The effects of whiplash-associated disorders on the kinematic and the electromyographic responses of individuals submitted to anterior surface translations in the sitting position

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January, 2007

A thesis submitted to McGill University in partial fulfilment
of the requirements of the degree of

Master of Science

Your file Votre référence
ISBN: 978-0-494-32768-5
Our file Notre référence
ISBN: 978-0-494-32768-5

CONTRIBUTIONS OF AUTHORS

Julie N. Côté, the candidate's supervisor and principal investigator of this research program, developed this research project and was involved in all decisions about the elaboration of the research protocol, the data collection and the data analysis. She was also implicated in the revision of both research articles.

Nancy St-Onge and the candidate were also involved in the elaboration of the research protocol. They participated to all data collection sessions and analyzed the data. Nancy St-Onge wrote the first research article with the help of the candidate who constructed the figures. The candidate wrote the second research article.

Joyce Fung is the director of the Research Centre of the Jewish Rehabilitation Hospital. She made the laboratory available for this research project and lent the equipment. She also helped in the elaboration of the protocol by making precious suggestions.

ACKNOWLEDGEMENTS

First and foremost, I would like to thank my supervisor Julie Côté. You believed in me from the onset of this project, and I thank you for your continued confidence in me, your availability, your encouragements and assurance, and your support, especially during this sometimes challenging task of manuscript composition. I would also like to thank Nancy St-Onge, another important member of "team whiplash". Thank you for your patience while teaching me all that I needed to know about data collection and analysis, and thank you for helping me recognize all that I have achieved since the beginning of this endeavor.

I can't forget to acknowledge those members of the research centre, each of whom helped me in their own way. Thank you to Caroline Paquette for sharing your knowledge and for your incredible ability to find a solution to just about any problem! Thank you to Richard Preuss for lending me your analysis programs and for your availability as a physical therapist during data collection sessions. Thank you to the other physical therapists who participated in this project: Nicoleta Bugnariu, Adriana Venturini, Elizabeth Dannenbaum, Michael Trivino and Petko Baltov. Also, thank you to Vira Rose, Eric Johnstone, Gevorg Chilingaryan and Leslie-Ann Stewart for each of your contributions. And thank you to Joyce Fung, the director of the research centre who generously shared her expertise in order to advance this project. This project could never have been realized without the assistance of team PÉDIP. Thank you to all the clinicians for your help with the recruitment of participants.

Of course I must send a big thank-you to the members of my family. Louise, Marcel, Sylvain, Julie and Guillaume, you have encouraged me unconditionally since I embarked on this journey. Each of you has been there for me when I needed you. I could never have made it this far without your love and support. Thank you. I would also like to send a special thank you to Joseph Bourgeois, Mario Houle, Lucie Lemelin, Andrée Dumouchel and Annie Tremblay. With you by my side, I have grown and developed far more than I could on my own. My experiences with you have been invaluable. Finally, I would like to thank all the people who agreed to participate in this project. Without you, this project would not exist.

ABSTRACT

The goal of this Master's project was to characterize the postural control patterns of individuals with chronic whiplash-associated disorders and to compare these patterns with those of healthy individuals. The postural reactions in response to low-intensity translations of the sitting position were assessed by way of kinematic and electromyographic analyses. We found that whiplash individuals display an earlier onset of their head displacement and a pattern of trunk displacement characterized by greater flexion at the upper levels of the spine, compared to the lower levels. Moreover, whiplash individuals present a tendency for a late recruitment of their neck flexors and for a greater use of a pattern of neck extensor muscle inhibition. These results suggest that individuals with whiplash-associated disorders may compensate their altered neck functional ability by modifying their relative movements along the spine and by adopting altered motor strategies to compensate for their painful muscles.

RÉSUMÉ

Le but de ce projet de maîtrise était de caractériser les mécanismes de contrôle postural chez des individus ayant des troubles associés à l'entorse cervicale. Les réactions posturales en réponse à des translations de faible intensité de la position assise ont été évaluées à l'aide d'analyses cinématiques et électromyographiques. Nos résultats indiquent que les individus pathologiques bougent la tête plus tôt et bougent le tronc avec plus de flexion aux niveaux supérieurs en comparaison des niveaux inférieurs. Aussi, les individus pathologiques ont tendance à recruter plus tardivement les fléchisseurs de cou et à utiliser davantage un patron d'inhibition au niveau des extenseurs de cou. Ces résultats suggèrent que les individus ayant des troubles associés à l'entorse cervicale compensent les habiletés fonctionnelles atteintes au niveau de cou en modifiant les mouvements interarticulaires le long de la colonne vertébrale et en adoptant des patrons moteurs qui compensent pour les muscles douloureux.

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INTRODUCTION

A whiplash is a trauma typically caused by acceleration-deceleration forces applied to the neck, usually during a motor vehicle collision (Cassidy et al. 2000). These forces cause a sudden movement of neck extension and flexion and result in bone or soft tissue injuries in the neck region (whiplash injuries) (Spitzer et al. 1995). In Quebec, whiplash injuries have been reported to be the most common type of injury for which claims are submitted to the Société de l'assurance automobile du Québec (SAAQ). These in turn represent an incidence of 70 per 100 000 inhabitants and annual costs of $18 millions in reimbursement and compensation. Moreover, whiplash injuries present a significant social and economic burden due to the considerable number of persons who develop chronic symptoms from their whiplash accident. As such, the SAAQ reported that 12.5% of patients were still compensated 6 months after the car accident, accounting for 46% of the total cost paid by the SAAQ (Spitzer et al. 1995).

Whiplash injuries may lead to a wide variety of clinical manifestations, termed whiplash-associated disorders (WAD), which are thought to be responsible for the development of chronicity associated with the whiplash accident. Barnsley et al. (1994) have reported that 14% to 42% of patients with whiplash injuries will develop chronic neck pain and approximately 10% will suffer constant, severe pain. More recently, Eck et al. (2001) have indicated that between 4% and 42% of patients with accident-related neck injuries report symptoms several years later. Despite several studies conducted into the general area of whiplash during the last decade, the mechanisms of whiplash and how it influences the systems involved in postural and motor control are not well understood. The literature suggests that the motor deficits associated with WAD can be due to an imbalance of postural central commands (Chester 1991) resulting from an impairment of the neck proprioceptive mechanisms (Gimse et al. 1997; Treleaven et al. 2003) or of the vestibular system (Rubin et al. 1995). The most often reported symptoms of WAD are neck pain, headache, visual disturbances and dizziness (Barnsley et al. 1994; Bogduk 1986; Eck et al. 2001; Spitzer et al. 1995). The symptoms of dizziness reported after a whiplash trauma suggest that postural control might be an aspect affected by a whiplash.

One's posture can be defined as the overall position of the body and limbs relative to one another and their orientation in space (Ghez 1991). For example, standing, sitting and lying are characteristic postures adopted by humans. Balance is the dynamic ability to maintain these postures so as to prevent falling. The visual, vestibular and somatosensory systems are the three sensory systems involved in postural control. In turn, these systems insure that appropriate corrections translate into postural adjustments and coordinated motor patterns that will successfully maintain the projection of the body's center of mass within the limit of the base of support (Ghez 1991, Winter 1995). One research area of postural control that has been importantly developed over recent decades is that conducted in conditions that pose a challenge to postural stability. As such, many protocols have been developed in which subjects were submitted to external mechanical perturbations. One way to induce an external perturbation on one's system is to suddenly move the support surface on which the subject stands or sits. The analysis of the kinematic and muscular patterns produced in response to support surface perturbations has contributed to a better understanding of the strategies adopted by the central nervous system to maintain a stable posture and avoid falls.

Although most of the studies using the postural perturbation paradigm have documented the postural stabilization mechanisms of healthy subjects, much less is known about the way that these are affected by pathologies, especially in the case of the seated posture. In particular, such postural perturbation protocols have never been included in studies of postural stabilization mechanisms of individuals with chronic WAD. Therefore, the general purpose of this Master's thesis was to characterize balance and postural control patterns of individuals with chronic WAD and to compare these patterns with those of healthy individuals. More specifically, we wanted to characterize postural reactions in response to anteroposterior translations in the sitting position by way of kinematic and electromyographic analyses.

In order to measure postural stabilization characteristics of individuals with WAD, we first had to select an appropriate perturbation that would be safe for average participants

of this pathological group while still eliciting a muscular response that could be quantified using our biomechanical analysis systems. For this reason, we began this investigation by first testing several intensities of perturbations on healthy subjects, in the forward and backward directions. Using data collected in this preliminary exploration, we could identify a perturbation of small intensity which produced observable and stereotypical postural reactions in the healthy population. This work is presented in the first research article (short paper), of which the candidate is co-author.

The second and main part of this Master's project was the application of the previously chosen perturbation to individuals with WAD and the comparison of their kinematic and muscular responses with those of a matched group of healthy subjects. The whiplash participants were recruited at the Programme d'évaluation, de développement et d'insertion professionnelle (PÉDIP), a clinical return to work program of the Jewish Rehabilitation Hospital in Laval. The results of this protocol are presented in the second research article (full-length paper), of which the candidate is first author.

REVIEW OF THE LITERATURE

Whiplash

Definitions and incidence

H.E Crowe was the first to use the term "whiplash" in 1928 to describe injuries to the cervical spine which could be attributable to external forces, exerting a "lashlike" effect. In 1995, the Quebec Task Force (QTF) on Whiplash-Associated Disorders (WAD) redefined the term and differentiated the mechanism, the injury and the various clinical manifestations of whiplash. They adopted the following definitions: "Whiplash is an acceleration-deceleration mechanism of energy transfer to the neck. It may result from rear-end or side-impact motor vehicle collisions, but can also occur during diving or other mishaps. The impact may result in bony or soft-tissue injuries (whiplash injury), which in turn may lead to a variety of clinical manifestations (WAD)" (Spitzer et al. 1995).

The incidence of whiplash differs depending on the country investigated. Barnsley et al. (1994) reported an incidence of whiplash injuries of 0.44 per 1000 inhabitants in Switzerland, 2 per 1000 in Norway, 1 per 1000 in Australia and 0.1 per 1000 in New Zealand. Using this data derived from insurance or compensation claim statistics, they estimated that the incidence of whiplash injury is approximately of 1 per 1000 inhabitants in western societies. In Quebec, the population-based incidence of compensated whiplash injury has been reported to be 0.7 per 1000 inhabitants in 1987 (Spitzer et al. 1995).

Chronicity and prognosis

Most patients who suffer from a whiplash injury will recover within approximately eight weeks (Suissa et al. 2006). However, a portion of patients will develop chronic neck symptoms that will linger beyond this duration. That proportion of patients varies in the literature. Chester (1991) reported that 10% of occupants in a rear-end car collision will develop a "whiplash syndrome." In their clinical review, Barnsley et al. (1994)

approximated that between 14% and 42% of patients will develop chronic neck pain and 10% will remain in a state of constant severe, pain. In a more recent review, Eck et al. (2001) reported similar results, that is, between 4% and 42% of patients with accident-related neck injuries still display symptoms several years after the accident.

In their review of the literature on whiplash, the QTF studied the area of prognosis and prognostic factors related to whiplash (Spitzer et al. 1995). According to them, the percentage of patients recovered after six months to a year varies depending on the study reviewed. They also reported studies that highlighted the initial neck pain intensity, the time of onset of neck pain, depression and well-being scores as prognostic factors for the persistence of headaches after a whiplash. In another study, the presence of musculoskeletal (decreased range of motion or point tenderness) or neurological (decreased or absence of deep tendon reflexes, weakness, sensory deficits) signs within three days following a motor vehicle accident were predictive of outcome six or more months later. In addition, older age was associated with persistence of symptoms.

More recently, Côté et al. (2001) updated the review on prognosis of whiplash injuries performed by the QTF in 1995. They found little consistency or conflicting evidence about the course of acute whiplash in patients, whether they were recruited from hospital-based emergency department, primary care medical practice or from the general population. They confirmed older age and baseline neck pain intensity as prognostic factors of delayed recovery. They also found that female gender, baseline headache intensity and baseline radicular signs and symptoms were associated with longer recovery.

In another systematic review of prospective cohort studies, Scholten-Peeters et al. (2003) analyzed the level of evidence for prognostic factors in whiplash recovery. Unlike Côté et al. (2001) who limited their review to articles in English, they included articles in English, German, French and Dutch. Their analysis of the studies selected also confirmed that high initial pain intensity was an adverse prognostic factor. However, they found strong evidence for no prognostic value for older age, female gender, high acute

psychological response, angular deformity of the neck, rear-end collision and compensation scheme.

In summary, the literature suggests that most patients effectively recover from their whiplash injuries. However, a significant proportion develops chronic and disabling symptoms that persist from months to years post-accident. Many studies were undertaken to identify prognostic factors of poor functional recovery. However there still remains some inconsistency in the literature. Although strong evidence points to the prognostic value of high initial pain intensity, many other factors have shown little to no evidence as prognostic indicators of the chronicity of WAD. As a result, much remains unknown about the causes of chronicity related to this prevalent pathology.

Injury mechanism

Most whiplash injuries result from rear-end impacts, the injured persons being in a stationary vehicle that is struck from behind. A clearly defined sequence of events follows the collision. First, the vehicle is accelerated forward and approximately 100 ms later, the person's torso and shoulders are also accelerated forward by the car seat. Because of its inertia, the head remains behind which results in a forced extension of the neck. Then, the inertia of the head is overcome and the head is thrown forward in flexion with the neck acting as a lever (Barnsley et al. 1994). This causes the head to accelerate at about twice the acceleration of the car (Magnusson et al. 1999).

In vitro studies performed on fresh cadaveric human cervical spines have allowed to more precisely describe the movements undergone by the cervical spine during a rear-end whiplash impact. Among the first to use whole cervical spines, Grauer et al. (1997) and Panjabi et al. (1998) have shown that when the lower cervical spine has reached its maximal extension (50 ms – 75 ms on Figure 1), the upper cervical spine is in flexion, this being for all the intensities of rear-end impacts tested. In other words, "the spine consistently exhibits an S-shaped curvature at the time of maximal lower level extension" (Grauer et al. 1997). Afterwards, the cervical spine moves in full extension (100 ms – 125

ms on Figure 1), forming a C-shape. At the time of their maximal extension, when the spine has formed an S-shaped curve (first phase), the lower levels of the cervical spine have exceeded their corresponding physiological range. The upper cervical spine then reaches maximum extension during the second phase, when the cervical spine exhibits a C-shape, but does not exceed its physiological level.

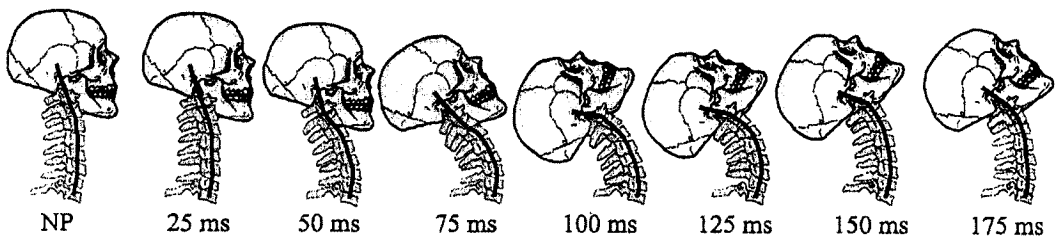

Figure1. Schematic of head and neck during a whiplash motion.

In a more recent study, Panjabi et al. (2004a) submitted to incremental whiplash trauma whole cervical spine specimens that were stabilized with a compressive muscle force replication system. This system had previously been found to produce head and neck kinematics similar to *in vivo* measurements (Panjabi et al. 2001). This new *in vitro* model confirmed previous findings in showing that the cervical spine exhibits the S-shaped and the C-shaped curvatures during a whiplash perturbation. However, even if the lower cervical spine already exceeded its physiological limits of extension during the S-shape phase, its peak extension was on average reached during the C-shape phase, when the upper cervical spine was also extending. In addition, this study indicated that while the lower cervical spine surpassed its physiological limit of extension for whiplash trauma of 5, 6.5 and 8 g, the upper cervical spine also did it but only at an acceleration of 8 g, and it never exceeded it in flexion. Taken together, these observations suggest that there is potentially more risk of injury at the lower levels of the spine (C6-C7 and C7-T1) but that there is also a risk at the upper levels if the impact acceleration is considerable. The risk of injury is present at all cervical levels as soon as peak extension of any segment surpasses its physiological limit.

This being said, a whiplash trauma can also be caused by frontal and side impacts, in which the neck is subjected to forced flexion and forced lateral flexion, respectively

(Barnsley et al. 1994). Therefore, both extension and flexion present theoretical risks of injury for many cervical structures. However, clinical, animal, cadaver and post-mortem studies have indicated that some structures are more prone to be injured. The anterior structures subjected to strain during the extension would be the capsule of the zygapophysial joints, the intervertebral discs, the anterior longitudinal ligament and the prevertebral neck muscles (longus capitis and longus colli). The posterior structures compressed would be the articular pillar of the zygapophysial joints and the intervertebral discs. During flexion, the capsule of the zygapophysial joints would be strained posteriorly and the intervertebral discs would be compressed anteriorly (Barnsley et al. 1994; Bogduk et al. 1986). More recent studies conducted on human cadaveric cervical spine specimens have confirmed the potential risk of injury to these structures (Ivancic et al. 2004; Panjabi et al. 2004b; Pearson et al. 2004).

Clinical symptoms

Several symptoms and clinical manifestations are associated with whiplash injuries. In their review, the QTF classified the ensemble of symptoms under the expression "whiplash-associated disorders" and proposed a clinical classification (Table 1) (Spitzer et al. 1995). The symptoms most commonly reported in the literature are neck pain, headache, visual disturbances, dizziness, weakness, paraesthesia, concentration and memory disturbances and psychological symptoms (Barnsley et al. 1994). Eck et al. (2001) added to this list neck stiffness, limited range of motion, shoulder pain and stiffness, arm pain, vertigo, temporomandibular joint symptoms, tinnitus, dysphasia and back pain.

Both studies of Barnsley and Eck highlighted the difficulty to find studies that associate a given whiplash symptom with a possible site of injury. Barnsley et al. (1994) suggested that injury to zygapophysial joints or intervertebral discs could be potential sources of neck pain. Headache, the most frequently reported complaint in association with neck pain, is assumed by some groups of researchers to be cervical in origin. Another important aspect of WAD is the associated dimension of pain, which in itself has been

thought to be the cause of physical symptoms such as movement avoidance and muscle hypertension. As such, Elert et al. (2001) found similar results of decreased ability to relax postural muscles and to produce shoulder torque in patients suffering from pain, whether from WAD or fibromyalgia. These findings point to pain as a potential mediator factor in the development of symptoms associated with WAD.

Table 1. Clinical classification of WAD.

Grade	Clinical presentation
0	No complaint about the neck No physical sign(s)
I	Neck complaint of pain, stiffness, or point tenderness only No physical sign(s)
II	Neck complaint AND Musculoskeletal sign(s)*
III	Neck complaint AND Neurological sign(s)†
V	Neck complaint AND Fracture or dislocation
	* Musculoskeletal signs include decreased range of motion and point tenderness. †Neurological signs include decreased or absent deep tendon reflexes, weakness and sensory deficits. Symptoms and disorders that can be manifest in all grades include deafness, dizziness, tinnitus, headache, memory loss, dysphagia, and temporomandibular joint pain.

Functional deficits

Several motor functions and abilities have been investigated in individuals with WAD. In these studies, the researchers have mainly used surface electromyography (EMG) and kinematic techniques in combination with several motor tasks to better characterize the functional deficits associated with the cervical region in persons with WAD.

Cervical range of motion (ROM) has frequently been used in the literature as an objective measure to characterize the severity of WAD and its evolution in time. Dall'Alba et al. (2001) measured cervical ROM in persons with chronic WAD and in asymptomatic individuals. Their results showed that in the WAD group, the cervical ROM was symmetrically reduced for all neck movements investigated. Sterling et al. (2003, 2004) corroborated these findings in a population of WAD patients one month post injury, regardless of the injury severity of these patients, as did Madeleine et al. (2004) in a chronic (at least six months post-injury) population. Finally, Sterling et al. (2003) showed that at three months post-injury, the deficit in ROM persisted only in patients with moderate or severe symptoms. These studies clearly indicate that decreased cervical ROM is a strong indicator of WAD as well as of WAD chronicity in the most severe groups.

Neck and shoulder muscle functions have been assessed in many studies in an attempt to understand the role of muscle dysfunction in the persistence of symptoms in individuals with WAD. Nederhand et al. (2000) measured the EMG activity of both upper trapezius (UT) in patients with chronic WAD Grade II (WAD II) during static tasks and during and after unilateral dynamic tasks of the upper extremities. In comparison with healthy individuals, patients with chronic WAD II presented higher coactivation during the dynamic task, both UT being recruited even during the unilateral task. Patients also demonstrated a generally decreased ability to relax their UT after exercise. In a subsequent study, Nederhand et al. (2002) compared the activity in the UT of patients with chronic WAD II, of patients with chronic nonspecific neck pain and of healthy controls. They used the same unilateral dynamic task to measure the mean level of muscle activity before and after the exercise, the ability to relax the UT after the exercise and the time course of muscle recovery. They could not find any statistically significant differences in any of their measures between patients with WAD II and those with nonspecific neck pain, suggesting similar mechanisms between these two diagnoses. Finally, in 2003, Nederhand et al. found that these muscle activity indicators, although sensitive to the levels of neck disability as measured using the Neck Disability Index

(NDI, Vernon and Mior 1991), did not vary over a 24 week period, suggesting an early reorganization of muscle strategies that persists over time. Falla et al. (2004a) extended Nederhand's studies to superficial neck muscles (sternocleidomastoid (SCM) and anterior scalenus (AS)) and found that EMG activity was greater in chronic WAD than in controls in all muscles except the right UT, which displayed the opposite trend across groups. This result indicates that the hyperreactivity previously observed in UT is also present in superficial neck flexors.

Falla et al. (2004b) continued their investigation and studied superficial and deep neck flexors during a motor task commonly used in the clinical setting to assess cervical dysfunction, the craniocervical flexion test (CCFT). This test provides a strategy to test the activity of the deep cervical flexor (DCF) muscles, which are thought to be more important for the control of stability of the cervical spine than superficial neck flexors. They found that subjects with chronic neck pain had decreased EMG activity in their DCF but increased EMG activity in their superficial neck flexors (SCM and AS) when compared to healthy subjects. They also noticed that the neck range of motion reached at each level of the CCFT was smaller in individuals with neck pain. These results indicate that in people with chronic neck pain, the decreased performance in the CCFT might be related to an impaired performance of DCF. The increased EMG activity of the superficial neck flexors and the decreased neck range of motion also suggest an altered muscle strategy employed to perform CCFT. Moreover, Sterling et al. (2003) demonstrated that this altered muscle response to the CCFT is already present in the acute stages of the injury and persists as chronicity develops, regardless of the degree of pain and disability. As a whole, these studies suggest that WAD individuals present characteristic neck muscle strategies, regardless of neck muscles acting as agonists or as postural stabilizers during the task.

Measures of cervical neck posture

The previously mentioned studies show strong evidence that the motor structures (muscles, joints) are directly affected during a whiplash accident. However, the

possibility that other structures of the motor control loops are also affected has received comparatively less attention. For instance, the ability of a whiplash patient to perceive proprioceptive information (afferences) and the capacity to integrate this information into appropriate motor control strategies (central) are factors that may also affect everyday tasks. In an attempt to shed some light into these questions, Sterling et al. (2003) sought to determine if the severity and chronicity of WAD were related to an inability to effectively control the head position in space. Joint position error (JPE) is a measure of a person's ability to relocate the head to a natural head posture following active cervical movements, usually performed in the clinical setting with eyes closed. Using this test, authors found that JPE measures could only differentiate the group with moderate/severe symptoms, who displayed greater JPE, from the groups with mild symptoms or from the recovered group. Their subsequent study indicated that patients with severe symptoms also displayed greater JPE than all other sub-groups (moderate, mild, healthy) following head extension (Sterling et al. 2004).

Finally, to better understand how WAD individuals coordinate internal perturbations produced by limb movements with cervical posture, some studies have measured the effects of rapid voluntary arm movements on neck response. It had been previously shown that in healthy persons, internal perturbations of the posture provoke the activation of superficial neck flexor and extensor muscles (termed "feedforward" adjustments) and are considered to be anticipated by the central nervous system (CNS) to provide stabilization of the cervical spine (Falla et al. 2004c). In a subsequent study, this paradigm was utilized to compare the behavior of the superficial neck flexors (SCM and AS) to that of the DCF in people with chronic neck pain and in healthy controls (Falla et al. 2004d). They found that deep and superficial cervical flexors demonstrated feedforward activity in healthy subjects and that this activity was delayed in people with chronic neck pain. These results indicated that the strategy used by the CNS to prepare the cervical spine for an internal perturbation involves coordinated activity of deep and superficial muscles of the neck, and that this central characteristic may be altered in persons suffering from neck complaints.

Symptoms associated with global posture and balance

As previously mentioned, dizziness and vertigo are common symptoms reported by people with WAD. Barnsley et al. (1994) described dizziness as a "sensation of disequilibrium" and brought the hypothesis that balance disturbance "may result from interference with postural reflexes that have cervical afferents." Indeed, it has been well demonstrated that important proprioceptive information arises from neck structures, suggesting that an impaired ability to receive feedback information from these structures may be detrimental to maintaining balance. Similarly, in 1991, Chester postulated that neck stiffness and pain reported by people who had a whiplash could be related to a central disorder of postural control which has evolved secondary to injury of the labyrinthine structures of the inner ear. In an attempt to measure the physical symptoms associated with dizziness, Rubin et al. (1995) examined the balance abilities of individuals who had expressed subjective complaints of dizziness following mild head injury or whiplash injury. They found that patients had a decreased postural stability (as measured by an increased body sway) in comparison with healthy individuals. In addition, dizziness and unsteadiness have been reported in different cervical pain syndromes such as cervico-brachial pain. These patients manifested increased vibration-induced and galvanic-induced body sway, indicating impaired postural control (Karlberg et al. 1995). Even if the exact cause of dizziness and vertigo is not known, these symptoms suggest that postural stability might be affected in populations suffering from these symptoms, such as individuals with WAD.

Posture and balance

The human posture is defined as the position of each body's segment relative to one another and their orientation in space. For example, lying, sitting and standing are common postures adopted by humans for the execution of several tasks. Balance is the dynamic concept of continuously adjusting posture to stay in equilibrium while accounting for motion and forces such as that of gravity. These postural adjustments

serve three behavioral functions. First, they are necessary to support the body against gravity and other external forces. Second, they stabilize the supporting parts of the body while others are moving. Third, they keep the body's center of mass (COM) aligned and balanced over the base of support, which is required in order not to fall (Ghez 1991). The body's COM is a point equivalent of the total body mass and is the weighted average of the COM of each body segment in the three-dimensional space (Winter 1995).

Sensory afferences are the first elements of the motor control system loop to come into play when a balance correction response is required. Indeed, the postural control system must be able to detect a loss of balance in order to trigger adequate postural responses. Three types of sensory inputs are involved in this specific process: visual, vestibular and somatosensory. Visual information allows the detection of motion in the visual field. Vestibular receptors can detect angular and linear movements of the head. Somatosensory inputs originate from receptors on the skin, in the muscles and in the joints and are involved in perceiving sensations of touch, pressure, temperature, pain and proprioception, defined as the sense of body position (Bear et al. 2001). Receptors involved in postural control are the muscle spindles, the Golgi tendon organs and the cutaneous receptors which detect muscle length, muscle tension and skin pressure, respectively. The combination of all these sensory inputs can be used in an integrative fashion to trigger postural adjustments.

A loss of balance is often synonymous of a perturbation of the equilibrated posture. Winter (1995) distinguishes two types of postural perturbations. Internal perturbations are related to the execution of voluntary movements. Moving the upper limbs, for example, modifies the position of the body's COM. In this case, the CNS will typically produce anticipatory (proactive) postural adjustments, which are preprogrammed responses, in order to maintain balance. This feedforward control is essential to coordinate posture with voluntary movements. External perturbations come from the environment and are unexpected. The postural responses are therefore reactive and the feedback from the sensory systems is used in order to calibrate the postural reactions. Ghez (1991) mentions that like reflexes, these responses are rapid and display a stereotyped spatiotemporal

organization. On the other hand, unlike reflexes, they are appropriately scaled to stabilize the posture, are refined with practice and learning, and are context-dependent.

In contrast, another hypothesis of postural control states that postural reactions in response to external and unexpected perturbations are not purely reactive but that the response amplitude is related to the expectation of the perturbation by the use of prior experience, suggesting an influence of central origin. According to Horak et al. (1989), central influences are important for the early component of the postural response "because postural muscle responses are often initiated before availability of the peripheral information characterizing the full nature of stimulus." The authors mention that this so-called central set leads to errors in postural response when the stimulus is different from that expected. In turn, peripheral sensory information can be used to adjust the postural response (late component) according to the real characteristics of the perturbation. Horak's findings have been supported by those of Di Fabio et al. (1990), who also found that postural responses are centrally initiated but regulated in amplitude by local sensory information. Another supporting argument for the involvement of central level structures is the regular finding that postural stabilization mechanisms are altered when the subject is aware of the incoming perturbation (Kumar et al., 2000, 2002), whether they have prior knowledge of its onset time (Siegmund et al. 2003a), and even more so when the perturbation is self-induced. In most of these cases, joint velocities are reduced (Simoneau et al. 2003) and muscle activation delays are generally shortened (Blouin et al. 2003a), suggesting feedforward postural control when the subject has prior knowledge of a perturbation.

Postural stabilization mechanisms in standing

Postural reactions to external perturbations have been extensively studied in standing. Most notably, the development of movable platforms as a perturbation device has allowed researchers to examine the stabilizing response of the postural control system following mechanical perturbations.

Among the first findings resulting from this broad research area were those of Nashner (1976), who was the first to describe stereotypical postural stabilization patterns following a variety of surface perturbations. They found that following translational or rotational surface perturbations, the response patterns of the ankle muscles resembled that of a long-latency stretch reflex, which could provide a rapid compensation for a postural disturbance when this response was appropriate to the task. This strategy was later described as the 'ankle strategy'. More specifically, when the ankle strategy is used, ankle muscles are typically activated first, followed by knee and finally hip muscles, in a distal-to-proximal sequence. In reaction to anteroposterior translations, a second strategy was observed to be adopted by the subjects (Horak and Nashner 1986). The 'hip strategy' was characterized by the activation of the hip muscles that increased the sagittal shear forces, rotating the body about the hip joints, with hip muscles activated first, followed by the knee muscles and finally by the ankle muscles in a proximal-to-distal sequence.

In more recent studies, Henry et al. (1998) showed that the muscle synergy organization employed to maintain stance was not limited to ankle and hip strategies or to a combination of both. They recorded EMG activity of ankle, knee and hip muscles of subjects submitted to horizontal surface translations in 12 different, randomly presented directions. They found that in general, hip muscles were recruited before or at the same time as more distal muscles, and ankle and knee muscles were recruited in a distal-to-proximal sequence. The EMG latencies of ankle and knee muscles were constant and independent of the direction of translation although the hip muscles were activated at two different EMG latencies depending on the direction of the perturbation.

In summary, these studies have been important in documenting muscle synergies that are typically utilized to restore balance in response to mechanical perturbations of the standing posture. Anteroposterior perturbations provoke mainly two stereotyped patterns: ankle and hip strategies. However, diagonal perturbations revealed that hip muscles could be recruited early or late in the synergies, with the same distal-to-proximal sequence for ankle and knee muscles. Therefore, these results suggest that these two principal strategies are modifiable depending on the characteristics of the perturbation and the

expectation of the subject, which is consistent with the hypothesis of a complex interaction between central and peripheral contributions for postural control.

Finally, some researchers were interested more specifically in the control of head stability and the coordination with the stabilization of the trunk during situations in which balance was challenged. Pozzo et al. (1995) extended previous studies done in the sagittal plane during gait tasks (Pozzo et al. 1989, 1990) by the investigation of head stabilization in the frontal plane in subjects maintaining challenging standing postures. Their overall results indicated that the head could be stabilized together with the trunk for small body instabilities, a result corroborated by Keshner (2003) in the sitting position. However, during great body oscillations, the head was stabilized independently from the trunk and stayed close to the vertical position, suggesting that the head could be a reference segment to provide an egocentric reference associated with the vertical gravity.

Postural stabilization mechanisms in sitting

Only few studies have been conducted to investigate the stabilization mechanisms of the sitting posture in healthy persons. The most complete study to date is that of Forssberg and Hirschfeld (1994), who aimed at describing the muscle activation patterns and the body kinematics during various types of perturbations. In their study, subjects were sitting on a platform with hip flexed at 90° and knees extended and were submitted to several types of postural perturbations, including forward and backward translations. Results showed that following a forward perturbation, the pelvis, then the thorax and finally the head rotated in the backward direction before moving back to their initial position (or nearly) in the forward direction. The opposite pattern was observed when the subjects were perturbed in the backward direction: the pelvis, followed by the thorax and finally the head rotated in the forward direction and then moved back to their initial position in the backward direction.

Moreover, results showed that the postural muscle activation pattern following a forward translation was consistent in all subjects. It was characterized by the early activation of

the rectus femoris followed by the activation of neck and trunk flexors, then of the gluteus maximus and finally by neck and trunk extensors. This sequence indicated that ventral muscles were activated before dorsal muscles. However, no clear caudo-cranial recruitment sequence could be identified either in ventral or in dorsal muscles. These EMG patterns were consistent with the kinematics: the activation of ventral muscles acted to stabilize the pelvis, the trunk and the head during the backward sway and dorsal muscles, during the forward sway. The backward translation of the platform did not evoke such consistent muscular activation patterns, however. The muscles were activated with smaller amplitude in comparison with that during forward translations and with large intra- and interindividual variability. In most subjects, weak activity of trunk and neck extensors was evoked, which theoretically serve to stabilize the trunk and head during the forward sway. The differences in muscle activation patterns between backward and forward body sway during sitting most likely reflect the asymmetry of the stability limit of the sitting posture. Indeed, in sitting (especially with the legs extended), the body's COM is closer to the posterior margin of the support surface. Therefore, a perturbation provoking a backward body sway is more susceptible to move the body's COM outside the limit of stability, therefore requiring more muscle activation to maintain posture.

Another important study on postural control of seated humans was conducted by Vibert et al. (2001). This team also compared postural adjustments of head and trunk in response to different types of postural perturbations, including anteroposterior surface translations. However, they submitted the subjects to high-jerk perturbations and the subjects were sitting on a standard chair. Results showed that anteroposterior translations produced a stereotypical sequence of head and trunk movements. Following a forward perturbation, the hips were translated forward while the upper part of the torso and the head rotated backward. Then, the whole trunk moved back to its initial position in a forward rotation and finally, the head also rotated in the forward direction to reach its initial position, aligned with the trunk. The sequence of movements was opposite for a backward perturbation: the head and trunk rotated first in the forward direction before moving back in the backward direction. The amplitude of the head movement was highly variable

across subjects, who were separated in two groups: stiff subjects had small head motion amplitude (approximately between 4 and 15°), with the head more or less in line with the trunk, while floppy subjects had larger head movement amplitude (approximately 30°), with their head lagging behind the trunk. These two patterns of kinematic responses were highly repeatable for both groups. Further analysis showed that stiff and floppy behaviors were end-points of a continuum rather than two discrete categories. However, the analysis of muscle activity revealed no stereotypical pattern for neck muscles. Regardless of the amplitude of the subject's head movement, a large proportion of subjects did not display any systematic activation pattern of their neck muscles.

As a whole, the kinematic patterns found by Vibert et al. (2001) are consistent with those of Forssberg and Hirschfeld (1994). Even if the starting postures of the subjects were different, both studies showed a distal-to-proximal sequence in the displacement of pelvis, thorax and head segments, in a direction opposite to the support surface translation for the thorax and the head. Moreover, Forssberg and Hirschfeld (1994) showed that following a perturbation, muscles that are elongated typically respond in a way that is consistent with the well-documented stretch reflex (Gordon and Ghez 1991), a finding that is supported by Zedka et al. (1998) who found similar results by investigating rotations of the support surface.

Experimental conditions of whiplash-like perturbations

When subjected to a whiplash impact, typically observed in car accidents and which provoke a sudden acceleration of the head, the human body is faced with a postural perturbation that is severe enough to provoke pathological symptoms. To better understand the mechanism of the whiplash lesion, several researchers have attempted to reproduce such conditions in a laboratory setting. Brault et al. (2000) investigated the possibility that the whiplash motion directly induces muscle tissue damage. In their experimental setup, the participants were sitting in a test car and submitted to two small rear-end accidents, producing 4 km/h or 8 km/h speed changes on the target vehicle. Their results indicated that the participants started to move their head earlier, with larger

amplitude, and they recruited their neck muscles earlier at 8 km/h. Combining kinematic and EMG data, the authors found that the SCM maximum lengthening velocity was reached when the muscle was activated while lengthening. These data suggest a possible muscle injury mechanism that is due to rapid and forceful eccentric contractions.

The goal of reproducing whiplash-like perturbations in a laboratory setting was also to determine which parameters of a whiplash impact play an important role in the development of a whiplash injury. In order to simulate a whiplash accident in the laboratory, the subjects are typically sitting in a car seat fixed to a sled and are submitted to forward translations. As highlighted previously, the awareness of the participants for the incoming perturbation affects their kinematic and neck muscular responses (Kumar et al. 2000, 2002). The magnitude of the platform acceleration is a second factor that was shown to modify the postural response in perturbed subjects. More specifically, greater sled accelerations provoked earlier and larger head acceleration and neck muscular activity (Kumar et al. 2000, 2002; Siegmund et al. 2002).

However, the attempt to reproduce as closely as possible real-life whiplash impacts poses certain challenges. The use of sequential whiplash-like perturbations within experimental protocols might trigger muscular and kinematic responses that do not represent those of individuals normally submitted to a unique whiplash impact in real life. Siegmund et al. (2003b) tested this factor and their results indicated that during the first trial, all the subjects rapidly restored their upright head position, which was not always the case in the subsequent trials. However, the sequence of 11 perturbations produced a large decrease in EMG amplitude but no changes in EMG latencies of SCM and cervical paraspinals (CP). Similarly, the sequential perturbations produced large variations in peak kinematic variables but did not affect the onset of head and torso acceleration. All together, these results indicate that habituation modifies the amplitude but not the timing of the postural response after multiple perturbations. This suggests that caution should be observed when interpreting results of studies using multiple perturbations.

Blouin et al. (2003b) found similar results of stereotypical muscular and kinematic responses to the first trial in all subjects. However, following subsequent perturbations, two different kinematic behaviors could be observed: in one group, there was an increase in head angular displacement and velocity in extension with the number of trials and in the second group, the head kinematics stayed stable. These results are slightly different from what was found by Siegmund et al. (2003b) who did not report dichotomic behaviors among subjects. On the other hand, the two kinematic groups found by Blouin are consistent with the 'floppy' or the 'stiff' strategy used by the subjects in Vibert et al. (2001). For both kinematic groups in Blouin's study, there was a decrease in EMG amplitude of neck muscles and no significant changes in EMG onsets, indicating that a neck stiffening strategy was not adopted by the subjects, which is in accordance with the findings of Siegmund et al. (2003b).

These studies underline that the support surface acceleration and the awareness of the subject with regards to the direction and the timing of the perturbation affect the muscular and kinematic patterns of the subjects. It was highlighted that a perturbation of greater acceleration or that is unexpected produces larger head kinematics and greater EMG activity in neck muscles. This suggests that whiplash impacts which are completely unexpected or of greater acceleration could potentially increase the risk of injury. However, other studies also revealed that protocols including multiple whiplash-like perturbations also affect the postural response of the subjects. Therefore, the data obtained with protocols that simulate whiplash perturbations must be carefully interpreted in function of the experimental conditions, and inferences on the injury mechanisms associated with whiplash injuries must be made with great caution.

Postural control in persons with WAD

Postural control and balance mechanisms have been well described in healthy individuals in the standing position, the sitting position and during functional tasks. In light of these findings, and using knowledge gained from studies that investigated the symptoms associated with WAD, it has been hypothesized that mechanisms responsible to maintain

postural stability could be affected in individuals with WAD. Balance and postural control of individuals with chronic WAD have been studied using posturography, a biomechanical assessment technique involving the subjects standing on one or two force plates that measure the body's center of pressure (COP) displacement. The COP is the weighted average of all the pressures under the feet, corresponding to the point location where the ground reaction force acts on the body (Winter 1995), and the displacement of this variable is typically used as an indication of body sway. Accelerometers attached to the trunk as well as other kinematic systems are additional devices commonly used in the laboratory setting to quantify body sway. These tools have been used, along with several visual, vestibular and support surface conditions, in experimental setups to better understand the mechanisms involved in some common postural disorders. Using a clinical protocol of perturbations, Chester (1991) found that most patients who had sustained a whiplash accident displayed inappropriate stabilization responses of the standing posture following repeated sensory perturbations. Chester also used several clinical procedures to verify the integrity of the inner ear and obtained abnormal results for each test in a portion of the patients, suggesting that the delicate labyrinthine structures can be injured during a whiplash accident and that the resultant inner ear dysfunction might be partly responsible for the inefficient control of balance in standing. The ability to stabilize the posture was further investigated in a laboratory posturographic protocol by Kogler et al. (2000) and Madeleine et al. (2004) who compared the performance of WAD and healthy individuals submitted to several sensory conditions from a standing position. They both found that patients had larger amplitude of their COP displacements compared with healthy individuals and that sensory perturbations had a greater effect on the postural stability of patients compared with that of healthy individuals. These results support the hypothesis that neck injuries might impair postural control mechanisms in standing.

Aside from using techniques of support surface displacements and modifications of the sensory environment, quiet standing can be perturbed mechanically by the subject executing movements with the limbs (Michaelson et al. 2003). Such experiments have shown that WAD individuals, while standing, display greater amplitude of COP

trajectory compared with individuals with work-related neck pain (WRP) or control subjects. WAD patients also showed greater amplitude of head translation. The same subjects were also tested for their ability to adopt standard standing positions such as Romberg stance, tandem stance and one legged stance. Patients with WAD were less able to maintain some positions for the required duration than patients with WRP and than healthy individuals. These results confirmed the previous findings that patients with WAD had greater postural sway in standing than healthy controls when balance is challenged and that these patients show a reduced ability to stabilize the head.

In a similar study, trunk sway of patients with chronic WAD was measured during the execution of 14 stance and gait tasks, in normal and perturbed conditions (Sjöström et al. 2003). The results showed a characteristic pattern of trunk sway for both stance and gait tasks of WAD individuals, in comparison with controls. WAD patients had greater trunk sway during stance tasks and during complex gait tasks that required task-specific gaze control. On the other hand, WAD individuals presented lesser trunk sway in simple gait tasks that demanded large head movement but no task-specific gaze control. This might be due to their smaller amplitude of head rotation during the task.

In summary, these studies clearly show that individuals with chronic WAD present a decreased postural stability in standing, especially when the sensory conditions are modified (eyes closed for example) or when mechanical perturbations are used to disturb the subjects from their tasks. These results bring the hypothesis that due to the neck trauma, these patients might have difficulty combining visual, vestibular, and neck proprioceptive signals to generate balance control commands. However, lacking from these studies are ones that document the characteristics of the sitting posture in WAD individuals, as well as more in-depth analyses of postural control mechanisms, which can be gained via the use of techniques of electromyography.

In conclusion, the literature demonstrates that WAD is an important health and socioeconomic problem that is not fully understood. Even if the injury mechanism is well

described, it is still difficult to identify the structures being injured during a whiplash impact and that could be responsible for the several symptoms associated with a whiplash trauma. Most patients recover from their injury within eight weeks. However, in a portion of them, some symptoms such as neck pain and headache (shown to be related to motor functional deficits) will develop chronicity. Moreover, we know that individuals with WAD typically manifest complaints of dizziness and unsteadiness that can be related to altered postural stability. The mechanisms implicated in maintaining postural stability have been well described in healthy individuals in the standing and sitting positions. In patients with chronic WAD, the ability to stabilize the posture has been investigated in quiet standing and during gait tasks in many studies that indicate increased body sway in comparison to healthy controls. However, these postural stability characteristics have not been examined in sitting. In addition, this population has not been tested for its capacity to restore balance following support surface perturbations, a paradigm more commonly used to better understand postural strategies. Protocols of mechanical perturbations combining kinematic and electromyographic data could be useful to analyze in more depth the postural control mechanisms in individuals with WAD.

FIRST RESEARCH ARTICLE

A paradigm to assess postural responses triggered by anteroposterior translations in sitting

Nancy St-Onge, Julie N. Côté, Isabelle Patenaude, Joyce Fung

Abstract

Postural reactions may be altered in the presence of pathologies, such as whiplash-associated disorders (WAD). The ability of individuals suffering from whiplash-associated disorders to stabilize their posture following a mechanical perturbation has not been characterized yet. The goal of this study was to identify a low-threshold perturbation that elicits postural reactions in healthy seated individuals. We hypothesize that such a perturbation would be safe for individuals with neck injuries and it could be used to assess the physical condition of WAD individuals. Six healthy subjects participated in this study. They sat on an adapted ergonomic chair fixed on a movable support surface which was submitted to forward and backward translations. The neck and trunk angular displacements as well as the activity of sixteen neck and trunk muscles were recorded. Displacements of low amplitude (15 cm) and long duration (500 ms) elicited stereotypical reactions across subjects. Although this perturbation is of low intensity compared to data from the literature, the stimulus is sufficient and can be used safely to quantify patterns postural reactions. This perturbation will be used to assess the postural stability of persons with WAD.

Keywords: low-intensity perturbation; neck injuries; postural reactions; kinematics; electromyography

Introduction

A whiplash can be described as a fast movement of the head which occurs during the 100 to 200 ms following an impact in any direction (Sjöström et al. 2003). About 10% of the victims of car accidents involving a rear impact develop a pathology, and 18% to 40% will have chronic symptoms. Whiplash leads to a variable prognosis which is difficult to foresee (Suissa 2003). Post-traumatic pathologies are generally grouped in a common class called whiplash-associated disorders (WAD). Those disorders usually attack soft tissues and cervical sprain is the most common sub-acute or chronic condition. Chronic symptoms associated with WAD include a decrease of movement amplitude and

proprioception at the level of the neck (Sterling et al. 2003), a decrease of the muscle force and endurance (Dumas et al. 2001), neck and back pain, headaches, vision troubles, and cognitive difficulties (Siegmund et al. 2001) as well as abnormal eye movements (Rubin et al. 1995).

The mechanisms of whiplash and how they influence the systems involved in postural and motor control are not well understood. The motor deficits associated with WAD can be due to an imbalance of postural central commands (Chester 1991) resulting from an impairment of the neck proprioceptive mechanisms (Gimse et al. 1997; Treleaven et al. 2003) or the vestibular system (Rubin et al. 1995). The level at which the systems are perturbed is not well defined and therefore the complexity of motor limits associated with this pathology is not well understood. Many anatomical structures can be injured during whiplash: muscles, ligaments, fascia, intervertebral discs, nerves, cartilage, and joint capsules (Loudon et al. 1997). Upright balance is maintained by the interaction of sensory systems (vestibular, visual and proprioceptive) and motor coordination (head, neck, trunk and extremities). The altered postural equilibrium observed in WAD individuals can result from disorders in one or more of the sensory-motor elements involved in maintaining balance.

Biomechanical tools are seldomly used to quantify the severity of WAD, evaluate the progress of treatment, or predict the chances of return to the workplace following injury. Support surface perturbations are commonly used in research to study the postural control system in humans. They trigger specific motor responses that can determine the integrity of the proprioceptive and musculoskeletal systems involved in the control of posture and balance. In standing healthy subjects, appropriate movement patterns and muscle actions are generated by the central nervous system to maintain the projection of the body's center of mass (COM) within the base of support following multidirectional surface translations (Henry et al. 1998; Horak and Nashner 1986; Nashner 1976; Nashner 1983; Okada et al. 2001; Pozzo et al. 1995; Simoneau et al. 2003). Other studies have characterized healthy response patterns to perturbations applied to the sitting posture (Blouin et al. 2003a; Blouin et al. 2003b; Brault et al. 2000; Forssberg and Hirschfeld

1994; Keshner 2003; Siegmund et al. 2002; Siegmund et al. 2003a; Siegmund et al. 2003b; Vibert et al. 2001). These studies have allowed the characterization of healthy postural stabilization responses across the spine's musculoskeletal system, which can in turn be used to highlight specific pathological states associated with postural response mechanisms.

Studying the postural stabilization mechanisms in WAD individuals implies submitting subjects to perturbations that do provoke a physical response. However, from a safety point of view, it is of prime importance to select minimal perturbations to elicit such responses in a pathological population. A protocol using low-intensity support surface translations could also be used to evaluate the integrity of the postural control system of WAD individuals. Moreover, although the instability caused by a whiplash occurs mainly at the level of the neck, it is possible that the whole postural chain be affected. The general objective of our research efforts is to quantify the biomechanical characteristics of individuals suffering from WAD. Our goal is to develop and validate quantitative approaches that will allow a better evaluation of the severity of the pathology in this population. This knowledge could be used to better evaluate the initial condition of a person suffering from WAD, measure the efficacy of a treatment and predict the ability to return to work. As a first step towards this objective, the goal of this specific study was to identify a low-intensity threshold perturbation that provokes postural reactions in seated individuals. Such a perturbation would be safe for individuals with neck injuries and it could be used to better assess the condition of WAD individuals.

Materials and Methods

Healthy subjects (n=6; 26-45 years old) with no history of back or neck musculoskeletal disorders participated in this study. The nature of their participation was explained to them and they gave their written consent by signing the forms approved by the institutional ethics committee.

Subjects sat on an adapted ergonomic chair (see Figure 1) firmly bolted on a movable support surface which is servo-controlled by electro-hydraulic actuators. Subjects were stabilized to the chair using a belt and stabilizing devices aligned with their hips and lower legs, so that movement was only allowed above the pelvis. They had their arms crossed in front of their chests. Subjects were submitted to forward and backward translations. For the first subject, the platform was moved with a ramp stimulus of 15 cm amplitude (maximum amplitude allowed by our system) and different durations. We chose this amplitude in consultation with the clinical team providing treatments to the WAD patients, who suggested that a longer displacement of the platform would help to avoid high-jerk perturbations. The duration of the perturbation was achieved by choosing the time it took for the platform to be displaced through the linear portion of the displacement. Nine durations were chosen and applied in the following order: 900 ms, 800 ms, 700 ms, 600 ms, 500 ms, 400 ms, 300 ms, 200 ms, 100 ms. Forward perturbations were applied first. Thus, the minimal stimulus duration which produced postural reactions could be determined. The threshold perturbation in the first subject was identified as the smallest intensity which produced observable and stereotypical postural reactions. After having found that threshold perturbation, we assessed the inter-subject reproducibility of these patterns by also subjecting the remainder of subjects to forward and backward perturbations at that same intensity. For those subjects, we assessed the intra-subject reproducibility of the response patterns by using a randomized sequence of 15 perturbation trials at the identified threshold intensity, with 5 forward translations, 5 backward translations and 5 unperturbed trials. For all subjects, a static trial with the subject sitting on the chair was recorded.

Motion of the head, arm and trunk segments was analyzed from 3D position coordinates of the body markers using a high resolution six-camera Vicon 512 Motion Analysis System (Vicon Peak, UK) (sampling frequency: 120 Hz). Reflective markers were placed on the following anatomical landmarks: left and right front head, left and right back head, C7 spinous process, left and right T1 transverse processes, T6 spinous process, left and right T8 transverse processes, left and right L1 transverse processes, right scapula, sternal notch, left acromion, left lateral epicondyle, left head of third metacarpal, right acromion,

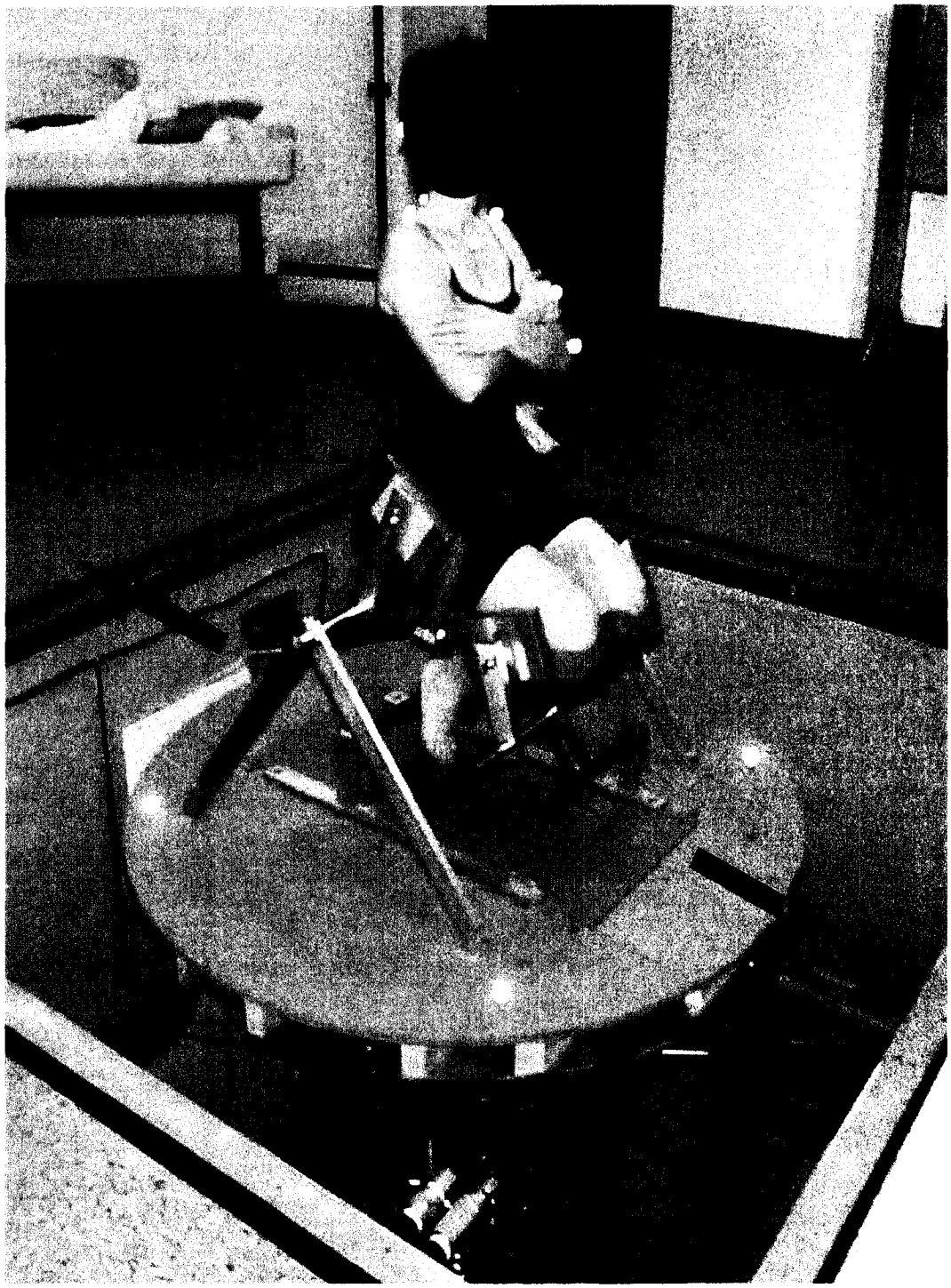

Figure 1. Adapted ergonomic chair bolted on a movable platform. The subject is stabilized to the chair using a belt and stabilizing devices aligned with her hips and lower legs, so that movement is only allowed above the pelvis.

right lateral epicondyle, right head of third metacarpal, S1, left and right postero-superior iliac spines, apex of sacrum. Reflective markers were also placed on each corner of the movable platform to record its position. Also, to allow the computation of COM, the following anthropometric measurements were taken: trunk depth at T1, T4, T10, and L3 levels; body height and body weight.

The activity of sixteen muscles of the neck and trunk was acquired using telemetric surface electromyography (Noraxon, USA). Data were sampled at 1800 Hz.
After preparing the skin, bipolar surface Ag/AgCl electrodes (Ambu, DE) were placed bilaterally on the skin overlying the following neck and trunk muscles: scalenus (SCA), sternocleidomastoid (SCM), cervical paraspinals (CP), upper trapezius (UT), erector spinae (thoracic level; TES), erector spinae (lumbar level; LES), rectus abdominis (RA), external obliques (EO).

Kinematic data was low-pass filtered (zero-lag Butterworth filter, 6 Hz). The head segment was defined using the four head markers and the trunk segment was defined using markers of S1 and both left and right T1 transverse processes. Head and trunk angular displacements were computed by quantifying the orientation of the head and trunk segments relative to the global space using Euler xyz rotations. We analyzed only angular displacements in the sagittal plane. Abdomen COM, lower thorax COM, upper thorax COM, both arms COM, and head COM were computed using segmental measurements and standard anthropometric tables. The COM of the head, arms and trunk (HAT) was computed from a combination of individual segment COMs. Heartbeat noise was removed from electromyographic (EMG) signals using a 40Hz low-pass Butterworth filter. Each heartbeat was filtered out from the EMG signal by applying the filter and was subsequently subtracted from the signal. EMG data with heartbeats removed was then band-pass filtered (zero-lag Butterworth filter, 10-350 Hz) and full-wave rectified. EMG envelopes were then computed using a 10-Hz zero-lag Butterworth filter. Platform displacement was computed using the four markers positioned on each corner of the platform. The position of those markers was averaged to find the position of the center of the platform. Platform onset was taken as the time when the velocity of its center reached

5% of the maximal velocity. Trials were aligned at platform onset and averaged for each subject.

Results

Figure 2 shows average head angular displacement and EMG envelopes for SCM, CP, RA, and LES in one subject for perturbations of different intensities for both forward and backward perturbations. Low-intensity (700 ms and 600 ms) perturbations in the forward direction generated head angular displacements of approximately 3°. Head and trunk angular displacement reached 7° when the platform displacement lasted 500 ms for forward perturbations. Bursts of muscular activity can be observed for all intensities in SCM, CP, RA, and LES muscles for forward perturbations. For backward perturbation, all intensities generated head angular displacements of approximately 4-5°. Also, bursts of activity were generated in CP, RA, and LES at all intensities. From the results of the first subject, we consider that the 500-ms perturbation is a threshold perturbation which produced observable postural reactions. We find that displacements of 3° are too small to be measured. For backward perturbations, the amplitude of head angular displacement reached 5° for perturbations of smaller intensities. However, we have decided to study the same intensity for both directions of perturbation. We therefore studied 500-ms perturbations both in the forward and backward directions in the remaining subjects.

Figure 3 shows average head and trunk angular displacement and HATCOM displacement in all subjects for 500-ms perturbations. For forward perturbations, head and trunk segments first moved into extension, followed by flexion whereas for backward perturbations, they first moved into flexion, followed by extension. HATCOM started moving 0-100 ms after platform onset, while trunk and head segment onsets occurred 0-100 ms and 100-500 ms relative to platform onset, respectively. Trunk and head angular displacements ranged from 3 to 10°. HATCOM stabilised after overshooting its final position.

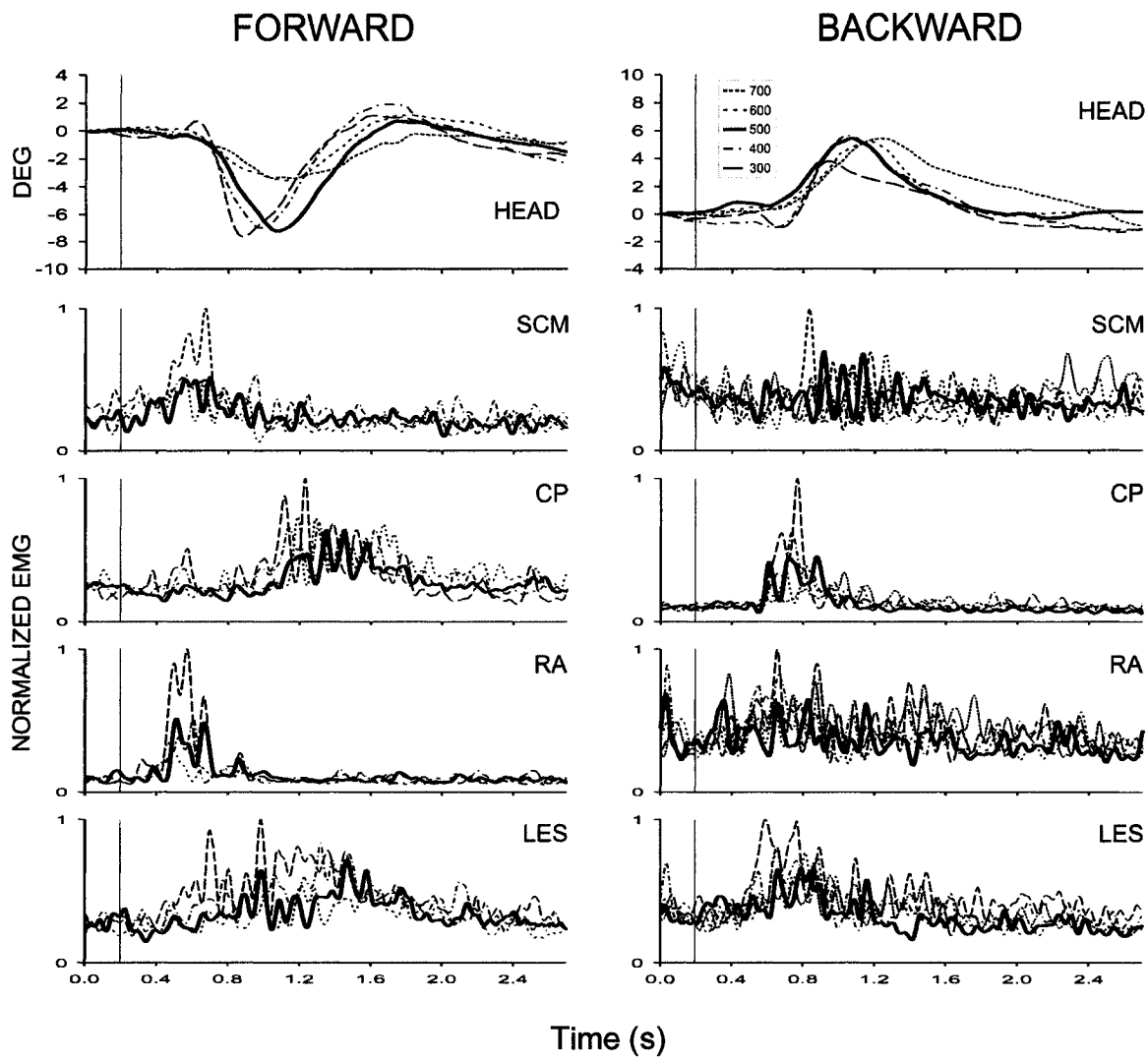

Figure 2. Averaged head angular flexion(+)/extension(-) and electromyographic (EMG) envelopes for sternocleidomastoid (SCM), cervical paraspinals (CP), rectus abdominis (RA), and lumbar erector spinae (LES) in one subject for perturbations of different intensities for forward and backward translations. Data were normalized to the maximum of averaged envelopes for all intensities. Vertical lines show onset of platform movement.

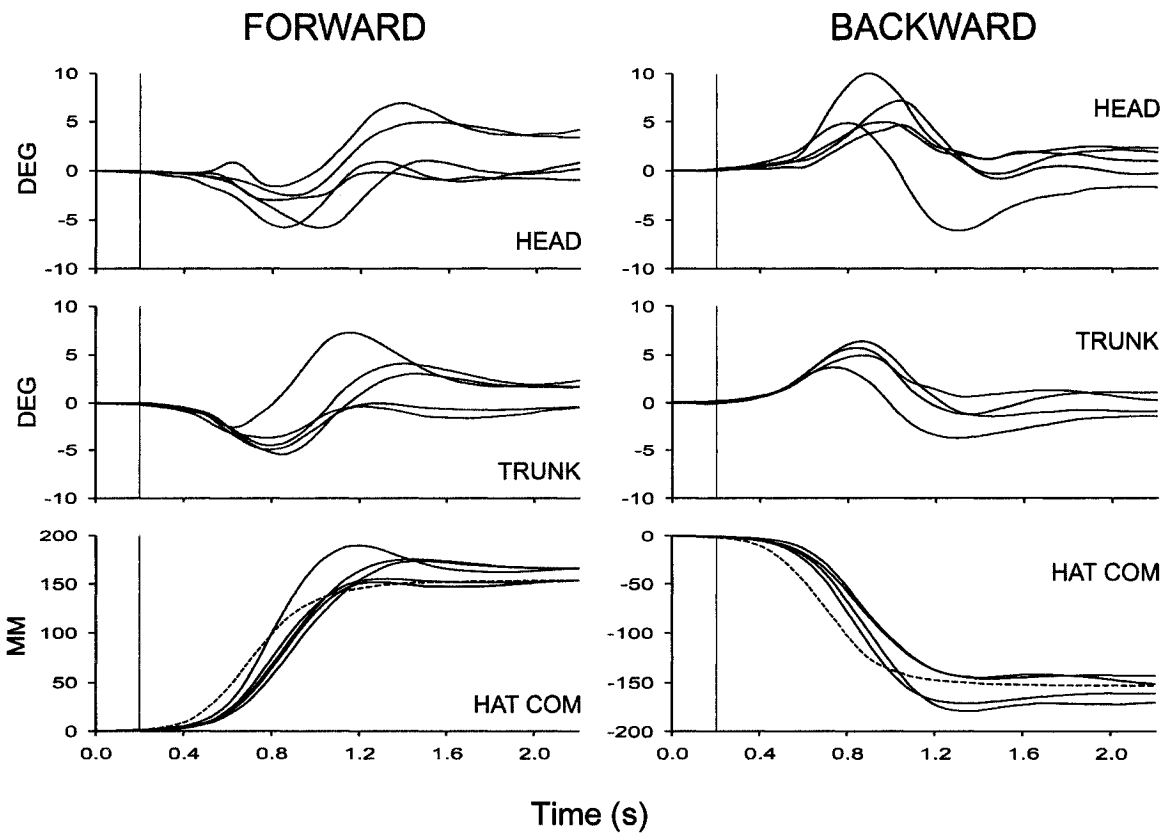

Figure 3. Head and trunk angular flexion(+)/extension(-) and HATCOM displacement in five subjects for 500-ms perturbations. Averaged data of up to five trials are shown for each subject. Dashed lines represent platform displacement. Vertical lines show onset of platform movement.

Figure 4 shows average EMG envelopes for SCM, CP, RA, and LES in all subjects for 500-ms perturbations. For forward perturbations, neck and trunk flexor muscles were activated first, followed by the extensor muscles. For backward perturbations, extensor muscles were activated first, followed by flexors.

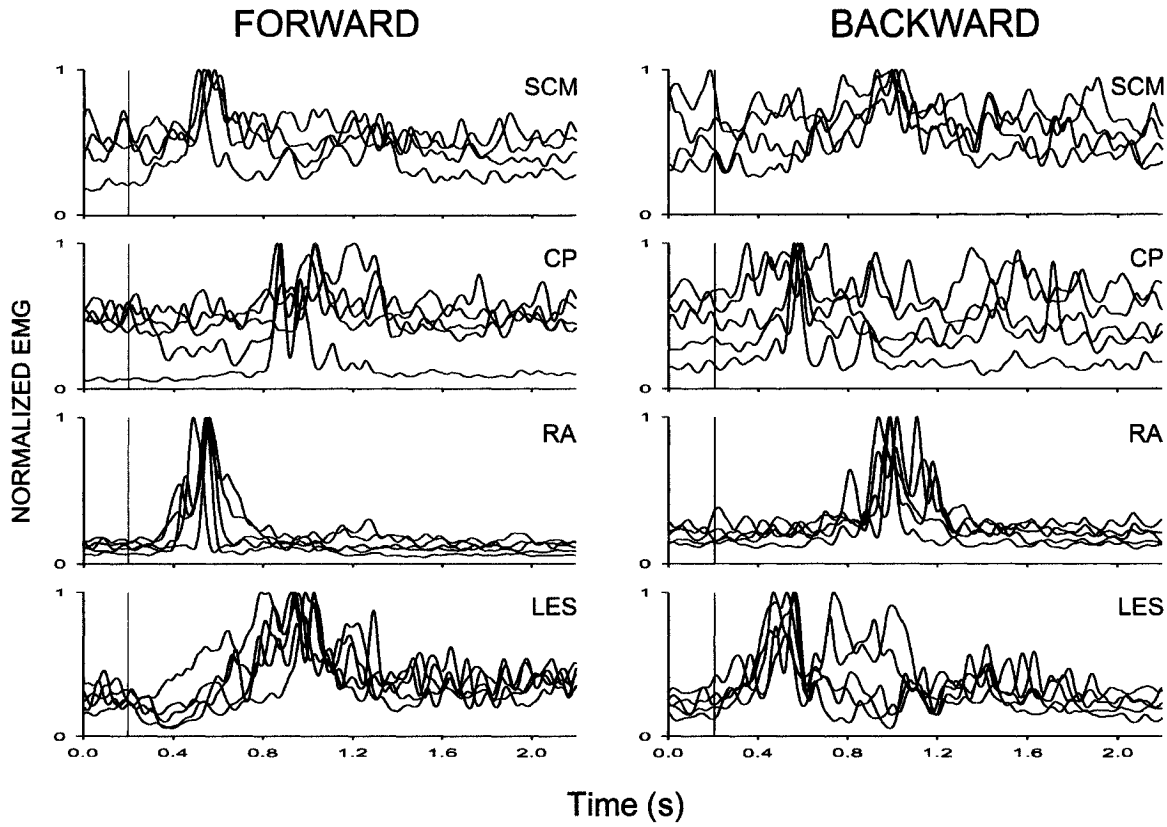

Figure 4. EMG envelopes for SCM, CP, RA, and LES in five subjects for 500-ms perturbations. Averaged data of up to five trials are shown for each subject. Data was normalized to the maximum of averaged envelopes for each subject. Vertical lines show onset of platform movement.

Discussion

We identified a low-intensity threshold perturbation (15 cm translation in 500 ms) that provokes postural reactions in healthy seated individuals. Using such a perturbation, we observed stereotypical electromyographic and kinematic responses in our subjects. Appropriate movement patterns and muscle actions were generated to restore the projection of the body's center of mass (COM) within the base of support following surface translations. Similar studies have been done in sitting (Blouin et al. 2003a; Blouin et al. 2003b; Brault et al. 2000; Forssberg and Hirschfeld 1994; Keshner 2003; Siegmund

et al. 2002; Siegmund et al. 2003a; Siegmund et al. 2003b; Vibert et al. 2001) but using higher accelerations. Siegmund et al. (2002) have observed that the onset and amplitude of the muscle and kinematic variables was dependent on the acceleration of perturbations. Since our ultimate goal is to study the reactions to a postural perturbation in a population that is vulnerable to the perturbation intensities, there is a necessity for a study aiming at finding the smallest perturbation that can elicit a stereotypical postural reaction. When cited, the accelerations used in different studies ranged between 4.45 and 14.80 m/s^2. Keshner (2003) tested three different accelerations (1.22, 2.43, and 4.45 m/s^2) and found that muscles responded only sporadically at the lowest intensities. She therefore decided to focus on the data recorded during the 4.45 m/s^2 translations to measure postural responses. In our study, the maximal acceleration reached by the movable platform was 1.5 m/s^2. The consistent responses observed in our study might be explained by the greater distance traveled by the platform (15 cm versus 2.5 cm).

Using biomechanical measures we hope to better understand the postural stabilization characteristics of WAD. We expect surface translations to elicit a variety of postural responses in WAD individuals that will be different from the stereotypical ones observed in healthy individuals. We also expect significant differences between the postural reactions of healthy subjects and those of WAD individuals. Also, an intensive rehabilitation treatment should have a significant effect on the postural reactions of WAD individuals. These results could have impacts at different levels. They could be used as an objective basis to decide about the management of WAD individuals in rehabilitation approaches. The results could also be used to make recommendations about clinical strategies. They could also indicate the improvement of the functional status of individuals at critical stages of their rehabilitation.

In summary, there is a need to objectify biomechanical parameters of postural stabilization associated with WAD. By measuring muscular and kinematic compensations during mechanical perturbations, postural adaptations with respect to task and environmental constraints can be evaluated and modified if necessary. We believe that

we found a low-intensity mechanical perturbation which will allow the characterization of postural reactions of WAD individuals before and after treatments.

Acknowledgments

We thank Richard Preuss for sharing scripts for data analysis. N.S. is supported by a MENTOR post-doctoral scholarship and I.P. is supported by a MENTOR and an NSERC M.Sc. scholarship. Equipment has been purchased with the help of CFI and JRH foundation.

SECOND RESEARCH ARTICLE

The effects of whiplash-associated disorders on the kinematic and the electromyographic responses of individuals submitted to anterior surface translations in the sitting position

Isabelle Patenaude, Julie N. Côté, Nancy St-Onge, Joyce Fung

Abstract

Individuals with whiplash-associated disorders have been shown to display decreased postural stability during stance and gait tasks as indicated by their increased body sway in comparison with healthy persons. However, their ability to stabilize their posture following support surface perturbations has not been studied yet. The goal of this study was to characterize balance and postural control patterns of individuals with chronic whiplash-associated disorders and to compare these patterns with those of healthy individuals. Nine individuals with whiplash-associated disorders and seven healthy controls were submitted to anterior support surface translations in the sitting position. Their head and trunk kinematics and their neck and trunk muscle activation responses were assessed. We found that whiplash individuals displayed an earlier onset of their head displacement and a pattern of trunk displacement characterized by greater flexion at the upper levels of the spine, compared to the lower levels. Moreover, whiplash individuals presented a tendency for a late recruitment of their neck flexors and a greater use of a pattern of neck extensor muscle inhibition. These results suggest that individuals with WAD may compensate their altered neck functional ability by modifying their relative movements along the spine and by adopting altered motor strategies to compensate for their painful muscles. The results of our study highlight the importance of considering whiplash-associated disorders as a pathology that involves all segments of the trunk and not only the cervical region.

Introduction

Whiplash injuries cause a significant societal and economic burden to most industrialized societies, in health care as well as in insurance costs. In the Canadian province of Quebec, the incidence of whiplash cases has previously been reported to be 70 per 100 000 inhabitants, representing approximately 5000 cases annually and accounting for 20% of all traffic injury insurance claims (Spitzer et al. 1995). A whiplash can be defined as a sudden movement of the head which occurs during the 100 to 200 ms following an impact in any direction (Sjöström et al. 2003). It may result in injuries to neck muscles,

ligaments, fascia, intervertebral discs, nerves, facet joints, articular cartilage, and joint capsules (Loudon et al. 1997), which may in turn lead to a wide variety of clinical manifestations grouped in a common class called whiplash-associated disorders (WAD). Most patients recover from whiplash injuries within eight weeks (Suissa et al. 2006). However, 4% to 42% will maintain chronic symptoms even several years after the trauma (Eck et al. 2001). Chronic symptoms associated with WAD include neck pain, headache, visual disturbances, dizziness, weakness, paraesthesia, concentration and memory disturbances (Barnsley et al. 1994), neck stiffness, temporomandibular joint pain, tinnitus, dysphagia, decreased range of motion (Spitzer et al. 1995), decreased proprioception at the neck level (Loudon et al. 1997) as well as abnormal eye movements (Rubin et al. 1995).

Symptoms of neck pain and of limited neck movement suggest that changes in neck muscle behavior might be characteristic of chronic WAD. It was previously reported that individuals with chronic WAD show coactivation of their upper trapezius (UT) in the execution of a unilateral upper limb dynamic task, in addition to a hyperreactivity in their UT (Nederhand et al. 2000, 2002), anterior scalenus (AS) and sternocleidomastoid (SCM) (Falla et al. 2004a), as well as a generally decreased ability to relax their neck muscles following an upper limb task. During the craniocervical flexion test (CCFT), a progressively staged performance of active craniocervical flexion (similar to a head nod action), chronic whiplash individuals have been reported to exhibit increased activity in the superficial neck flexors (SCM and AS; Falla et al. 2004b; Sterling et al. 2003) concomitantly with decreased activity in deep neck flexors. These findings indicate that WAD is characterized with muscle dysfunctions at the cervical level and altered neck muscle recruitment patterns during functional tasks. Moreover, we know that in certain trunk pathologies, alterations in the normal patterns can be observed not only at the directly injured structures but also at the neighboring regions of the trunk. This has been recently shown in patients with low-back pain (Larivière et al. 2000). However, the possibility that the trunk musculature may play a role in compensating for the cervical musculature deficits in WAD individuals has never been investigated.

Symptoms of dizziness and unsteadiness reported after a whiplash trauma suggest that postural control might be another aspect affected by a whiplash. It has been frequently observed that postural responses are generated in reaction to internal or external perturbations of the equilibrated posture. We know that visual, vestibular and somatosensory afferences are involved in the detection of postural disturbances and are combined in an integrative fashion by the central nervous system (CNS) to trigger postural adjustments. However, the mechanisms of whiplash and how it influences the systems involved in postural and motor control are not well understood. The literature suggests that the motor deficits associated with WAD can be due to an imbalance of postural central commands (Chester 1991) resulting from an impairment of the neck proprioceptive mechanisms (Gimse et al. 1997; Treleaven et al. 2003) or of the vestibular system (Rubin et al. 1995). However, the level at which the systems are perturbed is still not well identified and it follows that the complexity of the motor limitations associated with this pathology is not well understood. Kogler et al. (2000) and Madeleine et al. (2004) used posturographic protocols to show that WAD individuals display larger amplitude of their center of pressure (COP) displacements in standing and are more importantly affected by sensory perturbations while trying to maintain an equilibrated standing posture, in comparison with healthy individuals. Similarly, Michaelson et al. (2003) found that standing WAD individuals executing movements with the upper limbs display greater amplitude of COP trajectory as well as greater amplitude of head translation. In this study, WAD individuals were also less able to maintain standard standing positions (tandem, Romberg). A subsequent study showed that WAD patients display greater trunk sway during stance tasks and during complex gait tasks that required task-specific gaze control. However, they showed reduced trunk sway in simple gait tasks that demanded large head movement but no task-specific gaze control (Sjöström et al. 2003). These results indicate that chronic WAD affects the performance of both gait and standing stance tasks, even more so in altered sensory conditions, suggesting possible deficits in the integration mechanisms employed to produce stable motor tasks.

Postural strategies used to maintain an equilibrated posture can also be investigated using protocols of mechanical perturbations, typically performed by inducing sudden displacements of the support surface. In standing healthy subjects, it has been well described that appropriate movement patterns and muscle actions are generated by the system to maintain the projection of the body's center of mass (COM) within the limits of the base of support following multidirectional surface translations (Henry et al. 1998; Horak and Nashner 1986; Nashner 1976; Nashner 1983). For instance, in response to anterior and posterior support surface translations, stereotypical patterns occur in the leg and trunk muscles. One of these patterns is characterized by the early activation of the stretched ankle muscles followed by a response in the posterior thigh muscles and finally in the posterior trunk muscles. Larger and faster perturbations, as well as perturbations in various directions, rather trigger early activation of trunk muscles followed by the thigh muscles, indicating that muscles not necessarily stretched but that are functionally relevant to an appropriate postural response can also play important parts in the stabilization mechanisms. Other studies have characterized healthy response patterns to perturbations applied to the sitting posture (Blouin et al. 2003a; Blouin et al. 2003b; Brault et al. 2000; Forssberg and Hirschfeld 1994; Keshner 2003; Siegmund et al. 2002; Siegmund et al. 2003a, Siegmund et al. 2003b; Vibert et al. 2001; Zedka et al. 1998). These studies did not demonstrate a clear caudo-cranial sequence of muscle activation in response to anteroposterior translations. However, it remains that by combining kinematic and electromyographic (EMG) data, the literature on postural perturbations as a whole has consistently identified that trunk and neck muscles are activated when they are lengthening, during backward body sway for flexors and during forward body sway for extensors.

Although these studies have documented the sitting postural stabilization mechanisms in healthy subjects, much less is known about the way that these are affected by pathologies. In particular, such perturbation protocols have never been included in studies of postural stabilization mechanisms of individuals with chronic WAD. The purpose of this study was to characterize balance and postural control patterns of individuals with chronic WAD and to compare these patterns with those of healthy individuals. More specifically,

we wanted to characterize postural reactions in response to anterior translations in the sitting position by way of kinematic and EMG analyses. Since most studies have investigated the control of the standing posture in WAD individuals, and since several tasks of everyday life are conducted from a sitting position (driving, computer work,...), studies are needed to better understand how WAD affects the sitting posture. The sitting position was also selected in order to allow us to focus our analysis on the role of the spine segments in stabilizing posture, since in this position the potential contribution of the lower limbs in stabilizing posture is cancelled. We hypothesized that the deficit in the ability to stabilize the posture of WAD individuals could be characterized by changes in the amplitude and in the timing of trunk and head movements, as well as changes in the organization of the recruitment patterns of neck and trunk muscles. In turn, we hypothesized that with information on head and trunk kinematics and muscle activity we could characterize deficits in the ability to stabilize the sitting posture of WAD individuals.

Methods

Subjects

A group of nine patients with chronic whiplash-associated disorders (WAD) aged 22-57 years (mean age = 37.7; 6 women, 3 men) and seven healthy controls aged 25-45 years (mean age = 33.9; 4 women, 3 men) participated in this study. Patients were recruited from the clinical return to work program of the Jewish Rehabilitation Hospital in Laval, Qc. All patients were at least three months post injury (see Table 1). Patients who had suffered concussion, head trauma, cervical dislocation or fracture, spinal cord injury, or with previous history of whiplash injury were excluded. Healthy and WAD subjects having been diagnosed with any other neck or back, musculoskeletal or neurological problems were also excluded. Approval of the institutional ethics committee had been previously obtained and all participants signed an informed consent form before participating in the study.

Table 1. Characteristics of patients with chronic WAD.

Subject	Gender	Age	Grade*	Time elapsed since whiplash injury (months)
1	F	57	—	—
2	F	22	—	—
3	F	25	II	10.0
4	F	48	—	4.5
5	M	41	II	7.5
6	M	35	—	5.0
7	M	27	—	4.5
8	F	53	—	9.0
9	F	31	II	3.5

* The grade of WAD refers to the Quebec classification of WAD proposed by the Quebec Task Force on WAD (Spitzer et al. 1995). The grade is an indication of the severity of the whiplash injury and the associated symptoms.

Experimental protocol

Subjects were sitting on an experimental chair that supported the pelvis and the knees. A seat belt was attached to the thighs in order to fix the legs on the chair and firm cushions were adjusted on each side of the subject to stabilize the hips and the knees. This chair design also served to standardize the hip and knee angles (Figure 1). The trunk was free to move in all planes. The participants were instructed to cross their arms on their torso, keep their eyes open, look ahead and stay relaxed throughout trials. In this position, three static trials were first recorded. With the same instructions, participants were then submitted to forward or backward translations of 15 cm in 500 ms at a peak velocity of 0.3 m/s (peak acceleration of 1.5 m/s^2). This perturbation had been previously selected from pilot work that aimed at determining the weakest translation perturbation that elicited a postural stabilization reaction (St-Onge et al. submitted). The perturbations were generated by a movable platform that was under the control of a hydraulic servomotor and on which the chair was fixed. The protocol included a total of 15 trials (5 forward, 5 backward and 5 unperturbed trials) randomly presented. Subjects were informed prior to each trial ("Ready? One, two, three, go!"); however they were not warned if the trial would be a forward perturbation, a backward perturbation, or a no-

perturbation trial. After the aforementioned protocol, three static trials were again recorded.

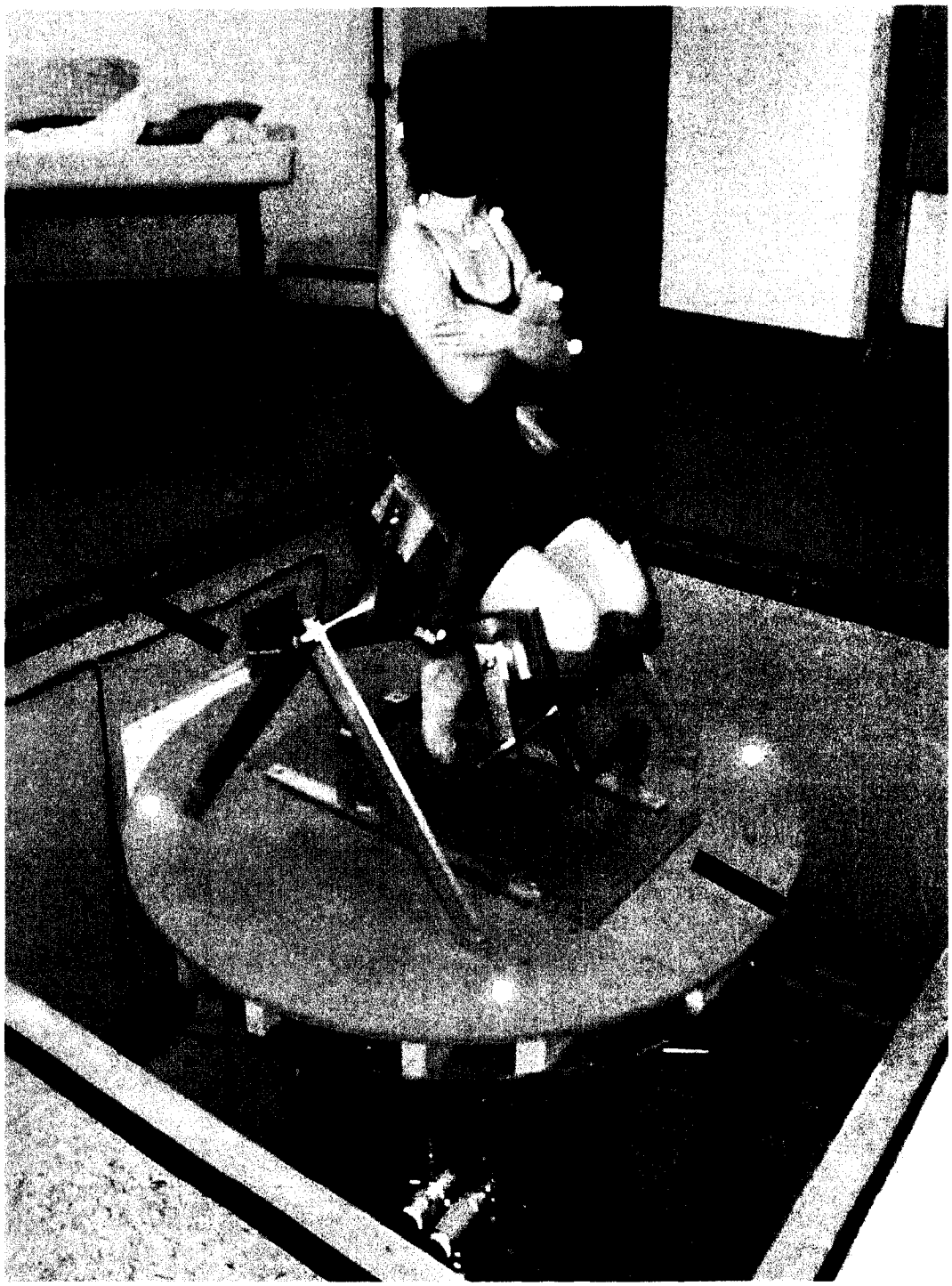

Figure 1. Adapted ergonomic chair bolted on a movable platform. The subject is stabilized to the chair using a belt and stabilizing devices aligned with her hips and lower legs, so that the movement is only allowed above the pelvis.

Data acquisition

Kinematics of the platform, the head, the arms and the trunk was recorded using a high resolution six-camera Vicon 512 Motion Analysis System (Vicon Peak, UK). Passive reflective markers were placed on the following anatomical landmarks: left and right front head, left and right back head, C7 spinous process, right scapula, left and right T1 transverse processes, T6 spinous process, left and right T8 transverse processes, T12 spinous process, left and right L1 transverse processes, S1, left and right postero-superior iliac spine, apex of sacrum, sternal notch, left and right acromia, left and right lateral epicondyle, and left and right head of third metacarpal. Reflective markers were also placed on each corner of the movable platform to record its position in tridimensional space. Kinematic data was sampled at 120 Hz. In order to allow the computation of the center of mass (COM), the following anthropometric measurements were taken: trunk depth at T1, T4, T10, and L3 levels; body height and body weight.

The activity of sixteen muscles of the neck and trunk was acquired using the Telemyo 900 electromyographic system (Noraxon, USA). After preparing the skin following standard procedures (cleaning, shaving, lightly abrading), bipolar surface Ag/AgCl electrodes (Ambu, DE) were placed bilaterally on the skin overlying the following neck and trunk muscles: scalenus (SCA), sternocleidomastoid (SCM), cervical paraspinals (CP), upper trapezius (UT), erector spinae (thoracic level; TES), erector spinae (lumbar level; LES), rectus abdominis (RA), external obliques (EO). A ground electrode was placed on the head of humerus. Electromyographic (EMG) data was sampled at 1800 Hz.

Data analysis

Kinematic data was low-pass filtered (zero-lag second order Butterworth filter, 6 Hz). The head segment was defined using the four head markers and the trunk segment was defined using markers of S1 and both left and right T1 transverse processes. Head and trunk angular displacements were computed by quantifying the orientation of the head and trunk segments relative to the global space using Euler xyz rotations. In this paper,

we analyzed only angular displacements in the sagittal plane. The segmental COMs of the head, the upper thorax (T1-T6) (including the arms), the lower thorax (T6-L1) and the abdomen (L1-S1) were computed using segmental measurements and standard anthropometric tables.

Platform displacement was computed using the four markers positioned on each corner of the platform. The positions of these markers were averaged to find the position of the center of the platform. Platform onset was taken as the time when the velocity of its center reached 2% of its maximal velocity. Angle and COM onsets were taken as the time when their velocities first surpassed 5% of the respective maximum velocities, and COM offsets when their velocities decreased under 5% of the respective maximum velocities. For head and trunk angles, the initial position was considered as an angle of zero. We computed the peak head and trunk angular displacements (corresponding to peak extension and flexion angles), the time to peak angular position (time between platform onset and the instant when each peak angle was reached) and the total head and trunk angular excursions (peak flexion angle – peak extension angle). For each segmental COM, we computed the amplitude of COM displacement (maximum position – initial position), the COM final position relative to platform final position, the COM overshoot (maximum COM position – final COM position at the time of COM offset) and the time to peak COM position (time between platform onset and the instant when each peak COM was reached).

The EMG signals were amplified (2000 X). Using a 40Hz low-pass second order Butterworth filter, each heartbeat was filtered out from the EMG signal and subsequently subtracted from the signal. EMG data with heartbeats removed was then band-pass filtered (zero-lag second order Butterworth filter, 10-350 Hz) and full-wave rectified. The mean and the standard deviation (SD) of each baseline signal were calculated using the signal from 300 ms to 100 ms before platform onset. EMG envelopes were then computed using a 20-Hz zero-lag second order Butterworth filter. The onset of each muscle burst was identified on the EMG envelopes as the first burst that was > 2 SD above the baseline mean for at least 25 ms. From this point, the EMG burst was followed

back to the baseline mean and the latency relative to the platform onset was recorded to represent the onset of the muscle burst. Inhibition onset was identified when the muscle displayed an activity level < ½ SD under the baseline mean for at least 25 ms. All analyses were computed using Matlab v. 6.5.1 (The MathWorks, Inc., Natick, MA, USA).

Statistical analysis

Data was first averaged over trials for each subject when at least three trials were available. For each kinematic dependent variable, the data was analyzed using a two-way repeated-measures analysis of variance (ANOVA) to assess differences related to the group (whiplash, healthy) and to the segment analyzed within the groups (for segmental angles: head and trunk; for COM: head, upper thorax, lower thorax and abdomen).
For EMG onsets of trunk muscles, the data was first analyzed using a two-way repeated-measures ANOVA which revealed no differences between left and right muscles in healthy and whiplash groups. EMG onsets of right trunk muscles were further analyzed in a two-way repeated-measures ANOVA to evaluate differences related to the group (whiplash, healthy) and to the muscle analyzed within the groups (RA, EO, TES, LES). When a significant main effect or interaction effect was found, *post-hoc* analyses were performed using a Tukey test. All statistical tests were performed using Statistica, v.7 (Statsoft, Tulsa. OK, USA) and statistical significance was set at $p < 0.05$.

Results

Kinematics

Angles

Following forward perturbations, the head and trunk segments first rotated in extension and then in flexion before moving back towards their initial position in a second extension (Figure 2 and Figure 3). A deeper analysis of head and trunk segment

Figure 2. Sequence of head and trunk movements following a forward perturbation. A. Position before the onset of the platform. B. Peak extension. C. Peak flexion. D. Final trunk and head position.

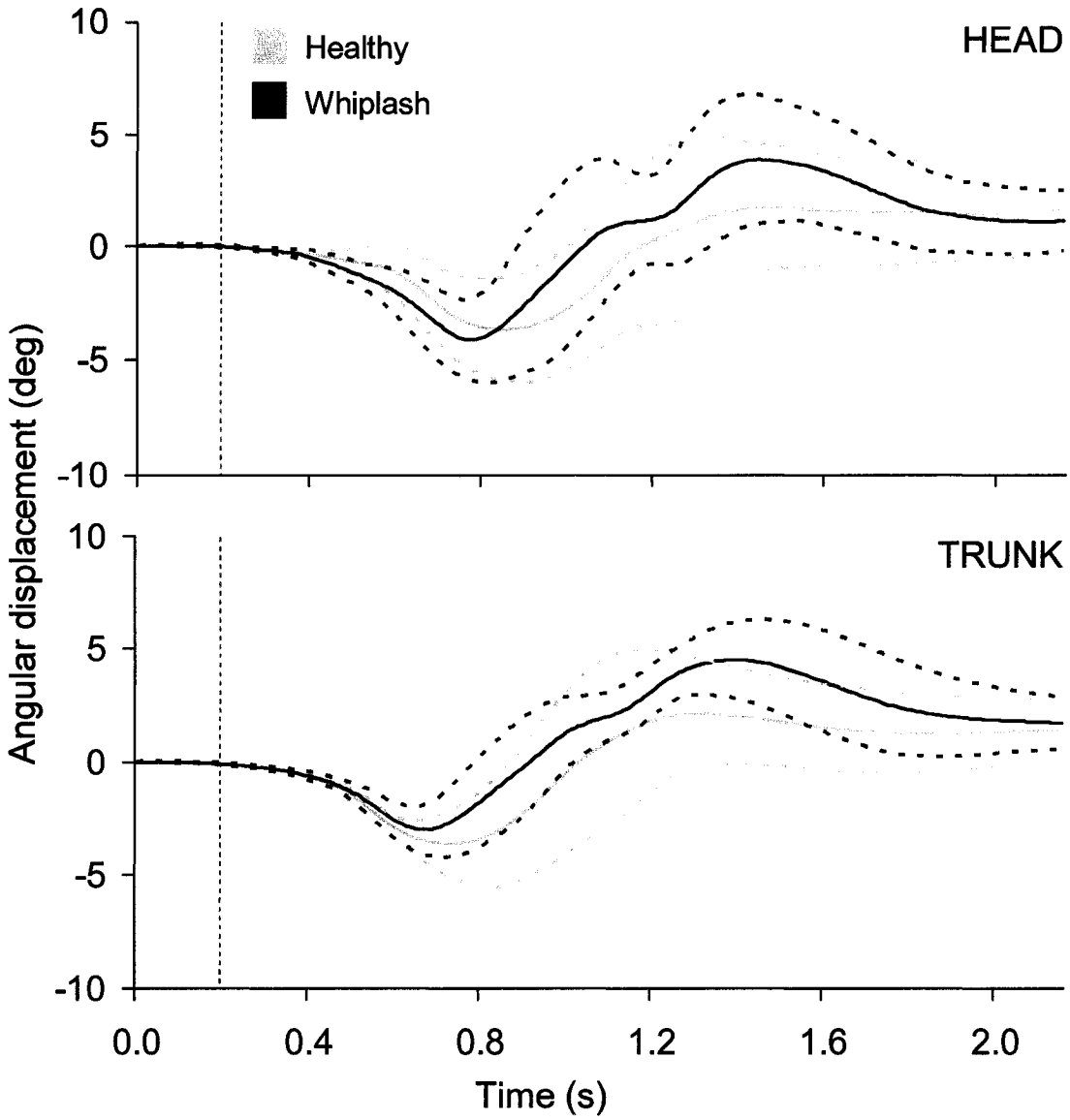

Figure 3. Averaged curves of head and trunk angular displacement over time following a forward perturbation in healthy and whiplash groups. The thick curves represent the average of all trials for all subjects within the group. The dashed thick curves represent intervals of one standard deviation. The dashed vertical line indicates the platform onset. Positive angular displacement indicates flexion.

displacements showed that the flexion movement brought the head, upper thorax, lower thorax and abdomen segments to overshoot their initial position. This kinematic behavior was observed in all subjects. The average trunk angle onsets were 77 ms ± 27 ms (healthy) and 66 ms ± 45 ms (whiplash), whereas average head angle onset values were 224 ms ± 87 ms (healthy) and 118 ms ± 79 ms (whiplash), relative to platform onset (Table 2). The ANOVA of angle onsets revealed significant effects of group, of angle and of interaction group by angle. *Post-hoc* analysis showed that in whiplash individuals, the head started to move earlier than in healthy controls ($p < 0.05$). Also, the trunk started to move before the head in healthy individuals ($p < 0.01$) but not in whiplash individuals ($p > 0.05$).

The average times to reach peak extension and flexion were calculated in order to better compare the kinematic behavior of both groups following trunk and head onsets (Table 2). On average, for both groups combined, the times to peak extension and flexion were 636 ms ± 74 ms and 1274 ms ± 171 ms, respectively for the trunk, and 753 ms ± 94 ms and 1342 ms ± 175 ms, respectively, for the head. The ANOVA analysis showed that there was a significant difference in the time to peak extension between healthy and whiplash groups, but no significant difference between both groups in time to peak flexion. This analysis also revealed differences between time to peak extension and flexion amongst different angles. *Post-hoc* analysis indicated that in general, whiplash individuals reached peak extension before healthy individuals ($p < 0.05$). Moreover, in both groups the trunk peak extension angle was reached before the head peak extension angle ($p < 0.001$) and the trunk peak flexion angle was reached before the head peak flexion angle ($p < 0.01$).

Table 2. Mean (SD) head and trunk onsets, time to peak extension and time to peak flexion (ms) in healthy and whiplash groups.

	Healthy				Whiplash				ANOVA		
	Trunk	n	Head	n	Trunk	n	Head	n	G	A	G x A
Onset	77 (27)	5	224 (87)	7	66 (45)	9	118 (79)	9	*	***	*
Peak extension	695 (78)	6	808 (119)	6	597 (40)	9	716 (52)	9	*	*****	
Peak flexion	1283 (221)	7	1350 (225)	6	1267 (134)	9	1337 (147)	9		*	

n = number of subjects for which the respective parameters could be successfully calculated.
G = Group, A = Angle.
* Significantly different at $p < 0.05$
*** Significantly different at $p < 0.001$
***** Significantly different at $p < 0.00001$

Finally, on average, the total angular excursion was 7.2° ± 2.2° (healthy) and 8.8° ± 2.0° (whiplash) for the trunk and 7.5° ± 3.3° (healthy) and 10.6° ± 5.5° (whiplash) for the head. The ANOVA did not reveal significant effects of group, of angle or of interaction group by angle for head and trunk total angular excursion, as well as for head and trunk peak extension and peak flexion (Figure 4). It should be noted that the data presented in all Tables and Figures are the mean (SD) of all subjects available for each variable. However, only the subjects with complete data for a given variable could be included in the corresponding ANOVA computations.

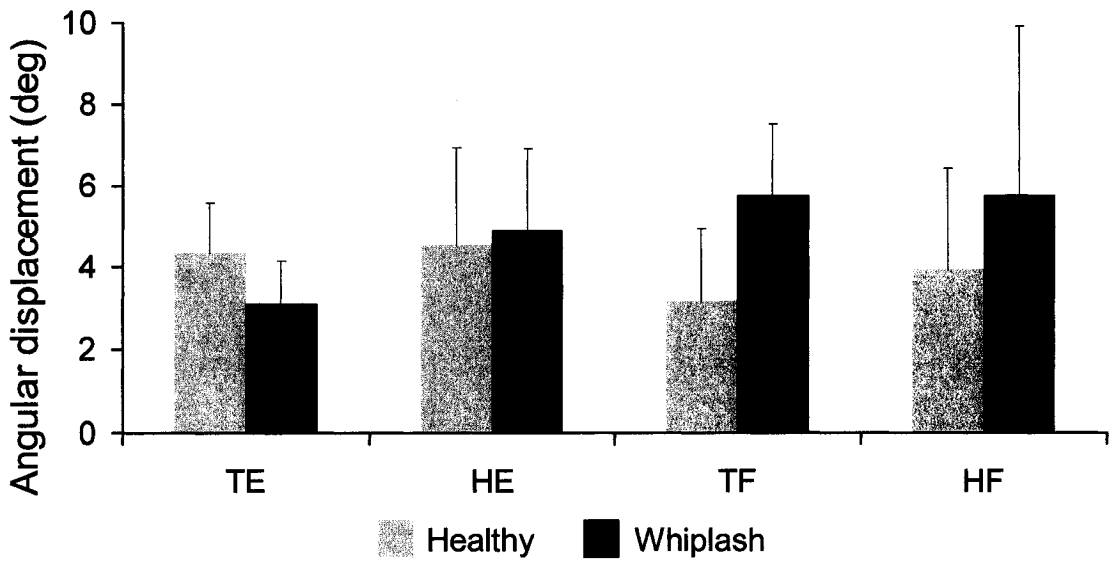

Figure 4. Mean values of peak extension and peak flexion angles in healthy and whiplash groups. The error bars correspond to one standard deviation. TE = trunk extension, HE = head extension, TF = trunk flexion, HF = head flexion.

COM

The head, upper thorax, lower thorax and abdomen COMs all moved forward during the perturbations, with greater displacements than the platform and these displacements lagging in time behind those of the platform (Figure 5). The COM analysis did not reveal a group effect (healthy versus whiplash) for any of the COM parameters. However, there

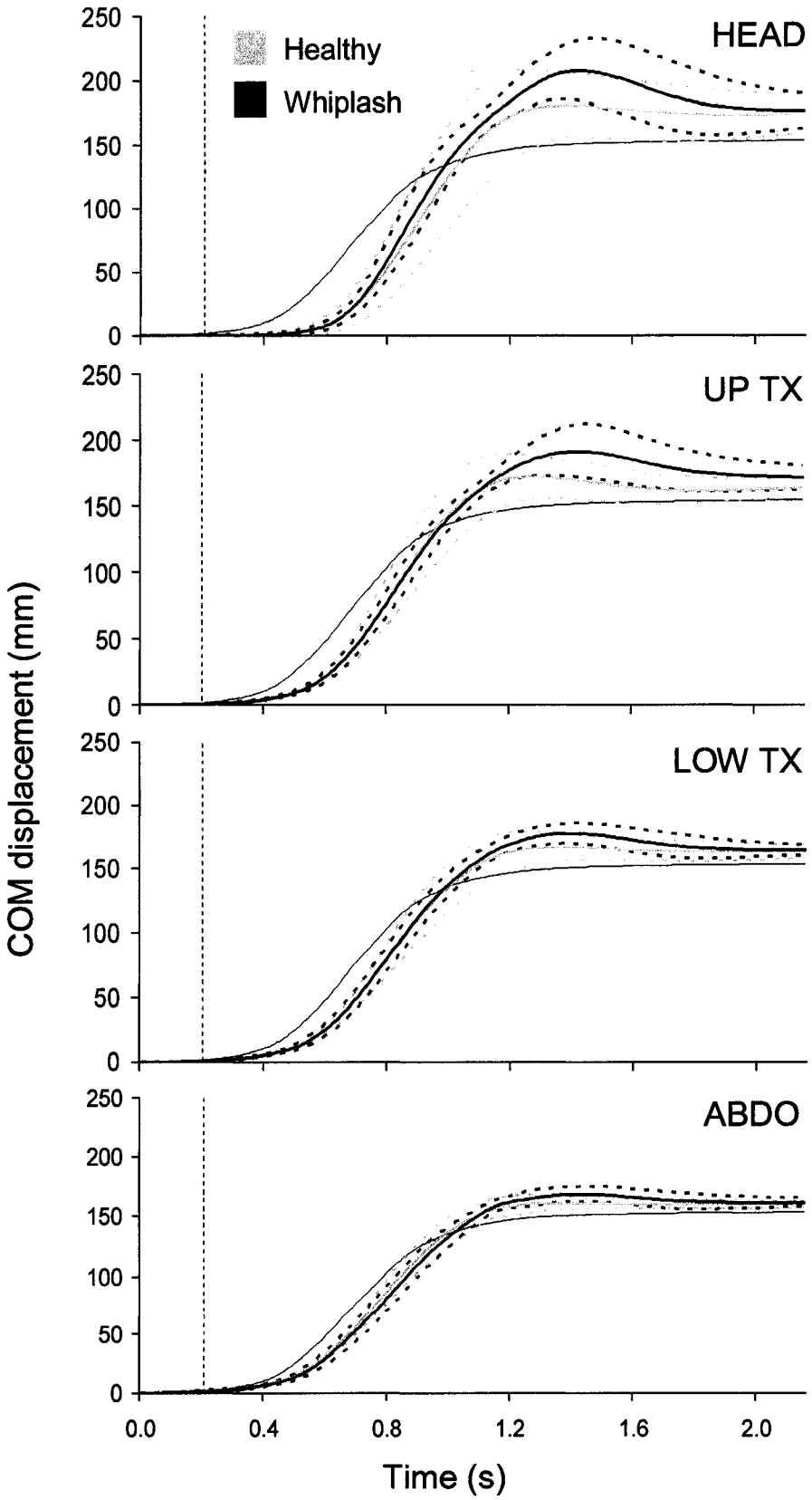
Healthy
Whiplash
HEAD
UP TX
LOW TX
ABDO
COM displacement (mm)
Time (s)
0
50
100
150
200
250
0.0
0.4
0.8
1.2
1.6
2.0

Figure 5. Averaged curves of head, upper thorax, lower thorax and abdomen COM displacement over time following a forward perturbation in healthy and whiplash groups. The thick curves represent the average of all trials for all subjects within the group. The dashed thick curves represent intervals of one standard deviation. The thin curve represents the platform displacement. The dashed vertical line indicates the platform onset. HEAD = head segment, UP TX = upper thorax segment, LOW TX = lower thorax segment, ABDO = abdomen segment.

was a significant interaction effect of group by COM for some variables. The onsets of COM displacement were calculated and the ANOVA revealed a significant effect of COM. *Post-hoc* analyses indicated that overall, the abdomen COM started to move first, followed by the lower thorax COM, then by the upper thorax COM and finally by the head COM (Table 3). The onsets of segmental COM for each group are presented in Table 4. For the time to reach peak COM position, no significant effect of group, of COM or of interaction group by COM was found (Table 5).

Differently to what was found with angles, the analysis of the amplitude of COM displacement of head, upper thorax, lower thorax and abdomen revealed some differences between the behavior of the head and that of the other segments of the trunk, indicated by significant effects of COM and of interaction group by COM. *Post-hoc* analyses showed that in the healthy group, the amplitude of COM displacement of the head (193 mm ± 25 mm) was greater than that of the lower thorax (171 mm ± 10 mm) ($p < 0.05$) and abdomen (163 mm ± 6 mm) ($p < 0.001$) segments. In the whiplash group, the amplitude of COM displacement of the head (220 mm ± 26 mm) was greater than that of the upper thorax (193 mm ± 19 mm), lower thorax (183 mm ± 11 mm) and abdomen (170 mm ± 7 mm) segments ($p < 0.001$). Additionally, the amplitude of COM displacement of the upper thorax was greater than that of the abdomen segment ($p < 0.05$) (Figure 6). The same differences between the segments and between the two groups were found for the COM overshoots (Figure 7). The COM final position relative to platform final position was calculated and the ANOVA indicated a significant effect of COM. *Post-hoc* analyses showed that in all subjects taken together, the head COM final position was more in front of the platform (14 mm ± 10 mm) than upper thorax (8 mm ± 6 mm), lower thorax (6 mm ± 4 mm) ($p < 0.01$) and abdomen (5 mm ± 3 mm) segments ($p < 0.001$).

Table 3. Mean (SD) onsets (ms) of COM displacement of both groups collapsed and the corresponding p-values of the *post-hoc* analyses.

	Onset	n	Abdomen	Lower thorax	Upper thorax	Head
Abdomen	153 (42)	15	—	*	***	***
Lower thorax	209 (41)	13	—	—	*	***
Upper thorax	263 (39)	12	—	—	—	***
Head	420 (54)	16	—	—	—	—

n = number of subjects for which the respective parameters could be successfully calculated.
* Significantly different at $p < 0.05$
*** Significantly different at $p < 0.001$

Table 4. Mean (SD) onsets (ms) of COM displacement in healthy and whiplash groups.

	Healthy	n	Whiplash	n
Abdomen	157 (39)	7	150 (46)	8
Lower thorax	200 (47)	7	219 (35)	6
Upper thorax	261 (48)	5	264 (36)	7
Head	416 (54)	7	422 (58)	9

n = number of subjects for which the respective parameters could be successfully calculated.

Table 5. Mean (SD) times (ms) to reach peak COM position in healthy and whiplash groups.

	Healthy	n	Whiplash	n
Abdomen	1439 (225)	7	1348 (47)	8
Lower thorax	1648 (550)	7	1341 (87)	8
Upper thorax	1628 (534)	7	1352 (92)	7
Head	1711 (634)	7	1331 (84)	9

n = number of subjects for which the respective parameters could be successfully calculated.

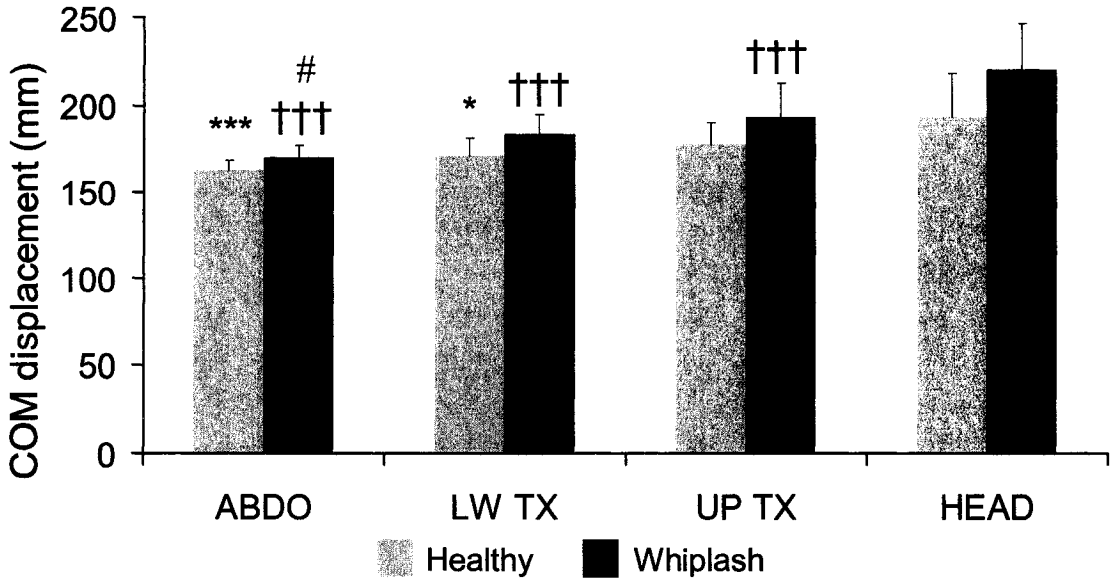

Figure 6. Mean values of COM displacement amplitudes in healthy and whiplash groups. The error bars correspond to one standard deviation. HEAD = head segment, UP TX = Upper thorax segment, LOW TX = lower thorax segment, ABDO = abdomen segment. * Significantly different from healthy head segment at $p < 0.05$, *** Significantly different from healthy head segment at $p < 0.001$, ††† Significantly different from whiplash head segment at $p < 0.001$, # Significantly different from whiplash upper thorax segment at $p < 0.05$.

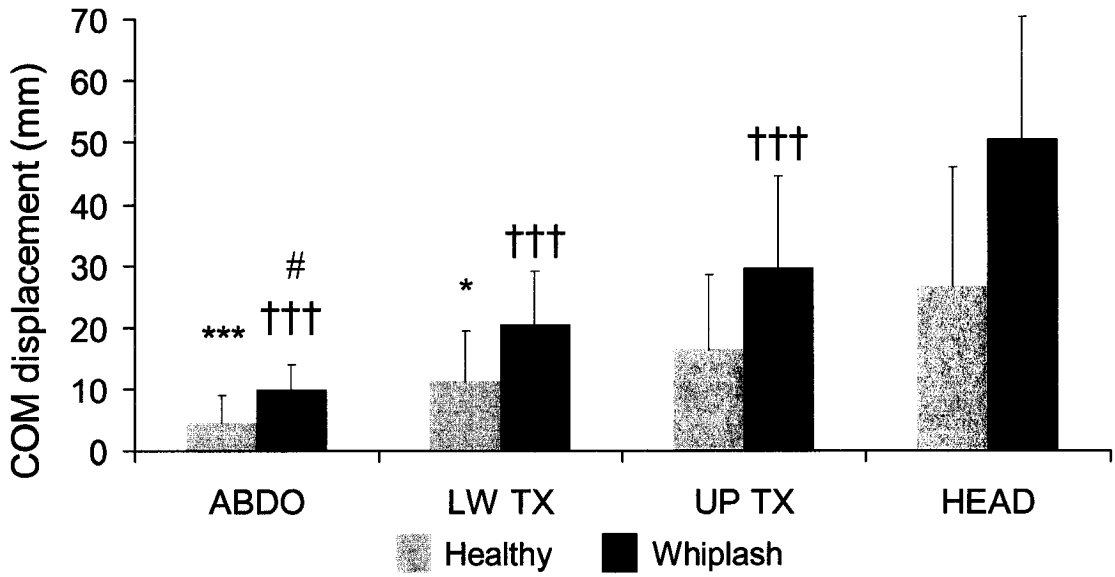

Figure 7. Mean values of COM displacement overshoot in healthy and whiplash groups. The error bars correspond to one standard deviation. HEAD = head segment, UP TX = upper thorax segment, LOW TX = lower thorax segment, ABDO = abdomen segment. * Significantly different from healthy head segment at $p < 0.05$, *** Significantly different from healthy head segment at $p < 0.001$, ††† Significantly different from whiplash head segment at $p < 0.001$, # Significantly different from whiplash upper thorax segment at $p < 0.05$.

EMG

Figure 8 shows a typical example of EMG signals obtained in a healthy subject. The selected forward perturbation provoked consistent activation of trunk muscles in almost all subjects and activation of neck muscles in about half of subjects, with the exception of the UT which was activated only in a few subjects. Moreover, some neck and trunk extensors were characterized by a period of muscle inhibition preceding the burst of activation. This specific pattern was more common in back extensors of healthy individuals (approximately n = 6) and in neck extensors of whiplash individuals (approximately n = 4). It was also observed in flexor muscles of a few whiplash subjects. While performing statistical analysis of EMG parameters, the data of neck muscles (Table 6) was not included in the statistical design because an insufficient amount of subjects displayed clear activation bursts for these muscles. For trunk muscles, the ANOVA did not reveal a group effect on muscle onset values; however it did reveal a significant effect of muscle. *Post-hoc* analyses indicated that for both groups taken together, the onsets of RA (319 ms ± 72 ms) and of EO (372 ms ± 181 ms) were smaller than the onsets of TES (574 ms ± 134 ms) and LES (611 ms ± 177 ms), indicating that trunk flexors (RA and EO) were recruited on average earlier than the trunk extensors (TES and LES) (Table 7). The onsets of trunk muscles for each group are presented in Table 8. For each muscle in which a period of inhibition was present, the onsets (Table 9) were calculated. The TES and LES muscles showed consistent inhibition periods in several subjects among both groups, with average inhibition onset times (right side) of 165 ms ± 40 ms (healthy) and 164 ms ± 74 ms (whiplash) for the TES, and 137 ms ± 60 ms (healthy) and 140 ms ± 59 ms (whiplash) for the LES. However, this pattern was not observed consistently enough in either group to warrant further statistical analysis.

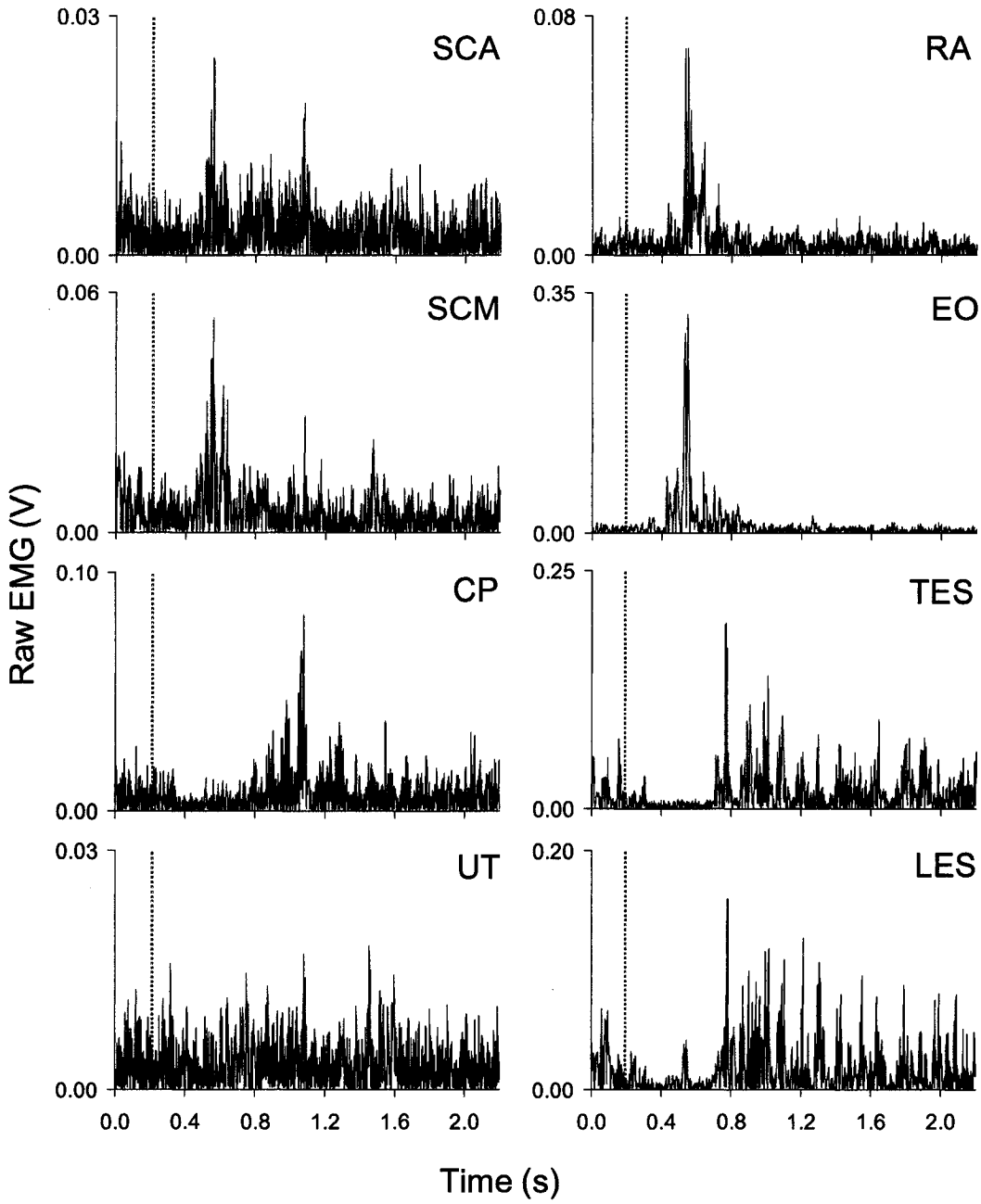

Figure 8. Typical example (healthy subject) of electromyographic signals obtained in response to a forward perturbation in the eight muscles tested. The vertical dashed line indicates the platform onset. SCA = scalenus, SCM = sternocleidomastoid, CP = cervical paraspinal, UT = upper trapezius, RA = rectus abdominis, EO = external oblique, TES = thoracic erector spinae, LES = lumbar erector spinae.

Table 6. Mean (SD) EMG onsets (ms) of neck muscles in healthy and whiplash groups.

	Healthy				Whiplash			
	Right	n	Left	n	Right	n	Left	n
SCA	313 (94)	6	328 (75)	4	467 (163)	5	420 (114)	5
SCM	351 (62)	4	318 (20)	5	467 (209)	7	421 (29)	4
CP	704 (263)	5	768 (166)	2	708 (191)	8	588 (151)	5
UT	1223	1	—		524 (193)	2	688	1

n = number of subjects for which the respective parameters could be successfully calculated.

Table 7. Mean (SD) EMG onsets (ms) of trunk muscles of both groups collapsed and the corresponding p-values of the *post-hoc* analyses.

	Onset	n	RA	EO	TES	LES
RA	319 (72)	15	—		**	***
EO	372 (181)	15	—	—	*	**
TES	574 (134)	13	—	—	—	
LES	611 (177)	16	—	—	—	—

n = number of subjects for which the respective parameters could be successfully calculated.
* Significantly different at $p < 0.05$
** Significantly different at $p < 0.01$
*** Significantly different at $p < 0.001$

Table 8. Mean (SD) EMG onsets (ms) of trunk muscles in healthy and whiplash groups.

	Healthy	n	Whiplash	n
RA	312 (62)	7	325 (83)	8
EO	357 (214)	7	384 (161)	8
TES	585 (182)	6	566 (90)	7
LES	667 (192)	7	567 (161)	9

n = number of subjects for which the respective parameters could be successfully calculated.

Table 9. Mean (SD) EMG inhibition onsets (ms) in healthy and whiplash groups.

	Healthy				Whiplash			
	Right	n	Left	n	Right	n	Left	n
SCA	—		—		164	1	—	
SCM	—		—		127	1	337	1
CP	230	1	—		212	1	248	1
UT	375	1	—		304 (131)	4	210 (150)	3
RA	—		—		—		—	
EO	—		—		213	1	86 (60)	3
TES	165 (40)	6	179 (37)	6	164 (74)	4	112 (50)	2
LES	137 (60)	5	167 (12)	3	140 (59)	3	135 (75)	4

n = number of subjects for which the respective variables could be successfully calculated.

Discussion

The purpose of this study was to characterize how whiplash-associated disorders may influence the systems involved in postural control from a sitting position. Previous work has shown a decreased ability to stabilize the posture in standing and during gait tasks in individuals with WAD (Michaelson et al. 2003; Sjöström et al. 2003). However, postural strategies adopted in the sitting position have not been investigated in this population. We compared the kinematic and muscular responses of WAD and healthy individuals submitted to anterior translations in the sitting position, following the hypothesis that using these techniques, we could quantify deficits in our WAD population.

Temporal sequence of postural stabilization

Our results show that in response to forward perturbations, all subjects displayed a stereotyped kinematic pattern of head and trunk extension, followed by a flexion that overshot the initial position of both segments and finally by a second extension that brought back the head and trunk almost to their initial position, aligned with the pelvis. In the healthy group, the trunk started to move before the head. This sequence of movement between trunk and head segments has been previously observed in sitting (Forssberg and

Hirschfeld 1994; Keshner 2003; Vibert et al. 2001). It has indeed been postulated that following mechanical perturbations, because of its inertia, the head remains stable in space for a longer period of time before moving, in comparison with the trunk (Vibert et al. 2001). However, in the whiplash group, the difference between the onsets of head and trunk angular displacements was not significant and the onset of head displacement was smaller in comparison with the healthy group (i.e. in the WAD group, the head began moving earlier, relative to platform onset, compared to the healthy group). This result suggests that in individuals with WAD, the head stays more in line with the trunk at the initial movement onset, with both segments moving *en block*. This could represent a simplifying strategy employed by the nervous system, possibly to stabilize the vulnerable neck segment against the trunk at the onset of a perturbation. Another interpretation could be that whiplash participants adopted a strategy of reducing head movements to avoid using their painful muscles. However these findings must be considered within the context of the present study (small sample size, low-intensity perturbations and whiplash participants presenting a large variability in their symptoms). Indeed, it is possible that a stronger perturbation may have revealed other protective strategies both in healthy and WAD groups. If faced with a faster perturbation, we may have observed similar protective strategies in both groups or an inability to adopt such strategies. This would be likely to occur especially in the whiplash group in cases where their systems would be unable to produce such protective reactions.

The trunk segment reached peak extension and peak flexion earlier than the head in both groups. For healthy individuals, this means that the sequence between the trunk and the head was conserved during the extension and flexion phases. In whiplash individuals, it indicates that the head began to be dissociated from the trunk after their onsets and maintained this dissociation throughout the remainder of the perturbation, similarly to what was observed in healthy individuals. However, the data also indicates that overall, peak extension was reached earlier in the whiplash group in comparison with the healthy group, and no group difference was observed in the case of peak flexion. Thus, although they displayed different latencies during the extension phase, at the time of peak extension and peak flexion, individuals with WAD displayed a sequence of movement

similar to that of healthy individuals. However, the group differences in onsets and latencies did not translate into modifications of the amplitudes of movement, neither in extension nor in flexion. Indeed, both groups displayed the same pattern in terms of amplitude of the head and the trunk angular displacement.

In terms of COM displacements, the kinematic pattern previously described was reflected by the forward displacement of abdomen, lower thorax, upper thorax and head COMs, with their onsets delayed relative to the platform onset. This delay is due to the first extension phase during which all segments lagged relatively to the pelvis. The onsets of COM displacement did not reveal any difference between the whiplash and the healthy groups. In both groups, the results indicated a caudo-cranial sequence of movement, from the abdomen to the head. This kinematic pattern is reminiscent of the ankle strategy observed in standing subjects submitted to support surface translations. That strategy is characterized by the whole body rotating around the ankle joint, with a consequence of bringing back the body's COM over the base of support (Horak and Nashner 1986; Nashner 1976; Nashner 1977). This caudo-cranial pattern has also been compared to that of the body acting as an inverted pendulum, since all segments above the pivot point move in phase, in the same direction (Keshner 2003; Winter 1995). The same observation has been reported by Forssberg and Hirschfeld (1994) for sitting subjects submitted to forward and backward translations, where in sitting, the hip joint plays the part of the pivot point around which the trunk is rotating in the sagittal plane.

After their delayed onset, the segments caught up to the platform and even overshot the platform final position. This is due to the flexion phase, which was shown to bring the head and the trunk to overshoot their initial position. Moreover, results indicate that whiplash individuals took the same time to reach peak COM position in comparison with healthy controls and in both groups, all segments reached their peak COM position at the same time. In contrast, the analysis of the amplitude of the forward displacement of the COM segments showed differences between the healthy and whiplash groups. In healthy individuals, there was no difference in the amplitude of COM displacement between the individual trunk segments, with only the head COM presenting a greater displacement

compared to lower thorax and abdomen COMs. However, whiplash individuals displayed a greater displacement of their upper thorax COM in comparison with the abdomen COM, and the head COM displacement was greater than upper thorax, lower thorax and abdomen COM displacements. These findings show that in individuals with WAD, the flexion of the trunk is more evenly distributed along the spine, with greater range of motion at the superior segments of the spine relative to the more inferior ones. This suggests that the behavior of the trunk in WAD individuals resembles less that of the inverted pendulum, with more evenly distributed movement along the trunk segments and reduced reliance on the trunk moving around the main pivot point (hips). Finally, the data shows that all segments finished their motion by moving backwards, resulting in the subjects almost reaching their initial position, with the trunk aligned with the pelvis. The segmental COM final position relative to the platform final position did not reveal differences between the two groups. In both groups, the final position of all segmental COMs was in front relative to the final position of the platform, with the head staying farther ahead than the other segments.

Effects of WAD on kinematic characteristics

We hypothesized that following anterior translations, individuals with WAD would present some changes in their kinematic patterns in comparison with healthy controls. Several characteristics of head and trunk kinematics confirmed this hypothesis.
Early after the onset of the platform displacement, at the beginning of the postural response, it appeared that individuals with WAD stabilized their head in line with the trunk, a pattern not present in the healthy group. This could be interpreted as a protective strategy adopted to avoid a too large extension at the neck level. This hypothesis was also based on the frequent reports that individuals with WAD generally display reduced cervical motion amplitude (Antonaci et al. 2002; Dall'Alba et al. 2001; Madeleine et al. 2004; Sterling et al. 2003, Sterling et al. 2004) and on recent work using the craniocervical flexion test (CCFT) which showed that persons with neck pain displayed smaller cervical motion amplitude at all levels of this clinical procedure (Falla et al. 2004b; Sterling et al. 2003). However, this strategy adopted by our whiplash subjects did

not result in smaller head extension in comparison with healthy controls, as was expected. This absence of group difference could be due to the fact that there may exist a comparatively large variability in head range of motion in persons subjected to mechanical perturbations, even within a healthy population. Indeed, in two recent studies, it was observed that whereas several subjects displayed relatively large head motion amplitude following perturbations (floppy subjects), certain healthy subjects adopted a stiffening strategy (stiff subjects) that resulted in smaller head motion relative to the trunk (Blouin et al. 2003b; Vibert et al. 2001). Moreover, in these studies, the peak translation accelerations were 6.37 m/s^2 (Vibert et al. 2001) and 10.8 m/s^2 (Blouin et al. 2003b), which is much greater than in the present study (1.5 m/s^2). Since slower perturbations have been shown to result in smaller head displacements and accelerations (Kumar et al. 2000; Kumar et al. 2002; Siegmund et al. 2002), it is possible that the stimulus used in our study was too small to allow us to observe a group difference in terms of head motion amplitudes. Therefore, the potentially large between-subject variability in head motion amplitude following mechanical perturbations, combined with our use of comparatively small perturbations, could explain why we did not observe a group difference in head motion amplitude following forward perturbations.

At the trunk level, our data indicates that individuals with WAD present changes in the distribution of the movement along the spine, illustrated by a greater spatial dissociation between the various segments of the spine. In both groups, all segmental COMs reached their maximal displacement at the same time. However, the amplitude of the COM displacements and the overshoot relative to their final position show that whiplash individuals display more flexion at the upper levels of the spine, compared to their lower levels. This is reflected in the final head COM position, which is more in front of the upper thorax COM, and the upper thorax COM which is more in front of the abdomen COM in WAD. This reorganization of the trunk movement suggests a compensation for the injury sustained at the neck level. Indeed, the mobility of the trunk and the neck is insured by the many joints that compose the vertebral column. Since the vertebral column has a redundant number of degrees of freedom (Bernstein 1967), several combinations of joint excursions can effectively produce flexion and extension of the trunk and the neck.

As such, our results suggest that individuals with WAD compensate their reduced cervical functional ability by increasing the flexion at the upper thorax level. Similar changes in mobility along the spine have been reported in subjects with low back pain (Larivière et al. 2000). In the execution of trunk flexion-extension tasks from an upright position, they have demonstrated that patients with low back pain displayed an increase in thoracic flexion to compensate for the decrease in lumbar flexion. Therefore, we suggest that similar compensation mechanisms across the spine are employed in individuals faced with WAD.

Muscle behavior

The forward perturbations also triggered the activation of trunk and neck muscles. At the level of the trunk, the muscles investigated were consistently activated in almost all subjects and trials. The comparison of muscle onsets revealed that whiplash individuals recruited their trunk muscles with similar latencies in comparison with healthy controls. In both groups, the trunk flexors (RA and EO) were activated before the trunk extensors (TES and LES). This muscle pattern fits well with our findings of trunk kinematics. Taken together, data from these two techniques indicate that the ventral muscles (flexors) were activated first, during the trunk extension, and the dorsal muscles (extensors) later. This pattern of muscle recruitment has been previously observed in subjects submitted to forward translations and legs-up rotations (Forssberg and Hirschfeld 1994; Zedka et al. 1998). Moreover, these findings are consistent with the general finding that following sudden perturbations, muscles that are stretched respond first, followed by their antagonists in order to minimize an overshoot and pursue a new stable configuration (Forssberg and Hischfeld 1994; Horak and Nashner 1986; Keshner et al. 1988).

The neck muscles were activated only in a portion of the participants of each group and the UT was activated in very few subjects. From these muscles, we could not observe consistent recruitment patterns across subjects, with some subjects recruiting the SCA, the SCM and the CP and other subjects only some of these muscles. For this reason, it was not possible to statistically compare the onsets of the neck muscles between the

whiplash and healthy groups, or to compare neck and trunk muscles to each other. However, mean values of muscle onsets were calculated with the data available and the results showed interesting tendencies. The first tendency suggests that whiplash individuals recruit their neck flexors (SCA and SCM) later in comparison with healthy controls. This result seems in contradiction with the kinematic pattern previously described of head and trunk moving *en block* at the onset of movement. Indeed, such a protective strategy has been postulated to be attributed to stiffening reactions in the neck muscles (Vibert et al. 2001). However, other studies have suggested that individuals with chronic WAD develop an altered motor strategy aimed at minimizing the activation of painful muscles, resulting in a reorganization of muscle recruitment in the neck (Falla et al. 2004a; Falla et al. 2004b; Nederhand et al. 2003; Sterling et al. 2003), in accordance with the pain adaptation model (Lund et al. 1991). Therefore, the tendency of whiplash individuals for a late recruitment of their neck flexors, in addition to their kinematic pattern of head and trunk moving *en block*, support the hypothesis of such a protective strategy. The second tendency observed indicates that neck flexors (SCA and SCM) are recruited before the neck extensor (CP), in both groups. Thus, similarly to what was found at the trunk level, our results suggest that neck ventral muscles would be recruited before the neck dorsal muscles following forward perturbations.

The lack of consistency in neck muscle recruitment patterns observed in the present study might be due to the low intensity of the perturbation. However, Vibert et al. (2001) also showed a lack of consistency in neck muscle activation patterns, in spite of using much higher intensity perturbations than the one we used in our study. Rather, we agree with the suggestion of Vibert et al. (2001) and of Jull (2000) that the recruitment of superficial neck muscles might not be the most favored mechanism of head stabilization compared to one involving the deep axial neck muscles. Therefore, it seems that in healthy and whiplash individuals, the inter-subject variability of superficial cervical muscle activation patterns could be due to the fact that because the deep axial neck muscles are in a better anatomical position to stabilize the cervical spine segments, individuals rather prioritize to consistently recruit this group of muscles rather than the superficial cervical muscles.

In turn, the choice to recruit superficial muscles to stabilize the head may be less consistent across subjects and across trials.

From our results, we also observed that some subjects presented an interesting pattern of muscle inhibition of their extensors. When a burst of activity was also present in the same muscle, this period of muscle inhibition preceded the muscle burst and presented some overlap with the burst of the antagonistic flexor muscle. This pattern is consistent with one version of the stretch reflex which provokes the contraction of the stretched muscle and its synergists concomitantly with the inhibition of the antagonist muscle (Gordon and Ghez 1991). This pattern of muscle inhibition was present in both groups of subjects. However, in neck extensors, the results indicate a tendency for a pattern more frequent in the whiplash group in comparison with the healthy group and in trunk extensors, a tendency for a pattern less frequent in the whiplash group in comparison with the healthy group (see Table 9). In neck extensors, the greater occurrence of the inhibition pattern in whiplash individuals might indicate a preference of using this strategy to compensate for the delayed onset in neck flexors. Indeed, the inhibition of neck extensors would counteract the fact that the contraction of neck flexors occurs later in WAD. We presume that this pattern would be a part of the altered motor strategy developed to protect the injured muscles. The less frequent use of muscle inhibition in trunk extensors of WAD individuals might reflect that the muscle reorganization also affects their trunk muscles in addition to their neck muscles.

In summary, our analysis of muscle behavior did not reveal significant differences in neck and trunk muscle activation patterns between whiplash and healthy individuals. However, our data suggests a tendency for a delayed recruitment of neck flexors in individuals with WAD, in addition with a greater tendency for a pattern of neck extensor muscle inhibition preceding the neck flexor burst. This suggests a possible reorganization of neck muscle recruitment strategies to minimize the use of painful muscles. In both groups, the trunk flexors were recruited before the trunk extensors. A similar tendency was observed in neck muscles. These results are consistent with the general accepted

finding that following a sudden perturbation, the muscles that are elongated are recruited first.

It should be noted that our data should be interpreted in light of some limitations associated with our experimental approach. This study was conducted with a relatively small number of participants in both groups, with participants presenting a large variability in their symptoms in the case of the whiplash group. For instance, the time between the whiplash trauma and their participation to this protocol varied from three and a half to ten months. In addition, even if all these participants had received a diagnosis of cervical sprain, some of them were also diagnosed with thoracic sprain, lumbar sprain or both, which is consistent with the definition of WAD. The results presented in the present study should therefore be interpreted within the limits of our sample of individuals with WAD. Also, our results should be interpreted while keeping in mind that we used a perturbation clearly weaker than those in the literature on healthy sitting subjects. Indeed, we were assessing the postural stabilization strategies of a pathological population that was somewhat vulnerable to further aggravation of their injury. Thus, the choice of our perturbation had to be made with this in mind. In light of our communications with professionals of the clinical team, we chose a perturbation with the highest amplitude allowed by our system (15 cm) so as to avoid high-jerk perturbations and the choice of the duration of the perturbation is further explained somewhere else (St-Onge et al. submitted). Thus, our results can only be interpreted within the context of the chosen perturbation. It should also be mentioned that we assumed that the perturbation used in the present study was a threshold perturbation for our healthy subjects (St-Onge et al., submitted). However, we should point out that this perturbation is not necessarily a threshold perturbation for all whiplash patients of our study, and that we may have found different results had we been able to measure the effects of patient-specific threshold perturbations on postural stabilization strategies. Nevertheless, we showed that our perturbation was sufficient to reveal specific characteristics in the kinematic patterns of whiplash individuals as well as to suggest some interesting tendencies in their muscle activation patterns.

In conclusion, we have shown that in response to sudden forward displacements of the support surface, WAD individuals display an earlier onset of their head displacement and a pattern of trunk displacement characterized by greater flexion at the upper levels of the spine, compared to the lower levels. In addition, WAD individuals present a tendency for a late recruitment of their neck flexors and for a greater use of a pattern of neck extensor muscle inhibition. These results suggest that individuals with WAD may compensate their altered neck functional ability by modifying their relative movements along the spine and by adopting altered motor strategies to compensate for their painful muscles. The results of our study highlight the importance of considering WAD as a pathology that involves all segments of the trunk and not only the cervical region, with implications in rehabilitation and return to work strategies.

Acknowledgments

We thank Richard Preuss for sharing scripts for data analysis. We also thank Nicoleta Bugnariu, Adriana Venturini, Elizabeth Dannenbaum, Michael Trivino and Petko Baltov for their presence during the data collection sessions. I.P. is supported by a MENTOR and an NSERC M.Sc. scholarship and N.S. is supported by a MENTOR post-doctoral scholarship. Equipment has been purchased with the help of CFI and JRH foundation.

CONCLUSION

Several general conclusions can be drawn from this global thesis effort. In the first manuscript, we identified a low-intensity threshold translational perturbation that provoked postural reactions in healthy seated individuals. Using such a perturbation, we observed stereotypical kinematic and electromyographic responses in our healthy subjects. It was thought that the observed movement patterns and muscle actions were generated to restore the projection of the body's center of mass (COM) within the base of support following surface translations. In turn, we believed that this threshold perturbation, the weakest one tested that provoked clear postural stabilization patterns, could be safely used to assess the postural stabilization mechanisms in a population of chronic whiplash-injured patients.

In the second step of this research, the protocol developed with healthy individuals was applied to a group of patients with whiplash-associated disorders (WAD) and their postural reactions were compared with those of healthy controls. We found that in response to sudden forward displacements of the support surface, WAD individuals displayed an earlier onset of their head displacement and a pattern of trunk displacement characterized by greater flexion at the upper levels of the spine, compared to the lower levels. In addition, WAD individuals present a tendency for a late recruitment of their neck flexors and for a greater use of a pattern of neck extensor muscle inhibition. These results suggest that individuals with WAD may compensate their altered neck functional ability by modifying their relative movements along the spine and by adopting altered motor strategies to compensate for their painful muscles. The results of our study highlighted the importance of considering WAD as a pathology that involves all segments of the trunk and not only the cervical region, with implications in rehabilitation and return to work strategies.

Our work opens the way to several possible research directions. For one, in conjunction with this project, we also acquired biomechanical data of persons (healthy, WAD) submitted to backward support surface translations. The analysis of the postural reactions

observed during the backward perturbations will be helpful in completing the picture of the postural stabilization mechanisms adopted by WAD individuals following antero-posterior translations. Also, in this study, our group of patients was evaluated at the moment they entered an intensive return to work rehabilitation program, and we are currently in the process of repeating our experimental protocol at their discharge from the program. Therefore, it will be interesting to test their condition after they complete this program to determine if this intensive multidisciplinary rehabilitation had a significant effect on their postural reactions. The global results following this second evaluation could have impacts at different levels. They could be used as an objective basis to decide about the management of other WAD individuals in rehabilitation approaches. The results could also be used to make recommendations about clinical strategies. They could also indicate the improvement of the functional status of individuals at critical stages of their rehabilitation.

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APPENDIX A

Ethics certificate

Comité d'éthique de la recherche
des établissements du CRIR

Certificat d'éthique

Par la présente, le comité d'éthique de la recherche des établissements du CRIR (CER) atteste qu'il a évalué le projet de recherche (CRIR-37-0404) intitulé:

« Évaluation de la validité de l'outil de mesure clinique d'amplitude du cou (CROM) comparativement à deux outils d'analyse cinématique ».

Présenté par: **Julie Côté, Ph.D.**

Le comité d'éthique de la recherche composé de :

NOM	**POSTE**
Mme Isabelle Bilodeau	Une personne possédant une vaste connaissance du domaine psychosocial en réadaptation
Mme Nicol-Korner-Bitensky	Une personne possédant une vaste connaissance du domaine biomédical en en réadaptation
Mme Julie-Anne Couturier	Clinicienne détenant une vaste connaissance des déficits sensoriel visuels ou auditifs
Mme Marie-Josée Drolet	Clinicienne détenant une vaste connaissance des déficits moteurs ou neurologiques
Mme Marie-Ève Bouthillier	Une personne spécialisée en éthique
Me Michel Giroux	Une personne spécialisée en droit
M. André Vincent	Une personne non affiliée à l'établissement et provenant de la clientèle des personnes adultes et aptes

APPENDIX B

Consent forms

Consent form
(WAD - biomechanical)

1 - Title of project

Biomechanical and psychosocial characterization of individuals suffering from whiplash-associated disorders and objectification of their chances of permanently returning to work after a personalized intensive rehabilitation protocole.

2 - Researchers in charge of project

Julie Côté, Ph.D. Assistant professor, Department of Kinesiology and Physical Education, McGill University, (450) 688-9550, ext. 4813
Debbie Feldman, Ph.D., Assistant professor, School of Rehabilitation/Department of Social and Preventive Medicine, University of Montreal, (514) 343-6111 ext. 1252
Gaétan Filion, M.D., Physiatrist, Medical director, Pediatric programme, Jewish Rehabilitation Hospital, (450) 688-9550
Joyce Fung, PT, Ph.D., Associate professor, School of Physical and Ergotherapy, McGill University, (450) 688-9550 ext. 529
Nancy St-Onge, Ph.D., Postdoctoral fellow, School of Physical and Ergotherapy, McGill University, (450) 688-9550 ext. 623

3 - Project description and objectives

The objective of this project is to better understand psychosocial and biomechanical characteristics of individuals suffering from whiplash-associated disorders. Our goal is to develop and validate quantitative approaches that will allow a better evaluation of the severity of the pathology in this population.

Twenty-five subjects suffering from a whiplash injury will be recruited from the patients taking part in the *Programme d'évaluation, de développement et d'intégration professionnelle* (PÉDIP). A group consisting of twenty-five healthy subjects will also be recruited. We want to compare postural characteristics between healthy subjects and those suffering from a whiplash injury. Whiplash individuals will be evaluated before and after the PÉDIP rehabilitation protocole. Using this information, we wish to define the pathological condition associated with a whiplash injury and therefore better evaluate the chances of safely returning to the workplace of individuals suffering from a whiplash injury.

4 - Nature and duration of participation

The research project to which I am invited to participate aims at understanding postural reactions in individuals suffering from a whiplash injury. The tests will be performed at the Research Center of the Jewish

Rehabilitation Hospital. I will have to come twice to the center for a period of two hours each time. The first session will take place at the beginning of the PÉDIP treatments and the second one when the treatments will be over. Each of the sessions will be organized into two parts: preparation and perturbations.

Preparation will last approximately one hour. Surface electrodes will be positioned on the skin overlying neck and trunk muscles. Reflecting markers will also be placed on my head, trunk, pelvis, arms and legs in order to record their positions. None of those procedures is invasive.

Perturbations will last approximately 20 minutes. I will sit on a stool fixed to a perturbation platform and will be attached using a belt. I will first have to stay immobile during a few seconds for three trials. Forward and backward translational perturbations will then be applied to the platform. I will then have to stay immobile during a few seconds for three trials. During all of the trials I will have to try to maintain my position as stable as possible.

5 - Advantages associated with my participation

I will not personally benefit from advantages by participating in this study. However, I will contribute to science.

6 - Risks associated with my participation

My participation in this project does not put me at any medical risk. Moreover, my participation will not affect care and services I receive at the Jewish Rehabilitation Hospital.

7 - Personal inconvenients

The duration of the testing (approximately two hours) might be an inconvenient for some individuals. The possibility that we have to shave the subject before positioning the electrodes on the skin might also be an inconvenient. Electrodes might cause skin irritation. Also, some people might experience fatigue, discomfort or vertigo during and after the perturbations. I have the right to withdraw from the study at any moment if I wish to. Note that a clinician will be present at all time should complications occur. It is also possible that some subjects feel pain following the participation to the project.

8 - Access to my medical file

I authorize access to my medical file to the persons responsible for this project. I understand that only the information concerning my whiplash-associated disorders and the evolution of the symptoms will be used. I also authorize the persons responsible for this project to give access to that information to other members of the research team.

9 - Confidentiality

All the personal information collected for this study will be codified to insure confidentiality. Information will be kept under locking key at the research center of the Jewish Rehabilitation Hospital by one of the persons responsible for the study for a period of five years. Only the people involved in the project will have access to this information. If the results of this research project are presented or published, nothing will allow my identification.

The results obtained will be used to set up a database. The information being codified, nothing will allow my identification and it will not be possible to associate me with the results.

10 - Questions concerning the study

The researchers present during the testing should answer my questions concerning the project satisfactorily.

11 - Withdrawal of subject from study

My participation in the research project described above is completely voluntary. I have the right to withdraw from the study at any moment without

affecting health cares and services I receive from the Jewish rehabilitation hospital.

Should I withdraw form the study, all audiovisual and written document concerning myself will be destroyed.

12 - Responsibility

By accepting to enter this study, I do not surrender to my rights and do not free the researchers, sponsor or the institutions involved from their legal and professional obligations.

13 - Monetary compensation

I will not receive a monetary compensation for participating to this study.

14 - Contact persons

If I need to ask questions about the project, signal an adverse effect and/or an incident, I can contact at any time Dr. Julie Côté, Assistant professor in the Department of Kinesiology and Physical Education, McGill University at (450) 688-9550, ext. 4813.

Also, if I have questions concerning my rights and remedy or my participation to this research project, I can contact Me Anik Nolet, Research ethics co-ordinator of CRIR at (514) 527-4527 ext. 2643 or by email at anolet.crir@ssss.gouv.qc.ca

CONSENT

I declare to have read and understood the project, the nature and the extent of the project, as well as the risks I am exposed to as describe in the present document. I had the opportunity to ask all my questions concerning the different aspects of the study and to receive explanations to my satisfaction.

I, undersigned, voluntarily accept to participate in this study. I can withdraw at any time without any prejudice. I certify that I have received enough time to take my decision and I know that a copy of this consent form will be added to my medical file.

A signed copy of this information and consent form should be given to me.

NAME OF SUBJECT **SIGNATURE**

_____ _____

Done in _____, _____, 20_____.

COMMITMENT OF RESEARCHER

I, undersigned, _____ , certify
(a) having explained to the signatory the terms of the present form ;
(b) having answered all questions he/she asked concerning the study ;
(c) having clearly told him/her that he/she is at any moment free to withdraw from the research project described above ;
and (d) that I will give him/her a signed and dated copy of the present document.

_____
Signature of person in charge of the project
or representative

Done in _____, _____ 20__.

Consent form
(Control)

1 - Title of project

Biomechanical and psychosocial characterization of individuals suffering from whiplash-associated disorders and objectification of their chances of permanently returning to work after a personalized intensive rehabilitation protocole.

2 - Researchers in charge of project

Julie Côté, Ph.D. Assistant professor, Department of Kinesiology and Physical Education, McGill University, (450) 688-9550, ext. 4813
Debbie Feldman, Ph.D., Assistant professor, School of Rehabilitation/Department of Social and Preventive Medicine, University of Montreal, (514) 343-6111 ext. 1252
Gaétan Filion, M.D., Physiatrist, Medical director, Pediatric programme, Jewish Rehabilitation Hospital, (450) 688-9550
Joyce Fung, PT, Ph.D., Associate professor, School of Physical and Occupational Therapy, McGill University, (450) 688-9550 ext. 529
Nancy St-Onge, Ph.D., Postdoctoral fellow, School of Physical and Occupational Therapy, McGill University, (450) 688-9550 ext. 623

3 - Project description and objectives

The objective of this project is to better understand psychosocial and biomechanical characteristics of individuals suffering from whiplash-associated disorders. Our goal is to develop and validate quantitative approaches that will allow a better evaluation of the severity of the pathology in this population.

Twenty-five subjects suffering from a whiplash injury will be recruited from the patients taking part in the *Programme d'évaluation, de développement et d'intégration professionnelle* (PÉDIP). A group consisting of twenty-five healthy subjects will also be recruited. We want to compare postural characteristics between healthy subjects and those suffering from a whiplash injury. Whiplash individuals will be evaluated before and after the PÉDIP rehabilitation protocole. Using this information, we wish to define the pathological condition associated with a whiplash injury and therefore better evaluate the chances of safely returning to the workplace of individuals suffering from a whiplash injury.

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The research project to which I am invited to participate aims at understanding postural reactions in individuals suffering from a whiplash injury. The tests will be performed at the Research Center of the Jewish

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5 - Advantages associated with my participation

I will not personally benefit from advantages by participating in this study. However, I will contribute to science.

6 - Risks associated with my participation

My participation in this project does not put me at any medical risk.

7 - Personal inconvenients

The duration of the testing (approximately two hours) might be an inconvenient for some individuals. The possibility that we have to shave the subject before positioning the electrodes on the skin might also be an inconvenient. Electrodes might cause skin irritation. Also, some people might experience fatigue, discomfort or vertigo during and after the perturbations. I have the right to withdraw from the study at any moment if I wish to.

8 - Access to my medical file

Access to my medical file is not required for this study.

9 - Confidentiality

All the personal information collected for this study will be codified to insure confidentiality. Information will be kept under locking key at the research center of the Jewish Rehabilitation Hospital by one of the persons responsible for the study for a period of five years. Only the people involved in the project will have access to this information. If the results of this research project are presented or published, nothing will allow my identification.

10 - Questions concerning the study

The researchers present during the testing should answer my questions concerning the project satisfactorily.

11 - Withdrawal of subject from study

My participation in the research project described above is completely voluntary. I have the right to withdraw from the study at any moment.

Should I withdraw form the study, all audiovisual and written document concerning myself will be destroyed.

12 - Responsibility

By accepting to enter this study, I do not surrender to my rights and do not free the researchers, sponsor or the institutions involved from their legal and professional obligations.

13 - Monetary compensation

A monetary compensation will be given to me at the end of the experiment to reimburse the travel expenses to participate in this study.

14 - Contact persons

If I need to ask questions about the project, signal an adverse effect and/or an incident, I can contact at any time Dr. Julie Côté, Assistant professor in the Department of Kinesiology and Physical Education, McGill University at (450) 688-9550, ext. 4813.

Also, if I have questions concerning my rights and remedy or my participation to this research project, I can contact Me Anik Nolet, Research ethics co-ordinator of CRIR at (514) 527-4527 ext. 2643 or by email at anolet.crir@ssss.gouv.qc.ca

CONSENT

I declare to have read and understood the project, the nature and the extent of the project, as well as the risks and inconveniences I am exposed to as described in the present document. I had the opportunity to ask all my questions concerning the different aspects of the study and to receive explanations to my satisfaction.

I, undersigned, voluntarily accept to participate in this study. I can withdraw at any time without any prejudice. I certify that I have received enough time to take my decision and I know that a copy of this consent form will be added to my medical file.

A signed copy of this information and consent form should be given to me.

NAME OF SUBJECT **SIGNATURE**

_____ _____

Done in _____, _____, 20_____.

COMMITMENT OF RESEARCHER

I, undersigned, _____ , certify
(a) having explained to the signatory the terms of the present form ;
(b) having answered all questions he/she asked concerning the study ;
(c) having clearly told him/her that he/she is at any moment free to withdraw from the research project described above ;
and (d) that I will give him/her a signed and dated copy of the present document.

_____
Signature of person in charge of the project
or representative

Done in _____, _____ 20__.

Formulaire de consentement (DACFC – volet biomécanique)

1 - Titre du projet

Caractérisation biomécanique et psychosociale des individus atteints de désordres associés au coup de fouet cervical et objectivation de leurs chances de retour permanent au travail suite à un protocole de réadaptation intensif personnalisé.

2 - Responsable(s) du projet

Julie Côté, Ph.D. professeure adjointe, Département de kinésiologie et d'éducation physique
Université McGill, (450) 688-9550, poste 4813
Debbie Feldman, Ph.D., professeure adjointe, École de réadaptation / Dép. de médecine sociale et préventive, Université de Montréal, (514) 343-6111 poste 1252
Gaétan Filion, M.D., physiatre, directeur médical, programme pédiatrique, Hôpital juif de réadaptation, (450) 688-9550
Joyce Fung, PT, Ph.D., professeure agrégée, École de physiothérapie et d'ergothérapie, Université McGill, (450) 688-9550 poste 529
Nancy St-Onge, Ph.D., chercheure post-doctorale, École de physiothérapie et d'ergothérapie, Université McGill, (450) 688-9550 poste 623

3 - Description du projet et de ses objectifs

Le but de ce projet est de mieux comprendre les caractéristiques psychosociales et biomécaniques d'individus ayant subi un coup de fouet cervical. Nous visons à développer et valider des approches quantitatives permettant de mieux évaluer l'atteinte pathologique de ces individus.

Vingt-cinq sujets ayant subi un coup de fouet cervical seront recrutés parmi la clientèle du Programme d'évaluation, de développement et d'intégration professionnelle (PÉDIP) de l'hôpital juif de réadaptation. Un groupe de vingt-cinq sujets sains sera également recruté. Nous désirons comparer les caractéristiques de la posture entre les sujets sains et les sujets ayant subi un coup de fouet cervical. Les individus souffrant d'un coup de fouet cervical seront évalués avant ainsi qu'après le protocole de réadaptation PÉDIP. Nous voulons ainsi définir la condition pathologique reliée au coup de fouet cervical et ainsi pouvoir mieux évaluer les chances de retour sécuritaire au travail des individus ayant subi un coup de fouet cervical.

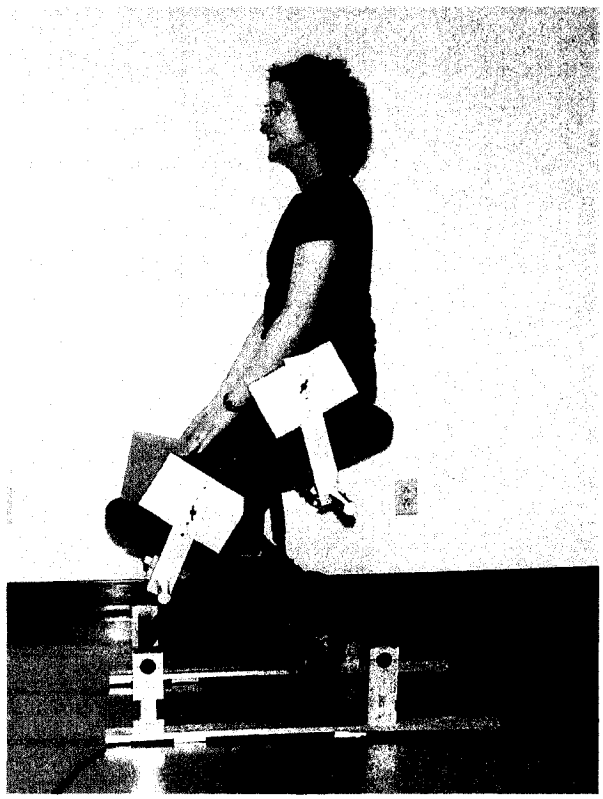
4 - Nature et durée de la participation

Le projet de recherche auquel je suis invité à participer vise à comprendre le contrôle de la posture chez les individus ayant subi un coup de

fouet cervical. Les tests se déroulent au centre de recherche de l'Hôpital juif de réadaptation. La participation qui m'est demandée comporte deux séances de deux heures. La première séance aura lieu au début des traitements PÉDIP et la deuxième lorsque les traitements seront terminés. Chaque séance sera séparée en deux parties : la préparation et les perturbations.

La préparation durera environ une heure. Des électrodes de surface seront fixées sur la peau recouvrant les muscles du cou et du tronc. Des marqueurs réfléchissant seront aussi collés sur ma tête, mon tronc, mon bassin, mes bras et mes jambes afin d'enregistrer leurs positions. Aucune de ces procédures n'est invasive.

Les perturbations dureront environ 20 minutes. Je serai assis sur un banc fixé à une plateforme de perturbations et serai attaché à l'aide d'une ceinture. Je devrai d'abord rester immobile pendant trois essais de quelques secondes. Ensuite, des perturbations seront appliquées en déplaçant la plateforme en translation vers l'avant et vers l'arrière. Je devrai ensuite rester immobile pendant trois essais de quelques secondes. Pendant tous les essais, je devrai essayer de rester le plus stable possible dans la position.



5 - Avantages pouvant découler de ma participation

Je ne retirerai personnellement pas d'avantages à participer à cette étude. Toutefois, j'aurai contribué à l'avancement de la science.

6 - Risques pouvant découler de ma participation

Ma participation à ce projet de recherche ne me fait courir, sur le plan médical, aucun risque que ce soit. Il est aussi entendu que ma participation au projet n'affectera pas les soins et les services que je reçois ou recevrai de l'Hôpital juif de réadaptation.

7 - Inconvénients personnels

La durée de la session d'environ deux heures peut représenter pour certaines personnes un inconvénient. La possibilité que l'on doive raser le participant pour poser les électrodes peut aussi représenter un inconvénient. Il arrive parfois que les électrodes occasionnent de l'irritation cutanée. De plus, certaines personnes pourraient ressentir de la fatigue, de l'inconfort ou une sensation de vertige pendant ou suite aux perturbations. Il est entendu que je pourrai à tout moment interrompre la session si je le désire. De plus, un clinicien sera présent à tout moment en cas de complications. Il est aussi possible que certains participants ressentent une légère douleur suite à leur participation au projet.

8 - Accès à mon dossier médical

J'autorise les responsables du projet à obtenir accès à mon dossier médical. Je comprends que seuls les renseignements relatifs à mes désordres associés au coup de fouet cervical et à l'évolution des symptômes seront consultés. J'autorise aussi les responsables du projet à permettre l'accès à ces renseignements à d'autres membres de l'équipe de recherche.

9 - Confidentialité

Tous les renseignements personnels recueillis à mon sujet au cours de l'étude seront codifiés afin d'assurer ma confidentialité. Ces données seront conservées sous clé au centre de recherche de l'Hôpital juif de réadaptation par un responsable de l'étude pour une période de cinq ans. Seuls les membres de l'équipe de recherche y auront accès. En cas de présentation de résultats de cette recherche ou de publication, rien ne pourra permettre de m'identifier.

10 - Questions concernant cette étude

Le(s) chercheur(s) présent(s) lors de la collecte des données s'engage(nt) à répondre de façon satisfaisante à toutes mes questions concernant le projet de recherche.

11 - Retrait de la participation du sujet

Ma participation au projet de recherche décrit ci-dessus est tout à fait libre et volontaire. Il est entendu que je pourrai, à tout moment, mettre un terme à ma participation sans que cela n'affecte les soins et les services de santé que je reçois ou recevrai de l'Hôpital juif de réadaptation.

En cas de retrait de ma part, les documents audiovisuels et écrits me concernant seront détruits.

12 - Clause de responsabilité

En acceptant de participer à cette étude, je ne renonce à aucun de mes droits ni ne libère les chercheurs, le commanditaire ou les institutions impliquées de leurs obligations légales et professionnelles.

13 - Indemnité compensatoire

Je ne recevrai pas de compensation financière pour ma participation à cette étude.

14 - Personnes-ressources

Si je désire poser des questions sur le projet, signaler un effet adverse et/ou un incident défavorable, je peux rejoindre en tout temps Dre Julie Côté, professeure adjointe au Département de kinésiologie et d'éducation physique de l'université McGill au (450) 688-9550, poste 4813.

De plus, si j'ai des questions sur mes droits et recours ou sur ma participation à ce projet de recherche, je peux communiquer avec Me Anik Nolet, coordonnatrice à l'éthique de la recherche des établissements du CRIR au (514) 527-4527 poste 2643 ou par courriel à l'adresse suivante: anolet.crir@ssss.gouv.qc.ca

CONSENTEMENT

Je déclare avoir lu et compris le présent projet, la nature et l'ampleur de ma participation, ainsi que les risques auxquels je m'expose tels que présentés dans le présent formulaire. J'ai eu l'occasion de poser toutes les questions concernant les différents aspects de l'étude et de recevoir des réponses à ma satisfaction.

Je, soussigné(e), accepte volontairement de participer à cette étude. Je peux me retirer en tout temps sans préjudice d'aucune sorte. Je certifie qu'on m'a laissé le temps voulu pour prendre ma décision et je sais qu'une copie de ce formulaire figurera dans mon dossier médical.

Une copie signée de ce formulaire d'information et de consentement doit m'être remise.

NOM DU SUJET **SIGNATURE**

_____ _____

Fait à _____, **le** _____, **20**_____.

ENGAGEMENT DU CHERCHEUR

Je, soussigné (e), _____ , certifie
(a) avoir expliqué au signataire les termes du présent formulaire;
(b) avoir répondu aux questions qu'il m'a posées à cet égard;
(c) lui avoir clairement indiqué qu'il reste, à tout moment, libre de mettre un terme à sa participation au projet de recherche décrit ci-dessus;
et (d) que je lui remettrai une copie signée et datée du présent formulaire.

_____
Signature du responsable du projet
ou de son représentant

Fait à _____, le _____ 20__.

Formulaire de consentement (témoin)

1 - Titre du projet

Caractérisation biomécanique et psychosociale des individus atteints de désordres associés au coup de fouet cervical et objectivation de leurs chances de retour permanent au travail suite à un protocole de réadaptation intensif personnalisé.

2 - Responsable(s) du projet

Julie Côté, Ph.D. professeure adjointe, Département de kinésiologie et d'éducation physique
Université McGill, (450) 688-9550, poste 4813
Debbie Feldman, Ph.D., professeure adjointe, École de réadaptation / Dép. de médecine sociale et préventive, Université de Montréal, (514) 343-6111 poste 1252
Gaétan Filion, M.D., physiatre, directeur médical, programme pédiatrique, Hôpital juif de réadaptation, (450) 688-9550
Joyce Fung, PT, Ph.D., professeure agrégée, École de physiothérapie et d'ergothérapie, Université McGill, (450) 688-9550 poste 529
Nancy St-Onge, Ph.D., chercheure post-doctorale, École de physiothérapie et d'ergothérapie, Université McGill, (450) 688-9550 poste 623

3 - Description du projet et de ses objectifs

Le but de ce projet est de mieux comprendre les caractéristiques psychosociales et biomécaniques d'individus ayant subi un coup de fouet cervical. Nous visons à développer et valider des approches quantitatives permettant de mieux évaluer l'atteinte pathologique de ces individus.

Vingt-cinq sujets ayant subi un coup de fouet cervical seront recrutés parmi la clientèle du Programme d'évaluation, de développement et d'intégration professionnelle (PÉDIP) de l'hôpital juif de réadaptation. Un groupe de vingt-cinq sujets sains sera également recruté. Nous désirons comparer les caractéristiques de la posture entre les sujets sains et les sujets ayant subi un coup de fouet cervical. Les individus souffrant d'un coup de fouet cervical seront évalués avant ainsi qu'après le protocole de réadaptation PÉDIP. Nous voulons ainsi définir la condition pathologique reliée au coup de fouet cervical et ainsi pouvoir mieux évaluer les chances de retour sécuritaire au travail des individus ayant subi un coup de fouet cervical.

4 - Nature et durée de la participation

Le projet de recherche auquel je suis invité à participer vise à comprendre le contrôle de la posture chez les individus ayant subi un coup de

fouet cervical. Les tests se déroulent au centre de recherche de l'Hôpital juif de réadaptation. La participation qui m'est demandée comporte deux séances de deux heures. Chaque séance sera séparée en deux parties : la préparation et les perturbations.

La préparation durera environ une heure. Des électrodes de surface seront fixées sur la peau recouvrant les muscles du cou et du tronc. Des marqueurs réfléchissant seront aussi collés sur ma tête, mon tronc, mon bassin, mes bras et mes jambes afin d'enregistrer leurs positions. Aucune de ces procédures n'est invasive.

Les perturbations dureront environ 20 minutes. Je serai assis sur un banc fixé à une plateforme de perturbations et serai attaché à l'aide d'une ceinture. Je devrai d'abord rester immobile pendant trois essais de quelques secondes. Ensuite, des perturbations seront appliquées en déplaçant la plateforme en translation vers l'avant et vers l'arrière. Je devrai ensuite rester immobile pendant trois essais de quelques secondes. Pendant tous les essais, je devrai essayer de rester le plus stable possible dans la position.

5 - Avantages pouvant découler de ma participation

Je ne retirerai personnellement pas d'avantages à participer à cette étude. Toutefois, j'aurai contribué à l'avancement de la science.

6 - Risques pouvant découler de ma participation

Ma participation à ce projet de recherche ne me fait courir, sur le plan médical, aucun risque que ce soit.

7 - Inconvénients personnels

La durée de la session d'environ deux heures peut représenter pour certaines personnes un inconvénient. La possibilité que l'on doive raser le participant pour poser les électrodes peut aussi représenter un inconvénient. Il arrive parfois que les électrodes occasionnent de l'irritation cutanée. De plus, certaines personnes pourraient ressentir de la fatigue, de l'inconfort ou une sensation de vertige pendant ou suite aux perturbations. Il est entendu que je pourrai à tout moment interrompre la session si je le désire.

8 - Accès à mon dossier médical

L'accès à mon dossier médical n'est pas requis pour cette étude.

9 - Confidentialité

Tous les renseignements personnels recueillis à mon sujet au cours de l'étude seront codifiés afin d'assurer ma confidentialité. Ces données seront conservées sous clé au centre de recherche de l'Hôpital juif de réadaptation par un responsable de l'étude pour une période de cinq ans. Seuls les membres de l'équipe de recherche y auront accès. En cas de présentation de résultats de cette recherche ou de publication, rien ne pourra permettre de m'identifier.

10 - Questions concernant cette étude

Le(s) chercheur(s) présent(s) lors de la collecte des données s'engage(nt) à répondre de façon satisfaisante à toutes mes questions concernant le projet de recherche.

11 - Retrait de la participation du sujet

Ma participation au projet de recherche décrit ci-dessus est tout à fait libre et volontaire. Il est entendu que je pourrai, à tout moment, mettre un terme à ma participation.

En cas de retrait de ma part, les documents audiovisuels et écrits me concernant seront détruits.

12 - Clause de responsabilité

En acceptant de participer à cette étude, je ne renonce à aucun de mes droits ni ne libère les chercheurs, le commanditaire ou les institutions impliquées de leurs obligations légales et professionnelles.

13 - Indemnité compensatoire

Une compensation financière me sera remise à la fin de la session expérimentale afin de défrayer les frais de déplacement encourus pour participer à cette étude.

14 - Personnes-ressources

Si je désire poser des questions sur le projet, signaler un effet adverse et/ou un incident défavorable, je peux rejoindre en tout temps Dre Julie Côté, professeure adjointe au Département de kinésiologie et d'éducation physique de l'université McGill au (450) 688-9550, poste 4813.

De plus, si j'ai des questions sur mes droits et recours ou sur ma participation à ce projet de recherche, je peux communiquer avec Me Anik Nolet, coordonnatrice à l'éthique de la recherche des établissements du CRIR au (514) 527-4527 poste 2643 ou par courriel à l'adresse suivante: anolet.crir@ssss.gouv.qc.ca

CONSENTEMENT

Je déclare avoir lu et compris le présent projet, la nature et l'ampleur de ma participation, ainsi que les risques et les inconvénients auxquels je m'expose tels que présentés dans le présent formulaire. J'ai eu l'occasion de poser toutes les questions concernant les différents aspects de l'étude et de recevoir des réponses à ma satisfaction.

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