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Contents

JCCA Vol 58 No 2 ISSN 0008-3194 (Print) and ISSN 1715-6181 (Electronic)

Contents

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Editorial Board

Special issue on spine neuromuscular control

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Why do a special issue on spine neuromuscular control?

Chiropractic research capacity in Canada, the United States and throughout the world has undergone tremendous development over the past 15 years. Unique to Canada is that chiropractors who are also full-time researchers, play an important role not simply in chiropractic teaching institutions (Université du Québec à Trois-Rivières and Canadian Memorial Chiropractic College), but they have also been integrated into more than a dozen research intensive universities throughout the country. Several of such researchers have expertise in biomechanics, motor control and neurophysiology.[1]

The chiropractic profession, especially in Canada, has supported the development of research, firmly believing that basic science, innovation and professional development are intertwined. Given the increasing number of chiropractic researchers in these closely related fields of fundamental research, the Journal of the Canadian Chiropractic Association (JCCA) invited a number of chiropractic clinician-scientists to submit their most recent and innovative work to a special issue dedicated to spine neuromuscular control. In this unique issue of the JCCA, the often uneasy and perhaps cloudy relationship between basic and applied chiropractic research will be explored, with a particular focus on spine neuromuscular control mechanisms.

Spine neuromuscular control has remained a topic of interest throughout chiropractic's history. In the early years of the profession, communication between the central and the peripheral nervous systems, as well as the afferent and efferent control mechanisms between the central nervous system and the spinal joints were key features of the chiropractic theories.[2] As illustrated in the following quotation, Verner's (1941)[3] views of the possible mechanisms governing spinal function were, in the early 1940s, not too far from our contemporary understanding of spinal neuromuscular control.

> *"Anatomical disrelation may be perpetuated through the somatic reflex arc, in some people. For example, a contracted muscle may irritate its own afferent nerve, which in turn may stimulate its own motor nerve. Thus the contraction may be perpetuated indefinitely in some people."*

Based on the anatomy and physiology knowledge of the time, and since chiropractic was primarily theory driv-

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en during its early years, several ideas and theories were put forward regarding its mechanism. Only one final step needed to be taken: conduct research that could test the different theories and the related hypotheses.

When compared to other health professions, research is relatively new to chiropractic and I (MD) have once suggested that its value resides mostly in the credibility and recognition gained by engaging, as a profession, in the act of research.[4] In 2014, we can undoubtedly state that the chiropractic profession has firmly tackled the challenge of engaging in research. Chiropractic researchers have made significant contributions to our understanding of spine neuromuscular control. The goal of the present JCCA special issue is to introduce and highlight contemporary research in the field of spine neuromuscular control and other related topics. By focusing on research areas related to our profession, and by showcasing the work of chiropractic researchers and their collaborators, we hope that clinical scientists, field practitioners and patients will discover (or rediscover) the breadth of expertise developed throughout the last decade as well as the most recent advances in fundamental and applied chiropractic research.

What will you find in this special issue of the JCCA?

The contents of the present issue represent the spectrum of approaches to research. The central theme is approached from methods including case studies, animal models, experimental studies, treatment interventions and narrative reviews.

The muscular response to both injury and spinal manipulative therapy (SMT) is explored. Mang, Siegmund, and Blouin induce whiplash and consider the role of a startle response using electromyography (EMG) outcome measures. The muscle impact from facet joint dysfunction is evaluated by Reed, Pickar and Long. Pagé, Nougarou, Dugas and Descarreaux consider the muscle response associated with mechanically delivered SMT in humans, while Cao and Pickar look at the muscle response from an animal model utilizing mechanically delivered SMT.

A distinction should be made that the mechanically delivered SMT was not from devices intended for immediate commercial use, but rather they are robotic laboratory-based systems that can deliver precise and consistent force, amplitude/depth of thrust and duration of thrust. Consistency of the characteristics of the thrust is essential in order to isolate the variability of the findings to that of the body's response to the intervention. An alternate approach to the ones mentioned in this issue would be to have a mechanical model, upon which a clinician manually thrusts, so the only variability measured is that of the clinician. If the thrust were manually delivered to an animal or human patient, or if the instrument was a "hand held" device, the approach would measure the combined variability of the thrust delivery coupled with the variability of the response of the body. A method to tease out the significance of that combined variability in a non-thrust style of spinal manipulation was utilized by Gudavalli and Cox. They compared the force output of experienced versus novice performers, which is a both recruitment and testing approach seen frequently in motor learning literature. Using "real time" or concurrent feedback, another approach utilized in motor skill learning, they comment on the factors that improve in the novice performer.

The present issue of this journal may serve as a tool for learning. The narrative review articles may stimulate individual practitioners, educational institutions, or researchers to consider different therapeutic or measurement approaches. In his review, Bruno clarifies issues of contention related to stabilization exercises, presents strategies to identify patients most likely to respond to interventions, and presents protocols for clinicians or educators to consider. Passmore, Murphy and Lee present the rationale and formula for employing a neurophysiological technique demonstrated to measure changes associated with chiropractic intervention.

This issue acknowledges that altered body mechanics, beyond muscular changes, can also impact the autonomic nervous system and sensory processing. Whiplash is a rapid flexion-extension event explored by Mang, Siegmund, and Blouin that occurs in less than seconds. In their study the authors investigated the recruitment of axial and appendicular muscles along with autonomic responses and showed that responses to whiplash-like events involves both a descending recruitment pattern of axial and appendicular muscles and increased sympathetic responses. Enix, Scali and Pontell describe the anatomical relationship of musculature and the spinal cord. The spinal distortion caused by scoliosis and observed changes in body sway and impaired sensory processing was identified by Pialasse and Simoneau to be worthy of further investigation. Perhaps that investigation of scoliosis in the future could utilize the technique of measuring sensory changes

using somatosensory evoked potentials described in the review by Passmore, Murphy and Lee.

It is our hope that the contents of this issue are used as a resource for education, and as a catalyst to inspire future research in spine neuromuscular control. Such scientific exploration may further facilitate our understanding of chiropractic intervention, its mechanisms, and the potential ailments that may respond to care.

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Whiplash evokes descending muscle recruitment and sympathetic responses characteristic of startle

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Whiplash injuries are the most common injuries following rear-end collisions. During a rear-end collision, the human muscle response consists of both a postural and a startle response that may exacerbate injury. However, most previous studies only assessed the presence of startle using data collected from the neck muscles and head/neck kinematics. The startle response also evokes a descending pattern of muscle recruitment and changes in autonomic activity. Here we examined the recruitment of axial and appendicular muscles along with autonomic responses to confirm whether these other features of a startle response were present during the first exposure to a whiplash perturbation. Ten subjects experienced a single whiplash perturbation while recording electromyography, electrocardiogram,

Le coup de fouet cervical est la blessure la plus fréquemment subie à la suite d'une collision arrière. Durant une telle collision, la réponse musculaire humaine comporte à la fois une réaction posturale et une réaction de sursaut qui peuvent exacerber la blessure. Toutefois, la plupart des études antérieures ont seulement évalué la présence de la réaction de sursaut au moyen de données sur les muscles du cou et la cinématique de la tête et du cou. La réaction de sursaut évoque aussi un recrutement musculaire descendant et modifie l'activité du système nerveux autonome. Nous avons examiné dans le présent article le recrutement des muscles axiaux et appendiculaires ainsi que les réponses autonomes afin de confirmer si ces autres caractéristiques d'une réponse de sursaut étaient présentes au cours de la première exposition à une perturbation de type coup de fouet. Dix sujets ont subi une seule perturbation de ce type et leurs réponses électromyographique, électrocardiographique et électrodermographique ont été enregistrées. Tous

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and electrodermal responses. All subjects exhibited a descending pattern of muscle recruitment, and increasing heart rate and electrodermal responses following the collision. Our results provide further support that the startle response is a component of the response to whiplash collisions.

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KEY WORDS: whiplash, startle, perturbation, chiropractic

les sujets ont présenté un recrutement musculaire descendant, une augmentation du rythme cardiaque et des réponses électrodermales suivant la collision. Nos résultats soutiennent l'idée selon laquelle la réaction de sursaut est une composante de la réponse aux collisions avec coup de fouet.

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MOTS CLÉS : coup de fouet, sursaut, perturbation, chiropratique

Introduction

Whiplash injuries are the most common injury caused by motor vehicle collisions, and rear-end collisions pose the greatest risk of whiplash injury.[1] Although the exact aetiology of whiplash injuries remains unclear, a startle response elicited by a multisensory stimulus (somatosensory, acoustic and vestibular) is part of the neuromuscular response to a rear-end collision.[2-5] The startle response increases neck muscle activity and increased activity in the posterior neck muscles, in particular the cervical multifidus muscle, may exacerbate strains in posterior neck structures (i.e. cervical facet joint) while these neck structures are already strained by the collision-induced intervertebral motion.[4,6] Thus, it is important to understand how the startle response contributes to the neuromuscular response during rear-end collisions.

Previous whiplash experiments have focused primarily on neck muscle responses and head/neck kinematics as indicators of the presence of a startle response.[3,6-8] However, the startle response, which is found in all mammals[9], elicits a descending pattern of involuntary, axial and appendicular muscle recruitment originating from the caudal brainstem[10]. The startle response also influences autonomic physiological responses: it activates sympathetic post-ganglionic neurons that innervate organs such as the heart, blood vessels and sweat glands.[11-17] As a result, startle-induced sympathetic responses include an increase in heart rate and electrodermal activity (EDA), which is a technique used to infer sympathetic drive from measurable changes in skin conductance at the surface of the skin.[15,18]

Here we attempt to confirm that a startle response forms part of the neuromuscular response evoked during a rear-end collision. Specifically, we investigate the recruitment of axial and appendicular muscle responses and changes in autonomic responses as additional indicators of the presence of a startle response during a rear-end collision. If a startle forms part of the response to a whiplash collision, we expect a whiplash-like perturbation to evoke a descending recruitment of muscles and an increase in heart rate and electrodermal activity characteristic of a startle response.

Methods

Subjects

Ten subjects with no history of neurological disorders participated in this experiment (5M/5F, 27±8 years, 169±11 cm tall, 70±14 kg). All subjects provided written informed consent and were paid a nominal fee for participating. The research protocol was approved by the UBC Clinical Ethics Review Board (H07-01281) and conformed to the Declaration of Helsinki.

Instrumentation

Surface electromyography (EMG) electrodes (Ambu Blue Sensors: N type, Ballerup, Denmark) were placed unilaterally on muscles on the left side of the body: sternocleidomastoid (SCM), cervical paraspinal (PARA) at the C4 level, triceps brachii (TRI), first dorsal interosseous (FDI), erector spinae (ES) at the L4 level and rectus femoris (RF). Due to the multi-layered architecture of the posterior neck muscles, we use the term paraspinals muscles (PARA) to describe the total muscle activity

recorded at these electrodes. Reference electrodes were placed bilaterally on the acromion to satisfy the internal grounding requirements of the EMG recording system. EMG recording sites were shaved, cleaned with alcohol and lightly abraded with NuPrep gel (D.O. Weaver and Co., Aurora, CO, USA). All EMG signals were amplified using a Neurolog system (Digitimer, Welwyn Garden City, Hertfordshire, England, UK) at subject dependent gains (ranging from ×1000-5000) and analogue band-pass filtered from 10 to 1000 Hz.

Electrocardiography (ECG) was measured in a bipolar recording configuration with a pre-amplification device (Grass Technologies P55 A.C. Pre-Amp, West Warwick, RI, USA). Disposable surface electrodes (Ambu Blue Sensors: M type, Ballerup, Denmark) were placed on the right side of the chest just below the clavicle medial to the deltoid muscle, on the left side of the chest at the level of the 5th intercostal space on the mid-clavicular line, and on the right side of the chest at the level of the 5th intercostal space adjacent to the mid-axillary line to act as reference. The ECG signals were amplified ×1000 and analogue band-pass filtered between 0.3 Hz – 100 Hz. Electrodermal activity (EDA) was recorded using a skin conductance module (Cambridge Electronic Design (CED) 2502, Cambridge, England, UK) and disposable surface electrodes were placed at the thenar and hypothenar eminences of the right hand. The EDA signals were passed through a second order low-pass analogue filter with a 10 Hz cut-off frequency to remove any high frequency noise in the recordings.

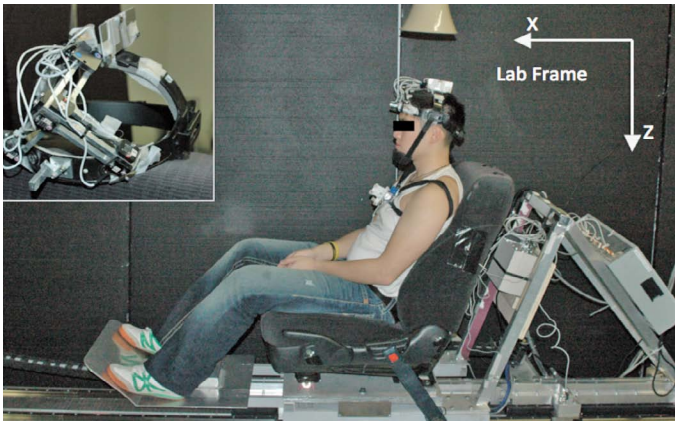

Figure 1.
Photographs of the experimental showing the location of the head and torso accelerometers, horn speaker and laboratory reference frame (X, Z). Inset: Close-up view of the nine accelerometer array on the headgear device. Note: electromyography (EMG) electrodes are not shown.

Kinematics of the head, torso and trunk were recorded with transducers to document the occupant responses and seat interaction. Head acceleration was measured using a nine accelerometer array (8 Kistler 8302B20S1; ±20g, Amherst, NY, USA. and 1 Silicon Design 2220-010; ±10g, Issaquah, WA, USA) arranged in a 3-2-2-2 configuration[19] and securely fastened to the subject's head. Upper torso acceleration was measured using a tri-axial linear accelerometer (Summit 34103A; ±7.5 g, Akron, OH) mounted to an aluminum plate that was securely fastened to the chest immediately below the sternal notch. Lower lumbar acceleration was measured using a uniaxial linear accelerometer (Silicon Design 2220-020; ±20g, Issaquah, WA, USA) fastened to the skin between the L5 and S1 spinal levels with the sensing axis orthogonal to the seatback/back interface. A motion capture system (Optotrak Certus, Northern Digital, Waterloo, ON, Canada) was used to measure head, torso and sled displacements. Twelve infrared (IRED) markers were affixed in groups of four to the head accelerometer array, torso chest plate, and car seat/sled platform. The location of the accelerometers and IRED markers were digitized relative to anatomical landmarks using Optotrak so that the kinematics could be transformed to anatomically relevant locations (i.e. atlanto-occipital joint and head centre of mass).[2,3,7] Sled acceleration was measured with a uni-axial accelerometer (Silicon Design Inc. 2220-100; ±100g. Issaquah, WA, USA). All accelerometer data were digitally low-pass filtered using a 4th order, dual-pass Butterworth filter with a cut-off frequency of 100 Hz.

EMG, ECG, EDA and accelerometer signals were sampled simultaneously at 2000 Hz using a National Instrument Data Acquisition (DAQ) PXI system and a custom Labview program, (National Instruments Corporation, Austin, Texas, USA). Optotrak data were acquired at 200 Hz per marker and the capture of each frame was triggered by the DAQ system to ensure synchronized data. For all trials, data were recorded for a total of 40 s: 10 s before and 30 s after the onset of sled acceleration.

Test Procedures

Subjects were seated on a feedback-controlled linear sled

fitted with the driver's seat of a 2005 Honda Accord (Figure 1). Subjects were instructed to sit comfortably facing forward, rest their forearms on their lap, and relax their head and neck muscles. The head restraint was removed from the top of the seat back to prevent head-to-head-restraint interaction that could affect the head/neck kinematics or generate additional sensory inputs. The sled generated no audible or mechanical pre-perturbation signals that could be used to predict the onset of a perturbation. The ambient background noise level in the lab was 64 dB. Each subject experienced a single forward horizontal translation with an average speed change of 75.00±0.03 cm/s, a peak acceleration of 19.5±0.2 m/s^2, and a duration of 53.20±0.05 ms. To closely replicate a real automotive collision, the onset of the acceleration matched the onset of a vehicle-to-vehicle collision with a speed change of 8 km/h (2.22 m/s;[20]) and was presented simultaneously with the sound recorded from of an actual 8-km/h vehicle-to-barrier crash (peak 109 dB, time-to-peak 34ms). To remain naïve to the experiment, subjects received neither practice nor demonstration trials of the perturbation.

Data Analysis

After data collection, all EMG data were digitally high-pass filtered using a 4th order, dual-pass Butterworth filter with a 30 Hz cut-off frequency to further remove any motion artifact. To determine the recruitment order of axial and appendicular muscle responses, we compared the onsets of activity in the different muscles recorded. EMG onset was defined as the time when the root-mean-squared (RMS) amplitude (20 ms window) reached 10% of its maximum value[8], and was then confirmed visually.

Instantaneous heart rate (IHR) was obtained from the R-R intervals on the ECG signals to detect changes in the beat-to-beat intervals during and following the whiplash perturbation. Baseline IHR and EDA were defined as the average value over 5 s immediately preceding each perturbation. Peak amplitude and time-to-peak for both IHR and EDA responses were determined as the first peak to occur within the 10 s period following the onset of the perturbation. The timeframe for IHR and EDA responses to return to baseline values were defined as the first instance IHR and EDA responses returned to their respective baseline values following the perturbation.

The head acceleration data were transformed from the head accelerometer array to the atlanto-occipital joint (AOJ) location and reported in the global reference frame (x-forward, y-right, z-down; for detailed procedures, see[6]). The AOJ was estimated to be 24 mm posterior and 37 mm inferior to the head's center of mass[21] and the head's center of mass was estimated to lie in the mid-sagittal plane, rostral to the inter-aural axis by 17% of the distance between the interaural axis and the vertex[22]. All head and trunk accelerometers were corrected for the earth's gravitational field. The onsets of head (x- and z-axis), chest (x-axis), lower lumbar (x-axis), and sled accelerations (x-axis) were determined directly from the transformed accelerometer data using a finite difference algorithm (5 ms moving window at a threshold value of 2 times the maximum pre-perturbation baseline value)[8] and then confirmed visually. All data analyses were performed using MATLAB (The Mathworks, Natick, MA).

Statistical Analysis

Non-parametric statistics were used to determine the significance differences between muscle response onsets by ranking the recruitment order of axial and appendicular muscle responses. A Friedman rank sum test was first used to determine whether the recruitment of EMG responses was different between muscles. A paired Wilcoxon rank sum test was then used to determine individual differences between each pair of muscles. Similar non-parametric statistics were performed to determine the significant differences within the order of acceleration onsets. A Friedman rank sum test was used to determine whether the acceleration onsets were different between accelerometer locations and a paired Wilcoxon rank sum test further determined individual differences between each pair of accelerometer locations. Autonomic responses were analyzed with a parametric paired-sample Student's T-Tests to compare pre-perturbation baseline IHR and EDA responses to the respective peak response occurring within the first 10 seconds following sled perturbation. All statistical analyses were performed using MATLAB at a significance level of $p = 0.05$.

Results

All subjects exhibited well-defined, axial and appendicular muscle responses elicited by the sled perturbations (Figure 2a). The onset of acceleration propagated upward from the sled (x-axis: defined as time zero) to the lumbar spine (x-axis: 15.1±2.9 ms), upper torso (x-axis: 25.6±2.3

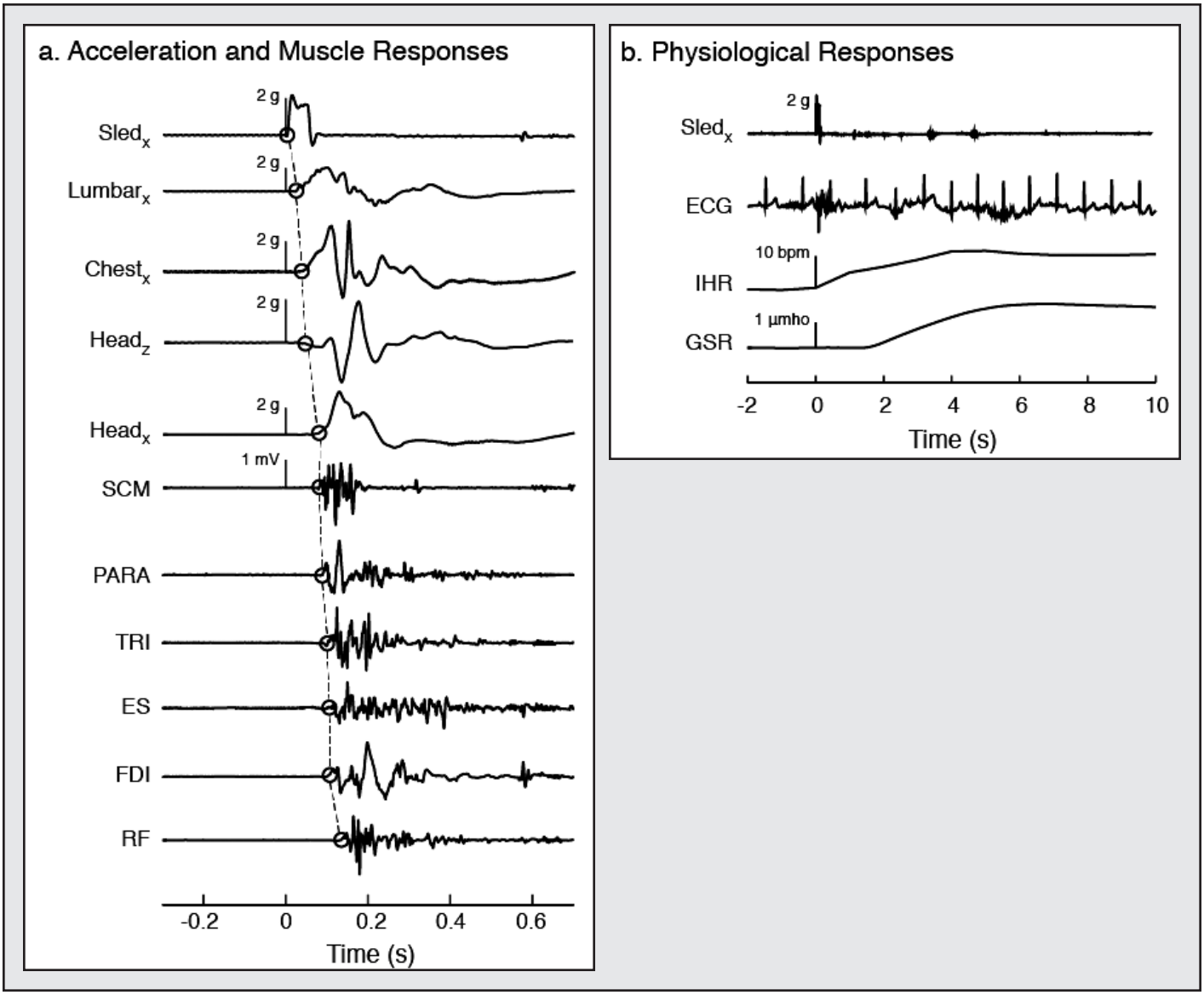

Figure 2.

A sample of kinematic, muscular and autonomic responses from a single subject during the first exposure to a whiplash perturbation. Due to the different timing of responses, kinematic and muscular data have been grouped in panel a., and autonomic responses in panel b. Hollow circles and dotted lines represent the onsets of accelerations and muscle responses to illustrate the propagation order of accelerations and the recruitment order of axial and appendicular muscles, respectively. The vertical scale bars are aligned with the onset of the sled perturbation and are consistent between trials. Kinematic data: subscript x and z refers to the x- and z-directions, respectively, for sled, lumbar, trunk and head accelerations. Electromyographic data: sternocleidomastoid (SCM), cervical paraspinal (PARA). triceps brachii (TRI), erector spinae at the level of L4 (ES), first dorsal interosseous (FDI) and rectus femoris (RF). Autonomic data: electrocardiogram (ECG), instantaneous heart rate (IHR) and electrodermal activity (EDA).

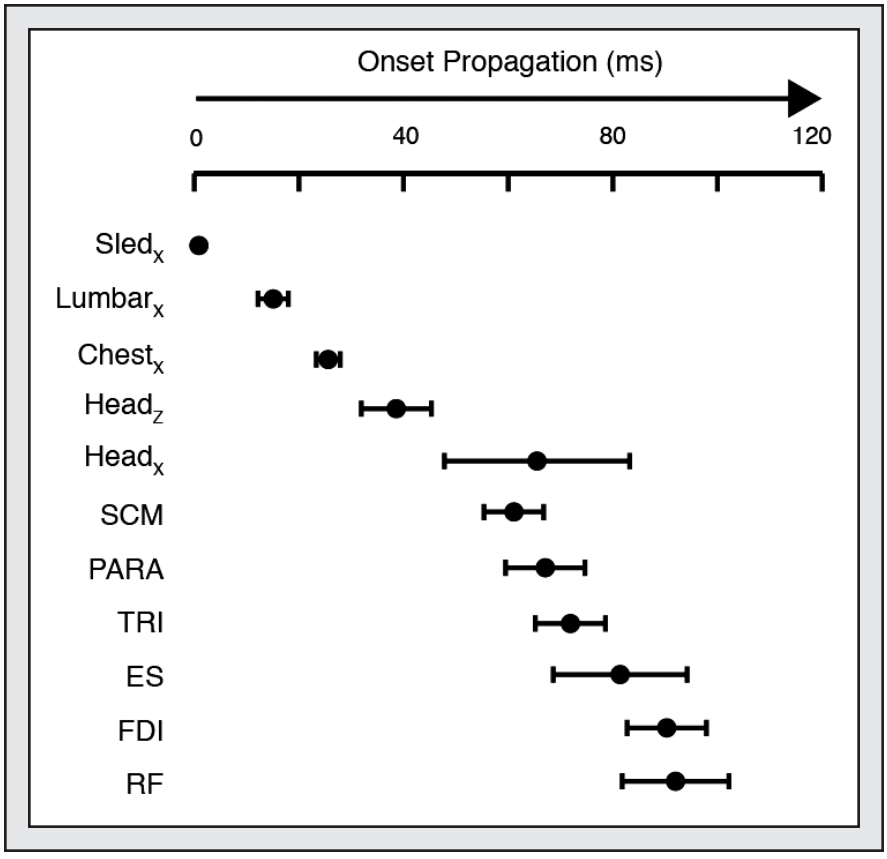

Figure 3.
Group mean and standard deviation for onsets of acceleration and muscle responses. Kinematic data: subscript x and z refers to the x- and z-directions, respectively, for sled, lumbar, trunk and head accelerations. Electromyographic data: sternocleidomastoid (SCM), cervical paraspinal (PARA), triceps brachii (TRI), erector spinae at the level of L4 (ES), first dorsal interosseous (FDI) and rectus femoris (RF).

ms) and then head (z-axis: 38.9±6.9 ms, x-axis: 65.5±17.7 ms) ($\chi^2(3)=30.00$, $p<0.0001$) (Figure 2a & Figure 3). Wilcoxon rank sum tests indicated a significantly earlier onset of sled acceleration than onsets of lumbar spine, upper torso and head (z-axis) accelerations (multiple p values<0.0001), earlier onset of lumbar spine accelerations than onsets of upper torso and head (z-axis) accelerations (multiple p values<0.0002), and earlier onset of upper torso accelerations than onset of head (z-axis) accelerations (p<0.0002), to establish an upward propagation of accelerations (Sled ⟶ Pelvis ⟶ Upper Torso ⟶ Head). In contrast to the upward propagation of acceleration onsets, we observed a downward recruitment of muscle onsets from the neck muscles to the appendicular muscles ($\chi^2(5)=43.08$, $p<0.0001$). Neck muscles were activated first (SCM: 61.1±5.7 ms & PARA: 67.1±7.6 ms) followed by TRI (71.9±6.7 ms), ES (81.4±12.8 ms), FDI (90.3±7.6 ms) and RF (92.0±10.2 ms). Wilcoxon rank sum tests indicated no difference between SCM and PARA onsets ($Z=-1.7047$, $p=0.0883$), but did show that SCM was active before TRI, ES, FDI, and RF (multiple p values<0.0028) and that PARA was active before ES, FDI, and RF (multiple p values<0.0058) though not TRI ($Z=-0.9085$, $p=0.3636$). Furthermore, TRI was active before FDI ($Z=-3.5920$, $p=0.0003$) and ES was active before RF ($Z=-2.1560$, $p=0.0311$). Thus, two descending muscle recruitment schemes were observed: 1.) SCM ⟶ TRI ⟶ FDI and 2.) SCM/PARA ⟶ ES ⟶ RF.

Concurrent sympathetic responses (IHR and EDA) were observed in all subjects following the simulated collision (Figure 2b). Baseline IHR ranged from 54 to 91 beats per minute (bpm) with an average IHR of 70±12 bpm. IHR increased by 14.3±5.7 bpm ($p<0.0001$) at 4.7±1.6 s after the onset of perturbation to 84±11 bpm. IHR returned to baseline levels in all subjects within 30 seconds following the collision. Baseline EDA values of -3.460±1.1 μmho increased by 2.08±1.1 μmho ($p=0.0002$) at 6.7±2.0 s after the onset of perturbation to an average

value of -1.405±1.7 μmho. In comparison to IHR, EDA did not return to baseline within the recording duration of the experimental trial (30s).

Discussion

The goal of this study was to confirm the presence of a startle response within the neuromuscular response to a rear-end collision using two indirect measures of the startle response: recruitment order of muscle responses and autonomic physiological responses. A single whiplash-like perturbation evoked a descending recruitment pattern of axial and appendicular muscles and increased sympathetic responses (IHR and EDA). These observations were consistent with responses evoked independently by an acoustic startling stimulus (muscle responses[10] and autonomic responses[11-17]) and provide further support that startle contributes to the overall response evoked during a rear-end collision.

Descending recruitment of muscle responses indicative of startle

A rear-end car collision is a complex, multi-sensory perturbation that stimulates the visual, vestibular, somatosensory, and auditory systems. Recent human volunteers studies involving seated transient perturbations have suggested that the startle reflex forms part of the neuromuscular response to a rear-end collision.[2,3,6,23] The startle response elicits a descending pattern of involuntary axial and appendicular muscle activity such as facial grimacing, abduction of the upper arms and bending of the knees.[10,24] From our study, we observed axial and appendicular muscle responses with a descending recruitment of muscle activations from neck muscles (SCM and PARA) to more distal axial muscles (ES) to appendicular muscles (FDI and RF). These results were similar to those elicited by the acoustic startle response and further support the presence of the startle responses.[10]

Alternatively, Forssberg and Hirschfeld (1994) proposed that somatosensory afferents derived from the backwards rotation and translation of the pelvis were responsible for triggering postural responses during sitting.[25] Somatosensory receptors located in both the trunk and the pelvis are the first detectors of the physical onset of a whiplash perturbation as we observed an ascending propagation of accelerations from the seat to the head ($lumbar_x$: 15ms, $torso_x$: 26ms, and $head_z$: 39ms). If the trunk and pelvis were indeed responsible for the triggering of the postural responses, one may expect segmental reflexes from the lumbar (ES muscle) to occur first through fast conducting monosynaptic stretch reflexes to maintain posture. These segmental reflex loops would then evoke an ascending recruitment of muscle activity along with the ascending propagation of accelerations. However, the current study observed two descending recruitment patterns of axial and appendicular muscles (SCM $\longrightarrow$ TRI $\longrightarrow$ FDI & SCM/PARA $\longrightarrow$ ES $\longrightarrow$ RF) despite an ascending propagation of accelerations. The observed downward recruitment of muscles responses further support the idea that startle reflex forms part of the neuromuscular responses to a rear-end collision.

Sympathetic responses indicative of startle

Sympathetic neural activity mediates the human body's fight-or-flight responses to maintain homeostasis following situations perceived as startling or dangerous.[26] Changes in instantaneous heart rate (IHR) and electrodermal activity (EDA) can be used to infer the body's regulation of this sympathetic drive during threatening situations. Following an unexpected rear-end collision, we observed an increase in sympathetic drive resulting in IHR and EDA increases of 14.1 bpm and 2.1 μmho, respectively. Similar increases in IHR and EDA were observed in volunteers who were driving on public roads and encountered a startling scenario involving an unexpected pedestrian crossing the road or a potential collision with another vehicle.[16] Moreover, a startling auditory (110 dB) stimulus has been shown to evoke an average IHR increase of 11 bpm in human volunteers lying in a supine position.[12] Thus, the sympathetic responses (within the first 10s) observed here support the presence of a startle response during a rear-end collision.

Implication for whiplash injury prevention

The cervical facet joints are a source of neck pain in 40-68% of patients with chronic whiplash injuries following a rear-end collisions.[27, 28] Excess strain can occur in the facet joint due to the intervertebral kinematic during the whiplash motion.[29] Due to their direct attachments onto the capsular ligaments[30], increased cervical multifidus, possibly related to the startle response, may further increase the capsular ligament strain and exacerbate injury[4,30,31]. The additional evidence of the startle response

observed here provides further support for investigating methods of reducing the startle response following low-speed rear-end collisions. If the startle component of the posterior neck muscle responses can be decreased, then the strain applied to posterior neck structures and the risk of whiplash injury may be reduced. We have previously shown that a loud (105 dB) pre-stimulus tone, presented 250 ms before the onset of impact, inhibits the startle component of the neuromuscular response evoked during a whiplash collision.[7] The pre-stimulus tone decreased the kinematics of the head (horizontal acceleration and angular acceleration in extension by 23%) and neck muscle responses (SCM by 16% and PARA by 29%). Thus, we suggest that startle responses should be addressed in the development of future anti-whiplash safety devices to reduce, and possibly prevent, the risk of whiplash injuries.

Our observations that a whiplash-evoked startle response elicits muscle activity throughout the body may have several clinical implications for the management of whiplash injuries. Although whiplash injuries remain primarily associated with neck pain (80%-100%), patients have also reported localized pain in the lumbar region (30%-60%) and extremities (12%-35%)[32-38]. In follow-up reports two years after the motor vehicle collisions, patients reported chronic pain in the lumbar region (6%-25%) and in the extremities (8%-17%).[32,34,35,37] The aetiology of the lumbar symptoms remains unclear, but the present findings imply that increased axial muscle activity can potentially lead to chronic low-back pain symptoms reported by patients with whiplash-associated disorders. It may be that increased activation of lower back muscles increases internal loads on lumbar structures by altering the kinematic and kinetic responses of the lumbar spine despite being supported by the car seat throughout the whiplash collision. Future in-vivo studies are needed to confirm this hypothesis and to characterize the kinematic and kinetic responses of the lumbar spine during whiplash collisions. Understanding the neuromechanics of whiplash injuries will ultimately lead to injury prevention, better management and improve the life quality of patients with whiplash-associated injuries.

The whiplash perturbation used in this study is less severe than many real-life whiplash injury-inducing collisions[39] and volunteer studies (higher speed changes: 4 to 16 km/h and peak accelerations: up to 6.0 g)[40-44]. However, startle responses have been shown to increase with stimulus intensity and rise time.[45] If the startle response is present in the neuromuscular response to the acceleration pulse used in this study, the startle response should increase as stimulus intensity increases. Nevertheless, further work is needed to confirm that our results are relevant at higher collision severities. Investigation into specific neurophysiological pathways responsible for triggering and modulating muscular and autonomic responses was outside the scope of this study. Thus, the exact nature of the sensory afferents triggering the startle reflex during rear-end collisions remains unanswered.

Conclusion:

This study provided further support that the startle response contributes to the neuromuscular response evoked during a rear-end collision. We observed a descending recruitment pattern of axial and appendicular muscles and increased sympathetic responses indicative of a startle response. Increasing our understanding of how the startle response contributes to the neuromuscular response during rear-end collisions will lead to the development of more effective anti-whiplash safety devices to reduce, and possibly prevent, the risk of whiplash injuries.

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The use of "stabilization exercises" to affect neuromuscular control in the lumbopelvic region: a narrative review

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It is well-established that the coordination of muscular activity in the lumbopelvic region is vital to the generation of mechanical spinal stability. Several models illustrating mechanisms by which dysfunctional neuromuscular control strategies may serve as a cause and/or effect of low back pain have been described in the literature. The term "core stability" is variously used by clinicians and researchers, and this variety has led to several rehabilitative approaches suggested to affect the neuromuscular control strategies of the lumbopelvic region (e.g. "stabilization exercise", "motor control exercise"). This narrative review will highlight: 1) the ongoing debate in the clinical and research communities regarding the terms "core stability" and "stabilization exercise", 2) the importance of sub-grouping in identifying those patients most likely to benefit from such

Il est bien établi que la coordination de l'activité musculaire dans la région lombo-pelvienne est vitale à la génération de la stabilité mécanique de la colonne vertébrale. Les ouvrages spécialisés fournissent la description de plusieurs modèles illustrant les mécanismes par lesquels les stratégies de contrôle neuromusculaire dysfonctionnelles peuvent être une cause ou un effet de la lombalgie. Le terme « stabilité du tronc » est employé de différentes manières par les cliniciens et les chercheurs, et ces variations dans l'emploi du terme ont mené à plusieurs approches en matière de réadaptation que l'on fait valoir comme affectant les stratégies de contrôle neuromusculaire de la région lombo-pelvienne (p. ex. « exercice de stabilisation », « exercice de contrôle moteur »). Cette revue narrative soulignera : 1) le débat continu dans les communautés clinique et de la recherche sur les termes « stabilité du tronc » et « exercice de stabilisation »; 2) l'importance du regroupement en sous-groupes lorsque l'on identifie les patients les plus susceptibles de bénéficier de telles interventions thérapeutiques;

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therapeutic interventions, and 3) two protocols that can assist clinicians in this process.

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KEY WORDS: stability, motor control, stabilization, exercise, chiropractic

3) deux protocoles qui peuvent aider les cliniciens dans ce processus.

(JCCA 2014;58(2):119-130)

MOTS CLÉS : stabilité, contrôle moteur, stabilisation, exercice, chiropratique

Introduction

Low back pain (LBP) is a significant public health problem and has been described as exhibiting epidemic proportions.[1] It has been estimated that 50-85% of the population will experience LBP at some point during their lives and that 10-30% of the population experiences LBP at any given moment.[2-3] LBP imposes a significant and increasing socioeconomic burden with estimated total costs comparable to those attributed to conditions such as heart disease and diabetes,[4-7] and results from the Global Burden of Disease Study 2010 indicate that it is now the leading contributor to global disability.[8] Importantly, it is the 5-10% of LBP cases that become chronic which account for a majority of the total costs attributed to the condition.[6,9,10]

Due to these high costs, investigating the most effective means of diagnosing and treating chronic LBP is a vital area of interest for health care authorities. To this end, international guidelines regarding the management of chronic LBP have been established.[11,12] These guidelines are consistent in recommending "exercise therapy" for patients with chronic LBP, and recent reviews support the effectiveness[13] and cost-effectiveness[14] of this approach. Despite the abundance of support for the use of exercise therapy for chronic LBP patients, there is much debate in the literature with regards to optimal exercise prescription. A large variety of exercise modalities have received attention in both the clinical and research literature over the years, including aerobic exercise, directional preference based (McKenzie) exercise, strengthening and/or endurance training of the abdominal/lumbopelvic musculature, and various forms of "stabilization exercise" (see next section). The evidence to date suggests that such exercise modalities are generally more effective than usual care in the treatment of chronic LBP.[13] However, there is currently no evidence to support the use of one exercise approach over another since the relative effectiveness of different approaches has been shown to be generally comparable.[12-13] Recently, it has been suggested that sub-groups of patients with LBP may respond differently to the various types of exercises that are used in clinical practice.[13]

Due to the multi-dimensional nature of LBP, the classification of the inherently heterogeneous LBP population into homogeneous sub-groups who are more likely to respond to a specific treatment approach based on factors in their history and physical examination has been advocated[15-18] and viewed as a research priority in the field for over a decade.[19-22] Attempts to establish specific causative factors or mechanisms of action associated with a particular patient's LBP would allow for more targeted treatments, which in turn will allow health care resources to be used more efficiently.[16,18,20,21] Chiropractors are well-placed to be leaders in both the development (research) and implementation (clinical practice) of such approaches to the management of LBP. Being able to recognize those individuals who are more likely to benefit from active care strategies, and implement targeted strategies that are individualized to each unique presentation, would have obvious benefits for our patients; it would also serve to expand our profession's position in the wider health care community.

"Core stability" and "stabilization exercise"

It is well-established that the coordination of muscle activity around the lumbopelvic region is vital to the generation of mechanical spinal stability.[23,24] Models illustrating mechanisms by which altered motor control strategies in this region serve as a potential cause and/or effect of LBP have been described by Panjabi[25-27] and others.[28-31] Panjabi[25] described three inter-coordinated subsystems that collectively are responsible for adapting to the stability requirements of the spine during various postures and

movements: a passive subsystem (e.g. vertebrae, intervertebral discs, ligaments), an active subsystem (i.e. the muscles surrounding the spinal column), and a neural control subsystem. Dysfunctional neuromuscular control strategies (e.g. muscle activation levels, coordination of muscle contractions) could therefore result in "clinical instability", which has been defined as the loss of the ability of the spine to maintain its pattern of displacement under physiologic loads resulting in no initial or additional neurological deficit, no major deformity, and no incapacitating pain.[26,27]

In a seminal paper on the topic, Bergmark[32] described and categorized two systems of muscles in the lumbopelvic region that contribute to spinal stability: 1) a "local system" of muscles that have an origin or insertion directly on the vertebrae, and 2) a "global system" of muscles that transfer the load directly to the thoracic cage and pelvic girdle. The "local system" has generally come to include deep muscles such as the multifidus, transversus abdominis, diaphragm, and pelvic floor muscles; whilst the "global system" is generally described as constituting the large superficial muscles such as the erector spinae, rectus abdominis, internal and external obliques, quadratus lumborum, gluteus maximus, and latissimus dorsi.[28,32-35]

The term "core stability" is commonly used to refer to the ability of these "core" muscles to stabilize the lumbar spine and pelvic girdle during static postures and dynamic movements. A host of theories and "stabilization exercise" programs have been developed to train these muscles as a means of treating and/or preventing LBP. However, there is still much inconsistency and debate both in the clinical and research communities with regards to what constitutes "core stability" and a "stabilization exercise". Additionally, several recent rehabilitative approaches emphasize the re-training of functional movement patterns as part of a "stabilization exercise" program, rather than focusing efforts on the training specific muscles.[33,34,36,37]

Although chronic LBP patients demonstrate a variety of apparently dysfunctional neuromuscular control strategies,[38-49] many stabilization exercise programs focus primarily on the training of the deep (local) muscles, particularly multifidus and transversus abdominis.[50] Localized atrophy of the multifidus[51] and a delayed onset of transversus abdominis during movements of the upper limbs[39,41] and lower limbs[40] have been shown in samples of LBP patients. However, the small magnitude and inconsistency of these apparent delays has led some authors to challenge their clinical significance.[28,52] Regardless, exercises have been proposed to selectively target multifidus and transversus abdominis.[50] Although there is some evidence that such exercises are able to change the recruitment of these muscles,[53-60] these findings are not universal.[52,61]

In addition to the changes to the multifidus and transversus abdominis that seem to be associated with LBP, samples of LBP patients also demonstrate altered neuromuscular control strategies in the superficial (global) muscles.[38,43-45,49] As such, rather than attempting to selectively recruit the deep muscles, an alternative approach is to use an "abdominal brace" that involves the contraction of all abdominal and low back musculature during exercise protocols.[33] This type of contraction has been shown to increase spinal stability[62] and paraspinal stiffness[63] compared to exercises that selectively target the multifidus and transversus abdominis. Several authors therefore recommend directing stabilization exercise programs toward grooving motor patterns that enhance spinal stability through repetition rather than specifically targeting one or two muscles.[64-67]

Due to the ongoing debate and inconsistency in the literature, assessing the evidence related to the effectiveness of stabilization exercise in the treatment of LBP is problematic. Several systematic reviews[68-72] and meta-analyses[73,74] have been published on the effect of stabilization exercise programs that selectively target the multifidus and transversus abdominis. The findings are relatively consistent in suggesting that, for *chronic* LBP, such exercises are more effective in reducing pain and disability in the short, intermediate, and long term compared to no treatment, regular medical treatment, education, or general exercise. There is, however, some controversy regarding their relative effectiveness compared to other treatment interventions. Some reviews suggest that they are more effective in reducing pain and disability in the short and long term compared to spinal manipulation, mobilization, and conventional physical therapy programs,[70,73] whilst others suggest that they are equally effective.[68,69,71,72]

Many of the trials included in these reviews incorporate stabilization exercise programs that attempt to selectively target the multifidus and transversus abdominis in the initial phases, and gradually progress to complex postural and dynamic tasks that involve both the deep and superficial muscles. Ergo, some authors have questioned

whether the apparently beneficial clinical effects of the programs (i.e. reduced pain and disability) are due to the "re-training" of the deep muscles, the subsequent stages of the program that engage all trunk muscles, or a combination of both.[29,70,73] To date, there are no published clinical trials that have directly compared a program focused on selective activation of the deep muscles with one focused on the contraction of all abdominal and low back musculature.

Another fundamental question related to the mechanism of action of stabilization exercise is whether the apparent clinical benefits are in fact related to changes in neuromuscular control strategies.[70,73,75] Surprisingly, very few studies have measured both clinical variables and physiological variables to assess the degree to which changes in one may be associated with the other. In a recent clinical trial,[54] patients who underwent an 8-week stabilization exercise program showed greater post-intervention improvement in the recruitment of the transversus abdominis than those who performed general exercise or received spinal mobilization. There was also a significant, moderate correlation between improved recruitment of transversus abdominis and reduction in disability. In a recent case series of four patients with LBP,[36] each patient was given verbal or manual cues to alter motion and muscle activation strategies to reduce the pain felt during the performance of specific provocation tests/movements. The results demonstrated that patient-specific interventions were effective in reducing pain during the tests/movements that initially caused pain. There were also corresponding measurable changes in biomechanical variables calculated using kinematic, kinetic, and electromyographic data.

Sub-grouping: an important consideration

An important consideration that is receiving increasingly more attention in the literature is the heterogeneity of the patient samples in previously-conducted clinical trials investigating the effectiveness of stabilization exercise.[67,70,72,73,75-77] There is preliminary evidence that treatment targeted at specific LBP patient sub-groups is more effective than non-targeted treatment.[78-80] However, definitive conclusions regarding the size of such matched treatment effects cannot be made based on the current evidence in this area.[77]

Certain sub-groups of chronic LBP patients have been shown to possess specific dysfunctional neuromuscular control strategies that are not apparent when these sub-groups are pooled with other LBP patients.[81-84] It has therefore been suggested that stabilization exercise may be more effective in a select sub-group of LBP patients.[36,54,61,73,76] A recent systematic review[76] investigated the level of participant sub-grouping in randomized controlled trials investigating the effectiveness of manual/exercise therapy for patients with *chronic* LBP. As of December 2008 (the last month included in the review's literature search), only five trials that met the review's search criteria reported using a clinical protocol to sub-group participants. None of these trials involved the use of a stabilization exercise intervention treatment arm. In another systematic review[77] investigating the relative effectiveness of targeted vs. non-targeted manual/exercise therapy for patients with LBP, one trial involving *acute and sub-acute* LBP patients met the review's search criteria and involved a stabilization intervention treatment arm.[78] This trial used a classification system proposed by Delitto and colleagues[85] (discussed further in the next section) to classify study participants into three sub-groups, including one for whom stabilization exercise was recommended. Since these reviews, one small trial has been published[86] that utilized a clinical prediction rule proposed by Hicks and colleagues[87] (also discussed further in the next section) to identify patients with "lumbar segmental instability" for selective inclusion in the trial.

To improve the current state of evidence related to sub-group classifications for LBP interventions, it has been recommended that: 1) future clinical trials investigating specific interventions for LBP (including stabilization exercise) incorporate the use of reliable and valid clinical protocols to create homogeneous patient samples,[76,88] and 2) such protocols should be based on identifying the underlying mechanism(s) of action for the specific disorders under investigation.[76] To this end, future trials investigating the effectiveness of stabilization exercise need to include both clinical and physiological variables in order to answer three fundamental questions:[75]

- Are neuromuscular control deficits actually present in the trial participants who receive interventions designed to treat these deficits?
- Does the intervention achieve the intention of changing the neuromuscular control deficit?

Table 1:
Summary of two clinical protocols proposed to identify low back pain patients who are more likely to respond to stabilization exercise intervention

Classification system	Patient type	Clinical features of interest	Intervention	Definition of a positive outcome (treatment success)	Features of the system	Limitations of the current evidence base
Treatment-Based Classification[85]	Acute LBP patients	• History of frequent recurrent episodes of LBP precipitated by minimal perturbations • History of alternating sides of a lateral shift deformity (i.e. antalgic posture) • History of frequent spinal manipulation with short-term relief • History of trauma, pregnancy, or use of oral contraceptives • Pain relief with immobilization (e.g. external support, abdominal bracing) • Clinical signs of generalized ligamentous laxity • Clinical signs of "segmental instability" (e.g. presence of aberrant movement during lumbar ROM testing, positive posterior shear test)	Not specified	Not specified	Not specified	• Has only been applied in clinical trials involving samples of *acute* and *sub-acute* LBP patients • The number of criteria that must be present to categorize a patient as being more likely to respond to the intervention has not been specified • The intervention to apply to patients who are deemed more likely to respond has not been specified • The definition of a positive outcome that can be expected has not been specified
Clinical Prediction Rule[87]	Not specified	• Age < 40 years • Average SLR > 91° • Presence of aberrant movement during lumbar ROM testing • Positive prone instability test	Abdominal bracing in various positions; progression directed by a physical therapist (8 week program)	≥ 50% reduction in disability score (ODI)	If ≥ 3/4 variables are present,+LR: 4.0 (95% CI: 1.6-10.0) If 2/4 variables are present, +LR: 1.9 (95% CI: 1.2-2.9)	• Has only been applied in one small clinical trial involving a sample of *chronic* LBP patients • Has not undergone full validation or impact analysis testing

Abbreviations: *CI: confidence interval; FABQ: Fear Avoidance Belief Questionnaire; LBP: low back pain; ODI: Oswestry Disability Index; ROM: range of motion; SLR: straight leg raise; +LR: positive likelihood ratio.*

- Are improvements in clinical outcomes (e.g. pain and disability) related to changes in neuromuscular control deficits?

Identifying patients more likely to benefit from "stabilization exercise"

The previous section highlighted the need for future trials to incorporate the use of reliable and valid clinical protocols to identify patient sub-groups in their study design. Such protocols would also be of obvious benefit to clinicians to assist them in identifying patients who are more or less likely to benefit from stabilization exercise. Although methods to objectively quantify spinal stability have been proposed,[23,89] these methods involve the use of advanced technology and mathematical modeling that make them of limited use in a routine clinical setting. A handful of clinical protocols have been proposed for identifying LBP patients who are more likely to respond favourably to stabilization exercise. Although none has gained universal acceptance, a non-systematic review of the literature revealed two protocols that have been cited in several recent systematic reviews on the topic.[77,90-93] Table 1 describes the features of these two protocols, and a summary of the evidential support for their clinical use

Table 2:
Operational definitions of the testing procedures described for use in the two clinical protocols proposed to identify low back pain patients who are more likely to respond to stabilization exercise intervention

Classification system	Clinical procedure	Operational definition
Treatment-Based Classification[85]	Clinical signs of generalized ligamentous laxity	Beighton score ≥ 4/9: One point is assigned for the ability to perform each of the following: 1) passive extension of the left fifth finger > 90°, 2) passive extension of the right fifth finger > 90°, 3) passive apposition of the left thumb to the flexor aspect of the forearm, 4) passive apposition of the right thumb to the flexor aspect of the forearm, 5) hyperextension of the left elbow > 10°, 6) hyperextension of the right elbow > 10°, 7) hyperextension of the left knee > 10°, 8) hyperextension of the right knee > 10°, 9) forward flexion of the trunk with the knees extended and the palms of the hands resting flat on the floor.
	Presence of aberrant movement during lumbar ROM testing	Aberrant movement: instability catch, painful arc of motion, Gower's sign, reversal of lumbopelvic rhythm
	Positive posterior sheer test	The patient is standing with arms across the lower abdomen. The examiner stands at one side of the patient and places one arm around the patient's abdomen, over the patient's crossed hands. The heel of the opposite hand is placed on the patient's pelvis for stabilization. The examiner produces a posterior force through the patient's abdomen and an anteriorly directed stabilizing force with the opposite hand. The test is repeated at all lumbar levels. A positive test is determined by the provocation of symptoms.
Clinical Prediction Rule[87]	SLR	The patient is supine. The inclinometer is positioned on the tibial crest just below the tibial tubercle. The leg is raised passively by the examiner, whose other hand maintains the knee in extension. The leg is raised slowly to the maximum tolerated straight leg raise (not the onset of pain).
	Presence of aberrant movement during lumbar ROM testing	Aberrant movement: instability catch, painful arc of motion, Gower's sign, reversal of lumbopelvic rhythm
	Prone instability test	The patient lies prone with the body on the examining table and legs over the edge and feet resting on the floor. While the patient rests in this position, the examiner applies posterior to anterior pressure to the lumbar spine. Any provocation of pain is reported. Then the patient lifts the legs off the floor (the patient may hold table to maintain position) and posterior compression is applied again to the lumbar spine. If pain is present in the resting position but subsides in the second position, the test is positive.

Abbreviations: *ROM: range of motion; SLR: straight leg raise.*

is provided below; please note that a formal critical appraisal process was not used to judge the quality or risk of bias of the original research papers that have been published related to the protocols. Table 2 summarizes the operational definitions of the clinical testing procedures described for the two protocols.

Treatment-based classification system

Nearly 20 years ago, Delitto and colleagues[85] described a "treatment-based classification approach" for *acute* LBP patients involving three levels of patient classification based on specific historic features and examination findings. The authors state that the development of this classification system was based on input from clinicians of various health care disciplines rather than from a formal derivation study. They also acknowledge that: "Although some of the tests and procedures discussed in this article have been subjected to peer-reviewed investigation, we would remind the reader that much of the decision-making rules that we propose have not been tested through prospective research."[85,p.471]

The classification system describes criteria that can be used to identify a sub-group of patients for whom stabilization exercise is recommended (the third level of classification). Importantly, in order to be placed into such a sub-group, a patient must first meet the following criteria: 1) he/she is deemed to have LBP that "can be managed independently and primarily by physical therapy" (the first level of classification), and 2) he/she is unable to stand for 15 minutes or more, sit for 30 minutes or more, or

walk for more than 0.4 km without worsening of pain (the second level of classification). In such cases, the following criteria are suggested to identify patients for a "stabilization exercise" sub-group:

- History of frequent recurrent episodes of LBP precipitated by minimal perturbations
- History of alternating sides of a lateral shift deformity (i.e. antalgic posture)
- History of frequent spinal manipulation with short-term relief
- History of trauma, pregnancy, or use of oral contraceptives
- Pain relief with immobilization (e.g. external support, abdominal bracing)
- Clinical signs of generalized ligamentous laxity
- Clinical signs of "segmental instability" (e.g. presence of aberrant movement during lumbar range of motion testing, positive posterior shear test)

The authors did not state a specific number of criteria that must be present to determine inclusion in this sub-group, nor a specific type of stabilization exercise program to prescribe for such patients.

The inter-rater reliability of classification assignment by physical therapists experienced with using the system has been found to be moderate[94] in two studies.[95,96] In addition, the results of another study provides preliminary evidence regarding the construct validity of the classification system.[97] A handful of clinical trials have evaluated the effectiveness of providing treatment based on this classification system. In one trial,[98] *acute* LBP patients were randomized to receive classification-based treatment or guideline-based treatment. The type of exercise performed by the patients in the stabilization exercise sub-group was not specified. The results indicated that improvement in clinical outcomes (e.g. disability, quality of life) was significantly greater after 4 weeks in the patients who received classification-based treatment. In another trial,[78] *acute and sub-acute* LBP patients were randomized to receive spinal manipulation, stabilization exercises (involving abdominal bracing and strengthening of the abdominal and lumbar musculature), or directional preference exercises. Clinical data collected at baseline were used to determine a classification for each patient, and comparisons were made between patients who received treatment matched to their sub-group classification and those who did not receive matched treatment. The results demonstrated that patients who received matched treatment had significantly less disability post-intervention (4 weeks) and at a 1-year follow-up. Unfortunately, the specific treatment effects for the stabilization sub-groups in both of these trials were not reported. As well, the magnitude of the overall matched treatment effect reported by Brennan and colleagues[78] has been called into question by the authors of a recent systematic review.[77] These authors stress the importance of distinguishing between *prognostic factors* (i.e. signs and symptoms that indicate a likely outcome regardless of treatment) and *treatment modifiers* (i.e. signs and symptoms that indicate a likely response to a specific treatment) when analyzing classification systems or clinical prediction rules. Using such methods, the results of this review demonstrated that although the classification system was able to identify individuals who were more likely to respond to a matched treatment, the actual treatment modifier effect size was not statistically significant.

Clinical prediction rule

More recently, Hicks and colleagues[87] published the results of a clinical prediction rule derivation study that explored the predictive value of various demographic, historic, and clinical examination variables for predicting outcome following a stabilization exercise program consisting of abdominal bracing in various positions. Four variables were found to be significantly related to treatment success (defined as ≥ 50% reduction in disability score): age < 40 years, average straight leg raise > 91°, the presence of aberrant movement during lumbar range of motion testing, and a positive prone instability test. The best rule for predicting treatment success was the presence of ≥ 3/4 of the significant variables (positive LR: 4.0; 95% CI: 1.6-10.0).

Teyhan and colleagues[99] used this clinical prediction rule to selectively recruit a sub-group of LBP patients who demonstrated ≥2/4 of the significant variables predicting treatment success. When this sub-group was compared to a sample of healthy controls, the authors were able to create a multivariate model of kinematic variables (as measured by digital fluoroscopic video) that was able to distinguish group membership. It would be useful to

repeat this study comparing a sub-group of LBP patients predicted to succeed with stabilization exercise with a sub-group predicted to fail with such treatment. In addition to this study, the results of a recent clinical trial demonstrated that an 8-week stabilization exercise program (involving abdominal bracing and abdominal hollowing exercises) plus routine exercise was more effective than routine exercise alone in reducing pain and disability in a simlar sub-group of *chronic* LBP patients, both postinterention and at a 3 month follow-up.[86]

Importantly, although this clinical prediction rule has been supported to some extent by a construct validation study[99] and applied in one small clinical trial,[86] it has not undergone full validation or impact analysis testing.[100] Ergo, definite conclusions regarding the clinical utility of this rule cannot be made, and caution must be used when applying it in clinical practice.[90-93]

Limitations

It must be stressed that this is a narrative review, rather than an exhaustive systematic review of the topic. Narrative reviews are inherently subjective with several limitations (e.g. selection bias of the studies included). The classification system and clinical prediction rule described herein have received a moderate amount of attention by the research and clinical communities. However, there may be additional methods related to the identification of LBP patients who are more likely to respond to stabilization exercise that have been described variously in the literature, which have not been included in this review.

Future areas of research

There are several interesting avenues of research based on the current gaps in the literature related to the identification of LBP patients more likely to benefit from stabilization exercise. First, further work should explore the potential usefulness of factors or procedures other than those included as potential predictors in the previous clinical prediction rule derivation study.[87] For example, the active straight leg raise test score has been shown to be a significant predictor for recovery in females with pregnancy-related pelvic girdle pain.[101] It would therefore be useful to include this test as a potential predictor in future derivation studies, along with other clinical procedures used to assess the neuromuscular control strategies of LBP patients. Second, once clinical protocols (e.g. clinical prediction rules) have been derived, they need to undergo appropriate and adequate validation testing in clinical trials. Importantly, the patient population to which the rule is intended to be applied needs to be represented in the participant samples in such trials.

Summary

This narrative review has attempted to highlight the variety and debate in the literature regarding the terms "core stability" and "stabilization exercise". Several recommendations for future research in this area have also been presented.

A handful of methods have been described over the years that purport to identify sub-groups of LBP patients who would likely benefit from stabilization exercise. Each has some degree of evidential support; however, all require further study before they can be used with confidence in practice. One of the main limiters regarding the use of the Treatment-Based Classification System proposed by Delitto and colleagues[85] in practice is that the evidence supporting its use is based on studies conducted with *acute* LBP patients. This is somewhat disconcerting since current guidelines generally do not recommend exercise therapy for *acute* LBP patients.[11,12] The clinical prediction rule proposed by Hicks and colleagues[87] has some degree of evidential support, but still requires full validation and impact analysis testing.

Evidence-based health care requires clinicians to use the best *available* evidence to assist in their clinical decision making. It is suggested that the two clinical protocols described here *may* be used in clinical practice; however, clinicians need to be aware of the limitations of each based on the current evidence available, and accordingly be judicious and cautious in their application.

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Effect of bracing or surgical treatments on balance control in idiopathic scoliosis: three case studies

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Scoliosis is the most frequent spinal deformity among adolescents. In 80 % of cases, it is defined as idiopathic as no individual cause has been identified. However, several factors linked to Adolescent Idiopathic Scoliosis (AIS) have been identified and are under investigation. One of these factors is neurological dysfunction. Increase in body sway has been observed either during or following sensory manipulation in AIS patients. It is believed that impairment in sensory processing could be related to scoliosis onset. Impairment in sensory processing could induce a body schema distortion. The aim of this case series was to evaluate if conventional orthopaedic treatments could improve balance control thus implying a better body representation. Although, no strong conclusion can be drawn from a case series, results suggest that alteration in body representation should be investigated in future studies.

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KEY WORDS: scoliosis, adolescent, sensory impairment, chiropractic

La scoliose est la déformation de la colonne vertébrale la plus fréquente chez les adolescents. Dans 80 % des cas, on la définit comme idiopathique, puisqu'on n'a jamais déterminé de cause unique. Toutefois, plusieurs facteurs liés à la scoliose idiopathique de l'adolescent (SIA) ont été déterminés, et font actuellement l'objet d'études. L'un de ces facteurs est la dysfonction neurologique. Une augmentation du déséquilibre corporel a été observée durant ou après la manipulation sensorielle chez les patients atteints de SIA. On croit qu'un trouble du traitement sensoriel pourrait être lié à l'apparition de la scoliose. Un trouble du traitement sensoriel pourrait entraîner une distorsion du schéma postural. Le but de cette série d'études de cas était d'évaluer si les traitements orthopédiques classiques pouvaient améliorer le contrôle de l'équilibre, et ainsi améliorer la posture du corps. Même s'il est impossible de tirer des conclusions solides d'une série d'études de cas, les résultats suggèrent néanmoins que les modifications de la posture du corps devraient faire l'objet d'études ultérieures.

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MOTS CLÉS : scoliose, adolescent, trouble sensoriel, chiropratique

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All participants gave their informed consent according to the Laval University Biomedical Ethics Committee.
This project has been partly supported by *Fondation Cotrel de l'Institut de France*. JPP has been awarded a scholarship *from Fond de Recherche du Québec en Santé*, and has been supported by *Fondation de Recherche Chiropratique du Québec* and the European Chiropractic Union.

Introduction

Scoliosis is the most common spinal deformity among adolescents.[1] It can be congenital or have an early onset between birth and 3 years of age (infantile), develop between 2 and 10 (juvenile), or it even develops during adulthood as a degenerative scoliosis. Scoliosis takes place mostly during adolescence, the prevalence is approximately 2-3% in children ages 10 to 16 years, and is more frequent in females.[2,3] Scoliosis is characterized or classically defined as a lateral deviation of the spine, but in fact, it is a three-dimensional (3D) deformation inducing geometric and morphologic changes in trunk and rib cage.[4]

Etiology

Harrington[5] has suggested that over 50 pathologies generate a secondary scoliosis. Among these pathologies, various neuromuscular diseases such as anterior poliomyelitis with trunk paralysis, multiple sclerosis, but also malformations such as congenital hemi-vertebra cause secondary scoliosis. Nonetheless, 80% of scoliosis is still considered as idiopathic.[5] It is unlikely, however, that the etiopathogenesis of idiopathic scoliosis results from a unique factor. In contrast, it is believed that various factors are involved and interact with various genetic predisposing factors.[6,7] The current trend in scoliosis research is to detect biomarkers that could predict either spine deformation onset or progression risk.[6] The common factors that are being investigated could be aggregated into 6 groups: genetic, neurological, hormonal and metabolic, skeletal growth, biomechanical, environmental.[8] During the last decades, various studies have investigated whether AIS patients had perceptual or sensorimotor impairments. It has been reported that AIS patients have deficits in sensorimotor adaptation and balance control and perceptual impairments.[9]

Vestibular system and scoliosis

An efficient control of upright balance implies the detection of instability (i.e., its direction and amplitude) and the selection of appropriate motor commands to restore stability.[10,11] Therefore, these processes require accurate sensory systems, optimal sensory processing and sensorimotor transformation. Altering the quality of sensory information allows studying the ability of the brain to reweight the sensory signal and select the appropriate motor commands to ascertain proper balance control. Results from studies assessing balance control have demonstrated that AIS patients have poorer balance control than controls and manipulating the availability of visual information or the quality of lower limb sensory information increased their disequilibrium.[12-15] The role of ankle proprioception, for controlling balance, has been studied in AIS patients by co-vibrating the tendon of the ankle joint, which altered the sensory information, and led to greater instability of AIS patients than controls.[16] Furthermore, following a brief period of sensory deprivation it has been shown that reintegration of ankle proprioception, whether vision was available or not, led to larger variability of the CP velocity in AIS patients whereas the age-matched controls reduced their CP velocity variability.[17]

Another sensory system that is worth investigating as a potential factor for scoliosis onset is the vestibular apparatus.[18-20] For instance, the vestibular nuclei occupy a prominent position in the brainstem. Since the lateral vestibulospinal tract controls axial muscles[21], it is thought that alteration in the brainstem or the cortical network involved in sensorimotor transformation, during body growth (i.e., preadolescent and adolescent period) may translate into abnormal trunk muscles activation causing permanent spinal deformities.[19,22] It has been reported that AIS patients, when asked to judge the amplitude of the whole body rotation, underestimated the amplitude of the angular displacement to a greater extent than controls.[18] However, in this last study, the vestibulo-ocular reflex (VOR) gain (defined as eye speed divided by head speed) of the AIS patients was similar to controls. These latest results promote the suggestion that it is the cortical mechanisms performing the sensory processing and sensorimotor transformation rather than the brainstem that is malfunctioning in AIS patients.[23-25]

One way to assess sensorimotor transformation capability is to manipulate sensory information and quantify its effect on motor control. For instance, the role of vestibular information on upright balance control can be evaluated using bipolar binaural galvanic vestibular stimulation (GVS).[22,26-28] With the head in neutral position, GVS evokes body sway mainly along the frontal plane and the direction is toward the side of the anode.[29] By changing the polarity of the stimulation (i.e., anode on the right or left mastoid), body sway can be induced on the right or left. Using vestibular stimulation, abnormal vestibulomotor control has

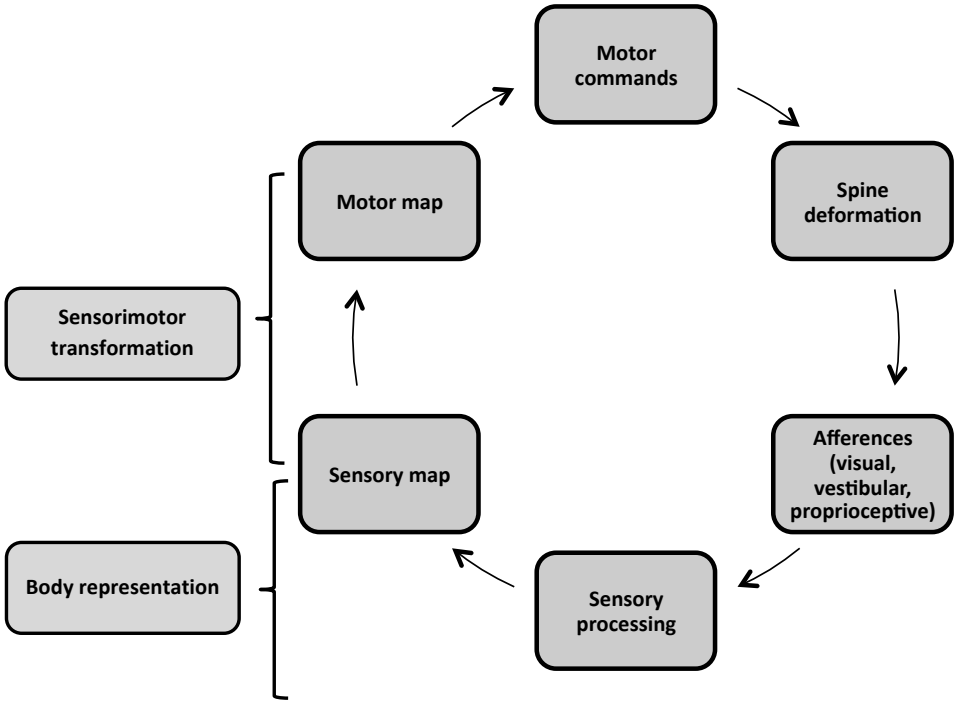

Figure 1:
Theoretical model of the association between a distorted body representation and the development of spine deformation. Alteration in the processing of sensory information could create a deformation of the body representation. Consenquently, the motor commands from the sensorimotor transformation process would be altered (e.g., asymmetrical). During a critical period of the development, this would create spine deformation. As a result, torso proprioception would be asymmetrical promoting body representation distortion.

been observed in AIS patients; compared to controls AIS patients demonstrated larger body sway either during or immediately after GVS cessation.[30]

It has been suggested that scoliosis could be related to a delay in the development or a distortion of the body schema.[9,31] Although attractive, this suggestion should be further investigated. Body schema refers to specific neural cortical networks holding an updated map of the body shape, dimension and posture. In other words, at the cortical level, the processing of the various sensory signals forms a sensory map of the body.[32] As an example, when using a tool to elongate the hand the brain needs to take into account the change in the body dynamics to ascertain proper movements.[33] In such a case, the body schema is updated; the participants perceive their arm as being longer.[34] Proprioception and vision are crucial for body schema updating, however, it has been recently suggested that vestibular information also contributes to body schema updating.[33,35-37] For instance, it has been demonstrated that vestibular stimulation enhances somatosensory input and even modulates visual processing.[36,38] Furthermore, it has been reported that patients with vestibular disorders might encounter distortions of their body schema.[37] Consequently, dysfunction in the mechanisms processing sensory information can cause asymmetry or a change in the amplitude of the vestibulomotor commands and alters the body schema. During rapid spine growth, this condition would lead to spine deformation and asymmetrical trunk proprioception promoting the updating of a distorted body schema (Fig. 1).[31,34,39]

The present study is part of a research programme assessing the vestibulomotor control of balance in AIS. The objective of the present study was to establish an experimental framework for testing whether spine deformation could be related to a distortion of the body schema. Since the body schema is continuously updated through sensory signals, it is possible that surgical intervention that drastically reduces spine deformation or bracing that creates proprioceptive rehabilitation, through torso proprioceptive cues, lead to a recalibration of the body schema. If this is the case, improvement in balance control either during or after sensory manipulation should be observed following spine surgery or long-term bracing. If this hypothesis is supported, it would indicate that the weight of proprioceptive information from the torso is larger than the weight of vestibular information (participants are tested in absence of vision) in the updating of body schema. An alternate hypothesis is that balance control improvement is caused by a decrease in the biomechanical forces acting on the spine due to a lessening of the spinal curvature. It has been demonstrated, however, that reintegration of sensory information altered balance in AIS patients which favours the first hypothesis.[17] In contrast, if body sway does not decrease following spine surgery or long-term bracing, it would suggest that the cortical mechanisms involved in sensorimotor transformation are impaired. In this case, although straightening the spine or bracing would improve torso proprioceptive cues, it would not be sufficient to recalibrate effectively the body schema.

Methods

Three participants were involved in this study. All of them gave their written informed consent according to Laval University biomedical ethics committee. Vestibular stimulations were delivered using a DS5 bipolar constant current stimulator (Digitimer Ltd, Garden City, UK). The skin behind the ears over the mastoid process was prepared bilaterally using electrode skin prep pad (Dynarex, Orangeburg NY, USA) before placing the PALS Platinum 3.2 cm electrodes (Axelgaard Manufacturing Co Ltd, Failbrook CA, USA). The electrodes were secured using 3M Transpore Tape 1527-1(3M). Participants performed the same tasks; they stood upright with their eyes closed and their feet 2 cm apart and with each foot standing on a force platform. Balance control was assessed using two force platforms (AMTI-model BP400600NC-1000, Watertown, MA, USA). The horizontal displacement of the torso along the frontal plane was evaluated using sensors (Polhemus – model Liberty 240/8, Colchester VT, USA) located at C7 and L5/S1. Because these measurements are influenced by either the height (i.e., L5/S1 and C7 displacement) or the weight (i.e., vertical force) of the participants, sensor horizontal displacement was normalized to participant's height and the vertical forces were normalized to participant's weight. For each trial, data acquisition started only when the participant's weight was evenly distributed according to the amplitude of each foot vertical force. Each trial was divided into four epochs. The first 2-seconds were used to assess baseline balance control prior to GVS (preGVS [2 0]). The following 2-seconds served to evaluate vestibulomotor control. A GVS of 1mA of amplitude and lasting 2 seconds was applied to assess vestibulomotor control (GVS [0 2]). For 15 trials, the anode was located on the left mastoid process (inducing a right to left body movement along the frontal plane) and for 15 trials the anode was located on the right mastoid process (inducing a left to right body movement along the frontal plane). The first second, following GVS, permitted to assess balance control during sensory reintegration ([2 3]) while the following 2-second was used to evaluate whether participants' balance control returned to baseline level (balance recovery [3 5]). The body sways of the two AIS patients were compared to normative data obtained from 15 age-related adolescents without spine deformities or neurological problems (control group – CTR). For the adult case, the control group is composed of 16 age-related young adults. AIS participants were evaluated twice; the second assessment occurred at least 12-month following the initial evaluation (hereafter, T0 and T1 are used to evoke the first and second evaluation). The same experimenter and the same material were used for both evaluations. From the force platform data, the Root Mean Square values (RMS) of the vertical forces were computed before vestibular stimulation (pre-GVS [-2 0] interval), during vestibular stimulation (GVS [0 2] interval), immediately after the cessation of the stimulation (sensory reintegration [2 3] interval), or later in time (balance recovery: post [3 5] interval). Normative data for the RMS vertical force value calculated in the two control groups are presented in Table 1.

Table 1:
Root mean square (RMS) values of the vertical force before (pre), during or after galvanic vestibular stimulation (GVS). These data are from a group of healthy adolescent (n=16) and a group of healthy young adult (n=15). Data are the means (standard deviation) of 15 trials per side.

	[-2 0] pre-GVS	**[0 2] GVS**	**[2 3] Sensory Reintegration**	**[3 5] Balance Recovery**
Adolescents Right /Left	0.27 (0.07) / 0.27 (0.07)	0.48 (0.11) / 0.51 (0.18)	0.64 (0.19) / 0.64 (0.20)	0.52 (0.15) / 0.56 (0.16)
Young adults Right / Left	0.23 (0.11) / 0.24 (0.10)	0.45 (0.16) / 0.45 (0.13)	0.61 (0.22) / 0.66 (0.24)	0.46 (0.17) / 0.45 (0.12)

Case 1: Effect of spine surgery on balance control

This case concerns a 17-year-old male. He was 14 years old when he first saw his orthopedic surgeon. The assessment of his balance control was performed when he was 15 year old. There were 3 other known cases of scoliosis in his family: his 2-year younger sister (mild scoliosis, Cobb angle = 20°), his mother (unknown Cobb angle), and his mother's sister (she probably had a severe spine deformation since she had had corrective spinal surgery). At the initial balance control assessment (T0), his Risser sign was 1 (i.e., index of osseous maturity based on iliac crest ossification, ranging from 0 to 5) and he had a 52° right thoracic curve and a 34° left lumbar curve. At the age of 16, he underwent surgery. Pre-surgery neurological routine examination did not report any findings. Motor conductance was normal in both lower limbs, sensory conductance was difficult to obtain on the right side but lumbar spine MRI was normal. The surgery consisted of reducing the curves and vertebrae rotations using transpedicular screws from the third thoracic to third lumbar vertebrae and two Harrington rods. Following the surgery (T1), 18-months later, he had an 18° right thoracic curve and a 14° left lumbar curve. His Risser sign was 5. Because spine deformation and surgical instrumentation necessarily constrained trunk mobility, the participant's trunk maximal voluntary range of motion along the frontal plane was quantified using the sensors located on the 5th lumbar vertebra (L5), and on the 7th cervical vertebra (C7). Right and left maximal voluntary trunk flexions were 30°and 38° before surgery (T0) and 23° and 27° following surgery (T1). Maximal torso deviations, due to vestibular stimulation, were smaller than his voluntary range of motion: 4° and 6° at T0 and 2° and 2° at T1 for right and left movements, respectively.

Before spine surgery, his balance instability was much larger than controls during and after vestibular stimulation; the vertical force RMS values were 2.4 times greater than controls during GVS ([0-2]) and 4.9 times immediately following GVS (i.e., sensory reintegration epoch, [2-3]) (Fig. 2). Following spine surgery (T1), however, his balance control slightly improved. For instance, his vertical force RMS values were both 1.3 times greater than controls for the GVS and sensory reintegration epochs, respectively. It is worth noting that, following spine surgery, his vertical force RMS values diverged slightly from controls during the GVS epoch mainly because the vertical force slightly increased toward the end of the interval whereas it leveled out for controls. Overall, for this AIS patient, it seems that the spine surgery improved his balance control.

Case 2: Effect of bracing on balance control

Case 2 is a 15-year-old girl and the sister of case 1. Her balance control assessments were performed the same day as her brother. At that time (T0), she was 13 when a 16° right thoracic curve and a 13° left lumbar curve

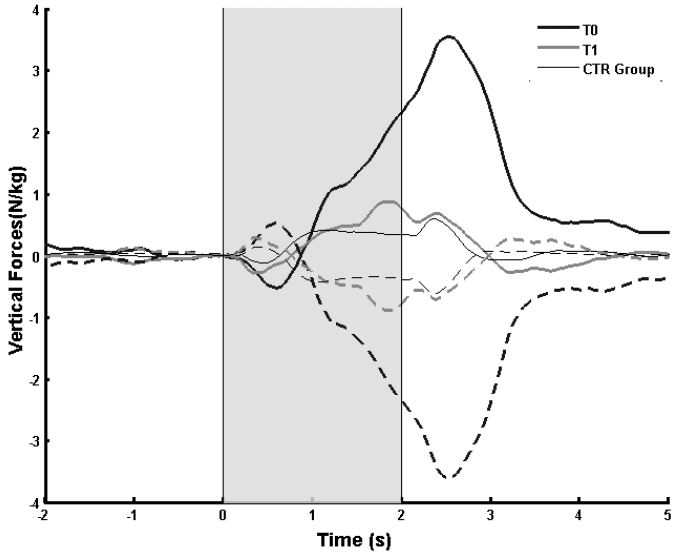

Figure 2:
Case 1 mean vertical forces from 2 seconds before GVS onset to 3 seconds after GVS cessation. GVS onset starts at 0-s and lasts 2-s (shaded area). Regular lines present data for the right stimulation whereas the dashed lines depict data for the left stimulation. The thin lines represent mean data for age-matched controls (CTR group) and thick lines illustrate the data of the AIS patients before (T0: thick gray lines) and after spine surgery (T1: thick light gray lines).

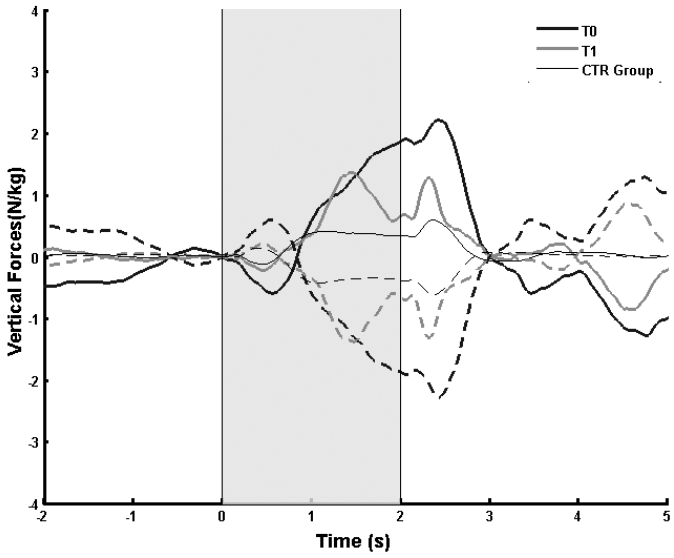

Figure 3:
Case 2 mean vertical forces from 2 seconds before GVS onset to 3 seconds after GVS cessation. GVS onset starts at 0-s and lasts 2-s (shaded area). Regular lines present data for the right stimulation whereas the dashed lines depict data for the left stimulation. The thin lines represent mean data for age-matched controls (CTR group) and thick lines illustrate the data of the AIS patients before (T0: thick gray lines) and 18-months after bracing (T1: thick light gray lines).

were detected. At the time, her Risser sign was 2. Before the first balance control evaluation, the patient had been wearing a Providence brace for 2 months and was still wearing it 18-months later (i.e., at T1). Bracing did not change much her spine deformation; she had a 17° right thoracic curve and a 23° left lumbar curve and her Risser sign was 4. At initial evaluation (T0), during the vestibular stimulation, her balance control was impaired compared to controls; the vertical force RMS value was 2.4 times larger (Fig. 3). Furthermore, her vertical force RMS value was 3 times larger than controls immediately following GVS (i.e., sensory reintegration interval, [2 3]) and she could not recover her balance to the same extent as the controls (balance recovery interval, [3 5]). Eighteen months later (T1), during GVS, her vertical force RMS value was 2.6 larger than control. Although it seems that the amplitude of her vertical force slightly decreased; her balance control was still impaired compared to controls. Immediately following the cessation of GVS (i.e., sensory reintegration interval), her vertical force RMS value was 2.6 times greater than controls. Finally, it is worth noting that compared to controls, she had trouble recovering her balance; the amplitude of her vertical forces did not reach a steady state. Overall, the present results suggest that long-term torso proprioceptive cue provided by the brace partly improved (but still larger than controls) balance control while her lumbar deformation increased by 10°. This latest result suggests that the amplitude of the spine deformation is not necessary related to balance control impairment.

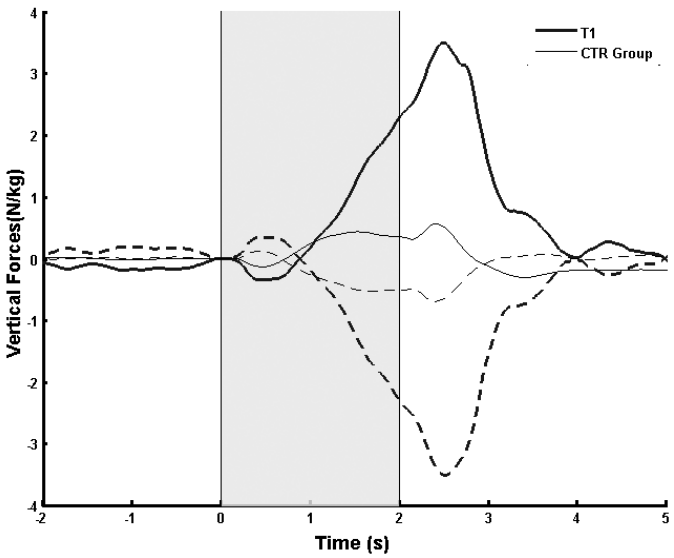

Figure 4:
Case 3 mean vertical forces from 2 seconds before GVS onset to 3 seconds after GVS cessation. GVS onset starts at 0-s and lasts 2-s (shaded area). Regular lines present data for the right stimulation whereas the dashed lines depict data for the left stimulation. The thin lines represent mean data for age-matched controls (CTR group) and thick lines illustrate the data of the AIS patients after spine surgery (T1: thick light gray lines).

Case 3: Effect of spine surgery in adult on balance control

This participant is a 20-year-old woman. There are two other known cases of scoliosis in her family: her grandmother and her older sister underwent spine surgery. Her scoliosis has been diagnosed when she was 11. Between the diagnosis and the surgery, she had been braced. A first surgery was performed when she was 14 and a second surgery when she was 16. Before the first surgery, she had a 70° right thoracic curve and a 55° left lumbar curve. The last assessment of her spine deformation revealed that she still had a 35° right thoracic curve and a 30° left lumbar curve. The balance control assessment was realized following both spine surgeries. The analysis of the vertical force time-series during GVS revealed that her balance control was worse than controls; her vertical force RMS value was 2.3 larger than controls (Fig. 4). Furthermore, immediately following vestibular stimulation (i.e., sensory reintegration epoch [2 3]), her balance control was still worse than controls; her vertical force RMS value was 4.5 times larger. Across time (i.e., balance recovery epoch, [3 5]), however, her vertical forces drastically decreased but her RMS value was still 1.9 larger than controls. Overall, it is concluded that despite the absence of a complete reduction in her spine deformation, compared to controls, the cortical mechanisms performing sensorimotor transformation are impaired.

Discussion

Visual, proprioceptive and vestibular information contribute to the perception of the body shape, dimension and relative limb position with respect to each other (body representation). Since it has long been reported that AIS patients have sensory processing impairments[16,17,19,40-43], it is plausible to suggest that AIS patients could have a distorted body representation. The aim of this study was to present an experimental framework to evaluate this suggestion. It was hypothesized that reducing spine deformation, through conventional treatment, should allow recalibrating body schema. As a result, reduction in spine deformation should translate into balance control improvement either during or following sensory manipulation.

Bracing or surgery effect

Results have demonstrated that for cases 1 and 2, either the spine surgery or bracing slightly improved balance control. For both cases, however, balance control was still impaired during or following vestibular stimulation. For these patients, altering the asymmetry in torso proprioception through spine surgery or providing torso proprioceptive cue via bracing partly improved balance control. The cortical mechanisms that update the body schema likely weight differently the sensory signals.[17,44] Consequently, for some patients, straightening the spine or wearing a brace could partly reduce body representation distortion. For these individuals, alteration in the sensorimotor transformation of vestibular information would not be completely eliminated by the torso proprioception. In conclusion, it is speculated that for these two cases, improvement in balance control during sensory deprivation or sensory reintegration implies a better body representation.

For case 3, the reduction in spine deformation, through two surgeries, did not reduce her balance sway to the same extent as controls either during or immediately following sensory manipulation. Nonetheless, it is worth mentioning that she still had a spine deformation post-surgery (i.e., 35° right thoracic curve and a 30° left lumbar curve). Therefore, one may suggest that balance control impairment was related to biomechanical factor. The increase in vertical force immediately following vestibular stimulation rule out this suggestion as performing sensory reintegration led to balance control impairment. As a result, it seems that asymmetrical torso proprioceptive information (i.e., distorted body representation) led to suboptimal sensorimotor transformation and inefficient balance control.

Treatment of AIS

The recommendation from the Scoliosis Research Society (SRS) indicates that for curves between 25° and 40° patients should be braced.[45-47] For these curve severities, surgical treatment is not necessary as long as the curve remains below 45° even if it progresses despite bracing. Surgical treatment is recommended for patients that are still growing with curve greater than 45°, or if the curve is larger than 45° and continues to progress even if growth has stopped. The purpose of surgical intervention is twofold: i) to prevent curve progression and ii) to reduce spine deformation. On the other hand, bracing only slows curve progression. Therefore, to be efficient, bracing must be prescribed as soon as possible. Bracing is considered an effective treatment with 72% of success (i.e., the curve did not worsen) compared to 42% after observation.[48] Furthermore, there is a significant positive association between hours of bracing and treatment success; 12.9 daily hours of bracing entails a success rate of 90 %.[48]

Limitations and research recommendation

Undoubtedly, scoliosis onset or progression involves multiple factors. Alteration in the processing of sensory information or in the mechanisms performing sensorimotor transformation could be related to a genetic defect, for example. Therefore, alterations in sensorimotor transformation, for example due to a distortion in body representation, might be related to scoliosis onset or progression in some patients. This case series propose a tentative experimental framework to explore whether a potential link between body representation and scoliosis exists. This study has various limitations. Obviously, to better test the experimental framework and draw any conclusion, more AIS patients need to be tested before and after spine surgery to thoroughly verify whether reduction in spine deformation translate into a better body representation. Because of its complex aetiology, it is proposed that grouping AIS patients based on the severity of the spine deformation could mix patients with various causes (e.g., genetic, neurological dysfunction, hormonal). Consequently, an approach based on detecting the prevalence of a biomarker (e.g., vestibular impairment) should be used.[49]

The motor response evoked by GVS is reliable in healthy individuals and individuals with vestibular pathology over weeks (personal communication with the authors).[50] Although in the present study balance control was studied after several months, we are confident that this period did not affect our results since the motor responses evoked by GVS are unaffected up to 60 years old.

Conclusion

Overall, the present results suggest that reducing spine deformation does not necessary translate in balance control improvement. The three cases demonstrated different behaviour following conventional treatment. For instance, spine surgery improved to a great extent balance control in case 1 either during or following sensory manipulation. In contrast, bracing had a slight effect for case 2 while her lumbar deformation increased by 10°. For case 3, reduction in spine deformation through surgeries did not translate in balance control similar to controls. The absence of clear-cut results supports the idea that AIS is a multifactorial pathology. Consequently, studying the effects of conventional treatment on balance control while manipulating sensory information (e.g., through GVS) could give some insights into the physiopathology of AIS patients with balance control impairment.

Acknowledgements

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The effect of spinal manipulation impulse duration on spine neuromechanical responses

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Introduction: *Spinal manipulation therapy (SMT) is characterized by specific kinetic and kinematic parameters that can be modulated. The purpose of this study is to investigate fundamental aspects of SMT dose-physiological response relation in humans by varying SMT impulse duration.*

Methods: *Twenty healthy adults were subjected to four different SMT force-time profiles delivered by a servo-controlled linear actuator motor and differing in their impulse duration. EMG responses of the left and right thoracic paraspinal muscles (T6 and T8 levels) and vertebral displacements of T7 and T8 were evaluated for all SMT phases.*

Results: ***S**ignificant differences in paraspinal EMG were observed during the "Thrust phase" and immediately after ("Post-SMT1") (all T8 ps < 0.01*

Introduction : *La manipulation vertébrale (MV) se caractérise par des paramètres cinétiques et cinématiques particuliers qui peuvent être modulés. L'objet de la présente étude est d'examiner des aspects fondamentaux de la relation dose-réponse physiologique de la MV chez des humaines en faisant varier la durée de l'impulsion de la MV.*

Méthodologie : *Vingt adultes en santé ont subi quatre différents profils force-temps de MV livrés au moyen d'un actuateur linéaire asservi et ayant des durées d'impulsion différentes. Les réponses EMG des muscles paravertébraux de gauche et de droite (au niveau des vertèbres T6 et T8) et les déplacements des vertèbres T7 et T8 ont été évalués pour toutes les phases de la MV.*

Résultats : *Des différences considérables ont été observées dans l'EMG des muscles paravertébraux au cours de la phase de la « poussée » et immédiatement après celle-ci (« post-MV1 ») (T8 : tous les p < 0,01 et T6 lors de la poussée : tous les p < 0,05). Les*

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and T6 during the thrust ps < 0.05). Sagittal vertebral displacements were similar across all conditions (p > 0.05).

Conclusion: *Decreasing SMT impulse duration leads to a linear increase in EMG response of thoracic paraspinal during and following the SMT thrust.*

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KEY WORDS: spine, manipulation, dose, impulse duration, chiropractic

déplacements sagittaux des vertèbres étaient semblables dans toutes les situations (p < 0,05).

Conclusion : *Une réduction de la durée de l'impulsion de la MV entraîne une augmentation linéaire de la réaction à l'EMG des muscles paravertébraux thoraciques au cours de la poussée de la MV, et après celle-ci.*

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MOTS CLÉS : colonne vertébrale, manipulation, dose, durée de l'impulsion, chiropratique

Introduction

Manual therapies are often used in the treatment of spinal conditions; they have been one of the most studied conservative treatment approaches for such conditions.[1-3] Recent systematic reviews show that manual therapies such as spinal manipulation and mobilization both have positive, but limited, short-term effects on pain and disability.[2,4] Failure to demonstrate larger clinical effects, such as the ones often described by clinicians, may partly be explained by the limited knowledge with regard to the mechanisms of action underlying manual therapies. Many scientists and clinicians have proposed that both spinal manipulation and mobilization exert biologic effects on the nervous system through mechanical deformation of musculoskeletal tissues[5-7], but actual data on human subjects remain sparse.

Spinal manipulative therapy (SMT) is usually defined as a dynamic thrust of high-velocity, low-amplitude applied at specific contact points over the spine.[7,8] Historically, spinal manipulation, otherwise known in the chiropractic profession as "adjustment", has been one of the defining elements of the chiropractic therapeutic approach.[9] Early conceptualisation of possible SMT biologic effects were based on the premises that biomechanical parameters play a critical role in the nature and amplitude of physiological responses.[10]

> *"Attention to the amount of force and speed used, the direction of the thrust, the recording of the places worked upon, all make for a fair amount of predictability that the same procedure followed again can give the same result".*
> *Verner 1941*

SMT is characterized by specific kinetic and kinematic parameters that vary according to the region where it is applied[11], the clinician's experience[12,13], and its method of application[14]. It has been suggested that the clinical effects of SMT are related to the modulation of these parameters, and our research group has undertaken a series of exploratory experiments aimed at evaluating biomechanical and neuromuscular responses to varying dosages of SMT parameters.[15] These studies have showed a clear dose-response relationship between forces[16] and preload forces (Conference abstract at ACC-RAC 2014) and paraspinal neuromuscular responses. However, impulse duration has not been investigated by our research group. The next section presents the current state of knowledge related to the effects of SMT impulse duration.

Specific effects of SMT impulse duration

Thoracic spine SMT are usually performed within an impulse duration (time-to-peak force) of 130 to 200ms[13,17], however, a wide range of impulse durations have been reported when SMT is performed by humans (30-250ms)[6]. Systematic modulation of biomechanical and physiological responses to varying levels of impulse durations have mostly been investigated in anaesthetised animals. Studies evaluating vertebral displacements or neurophysiological effects of different impulse durations or

velocities on cadavers, anesthetized or healthy humans reported few or no result regarding these parameters.[18-21] Studies on anaesthetised animals showed that varying impulse phase durations produces changes in the displacement and acceleration of the contacted and adjacent vertebras.[6] Shorter impulse durations produce larger adjacent and fewer contacted vertebral segment motions than longer impulse durations.[22] Moreover, recordings of physiological responses in animals showed that changing impulse durations evokes a variety of responses from afferents innervating muscle spindles and Golgi tendon organs. When peak force remains constant, the muscular activity amplitude increases with increasing impulse duration plateauing around 200ms.[22] Recent studies revealed that resting muscle spindle discharge is significantly modified by impulse duration in anaesthetized cats, when thrust displacement or thrust force amplitude are unchanged. Muscle spindle responses to increasing speed (shorter impulse duration) are characterized by a curvilinear increase in discharge frequency[23] with the steepest increase occurring at an impulse duration of 100 ms or shorter[24,25]. Overall, these results suggest a possible impulse duration threshold for which spindle responses are specifically and significantly increased under mechanical deformation of the spine. The SMT impulse duration dose-response relationship, however, remains to be investigated in humans.

The purpose of the present study is, therefore, to investigate fundamental aspects of SMT dose-physiological response relation in humans by investigating how different SMT impulse durations could modify biomechanical and neuromuscular responses to spinal manipulation.

Methods

A total of twenty healthy participants aged between 20 and 35 years old were recruited (10 female and 10 male with mean ± standard deviation age and body mass index of 23.75 ± 3.29 years and 23.43 ± 2.58 kg/m^2). A general "screening" was performed by an experienced chiropractor in order to rule out any contraindication to SMT. Participants were excluded if they presented thoracic or lumbar pain, previous history of back trauma surgery, severe osteoarthritis, inflammatory arthritis, vascular problems, or any other condition that would limit the usage of SMT. Once included in the study, all participants gave their informed written consent according to the University's Human Research Ethics Committee certification (No. CER-12-181-06.37).

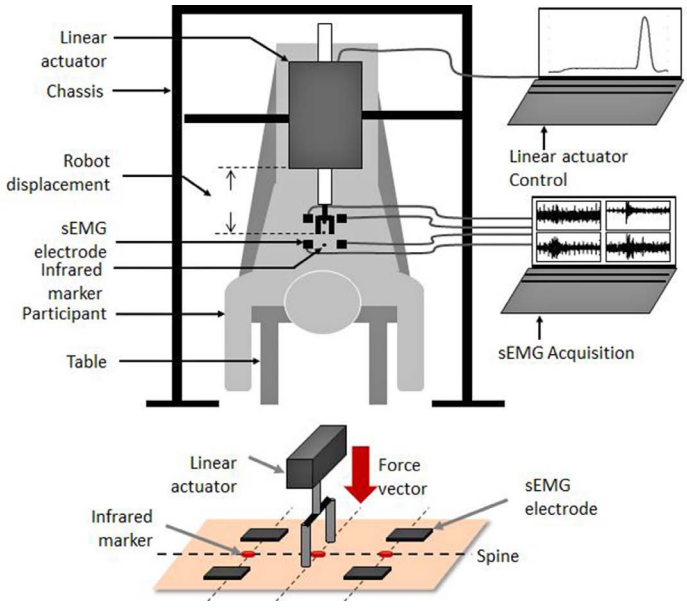

Figure 1:
Illustration of the experimental set up and the main components of the servo-controlled linear actuator motor. Surface EMG (sEMG).

Experimental protocol:

Each participant was first shown a demonstration of a simulated spinal manipulation performed by a servo-controlled linear actuator motor on a rigid body, in order to explain and highlight the basic operating and main security features of the apparatus. Electromyography (EMG) electrodes were applied over the left and right thoracic paraspinal muscles (T6 and T8 levels) following fiber orientation, and kinematic was collected by positioning light-emitting diodes on the spinous processes (T7 and T8 levels). The experimental set up is illustrated in figure 1. Each participant lied down in a prone position on a chiropractic table and was subjected to four different SMT force-time profiles. These four simulated SMT curves consisted of a 20N preload force for 1000ms followed by a "Thrust phase" composed by an "Impulse phase" leading to a peak force of 255N[16] and a "Resolution phase". The four SMT force-time profiles differed in their impulse phase duration respectively set to 125ms, 175ms, 225ms,

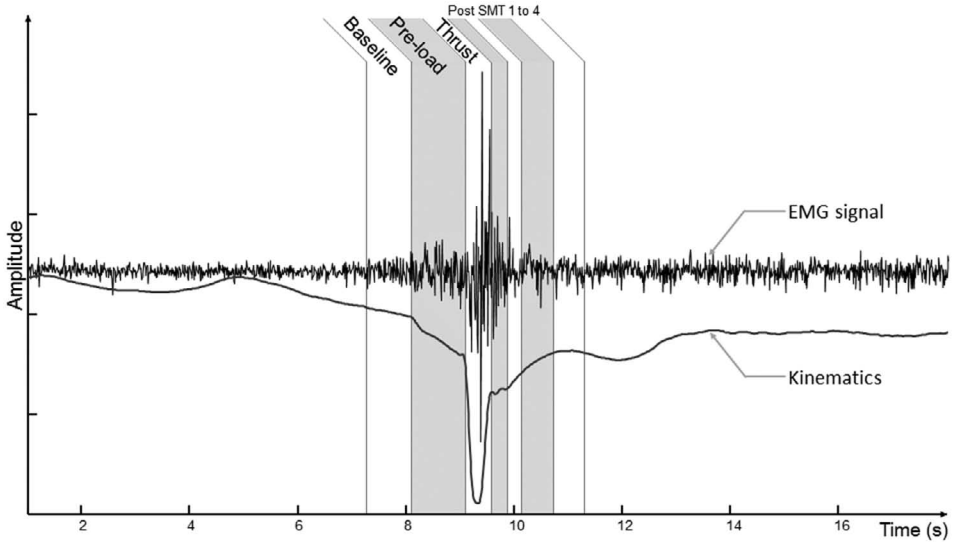

Figure 2:
Typical EMG and kinematic responses throughout the various SMT time-windows defined in the methods section.

and 275ms. Resolution phase duration was identical to impulse phase duration. A 20N preload force was chosen to limit the potential physiological responses related to preload forces. A typical SMT force-time profile is illustrated in figure 2. Five minutes of rest were given between each of the four trials, and the various impulse duration conditions were randomized across participants to avoid any sequence effect.

Apparatus:

EMG activity was recorded using a Delsys Surface EMG sensor with a common mode rejection ratio of 92dB at 60Hz and input impedance of 1015Ω (Model DE2.1, Delsys Inc., Boston, MA, USA). Interelectrode distance was fixed at 20mm, and electrode diameter was 10mm. Electrodes were applied over the thoracic paraspinal muscles on each side of the spine, approximately 2cm from the T6 and T8 spinous processes. The reference electrode was positioned on the left acromion of each participant. For each electrode, (1) the desired body part (region) was gently shaved, (2) the skin was gently abraded with fine-grade sandpaper (Red Dot Trace Prep, 3 M, St. Paul, MN, USA) and (3) the skin was wiped with alcohol swabs. These three steps were systematically done for each electrode and each participant in order to reduce skin impedance. Data were sampled at 1,000Hz with a 12-bit A/D converter (PCI 6024E, National Instruments, Austin, TX, USA). The data were collected by LabView (National Instruments, Austin, TX, USA) and processed by Matlab (MathWorks, Natick, MA, USA). A motion analysis system (Optotrak Certus; Northern Digital, Waterloo, Ontario, Canada) was used to perform the kinematic data acquisition. Kinematic markers were placed on T7 and T8 spinous processes and data were collected at 100Hz.

A servo-controlled linear actuator motor (Linear Motor Series P01-48x360, LinMot Inc., Zurich, Switzerland) was developed and used to precisely simulate SMT for the four different impulse duration conditions. The linear motor vertically displaced a slider applied directly to the spine. A twin tip padded rod (14mm of diameter and 36mm inter-rod distance), was used as the contact point between the servo-controlled linear actuator motor and transverse processes of T7. A microcontroller accurately controlled the linear motor in order to reproduce a target SMT force-time profile loaded from a computer. A close loop force control constantly provided the needed intensity to maintain the output force as close as possible to the target force-time profile. A complete technical description and details of the safety features are presented in a previous article.[15]

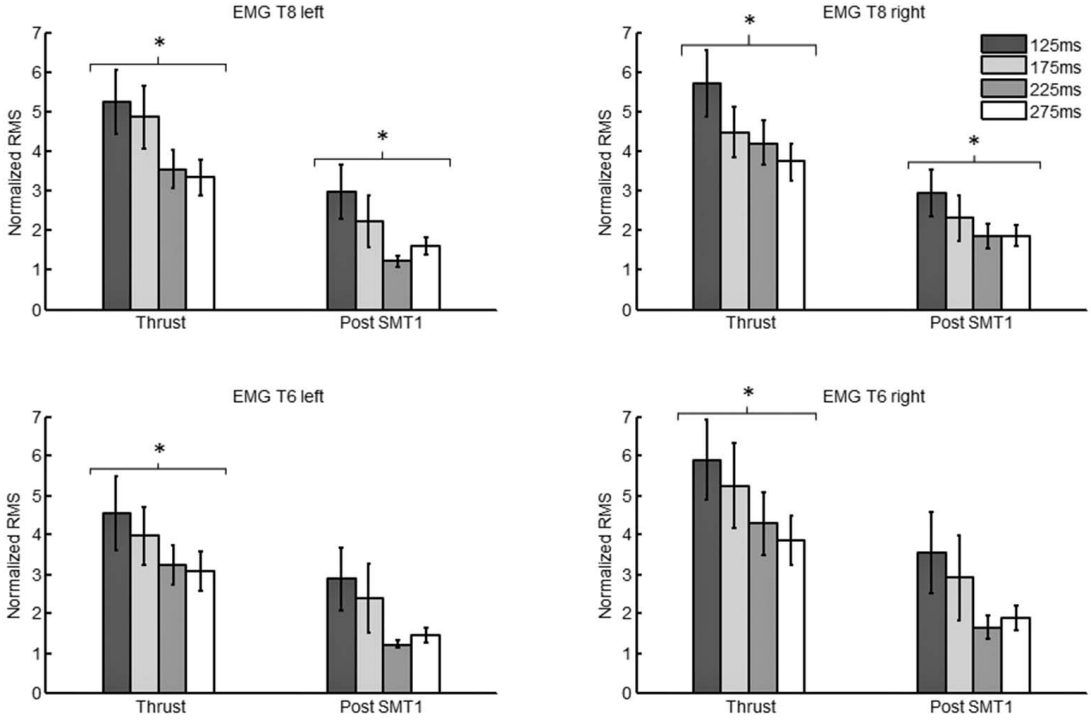

Figure 3:
EMG responses to varying levels of impulse duration during the "Thrust phase" and "Post-SMT1". Mean (standard error) normalized RMS values (T6 left and right, T8 left and right paraspinal muscles) during the "Thrust phase" and "Post-SMT1" are presented.

Data analysis:

EMG data were filtered digitally by a 20 to 450Hz band-pass 4th order Butterworth filter. A band-stop 4th order Butterworth filter was also applied to remove the power supply contribution of 60Hz. Because surface EMG electrodes were positioned in the thoracic spine area, a custom designed digital filter was used to remove ECG artefacts from surface EMG.[26]

In order to analyse EMG responses according to SMT force events, seven time windows (see figure 2) that spanned across the entire SMT force curve were defined: a "Baseline" of 500ms duration to observe EMG activity before the SMT, a "Preload phase" of 1000ms, a "Thrust phase" and four phases which successively followed the "Thrust phase" with two windows of 250ms and two windows of 500ms (referred as "Post-SMT1" to "Post-SMT4" in figure 2 and 3). Therefore, because the "Thrust phase" duration depended on the imposed impulse phase duration, its possible durations were respectively 250ms, 350ms, 450ms, and 550ms. For each trial, the four EMG recordings were divided in seven normalized root mean square (RMS) values corresponding to each time window. Normalized RMS values were obtained by dividing each RMS value by the RMS value obtained during the "Preload phase". A posterior to anterior force vector was used to perform spinal manipulations, and sagittal plane displacements were calculated. The vertebral displacement from "Preload phase" to peak force was considered for kinematic variable in the study. This value was calculated for the two kinematic markers (T7 and T8).

Statistical analyses:

All dependent variables were found to be normally distributed and were submitted to 1-way repeated-measures ANOVA (4 different impulse durations). Whenever ANOVA yielded a significant time effect, polynomial

contrasts were conducted to test for the linear trend (linear relationship between impulse duration applied and EMG response). Polynomial contrasts provide the opportunity to look at the response curve of the data and determine the nature of the relationship between SMT and EMG responses. The level of statistical significance was set at $p<0.05$ for all analyses.

Results

Figure 2 illustrates typical kinematic and EMG responses to a given SMT force-time profile. Overall, modulating the impulse duration (125ms, 175ms, 225ms, and 275ms) led to significant differences in paraspinal EMG not only during the "Thrust phase" but also during "Post-SMT1" (all T8 ps < 0.01 and T6 ps < 0.05). Testing for linear trend showed significant linear relationship between impulse duration and EMG responses for T8 (during both the "Thrust phase" and the Post-SMT1) and T6 ("Thrust phase" only) (all ps < 0.05). The linear relationship showed that decreasing the impulse duration led to a significant increase in paraspinal muscle activity. Paraspinal EMG activity was similar across all impulse duration for the remaining time-windows i.e. "Baseline phase", "Preload phase", "Post-SMT2", "Post-SMT3" and "Post-SMT4" time-windows, indicating that changes in impulse duration did not affect muscular activity during these components of SMT ($p > 0.05$). EMG responses to varying impulse durations during the "Thrust phase" and "Post-SMT1" are presented in figure 3.

Sagittal vertebral displacements from "Preload phase" to peak force were similar across all impulse duration conditions, indicating that spinal displacement during SMT did not change when modulating impulse duration ($p > 0.05$). Sagittal vertebral displacement ± standard deviation of T7 and T8 are reported in table 1.

Table 1:
Sagittal Vertebral displacement ± standard deviation of T7 and T8 from "Preload phase" to peak force.

Impulse duration	T7 (mm ± SD)	T8 (mm ± SD)
125 ms	14.52 ± 2.94	13.27 ± 3.02
175 ms	15.18 ± 2.84	13.84 ± 2.95
225 ms	15.58 ± 1.83	13.78 ± 2.65
275 ms	15.51 ± 1.91	14.01 ± 2.53

Discussion

The main objective of the present study was to investigate the SMT dose-physiological response using systematic modulation of SMT impulse duration. The main findings indicate that EMG responses of thoracic paraspinal muscles increased linearly with decreasing SMT impulse duration. Such dose-response relationship was observed during the SMT "Thrust phase" for both paraspinal muscle levels recorded (T6 and T8), but also in the first 250ms time window following the spinal manipulation impulse for T8 paraspinal muscle level. These muscle activations, however, quickly attenuated in the following time windows (from 250ms to 1.25ms after spinal manipulation impulse).

Neuromuscular responses

Other studies have previously attempted to document the relationship between the impulse duration and paraspinal EMG responses. Using an animal model (Merino sheep), Colloca et al. (2006) reported an increase in the percentage of higher amplitude EMG response to pulse duration of 100ms and 200ms compared to pulse duration of 10ms.[22] Comparisons to others studies remain difficult, as the effect of impulse duration variation has not yet been evaluated while controlling (or describing) other parameters such as peak and preload forces in human or cadaveric studies.[19,20] Furthermore, studies evaluating instrument assisted SMT were conducted using impulse duration of less than 5ms, which results in spine oscillation for up to 150ms following the application of the force impulse. According to the authors, this oscillation may contribute to impulse-triggered EMG responses and may explain differences between the studies.[20,27]

The increasing EMG responses observed with shorter SMT impulse duration in the present study seem to be coherent with the results obtained from muscle spindle recordings for which a curvilinear increase in discharge frequency[23] was observed when decreasing impulse duration[24,25].

Kinematic responses

Colloca et al. (2006) reported that a short impulse duration (10ms) produces a smaller movement of the contacted segment as well as a larger adjacent movement than a longer impulse duration (100-200ms).[22] Nevertheless, they did not report clear differences between impulse durations of 100 and 200ms, which is consistent with the present results. Interestingly, Lee et al. (1992) reported larger adjacent vertebral segment displacement during shorter impulse duration.[18] However, their fast and slow conditions consisted of 500ms and 30sec impulse durations respectively, which are considerably slower durations than those used by Colloca et al.[22] and the ones presented in this study. With regard to vertebral displacement, these results suggest an inverted U dose-response relationship where very short impulse (e.g. 10ms) and long duration (30sec) produce less vertebral movement than 100ms and 200ms impulse duration. Studies evaluating a wider range of impulse duration using controlled force and displacements are needed in order to adequately evaluate this relationship.

Practical implications and study limitations

Impulse duration tends to decrease in experienced chiropractic students[12], but remains highly variable between clinicians and across repeated SMT[28]. The results of the current study highlight the possible relationship between SMT impulse duration and neurophysiological responses. Although speed (short impulse duration) is often associated with clinical expertise in the delivery of SMT, its specific contribution to the clinical effects is unknown.

The physiological responses described in this study were obtained from young healthy participants and may not be generalizable to other populations, including patients with spinal pain. Future studies of SMT dose-response relationship in patients with cervical, thoracic and lumbar spine pain are needed. During testing, each kinematic marker were mounted on wooden supports in order to minimise masking of kinematic markers caused by linear actuator motor displacement during SMT. This procedure, in addition to skin motion during SMT may have led to an increased variability in sagittal vertebral displacement, thus minimizing the possibility to identify significant changes in vertebral displacements. An additional floor mounted camera should be added in future studies to allow direct skin positioning of the markers.

Conclusion

The present study objective was to investigate fundamental aspects of the SMT dose-physiological response relation in humans by investigating how different SMT impulse duration can modify biomechanical and neuromuscular responses to spinal manipulation. The main results indicate that while decreasing SMT impulse duration, EMG response of thoracic paraspinal muscles increased linearly during and following the SMT thrust. Whether or not these differences are of any clinical importance remains to be determined.

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Effect of spinal manipulation on the development of history-dependent responsiveness of lumbar paraspinal muscle spindles in the cat

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We determined whether spinal manipulation could prevent and/or reverse the decrease and increase in paraspinal muscle spindle responsiveness caused respectively by lengthening and shortening histories of the lumbar muscles. Single unit spindle activity from multifidus and longissimus muscles was recorded in the L_6 dorsal root in anesthetized cats. Muscle history was created and spinal manipulation delivered (thrust amplitude: 1.0mm, duration: 100ms) using a feedback-controlled motor attached to the L_6 spinous process. Muscle spindle discharge to a fixed vertebral position (static test) and to vertebral movement (dynamic test) was evaluated following the lengthening and shortening histories. For the static test, changes in muscle spindle responsiveness were significantly less when spinal manipulation followed muscle history ($p<0.01$), but not when spinal manipulation preceded it ($p>0.05$). For the dynamic test, spinal manipulation did not significantly affect the history-induced change in muscle spindle

Nous avons déterminé si les manipulations vertébrales pouvaient prévenir ou inverser la diminution et l'augmentation de la réactivité du fuseau musculaire paravertébral causé respectivement par les antécédents d'allongement et de raccourcissement des muscles lombaires. L'activité des fuseaux musculaires des muscles multifidus et longissimus prise isolément a été notée pour la racine dorsale de la vertèbre L_6 chez des chats anesthésiés. Les muscles ont été soumis à un antécédent musculaire et la manipulation vertébrale a été effectuée (amplitude la pulsion : 1,0 mm, durée : 100 ms) au moyen d'un moteur contrôlé par rétroaction fixé à l'apophyse épineuse de L_6. Les décharges du fuseau musculaire à une position vertébrale fixe (test statique) et au mouvement vertébral (test dynamique) ont été évaluées à la suite des antécédents d'allongement et de raccourcissement musculaires. Pour ce qui est du test statique, les changements dans la réactivité du fuseau musculaire étaient significativement moindres lorsque la manipulation vertébrale était effectuée après l'antécédent musculaire ($p<0,01$), ce qui n'était pas le cas lorsque la manipulation vertébrale la précédait ($p>0,05$). Pour ce qui est du test dynamique, la

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responsiveness. Spinal manipulation may partially reverse the effects of muscle history on muscle spindle signaling of vertebral position.

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KEY WORDS: Muscle spindle, proprioception, spinal manipulation, lumbar spine, paraspinal muscle, thixotropy, muscle history, chiropractic

manipulation vertébrale n'a pas eu d'effet significatif sur le changement de la réactivité du fuseau musculaire provoqué par l'antécédent. La manipulation vertébrale peut partiellement inverser l'effet de l'antécédent musculaire sur la signalisation de la position vertébrale du fuseau musculaire.

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MOTS CLÉS : Fuseau musculaire, proprioception, manipulation vertébrale, colonne lombaire, muscle paravertébral, thixotropie, antécédent musculaire, chiropratique

Introduction

Spinal manipulation is often applied to correct disturbances in the mechanical behavior of spinal motion segments. Motion between facet joints is thought to become restricted or functionally asymmetric due to paraspinal muscle dysfunction, synovial meniscoids or inclusions trapped between articular surfaces of the facet joints, intra-articular or myofascial adhesions, and/or distortion of the annulus fibrosus.[1-5] The disturbance, a spinal lesion, has had at least 100 synonyms used to describe it.[6] Chiropractic labels it a subluxation, osteopathy labels it somatic dysfunction, and manual medicine labels it fixation or functional blockage. Regardless of professional discipline, a consensus opinion is that altered segmental motion characterizes the spinal lesion for which spinal manipulation is delivered.[7,8] Controlled randomized studies indicate that spinal manipulation can induce short lasting changes in the spine's passive range of motion and longer lasting changes in its active range of motion.[9,10, but see 11]

Recent findings in humans demonstrate the importance of proprioceptive input from paraspinal muscle spindles for controlling spinal motion including regional repositioning of the lumbar spine and eliciting paraspinal muscle reflex activity. In the human lumbar spine, paraspinal muscle spindles are known to contribute to conscious awareness of low back position and movement velocity.[12-14] While healthy individuals can accurately reposition their lumbosacral spine, their repositioning ability is impaired when muscle spindle discharge is increased by applying vibration to the lumbar paraspinal muscles.[12,15] During vibration, the correct position is consistently undershot due to the misperception of paraspinal muscle length; lumbosacral orientation is "sensed" as being flexed more than it actually is. Interestingly, lumbosacral repositioning ability is impaired in individuals with a history of low back pain even in the absence of vibration[15] suggesting that abnormal proprioceptive signals can contribute to the pathophysiological mechanism of idiopathic low back pain. Additional evidence shows that simply increasing the background discharge from paraspinal muscle spindles affects paraspinal muscle reflexes. For example, vibration-induced stimulation of lumbar paraspinal muscle spindles inhibits the short latency paraspinal EMG activity normally evoked by tapping the erector spinae muscles.[16]

Paraspinal muscle dysfunction may arise from the history-dependence of muscle spindles in paraspinal muscles. This thixotropic property was first shown clearly for spindles in limb muscles of the anesthetized cat.[17,18] A history of having stretched and held the triceps surae muscles at a relatively long length (hold-long) followed by returning them to a shorter, initial length and slowly stretching them decreases the responsiveness of their muscle spindles to both the initial length and the slow stretch when compared to a history of only having held the triceps surae muscles at the shorter, initial length. It was proposed[18] that the muscle spindle apparatus stiffens at each held length. However, as the muscle is shortened following the hold-long history, the spindles kink or buckle and their ability to take up the new muscle length decreases.[18] This decrease in spindle responsive following a hold-long history alters afferent

inflow to the central nervous system and changes the biasing of spinal cord excitability.[19] In the leg's of humans and cats, the lengthening history alters the magnitude and timing of stretch-reflexes.[20,21] In the arm's of humans both lengthening and shortening histories relative to an intermediate length adversely affects repositioning accuracy.[22]

Muscle spindles in the lumbar multifidus and longissimus muscle also act thixotropically wherein the fidelity of their proprioceptive signaling is influenced by very small, maintained changes in the position of a vertebra.[23-27] Maintaining a lumbar vertebra in a position that holds the attached paraspinal muscles at a relatively long versus short length compared to an intermediate length decreases or increases, respectively the subsequent responsiveness of the lumbar muscle spindles to both the intermediate position and to subsequent muscle lengthening from the intermediate position. The magnitude of the altered responsiveness is graded with the magnitude of the change in vertebral position[26] and the plane in which the position occurs[24]. The changes are also graded with the duration over which the vertebral position is maintained.[25,27] The effect is maximal by approximately 4 s of lengthening history with a time constant of 1.1 s.[25] These changes in spindle behavior represent inaccuracies in the proprioceptive information they provide because the afferent inflow does not represent the actual position of the vertebra. It has been speculated that a history-induced reduction in feedback support from muscle spindles could be a causal element contributing to segmental tissue strain and injury in the low back.[27]

Based upon a suggestion that spinal manipulation may alter spindle sensitivity and affect muscle activity in the low back[27], the aim of the present study was to determine whether spinal manipulation in an animal preparation can correct errors in muscle spindle input that may arise from the thixotropic property of muscle spindles. Specifically, we determined whether spinal manipulation *prevented* changes in muscle spindle discharge caused by the history of vertebral position and whether spinal manipulation *reversed* the changes in muscle spindle discharge caused by the history of vertebral position.

Materials and Methods

Preparation

Experiments were performed on 27 deeply anesthetized adult cats (22 males and 5 females) weighting 3.0-5.7 kg. All cats were treated in accordance with the Guiding Principles in the Care and Use of Animals approved by the American Physiological Society. All procedures were initially described by Ge et al.[27] Briefly, deep anesthesia was initiated with pentobarbital sodium (35 mg/kg, iv) and maintained with additional dosages (~5 mg/kg, iv). Cats were mechanically ventilated (model 681; Harvard Apparatus Company, Inc., Millis, MA, USA). Arterial pH, P_{CO2}, and P_{O2} were measured every 90 minutes using i-STAT System (i-STAT Corporation, East Windsor, NJ, USA) and were maintained within normal range (pH 7.32-7.43; Pco_2, 32-37 mm Hg; Po_2, >85 mm Hg).

Paraspinal tissue dissection and a bilateral laminectomy limited to the caudal half of L_4 and the entire L_5 vertebra provided access to the L_6 dorsal roots. The low back from L_6 caudalward remained intact. To record from muscle spindle afferents from these muscles, thin filaments were teased from L_6 dorsal root using sharpened forceps under a dissecting microscope until impulse activity from a single unit with a receptive field in the paraspinal muscles could be identified. Action potentials were identified using a PC-based data acquisition system (Spike 2, Cambridge Electronic Design, Cambridge, UK). Activity from a putative muscle spindle in the lumbar spine was first identified when gentle, manual compression of the lumbar paraspinal tissues evoked a high frequency discharge. Afferents whose discharge was highest in response to probing the back muscles compared with the gluteal, hip, or leg regions, and which responded to manual movement of the L_6 vertebra in the dorsal-ventral directions were used. Following the experimental protocols, the back muscles were mechanically isolated by removing the lumbococcygeus muscle. That a receptive ending in the lumbar longissimus or multifidus muscles was the source of neural activity was determined using von Frey hairs (Stoelting Co, Wood Dale, Il, USA) to confirm that the most sensitive area for mechanically activating the afferent was actually located in the back muscles. Three methods were used to confirm that neural activity was from a muscle spindle as described previously:[28,29] 1) the afferent's ability to follow vibration (90 Hz, 0.06 mm; Mini-Vibrator, Model NC70209, Morgan Hill, CA, USA) applied to the muscle, 2) decreased discharge to a direct muscle twitch, and 3) sustained increase in discharge to succinylcholine injection (100-300 μg/kg, intra-arterial).

While recording afferent activity from lumbar para-

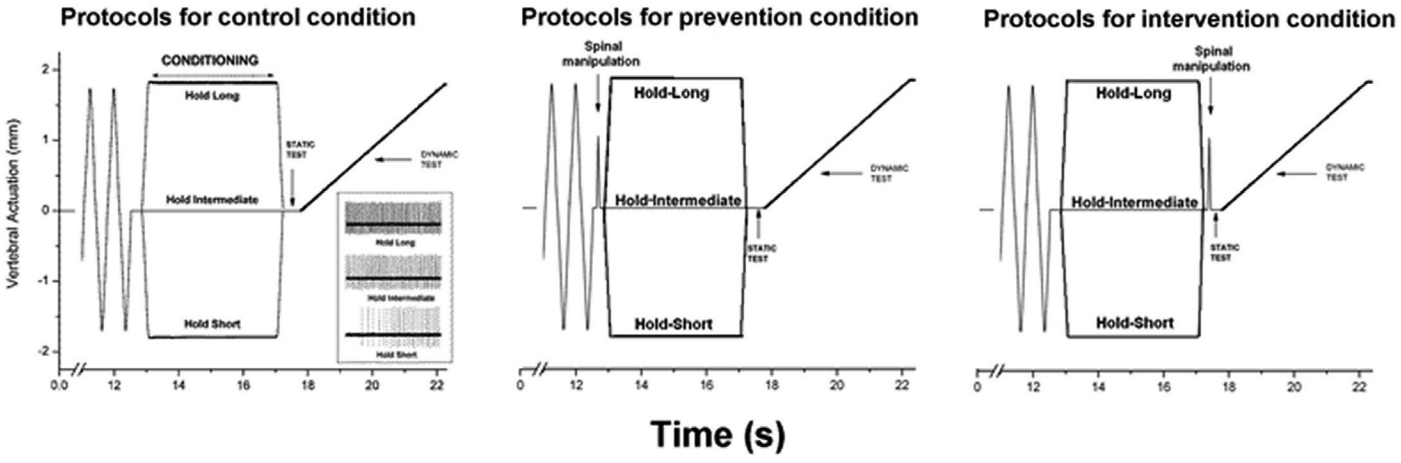

Figure 1.
Schematic of the experimental protocol during 3 spinal manipulation conditions and a representative response (inset during the control condition) of one spindle to three the 3 muscle history conditions. Loading protocol shows the change in vertebral position relative to the intermediate position.

spinal muscle spindles, actuation of the L_6 vertebra was induced using an electronic feedback control system (Lever System Model 310; Aurora Scientific, Aurora, Ontario, Canada). A horizontally-aligned lever arm attached to the motor's rotary drive shaft was coupled to the L_6 spinous process via a pair of adjustable tissue forceps (152.4 mm long, 1 × 2 teeth) vertically aligned. The forceps were clamped tightly onto the lateral surfaces of the L_6 spinous process through thin slits along either side of the vertebra. Controlled displacements of the lever arm were applied along the cat's dorsal-ventral axis thus actuating the vertebra in a dorsal-ventral direction.

Muscle History Caused by Changes in Vertebral Position

Muscle history was created by holding the L_6 vertebra at an intermediate position for 4.0s (hold-intermediate), or by moving it ±1.7mm and then holding it for 4.0s at the new position. Moving the vertebra ±1.7mm maintained the attached muscles at lengths relatively shorter (hold-short) or longer (hold-long) than the hold-intermediate length (see Fig. 1). At the hold-intermediate position, paraspinal tissues exerted no force against the motor's drive shaft. The direction that constituted hold-short was identified by a reduction in spindle discharge and hold-long by an increase in spindle discharge. Prior to creating each type of muscle history, the system was placed in a similar mechanical state by rapidly moving (10 mm/s): the L_6 vertebra back and forth 10 times, stretching and shortening the attached muscles to the same magnitude as the hold amplitude (Fig. 1).

The effects of each muscle history were assessed using a static test and a dynamic test as performed previously.[23-27] The static test occurred immediately following each "hold" condition by returning the vertebra to the intermediate position for 0.5s. The dynamic test followed the static test. The vertebra was slowly moved at 0.2 mm/s to the same displacement as the hold condition (1.7mm) in a direction that stretched the paraspinal muscle. Muscle spindle discharge during each of these tests in response to the hold-intermediate history was compared with the hold-long and with the hold-short histories.

Spinal Manipulation

Spinal manipulations were delivered in a fashion similar to those described previously.[30-33] Forceps were attached at the L_6 vertebra to guide its motion. The forceps were positioned perpendicular to the lever arm so that force and displacement at the end of the lever arm were the same as that at the back of the cat where it was contacted

by forceps. With the cat lying prone, spinal manipulation was applied in a vertical direction from dorsalward to ventralward. The displacement-time profile of the manipulation simulated that delivered clinically [discussed in [29,30]]. The manipulation was always delivered with the motor in displacement control and at constant velocity (0.01m/s: thrust amplitude = 1.0mm; thrust duration = 100ms).

Experimental Design

Each cat received 3 muscle history conditions: hold-intermediate, hold-long, hold-short. Each cat received 3 manipulation conditions: no spinal manipulation (control), spinal manipulation before creating muscle history (prevention), and spinal manipulation after creating muscle history (intervention). Thus, each cat received 9 protocols and served as its own control. Each of the 9 protocols was separated by at least 5 minutes. The presentation order of the 3 manipulation conditions was randomized across cats. The presentation order of the 3 muscle history conditions was randomized within a manipulation condition.

Data Analysis

Spindle activity was quantified as mean instantaneous frequency (MIF) for the static test and mean frequency (MF) for the dynamic test.[23-27] MIF was calculated by averaging the reciprocal of each time interval between consecutive action potentials. MF was calculated by dividing the number of action potentials by the dynamic test's duration. The responsiveness was defined as the change in MIF or MF between the hold-intermediate and the hold-short (ΔMIF_{short}, ΔMF_{short}) or hold-long protocols (ΔMIF_{long}, ΔMF_{long}). A positive value indicated an increase in muscle spindle responsiveness and conversely, a negative value indicated a reduction in muscle spindle responsiveness. Values close to zero indicated that conditioning had little or no effect. Spindle responses are reported as means (lower 95% confidence limit, upper 95% confidence limit) unless otherwise indicated.

One-way ANOVA was used to compare the effects of the control, prevention and intervention conditions on muscle spindle responsiveness during the static and dynamic test. Statistical significance was set at the $P < 0.05$ level for the entire study. Post-hoc pairwise comparisons were performed when significance reached $P < 0.05$ and were adjusted for multiple comparisons using the Bonferroni method. Statistical analyses were conducted using SAS (version 9.1, SAS Institute, Cary, NC).

Results

Physiological Characteristics of the Spindles

Twenty-eight paraspinal muscle spindle afferents were studied. Receptive fields from 8 afferents were in the lumbar multifidus muscle and 20 were in the longissimus muscle. The most sensitive portion of each receptive field was located medially (i.e., either in the multifidus muscle or the medial border of the longissimus muscle) and near the L_{6-7} or L_7-S_1 facet joint. Mechanical thresholds of lumbar paraspinal muscle spindles ranged between 4.0 and 115.2 mN [35.7 (39.2) mN; mean (SD)].

The discharge of all 28 afferents increased in response to succinylcholine injection. Twenty-seven afferents were silenced by bipolar muscle stimulation; 1 afferent could not be tested because the unit was damaged by insertion of the stimulating electrode. Twenty-seven afferents were tested with vibration applied indirectly to the muscle through the thoracolumbar fascia and 28 were tested with vibration applied directly to the muscle's exposed surface after removing the overlying fascia. During vibration through the fascia, all 27 spindle afferents were activated. Twenty-six were driven 1:1 (70 – 93 imp/s; i.e. with a discharge frequency similar to the vibration frequency) and 1 responded with a subharmonic discharge frequency (44 imp/s), however this latter unit was driven with direct muscle vibration. During vibration applied directly to the surface of the exposed muscle, 27 units were driven by direct muscle vibration. One unit could not be tested by direct muscle vibration because it died before the protocol was completed.

Responses to Conditioning and Spinal Manipulation

Before analyzing how the 3 spinal manipulation conditions affected the history-dependent responsiveness of muscle spindles during the static and dynamic tests, we wanted to be sure that the spinal manipulation given prior to the creation of muscle history did not differentially affect the creation of muscle history. Therefore,we compared between each of the 3 spinal manipulation conditions spindle activity during the conditioning phase (see "conditioning" label in left panel of figure 1) of both the

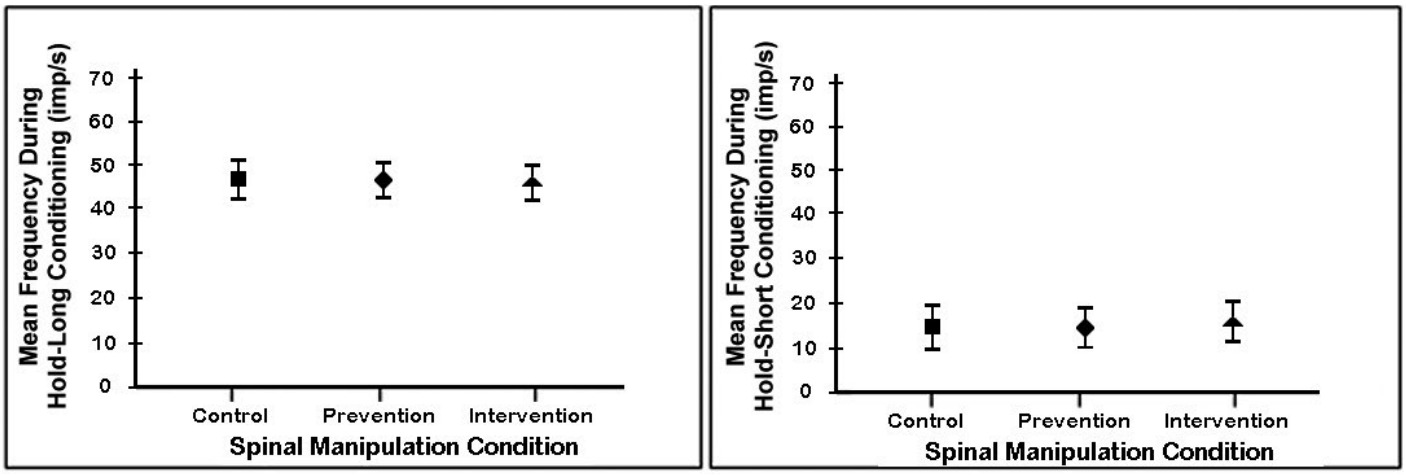

Figure 2.

Mean discharge frequency of paraspinal muscle spindles for each of the 3 spinal manipulation conditions during the conditioning phase used to create muscle history. Conditioning phase identified graphically in left panel of figure 1. Each symbol represents the mean ± 95% confidence interval of 28 spindles.

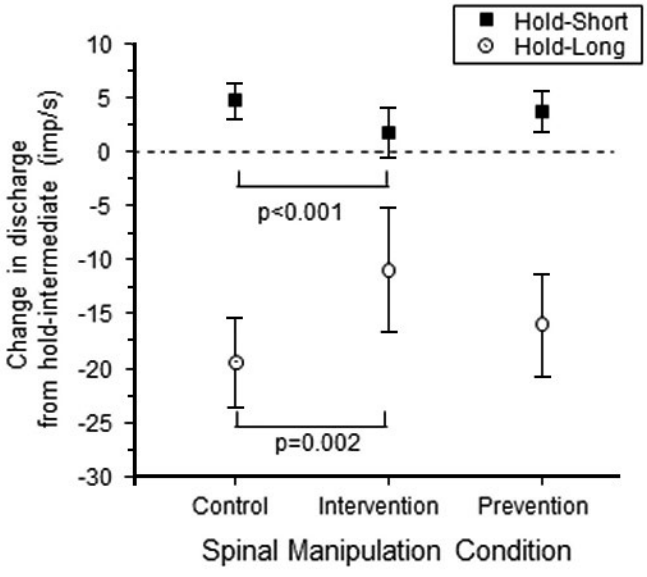

Figure 3.

Mean change in resting spindle afferent discharge during the static test for the 3 spinal manipulation conditions. Y-axis represents the change in muscle spindle discharge following the hold-long or hold-short compared with the hold-intermediate conditionings (ΔMIF_{long} or ΔMIF_{short}). Each symbol represents the mean ± 95% confidence interval of 28 spindles.

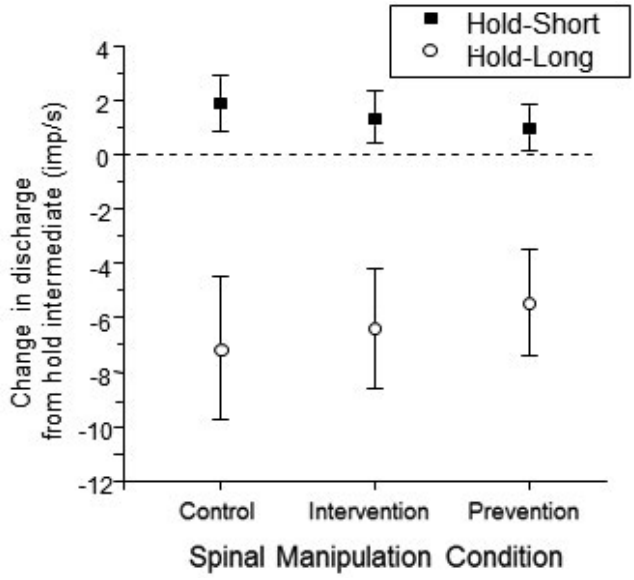

Figure 4.

Mean change in spindle afferent discharge during the dynamic test for the 3 spinal manipulation conditions. Y-axis represents ΔMF averaged over the entire movement of the dynamic test. Each symbol represents the mean ± 95% confidence interval of 28 spindles.

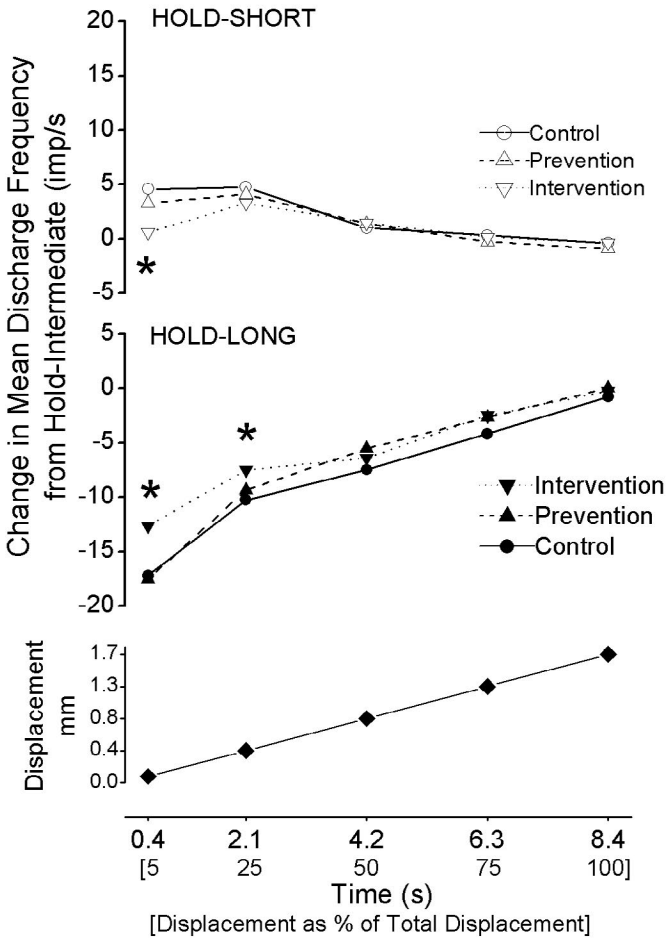

Figure 5.
*Time course of changes in muscle spindle discharge during the dynamic test for hold-short compared with hold-intermediate (upper panel) and for hold-long compared with hold-intermediate (middle panel). Bottom panel shows the magnitude of the vertebral movement over which the dynamic test was analyzed. * p<0.05 compared with the spinal manipulation control condition. Each symbol represents the average value between its time position and the time position of the previous data point, except for 5% which represents the average value between its time position and time 0 s.*

hold-long and hold-short histories. As shown in figure 2, spindle activities regardless of manipulation condition were similar during the 4 second lengthening histories and during the 4 second shortening histories. Thus, the spinal manipulation given prior to the creation of muscle history did not affect the process of creating history in the spindles.

Static Test

Results from the static test are summarized in figure 3. For the control condition with no spinal manipulation, hold-long compared with hold-intermediate (ΔMIF_{long}) decreased resting muscle spindle discharge by –19.4 (–23.6, –15.3) imp/s on average whereas hold-short compared with hold-intermediate (ΔMIF_{short}) increased it by 4.7 (3.0, 6.4) imp/s. This result is consistent with findings from previous studies.[23-25,27] For the prevention condition where spinal manipulation was given prior to creating muscle history, ΔMIF_{long} decreased by –16.0 (–20.9, –11.1) imp/s and ΔMIF_{short} increased by 3.7 (1.8, 5.7) imp/s. There was no significant difference in the responsiveness between the control and prevention conditions for either the hold-long or hold-short condition ($P = 0.33$ for ΔMIF_{long}, $P = 0.38$ for ΔMIF_{short}). For the intervention condition where spinal manipulation was given following the creation of muscle history, ΔMIF_{long} decreased by –10.9 (–16.7, –5.1) imp/s and ΔMIF_{short} increased by 1.7 (–0.6, 4.1) imp/s. Responsiveness to the effects of muscle history during the intervention condition was significantly less than that during the control condition both for hold-long and hold-short muscle history ($P = 0.002$ for ΔMIF_{long}, $P < 0.001$ for ΔMIF_{short}).

Dynamic Test

Averaged over the 8.4 s duration of the dynamic test which displaced the vertebra the same amount as the hold-long condition, ΔMF_{long} for the control, prevention, and intervention conditions was –7.2 (–9.8, –4.5) imp/s, –6.4 (–8.6, –4.2) imp/s, and –5.5 (–7.4, –3.5) imp/s, respectively. ΔMF_{short} for the three hold conditions was 1.9 (0.9, 2.9) imp/s, 1.3 (0.4, 2.3) imp/s, 1.0 (0.1, 1.9) imp/s, respectively. The magnitudes of the absolute changes in ΔMF_{long} and ΔMF_{short} were substantially larger to the lengthening compared to the shortening history. There were no significant differences in either ΔMF_{long} or ΔMF_{short} among the 3 spinal manipulation conditions

($F_{2, 83} = 2.45$, $P = 0.10$ and $F_{2, 83} = 2.54$, $P = 0.09$, respectively, Fig. 4).

The effect of spinal manipulation on spindle responsiveness during the dynamic test was also averaged over smaller increments of the test. Because the dynamic test was always applied at the same velocity (0.2 mm/s) and to the same magnitude of vertebral movement (1.7mm), identical time points during the test represent the same magnitude of vertebral movement. Therefore responsiveness during similar amounts of vertebral movement could be compared based upon time points of the dynamic test. As shown in Figure 5 (bottom panel), comparisons were made for vertebral movement between the intermediate position and the first 0.09mm of movement (5% of total movement), between 0.09 and 0.42mm (next 20% of total movement), between 0.42 and 0.85mm (25 -50 % of total movement), between 0.85 and 1.28 mm (50-75% of total movement), and between 1.28 and 1.70 mm (75-100% of total movement (Fig. 5 bottom panel). Comparisons averaging over 100% of the movement (1.70 mm) represent the average over the entire duration as described in the preceding paragraph. The spinal manipulation intervention condition returned dynamic spindle responsiveness toward normal (i.e., ΔMF_{long} approached zero) significantly more than either the control or prevention conditions when the vertebra was moved 5% and 25% of the full movement ($F_{2, 83} = 6.22$, $P = 0.004$ and $F_{2, 83} = 3.16$, 0.05, respectively, Fig. 5 middle panel). Similarly, the spinal manipulation intervention condition returned dynamic spindle responsiveness toward normal (ie ΔMF_{short} approached zero) significantly more than either the control or prevention conditions when the vertebra was moved 5% of the full movement ($F_{2, 83} = 7.95$, $P < 0.001$, Fig. 5 top panel). While the effects of the hold-short and hold-long muscle history conditions were present throughout the dynamic test, the specific effect of the spinal manipulation intervention condition was not present after 25% of the dynamic test.

Discussion

One clinical consequence of spinal manipulation is thought to be the normalization of paraspinal neuromuscular dysfunction. The present study demonstrated that spinal manipulation partially reversed but did not prevent the decrease in muscle spindle responsiveness caused by the lengthening history of lumbar paraspinal muscles (i.e. by hold-long), This suggests that spinal manipulation could reduce proprioceptive errors caused by the thixotropic property of muscle spindles in paraspinal muscles. Although the nature of the paraspinal muscle dysfunction amenable to spinal manipulation is not clear, changes in proprioceptive input or processing have often been proposed as a cause.[34,35] In the limbs, muscle history has been shown to disrupt neuromuscular integration by altering proprioceptive feedback from muscle spindles which creates positioning errors and modifies the timing and magnitude of reflex support.[20-22] In the vertebral column, we do not know with any certainty whether paraspinal muscle history contributes to the dysfunction for which spinal manipulation is applied clinically.[36]

The effect of both the hold-long and hold-short history during the control condition (no spinal manipulation) was similar to our previous studies showing that the positional history of a lumbar vertebra differentially alters the responsiveness of the paraspinal muscle spindles.[25,27] The discharge of spindles with a vertebra held at an intermediate position and during vertebral movement from that intermediate position decreases significantly when the intermediate position has been preceded by a vertebral position that maintains the spindle apparatus at longer length. Conversely, maintaining the spindle apparatus at shorter length relative to that at the intermediate position increases spindle responsiveness to both vertebral position and movement.

Several studies suggest that small changes in paraspinal muscle force can have a large impact on a motion segment's biomechanical behavior and stability.[37-40] These studies have contributed to the idea that damage to structures of the vertebral column and the risk of injury to the spine can be great during easy, non-demanding tasks.[38] For example, in vitro experiments accompanied by a modeling approach that incorporates graded increases in the activity of 1 lumbar paraspinal muscle show an increase in vertebral stabilization.[37] Graded increases in the muscle's modeled activity decreases the intersegmental neutral zone (range: 33%-40%) during flexion, extension, and axial rotation but not lateral bending, and decreases intersegmental range of motion (range: 7-27%) during extension and axial rotation but not flexion or lateral bending. The largest decrease in the neutral zone (hence greatest stabilization) and range of motion during these maneuvers occurs at low muscle forces (20N

compared with 40N and 60N). Similarly, a very small increase (1-3% of maximal voluntary contraction) in lumbar multifidus, iliocostalis and thoracic longissimus muscle activity at L_2-L_4 is sufficient to restore segmental stability of the lumbar spine even when the loading moments are increased to 75% of body weight.[38] When the force vectors from 5 paraspinal muscles are incorporated into the modeling approach, stabilization of an individual lumbar motion segment also increases: the intersegmental neutral zone decreases (range: 76-83%) during flexion, extension, axial rotation, and lateral bending and intersegmental range of motion decreases (range: 55-93%) during flexion, extension, axial rotation, and lateral bending.[39] Multifidus muscle accounts for 40-80% of the increased stability during sagittal flexion-extension, 45% during axial rotation, and 10-20% during lateral bending suggesting that neuromuscular mechanisms controlling multifidus muscle activity alone could functionally impact the motion segment especially during flexion-extension and axial rotation. Abnormal control of multifidus muscle may contribute to the fact that mechanical injury to the intervertebral disk occurs most often during loading moments that combine flexion, lateral bending, and axial rotation.[40] We speculate that intersegmental and regional spinal postural history, when it changes the responsiveness of paraspinal muscle spindles, represents a source of inaccurate proprioceptive information from the paraspinal muscles that could affect development of low level muscle activity and compromise neuromuscular control of spinal stability.

The phenomenon of muscle history is thought to arise from the spontaneous formation of stable, non-recycling intrafusal cross-bridges between actin and myosin filaments when muscle is held at constant length.[41] During voluntary muscle contraction in the limbs, co-activation of gamma- with alpha-motoneurons is thought to break these non-recycling crossbridges and return spindle afferent signaling to normal.[18,42,43] However, in the spine, voluntary paraspinal extensor contractions may not be as effective at reversing the effects of muscle history[44] in that forward flexion does not eliminate proprioceptive changes whose origins are consistent with a lengthening history[44,45]. The present study demonstrated that spinal manipulation helped reduce errors in muscle spindle signaling caused by the history of vertebral position. While passive stretching will eventually break the crossbridges as indicated in Figure 5, spinal manipulation may reduce the effects of history when voluntary movement is unable to stretch the muscles to a length that created the history in the first place. In clinical practice, spinal manipulation may have a greater influence on reducing the effects of muscle history than shown in this study because when applied manually, the practitioner typically brings a joint to its end range of motion and then moves it slightly beyond what the patient can accomplish through voluntary activity alone.[46]

Relevance and application

Well-designed, scientific studies using animal preparations are a means to understand neural mechanisms that contribute to the physiological effects of spinal manipulation. The knowledge gained through such studies can provide biological validation for the use of spinal manipulation, and help improve its delivery for the healthcare of patients.

In clinical practice, palpatory examination of the back identifies abnormalities in the texture and tone of paraspinal soft tissues, the presence of pain and/or tenderness in these tissues, and restrictions in spinal joint motion in or near these areas.[47,48] The idea that altered proprioceptive input from paraspinal tissues can cause these abnormalities and that spinal manipulation corrects these inputs is not new. Nearly 4 decades ago Korr[35,49] presented the idea the central nervous system's ability to appropriately control and coordinate activities of the paraspinal musculature and its autonomic support requires an accurate representation of their conditions. Such assessment arises in part from reliable, coherent patterns of neural feedback from sensory receptors in the paraspinal tissues.

Korr originally proposed[35] that decreased muscle spindle input from paraspinal tissues causes the central nervous system to increase gamma motoneuron activity in an effort to regain or normalize sensory feedback from these proprioceptors. One consequence of this increased gain was thought to be the change in paraspinal tissue texture and tone described above and previously measured by Denslow.[50] Spinal manipulation was thought to induce a barrage of sensory input form the paraspinal muscle spindles which enabled the central nervous system to normalize gamma motoneuron activity. Although there are no data regarding spinal manipulation's effect on gamma motoneurons, animal studies have shown that a barrage

of sensory input from muscle spindles does occur during spinal manipulation when it is delivered with biomechanical characteristics similar to those used clinically.[29,51] The present study confirmed that lengthening histories of paraspinal muscles reduces normal muscle spindle input, creating errors in the assessment of segmental vertebral and revealed that spinal manipulation under these conditions can return spindle input toward normal.

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Effect of changing lumbar stiffness by single facet joint dysfunction on the responsiveness of lumbar muscle spindles to vertebral movement

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Objective: *Individuals experiencing low back pain often present clinically with intervertebral joint dysfunction. The purpose of this study was to determine whether relative changes in stiffness at a single spinal joint alters neural responsiveness of lumbar muscle spindles to either vertebral movement or position.*

Methods: *Muscle spindle discharge was recorded in response to 1mm L_6 ramp and hold movements (0.5mm/s) in the same animal for lumbar laminectomy-only (n=23), laminectomy & $L_{5/6}$ facet screw (n=19), laminectomy & $L_{5/6}$ facetectomy (n=5) conditions. Mean instantaneous frequency (MIF) was calculated for the ramp-up, hold, ramp-down and post-ramp phases during each joint condition.*

Objectif : *La lombalgie se manifeste souvent cliniquement sous forme de dysfonction articulaire intervertébrale. Cette étude a pour objet de déterminer si des changements relatifs dans la rigidité d'une seule articulation vertébrale modifieraient la réactivité des fuseaux musculaires lombaires envers le mouvement ou la position des vertèbres.*

Méthodologie : *Les décharges des fuseaux musculaires ont été notées en réponse à des mouvements de rampe et de maintien de 1 mm à L_6 (0,5 mm/s) chez le même animal pour le groupe laminectomie lombaire seulement (n=23), laminectomie et vis translaminofacettaire $L_{5/6}$ (n=19), laminectomie et facettectomie $L_{5/6}$ (n=5). La fréquence instantanée moyenne (FIM) a été calculée pour les phases d'intensification, de maintien, d'atténuation et post-rampe pour chacun des groupes.*

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Results: *Mean MIFs were not significantly different between the laminectomy-only and the other two types of joint dysfunction for the ramp-up, hold, ramp-down, or post-ramp phases.*

Conclusion: *Stiffness changes caused by single facet joint dysfunction failed to alter spindle responses during slow 1mm ramp and hold movements of the L_6 vertebra.*

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KEY WORDS: stiffness, joint, muscle spindle, chiropractic

Résultats : *Les FIM n'étaient pas significativement différentes entre le groupe laminectomie seule et les deux autres types de dysfonction articulaires pour les phases d'intensification, de maintien, d'atténuation et post-rampe.*

Conclusion : *Les changements de rigidité causés par une dysfonction articulaire à facette unique n'ont pas réussi à modifier la réponse des fuseaux au cours de mouvements lents de rampe et de maintien de 1 mm de la vertèbre L_6.*

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MOTS CLÉS : rigidité, articulation, fuseau musculaire, chiropratique.

Introduction

Aberrant neuromuscular control of the trunk along with the inability of individuals with LBP to adopt optimal postural control strategies is thought to be involved in the etiology of low back pain (LBP).[1-10] Individuals with LBP demonstrate reduced lumbar muscle activation or earlier onsets of muscle activation following predictable and unpredictable trunk loading.[6] It has been reported that individuals experiencing an active episode of LBP demonstrate inadequate trunk muscle activation or inappropriate trunk muscle co-activation in response to rapid and/or unexpected perturbation.[11-13] In addition, LBP patients exhibit altered movement patterns between recurrent episodes but it is unclear whether the patterns develop prior to or following the first LBP episode.[10] These altered neuromuscular responses that accompany LBP have been attributed to a number of mechanisms including segmental neural circuitry and cognitive responses due to stress, pain avoidance and/or anticipation of pain.[14,15]

Muscle spindles are proprioceptors which provide a continuous sensory input to the central nervous system related to muscle length and rate of change in muscle length, and thereby potentially supply information regarding joint position and movement. This sensory input may help to optimize neuromuscular control of the trunk and intervertebral motion during intended movement trajectories. Compared to muscle spindles in appendicular muscles, much less is known about the functional characteristics of these proprioceptors in trunk musculature. However differences in structural complexity, organization and response to changes in muscle length have been described in muscle spindles of the trunk relative to appendicular muscles.[16-21] For example, we have recently shown that measures of dynamic responsiveness in trunk muscles are 5-10x higher than values reported for appendicular muscles.[18] Table 1 provides an abbreviated summary of recent findings regarding the responses of paraspinal muscle spindles to changes in both vertebral position and movement as well as to high velocity low amplitude spinal manipulation using variations of the experimental model employed in the present study.

Impaired spinal biomechanics are thought to have adverse physiological consequences by producing less than optimal neuromuscular control of the trunk. Individuals experiencing acute or chronic LBP episodes often present clinically with intervertebral joint dysfunction.[29-31] The relationship between intervertebral joint mobility and alterations in trunk mechanoreception has received little direct investigation but is of clinical interest due to the frequent assessment of intervertebral joint mobility by manual therapy practitioners during their clinical decision making process when treating patients experiencing LBP.

There is evidence suggesting that clinical identification of spinal joint hypo- and hypermobility subgroups along with correspondingly tailored manual therapy treatment approaches can lead to more successful therapeutic out-

Table 1

Muscle spindle response to vertebral position, movement, & spinal manipulation in an animal model

I. Lumbar Vertebral Position and Movement	
The history of vertebral position can change the responsiveness of muscle spindles. Static postures that changed lumbar vertebra position by as little as 0.07mm for as little as 4sec altered muscle spindle responsiveness to passive movement.	Cao and Pickar 2011[22] Ge and Pickar 2012[24]
Dynamic responsiveness of paraspinal muscle spindles are at least 5-10x higher compared to values reported for appendicular muscles.	Cao et al. 2009[18]
Muscle spindle responsiveness to lumbar vertebral movement and to a new position did not change in response to HVLA-SM regardless of thrust force, displacement, or duration.	Cao et al. 2013[23]
II. High Velocity Low Amplitude Spinal Manipulation (HVLA-SM)	
As HVLA-SM thrust duration approaches those used clinically, discharge frequency greatly increases and the increase depends more upon the amplitude of the thrust opposed to the thrust force.	Reed et al. 2013[25] Pickar & Kang 2006[27] Pickar et al. 2007[28]
Lumbar muscle spindles show more sensitivity to smaller HVLA-SM thrust displacements (1 vs 2mm).	Pickar et al. 2007[28]
HVLA-SM thrust duration effects baseline spindle discharge at 1, 2, 3mm displacements and 25, 55, 85% body weight thrust force.	Cao et al. 2013[23]
Intersegmental mobility changes at a single facet joint alters spindle response to clinically relevant HVLA-SM thrust durations (≤ 150ms).	Reed et al. 2013[26]

comes.[32-34] In a randomized clinical trial categorizing 131 LBP patients with respect to the clinical determination of spinal joint hypo- and hypermobility, it was reported that individuals with spinal joint hypomobility had greater improvement with spinal manipulation than individuals with spinal joint hypermobility.[33] This clinical study highlights the need not only to understand the underlying biological mechanisms of manual therapy intervention but suggests that the physiological response to the same therapeutic intervention differs based on the clinically identified types of spinal joint dysfunction (hypo- or hypermobility).

Motivated by the lack of knowledge regarding how different types of spinal joint dysfunction affect trunk mechanoreceptor activity and possibly clinical outcomes to the same manual therapeutic intervention, we undertook a series of basic science experiments investigating the effect of spinal joint dysfunction on sensory input related to vertebral movement and spinal manipulation. We previously reported the effects that single facet joint dysfunction has on sensory input during spinal manipulation.[26] The purpose of this paper is to report the effects that single facet joint dysfunction have on the mean instantaneous frequency of muscle spindles located in trunk musculature during 1mm ramp and hold movements of the L_6 lumbar vertebra derived from secondary analyses of the previous study involving facet joint dysfunction and spinal manipulation.[26]

Methods

Electrophysiological recordings were made from lumbar paraspinal muscle spindles in 23 Nembutal-anesthesized male cats weighing an average of 4.46kg (SD 0.31). All experiments were reviewed and approved by the Institutional Animal Care and Use Committee and comply with the Canadian Council on Animal Care. One neuron was investigated per animal because of the irreversible nature of the $L_{5/6}$ facetectomy surgical procedure. The experi-

Table 2

	Laminectomy Only	Laminectomy & Facet Screw	Laminectomy & Facetectomy
Experimental Order	1st →	2nd →	3rd
Vertebra Movement	L6	L6	L6
Number of L6 Neurons Analyzed	20	19	5

mental approach has been described previously in detail[25,26,35] and is presented only briefly.

A mixture of O_2 and isoflurane was delivered through a facemask (2L/min and 2%) in order to place catheters in a common carotid artery and an external jugular vein to monitor blood pressure and introduce fluids respectively. Following catheterization, deep anesthesia was maintained throughout the experiment with Nembutal (35 mg/kg, iv). Deep anesthesia was identified by absence of withdrawal reflex to noxious pinching of the toe pad, mean arterial pressures less than 120mmHg and the absence of a pressor response to surgical manipulation.

The proximal portion of the L_6 dorsal roots (cats have 7 lumbar vertebrae) was exposed after a bilateral laminectomy at the L_5 vertebra. The musculature on the right side of the spinal column (multifidus, longissimus and iliocostalis muscles) remained intact except for any attachments to the posterior portions of the $L_{4\text{-}5}$ vertebrae and for small slit incisions (3mm) on either side of the L_6 spinous process for forceps attachment by which the vertebra was moved. Most of the multifidus muscle remained attached to the L_6 vertebra using this method because it's aponeurotic tendon inserts onto the process's caudal edge.[36] In addition, the L_6 dorsal root enters the spinal cord 1 to 1½ vertebral segments cranial to the L_6 paraspinal soft tissues. The L_6 dorsal root was cut close to its entrance into the spinal cord and placed on a small platform. Thin filaments from the cut proximal dorsal rootlets were teased apart until muscle spindle activity from a single neuron with the most sensitive part of its receptive field being in the low back could be identified. At the end of the experimental protocols several approaches were used to confirm receptor location and its identity as a muscle spindle including: (1) vonFrey filaments (Stoelting, USA) to confirm the most sensitive area for mechanically activating the neuron was in the multifidus or longissimus muscles (the intervening lumbococcygeus muscle innervated by sacral nerves was removed; (2) a sustained increase in discharge response to succinylcholine injection (100 ug/kg, ia); (3) a sustained increase in response to a fast vibratory stimulus and (4) decreased discharge to paraspinal muscle electrically induced muscle twitch.

Ramp and hold movement of the L_6 vertebra was controlled using an electronic feedback control system (Lever System Model 310; Aurora Scientific) under displacement control. Attached to the control system's lever arm was a pair of adjustable tissue forceps which were clamped tightly onto the lateral surfaces of the L_6 spinous process. Ramp and hold movements of 1mm peak amplitude were applied at a rate of 0.5mm/s. Due to the facetectomy, testing order for the three joint conditions in the same animal was fixed. Therefore, determination of muscle spindle responses to the 1mm ramp and hold displacements was conducted in the following order: laminectomy-only, laminectomy & facet screw, laminectomy & facetectomy (Table 2). It should be noted that ramp testing for each spinal joint condition was performed prior to conducting a series of 5 randomized spinal manipulative thrust protocols (time control-0 ms, 75, 100, 150, 250ms) each separated by 5 minute intervals as previously described in detail.[26] In addition, insertion of the facet screw and facetectomy procedure typically required 30-35 minutes to accomplish equating to approximately 1 hour elapsing between ramp and hold testing per spinal joint condition once a paraspinal muscle spindle was isolated.

Creating Spinal Joint Conditions and Determining of Lumbar Stiffness

Changes in spinal stiffness were created by unilateral (left) $L_{5/6}$ facet-fixation (to increase intervertebral stiffness) or $L_{5/6}$ facetectomy (to decrease intervertebral stiffness). The left $L_{5/6}$ facet joint was fixated by inserting a single 10mm titanium endosteally-anchored mini-screw (tomas®-pin; Dentaurum, Germany) through the articular pillars of the $L_{5/6}$ facet joint. For the facetectomy, the left L_5 inferior facet and left L_6 superior facet were removed using bone rongeurs.

Lumbar stiffness testing was first performed in the laminectomy-only condition, as opposed to the intact spine, as this was the spinal condition in which neural recordings were first obtained. To determine lumbar stiffness in each joint condition, a 1mm ramp and hold movement of the L_6 vertebra was applied in the dorsal ventral direction at a rate of 0.5mm/s using the feedback-controlled motor. During the 1mm ramp and hold, forces and displacements were being measured so that force-displacement curves of the ramp portion could be constructed. The slope of the most linear portion of the force-displacement curve (between 2.16 – 8.83N) of the 1mm ramp was calculated and represented the spinal joint stiffness for each condition. Ramp pre-conditioning was not performed in order to minimize the total number of facet screw/bone engagements.

Twenty-three animals were used in this study. As described previously,[26] animals in which the laminectomy & facet screw (n=4) failed to increase ramp stiffness by at least 2 % when compared to the laminectomy-only condition were excluded from further analysis. Similarly, animals in which the laminectomy & facetectomy (n=8) failed to decrease ramp stiffness by at least 2% when compared to laminectomy-only were also excluded. In addition, during the laminectomy & facetectomy condition the neural recording was lost in 10 animals due to facetectomy-associated bleeding. Therefore, of the 23 animals used in this study, 20 neurons were included in the analysis: 4 had data for all 3 conditions (laminectomy-only, laminectomy & facet screw, laminectomy & facetectomy), 15 had data for the laminectomy-only and laminectomy & facet screw conditions, and 1 had data for the laminectomy-only and laminectomy & facetectomy conditions (Table 2).

Data Analysis

Muscle spindle activity was converted to instantaneous frequency (IF) by taking the reciprocal of the time interval between successive action potentials. IFs during the constant velocity ramp movement and hold position were used to obtain the following 5 measures of afferent response: (a) baseline during the 2 seconds that immediately preceded each ramp-up; (b) ramp-up; (c) during the last 2 seconds of the hold phase; (d) ramp-down and (e) post-ramp during 2 seconds that immediately follow the ramp-down (Fig. 1). Mean IF (MIF) was calculated over the durations of baseline, ramp-up, hold, ramp-down and the post-ramp phase.

Changes from baseline MIF due to the laminectomy-only, laminectomy & screw and the laminectomy & facetectomy condition during the 1mm ramp and hold constituted the response measures. All neural activity was reported in impulses per second (imp/s). Data were analyzed in SAS System for Windows (Release 9.2) (SAS Institute Inc., Cary, NC). Statistical significance was set at 0.05. Each response variable was analyzed with a linear mixed effects longitudinal regression model including individual random effects to account for correlation between repeated measurements for an individual neuron based on a compound symmetry covariance structure. Adjusted means and 95% confidence intervals based on this model are reported.

Results

Muscle spindle recordings were analyzed from neurons with receptive fields located in the multifidus muscle (n=3) and longissimus muscle (n=17). In the cat, these lumbar paraspinal muscles are the two most medial to the spinous process.[36] In response to succinylcholine injection, all neurons exhibited long lasting high frequency discharges relative to baseline. In addition, all neurons exhibited a sustained response to vibratory stimulus and were silenced by muscle twitch during bipolar muscle stimulation (amplitude 0.1-0.3mA: 50 μs).

Lumbar stiffness in the laminectomy-only condition was 11.51N/mm (range 6.39 to 18.23N/mm). Compared to the laminectomy-only condition, laminectomy-facet fixation increased spinal stiffness 4.02N/mm (range: 1.08 to 7.75N/mm; 12.56% to 69.45%) while laminectomy-facetectomy decreased spinal stiffness –1.18N/mm

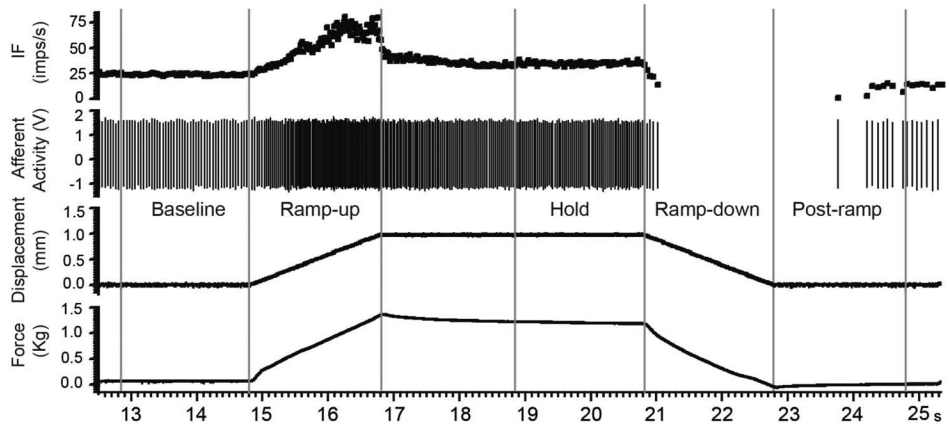

Figure 1.
An example of a 1mm ramp and hold movement of a L6 vertebra in a laminectomy-only preparation. Force, displacement, primary afferent activity and instantaneous frequency recordings are shown. Baseline, ramp-up, hold, ramp-down, and post-ramp regions used to calculate mean instantaneous frequencies are demarcated. Note the increase in afferent activity during the ramp-up and hold phase and the cessation of discharge due to unloading of the muscle spindle during the ramp-down phase.

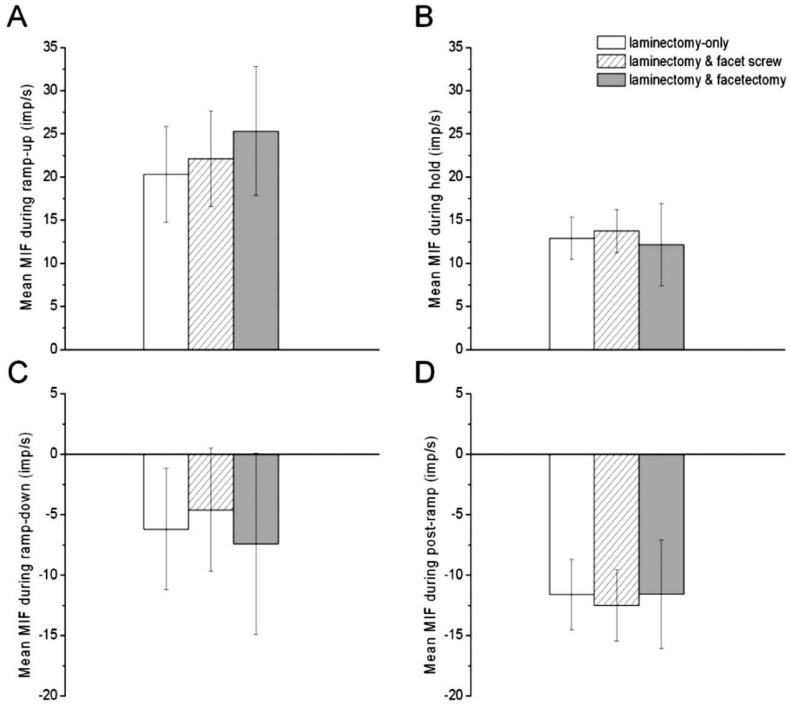

Figure 2.
The mean change in mean instanteous frequency relative to baseline discharge during (A) ramp-up, (B) hold, (C) ramp-down, and (D) post-ramp for laminectomy-only, laminectomy-facet fixation, and lamectomy-facetectomy conditions. There were no significant differences between conditions during any phase of vertebra movement.

(range: –0.69 to –2.26N/mm; –3.25% to –21.09%) as reported previously.[26]

Figure 1 shows an original recording from a muscle spindle with a receptive field in the longissimus muscle during a 1mm ramp and hold experimental protocol in the laminectomy-only condition. There was an increase in neural activity during the ramp-up and hold phases which was typically followed by a cessation of muscle spindle discharge due to spindle unloading and a resumption of resting discharge.

Figure 2 shows the adjusted mean MIF and 95% confidence intervals for each response measure during the ramp and hold movements for each facet joint condition. Mean $MIF_{ramp\text{-}up}$ was not significantly different among the 3 conditions (Fig. 2A; $F_{2,22}$=1.71, p=.20). The adjusted mean difference in $MIF_{ramp\text{-}up}$ between the laminectomy-only and the laminectomy & facet screw condition was 1.82imp/s (-1.61, 11.04) and 4.99imp/s (-1.07, 11.04) between the laminectomy-only condition and the laminectomy & facetectomy condition. Mean MIF_{hold} (Fig. 2B; $F_{2,22}$=0.27, p=.76), $MIF_{ramp\text{-}down}$ (Fig. 2C; $F_{2,22}$=0.56, p=.58) and $MIF_{post\text{-}ramp}$ (Fig. 2D; $F_{2,22}$=0.33, p=.72) were also not significantly different among conditions.

Discussion

The potential for interactive effects between intervertebral joint mobility and sustained changes in sensory signaling from peripheral paraspinal tissues is of fundamental importance to all researchers and clinical practitioners interested in optimizing neuromuscular control of the trunk. Spinal manipulation and/or spinal mobilization are typically delivered to patients at anatomical locations exhibiting signs and symptoms of biomechanical dysfunction.[37-39] The present study is a first step toward investigating the relationship between muscle spindle signaling and acute spinal joint dysfunction during passive movements applied to the lumbar spine.

This study indicated that acute biomechanical dysfunction (laminectomy & facet screw, laminectomy & facetectomy) at a single facet joint failed to alter mechanoreceptive afferent response during slow (0.5mm/s) 1mm dorsal-ventral ramp and hold movements of a lumbar vertebra. These findings mirror results from the recent study investigating the effects spinal manipulation thrust durations under the same spinal joint conditions in which the longest thrust duration (250ms) also failed to demonstrate changes between conditions.[26] Acute spinal joint dysfunction at multiple joints, chronic spinal joint dysfunction, increased vertebral displacement, rotary displacement, and/or a faster ramp rate may be required to affect neuromuscular sensory input from trunk muscle proprioceptors during slow ramp and hold movements and/or longer spinal manipulative thrust durations.

It is interesting to note that in two previous feline studies using the laminectomy-only condition, muscle spindle responses to ramp and hold movements (1, 2, and 3mm; 0.5mm/s), both similar to and greater than the hold amplitude used in the current study (1mm, 0.5mm/s) were not affected by an interposed high velocity low amplitude spinal manipulative thrust;[23] yet the afferents were almost twice as sensitive during the manipulative thrust itself when the peak amplitude was 1mm compared to 2mm.[28] These previous studies along with the present study suggest that mechanoreceptive trunk responses to slow vertebral movements (0.5mm/s) are neither affected by acute single facet joint dysfunction nor by high velocity low amplitude spinal manipulation regardless of ramp amplitude.

Limitations

The present study was limited to the effects of acute spinal joint dysfunction at a single facet joint with all other spinal joints remaining intact. While this was a model investigating the simplest degree of intervertebral joint dysfunction on paraspinal sensory input, a greater degree of joint dysfunction (e.g. involving multiple facet joints and/or the intervertebral disc as often encountered clinically) or the chronic presence of joint dysfunction may be required to affect trunk muscle spindle signaling. There were no differences in mean MIF between the conditions for ramp-up, hold, ramp-down, and post-ramp, but these findings should be confirmed in a powered study with minimal loss of preparations particularly within the laminectomy & facetectomy condition. Additional factors that should be taken into consideration in future studies include: making contact on the paraspinal muscles themselves as opposed to making direct contact with the vertebra itself via forceps attached to the spinous process, a greater degree of vertebra displacement (>1mm), increasing the ramp rate, incorporating a rotary or lateral component to vertebral displacement, chronic spinal joint dysfunction and/or testing in the presence of a musculo-

skeletal inflammatory milieu that frequently accompanies spinal joint dysfunction clinically. One or more of these factors may be required to physiologically affect changes in sensory input from trunk muscle spindles during slower and/or small intervertebral joint movements.

Failure to create a minimum 2% change in lumbar stiffness in a dozen preparations is likely due to a number of factors including but not limited to the greater inherent flexibility of the feline spinal column, inadequate placement of the facet screw, partial splintering of the facet joint, incomplete facetectomy, and/or lack of a rotary or lateral displacement component of the spine during biomechanical testing. Dorsal-ventral ramp testing was the only direction used in current study due to the increased risk of tearing the afferent fiber off the recording electrode that accompanies rotary or lateral movements in this type of experimental preparation.

Neurophysiological and biomechanical studies using anesthetized animals where measurements from the spinal tissues can be obtained directly are of growing importance in the quest to understanding the underlying mechanisms of spinal manipulation despite certain inherent limitations of this work. Since the chiropractic profession's first basic science white paper was published in 1997,[40] much basic work has been accomplished (see [41-44] for review), and yet there remains a great need for more and better animal models if the goal is to identify the biological mechanisms involved in spinal manipulation intervention. Once mechanisms are identified, this knowledge can then be translated into providing better clinical care for individuals seeking chiropractic services. As shown in Table 1, much information relevant to the practice of chiropractic has been learned over a relatively short period using slight variations of the animal model used in the current study.

Conclusion

Coordination of paraspinal muscles is required to provide optimal neuromuscular control of dynamic intervertebral mobility during intended bodily movements. It is possible that distorted proprioceptive input related to acute or chronic spinal joint dysfunction could result in suboptimal neuromuscular trunk control; however, the results of this study indicate that changes in lumbar stiffness due to dysfunction at a single facet joint fails to alter paraspinal muscle spindle responses during slow (0.5mm/s) 1mm ramp and hold movements. Spinal joint dysfunction at multiple joints, chronic joint dysfunction, and/or more rotary/combinatorial motions in a facet dysfunctional model may be necessary to alter responses of trunk spindle afferents during small slow movements of the lumbar spine.

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The origin, and application of somatosensory evoked potentials as a neurophysiological technique to investigate neuroplasticity

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Somatosensory evoked potentionals (SEPs) can be used to elucidate differences in cortical activity associated with a spinal manipulation (SM) intervention. The purpose of this narrative review is to overview the origin and application of SEPs, a neurophysiological technique to investigate neuroplasticity. Summaries of: 1) parameters for SEP generation and waveform recording; 2) SEP peak nomenclature, interpretation and generators; 3) peaks pertaining to tactile information processing (relevant to both chiropractic and other manual therapies); 4) utilization and application of SEPs; 5) SEPs concurrent with an experimental task and at baseline/control/pretest; 6) SEPs pain studies; and 7) SEPs design (pre/post) and neural reorganization/ neuroplasticity; and 8) SEPs and future chiropractic

Les potentiels évoqués somesthésiques (PES) peuvent servir à élucider les différences dans l'activité corticale liée à une manipulation vertébrale (MV). La présente revue narrative a pour objet de donner un aperçu de l'origine et de l'application des PES, une technique neurophysiologique servant à étudier la neuroplasticité. Les sujets suivants feront l'objet de résumés : 1) paramètres pour la génération de PES et l'enregistrement des formes d'ondes; 2) nomenclature, interprétation et générateurs du point maximum de PES; 3) points maximums relatifs au traitement de l'information tactile (pertinent pour la chiropratique et les autres thérapies manuelles); 4) l'utilisation et l'application des PES; 5) PES en même temps qu'une tâche expérimentale et au point de référence/prétest; 6) les PES et les études sur la douleur; 7) conception des PES (pré/post) et réorganisation neuronale/ neuroplasticité; 8) les PES et la recherche future en chiropratique. Comprendre ce que sont les PES ainsi

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research are all reviewed. Understanding what SEPs are, and their application allows chiropractors, educators, and other manual therapists interested in SM to understand the context, and importance of research findings from SM studies that involve SEPs.

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KEY WORDS: somatosensory evoked potential, neuroplasticity, manipulation, chiropractic

que leur application permet aux chiropraticiens, aux éducateurs et aux autres thérapeutes manuels qui s'intéressent à la MV de comprendre le contexte et l'importance des conclusions des recherches sur la MV où l'on a recours aux PES.

(JCCA 2014;58(2):170-183)

MOTS CLÉS : potentiel évoqué somesthésique, neuroplasticité, manipulation, chiropratique

Introduction

Evoking and recording somatosensory evoked potentials (SEPs) is appearing in scientific literature that pertains to spinal manipulation (SM). There is evidence to support that SEPs are a neurophysiological technique capable of elucidating differences in cortical activity associated with an SM intervention.[1,2] Haavik and Murphy[3] hypothesized that appropriate spinal movement normalizes afferent input and restores sensorimotor function and integration by filtering and processing appropriate somatosensory input. The purpose of this manuscript is to provide an overview of the origin, and application of somatosensory evoked potentials as a neurophysiological technique to investigate neuroplasticity. Neuroplasticity is defined as how one's central nervous system adapts to their ever-changing environment. Neuroplastic changes can be subjectively positive for the individual (adaptive) such as learning, or they can be subjectively negative (maladaptive) such as pain.[3] Understanding what the SEPs technique is, and how it has been applied will allow chiropractors, manual therapists and educators with an interest in SM to better understand the context, and importance of research findings from SM studies that involve SEPs as an outcome measure.

The most basic form of electrical communication between cells in the human body is the action potential.[4] A neuron, stimulated by other cells or other external stimuli, will reach a point at which an "all or none" burst of electricity is generated, and propagated. Depending on the type of neuron where this propagation is generated the result will be either inhibitory, or excitatory in nature at the synapse where it terminates. Excitatory post synaptic potentials facilitate action potential generation at the cells upon which they synapse. Such changes in electrical activity occur as a result of positive and negative ions crossing the cellular membrane. The ion flow results in changing regional polarity, and the resulting voltage changes in the area can be measured to demonstrate activity in the brain.

The brain is the site of integration, and perception of all external and internal stimuli, it is the keystone of the central nervous system. The somatosensory system is comprised of elements of the peripheral nervous system and central nervous system that serve the modalities of touch, vibration, temperature, pain and kinesthesia.[5] Neurologically this pathway consists of peripheral receptors and afferent neurons that enter the dorsal root ganglion prior to ascending the spinal cord to the medulla where they synapse with the ipsilateral dorsal column nuclei (Figure 1). Once in the medulla they cross to the contralateral side of the brain (decussate) and the pathway continues to the contralateral ventral posterior lateral nucleus of the thalamus prior arrival at the primary somatosensory cortex for processing.[6] This pathway consists of the dorsal column – medial lemniscal, and thalamo – cortical sensory systems.[7] Knowledge of the anatomical pathway of afferent and subsequently perceptual information can serve as a roadmap to the study of information acquisition and processing.

Origins of Somatosensory Evoked Potentials

An evoked potential occurs when the stimulation of sensory receptors or afferent nerve bundles past their resting threshold results in the generation of a compound action potential. While not mutually exclusive the evocative stimulation can consist of tactile, vibrational, painful or electrical elements.[8] The compound action potential

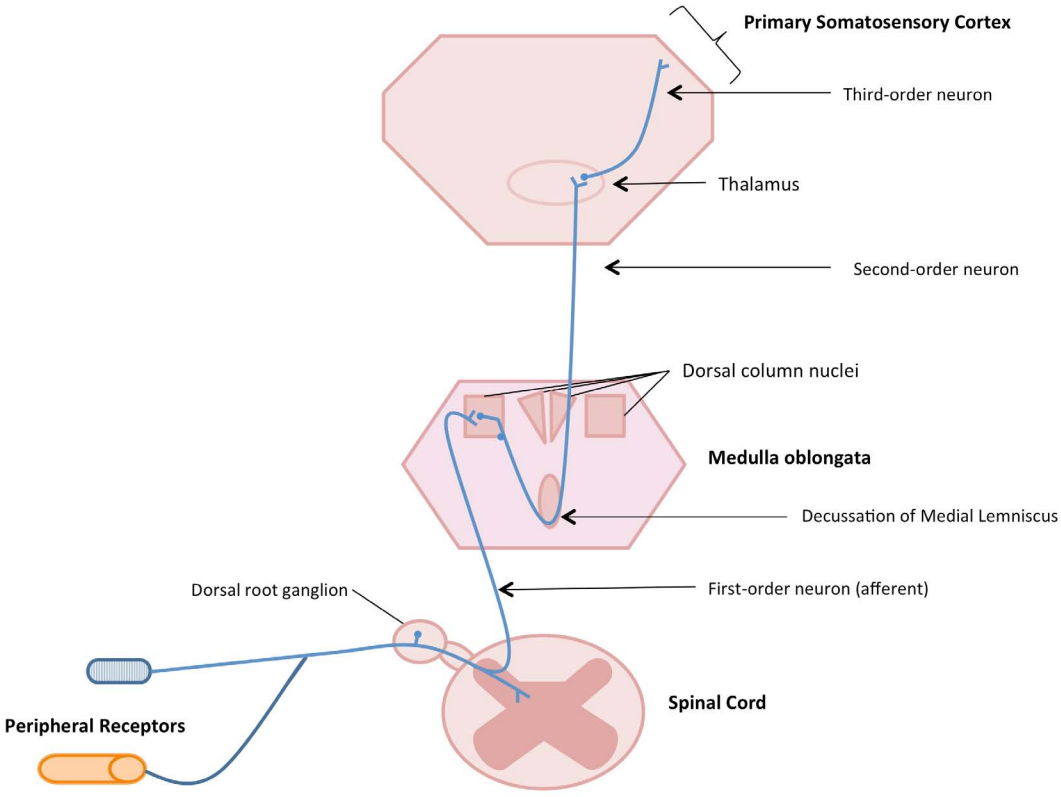

Figure 1.
Dosal column-medial lemniscal pathway.

transmitted can be recorded using electrodes to study the post-stimulus characteristics.[9] Potentials evoked by peripheral nerve stimulation can be recorded in the sensorimotor cortex and multiple other sites along the pathway.[10]

A somatosensory evoked potential (SEP) is the electrical activity response measured at the skin's surface following controlled peripheral nerve stimulation. Electrical activity from peripheral stimulation measured over the scalp reflects cerebral action potentials and are best recorded contralateral to peripheral nerve stimulation.[11] The recorded electrical potential of this afferent volley bombardment generates a complex waveform.[12]

Waveform reproducibility is confirmed by taking the average of several controlled stimuli to waveform generation time-locked trials. The resulting average waveform can then be analysed in terms of the peaks and troughs present at different time points relative to the stimulation. To understand the significance of the waveforms, their components and their neurological interpretation, Giblin[13] observed SEPs in both healthy participants and patients with impairments including lesions of the peripheral nerves, spinal cord, and the brain. He described "early potentials" as those of brief duration that occur within the first 35 msec after stimulation of the median nerve at the wrist. Recorded latency will vary based on the distance from anatomical stimulation site. For example, lower extremity potentials have a slightly longer latency than upper extremity potentials as they have a longer distance to travel. Early potentials were accurately reproducible and Giblin[13] noted the positive and negative voltage changes at particular times in milliseconds.

Early SEP studies had substantial variability in many

facets of technique application. This variability included, but was not limited to: the stimulus intensity and inter-stimulus interval of the peripheral evoked potentials, the impedance and location of recording electrodes, the number of signals recorded to generate an average waveform, the filtering and amplification of recorded signals, and the measurement and recognition of specific peaks. Acknowledging this heterogeneity of method, but the usefulness of this approach to the study of the nervous system, the International Federation of Clinical Neurophysiology (IFCN) generated a report from a committee of recommended standards for short latency somatosensory evoked potentials.[14] The findings from the report have been used in part to generate suggested SEP stimulating and recording parameters as detailed in the following section of this manuscript. A brief overview comparisson of SEPs and other common neurological recording techniques can be found in Table 1.

Parameters for SEP generation & recording of waveforms

Different from electroencephalography (EEG) which reflects the brain's spontaneous electrical activity over a short period of time, SEPs are not recorded continuously to spontaneous stimuli but are time locked to a stimulus with a pretrigger.[15] SEP peak amplitudes are traditionally in the under 10μV range (smaller then EEG [tens of μV], EMG [mV], ECG [V]).[15] The stimulation most favoured is electrical stimulation as it has parameters that are easily manipulated and controlled.[16]

According the updated IFCN guidelines[17] the recommended electrical stimulus should consist of a 0.1-0.2 ms duration square wave pulse. These pulses can be delivered by constant current stimulators applied transcutaneously over the targeted nerve. When stimulating a mixed (motor and sensory fibre containing) nerve, stimulus intensity should exceed the motor threshold for eliciting a muscle twitch. But, the intensity should not be so high as to excite a-delta or c-fibres that are excited by nociceptive input. Gandevia and colleagues[18,19] have demonstrated that muscle afferents most likely dominate the cerebral potentials produced by stimulation of the mixed median nerve at the wrist. IFCN guidelines recommend that the pulse delivery should repeat at a frequency between 3 and 5 Hz. Stimulation frequencies up to 8 Hz can be used for pulse delivery if the latency of a target peak to be measured occurs before 30 ms. After 30 ms peaks resulting from this higher (8hz) frequency of stimulation are subject to reduction or attenuation which is why a bandwidth of 3-5Hz is preferred.[20] Recently however, Haavik and Murphy[21], have demonstrated that stimulation rate may impact early peaks differentially. Specifically, a rate of 5 Hz enhanced the N24 SEP peak amplitude, while a rate of less than 3 Hz was needed to reliably record the N30 peak. Electrodes for stimulation should be placed over the course of the desired nerve, with the cathode placed 2 cm proximal to the anode.[17]

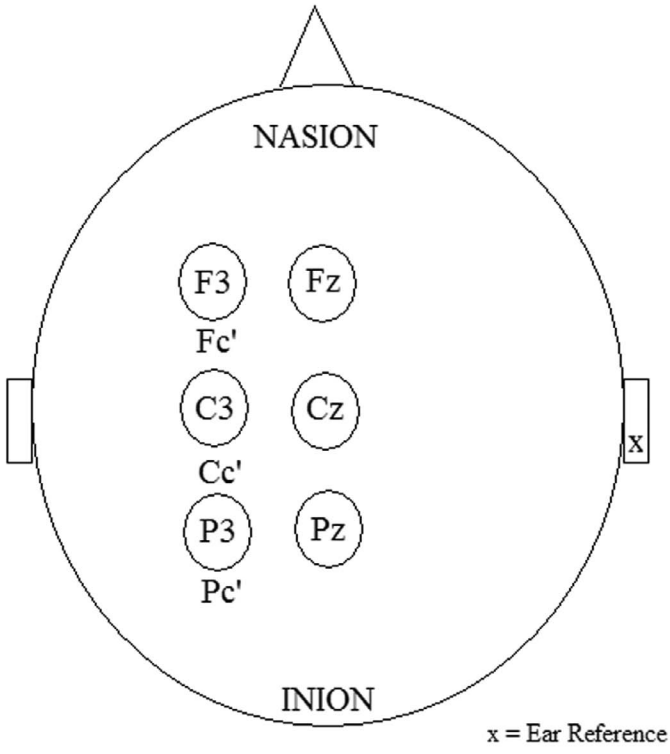

Figure 2.
Relevant 10-20 system electrode placement sites.

To most effectively, and efficiently record SEPs signals to measure the changing activity in the brain and central nervous system, it is recommended that one centimetre surface recording EEG electrodes should be placed as per the 10-20 international EEG system (Figure 2).[15] The cortical locations that should be used contralateral to the site of stimulation are the Fc' (contralateral frontal) and Pc

Table 1.
Neurological imaging and measurement techniques

Technique	Role of Technique	Advantages	Limitations
Positron Emission Tomography (PET)	Three-dimensional functional imaging of a radioactive tracer (injected the body).	Reasonable spatial resolution.	Requires injection of radionucleotide, expensive (require full time staff & radionucleotide cost), restricted movement environment, not portable.
Functional Magnetic Resonance Imaging (fMRI)	Measure three-dimensional changes in cerebral blood flow overlaid on a magnetic resonance imaging brain map.	High spatial resolution.	Expensive (require full time staff), restricted movement environment, not portable. Reliant upon neurovascular coupling, which is the interpretation of the blood oxygen level dependent (BOLD) signal. Low temporal resolution.
Transcranial Magnetic Stimulation (TMS)	Sends a magnetic burst targeted toward a general anatomic region.	Non-invasive, can be used to elicit motor evoked potentials (MEPs) with measurable amplitude and latency using surface electromyography (EMG).	Requires high level of training to deliver, and interpret. Variability can be high depending on relative positions of coil, and participant. Low spatial resolution.
Magneto-encephalography (MEG)	Measures magnetic fields from cerebral sources.	High temporal resolution.	Expensive (require full time staff), requires high level of training to deliver, and interpret, restricted movement environment, not portable.
Electro-encephalography – Surface (EEG)	Records spontaneous electrical activity from the central nervous system relative to a reference electrode.	High temporal resolution, records electrical responses concurrently with presentation of other stimuli.	Requires high level of training to deliver, and interpret. Surface recorded, and not time locked to external pre-cognitive stimuli, spatial resolution limited.
Somatosensory Evoked Potentials – Short Latency (SEPs)	Responses of PNS and CNS to time locked and consistent stimulation producing electrical activity as consistent measureable waveforms that can be averaged for clean interpretation. Measured using surface EEG.	Time locked to stimulus (consistent), early peaks are pre-cognitive in response to stimulation.	Requires high level of training to deliver, and interpret. Surface recorded, spatial resolution limited to peak interpretation.
Event Related Potentials (ERPs)	Brain response directly related to a sensory, motor or cognitive event. Waveforms are averaged by repeated exposure to the "event" or stimuli of interest. Measured using surface EEG.	High temporal resolution (<1 ms)	Poorly defined spatial resolution. Requires high level of training to deliver, and interpret.
Source Localization	Mathematical model for data de-convolution, allows for de-blurring of scalp EEG.	Improves EEG spatial resolution from a centimetre scale on the cortex to a millimetre scale.	Requires high level of level of training to deliver, and interpret
Standardized Weighted Low-Resolution Brain Electromagnetic Tomography (swLORETA)	Mathematical model used to determine the depth of source localization of EEG signals.	Improves EEG and Source Localization interpretation.	Requires high level of level of training to deliver, and interpret.

(contralateral parietal).[17] Skin at the scalp EEG electrodes should have less than 5 Kohms impedance. The number of waveforms that need to be averaged are from between 500 and 2000 stimuli presentations, in order to clearly differentiate the signal from noise. Updated IFCN guidelines[17] recommend a filtering bandwidth with a high pass of 3 Hz and low pass of over 2000 Hz to isolate reproducible waves from background noise. Scalp electrodes may utilize an earlobe reference[17] and a lip placement for the ground electrode[22]. Adherence to these recommendations will allow the optimal uniform technical recording environment to assess the neurophysiological changes associated with behavioural or perceptual experimental interventions and the resulting information processing.

SEP peak nomenclature, interpretation and generators

Waveform peaks are assigned a letter representing their polarity (positive or negative). By convention an upward wave deflection is a negative polarity (N), while a downward deflection is positive (P), and also assigned an integer based on the post stimulus latency (in ms) at which they appear in a healthy population.[17] Both the latency and the amplitude (uV) of these peaks can be used to interpret changes in neural activity. The amplitudes and latencies of the peaks are thought to represent a combination of the peripheral and central nervous system reception of the external stimulus, and the early processing by a given neural structure of that stimulus. Specifically, amplitude represents the magnitude of the incoming afferent volley.[15] Latency reflects the anatomical location along the somatosensory pathway impacted by the peripheral stimulus.[15]

The waveform is a post-stimulation cortical-electrical potential with predictable and reproducible peak and trough amplitudes and latencies based on recording site. The signals recorded are reflective of their neural generators.[17] The neural generator can be "near-field", or anatomically close to the electrode (cortex surface), or "far-field", relatively anatomically distant (subcortical).[6] This means that the near-field potentials represent the direct region of polarity change proximal to the electrode. Far-field potential responses reflect structures with a diffuse signal to a larger area of the surface, they are more likely to be detected at multiple electrode sites.[15]

Early SEP peaks also referred to as "short latency" SEPs are considered to be the most useful for the study of neurological activity as they are the least variable among participants with intact nervous systems free from pathology considered to represent the normal population.[15] Short latency refers to the peaks and troughs present within the first 40 msec following a single stimulation to the upper limb, and less than 50 msec for the lower limb.[23] Peaks of longer latency than 45 ms may be susceptible to cognitive factors, which may further increase their variability.[17]

Identification and meaning associated with specific temporal peaks have been derived from several different methodologies. One methodological example are the techniques used in laboratory obliteration studies which are traditionally performed with animal populations. Severe attenuation or abolishment of all SEPs occurs in primates when the dorsal columns of the upper thoracic, or mid cervical aspects of the spinal cord are ablated.[24] No SEP anomalies occur when there are lesions to other parts of the spinal cord but the dorsal columns are left intact. This finding suggests the dorsal column tracts are essential in the mediation of SEPs.[16] Additionally, SEP peaks have been shown to result mainly from stimulation of large myelinated sensory afferents such as 1a muscle afferents and, possibly, cutaneous afferents.[18,19] The low intensity of stimulation applied, which is just above motor threshold, means that large myelinated sensory afferents which are also the most rapidly conducting afferents are preferentially excited, and reach the cortex prior to other afferent fibres.

The presence of a specific pathology is another factor that impacts SEP peak amplitude, latency or total absence. Peaks may be delayed or absent in pathology cases with an etiology that is degenerative, traumatic or congenital.[16] Degenerative pathologies such as multiple sclerosis (MS)[25,26,27,28], spinal cord tumours affecting the posterior columns[29] amyotrophic lateral sclerosis (ALS), Freidrich's ataxia[30,31] and Guillain-Barré Syndrome[32] will alter SEP waveforms. Traumatic or compressive pathologies including focal nerve lesions[33,34], brachial plexus lesions/nerve root avulsion[35,36,37], meralgia paresthetica[34], or nervous system lesions from a traumatic brain injury (TBI)[38] or surgery[16] are visible in the presence or absence of SEP components. Congenital pathology such as achondroplasia with associated foramen magnum stenosis will yield an abnormal SEP study.[39] SEPs can even be used to conclusively identify brain death. A peak at N13/N14

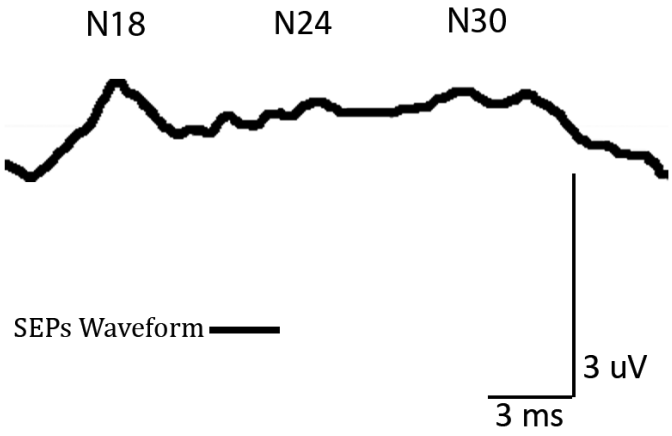

Figure 3.
Typical SEPs waveform.

with no peaks of further latency indicate that signals are reaching the cervical spine close to the medulla, but with no cerebral activity.[16]

It is possible to identify many SEP peaks, their origin, and significance (Figure 3). For the purpose of brevity, this review will focus on the origin of the P14-N18 complex for N18, N20-P27 complex for N20, P22-N24 complex for N24, and P22-N30 complex for N30. These peaks have different possible implications for the study of tactile information processing (N18[40]; N20[41]; N24[42]; N30[43]). Tactile information processing is particularly relevant in the study of chiropractic and other manual therapies. Clinicians use their tactile sense to both assess (ex. joint and muscle palpation) and treat patients. Patients receive tactile input (ex. manipulative thrust, soft tissue mobilization) delivered with therapeutic intent from the clinician.

The N18 Peak

The far field, widespread, N18 component is distinct in SEP traces. It has the broadest elevation from baseline following the P13-14 potential.[44] Mauguiere[15] suggested that there are multiple generators of the N18 scalp-recorded potential. Clinical evidence indicates that the N18 component is generated in the brain stem at the level of the midbrain-pontine region.[45] Such brainstem lesions significantly attenuate the N18 amplitude.[45] Noel, Ozaki and Desmedt[46] suggested that the N18 peak originates in the lower medulla nuclei including the accessory inferior olives and dorsal column nuclei. Noel, Ozaki and Desmedt[46] presented three patients whose N18 component remained intact although they had lesions at the medial lemniscus levels including the midbrain and upper medulla. The finding that N18 is related to the dorsal column nuclei is also supported by Manzano, Negrao and Nobrega[47] who found N18 as the only SEP component resistant to tactile cutaneous vibratory changes. Sonoo, Sakuta, Shimpo, Genba, and Mannen[48], and later Sonoo et al.[49] concluded that the cuneate nucleus was likely responsible for the N18 potential based on several observed cases of patients with deep sensation disturbance and high cervical brain stem, thalamic, and pontine lesions. A review by Sonoo[50] expanded on the mechanism for the N18 peak. He concluded it is likely generated in the cuneate nuclei through primary afferent depolarization. Specifically, by collaterals from dorsal column afferents to cuneate nuclei interneurons that synapse on dorsal column fibers' presynaptic terminals that become depolarized and function as presynaptic inhibition.[50]

The N20 Peak

The primary somatosensory cortex lies in the posterior bank of the rolandic fissure representing Brodmann's area 3b in the parietal lobe. This is the site of N20 peak generation.[51] It is known to respond to contralateral tactile stimuli.[52] The parietal N20 peak is consistent and occurs contralateral to the site of stimulation.[15] Brodmann's area 3b (the primary somatosensory cortex) responds to cutaneous inputs, but not joint movement input. Desmedt and Osaki[53] confirmed this N20 cutaneous response, and not joint movement, in a study on passive finger movement. In healthy normal participants the N20 peak is the earliest cortical processing in the primary somatosensory cortex.

The N24 peak

The origin of peak N24 is located close to the location of N20. N24 is a frontal lobe negativity that appears on the ascending slope of peak N30. Garcia Larrea, Bastuji and Mauguiere[54] found that N24 is best revealed at higher stimulus rates (greater than 3 Hz) that selectively decrease the N30 peak. As discussed previously in this review, Haavik and Murphy[21] have recently shown that 5 Hz stimulation is sufficient to enhance the N24 record-

ing while ensuring detection of changes subsequent to a motor training task. Due to its mild variability in latency the N24 peak has also been referred to as N23[55], or N25[43]. Waberski et al.[43] used source localization to identify to the posterior wall of the central sulcus in area 3b of the somatosensory cortex as the site of N24 generation. In order for this pathway to continue the input sent to the somatosensory cortex travels through the cerebellar cortex and deep cerebellar nuclei.[56] The N24 amplitude is enhanced if the cerebellar cortex is disrupted. N24 is reduced or absent, but all preceding peaks are left intact if the cerebellar cortex and deep cerebellar nuclei are lesioned.[42] The characteristics of N24 are linked directly to the integrity of the cerebellum through its cortex and its deep nuclei. In summary, when a deep structure is lesioned the peak is obliterated, while if only the cortex is disrupted the peak is enhanced. The aforementioned findings provide evidence of the possibility that the deep structures generate increased activity in an attempt to relay signals to the cortex in the event that the cortex is damaged and fails to appropriately received signals.

The N30 Peak

The N30 frontal lobe peak reflects sensory integration.[57] This peak is negatively impacted by imagined or actual voluntary muscle contraction. Cheron and Borenstein[55] demonstrated that both imagined and actual finger movements attenuated the N30 peak. As a result this peak is believed to reflect complex cortical and subcortical loops that link the basal ganglia, thalamus, pre-motor areas, and primary motor cortex.[58-61] Parkinson's disease (PD) is known to degrade components of the basal ganglia, including but not limited to the internal globus pallidus, and the subthalamic nucleus. A PD patient population has demonstrated a decreased N30 peak compared to a control population.[62,63] Muscle tone rigidity decreases and N30 amplitude increases in PD patients when the neuromuscular junction is blocked.[63] Basal ganglia deep brain stimulation also produces increased N30 amplitude, which is attributed to improved supplementary motor area (SMA) activity.[62] Basal ganglia efferents are anatomically found to terminate in the ventrolateral thalamus, from where they project to the SMA.[64,65] Waberski et al.[43] employed a mathematical technique known as "source localisation" to suggest that primary motor cortex or more specifically the pre-central motor cortex is the N30 peak generator. Primate[66,67], and subsequently human[68] intracortical recordings support that N30 is generated at the motor cortex.

The neural generators of the N30 SEP peak have recently been explored using novel technology. Cebolla, Palmero-Soler, and Cheron[69] used swLORETA (standardized weighted Low Resolution Brain Electromagnetic Tomography) and determined that the N30 is generated by network activity in the motor, premotor and prefrontal cortex. This finding sheds light on the role N30 plays as a marker of neural processing relevant to sensorimotor integration. The role of the prefrontal cortex is a finding of particular interest since it is a site of executive function including cognitive planning and decision-making. The prefrontal cortex receives somatosensory input and other internal and external sources of information that can be used to inform decision- making. Clinicians who deliver manual therapies use their tactile sense via palpation of muscles and joints to make clinical decisions.

Utilization and Application of SEPs

Clinical

SEP recording is an objective technique and is often more sensitive than the traditional neurological component of physical examination.[7] For example, SEPs can be used in comatose, anesthetised patients.[7] Interpretation of the presence and absence of specific waveforms can be utilized to predict comatose patient prognosis. When SEPs are recorded within 72 hours of entering the comatose state prediction of prognosis is >99% accurate.[70]

Based on the reliability of SEP peaks, it is increasingly accepted for use in the operating room. Operating room monitoring of SEP peaks is done to correct spinal cord ischaemia, prior to it becoming a debilitating issue. SEPs are used in repetition to continuously monitor for detection of neurological impairment during scoliosis surgery. This technique has resulted in a 50-60% decrease in paraplegia post surgery.[71]

Surface recording electrodes, while relatively non invasive, cause the spatial accuracy of SEP recording to be decreased compared to other direct neuromeasurement techniques. SEPs are regarded as having high temporal and low spatial resolution.[72] The meaningfulness of the interpretation of SEP waveforms is established enough that is has been used as a pre-screening tool for inclusion or exclusion of participants in scientific research. SEPs were

collected prior to selection for experimental inclusion in a traumatic brain injury (TBI) study by Sarno, Erasmu, Lipp and Schlaegel.[38] This technique allowed the reduction and refinement of a pool of participants for a reaction time study. Understanding limitations and performance of a TBI population can otherwise be problematic to test due to the possible heterogeneity of symptoms. Examination of the quality of the N20 peak allowed the exclusion of participants with severe sensory impairment, thus yielding an objective test to produce a more homogenous experimental group. SEPs may be used as a neurophysiological outcome measure when behavioural findings are absent (clinically silent).[16] Whether or not SEPs also have the potential to reveal clinically silent musculoskeletal lesions is an area that requires further research.

SEPs Concurrent with an Experimental Task and at Baseline/Control/Pretest

Buchner et al.[41] measured immediate cortical plasticity related to attention and anesthesia. They first elicited SEPs at base line, then again concurrent with conditions of directed attention. They found that an immediate cortical reorganization occurs at peak N20 when partial deafferentation was present. They used an electrical stimulation attention task on fingers 1, 3 and 5. Temporary deafferentation was achieved via injection of 1.5-2 ml of a 2% Meaverin solution to digits 2-4. They found that when participants were anesthetized directed attention to the dorsal hand increased the accessibility of neighboring cortical areas. Waberski, Gobbele, and Buchner[73] found similar results before and during air puff stimulation of the anesthetised thumb. Cortical representation of the thumb decreased in the presence of anesthesia compared to a preanesthetic condition. They interpreted this finding to indicate that anesthesia yields an immediate cortical reorganization of the representation of the affected and adjacent digits. From a clinical perspective even an acute peripheral injury or sensory perturbation may cause immediate cortical reorganization measurable using SEPs.

Psychophysical literature that pertains to tactile stimulation raises concerns regarding the generation and recording of SEPs concurrent with perception or performance related to another task. It is possible that concurrent SEPs stimulation could negatively impact accurate performance when responding to multiple tactile stimuli, or distractors, leading to unintended masking or enhancement. For example Giblin[13] determined that SEP peaks are attenuated or masked in the presence of additional tactile stimulation meant to be irrelevant to SEPs technique recording. The phenomenon is now known as "sensory gating". Morita, Petersen, and Nielsen[74], cautioned that SEPs gating can occur with concurrent motor activation in the lower extremity.

When designing a movement study with concurrent SEPs recording, experimentors need to be aware that factors leading to gating can result in the decreased amplitude of an expected waveform signal. For example, in as few as 60 ms post contraction tibial nerve SEPs would become attenuated when either foot was plantar or dorsi flexed concurrent with SEPs recording. In as few as 60 ms post contraction tibial nerve SEPs would become attenuated when either foot was plantar or dorsi flexed concurrent with SEPs recording.[74] If such factors are not controlled for the misinterpretation of results is possible.

SEPs Pain Studies

Tinazzi et al.[75] explored the impact of tactile sensory disruption using a passive tactile stimulus (no other cognitive, perceptual or motor intervention), in a within-participant SEPs study. Spinal (N14) and subcortical (N18) peaks remained unchanged. The parietal lobe N20 and frontal lobe N30 cortical SEP amplitudes were increased during anesthetic block of the ipsilateral ulnar nerve. This anesthesia, which the authors termed "transient deafferentation" was induced via injection of a 2% lidocaine solution. The amplitudes differed significantly during anesthesia compared to baseline, and following when anesthesia was worn off. The authors interpreted that increased peak amplitudes reflected increased activity that may be intracortical in origin, specifically in subareas of the somatosensory cortex. Clinicians need to be aware that peripheral changes in sensation, may lead to amplified changes in central (cortical) activity.

Unilateral radicular pain from the C-6 nerve root level demonstrates SEP amplitude differences compared to both the unimpaired side and to healthy controls.[76] Ten participants with a cervical disc protrusion compressing the C-6 nerve root, and ten healthy age matched controls were recruited. SEPs were recorded in a between-limb, and between-participants design. Amplitudes of peaks N13, P14, N20, P27, N30 were all significantly amplified in the limb with the presence of pain. This suggests

that peak enhancement can reflect a positive correlation between the presence of pain and SEP amplitude. Tinazzi et al.[75] concluded that SEPs might be a sensitive neurophysiological tool to investigate physiopathological changes in humans before the appearance of hard neurological (absent reflex, or motor impairment) symptoms. The same experimental design was used earlier to examine a population with EMG evidence of chronic unilateral carpal tunnel syndrome (Tinazzi et al., 1998).[76] Identical to the radiculopathy study, peaks N13, P14, N20, P27 were all increased in amplitude when generated from the pathological limb compared to the healthy limb, and to an asymptomatic healthy age-matched control group. While all pain and function loss in patient participants impacted the median nerve, ulnar nerve stimulation was used to generate and record the SEPs. Based on their finding Tinazzi et al. concluded that changes associated with chronic pain detected by peripheral nerves may cause plastic changes that can be detected in the brainstem prior to reaching the cortex. Limitations to both studies are the inability to completely homogenize the onset, duration, and intensity of the symptoms in the pain-participant population. Future research is needed to explore the possible neurophysiological quantification of unilateral pain. Studies on clinical interventions that decrease self-reported musculoskeletal pain could utilize a pre- post-intervention SEPs design with the predication that peaks will attenuate as pain decreases.

The issue of standardizing pain delivered to participants has been overcome using an experimentally-induced pain model.[77] Rossi et al.[57] built on their foundation to understand how their induced perturbation impacted behavioural, specifically motoric and imagined movement findings in a subsequent study. The induced tonic hand pain using a Levo-Ascorbic solution injection in the first dorsal interosseous muscle. They found that the N18 SEP peak was significantly increased when the pain was present. There was a significant decrease in N30 amplitude when asked to imagine finger movement during the pain condition. The attenuation of N30 was even more pronounced during actual motor recruitment. The strength of this study is the consideration of neurophysiological measurement, and behavioural or imagined movement. A weakness is that no behavioural outcome measures were recorded to quantitatively assess motor task performance.

SEPs Design (Pre and Post) and Neural Reorganization/Neuroplasticity

SEPs when recorded at baseline and compared to SEPs recorded following a separate perceptual, sensory or motor task reflect the neuroplasticity associated with a perceptual[78] or motor task[79]. A pre-test and post-test experimental design can be used to avoid inadvertently masking the tactile system while utilizing the SEPs technique. Pellicciari, Miniussi, Rossini and De Gennaro[78] compared SEP recordings in the elderly and in a young population, pre- and post-exposure to paired-associative stimuli. In their study paired-associative stimuli were the combination of median nerve electrical stimulation, and 20 ms later transcranial magnetic stimulation (TMS) of the S1 region. The 20 ms time delay reflects the time needed for the afferent signal from the Median nerve to arrive at S1. Essentially it is the reason for the N20 latency SEPs peak. The limitation of TMS is that it is not focal to a single structure and is a gross activation or inhibition. While neuroplasticity may take place in both populations with learning, the patterns and underlying structures reflecting plastic changes may differ. This suggests possible compensatory changes to accommodate the abilities of the elderly population. Murphy, Haavik-Taylor, Wilson, Oliphant, and Mathers[79] used pre- and post-task SEPs as a neurophysiological measure for plasticity related to motor output. In a within-participants design 10 individuals had SEPs recorded at baseline, then immediately after a 20-minute repetitive-typing task. Attenuation of the N13 peak, N14-18 complex, and N30-P40 complex all occurred immediately following the typing task. Had Murphy et al.[79] attempted to concurrently record SEPs while performing the typing task, the stimulus intended to be used to stimulate the somatosensory system may have served as an attentional, cognitive, or peripheral perturbation to motor performance that could have masked changes in the targeted SEP peaks. To ensure accurate interpretation, appropriate control groups are an asset to pre- and post-task designs.

Haavik-Taylor and Murphy[1] used a pre- and post-SEPs design to consider plasticity associated with the clinical intervention of spinal manipulation. Prior to the intervention, in a between-participant design, 24 individuals were pseudorandomized to receive either manipulation, or passive head motion. Only the spinal manipulation group yielded a significant attenuation of peaks N20 and N30,

for about 20 minutes post-manipulation. This plasticity effect provides evidence for altered cortical somatosensory processing and sensorimotor integration following spinal manipulation. The authors concluded that their findings may aid in the further study of the understanding of mechanisms for functional restoration and pain relief following spinal manipulation. An understanding at the mechanistic level, would aid clinicians in communicating the clinical significance of their intervention to patients, and colleagues from other healthcare disciplines.

A more recent somatosensory evoked potential (SEP) study investigated patients with a history of reoccurring neck pain or stiffness. SEPs were elicited via 3 methods. First from the median nerve and second from the ulnar nerve. The third method included simultaneous median and ulnar nerve stimulation. The ratios of the individual sum were compared to the dual simulatanous SEPs.[80] In a pre- post-task design participants had baseline SEPs recordings, performed a thumb tapping task on a single key for 20-minutes at a rate of 180 strikes per minute, then had post-task recordings. There was a significant increase in the dual SEP ratio for the N20-P25 complex, and the P22-N30 SEP cortical SEP components after a 20-minute motor task. However this increase did not occur when the motor training task was preceded with spinal manipulation. Spinal manipulation prior to the motor training task actually caused a significant decrease in the dual SEP ratio for the P22-N30 SEP component, most likely due to changes in the ability to appropriately filter somatosensory information at the cortical level.

SEPs and Future Chiropractic Research

The future usefulness of SEPs for the chiropractor or other manual therapists can be viewed from 2 distinct vantage points. First, SEPs can by used to measure if changes are present in the patient pre- compared to post-intervention. Hypothetically, a patient with concussive symptoms of mechanical origin may demonstrate central changes associated with a course of chiropractic intervention. A patient with a peripheral nerve entrapment, may yield changes in peripheral, central, or a combination of regions following a course of care compared to baseline. There is precedent for using a pre- post- intervention SEPs design with a clinical population. For example as mentioned in the previous section Haavik Taylor and Murphy recorded SEPs on a population with neck pain[80], they have also previously studied SEPs in patients with reoccuring neck stiffness[1], and pain-free people with a history of cervical spine issues[2] pre- and post-spinal manipulation. In a recent review regarding their work related to SEPs and spinal manipulation, Haavik and Murphy hypothesize that spinal manipulation leads to appropriate joint movement, which in turn yields normal afferent input allowing for appropriate somatosensory processing and integration to occur.[3] Second, SEPs could in the future also be used to measure if there are changes in the clinician, either: a) with learning the motor skill of spinal manipulation delivery; or b) if the clinician suffers an injury or pathology but is still trying to deliver manual therapies. When measuring changes in the clinician it would be most useful to use SEPs in tandem with a behavioural performance measure (reaction or movement time, and with kinetic or kinematic data) in order to determine if there is a correlation between behavioural and neurophysiological measures. The addition of behavioural measures allows for the intrepretation of not just the neurological regions impacted by clinical intervention, but also the functional performance differences that are possible.

Conclusion

Somatosensory evoked potential recording has been established as a meaningful neurophysiological measurement technique in both clinical and research contexts. Specific parameters for eliciting and measuring SEPs have been created as recommendations for uniform testing conditions. Obliteration and pathology studies have allowed understanding of the significance and origin of several peaks. Changes in activity resulting in peak latency and amplitude modulation allow the visualization and quantification of precognitive neural plasticity associated with perceptual, cognitive, and motor tasks or phenomena. SEPs have also been used to show changes with both transient and chronic pain, and changes following spinal manipulation. Future studies should extend the work on altered sensory input, including pain, joint dysfunction, paresthesia, as well as their interaction with motor training and sensory perception.

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The cervical myodural bridge, a review of literature and clinical implications

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The role of posterior cervical musculature in sensorimotor control, cervicocephalic pain, and stabilization of the spinal cord has been recently described. Anatomical soft tissue connections which cross the cervical epidural space link suboccipital muscle fascia and dura. These myodural bridges provide passive and active anchoring of the spinal cord. They may also be involved in a dural tension monitoring system to prevent dural infolding, and maintain patency of the spinal cord. Modulation of dural tension may be initiated via a sensory reflex to muscular contractile

Le rôle de la musculature cervicale postérieure dans le contrôle sensorimoteur, la douleur cervico-céphalique et la stabilisation de la moelle épinière n'a que récemment fait l'objet d'une description. Les connexions anatomiques des tissus mous qui traversent l'espace épidural cervical lient le fascia et la dure-mère des muscles sous-occipitaux. Ces ponts myoduraux offrent un point d'ancrage passif et actif à la moelle épinière. Ils peuvent aussi participer au système de contrôle de la tension durale afin de prévenir le repliement dural et de maintenir la perméabilité de la moelle épinière. Les modulations de la tension durale peuvent être provoquées par un réflexe sensoriel aux tissus musculaires contractiles. Les mouvements non anticipés

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Institutions where work was performed: Cadaveric tissue samples were obtained at the Department of Anatomical Sciences at Logan University and the Department of Anatomical Sciences at St. George's University, School of Medicine. Histological analysis of tissues was performed at the Research Microscopy Core in the Department of Pathology at Saint Louis University School of Medicine.

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tissues. Unanticipated movements such as hyperflexion extension injuries stimulate deep suboccipital muscles and transmit tensile forces through the bridge to the cervical dura. Due to its larger cross sectional area, the rectus capitis posterior major myodural bridge may exert greater mechanical traction on the dura than the rectus capitis posterior minor. University ethics committee approval and anatomical donor consent was obtained for this study.

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KEY WORDS: rectus capitis posterior major, obliquus capitis inferior, myodural bridge, dura mater

comme les blessures résultant d'une hyperflexion-extension stimulent les muscles sous-occipitaux profonds et transmettent des efforts de traction par le pont sur la dure-mère cervicale. En raison de sa plus grande section transversale, le pont myodural grand droit postérieur peut exercer une plus grande traction mécanique sur la dure-mère que le muscle petit droit postérieur. L'approbation du comité d'éthique de l'université et le consentement du donneur anatomique ont été obtenus pour la présente étude.

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MOTS CLÉS : grand droit postérieur, oblique inférieur de la tête, pont myodural, dure-mère, chiropratique

Introduction

The cervical spine is a complex anatomical structure that is of interest to anatomists, biomechanists, and clinicians. Soft tissue communications linking suboccipital muscle fascia and the dura and its role in cervical neuromuscular control have been examined recently.[1-8] These myodural bridges have been associated with the etiology of cervicocephalic headaches, and cervicocephalic pain syndromes.[3,9] These epidural connections may also be involved passively as a dural anchor and as an active stabilizer of the spinal cord.[4,6,10]

Recent studies have described myodural communications bridging the epidural spaces between the rectus capitis posterior minor (RCPmi), rectus capitis posterior major (RCPma), and obliquus capitis inferior (OCI) suboccipital muscles and the dura mater of the cervical spine.[1-8] Anatomical studies by Khan, Hack, Scali, Pontell, and others have reported on the presence of myodural bridges linking sub-occipital muscles with the dura mater of the cervical spine.[1,2,4,6] Additional studies by Shinomiya, Humphreys, Nash, Zumpano and Tagil confirmed these findings.[13,10-13] Connections between the suboccipital musculature fascia and cervical dura mater have implications in cervicocephalic pain syndromes, sensorimotor function, and postural control.[14-17] The clinical relevance of these cervical epidural membranes and their relationship to cervicogenic and tension headache syndromes has been discussed by multiple authors including Bates, Schoenen, Haldeman, Fernandez-De-Las-Penas and others.[9,14-17] Changes in cervical proprioception, balance, sympathetic tone, conversion of muscle type, and dural enfolding secondary to cervical spine injuries have been described by several authors including Palmgren, Rix, Uhlig, Cailliet, Lusczyk.[18-25,]

Anatomical research and reviews of existing literature concerning the fascial connections between the suboccipital muscles and the cervical dura have been reported recently.[1-8] In 1992 a fascial connection between was briefly mentioned by Kahn et al., in a report of the posterior intervertebral spaces of the cervical craniovertebral joint.[1] It was noted however, that a true membrane connecting the posterior arch of the atlas to the laminae of the axis did not exist, but rather two fibrous planes that transected this space.[1] Hack et al, reported on a myodural bridge between the fascia of the rectus capitis posterior minor (RCPmi) muscles in 1995.[2,26] An examination of the posterior atlantooccipital interspace showed a continuous band of tissue with fibers oriented primarily perpendicular to the muscle and dura.[2,26] A animal model study of 43 specimens in 1996 by Shinomiya et al. examined the ligamentous attachments within the cervical posterior epidural space.[10] They confirmed the presence of abundant posterior epidural ligaments attaching to the posterior dura mater.[10] Dissections of seven human cadaveric specimens by Rutten et al. in 1997, agreed with previous findings of an epidural connection between the RCPmi and the pos-

terior wall of the spinal cord dura mater.[24] Alix and Bates reported on previous anatomical findings and suggested that the ligamentous connection between muscle and the pain sensitive dura mater may provide an anatomic and physiological basis for cervicogenic headaches.[9] After a gross anatomical dissection of 30 cadaveric specimens in 2003, followed by magnetic resonance imaging (MRI) of 4 specimens, Humphreys' et al, also confirmed the presence of a fibrous connection bridging the epidural space linking the dura and RCPmi.[13] In a case report describing an anatomical variation of a bifurcated RCPmi discovered during a routine dissection in 2005, Tagil confirmed the presence of dense connective tissue linking suboccipital muscle fascia to the cervical dura mater.[3] Nash et al, employed sheet plastinations and confocal microscopy in an examination of the posterior atlanto-occipital interspace.[12] They noted the presence of connective tissue attaching the RCPmi fascia and the spinal dura mater in the posterior cranio-cervical region in adult human cadavers.[12] A larger study of seventy five cadavers by Zumpano et al. in 2006, examined variations in prevalence, tissue-type, gender in the soft-tissue bridge between RCPmi and dura mater.[11] They reported similar findings in the structure of the RCPmi myodural bridge as previous authors.[27] According to Kahkeshani and Ward, the myodural bridge has been underreported due the time necessary to properly dissect this region.[27] To encourage further study; they describe a method for the deep dissection of the suboccipital triangle which preserves the RCPmi and its attachments for further inspection.[27]

We recently reported on two additional epidural connections to the cervical dura.[4-7] Myodural bridges extending from the anterior fascia of the rectus capitis posterior major (RCPma) and obliquus capitis inferior (OCI) muscles, attaching on the cervical dura mater was documented recently by Scali, and Pontell in 2011 and 2013 (fig1).[4-7] In an anatomical study by Scali et al., 13 embalmed human cadaveric specimens showed broad fascial connections traversing the cervical epidural space from the RCPma muscle fascia, anterio-inferiorly to the posterior dural surface (fig 2).[4] RCPma myodural tissues from 11 specimens stained with hematoxylin and eosin indicated that the fascial connection inserted directly into the fascia surrounding the RCPma and attached to the posterior surface of the dura (fig 3).[5] Immunohistochemical analysis using anti-neurofilament protein fluorescent antibody staining showed a pattern of nerve distribution throughout one tissue sample (fig 4).[5] Pontell et al. dissected nine human cadaveric specimens, examining 14 OCI muscles and surrounding tissue.[6] We documented a continuous fibrous tissue originating at the anterior fascia of the OCI muscle belly and projecting anteriorly across the atlantoaxial interspace and attaching to the posterolateral aspect of the cervical dura mater between the first and second cervical vertebrae.[6] Histological analysis was performed on 12 OCI suboccipital muscles, connective tissue, and dura mater from human cadavers between the ages of 49 to 81.[7] Microscopic examination of OCI myodural tissues stained with hematoxylin and eosin showed the connective tissue emanating from the ventral OCI muscular fascia and inserted directly into the posterolateral aspect of the cervical dura mater.[7] A single OCI myodural connection stained for immuno-peroxidase using Dako's neurofilament protein monoclonal antibodies revealed fascicles traveling perpendicular, and parallel with the OCI myodural bridge.[7] Due to proximity, the RCPma and OCI muscles appear to form a single atlantoaxial myodural bridge, they are however, separate structures.[7] We were unable to find a similar connection between the obliquus capitis superior and the dura mater.[7] To examine the prevalence of these structures, T-2 weighted magnetic resonance imaging (MRI) of the atlanto-axial interspace of 240 individuals was performed in 2012 by Scali et, al.[8] Sixty four percent of the MRI's reviewed demonstrated a posterior concavity of the cervical dura mater consistent with a ligament attachment site.[8] Of this group, 24% also had oblique, linear hypointense fibers which appeared to attach to the cervical dura mater.[8] The breadth of studies which have examined these soft tissue dural connections, precludes them being considered variations of normal anatomy.[1-8,11-13,27]

In this article, we focus on the anatomical, functional significance, and clinical relevance of the RCPma and OCI myodural bridge. Consideration of its function as a passive and active stabilizer of the cervical spinal cord is discussed.

Anatomy of the suboccipital muscles

The RCPma and OCI muscles share a common innervation by the suboccipital nerve and some actions, with the RCPma acting to extend and slightly ipsilateral rotate the head and neck, while the OCI muscle ipsilaterally rotators

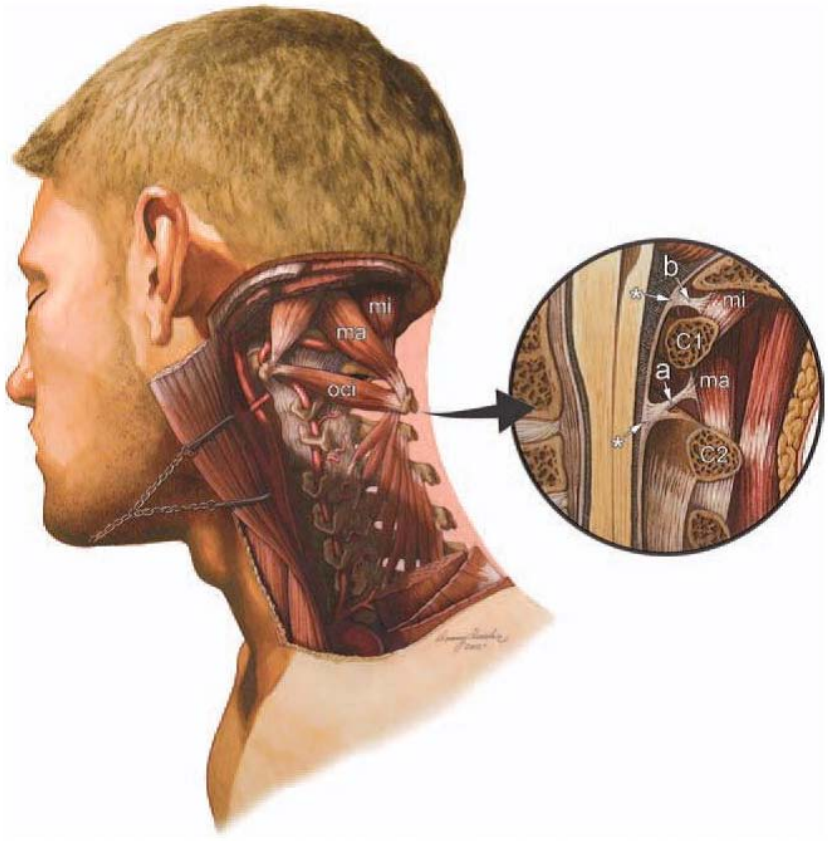

Figure 1
Illustration of a dissection of the deep suboccipital region of the cervical spine. The rectus capitis posterior minor (RCPmi), rectus capitis posterior major (RCPma), and the obliquis capitis inferior (OCI) muscle fascia have communications with the dura mater via soft tissue. The encircled illustration (right) depicts a midsagittal dissection revealing the RCPma, RCPmi, and OCI muscles. The cervical myodural bridge (a) traverses the epidural space between the posterior elements of the C1 and C2 vertebrae. Both myodural structures link the suboccipital muscle fascia in to the cervical dura mater (*).Used with permission from: Magnetic resonance imaging investigation of the atlanto-axial interspace. Clin Anat. 2013 May; 26(4):444-9. Scali et al. (Original anatomical artwork by Frank Scali, D.C., and Danny Quirk)

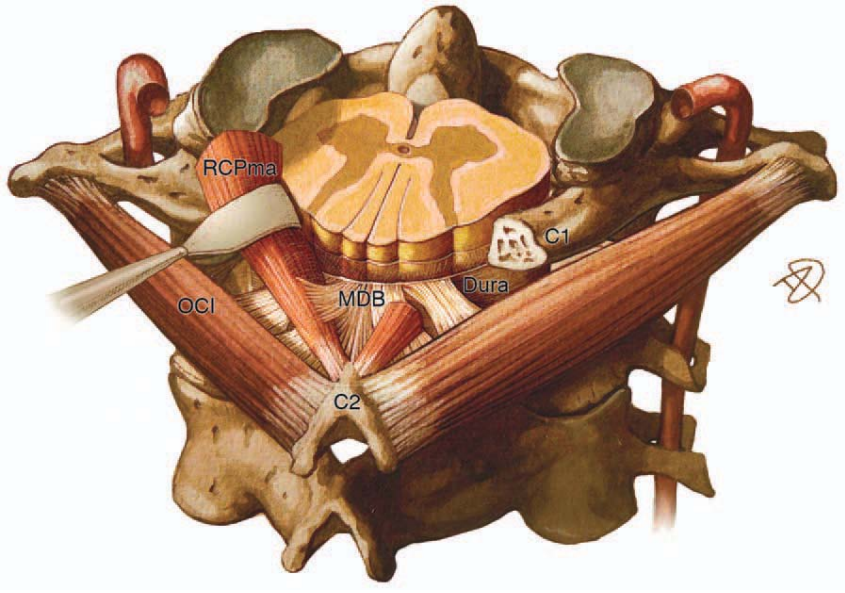

Figure 2
A myodural bridge (MDB) extending from the anterior fascia of the rectus capitis posterior major (RCPma) and obliquus capitis inferior (OCI) muscles, attaching on the cervical dura mater. The myodural bridge (MDB) communicates with the posterior aspect of the cervical dura mater between the C1 and C2 vertebrae. Used with permission from: The Obliquis Capitis Inferior Myodural Bridge, Clin Anat 2013 26:450 45. Pontell M, Scali F, Marshall E, Enix D. (Original anatomical artwork by Danny Quirk)

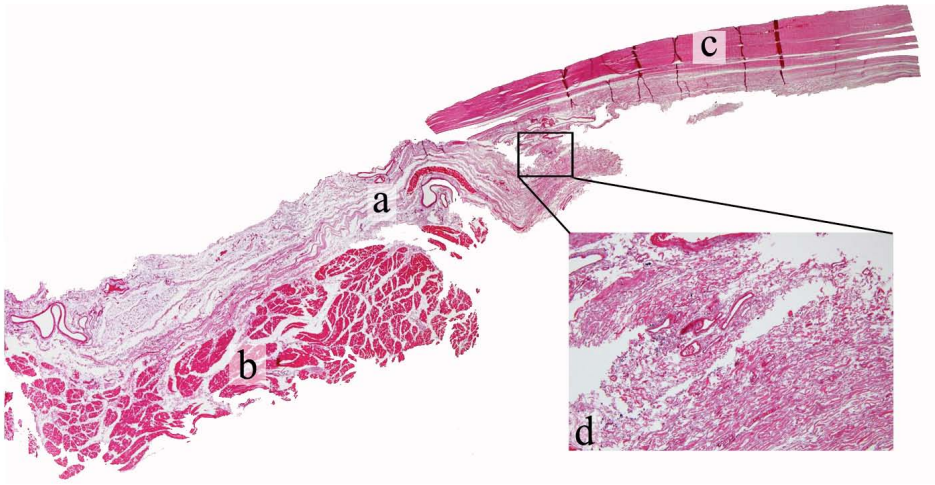

Figure 3

Hematoxylin and eosin stained tissues of left side sagittal section showing the soft tissue communication (a) between the RCPma (b) and the cervical dura mater (c) in a male cadaveric specimen. The magnified area shows the soft tissue communication at the point of contact with the dura mater (d). Used with permission from: Histological Analysis of the Connection between the Rectus Capitis Posterior Major's myodural bridge, The Spine Journal 13 (2013) 558-563., Scali F, Pontell M, Enix D, Marshall E.

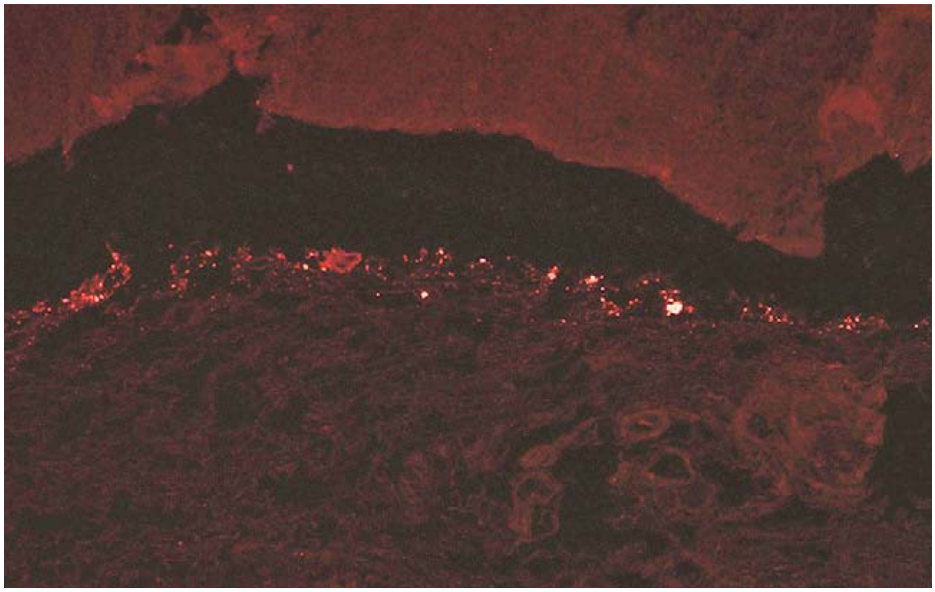

Figure 4

Sagittal section of the myodural bridge between the rectus capitis posterior major and the cervical dura mater depicting positive fluorescence after staining with antineurofilament protein antibodies. Used with permission from: Histological Analysis of the Connection between the Rectus Capitis Posterior Major's myodural bridge, The Spine Journal 13 (2013) 558-563., Scali F, Pontell M, Enix D, Marshall E.

the cervical spine.[3,4,22] Cervical suboccipital muscles are richly innervated and contain relatively high muscle spindle content.[18,21] Muscle spindle fibers found in the RCPma and OCI muscles are a source of primary afferents, representing major contributors to cervical spine neuromuscular control.[3,18,19,28,29] Consistent with their function of complex coordination and organization, high muscle spindle concentrations are typically found in smaller muscle groups responsible for fine motor skills.[18,29,30] Kulkarni et al. documented the density of muscle spindles in fetal tissue per gram of muscle tissue.[18] A large number of muscle spindles were noted in both the OCI and RCPma suboccipital muscles, with 242 and 98 spindles per gram of muscle tissue, compared to the trapezius and latissimus dorsi postural muscles with 2.2 and 1.4 muscle spindles per gram.[18] Muscle spindles are typically more concentrated in regions richest in slow fibers, and while the OCI muscles have been reported as a blend of unevenly dispersed type I and II fibers, their muscle spindles are distributed disproportionately in the deep areas richest in slow twitch fibers.[18,29] This unique configuration of muscle types suggests that these muscles may serve multiple functions including monitoring kinesthetic changes, maintaining constant force for eccentric head posture, and creating fast phasic movements when needed.[18,29,31] An electromyographic examination showed the RCPmi to be under active contraction while the head is in an upright neutral position, with muscle activity increasing significantly during cervical retraction.[32] The cervical muscles are subject to conversion from slow twitch to fast twitch muscles with injury.[20,21] Alteration in muscle type, to a more glycogenic morphology creates a muscle prone to facilitation.[21,31] This change can alter the discharge of primary afferents to the central nervous system effecting cervical neuromuscular control.[19-21,31] Additionally, a loss of proprioceptive inhibition of nociceptors at the dorsal horn of the spinal cord can result in chronic pain.[3,25]

Clinical implications

The spinal canals' midsagittal diameter of 10 mm which increases with flexion and decreases with extension, can impact the patency of the cervical cord.[22,25] In a study of 19 cadaveric specimens by Hong et. al., showed significant differences in dural thickness between different levels in the thoracic and lumbar spines, with the dura slightly thicker in men than women.[33] Buckling of cervical ligaments and dura mater have been reported with cervical spine extension.[22-25,34] Cervical extension can create infolding of the ligamentum flavuum, which encroaches upon the cord.[30] It is the elastin fibers found in the in the ligamentum flavuum that function to inhibit this inward buckling of the ligament into the spinal cord.[22,24,34,35] The dura mater, which is densely populated with nociceptors, also contains elastin fibers which are oriented in such a way as to resist the load placed on them.[34,35] The strain on the posterior side being greater than on the anterior side.[25,34,35] Our recent histological analysis of cervical dura elastin fibers in eight cadaveric specimens, confirms that elastin density changes from caudal to cranial as well as the orientation of fibers which run parallel and perpendicular to each other. Cervical extension may therefore also cause inward buckling of the dura itself, compromising the dorsal subarachnoid space.[31,34,36] In a prospective analysis of fifty patients receiving cervical injections under myelography; six percent demonstrated dural infolding on cervical extension, narrowing the posterior subarachnoid space.[36] Traumatic and iatrogenic tears to the cervical dura can effect up to 36% of cervical spine injuries.[23] Inflammation of the meninges, subdural hematomas, epidural infections, and nerve root compression and postural headaches are all the sequel of cerebral spinal fluid leaks with dural tears.[23,25] Meningeal vascular irritation can also cause hypertonicity of the posterior neck muscles, resulting in permanent tension on the dura, stimulating nociceptive dural fibers.[23,26,37] Considering the close proximity of the leptomeninges to the dura mater, a system to maintain the integrity of the subarachnoid space and, cerebrospinal fluid flow may exist.[14,15,25,28] Similar to the denticulate ligaments which secure the spinal cord within the subarachnoid space, the myodural bridge crosses the epidural space to anchor the spinal dura during head and neck motion.[4,6,10,23,25,28]

Neuromuscular stabilization of the spinal cord

Contraction of the RCPma, RCPmi, and OCI sub-occipital muscles which puts the myodural bridge under tension, transmitting forces across it to place the dura under tension and stabilize the spinal cord.[4,6,26,38,39] In addition to active contraction, the suboccipital muscles respond reflexively to involuntary and unanticipated movements of the head and neck.[20,25] Modulation of dural tension may also be initiated via a sensory reflex to muscular contractile

tissues.[5,7] Central nuclei that exert control over the deep suboccipital muscles, including the RCPma, RCPmi and OCI muscles could respond reflexively as a feedback control of dural tension.[5,7,25,28] Like many systems involving a feedforward or feedback mechanism of control, dural tension regulated through muscular contraction of suboccipital muscles is dependent on internal and external factors affecting those systems.[4,6,28,31] The regulation of tension across the myodural bridge as a spinal cord stabilizer may prevent dural infolding, reducing stimulation of nociceptive pain mechanisms.[5,7,39] Hypertrophy of suboccipital muscles or a failure of this system to maintain constant tension, may result in clinical manifestations arising from increased dural tension.[1,4,6,9,20]

Discussion

Functional anatomy of the myodural bridges

Many authors have speculated on the functional significance of the myodural bridge, generally attributing a mechanical advantage to it in stabilizing the spinal cord from dural enfolding.[1-13] The reflexive myotatic response of suboccipital muscles has been proposed by several authors as a likely mechanism to place the dura under tension.[4-9,38,39] In a study of 20 cadavers, Nakagawa reported that cervical cephalocaudal stresses may be due to the parallel orientation of elastin fibers in the dura and concluded its function was to resist hyperextension and compressive infolding of the dura.[34] Hack suggested that the purpose of the RCPmi myodural bridge might be to assist in resisting dural infolding, previously noted by Burt.[2,36] Shinomiya et. al., concluded that the role of the posterior cervical epidural ligaments is to provide an anchor to stabilize the dura mater from anterior translation during flexion.[10] Without a posterior epidural attachment, the dural canal can shift anteriorly compressing the spinal cord causing flexion myelopathy.[10] Rutten reported on high muscle spindle content in the RCPmi, postulating that the myodural bridges function may to monitor stresses on the cervical dura mater, reflexively preventing infolding.[24] They reported that tissue injury from cervical whiplash could affect mechanoreceptive properties, causing the monitoring system that maintains dural tension to fail.[21,24,31] Alix & Bates also discussed the duras tendency to fold inward on the spinal cord, and the myodural bridges ability to resist this movement.[9] McPartland and Brodeur proposed that the RCPmi plays a role in preventing dural crimping when the head is extended or moved backward, inhibiting normal circulation of cerebrospinal fluid.[38] Humphries reported on previous studies describing the primary mechanical function of the RCPmi to resist dural buckling during cervical extension, preventing damage to the spinal cord.[13] In a case report describing an anatomical variation of the suboccipital muscles, Tagil also noted that the spinal cord is believed to be protected by the dense connective tissue that links the suboccipital muscles to the cervical dura mater.[3] Kahkeshani and Ward indicated that a direct connection linking the musculoskeletal system to the dura mater provides a mechanical explanation for the efficacy of cervical massage and manipulative treatment for headaches.[27]

Along with the description of the RCPma myodural bridge by Scali et al., we proposed that modulation of cervical dural tension may include factors other than a myotatic reflex.[4,5] Myodural biofeedback may play a role in maintaining the integrity of the subarachnoid space.[4,5] We noted during dissection that manual traction applied to the RCPma caused movement of the spinal root within several levels.[4] Due to its larger cross sectional area, the rectus capitis posterior major myodural bridge may exert greater mechanical traction on the dura than the rectus capitis posterior minor.[4] We described another soft tissue connection traversing the epidural space between the OCI muscle fascia and the posterior sleeve of the dura mater in a paper by Pontell et al.[6] It was reported that the OCI myodural connections function dynamically to prevent dural infolding during cervical extension, similar to the RCPmi and RCPma bridges.[6,7]

We agree with previous authors who describe a stabilizing function of the RCPmi muscle myodural bridge and propose a similar role for the RCPma and OCI muscle myodural bridges. In addition to the passive anchoring of the dura described by Nakagawa, Hack, Humphries, and Shinomiya, a myo-reflexive response described by Rutten provides an active stabilizing component.[2,7,10,13,19,34,28,34] Besides the reflexive myotatic response of the suboccipital muscles, the presence of neuronal fibers in these tissues, may suggest functions other than the passive anchoring of these muscles to the posterolateral dura mater.[5,7]

The clinical relevance of these soft tissue connections to dural generated pain mechanisms, cervicocephalic headaches, subarachnoid space and cord impingement

make this an area of great interest.[9,13-17,20,39] The anatomical and histological evidence of these soft tissue communications bridging the epidural space from the RCPma and OCI muscles to the dura mater offers insight into the cervical spines' complex function of neuromuscular control.[5,7,18,39] Further examination of the tensile forces in the myodural bridge is needed. Biomechanical testing to confirm the tensile forces on these tissues currently in progress.

Conclusion

Anatomical soft tissue bridges which cross the cervical epidural space, connecting suboccipital muscle fascia and dura have passive and active functions to anchor the spinal cord. These myodural bridges may be involved in a dural tension monitoring system to prevent dural infolding and maintain patency of the spinal cord. Failure of this system could result in altered cerebral spinal fluid flow, changes in sensorimotor function, cervicocephalic headaches, and dural related pathologies.

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Real-time force feedback during flexion-distraction procedure for low back pain: A pilot study

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A form of chiropractic procedure known as Cox flexion-distraction is used by chiropractors to treat low back pain. Patient lies face down on a specially designed table having a stationery thoracic support and a moveable caudal support for the legs. The Doctor of Chiropractic (DC) holds a manual contact applying forces over the posterior lumbar spine and press down on the moving leg support to create traction effects in the lumbar spine. This paper reports on the development of real-time feedback on the applied forces during the application of the flexion-distraction procedure. In this pilot study we measured the forces applied by experienced DCs as well as novice DCs in using this procedure. After a brief training with real-time feedback

Une forme de procédure chiropratique connue sous le nom de flexion-distraction Cox est employée par les chiropraticiens dans le traitement de la lombalgie. Le patient se couche sur le ventre sur une table spécialement conçue, qui comporte un support thoracique stationnaire et un support caudal mobile pour les jambes. Le docteur en chiropratique (DC) maintient un contact manuel en appliquant une force sur la colonne lombaire postérieure, et appuie sur le support mobile pour les jambes afin de créer un effet de traction dans la colonne lombaire. Le présent article se veut un rapport sur le développement d'une rétroaction en temps réel au sujet des forces appliquées au cours de l'utilisation de la procédure de flexion-distraction. Dans cette étude pilote, nous avons mesuré les forces appliquées par des DC ayant de l'expérience et des DC débutants pendant l'application de cette procédure.

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Conflicts of Interest: Dr. Cox is the developer of this technique, he teaches these procedures to practicing chiropractors, and is a paid consultant to Haven Innovations Inc.

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novice DCs have improved on the magnitude of the applied forces. This real-time feedback technology is promising to do systematic studies in training DCs during the application of this procedure.

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KEY WORDS: Cox, flexion-distraction, technique, real-time, chiropractic

Après une brève formation avec rétroaction en temps réel, les DC débutants s'étaient améliorés relativement à la magnitude des forces appliquées. Cette technologie de rétroaction en temps réel est prometteuse pour la réalisation d'études systématiques sur la formation des DC durant l'application de cette procédure.

(JCCA 2014; 58(2):193-200)

MOTS CLÉS : Cox, flexion-distraction, technique, temps réel, chiropratique

Introduction

Musculoskeletal conditions are common causes of pain and disability with low back pain representing a prevalent complaint and costly societal burden.[1-7] Doctors of chiropractic (DCs) treat low back pain patients to relieve discomfort and improve function. DCs may deliver several types of chiropractic adjustments or spinal manipulation therapy (SMT) to the spine for the treatment of musculoskeletal (MSK) conditions. SMT includes manual high velocity low amplitude spinal manipulative (HVLA-SM) procedures, handheld instrument assisted techniques, low-velocity distraction procedures, drop piece high-velocity techniques.[8]

Chiropractic students traditionally learn the technique of delivering SMT procedures by observing someone skilled in a procedure. The expert teacher demonstrates a technique and the student then practices its delivery on other students or volunteer patients. The teacher observes the student performing a manual procedure and provide hands-over-hands guidance, and provide verbal feedback as the student develops proficiency. Experienced DCs provide training in a similar manner with student interns in clinical situations. Triano et al. have reviewed on the training methods used in the literature.[9]

Chiropractic techniques are measurable biomechanical events involving the application of forces to specific regions of interest, causing vertebral movements.[10-13] Several investigators have measured the forces delivered by DCs during manipulations of the lumbar, thoracic and cervical spine.[14-20] HVLA-SM is characterized by clinical force delivery, loading durations, loading rates, coordination index, and transmitted loads to the spine.

Over the past decade, educators have incorporated innovative bioengineering technologies into the training of chiropractic students and licensed doctors to give feedback on the forces, durations, loading rates, and coordination indexes. Mechanical instruments, mannequins, and human volunteers were used for training. Subsequently, researchers have demonstrated quantified force-time profile characteristics.[16,21-25] Most of these studies focused on HVLA-SM, with the majority evaluating the thoracic and lumbar spine.[16;21-24] Few studies have measured the biomechanical characteristics of HVLA-SM delivery to the cervical spine[24,25], and few studies on these parameters with mobilization procedures[26-29].

James Cox, DC developed manual distraction, or the flexion distraction procedure, to treat patients with spinal problems.[30,31] Several case reports, case series, and a randomized clinical trial have been published for treating neck and low back pain problems using this procedure.[32-38] During Cox Flexion-Distraction procedure, the patient lies face down on a specially designed chiropractic table. The DC gently moves the caudal section of the table while holding a broad manual contact over the posterior part of the low back with a vertebral level selected, with an intent to create traction effects in the lumbar spine.

This paper reports on the development of real-time force feedback at the Palmer Center for Chiropractic Research, which provides clinicians with real time visual graphical feedback on the magnitude of forces at the

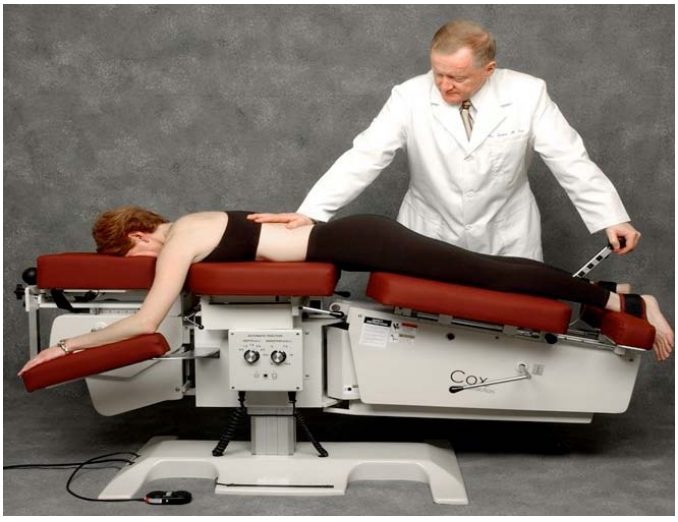

Figure 1.
Cox Flexion-Distraction Table with hand contacts for treating low back.

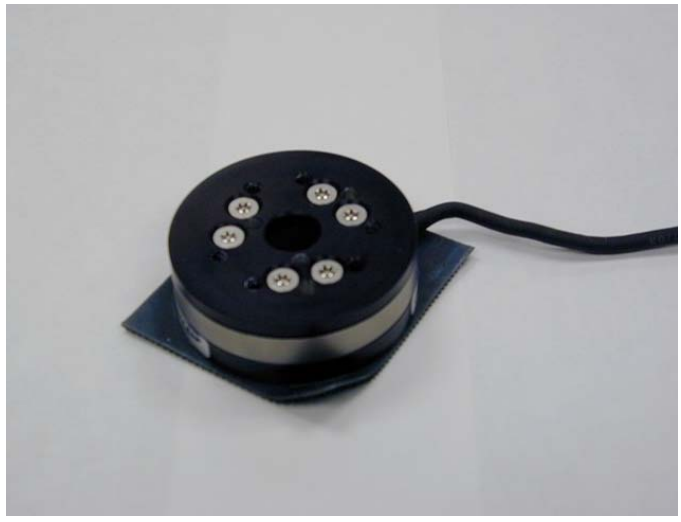

Figure 2.
Three Dimensional Force Transducer used in the study along with rubber padding.

contact hand of the DC on the participant's lumbar spine. This novel training tool was used to collect pilot data while Cox Flexion-Distraction was applied to simulated asymptomatic volunteers by experienced DCs as well as novice DCs.

Methods

The Palmer College of Chiropractic (PCC) institutional review board approved this study. Human simulated patient volunteers and the doctors of chiropractic volunteers signed written informed consent to participate in the study.

Recruitment

Four asymptomatic volunteers (2 male and 2 female age range 22-52 years old) served as simulated patients, recruited from the doctors attending a Cox certification course. DCs screened volunteers for any contraindications and safety considerations relative to receiving the Cox flexion-distraction procedure before study inclusion. Five experienced (>15 years experience in using flexion distraction procedure) DCs and 5 Novice DCs (<1 year experience in using flexion-distraction procedure) participated in the measurement of force delivery.

Force Transducer and Force Feedback Software

During the Cox Flexion-distraction procedure the DC contacts the posterior aspect of the lumbar spine using one hand and applies downward motion of the caudal section of the table where the ankles are cuffed to the table. DCs apply posterior-to-anterior forces (PAF) as well as inferior-to-superior forces (ISF) at the stabilizing hand contact on the posterior aspect of the lumbar spine. Figure 1 shows the table, the patient in a prone position, and the hand contacts. A three dimensional force transducer (Model # Mini-45, ATI-Industrial Automation, Apex, NC) was used to measure the three dimensional forces applied by the DC at the lumbar spine contact. Figure 2 shows the force transducer and the negative Fz axis is directed in the posterior-to-anterior direction of the patient, positive Fx axis is directed along the inferior-to-superior-direction of the volunteer participant. A rubber padding is placed between the patient and the transducer. The measurement of forces is achieved with the help of a three-dimensional force transducer, amplifiers, analog-to-digital converters, laptop portable computer, and custom written Labview software. A custom written software provides the graphical visual feedback in real time as a function of time during the delivery of

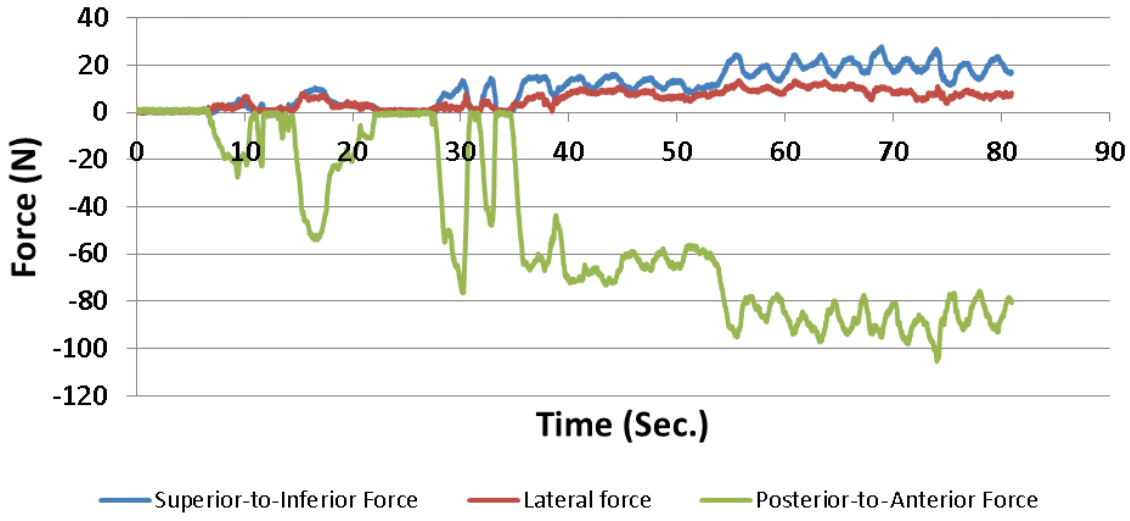

Figure 3.
A typical Force time graph displayed by the computer and the clinician's ability to alter the forces

the treatment. Figure 3 shows force-time graph with the possibility to change the applied force while delivering the treatment (visual real-time graphical feedback). The software was written in Labview (Version 7, National Instruments, Austin, TX). The data is collected at a sampling rate of 100Hz. Magnitude of forces in the inferior-to-superior direction and posterior-to-anterior direction at the hand contact can be simultaneously incorporated into the training.

We have independently tested the force transducer measures (Model: Mini45, ATI industrial Automation, Apex, NC) against a 3-D force plate (Model 4060NC, Bertec Corporation, Columxbus, OH) (20) in both normal and shear directions and found good agreement (less than 3% difference). During Cox flexion-distraction procedure for treating low back, forces are delivered in a gentle slow manner at a rate of approximately 0.5 Hz. Cox flexion distraction for low back pain is a form of low velocity variable amplitude spinal manipulation (LVVA SM). The procedure is performed with a participant lying prone on a specially designed table with a fixed section of the table under the trunk, and a moveable caudal section that allows guided flexion and traction movement in the lumbar spine. The clinician gently grasps the posterior aspect of the participant's back with a thenar contact (contact hand) at a specific vertebral level. With the opposite hand, the clinician grasps the control handle of the moving piece near the ankles. Using the contact hand, the clinician exhibits traction while attempting to maintain a contact at a single vertebral level and ensuring a gentle movement of the caudal section via contact with the control handle. The goal is to create a slow rhythmic distractive movement.

Figure 1 shows a manual contact used by DCs while performing the low back pain procedure. Because low back stiffness and lumbar spine anatomy differ between patients, force-feedback training provides clinicians an opportunity to perceive and gauge force magnitudes on different body types.

We have collected the data from five experienced clinicians with 15, 17, 21, 26, and 35 years experience in using the flexion-distraction technique and five novice clinicians (less than one year experience) in using this technique. Novice clinicians were given a brief training approximately 5 minutes while practicing using this force transducer and the real-time visual graphical feedback. After brief training we measured the forces of the five novice clinicians while delivering on asymptomatic volunteers.

The data is exported into an excel file and then to a custom written MathCad software program (version12,

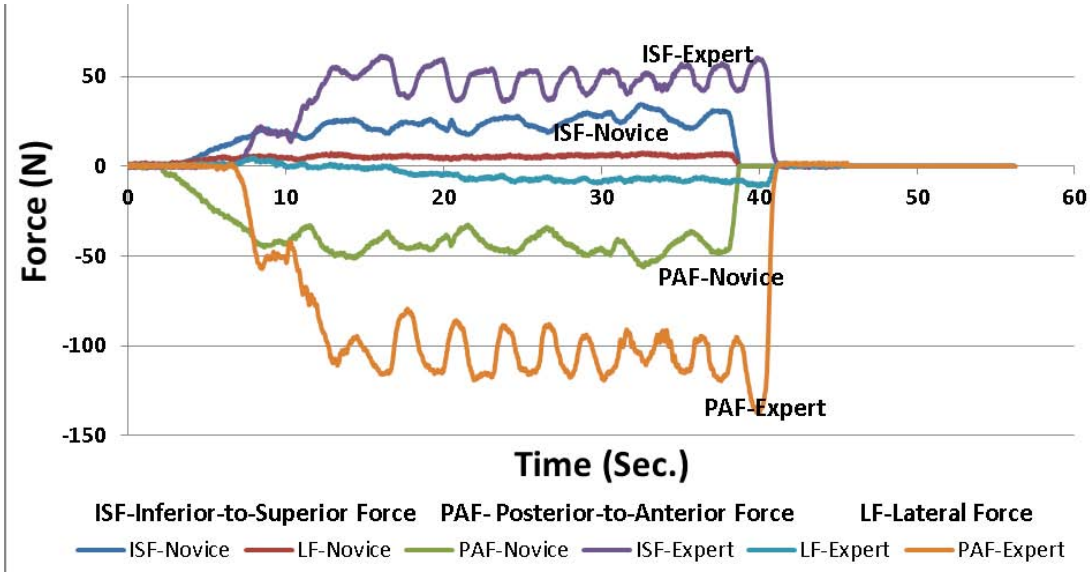

Figure 4.
A typical force time graph of experienced and novice Doctors of Chiropractic

Parametric Technologies, Natick, MA). The magnitudes of forces corresponding to the preload and peak force are extracted and averaged for each doctor over five cycles. The averages for the experienced and the novice doctors are averaged and compared descriptively.

Results

Participants who received the lumbar Cox flexion-distraction procedure consisted of 2 males and 2 females (total of 4 participants). The mean age was 45 years old (SD: 12). The mean height of participants was 172.8cm (SD: 7.7cm) and mean weight was 79.6kg (SD: 22.0kg).

Five experienced field clinician DCs (2 males and 3 females) with a wide range of clinical experience (15-35 years experience using flexion-distraction procedure) performed the lumbar flexion-distraction on all the four participants. This provided a reference data for comparison. Five recently graduated DCs with less than one year experience using flexion-distraction procedure (3 male and 2 female) participated in training. Figure 4 shows the graphical data on the forces used by a typical experienced DC as well as a novice DC. Table 1 provides the forces comparing the experienced and novice doctors. Table 2 provides the data on the novice doctors before and after a brief training using the software developed for training.

Discussion

To the best of these authors' knowledge, this is the first investigation in developing real-time force feedback and visual graphical display to deliver Cox flexion-distraction for lumbar spine. This real-time force feedback provides a foundation to monitor clinician force delivery and train clinicians to alter the delivery of force ranges. This real-time force feedback developed in this study is portable and could be easily implemented in classrooms, teaching clinics, and field settings.

This is a pilot study in collecting data on experienced and novice DCs using flexion-distraction procedure. Forces applied by experienced DCs are higher compared to the novice DCs. After a brief training of 5 minutes the force magnitudes have improved in preload as well as peak forces for the novice doctors. This improvement was observed for both posterior-to-anterior forces as well as inferior-to-superior forces.

Traditional approaches to technique training for Cox-flexion distraction have included observation and feed-

Table 1.
Descriptive values of Forces by experienced and novice Doctors of Chiropractic

Variable	Novice DCs (N=5) Mean (SD)	Experienced DCs (N=5) Mean (SD)
Inferior-to-Superior Forces		
Pre-load (N)	19 (6)	44 (16)
Peak Force (N)	41 (12)	65 (10)
Posterior-to-Anterior Forces		
Pre-load (N)	46 (27)	95 (34)
Peak Force (N)	86 (45)	140 (43)

N-Newtons

Table 2.
Descriptive comparison of forces of novice Doctors of Chiropractic before and after training

Variable	Before Training (N=5) Mean (SD)	After Training (N=5) Mean (SD)
Inferior-to-Superior Forces		
Pre-load (N)	19 (6)	31 (12)
Peak Force (N)	41 (12)	52 (12)
Posterior-to-Anterior Forces		
Pre-load (N)	46 (27)	69 (30)
Peak Force (N)	86 (45)	102 (43)

N-Newtons

back by an instructor/mentor. This method is based primarily on the subjective evaluation of distraction technique as a complex psychomotor skill rather than measuring the biomechanical event. The real-time visual graphical feedback of forces developed in this project extends this subjective evaluation process by providing real-time quantitative force data. As seen in Figure 3 one can notice the improvement of the application of forces during training. Initially the novice DC was applying light forces with no pre-load, gradually improving on the magnitude of the pre-load as well as peak forces by using the real-time visual graphical feedback on the computer monitor. This allows clinicians and students the opportunity to hone in their ability to deliver specific biomechanical forces. Peer and participant feedback/debriefing, delivered verbally, remained an essential component of clinician training.

Other investigators have used training instruments and instrumented mannequins to obtain visual feedback on forces and force-time profiles[16-22] during HVLA-SM, comparing force-time characteristics of students and clinicians. Our study is different from these studies in two ways: a) our study is based on real time graphical feedback while delivering treatment on human volunteers and b) for delivering a low velocity procedure such as flexion distraction and the DC can vary the treatment forces during the delivery with visual graphical feedback similar to the study reported on posterior to anterior mobilization forces on cervical spine[29].

Manual therapists apply forces to the spine for several reasons including improving joint mobility, reducing muscular hypertonicity, stimulating proprioceptive activity, and to relieve pain.[26] Force-magnitude related therapeutic effects have not been studied, but this technology will also allow to train clinicians to deliver treatment within specified force values. Applying treatment within specific force ranges can be a first step toward developing clinical studies designed to investigate optimum force-dosage in clinical settings. This will also allow clinical/physiological outcomes evaluation of patients as a function of different force ranges as an intervention.

Limitations

This study with a small sample size is not designed to test the differences between experienced DCs and novice DCs. Neither the study is designed to test the training process using a control group. This study is designed to provide real-time visual graphical feedback. This real-time force feedback could be used to design and conduct control studies to evaluate training and proficiency of novice DCs, and chiropractic students. The improvement in the delivery of the forces could be related to immediate learning effect. Considering this possibility, future studies should be undertaken to quantify the retention of this training procedure.

Conclusions

Real-time visual graphical feedback was developed and used to train novice DCs to change the force magnitudes applied during flexion-distraction procedure. This technology has the potential to design and undertake well de-

signed studies in training and assessing the delivery of forces during flexion-distraction procedure. The system developed in this study is portable with a laptop computer and can be easily implemented in any field clinician's office.

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