000).

Spinal manipulation is used therapeutically by a number of health professionals, including physical medicine specialists, physiotherapists, osteopaths and chiropractors. The different professions have different terminology for the "entity" or "manipulable lesion" that they manipulate. This manipulable lesion may be called "vertebral (spinal) lesion" by physical medical specialists or physiotherapists, "somatic dysfunction" or "spinal lesion" by osteopaths, and "vertebral subluxation" or "spinal fixation" by

* Corresponding author. Tel.: +649 3737599x82544; fax: +649 3737043.
E-mail address: h.taylor@auckland.ac.nz (H. Haavik-Taylor).

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chiropractors (Leach, 1986). Joint dysfunction as discussed in the literature ranges from experimentally induced joint effusion (Shakespeare et al., 1985), to 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). For the purposes of this paper, the "manipulable lesion" will be referred to as an area of spinal dysfunction.

There is a growing body of evidence suggesting that the presence of spinal dysfunction of various kinds has an effect on central neural processing. For example, several authors have suggested spinal dysfunction may lead to altered afferent input to the CNS (Bolton and Holland, 1996, 1998; Murphy et al., 1995; Zhu et al., 1993, 2000). Altering afferent input to the CNS is well known to lead to plastic changes in the way that it responds to any subsequent input (Brasil-Neto et al., 1993; Byl et al., 1997; Hallett et al., 1999; Pascual-Leone and Torres, 1993). Neural plastic changes take place both following increased (Byl et al., 1997; Pascual-Leone and Torres, 1993) and decreased (Brasil-Neto et al., 1993; Hallett et al., 1999; Ziemann et al., 1998) afferent input.

Altered afferent input from joints can lead to both inhibition and facilitation of neural input to related muscles. Numerous studies of painful joints have shown arthrogenous muscle inhibition (Hides et al., 1994; McPartland et al., 1997; Stokes and Young, 1984). However, even painless experimentally induced joint dysfunction (joint effusion) has been shown to inhibit surrounding muscles (Shakespeare et al., 1985). This altered motor control was also shown to persist even after aspiration of the joint effusion (Shakespeare et al., 1985). In the early 1980s, Steinmetz et al. demonstrated that relatively short (15–30 min) episodes of moderately intense afferent input to the spinal reflex pathways of rats causes increases in neural excitability that persists for several hours (Steinmetz et al., 1982, 1985). Once these facilitated areas are established, there may be no need for ongoing afferent input to maintain the altered output patterns. Since these early experiments, numerous studies have shown rapid central plastic changes after injuries and altered sensory input from the body (for review, see Wall et al., 2002). This can explain the findings of Shakespeare et al. (1985) of altered motor control persisting even after aspiration of the joint effusion. This process provides a potential explanation for altered neural processing as a result of joint dysfunction, and a rationale for the effects of spinal manipulation on neural processing that have been described in the literature.

Given that spinal dysfunction would alter the balance of afferent input to the CNS we propose that this altered afferent input may over time lead to potential maladaptive neural plastic changes in the CNS. We further propose that spinal manipulation can effect this. By recording SEPs and monitoring the peripheral nerve afferent volley, it is possible to determine where in the somatosensory pathway changes induced by spinal manipulation may be occurring.

2. Methods

2.1. Subjects

Twelve subjects (five women and seven men), aged 20–53 (mean age 29.9), participated in the spinal manipulation study. An additional twelve age-matched subjects (4 males and 8 females), aged 21–35 (mean age 27.1 years), participated in the passive head movement control study. The subjects were allocated into either group in a pseudo-randomized order. It was decided in advance that the first 12 volunteers that fit the inclusion/exclusion criteria for the study would become the manipulation group as this would enable the next 12 subjects to be age matched, if needed. However, both groups were of similar ages, so no additional age-matching was necessary. All 24 subjects agreed to have their cervical spines manipulated and/or their head moved by the researcher, and no subject knew which group they were taking part in prior to their experimental session taking place. To be included subjects could not have a history of neurological disease. The subjects were required to have a history of reoccurring neck pain or stiffness (e.g., repeatedly present during the performance of certain tasks such as work or study). However, at the time of the experiment all subjects were required to be pain free. This was done in order to assess the potential effects of joint manipulation delivered to dysfunctional joints alone without the presence of acute pain, as the presence of pain alone is known to induced a significant reduction of the post-central N20–P25 complex and a significant increase of the N18 wave (Rossi et al., 2003). Table 1 contains the experimental subjects' details, including their neck complaint history and known past neck (and/or head) trauma. Informed consent was obtained and the local ethical committee approved the study.

2.2. Somatosensory evoked potentials

All SEP recording electrodes (7 mm Ag/AgCl Hydrospot™ disposable adhesive electrodes from Physiometrix) were placed according to the International Federation of Clinical Neurophysiologists (IFCN) recommendations (Nuwer et al., 1994). 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 cites C3/4 and F3/4, which will be referred to as Cc′, and Fc′, respectively. All recording electrodes were referenced to the ipsilateral earlobe. The C6 spinous electrode was also referenced to the anterior neck (tracheal cartilage). The Erb's point electrode and the central Cc′ electrode were also referenced to the contralateral shoulder, as SEP components originating from subcortical regions are best recorded with a non-cephalic reference (Ulas et al., 1999). A ground electrode was attached to Fz. Stimuli consisted of electri-