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Cervical Multifidus Muscle Activity during Whiplash and Startle Responses

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ABSTRACT

The cervical multifidus muscles insert onto the lower cervical facet capsules and the cervical facet joints are the source of pain in some chronic whiplash patients. Our goal was to determine if the cervical multifidus muscles were active during a simulated rear-end collision and a loud acoustic stimulus, and thereby assess whether they can contribute to injuring the facet capsular ligaments during a whiplash exposure. Wire electromyographic (EMG) electrodes were inserted unilaterally into the cervical multifidus muscles of 9 subjects (6M, 3F) at the C4 and C6 levels. Seated subjects were then exposed to two external stimuli: a loud acoustic tone (124dB, 40ms, 1kHz) and a rapid forward acceleration (peak acceleration 1.4g, speed change 1.8 km/h). Aside from one female, all subjects exhibited multifidus activity after both stimuli (8 subjects at C4, 6 subjects at C6). Neither onset latencies nor EMG amplitude varied with stimulus type or electrode level (p=0.13). Onset latencies and amplitudes varied widely, with EMG activity during the acceleration stimulus occurring early enough in 5 subjects to exacerbate the peak collision-induced loading of the facet capsular ligaments reported by others. These data indicate that the multifidus muscles of some individuals are evoked during a rear-end collision and support a role for the multifidus muscles in the genesis of facet capsule injury in whiplash.

INTRODUCTION

The cervical facet joints are a source of neck pain in about half of chronic whiplash patients (Lord et al, 1996a). In addition to guiding better diagnostic and treatment techniques (Lord et al., 1996b; McDonald et al., 1999), this finding provides an anatomical focus to biomechanical studies aimed at understanding the aetiology of whiplash injuries. Pinching of the posterior synovial fold of the cervical facet capsular ligament is one possible injury mechanism (Kaneoka et al., 1999), but more attention has been devoted toward excess strain of the capsular ligament itself (Yoganadan et al., 1998; Winkelstein et al., 2000; Siegmund et al., 2000; Pearson et al., 2004). Injurious levels of strain have been observed in some capsular ligaments when loads simulating a rear-end collision are applied in-vitro (Siegmund et al., 2000; Pearson et al., 2004). More recently, allodynia—measured as paw withdrawals in a rat model—has been correlated to levels of capsular

ligament strain relevant to whiplash injury (Lee et al., 2004), and Group III and IV afferents from the facet joint capsule have demonstrated a graded response to mechanical loading in an in-vivo goat model (Lu et al., 2005).

Anatomically, the cervical facet capsule contains fine, unmyelinated nerves that likely have nociceptive function (McLain, 1994). Distending these ligaments by injection of contrast media has produced whiplash-like pain patterns in normal individuals (Dwyer et al., 1990). Tendons of the cervical multifidus muscles insert directly onto the capsular ligaments (Winkelstein et al., 2001; Anderson et al., 2005) and it has been postulated that multifidus activation during the neuromuscular response to a rear-end automobile impact could increase the strain in the capsular ligaments above that imposed passively by the impact-induced head and neck dynamics (Winkelstein et al., 2001; Siegmund et al., 2000). It remains unclear, however, if the multifidus muscles activate reflexively during a whiplash response.

The neuromuscular response to a whiplash exposure contains both a postural and a startle response (Blouin et al., 2006a). This combined postural/startle response was observed in surface electromyograms of the sternocleidomastoid and cervical paraspinal muscles with and without the loud sound that accompanies a vehicle crash (Blouin et al., 2006a), although the muscle response amplitude was larger with the loud sound (Blouin et al., 2006b). These prior findings suggest that the startle response amplifies the neck muscle response and could increase the potential for a capsule-based whiplash injury. But because these prior studies used only surface recordings of muscle activity, it remains unclear whether the deep multifidus muscles are active during a startle response.

The goal of this study was to measure the electromyographic (EMG) response of the cervical multifidus muscle during exposures to both a simulated rear-end collision and a loud acoustic stimulus capable of evoking a startle response. Evidence of reflex activation of the multifidus to one or both of these stimuli would provide evidence supporting a potential neuromuscular contribution to the genesis of facet-based whiplash injuries.

METHODS

Nine subjects (6M, 3F) participated in the experiment. Male subjects were 30±6 years old, 177±6 cm tall and weighed 79±5 kg; female subjects were 30±1 years old, 166±5 cm tall and weighed 68±6 kg. None of the subjects had a history of whiplash injury, medical conditions that impaired sensory or motor function, or prolonged neck or back pain during the preceding 2 years. Subjects did not ingest caffeine or nicotine for two hours before the experiment. All subjects gave written inform consent and the experiment was approved by the UBC Clinical Research Ethics Board and conformed to the Declaration of Helsinki.

EMG activity of the left multifidus muscles was measured using twisted pairs of insulated 0.05 mm wire (Stablohm 800A, California Fine Wire, Grover Beach, CA) with 0.5 mm of wire exposed at each recording tip. The recording tip of each wire was hooked to anchor it in the muscle tissue. After first identifying multifidus and any major vessels on each subject's MR scan (Phillips Gyroscan Intera 3.0T), wires were inserted into the cervical multifidus muscles at the C4 and C6 levels using 25 gauge needles under ultrasound guidance (Sonos 5500, Agilent Technologies, Andover, MA). Ultrasound was again used during wire extraction to confirm the wires had not moved during the experiment. Although the wires were harder to visualize during extraction (without the needle present), tissue displacement at the hooked end was readily apparent during wire extraction. EMG signals were amplified and band-pass filtered (30–1000 Hz) using a Neurolog system (Digitimer, Welwyn Garden City, Hertfordshire, England). Head acceleration was measured with a nine accelerometer array (Kistler 8302B20S1, Amherst, NY) and sled acceleration was measured with a uniaxial accelerometer (Sensotec JTF3629-05, Columbus, OH). Head and torso displacements were measured with a motion analysis system (Phoenix VZ4000, Burnaby, BC). Transducer signals were low-pass filtered (1000 Hz) and, together with the EMG signals, simultaneously sampled at 2 kHz. Displacement data were acquired at 100 Hz per marker.

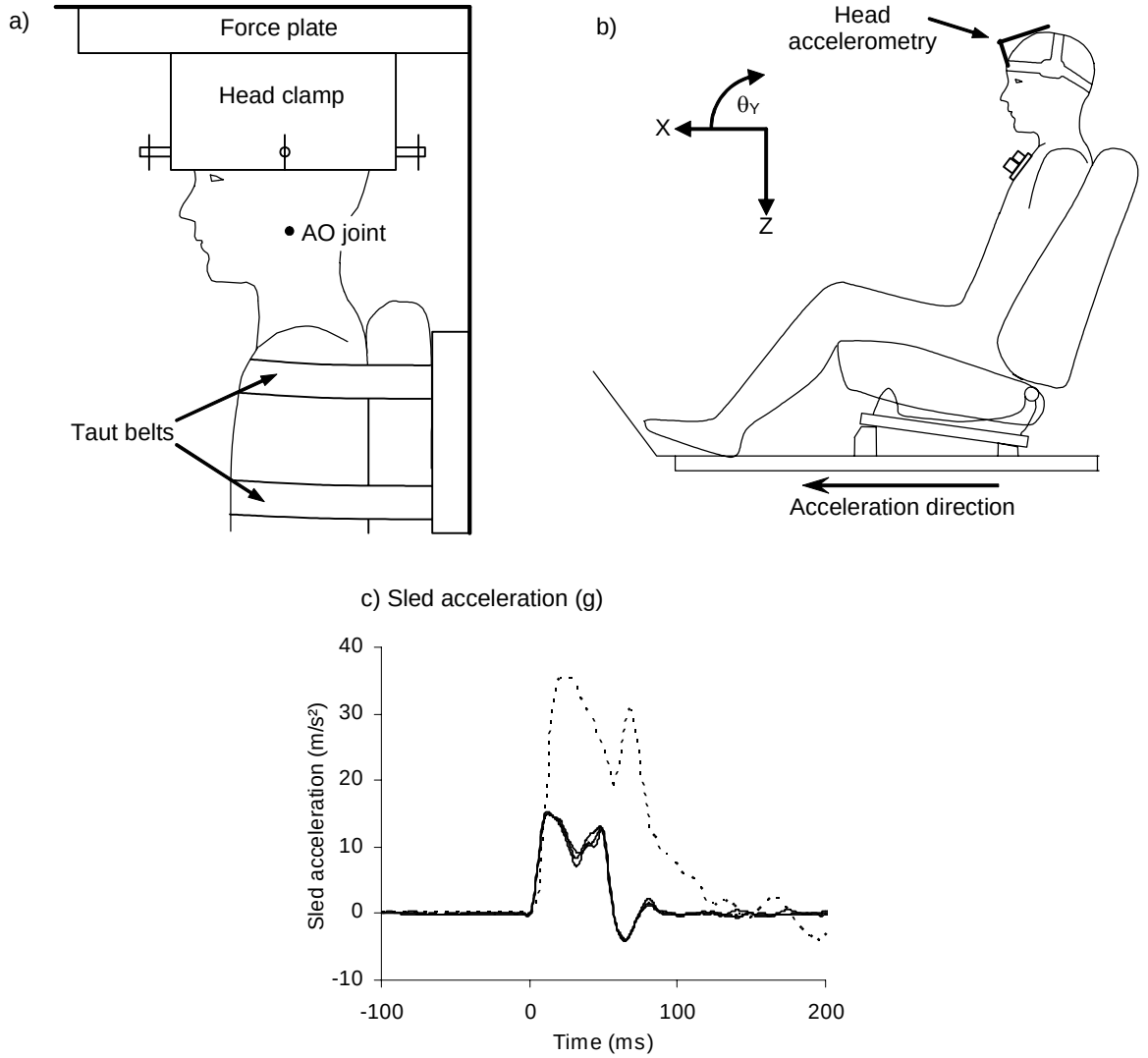

Figure 1: Schematic showing a) the head clamp and force plate used for the isometric contractions, b) the sled configuration, and c) the sled acceleration pulses. Three pulses are shown to illustrate the repeatability of the pulse. The dashed line shows a vehicle-to-vehicle collision pulse with a speed change of 8 km/h recorded during earlier experiments (Siegmund et al., 2001a).

Subjects first performed 5-second isometric maximal voluntary contractions (MVCs) from the neutral position in eight directions (flexion, extension, left and right lateral flexion, and the 45º points between these 4 primary directions) to provide normalizing data for the dynamic EMG recordings. For the MVCs, a subject's head was firmly clamped to an inverted force plate (Bertec 4060H, Worthington, OH) and their torso firmly strapped to a rigid seat back (Figure 1a). For their exposure to the whiplash and acoustic stimuli, subjects were seated in an automobile seat (1991 Honda Accord front passenger with the head restraint removed) mounted to a feedback-controlled linear sled (Figure 1b). Prior to the sled test, subjects were instructed to adopt a comfortable seated posture, face forward, rest their forearms on their lap, and relax their face and neck muscles. Seated subjects were first exposed to a single unexpected loud acoustic stimulus (124dB, 1000Hz, 40ms duration) capable of evoking a startle reflex (Siegmund et al., 2001b). After a rest period of at least 3 minutes, subjects experienced a single forward horizontal acceleration pulse (a_{peak}=1.55g; t_{peak}=16.6ms; Δt=59ms; Δv=0.50m/s, Figure 1c). A single exposure to each stimulus was used because

multiple exposures could be confounded by the rapid habituation observed to both acoustic startle (Brown et al., 1991; Bisdorff et al., 1994) and whiplash perturbations (Blouin et al., 2003; Siegmund et al., 2003b).

All EMG data were first high-pass filtered (50 Hz) to remove motion artifact present in some of the dynamic EMG data. The onset of EMG activity was determined using a log-likelihood-ratio algorithm (Staude and Wolf, 1999; Staude, 2001) and then confirmed visually. For both the whiplash and acoustic stimuli, the root-mean-squared (RMS) amplitude of the EMG signals was computed using a moving 20 ms window. These RMS values were then normalized by the maximum RMS EMG (also using a 20 ms window) observed during the MVC contractions.

For the whiplash and acoustic stimuli, three kinematic parameters were calculated: i) the horizontal acceleration of the head's centre of mass in the lab frame (a_x), ii) the horizontal displacement (retraction, r_x) of the atlanto-occipital (AO) joint with respect to the centre of the T1 vertebral body, and iii) the extension angle (θ_y) of the head in the lab frame. The center of the T1 vertebral body relative to the manubrium and C7 spinous process was determined from each subject's pre-test MR scan. All time-varying kinematic signals were set to zero at the start of the stimulus.

All dependent variables were first tested for normality using a Shapiro-Wilks test. Differences between the stimulus (sled/acoustic) and spine level (C4/C6) were tested using a two-way repeated-measures ANOVA and Tukey post-hoc tests for normally distributed variables. For variables not normally distributed, a non-parametric Friedman ANOVA and post-hoc Wilcoxon matched pairs tests were used. All tests were performed using Statistica (v.6.1, Statsoft, Tulsa, OK) with a significance level set at $p=0.05$.

RESULTS

Eight of the nine subjects exhibited multifidus activity after both stimuli (Figure 2). The remaining subject exhibited no multifidus activity to either stimulus—despite clear multifidus activity during her MVC—and was excluded from further analysis of the EMG data. Within the eight analyzed subjects, multifidus activity was present in all subjects at the C4 level and in six subjects at the C6 level. Supramaximal muscle activity was observed either at one level or to one of the two stimuli in six subjects. There were no differences in the muscle response amplitude between the sled and acoustic stimuli or between the two spinal levels ($p=0.32$).

There were also no differences in the onset latencies between either stimulus types or recording levels ($p=0.41$). There was however considerable variation in the onset latencies between subjects (Table 1), with multifidus onset occurring within 160 ms of stimulus onset in five subjects during the sled acceleration and six subjects during the acoustic stimulus.

All subjects exhibited a well-defined forward head acceleration, head extension, and retraction of the AO joint relative to T1 when exposed to the sled stimulus (Figure 2). The peak kinematic responses for the acoustic stimulus were smaller ($p<0.02$; Table 1) and also varied in direction, with the initial head acceleration forward in four subjects and rearward in the other subjects.

DISCUSSION

Our data indicate that the cervical multifidus muscles of most individuals respond to both a forward, seated acceleration and an acoustically startling tone. The wide intra-subject variability in the onset latency and response amplitude is similar to that observed in the lumbar multifidus muscles (Moseley et al., 2002), and suggests that if multifidus plays a role in the genesis of whiplash injuries, this role may vary considerably between individuals. This large variability is consistent with the whiplash-related epidemiological data: acute whiplash injury affects only a portion of the exposed population and chronic symptoms affect only a subset of those with acute injuries (Freeman et al., 1998; Pobereskin, 2005). The challenge for whiplash researchers then is to explain why some individuals are injured and others are not. To address this challenge, researchers need to consider how the variability within individuals and the variability between individuals combine to injure only a portion of the exposed population. The variability in multifidus muscle responses observed here, combined with the prior reports of the variability in cervical facet anatomy and morphology discussed below, may explain the observed variability in whiplash injury potential.

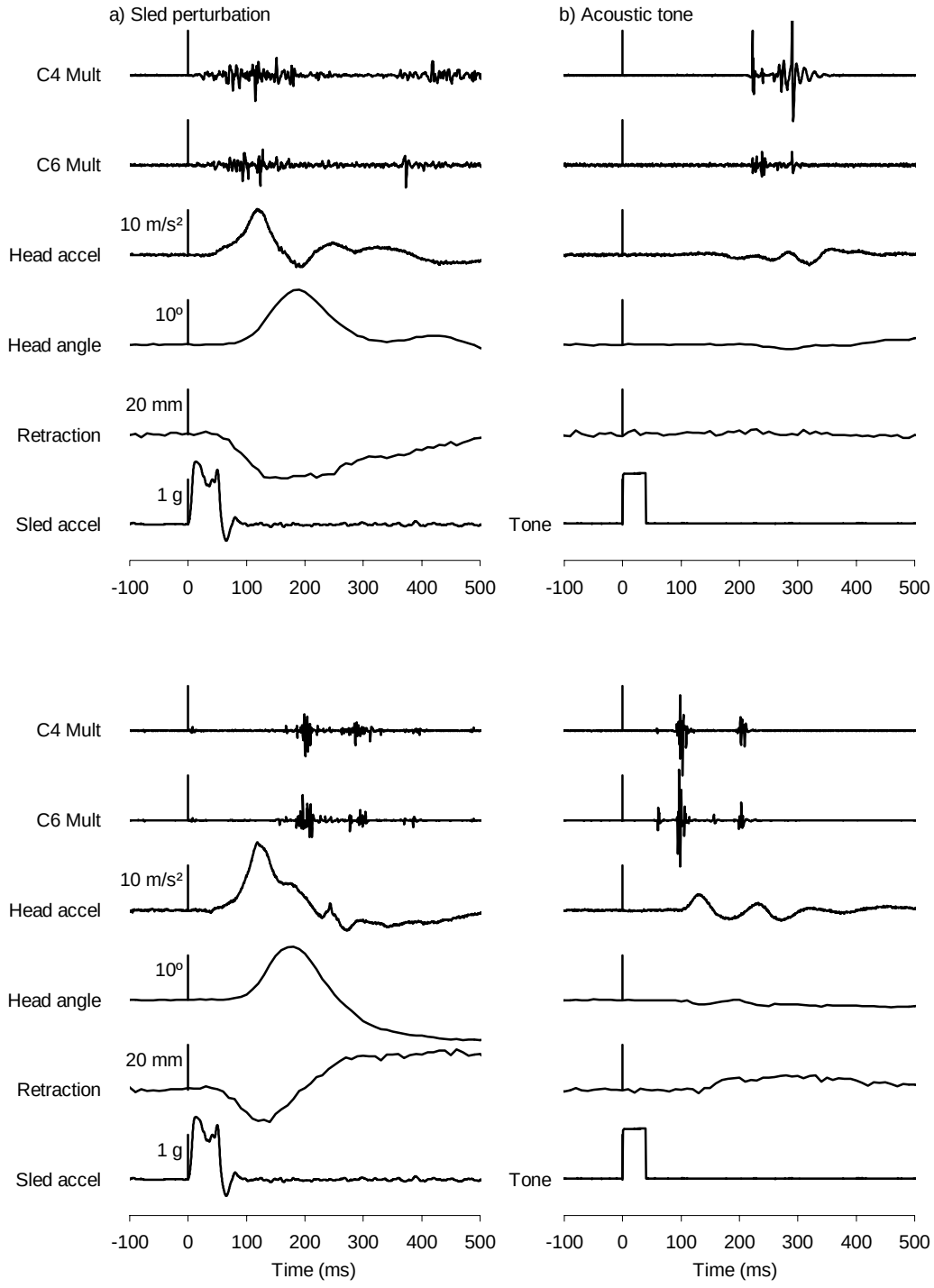

Figure 2: Exemplar data for two subjects showing the raw electromyographic activity and kinematics parameters observed during a) the sled perturbation and b) the loud acoustic tone. The vertical scale bars are aligned with time t=0 ms. EMG are presented in arbitrary units that are the same for both stimuli.

Table 1. Mean ± SD and median (range) of muscle onset times, normalized muscle amplitudes and kinematic amplitudes.

	Sled	Startle
EMG onset time (ms)		
C4 level	132 ± 57	117 ± 62
	153 (34 - 191)	92 (58 - 222)
C6 level	215 ± 169	186 ± 112
	175 (66 - 571)	169 (60 - 417)
EMG amplitude (%MVC)		
C4 level	1.37 ± 1.67 [a]	1.36 ± 1.23 [a]
	1.07 (0.12 – 5.31)	0.80 (0.39 – 3.37)
C6 level	0.40 ± 0.24	0.57 ± 0.53 [a]
	0.36 (0.09 – 0.77)	0.28 (0.15 – 1.50)
Kinematics		
Head accel (m/s²)	9.7 ± 3.3	1.9 ± 1.0
	10.1 (5.6 - 15.2)	1.6 (0.5 - 3.5)
Head extension (º)	15.6 ± 5.6	1.9 ± 3.1 [a]
	12.6 (10.0 - 25.9)	0.5 (0.1 - 8.8)
Retraction (mm)	18.4 ± 6.9	4.3 ± 2.4 [a]
	19.4 (8.7 - 31.3)	5.2 (1.0 - 6.6)

[a] non-normally distributed, Shapiro-Wilks $p<0.05$

Multifidus muscle activity must occur early to be relevant to whiplash injury. Peak capsular ligament strain occurs about 200 ms after the onset of T1 acceleration (Pearson et al., 2004) and the onset of T1 acceleration occurs about 25 to 35 ms after vehicle impact (Siegmund et al., 1997). Assuming muscles can generate meaningful levels of force by 75 to 100 ms following activation (Tenneyson et al., 1977; Andersen and Aagaard, 2006), multifidus activation must occur within 125 to 160 ms after impact (i.e., sled onset) for the muscle fibres that insert onto the capsular ligament (Winkelstein et al., 2001) to load the ligament at the same time the collision-induced strain peaks. Of the nine subjects we tested, multifidus activation occurred prior to 160 ms in 5 subjects following the sled perturbation and 6 subjects following the startling tone. Thus, in more than half our subjects, multifidus was active during an interval in which it can contribute to a facet capsular injury.

To be meaningful in the context of whiplash injury, multifidus must also generate a relatively large force in relation to the failure loads of the capsular ligament. The area of multifidus insertion onto the facet capsule varies between 9 and 96 mm² (average of 48±22 mm²; Winkelstein et al., 2001), which for an isometric tetanic stress of 0.44 MPa (Myers et al., 1998), corresponds to an applied force of 9 to 42 N (average 21 ± 10 N). During elongation, force increases by a factor of 1.2 to 1.6 depending on lengthening velocity (Scott et al., 1996) and therefore the force applied to the capsule would be between 11 and 67 N (average 29 ± 14 N; a factor of 1.4 is assumed for these calculations). Compared to the loads required to cause partial rupture of the capsular ligament (21 to 93 N; 45±21 N; Siegmund et al., 2000), the potential loads applied by early activation of the multifidus muscle reach a large proportion (64% on average) of the loads needed to injure the capsular ligament. Intervertebral motion induced by the collision is estimated to generate about 14 N of loading to the capsular ligament (Winkelstein et al., 2001), sufficient to bring the total capsular loading up to 43 N, just below the average load to cause partial rupture. This analysis shows that reflex activation of the cervical multifidus muscle during a rear-end collision could generate the dominant loads applied to the capsular ligaments of the lower cervical spine. In particular, joints with large multifidus/capsular insertion areas, low partial-rupture loads, and high levels of multifidus activation during

the collision would be particularly prone to injury. Unfortunately, the large within-individual variations in insertion area (Winkelstein et al., 2001) and partial-rupture load (Siegmund et al., 2000), combined with the large between-subjects variability in multifidus activation timing and amplitude observed here, make it difficult—if not impossible—to predict who is a risk for this type of injury.

The high percentage of Type I muscle fibres (Boyd-Clark et al., 2001), the small number of vertebra spanned (Anderson et al., 2005), and the low moment generating capacity (Anderson et al., 2005) of the cervical multifidus muscles suggest a primarily postural role—perhaps providing stability to the intervertebral joints during movements driven by larger, more superficial neck muscles. Thus posture-related activity in the multifidus muscle could conceivably provoke pain in a whiplash–injured capsular ligament. Moreover, the high number of muscle spindles in multifidus (Boyd-Clark et al., 2002) suggests an important role for multifidus in directly sensing spine joint position and perhaps contributing to head position sense. If pain from an injured capsular ligament inhibits or alters tonic or focal multifidus activity levels, then altered somatosensory information from muscle spindles could also contribute to the dizziness experienced by many whiplash patients (Evans, 1992). Indeed, increased joint position error in whiplash patients has been previously attributed to mechanoreceptor dysfunction in the cervical spine (Treleaven et al., 2003; Heikkila and Wenngren, 1998), and a dysfunctional interaction of the capsular ligament and the multifidus muscle provides a rational explanation for these symptoms.

The present study supports the hypothesis that neck muscles contribute to whiplash injury and suggests that individuals more prone to startle may be more prone to whiplash injury. Startle forms part of the neuromuscular response to a rear-end collision (Blouin et al., 2006a) and the combination of a loud noise and a simulated rear-impact generates larger posterior neck muscle responses (measured using surface EMG) than an impact alone generates (Blouin et al., 2006b). If this apparent summation of impact noise and impact-induced motion applies to the deep multifidus muscles as well, then easily startled individuals may experience larger capsular strains than less-easily startled individuals, and may therefore be more prone to whiplash injury. Since females are more prone to startle than are males and females have larger amplitude muscle responses to startle than do males (Kofler et al., 2001), this proposition may partially explain the greater frequency of whiplash injuries in females than in males (Jakobsson et al., 2000; Berglund et al., 2003).

The preceding analysis assumes maximal or near maximal levels of multifidus muscle activation. The present data shows considerable variation in the amplitude of the multifidus muscle response, with about half the subjects exceeding their MVC levels. Supramaximal muscle activity has been observed by others studying whiplash (Kumar et al., 2002), but it remains unclear why or how this occurs. Subjects simply may have not exerted maximal efforts during their MVCs; however, the neck moments measured in our male subjects (flexion 12 ± 6 Nm; extension 20 ± 8 Nm) are similar to maximal neck moments (flexion 13 ± 3 Nm; extension 24 ± 7 Nm) measured by others (Vasavada et al., 2001). Alternatively, multifidus may not recruit maximally during contractions designed to maximize the horizontal force at the forehead. Or perhaps short, reflex muscle activations generate more synchronous bursts of action potentials than do 5 second maximal voluntary efforts, and summation of these synchronous action potentials yields a greater EMG signal. Further work is needed to determine why this supramaximal activity occurs and how to estimate multifidus muscle force from these data.

We presented the sled and tone stimuli separately to subjects, whereas the acceleration and noise occur simultaneously during an actual collision. Thus vehicle occupants who are unexpectedly struck from behind may experience a superposition of the two neuromuscular responses we observed. The acoustic stimulus is also greater and sled perturbation is less than that experienced by many whiplash-injured patients, although the leading edge of the sled pulse simulates the onset of a vehicle collision with a speed change of 8 km/h (Figure 1c). Both stimuli levels were chosen because they were known to evoke measurable neck muscle activity using surface EMG with little risk of injury (Blouin et al., 2003; Siegmund et al., 2003a,b).

CONCLUSIONS

In summary, cervical multifidus muscle activity is evoked by both horizontal sled accelerations and acoustically startling tones. Direct insertion of the multifidus muscle onto the cervical facet capsular ligaments provides a mechanism by which multifidus activation can exacerbate whiplash injury potential,

however the large variability in both onset latency and activation amplitude suggests that this mechanism of injury may not operate in all individuals.

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DISCUSSION

PAPER: **Cervical Multifidus Muscle Activity during Whiplash and Startle Responses**

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QUESTION: *Frank Pintar, Medical College of Wisconsin*
Very nice. I might have missed it, but because the multifidus is involved in posturally keeping your head up right, were you able to control for that? I mean, how do you know if it's partially activated just because they had their head upright?

ANSWER: It is partially active when you are sitting upright. It's very quiet compared to the levels that we see during the stimuli that we use; but if you zoom in on the EMG, you can see there's some motor units ticking away. So, the way we control for it is we subtract off the baseline EMG immediately for the stem from the stem.

Q: But the variability is such that you're subtracting off different amounts for each individual, then, right?

A: Not only each individual; but if we did multiple trials on a single individual, they would likely have a different level of tonic activity immediately because of the stem for every trial.

Q: Okay. So does that affect when the person responds or to what magnitude they respond, or was there no pattern—in terms of the baseline?

A: In the limited data set we have, I wouldn't be able to discern that.

Q: Okay. Thank you.