The effect of spinal manipulation on biceps brachii muscle activity

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ABSTRACT

Objectives: This study investigated whether spinal manipulation of the ipsilateral C5/C6 segment facilitates linear summation of golgi tendon organ Ib afferent activity of the biceps brachii muscle as part of the convergent input on homonymous motor neuron pool excitability.
Methods: This placebo-controlled, single-blind, repeated measures study employed asymptomatic sample (n = 20) investigating two conditions: Activator Adjusting Instrument placebo spinal manipulation and spinal manipulative therapy.
Results: The immediate post-spinal manipulation therapy revealed a decrease in the biceps muscle electromyography Root Mean Square by 9.03 % (p = 0.39) in the face of an increase in biceps muscle force by 4.76 % (p = 0.155). Previous studies showed improved functional capacity of the homonymous muscle post-spinal manipulation. The presumed mechanism is a decrease in arthrogenic muscle inhibition through uncertain neural mechanisms. This study showed that C5/C6 spinal manipulation may facilitate biceps muscle autogenic inhibition, and suggests that through repeated spinal manipulation over time and the resulting habituation, altered arthrokinetic spinal reflex arcs caused by raised Aβ-fiber afferent discharge from somatic receptors in facet joint tissue may be desensitized with subsequent improved α-motor neuron functioning.
Conclusions: This study suggests a neural mechanism underlying the beneficial effect of spinal manipulation on arthrogenic muscle inhibition and α-motor neuron functioning in the symptomatic and asymptomatic individual. However, given the small sample size and large standard deviation, further research is warranted to add statistical significance to this finding.

INTRODUCTION

Spinal manipulation or known as spinal manipulative therapy (SMT) is a high-velocity low-amplitude (HVLA) thrust delivered at the end range of motion of facet joints in the spine, in the direction of the orientation of the facet joint articulation, often accompanied with an audible cracking sound (Millan *et al.*, 2012; Pickar, 2002). One of the most foremost theories for the neurophysiological mechanism underlying SMT entails the reduction of central sensitization. Central sensitization has been postulated to induce abnormal spinal reflex arcs which can affect the homonymous motor, nociceptive and possibly the autonomic neuronal pools by sensitizing mechano-insensitive nociceptors within and around the facet joint tissue (Olsen, 2015; Vernon, 2010). SMT is believed to correct alterations in the anatomical, physiological and / or biomechanical dynamics of individual vertebral segments which lead to the induction of the central sensitization, also known as the chiropractic subluxation complex, to its pre-injury / normal state, and in doing so restores the normal functioning of the nervous system (Gatterman, 2005; Pickar, 2002). However, the exact underlying neurophysiological effect of SMT on motor activity is uncertain and there is a lack of knowledge in the literature regarding its effects on asymptomatic individuals.

The electromyographic (EMG) response post-SMT may elucidate the neurophysiological effect of SMT on motor activity (Olsen, 2015; Pickar, 2002). Although many studies have showed that SMT can alter the excitability of the homonymous motor neuron pool and evoke spinal reflex activity (Olsen, 2015; Pickar, 2002), the literature shows conflicting evidence regarding the excitatory or inhibitory nature of the reflexive EMG response and excitability of the homonymous motor neuron pool post-SMT (Dunning and Rushton, 2009; Olsen, 2015). The immediate EMG response post-SMT entails an initial latent period consisting of a few milliseconds in duration followed by a transient increase in EMG amplitude, or, solely, a transient decrease in the EMG amplitude or decrease in the excitability of the homonymous motor neuron pool (Olsen, 2015; Pickar, 2002). The neural mechanism responsible for the EMG response latency occurring immediately post-SMT is largely unknown (Pickar, 2002). Several studies in the literature have postulated that a decrease in arthrogenic muscle inhibition (AMI) through either facilitation or disinhibition of the involved neural pathways is responsible for the transient increase in EMG amplitude and improved functional capacity of the homonymous muscle post-SMT (Dunning and Rushton, 2009; Suter and McMorland, 2002).

From a neurophysiological perspective, the literature has not yet linked the stimulatory effect of SMT on mechanoreceptors (type I-III nerve ending / somatic receptors) in the facet joint tissue with AMI; an increased discharge of Aβ-fiber afferents from mechanoreceptors in joint tissues causes Ib inhibition on the homonymous motor neuron pool, in the presence and absence of pain (Konishi *et al.*, 2003; Rice and McNair, 2010). Furthermore, although occupational and physical therapists use the principles behind habituation (neuroplasticity) to decrease abnormal neural responses to a stimulus by using specific techniques and exercises based on the effects of habituation, such as in the treatment of hyperesthesia (Lundy-Ekman, 2013), the concept of SMT achieving habituation to abnormal arthrokinetic spinal reflex arcs is not evident in the literature. This study investigated whether ipsilateral C5/C6 SMT facilitates linear summation of golgi tendon organ (GTO) Ib afferent activity (autogenic inhibition) of the biceps brachii muscle as part of the convergent input on the homonymous motor neuron pool excitability.

METHODS

Subjects

A convenience sample of 20 individuals asymptomatic for neck pain and bilateral upper extremity pain who presented himself or herself to the Durban University of Technology Chiropractic Day Clinic (8 females and 12 males) with a mean age of 26 ± 3.3 years was recruited. Only subjects between the ages of 18 and 40 were selected. Screening questions for the exclusion criteria and the clinical presence of any contraindication for cervical SMT (Puentedura *et al.*, 2012: 66) or any indication for further special investigations during a thorough case history, physical examination, and cervical spine regional examination were negative. This study was approved by the Institutional Research Ethics Committee of the Durban University of Technology |(IREC Reference Number: REC I I I/ 1 5), and written informed consent was obtained from all the subjects prior to the testing.

Equipment

Active EMG and dynamometry recording of the biceps muscle were made using the Bionomadix® (Biopac) complete wireless research system with four channel EMG recording, and TSD121C (Biopac) isometric hand dynamometer (0 kg to 100 kg) and amplifier. EL509 (Biopac) detection electrodes were made of Ag/AgCl laminated carbon with incorporated electrode gel cavity (16 mm diameter and 1.5 mm deep) in the form of parallel bars 27 mm long and 36 mm wide with inter-detection surface spacing of 1.0 cm. The full frequency spectrum of the biceps muscle EMG signal between 20 Hz and 450 Hz was captured (Dunning and Rushton, 2009: 509; Suter and McMorland, 2002). An– II Activator Adjusting Instrument (AAI) (Activator Methods) with a force setting of zero force was used to deliver an AAI placebo SMT (Humphries *et al.*, 2013).

Procedure

Each subject was instructed by the researcher to sit on a chair in front of an adjustable small table and to place his or her arm on the table. The adjustable table was set so that the subject's arm was horizontal to his or her shoulder. The subject's elbow was extended and the forearm supinated passively. The extended elbow remained in contact with the table and the distal supinated forearm hung off the table. The researcher correctly identified the C5/C6 spinal level by using specific palpation techniques (Benzel, 2012; Magee, 2008). The C5/C6 spinal levels were marked clearly on each subject's skin using a non-permanent marker pen. The proper skin preparations were administrated over the biceps muscle at the sites of the EMG electrode placement (Dunning and Rushton, 2009; Quach, 2007) before placing the electrodes correctly onto the biceps muscle (De Luca, 2002; Quach, 2007). The reference EMG electrode (27 mm x 36 mm) was placed on the posterior aspect of the ipsilateral deltoid muscle of each subject.

Thereafter each subject performed three sets of modified stretching of the biceps muscle based on the principles of the autogenic inhibition phase of proprioceptive neuromuscular facilitation (PNF) stretching. All three sets of modified stretching were 10 seconds in duration with two minute rest intervals and occurred in a single appointment. Before each set started, a 10 mm by 2 m tie down strap was strapped to the distal forearm of each subject by the researcher. The pull side of the TSD121C hand dynamometer 100 kg was attached to the tie down strap. A second 10 mm by 2 m tie down strap was used to attach the push side of the dynamometer to a 1 kg plate, which hung below the distal forearm of the subject (Figure 1). The dynamometer was then correctly calibrated with the attached 1 kg plate.

The weight of the 1 kg plate fully extended the elbow and passively stretched the biceps muscle to elicit the autogenic excitation phase of the modified stretching. The subjects were then instructed by the researcher to "resist against the weight of the 1 kg plate for 10 seconds without bending your elbow" to perform an isometric contraction of their biceps muscle and thereby to elicit the autogenic inhibition phase of the modified stretching. The subject's elbow must have remained stationary to hold the biceps muscle in a passively stretched position. The subjects were also instructed beforehand to "maintain the same constant biceps muscle contraction as possible for the whole duration of the 10 seconds of resistance" to rule out unwanted additional voluntary effort by the subjects. The raw EMG and dynamometry readings were recorded simultaneously during the entire duration of biceps muscle activity of each set of modified stretching. After the 10 seconds of resistance of each set, the tie down strap with the attached dynamometer and 1 kg plate was temporarily removed from the subject's distal forearm by the

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researcher during the rest periods. The EMG electrodes were not removed from the subject's biceps muscle during the rest periods.

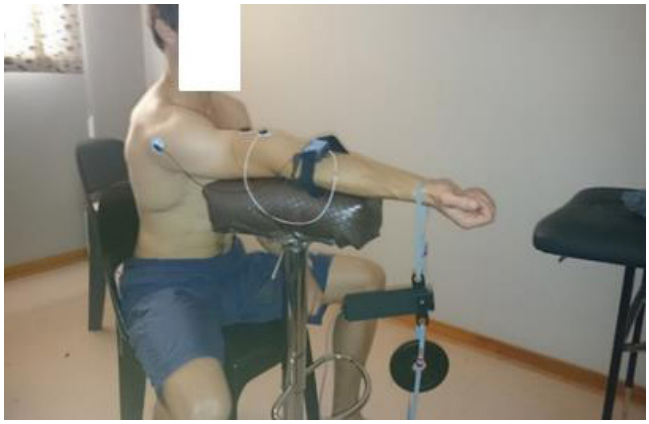

Figure 1: *The setup of the modified stretching of the biceps muscle based on the principles of the autogenic inhibition phase of PNF stretching.*

During the first set (AAI 1 intervention) and second set (AAI 2 intervention) of modified stretching, at the 4th second an AAI placebo SMT was correctly administered to the ipsilateral C5/C6 segment (Humphries *et al.*, 2013: 153). Although the AAI was set to a force setting of zero, the AAI placebo SMT still caused an audible cavitation type sound (Humphries *et al.*, 2013: 153). The researcher removed the contact of the AAI from each subject's skin immediately after the interventions. AAI placebo SMT cannot stimulate mechanoreceptors in the facet joint tissue (Huggins *et al.*, 2012: 53; Humphries *et al.*, 2013: 153) and may therefore have no effect on the linear summation of GTO Ib afferent activity of the biceps muscle during the modified stretching as part of the convergent input on the homonymous motor neuron pool excitability.

During the third set (SMT intervention) of modified stretching, at the 4th second the researcher correctly locked up the ipsilateral C5/C6 facet joint prior to administering the standardized posterior-to-anterior HVLA thrust at the 6th second (Bergmann and Peterson, 2010; Redwood and Cleveland, 2003). A two second time interval was given in order to lock up the C5/C6 facet joint. The researcher then re-positioned the participant's head to the starting position and removed any contact by the researcher from each subject immediately. The C5/C6 SMT can stimulate mechanoreceptors in the facet joint tissue (Dunning and Rushton, 2009; Olsen, 2015) and may therefore have an effect on the linear summation of GTO Ib afferent activity of the biceps muscle during the modified stretching as part of the convergent input on the homonymous motor neuron pool excitability.

Data and statistical analyses

The MP 150 data acquisition system and AcqKnowledge® analysis software was used to capture one-second EMG segments during the entire force plateau of each set of the modified stretching of the biceps muscle. The raw EMG signal was processed through Root Mean Square (RMS) analysis and the dynamometer data by the MP 150 data acquisition system and AcqKnowledge® analysis software, to obtain the variables of biceps muscle EMG RMS and force. Objective analyses made use of the mean values of each variable for the 500 millisecond period before and after, at the 4th second for the AAI 1 and AAI2 interventions and at the 6th second for the SMT intervention, during each set of modified stretching. These values were also subtracted from one another to calculate change scores for each variable. This process resulted in 120 biceps muscle EMG RMS and 120 biceps muscle force means in total encompassing a pre- and post-values for the AAI and SMT conditions. The mean percentage of change (Dunning and Rushton, 2009) in active biceps muscle activity for the variables was calculated.

Bivariate analyses consisted of independent t-tests, adjusted for unequal variance where appropriate. All analyses were performed with a confidence interval of $\alpha = 0.05$, as well as an optimal alpha (α) to account for the lack of power inherent in small sample designs as per Mudge *et al.*'s (2012) recommendations. The optimal αs were determined separately for the two sample comparisons between the AAI 1 and AAI 2 interventions; optimal $\alpha = 0.232$, and those between the SMT intervention and both AAI interventions (AAI 1 + AAI 2), optimal $\alpha = 0.203$. The calculation optimized α to find a medium effect size of $d = 0.5$ which was an equal weight for the cost of errors and a prior probability ratio of 1. The optimal α for the AAI intervention comparison yielded a power of 0.647, a large improvement from that of the conventional α, power = 0.338. Similarly, the optimal α for the SMT intervention and AAI interventions (AAI 1 + AAI 2) comparison yielded a power of 0.707, a large improvement from that of the conventional α, power = 0.429.

RESULTS

The 60 interventions applied to the 20 participants compromised 20 SMT interventions (33.3 %) and 40 AAI interventions (66.7 %). For the AAI interventions, 40 experienced an audible cavitation (100 %) and null experienced a non-audible cavitation (0 %). For the SMT interventions, 17 experienced an audible cavitation (85 %) and 3 experienced a non-audible cavitation (15 %).

The mean scores of the immediately pre- and post-AAI 1 and AAI 2 interventions are fairly similar across the variables for biceps muscle EMG RMS and force (Table 1). While differences are present between the AAI 1 and AAI 2 interventions, they are generally much smaller than the differences between them and the immediately pre- and post- SMT intervention, although the standard deviation of the variables in relation to the differences between the interventions is also fairly large (Table 1). The AAI 1 and AAI 2 interventions show a decrease in the biceps muscle EMG RMS along with a decrease in biceps muscle force,

Table 1: *The EMG RMS and muscle force of the biceps brachii immediately pre- and post-interventions (M ±SD)*

	Baseline (500 ms pre-intervention)	Post-intervention (first 500 ms)	Percentage difference (%)	
RMS (µV)				
Placebo AAI 1	41,98 ± 20,83	41,21 ± 20,27	1.86	$p = 0.39$
Placebo AAI 2	37,41 ± 20,19	37,4 ± 22,36	0.05	
SMT	49,53 ± 23,48	45,25 ± 23,93	9.03	
Force (kg)				
Placebo AAI 1	$2{,}397\text{-}1 \pm 2{,}238^{-2}$	$2{,}377\text{-}1 \pm 2{,}304^{-2}$	0.85	$p = 0.155$
Placebo AAI 2	$2{,}369\text{-}1 \pm 2{,}153^{-2}$	$2{,}323\text{-}1 \pm 2{,}162^{-2}$	1.97	
SMT	$2{,}327\text{-}1 \pm 3{,}69^{-2}$	$2{,}440\text{-}1 \pm 5{,}419^{-2}$	4.76	

whereas the SMT intervention reveals a decrease in biceps muscle EMG RMS and an increase in the biceps muscle force immediately post-interventions (Table 1).

In an analysis of the similarity of biceps muscle EMG RMS between the AAI 1 and AAI 2 interventions, no significant difference was found at both a conventional α level, $t(38) = 0.5344$, $p > 0.05$ ($p = 0.5962$), and an optimal α level, $t(38) = 0.5344$, $p > 0.232$. In an analysis of the difference in biceps muscle EMG RMS between the AAI interventions (AAI 1 + AAI 2) and the SMT intervention no significant difference was found at a conventional α level, $t(57) = 0.881$, $p > 0.05$ ($p = 0.3895$), or an optimal α level, $t(57) = 0.881$, $p > 0.203$. In an analysis of the similarity in biceps muscle force between the AAI interventions (AAI 1 + AAI 2) and the SMT intervention no significant difference was found at the conventional α level, $t(57) = 1.482$, $p > 0.05$ ($p = 0.1549$), but displayed a difference at the optimal α level, $t(57) = 1.482$, $p < 0.203$.

DISCUSSION

Proprioceptive neuromuscular facilitation stretching is the most effective stretching technique to increase muscle flexibility and range of motion, and is commonly used in the athletic and clinical environments (Sharman *et al.*, 2006). The underlying neural mechanisms involving PNF stretching consist of: firstly, autogenic excitation phase by way of facilitation of the Ia muscle spindle spinal reflex arc (gamma loop); secondly, autogenic inhibition phase by way of facilitation of the GTO Ib inhibitory di-synaptic spinal reflex arc; and thirdly, reciprocal inhibition phase via facilitation of the Ia inhibitory spinal pathways as a result of contraction of the antagonist muscle group (Bandy and Sanders, 2007; Sharman *et al.*, 2006). The autogenic inhibition phase of PNF stretching is performed by placing the targeted muscle passively in a lengthened position followed by an active low force muscle contraction for several seconds to activate the Ib afferent maximally (Bandy and Sanders, 2007; Sharman *et al.*, 2006). Many studies have demonstrated that the reduced efferent (motor) drive to the muscle by way of autogenic inhibition is a major factor that assists in elongation of the targeted muscle (Sharman *et al.*, 2006; Umphred *et al.*, 2013).

Historically the function of the GTO was thought to be a protective reflex in which a strong and potentially damaging muscle force from excessive loading will reflexively inhibit the muscle by way of the autogenic inhibition, to cause lengthening of the muscle instead of trying to maintain the muscle force and risking damage (Khurana, 2014; Lundy-Ekman, 2013). Although the GTO provides some protection by way of the autogenic inhibition and has been shown to facilitate decreased excitability of the homonymous motor neuron pool, the role of the GTO resides more in supplying the central nervous system with sensory information regarding active muscle tension and thus force in the muscle generated, via their Ib afferent fibers (Khurana, 2014; Lundy-Ekman, 2013). The effect of GTO input to the homonymous motor neuron pool is not on its own powerful enough to inhibit voluntary muscle contraction by way of the lateral corticospinal tract (LCST, pyramidal motor pathway), because recent studies have affirmed that maximal GTO Ib afferent activity occurs before 50% of maximal voluntary muscle contraction (FitzGerald *et al.*, 2012; Lundy-Ekman, 2013). A protective reflex for a skeletal muscle is considered to be predominantly provided by the LCST which causes presynaptic inhibition of the homonymous actively stretched prime mover muscle spindle afferents close to the contact points with their α motor neurons by way of the interpolation of inhibitory internuncials in the intermediate grey matter of the spinal cord (FitzGerald *et al.*, 2012).

The behaviour of GTOs demonstrates an immediate response to muscle tension and consists of an initial dynamic response – a burst in GTO discharge within 0.5 seconds. A static response immediately follows and consists of a gradual decline to a constant GTO discharge (Mileusnic and Loeb, 2006; Plowman and Smith, 2007). Historically it was thought that GTOs only respond to high forces but several studies have demonstrated that the activation of multiple motor units simultaneously, such as during high muscle force output, cause the GTOs to demonstrate non-linear summation and produce Ib afferent activity that is smaller compared to the activation of a single or two motor units over time. Studies

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have affirmed that the activation of a single or two motor units such as during very low muscle force output, causes the GTOs to demonstrate linear summation and produce higher Ib afferent activity (Mileusnic and Loeb, 2006; Sharman *et al.*, 2006). Although the GTO behaviour depends on the generated muscle tension that the extrafusal muscle fibers of the motor unit exert on the loosely packed innervated collagen fibrils found inside the lumen of the GTO, the response of the GTO also depends on the type of motor unit being activated (Mileusnic and Loeb, 2006).

Motor unit recruitment as well as the firing frequency of lower motor neurons (alpha/α motor neurons and gamma/γ motor neurons) are entirely dependent on the level of force and speed of muscle contraction by the voluntary effort of an individual (LCST), in the normal non-pathological state (Lundy-Ekman, 2013; Merletti and Parker, 2004). A direct relationship thus exists between the motor unit recruitment frequency and the homonymous muscle's generated force in relation to the voluntary effort of the individual (Lundy-Ekman, 2013; Merletti and Parker, 2004). A low force muscle contraction will cause the recruitment of low-threshold motor units with at least one of their extrafusal muscle fibers inserting into a GTO and intertwining with the loosely packed innervated collagen fibrils inside the GTO, resulting in linear summation of Ib afferent activity. As the muscular force becomes higher / faster by the voluntary effort of the individual, so the higher-threshold motor units will be recruited with at least one of their extrafusal muscle fibers inserting into a GTO and intertwining with the loosely packed innervated collagen fibrils inside the GTO, resulting in more non-linear summation of Ib afferent activity (Mileusnic and Loeb, 2006). Because the wiring of the α motor neuron entails multiple spinal segmental and supraspinal inputs, a single input may be insufficient to trigger the firing threshold of the homonymous α motor neurons and may solely influence the excitability of them. The summate balance between all these influences may decide if the homonymous α motor neurons increase or decrease their discharge frequency or excitability (Mense and Gerwin, 2010).

Linear summation of biceps muscle GTO Ib afferent activity may produce inhibitory post-synaptic potentials (IPSPs) in the homonymous α motor neurons and thereby solely decrease the excitability state of the homonymous motor neuron pool (Bandy and Sanders, 2007: 59; Mileusnic and Loeb, 2006) that facilitates the elongation of the biceps muscle during the modified stretching in this study (Sharman *et al.*, 2006; Umphred *et al.*, 2013). A decrease in the biceps muscle EMG amplitude (RMS) along with a decrease in biceps muscle force may result (Lundy-Ekman, 2013; Merletti and Parker, 2004). The results of this study supports the notion of the autogenic inhibition phase of PNF stretching reducing the efferent drive to the homonymous muscle; both the placebo SMT groups (AAI and AAI 2 interventions) showed a mean decrease in biceps muscle EMG RMS by less than 1.87 % and a mean decrease in biceps muscle force by less than 1.98 % (Table 1). No significant difference was found between the placebo SMT groups.

The C5/C6 SMT showed a much larger difference than the placebo SMT groups for the EMG RMS and muscle force of the biceps muscle immediately pre- and post-interventions (Table 1). To the contrary, this large difference could not be quantified in terms of statistical significance due to the large standard deviation of the variables relative to the differences between the interventions and possibly due to the small population group (Table 1). The immediate post-C5/C6 SMT revealed a mean decrease in the biceps muscle EMG RMS of 9.03 % (p = 0.39) in the face of a mean increase in the biceps muscle force of 4.76 % (p = 0.155) and a summation of percentage difference between the biceps muscle force and EMG RMS of 13.79 %; the immediate post-placebo SMT groups showed a summation of percentage difference between the biceps muscle force and EMG RMS by less than 1.93 % (Table 1). This finding is noteworthy because the variables of EMG RMS and muscle force are also entirely dependent on the voluntary effort of the subject (Lundy-Ekman, 2013; Merletti and Parker, 2004).

The large standard deviation (Table 1) is possibly caused by each subject contracting their biceps muscle at their own intensity by their free will. The variables of biceps muscle EMG RMS and force could therefore not be standardized by depending on the subjects to perform a constant low force contraction of the biceps muscle during the modified stretching to rule out unwanted voluntary effort (Lundy-Ekman, 2013; Merletti and Parker, 2004). This is problematic because an increase in voluntary effort by the subjects during the modified stretching of the biceps muscle can cause the same EMG response post ipsilateral C5/C6 SMT: an increase in voluntary contraction of the biceps muscle by way of the LCST will cause an increase in both the biceps muscle EMG RMS and force (Lundy-Ekman, 2013; Merletti and Parker, 2004); Suter and McMorland (2002) found a transient increase in biceps muscle EMG RMS and force immediately post-C5/C6 SMT. A subconscious adjustment in voluntary effort by the mean subject could have been the mechanism responsible for the increase in biceps muscle force immediately post the C5/C6 SMT, due to an emotional component such as fright, excitement or fear experienced (Engelhardt *et al.*, 2001; Rice and McNair, 2010). A significant difference in biceps muscle force was found between the placebo SMT groups and the C5/C6 SMT at the optimal α level, $t(57) = 1.482$, $p < 0.203$ immediately post-interventions.

The anomalous decrease in biceps muscle EMG RMS during an increase in biceps muscle force observed immediately post the C5/C6 SMT is theoretically due to the spatial summation of combined, peak linear / non-linear biceps muscle GTO Ib afferent activity caused by the modified stretching and possibly an increase in voluntary effort by the mean subject, plus the transient increase in facet joint mechanoreceptor Aβ-fiber afferent activity caused by the stimulatory effect of the C5/C6 SMT; that produced the

transient summation of Ib inhibition on the homonymous α motor neurons that was larger than the summation of excitation produced by the LCST innervating the biceps muscle α motor neurons. As part of the convergent input on the α motor neurons innervating the biceps muscle; the C5/C6 SMT caused transient facilitation of the vertebral segment's facet joint Ib inhibitory spinal pathway(s) and thereby facilitated (reinforced) the autogenic inhibition of the biceps muscle (facilitated GTO Ib inhibitory di-synaptic spinal reflex arc) produced by the modified stretching, which resulted in sufficient IPSPs in the biceps muscle α motor neurons that drove the membrane potential at the axon hillock of each depolarized biceps muscle α motor neuron away from firing threshold and thereby prevented the LCST from transiently generating sequential action potentials in the biceps muscle α motor neurons. When the stimulus is removed that caused the generation of excitatory post-synaptic potentials (EPSPs) or IPSPs in the target neuron, the disturbance in the membrane potential of the target neuron will fade away and return to baseline (Rastogi, 2006; Starr and McMilan, 2015). The HVLA thrust delivered during the C5/C6 SMT is a rapid short-lived propulsive thrust that can produce a force between about 220 N to 550 N with a duration of between about 200 ms to 420 ms (Herzog *et al.*, 1993). The C5/C6 SMT would have therefore activated the mechanoreceptors in the C5/C6 facet joint tissue transiently with subsequent transient facilitation of biceps muscle autogenic inhibition during the modified stretching. This finding suggests that spinal manipulation may cause transient Ib inhibition on the homonymous motor neuron pool, theoretically by causing transient facilitation of the facet joint Ib inhibitory di- or tri-synaptic spinal reflex arc(s) via interpolation with Ib internuncials.

The Ib internuncials found in Rexed lamina VI and VII in the intermediate grey matter of the spinal cord were initially defined by their response to input from the GTOs. The more recent literature shows that a range of sensory input, including Aβ-fiber afferents from mechanoreceptors in joint tissue, can reach the Ib internuncials through several independent spinal reflex arcs and that there is little specialization of Ib internuncial by the type of sensory input (Brushart, 2011; Greger and Windhorst, 2013). Studies have substantiated that joint Aβ-fiber afferents can cause similar Ib inhibition on the homonymous α motor neurons (Brushart, 2011; Greger and Windhorst, 2013).

A current accepted neurophysiological mechanism underlying both spinal manipulation (DePalma, 2011; Sterling and Kenardy, 2011) and AMI in the literature entails affecting the afferent discharge of somatic receptors (type I-IV nerve endings / mechanoreceptors and nociceptors) found in joint tissues (Rice *et al.*, 2014; Rice and McNair, 2010). Spinal manipulation can stimulate and cause a transient increase in Aβ-fiber afferent discharge (Millan *et al.*, 2012; Pickar, 2002) of type I Ruffini end-organs around the collagen fibers of the superficial layers of the facet capsular tissue (slowly adapting, low-threshold, static and dynamic mechanoreceptors); type II Pacinian corpuscles in the deeper layers of the facet capsular tissue (rapid adapting, low threshold and dynamic mechanoreceptors); and type III Golgi ending nerve endings at the junction between the inner and more superficial layers of the facet joint capsular ligament (very slowly adapting, high threshold and dynamic mechanoreceptors), their existence having been vindicated in many studies (McPetty, 2011; Sterling and Kenardy, 2011). Studies have affirmed that type I-III nerve endings in the facet joint tissue respond to the impulse of a HVLA load applied during SMT and not to loads with a slower force-time profile (Colloca *et al.*, 2000).

An increase in joint mechanoreceptor Aβ-fiber afferent discharge is strongly associated with AMI and it is postulated that joint afferent input has competing excitatory and inhibitory influences on the homonymous motor neuron pool; in a dysfunctional joint, the net effect can be inhibitory (Konishi *et al.*, 2003; Rice and McNair, 2010). The arthrokinetic spinal reflex arcs, in which somatic receptors in the joint tissue affect the spinal segmental innervated muscle activity, is dependent on the type of receptor activated. Activation of joint mechanoreceptors can, in addition to affecting the spinal segmental innervated muscles directly by affecting the homonymous α motor neuron's excitability, also affect the muscles by affecting the homonymous γ motor neuron-muscle spindle loop (Petty, 2011). Any joint dysfunction which can affect its mechanoreceptor Aβ-fiber afferent discharge can cause impairment of its arthrokinetic reflex functioning and produce abnormal patterns of spinal reflex arc activity (Middleditch and Oliver, 2005) and thereby result in weakness of the spinal segmental innervated muscles (Middleditch and Oliver, 2005; Porter, 2013), namely AMI (Rice *et al.*, 2014; Rice and McNair, 2010). Furthermore, a weak correlation exists between pain and AMI; several studies have affirmed the presence of significant AMI in the absence of pain. As little as 10 ml of fluid infused into joints can cause notable muscle inhibition, and even small, clinically undetectable joint effusions can cause significant AMI (Hopkins, 2006; Hopkins *et al.*, 2001; Rice *et al.*, 2014; Rice and McNair, 2010).

The finding of SMT facilitating Ib inhibition on the homonymous motor neuron pool suggests that by applying SMT over time to facet joints with raised Aβ-fiber afferent discharge and thus altered arthrokinetic reflex functioning, may desensitize the vertebral segment's facilitated facet joint Ib inhibitory spinal pathways by causing Ib inhibition on the homonymous lower motor neurons that is already subjected to AMI, with subsequent improved α-motor neuron functioning. By gradually or repeatedly inducing an abnormal stimuli or abnormal response over time, habituation can be achieved and result in a reduction of the abnormal stimuli or abnormal response (Lundy-Ekman, 2013; Sweetow and Sabes, 2010). Du Plessis (2014) investigated the effect of C5/C6 SMT on the EMG and strength of the biceps muscle in participants with chronic neck pain over a three week

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period. Three measurements were recorded spanning the three weeks. The mean dynamometry increased from the first reading recorded as 20.67 kg to the second reading recorded as 21.49 kg, and increased from the second to the third reading recorded as 22.99 kg, with a significant increase in the biceps muscle strength over the three weeks ($p = 0.005$). The mean EMG amplitude increased from the first reading recorded as 150.76 mV to the second reading recorded as 151.66 mV, and increased from the second to the third reading recorded as 152.02 mV, with a significant increase in the biceps muscle activity over the three weeks ($p = 0.000$). Du Plessis (2014) concluded that the underlying neural mechanism responsible for the significant increase in the muscle force and activity post the SMT is unclear. The incremental increase in muscle force and activity over the three week period is possibly due to the SMT desensitizing altered arthrokinetic spinal reflex arcs present in the mean subject.

The theory of SMT causing transient Ib inhibition on the homonymous motor neuron pool is substantiated by many studies that observed an EMG response latency, a transient decrease in EMG amplitude, as well as the transient decrease in excitability of the homonymous motor neuron pool immediately post-SMT. Herzog *et al.* (1999) investigated the effect of SMT applied to the cervical, thoracic and lumbar spine and sacroiliac regions on the muscle activity of their associated paraspinal musculature in asymptomatic participants. They reported an EMG response latency occurring within 50 ms to 200 ms immediately after the HVLA thrust. Colloca and Keller (2001) confirmed these latter findings in symptomatic patients with low back pain. They reported an EMG response latency occurring within 2 ms to 3 ms immediately after AAI SMT. Dishman *et al.* (2002) investigated the immediate pre- and post- SMT effects of lumbar SMT on the homonymous motor neuron pool excitability in participants with low back pain, by measuring and recording the amplitude of the tibial nerve H-reflex recorded from the gastrocnemius muscle. They found a significant transient decrease in the homonymous motor neuron pool excitability immediately post the SMT.

The notion of SMT causing transient disinhibition of the homonymous α motor neurons are plausible (Dunning and Rushton, 2009; Olsen, 2015), but solely after the SMT has caused transient Ib inhibition on the homonymous motor neuron pool. Numerous studies have showed a transient increase in EMG (spike) following the EMG response latency immediately post-SMT (Olsen, 2015; DeVocht *et al.*, 2005; Pickar, 2002; Colloca and Keller, 2001; Herzog *et al.*, 1999), as well as an increase in muscle strength and / or decrease in AMI immediately post-SMT (Dunning and Rushton, 2009; Pickar, 2002,; Suter and McMorland, 2002; Suter *et al.*, 2000).

The transient increase in EMG following the EMG response latency may be due to the SMT mechanically reducing a chiropractic subluxation complex present (Olsen, 2015; Pickar, 2002), and thereby causing disinhibition of the altered arthrokinetic spinal reflex arcs. Repetitive postural strain or trauma to the spine can cause alterations in the normal anatomical, physiological and / or biomechanical dynamics of individual vertebral segments and produce relatively large vertebral motions that achieve a new position of stable equilibrium. The higher energy level needed to achieve the new position of stable equilibrium can place additional mechanical stress or overload on the facet joint capsular tissue and / or cause uneven or increased unilateral facet joint loading. These alterations in the vertebral segment can cause tension, pressure, stretching or irritation of the facet joint capsular tissue as well as the displacement of collagen in the facet joint capsular ligament (Gatterman, 2005; Vernon, 2010), and thereby stimulate (depolarize and sensitize) mechanoreceptors within the facet joint tissue and subsequently increase their Aβ-fiber afferent discharge frequency (Dunning and Rushton, 2009; Vernon, 2010), in the presence and absence of pain (Rice *et al.*, 2014; Rice and McNair, 2010). Raised Aβ-fiber afferent discharge from facet joint tissue caused by a chiropractic subluxation complex can theoretically cause alterations in its arthrokinetic reflex functioning with subsequent facilitation of the vertebral segment's facet joint's Ib inhibitory spinal reflex arcs and thereby AMI.

Prior to suggesting that the C5/C6 SMT facilitated biceps muscle GTO Ib afferent activity as part of the convergent input on the homonymous motor neuron pool excitability, it is vital to explore and consider other causes or contributors to the decrease in biceps muscle EMG RMS during the increase in biceps muscle force immediately post the C5/C6 SMT. Although several contributors may exist, the most relevant will be emphasised in this article.

Although all subjects in the this study were asymptomatic, nociceptors in the cutaneous and facet joint tissue may have also been stimulated due to the propulsive HVLA thrust applied during the SMT intervention (Millan *et al.*, 2012; Pickar, 2002). It is unlikely though that an increase in nociceptors Aδ- and / or C-fiber afferent discharge caused or contributed to the decrease in biceps muscle EMG RMS immediately post the C5/C6 SMT, because the literature has shown that activated nociceptors afferent from joint and cutaneous tissue are more likely to cause an excitatory influence on the homonymous motor neuron pool excitability to result in or contribute to the induction of a muscle spasm (Mense and Gerwin, 2010; Steward, 2012). An excitatory influence on the homonymous motor neuron pool may contribute to an increase in the EMG RMS of the homonymous muscle during voluntary muscle contraction by causing disinhibition of the homonymous α motor neurons (Fitzgerald *et al.*, 2012; Merletti and Parker, 2004). Facilitation of an increased state of firing of the homonymous α motor neurons will not cause an increase in muscle strength (Mense and Gerwin, 2010), but may cause a decrease in muscle strength during voluntary muscle contraction if the muscle spasm is severe enough (Page *et al.*, 2010). The facilitation of increased excitability of

the homonymous motor neuron pool can cause alterations in the motor unit recruitment, lower the homonymous α motor neurons activation threshold, or lower their irritability threshold (Page *et al.*, 2010).

In addition, many studies have established that the input of Aβ-fiber afferents of mechanoreceptors is more powerful than the input of Aδ- and / or C-fiber afferents of nociceptors into the central nervous system (Haines, 2012; Steward, 2012). Many studies have shown that SMT stimulates and causes an increase of Aβ-fiber afferent discharge of mechanoreceptors in facet joint tissue (DePalma, 2011; Millan *et al.*, 2012). Also, the literature has establish that branches of large Aβ-fibers afferent from joint tissues have shared innervation with the homonymous wide dynamic range neurons in the dorsal horn of the spinal cord that enter the posterior column-medial lemniscal pathway in the white matter of the spinal cord (Haines, 2012; Steward, 2012). Stimulation of the posterior column by way of the joint Aβ-fibers afferent sends antidromic conducted action potentials via collateral braches into the dorsal horn which in turn stimulate the enkephalinergic interneurons that inhibit the transmission of nociceptive signals of the Aδ- and / or C-fibers via the anterolateral system. This is the physiological basis for the gate theory of pain (Haines, 2012; Steward, 2012). Further, none of the participants of this study reported experiencing pain during the SMT intervention.

The activation of mechanoreceptors in the cutaneous tissue can exert an excitatory or inhibitory influence on the homonymous motor neuron pool excitability (Mense and Gerwin, 2010; Steward, 2012). However, it is unlikely that stimulation of mechanoreceptors in the cutaneous tissue by the C5/C6 SMT (Millan *et al.*, 2012; Pickar, 2002) solely caused the decrease in biceps muscle EMG RMS but may have served as a contributor. The placebo SMT may have also stimulated mechanoreceptors in the cutaneous tissue at the C5/C6 segment due to the contact of the AAI with the skin (Huggins *et al.*, 2012; Humphries *et al.*, 2013), but showed a much smaller decrease in the biceps muscle EMG RMS compared to the post-C5/C6 SMT, a difference of more than 7.16 %.

It is unlikely that the normal physiological functioning of the elbow joint arthrokinetic reflex activity caused the decrease in biceps muscle EMG RMS immediately post the C5/C6 SMT. When a joint capsule is stretched during joint movement the joint mechanoreceptors will cause activation of the muscles which will reduce the joint capsular stretch and cause inhibition of the muscles which will increase the joint capsular stretch (Middleditch and Oliver, 2005; Petty, 2011). During the modified stretching of the biceps muscle in this study; the mechanoreceptors in the elbow joint tissue would have exerted powerful tonic excitatory influences on the α motor neurons innervating the elbow flexor muscles with subsequent contribution to an increase in biceps muscle EMG RMS, and exerted inhibitory influences on the α motor neurons innervating the elbow extensor muscles, to reduce the capsular stretch of the extended elbow joint (FitzGerald *et al.*, 2012; Mense and Gerwin, 2010). To the contrary, the C5/C6 SMT facilitating effect on the biceps muscle autogenic inhibition during the modified stretching was theoretically larger than the spatial summation of excitation produced by the LCST and elbow joint arthokinetic reflex activity as part of the convergent input on the homonymous motor neuron pool excitability.

There are several limitations to this current study that need to be acknowledged. No verifications may exist to ensure that the desired spinal levels of manipulation will be indeed the specific C5/C6 level (Dunning and Rushton, 2009). By contrast, specific palpation of the vertebral bodies can lead to correct spinal level identification (Benzel, 2012; Magee, 2008). Although the same person (the researcher) applied the C5/C6 SMT to all participants, the magnitude of the thrusting force of the SMT applied cannot be standardized between all of the subjects. The exact replication of EMG electrode placement between all participants cannot be standardized and verified (Dunning and Rushton, 2009: 502). The exact replication of a low force isometric contraction of the biceps muscle during the modified stretching between all the subjects cannot be standardized due to voluntary effort (Lundy-Ekman, 2013; Merletti and Parker, 2004).

CONCLUSION

A decrease in biceps muscle EMG RMS in the face of an increase in biceps muscle force immediately post-C5/C6 SMT during modified stretching of the biceps muscle based on the principles of the autogenic inhibition phase of PNF stretching suggests that the C5/C6 SMT facilitated biceps muscle GTO autogenic inhibition as part of the convergent input on homonymous motor neuron pool excitability. This phenomenon has not been reported in the literature, due to EMG studies having investigated the effects of SMT on muscle activity pre- and post-maximum voluntary muscle contraction and not the effect of SMT on muscle activity during facilitated GTO Ib afferent activity which is what is required to produce the spatial summation of sufficient IPSPs in the homonymous α motor neurons to drive the membrane potential at the axon hillock of each depolarized homonymous α motor neuron away from firing threshold in order to prevent the LCST from transiently generating sequential action potentials in the homonymous α motor neurons. Also, the LCST (voluntary effort) has a stronger input on the lower motor neurons than Ib inhibition and thereby masked the Ib inhibitory effect of the SMT in these EMG studies. The theory of SMT causing transient Ib inhibition on the homonymous motor neuron pool is substantiated throughout the literature that showed an EMG response latency, a transient decrease in the EMG and decrease in excitability of the homonymous motor neuron pool immediately post-SMT (Colloca and Keller, 2001; DeVocht *et al.*, 2005; Dunning and Rushton, 2009; Dishman *et al.*, 2002; Herzog *et al.*, 1999; Pickar, 2002; Suter and McMorland, 2002; Olsen, 2015).

Further research is warranted to add statistical significance to this finding in order to substantiate the suggestion that for optimal management of the symptomatic and asymptomatic patient with muscle weakness suspected to be of arthrogenic nature, the application of SMT to the segmentally innervated facet joints may be a beneficial approach before traditional strength rehabilitation or training is initiated. SMT may desensitize altered arthrokinetic spinal reflex activity present with subsequent improved α-motor neuron functioning.

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