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Sensory pathways mediating the neck muscle response in rear-end impacts

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ABSTRACT

The goal of this study was to examine whether the vestibulocollic (VCR) and cervicocollic (CCR) reflexes play a primary or secondary role in the activation of neck muscles to transient perturbations simulating a rear-end collision. To achieve this goal, we exposed subjects to transient surge perturbations and compared the onset and amplitude of their neck muscle responses to the onset and amplitude of their torso and head accelerations. We hypothesized that neck muscle activation times and response amplitudes would correlate to the onset and amplitude of either i) torso acceleration, if the CCR was a primary mediator of the neck muscle response, or ii) head acceleration, if the VCR was a primary mediator of the neck muscle response. Thirty subjects (15F, 15M) underwent 36 consecutive forward horizontal perturbations consisting of three different peak accelerations (7.8, 14.7, and 21.7 m/s²) and three different final velocities (0.25, 0.50, and 0.75 m/s). Onset and amplitude of the sternocleidomastoid (SCM) and cervical paraspinal (PARA) muscle responses were measured using surface electromyography. Onset and amplitude of the torso and head acceleration were measured using accelerometers on the torso (manubrium) and head. Weak correlations between the onset of muscle activation and the onset of both torso and head accelerations suggested that neither the cervicocollic nor vestibulocollic pathways were primarily responsible for activation of the neck muscles.

INTRODUCTION

To counter the inherent instability of a large head mass atop a multi-articulated neck, reflexes such as the vestibulocollic reflex (VCR), cervicocollic reflex (CCR), and longer-latency polysynaptic reflexes have developed. This actively-controlled system of reflexes manages to balance the competing demands of voluntary head movement and head stability – both in space and relative to the torso – during both familiar body movements and novel perturbations.

In addition to the knowledge gained from studying the head/neck system dynamics, insight into the neck neuromusculature has also been gained by studying the onset latencies and amplitude of the human neck muscle responses to transient perturbations in standing, supine and seated postures. In studies of perturbed standing, neck muscles have been observed to activate at or before abdominal muscles (Keshner et

al., 1988). Although this deviation from a sequential upward muscle activation pattern could suggest that the VCR triggered the neck muscle response (Woollacott et al., 1988), a comparison between normal and avestibular subjects demonstrated only minor differences in neck muscle onset latencies and response amplitudes (Keshner et al., 1988) and thus indicated that the VCR was not primarily responsible for neck muscle activation in perturbed standing.

During head drop tests in supine subjects, short onset latencies (24 ± 3 ms after onset of head acceleration) were observed in the sternocleidomastoid (SCM) muscles of normal subjects, whereas longer onset latencies (67 ± 7 ms) were observed in avestibular subjects (Ito et al., 1995, 1997; Bisdorff et al., 1999). These studies showed that the VCR could activate neck muscles at very short latencies. The longer onset latencies observed in the avestibular subjects, however, were still about 40 ms shorter than voluntary SCM activation times (Mazzini and Schieppati, 1992; Siegmund, 2001). This suggested that other reflexive pathways, possibly polysynaptic in nature, also mediate neck muscle activation.

In seated subjects exposed to transient perturbations, Gresty (1989) reported bursts of neck muscle activity 25 to 50 ms after torso acceleration onset during surge perturbations and Kanaya et al. (1995) reported onset of neck muscle activity as short as 25 ms after onset of angular head acceleration in two of seven subjects exposed to pitch perturbations. Various other groups have reported neck muscle activation times of 69 to 82 ms in seated subjects during surge perturbations (Forssberg and Hirschfeld, 1994; Brault et al., 2000; Vibert et al., 2001; Siegmund et al., 2002), although these latencies were reported relative to platform motion and thus likely overestimated the time between mechanoreceptor stimulation and neck muscle activation.

Based on similarly short neck muscle onset latencies in seated normal and avestibular subjects exposed to transient surge perturbations, Gresty (1989) concluded that the neck muscle response was mediated by the CCR rather than the VCR. Using consecutive transient pitch and surge perturbations, Forssberg and Hirschfeld (1994) also excluded a major vestibular component to the postural response of the neck muscles, but rather than the CCR, these researchers suggested that pelvic rotation relative to the support surface triggered the neck muscle response. Based on a shortening of the sternocleidomastoid muscle during their pitch perturbations, Kanaya et al. (1995) eliminated a homonymous stretch reflex as a likely trigger for neck muscle activation. Instead, these authors concluded that the neck muscle response was likely mediated by the VCR in normal subjects, and by an unloading response or trunk-based mechanoreceptors in avestibular patients.

These perturbation studies in standing, supine, and seated postures have shown that the VCR can operate quick enough to trigger the observed neck muscle responses, though it apparently does not do so under all circumstances. When the perturbation is isolated to the head (e.g., head drop tests), the VCR plays a dominant role in triggering the muscle response; whereas, under other circumstances (e.g., standing or seated perturbations), the VCR appears to be less consistent. To further examine whether the VCR and CCR play a primary or secondary role in the activation of neck muscle responses to transient perturbations, we compared the onset and amplitude of neck muscle responses to the onset and amplitude of the torso and head accelerations in subjects exposed to transient surge perturbations. To generate the necessary stimulus-response data, seated subjects were exposed to abrupt transient surge perturbations of different accelerations and velocities. It was hypothesized that neck muscle activation times and response amplitudes would correlate to the onset and amplitude of either i) torso acceleration if the CCR was a primary mediator of the neck muscle response, or ii) head acceleration if the VCR was a primary mediator of the neck muscle response.

METHODS

Subjects

Thirty subjects (15F, 15M) with no history of sensorimotor dysfunction or neck/back pain participated in the experiment. Female subjects were 21 ± 3 years old, 166 ± 7 cm tall and 59 ± 8 kg; male subjects were 25 ± 6 years old, 175 ± 8 cm tall and 78 ± 14 kg. All subjects gave their informed consent and

were paid a nominal amount. The use of human subjects was approved by the university's Ethics Review Board and the study conformed to the Declaration of Helsinki.

Instrumentation

Electromyographic (EMG) activity in the sternocleidomastoid (SCM) and paraspinal (PARA) muscles was recorded bilaterally using pairs of 10-mm diameter disposable surface electrodes (H69P, Kendall-LTP, Huntington Beach, CA) spaced 10 mm apart and amplified through an Octopus AMT-8 amplifier (Bortec, Calgary, AB). SCM electrodes were placed mid-belly and PARA electrodes were placed at the C4 level, 2 cm either side of the midline. At this level, the PARA electrodes were overlying portions of the trapezius, splenius capitis, semispinalis capitis, and semispinalis cervicis muscles. Two orthogonally-mounted, uni-axial, linear accelerometers (Kistler 8302B20S1, Amherst, NY) were used to measure horizontal (x') and vertical (z') head acceleration and an uni-axial angular rate sensor (ATA Sensors ARS-04E, Albuquerque, NM) was used to measure flexion and extension velocity. All three transducers were strapped to the forehead immediately above the glabella (Figure 1). Torso acceleration was measured using a similar uni-axial linear accelerometer located 2 cm below the superior margin of the manubrium. Horizontal sled acceleration was measured using a uni-axial linear accelerometer (Sensotec JTF3629-05, Columbus, OH). Head and torso positions before and during the perturbation were measured using a 3D position tracking system (Optotrak 3020, Northern Digital, Waterloo, ON) with infrared-emitting markers on the left seat hinge, left mastoid process, and midline at the forehead, vertex, manubrium, and C7 spinous process (Figure 1). The RMS accuracy of the position measurements from the Optotrak system was less than 0.1 mm and, based on marker separation, the RMS accuracy of the calculated angles was less than 0.1 degrees. EMG signals were bandpass filtered (10 to 1000 Hz) and transducer signals were low-pass filtered (1 kHz) before being simultaneously sampled at 2 kHz and stored for subsequent analysis. Optotrak data were acquired at 200 Hz per marker.

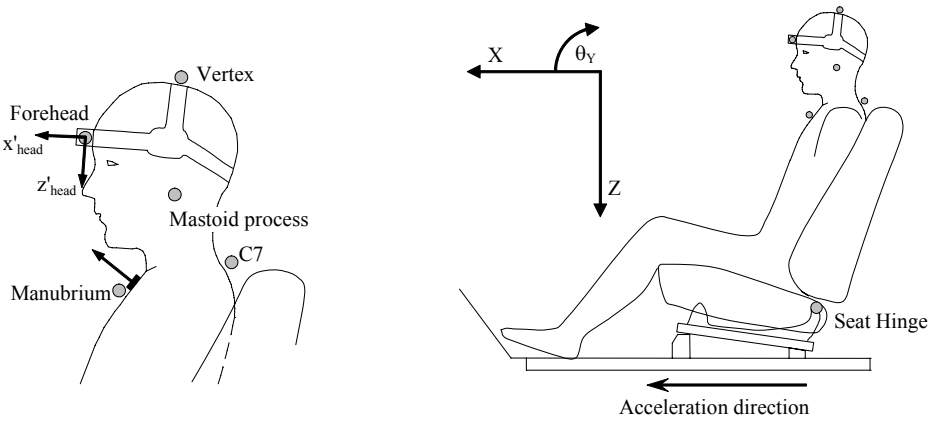

Figure 1: Schematic of the test configuration showing the locations of the Optotrak markers (shaded circles), the lab reference frame (X, Z), and the head reference frame (x', z'). The initial orientation of the head reference frame was governed by how the head band fit the subject and varied between +12 ± 4 deg relative to the lab reference frame. The sensitive axis of the torso accelerometer was also governed by body shape and varied between +30 ± 5 deg relative to the lab reference frame.

Test Procedures

The front passenger seat of a 1991 Honda Accord was mounted on a sled powered by a feedback-controlled linear induction motor (Kollmorgen IC55-100A7, Kommack, NY). The head restraint was removed from the top of the seat back to eliminate externally applied loads to the head during the perturbations. The sled generated no pre-perturbation signals, either audible or mechanical, which could be used to predict perturbation onset.

Initially-stationary subjects were exposed to seven different perturbations (Figure 2). Peak accelerations of 0.79 g, 1.50 g, or 2.21 g (SD < 0.02g) were used to reach velocities of 0.25, 0.50, or 0.75 m/s

(SD < 0.003 m/s). Peak accelerations were achieved in 17.1 ± 1.1 ms in all trials. Low, medium and high accelerations were designated La, Ma, and Ha, respectively, and low, medium, and high velocities were designated Lv, Mv, and Hv, respectively. The combination of medium acceleration (Ma) and medium velocity (Mv) was designated the standard perturbation. Depending on the specific pulse, acceleration occurred over a distance of 4 to 32 mm. The sled then traveled briefly at a constant velocity before slowing to rest at the end of its 750-mm throw at 0.05 m/s², about 10 percent of the low acceleration (La) rate. The total elapsed time from onset of initial acceleration to rest varied between 1.8 and 3.3 seconds.

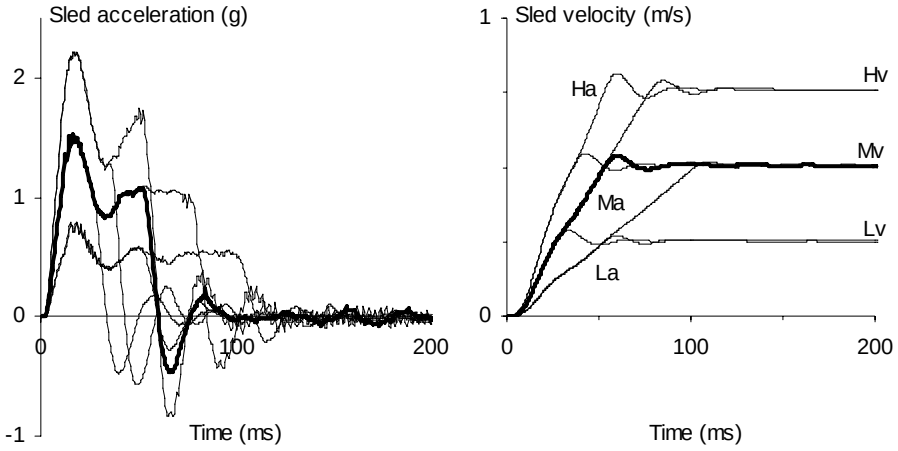

Figure 2: Acceleration and velocity of the seven perturbations. Superimposed acceleration vs. time traces (a) and velocity vs. time traces (b) show similarities between the perturbations. The bold lines depict the standard perturbation (MaMv) in both upper panels. Sled acceleration: low La, medium Ma, and high Ha; and sled velocity: low Lv, medium Mv, and high Hv.

Each subject underwent a single block of 36 perturbations: 12 standard perturbations and 4 each of the other six perturbations. After an initial standard perturbation, the order of presentation was randomized between subjects and the time between individual perturbations varied randomly (uniform distribution) between 20 and 30 seconds. Subjects were instructed to sit normally, face forward, rest their forearms on their lap and to relax their face and neck muscles. Subjects had their eyes open and were instructed to maintain an upright head posture. To achieve a stabilized response, each subject first underwent an additional eleven standard perturbations prior to testing.

Data Reduction

The ability of the subjects to reproduce their initial head and torso position and angle was assessed using Optotrak data taken immediately preceding each perturbation. The onset of head and torso movement and the amplitude of peak head and torso acceleration were determined directly from the accelerometer data. Forehead acceleration data were reported in the head reference frame (x', z'; Figure 1).

Pre-stimulus noise in each channel of the EMG data was quantified using the root mean squared (RMS) amplitude of the EMG signal over the 100 ms preceding the perturbation. EMG onset was defined as the time at which the RMS amplitude, computed from the raw EMG data using a moving 20 ms window, reached 10 percent of it maximum value and was then confirmed visually. For each muscle, the maximum RMS amplitude of the EMG signal was determined for the interval between EMG onset and peak head extension angle. The corresponding pre-stimulus noise for each muscle was then subtracted from this quantity.

Statistical Analysis

Within-subject means for each dependent variable were calculated for each of the seven perturbations. Amplitude measures were then normalized by the within-subject mean of the twelve standard perturbations. A one-way repeated-measures ANOVA was then used to assess differences between the seven perturbations for each dependent variable. Post-hoc testing was performed using a Scheffé test to identify homogenous groups. All ANOVAs were performed using Statistica (Statsoft Inc., Tulsa, OK) and a significance level of α=0.05. Separate linear regression analyses were used to examine the relationship between the onset and amplitude of muscle activity and the onset and amplitude of head and torso acceleration. Regression analyses were conducted using Excel (Microsoft Corp., Redmond, WA).

RESULTS

Subjects consistently reproduced the initial position and angle of their head and upper torso prior to each of the seven perturbations ($p>0.25$). Most subjects responded to a perturbation by rapidly restoring their upright head position (Figure 3), although some subjects periodically delayed restoration of their upright head position (right column, Figure 3). Despite these occasionally delayed responses, both acceleration and EMG onset times remained consistently short with little within-perturbation variability (Figure 4a-d).

Motion of the head, neck, and upper torso was observed first in the torso accelerometer and then in the vertical head accelerometer (Figures 3 and 4a,b). Acceleration onset times for both the torso and head varied between perturbations ($p<0.0001$) and the pattern of homogeneous groups showed that both onset times were sensitive to perturbation acceleration but not to perturbation velocity (Figure 4a,b). The onset latencies for the SCM and PARA muscles also varied between perturbations ($p<0.0001$), though the pattern of homogeneous groups in both muscles revealed shorter latencies for combined increases in perturbation acceleration and decreases in perturbation velocity (Figure 4c,d). As with the onset times, the peak amplitude of the head and torso acceleration and maximum RMS amplitude of the SCM and PARA EMG also varied significantly between perturbations ($p<0.0001$) (Figure 4e-h). High acceleration, high velocity (Ha,Hv) perturbations consistently produced the largest amplitude responses, and low acceleration, low velocity (La,Lv) perturbations, though not always significantly different from adjacent cells in the test matrix (e.g., La,Mv and Ma,Lv), consistently produced the smallest amplitude responses.

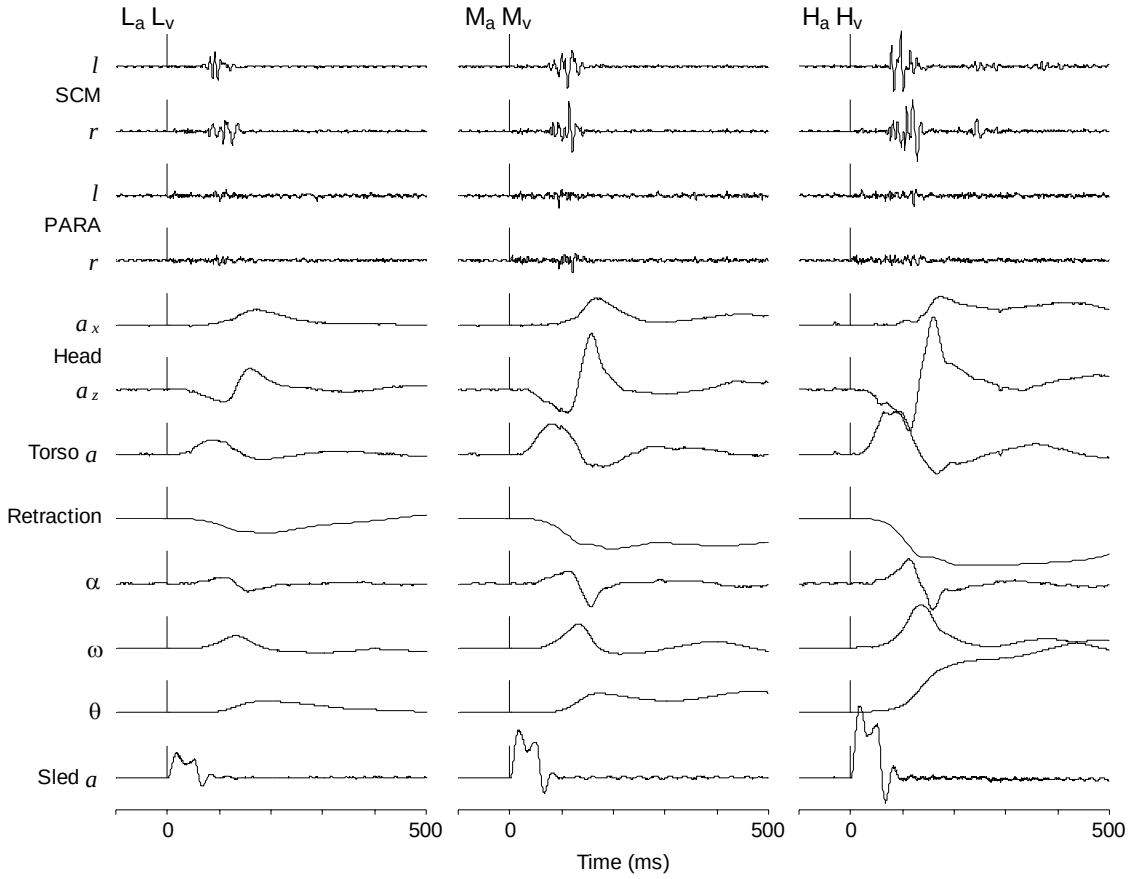

Figure 3: Sample data from a low velocity, low acceleration (LvLa), medium velocity, medium acceleration (MvMa), and high velocity, high acceleration (HvHa) perturbation for a single subject. Labeled hollow circles in the left panel represent kinematic peaks were used for subsequent analyses. The vertical scale bars are aligned with perturbation onset and equal to 1g, 50 mm, 200 rad/s², 5 rad/s and 20 deg, for their respective variables. SCM, sternocleidomastoid; PARA, cervical paraspinal - l, left; r, right; a, linear acceleration - subscript x and z refers to the x- and z-directions; r_x, retraction; α, head angular acceleration; ω, head angular velocity; θ, head angle.

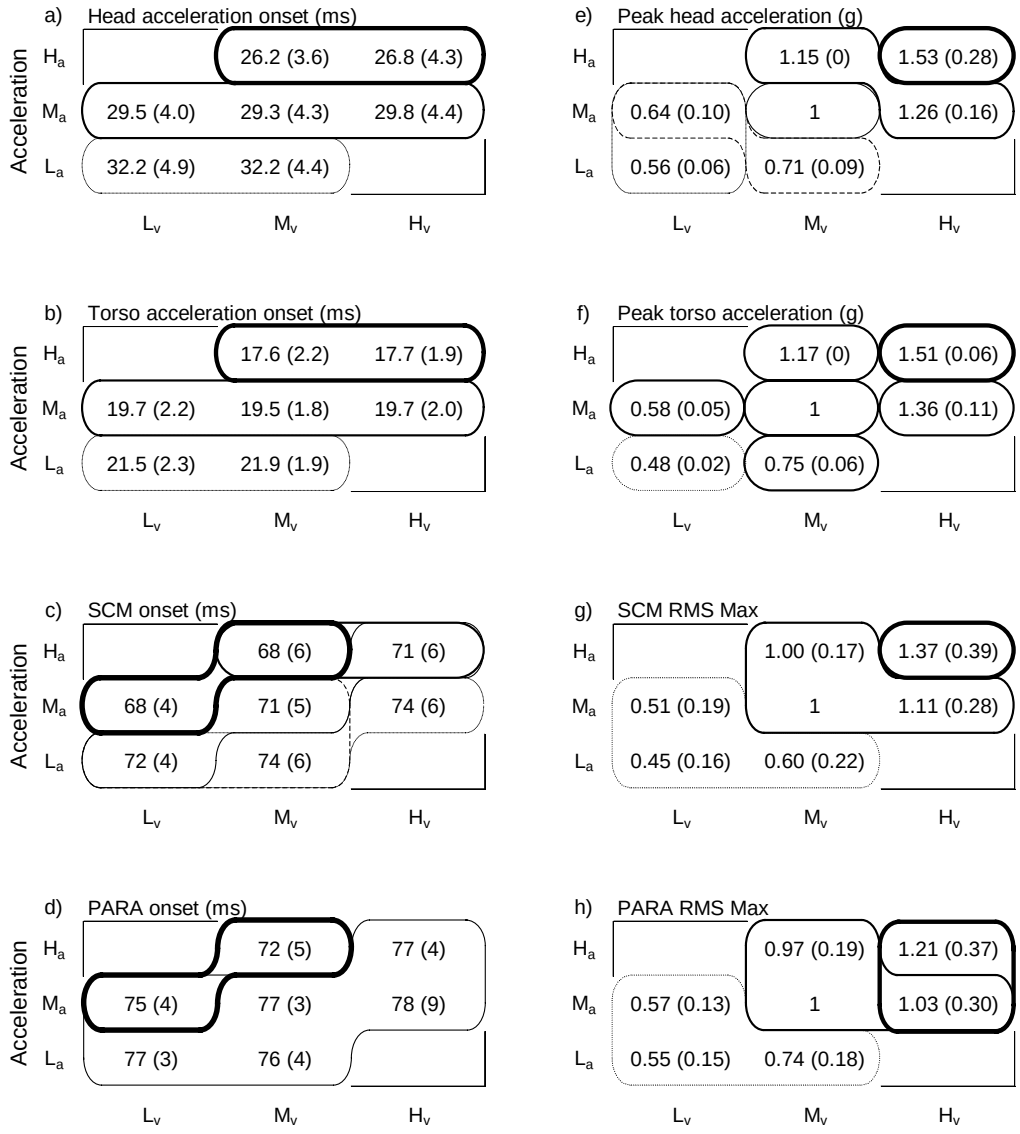

Figure 4: Mean and standard deviation of times for the (a) head acceleration onset, (b) torso acceleration onset, (c) SCM onset latency and (d) PARA onset latency; and normalized peak amplitudes for (e) head acceleration, (f) torso acceleration, (g) RMS SCM activity and (h) RMS PARA activity as a function of perturbation acceleration and velocity. The borders encircle homogeneous groups of data and increasing line weight corresponds to earlier times and increasing amplitude.

The correlation between neck muscle activity and head/torso acceleration was poor for onset times (Figure 5), but strong for peak amplitudes (Figure 6). A significant positive correlation coefficient ($r > 0.73$) between the onset of head or torso acceleration with the onset of SCM or PARA muscle activity was observed in fewer than 15 percent of subjects (Table 1). In contrast, the peak amplitude of both torso and head acceleration correlated significantly with peak SCM amplitude in about 90 percent of subjects and with peak PARA amplitude in about 65 percent of subjects (Table 1). The average slopes for the linear regressions between the normalized peak EMG amplitude (SCM/PARA) and normalized peak acceleration (head/torso) (see Figure 6) were not significantly different from each other ($p>0.09$) and only the slope for the relationship between peak PARA amplitude and peak torso acceleration was significantly greater than unity ($p=0.015$).

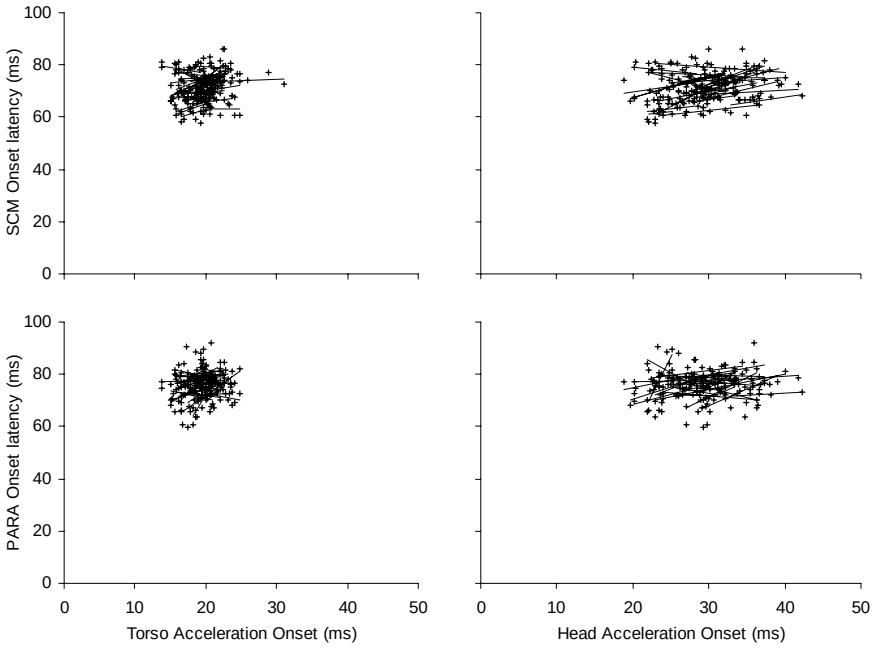

Figure 5: Linear regression analyses demonstrating the absence of a correlation between either SCM (top row) or PARA (bottom row) activation times and the onset of either torso (left column) or head (right column) acceleration. Each straight line represents the least-squares best fit line through an individual subject's data.

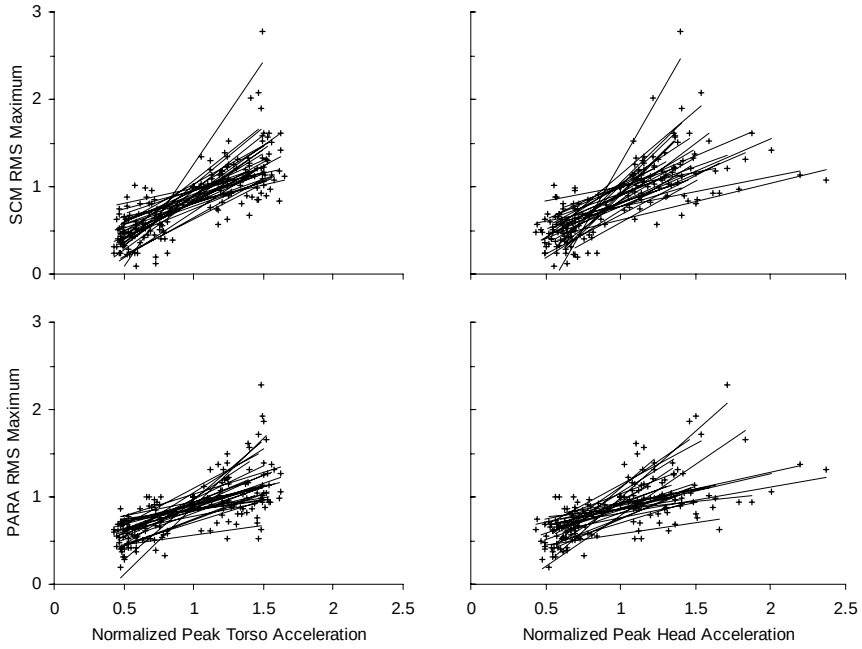

Figure 6: Linear regression analyses demonstrating the presence of a correlation between the amplitude of the neck muscle EMG (SCM, top row; PARA, bottom row) and the amplitude of both the torso (left column) and the head (right column) accelerations. Each straight line represents the least-squares best fit line through an individual subject's data.

Table 1. Mean (SD) of the correlation coefficients and best-fit slopes for the within-subject linear regressions of the onset and amplitude of head and torso acceleration with respect to the onset and peak amplitude of SCM and PARA muscle EMG activity. Also shown is the pooled mean (SD) of the difference between the onset of acceleration and muscle activity for all subjects and perturbations.

Acceleration Signal	Muscle	Difference	Correlation Coefficient	Regression Slope	n / N
Onset latency					
Head	SCM	42 (7)	0.34 (0.35)	0.29 (0.37)	2 / 30
	PARA	43 (14)	0.21 (0.44)	0.12 (0.44)	3 / 28
Torso	SCM	52 (6)	0.32 (0.37)	0.17 (0.23)	4 / 30
	PARA	52 (16)	0.20 (0.41)	0.09 (0.22)	4 / 28
Peak amplitude					
Head	SCM	-	0.89 (0.11)]*	0.97 (0.44)	28 / 30
	PARA	-	0.78 (0.15)]*	1.11 (0.50)	18 / 28
Torso	SCM	-	0.88 (0.09)]*	1.05 (0.39)	27 / 30
	PARA	-	0.79 (0.13)]*	1.22 (0.45)	19 / 28

Within-subject correlation coefficients of $r > 0.73$ were significant at the $p=0.05$ level. n, number of subjects with significant correlation coefficients; N, total number of subjects in each analysis.

DISCUSSION

In order to examine the response of the head/neck system to transient perturbations, seated human subjects were repeatedly and randomly exposed to surge perturbations of different intensities. Based on the analysis of response variables (e.g., onset latency, and peak or RMS amplitude), the results of the current study indicated that the head/neck system responded repeatably over the range of presented stimuli and that both the VCR and CCR play a secondary, rather than a primary, role in neck muscle response to this kind of stimulus.

The mechanoreceptors responsible for triggering neck muscle responses in postural perturbations have been debated in the literature (Woollacott et al, 1988; Keshner et al., 1988; Horak et al., 1994; Forssberg and Hirschfeld, 1994; Allum et al., 1997, 1998). Rotation about the ankles was originally thought to trigger automatic postural responses in perturbed standing (Nashner, 1976), however, Keshner et al. (1988) showed that neck muscle activation occurred at about the same latency as lower limb muscle activation (about 95 ms) and concluded that ankle rotations alone did not adequately explain the neck muscle response. Forssberg and Hirschfeld (1994) subsequently perturbed seated subjects and observed SCM muscle activation at about 77 ms. Using various perturbation directions, they observed different muscle activation patterns despite the presence of similar head kinematics and therefore excluded a major vestibular component to the postural response. Instead, these authors concluded that mechanoreceptors in the pelvis, possibly related to contact with their rigid platform, were likely responsible for triggering the postural response. Bisdorff et al. (1994, 1999) used a whole-body free-fall in supine subjects to evoke large postural responses in the neck muscles at 54 to 60 ms. Based on similar responses in normal and avestibular subjects, these authors concluded that sudden changes in cutaneous pressure and joint forces might be responsible for triggering the neck muscle response. Head-only drop tests in supine subjects have produced short SCM activation times of 22 to 25 ms (Ito et al., 1995, 1997; Bisdorff et al., 1999). Due to an absence of this short latency response in avestibular patients (onset occurred at 67 ms in these patients), these short activation times were attributed to a vestibulocollic reflex (Ito et al., 1995). Rapid release of a load generated by a tonic

neck muscle contraction has also been shown to evoke vestibulocollic reflexes at about 25 ms in the SCM muscles of normal subjects and stretch reflexes at about 41 ms in the SCM muscles of avestibular patients (Corna et al., 1996). Others have also reported stretch reflex latencies in the neck muscles of 50 to 60 ms (Horak et al., 1994; Ito et al., 1995), although the long duration of these stretch reflexes indicated that they were likely not similar to the monosynaptic stretch reflex observed in limb muscles, but more likely the polysynaptic cervicocollic reflex (Peterson, 1988).

Taken together, the results of these previous studies showed that the input of multiple mechanoreceptors converges on the motoneurons of the neck muscles and that all of them could play a role in triggering or modulating a postural response in the neck muscles. In the current study, mean onset times for torso acceleration varied from 17 to 22 ms, mean onset times for head acceleration varied from 26 to 32 ms, and mean onset latencies for the SCM muscles varied from 68 to 74 ms. Therefore SCM activation occurred about 50 ms after the onset of upper torso acceleration and 40 ms after the onset of head acceleration. Based on the previously-reported SCM onset latencies reviewed above, this was sufficient time for both the cervicocollic and vestibulocollic reflexes to mediate the SCM response observed in the current study and a simple timing analysis could not be used to exclude either reflex. The pattern of activation observed over the different perturbations used in the present study, however, provided some additional evidence with which the relative contribution of these reflex pathways could be evaluated further.

Torso acceleration was measured at the manubrium – the origin for the sternal heads of both sternocleidomastoid muscles. Forward acceleration of the manubrium relative to an initially stationary head would stretch the SCM muscle and could provide the necessary afferent input to trigger a cervicocollic reflex. If this was the primary sensory pathway mediating the SCM activation, then a strong correlation between the onset time of torso acceleration and the onset of SCM activation would be expected. A strong correlation, however, was not observed in the current data. This low correlation suggested that cervicocollic reflexes played at most a minor role in the activation of the SCM muscles in the perturbations studied here.

The onset of head acceleration occurred about 40 ms before SCM activation, an interval about 15 to 20 ms longer than that needed for the vestibulocollic reflex to act (Ito et al., 1995; Bisdorff et al., 1999). Upwards acceleration of the head could provide the necessary afferent input to trigger a vestibulocollic reflex. If this was the primary sensory pathway mediating the SCM activation, then a strong correlation between the onset time of head acceleration and the onset of SCM activation would be expected. Like the onset of torso acceleration, however, the onset of head acceleration correlated only weakly with the onset of SCM activation, a result that suggested that the role of the vestibulocollic reflex in triggering SCM activation was small as well.

The apparently minor roles for both the cervicocollic and vestibulocollic reflexes in the SCM activation observed here were consistent with the conclusions reached by Forssberg and Hirschfeld (1994), even though peak linear head accelerations in the current study were 5 to 10 times larger and peak angular head accelerations were 10 to 15 times larger than those observed in this previous study. Despite the presence of considerably larger muscle stretch stimuli and vestibular inputs, these reflex pathways still appeared to play only a minor role in triggering the SCM response. These large differences in head kinematics between the current study and the previous study of seated subjects by Forssberg and Hirschfeld (1994) were likely related to their perturbations pulse, which consisted of a fixed displacement of 80 mm at 0.36 m/s over 240 ms, and the absence of back support for their seated subjects. The perturbations used in the present study were both larger (up to 0.75 m/s), longer (750 mm), and consisted of a single large-amplitude initial acceleration with a much lower amplitude final deceleration. In addition, the presence of a seat back in the current study concentrated the kinematic response to the head and neck. The combined effect of these differences between studies likely explained these large differences in the amplitude of the head kinematics.

Horak et al. (1994) reported that the contribution of the vestibular system to postural control in standing was down-regulated in the presence of good somatosensory cues and up-regulated in their absence. In the current study, the entire posterior aspects of the back, pelvis, and thighs were in contact with the seat cushion and seat back, and would have provided subjects with widespread somatosensory information regarding the onset, duration, and intensity of the perturbing stimulus. Given the apparently minor contribution of the two dominant head-and-neck-based reflexes for neck muscle activation, the trunk

appeared to be the next most likely source of the somatosensory information that triggered SCM activation. Because of compliance in the seat back, the pelvis was likely the first part of the body to be accelerated forward by the perturbation. Spinal conduction velocities of 40 to 50 m/s have been measured in the ascending afferent pathways of the dorsal column of the human spinal cord (Cioni and Meglio, 1986; Halonen et al., 1989). Assuming a distance of 60 to 80 cm between the pelvis and brainstem, the afferent signals in the pelvis would require 12 to 20 ms to ascend the spinal cord. Given SCM onset latencies of about 70 ms, this analysis indicated that there was sufficient time for afferent signals in the pelvis and trunk to mediate the SCM response observed in the current study. Previous evidence of a 10 ms delay from the onset of lumbar paraspinal muscles to the onset of neck muscles in whiplash-like perturbations was consistent with this proposal (Szabo and Welcher, 1996). Therefore, the current findings supported a conclusion that the sensory information triggering a neck muscle response in seated perturbations originated remote from the head and neck and possibly in the pelvis, as predicted by Forssberg and Hirschfeld (1994), or more broadly from the trunk, as postulated by Allum et al. (1998). Whether the diffuse stimulation generated by seat contact excited mechanoreceptors in the skin, joints, muscles, or other tissues could not be discerned from the results of the current study.

It has been postulated that the overall pattern of the response to a postural perturbation is centrally generated and then shaped by additional sensory input and biomechanical demands (Horak et al., 1994; Allum et al., 1998). Despite the poor correlation observed between SCM activation and acceleration onset in the upper torso and head, the amplitude of the SCM activity was strongly correlated to the peak amplitude of the torso acceleration. This peak occurred at about the same time as SCM activation and well before peak SCM activity (102 to 120 ms). Although these strong correlations were not indicative of a causal relationship, they did indicate that the cervicocollic and vestibulocollic reflexes might have played a role in shaping the SCM muscle response.

Due to habituation, the absolute magnitude of the muscle and kinematic responses observed in the current study likely did not accurately reflect the response that would have been evoked in unprepared subjects exposed to only one perturbation (Siegmund, 2001). The onset latencies observed in the current study, however, were similar to those observed in subjects exposed to only one perturbation using the same experimental setup (Siegmund, 2001), and suggested that the same reflex pathway was mediating the SCM response despite habituation of the response magnitude. Therefore, the relative magnitude of the muscle and kinematic responses observed between the different perturbations used in the current experiment likely reflects the gradation in muscle and kinematics responses that would occur in unprepared subjects.

CONCLUSIONS

The temporal pattern of muscle activation and the onset of both torso and head acceleration over the seven perturbations used in this study suggested that neither the vestibulocollic reflex nor the cervicocollic reflex played a major role in activation of the neck muscles. Other somatosensory information, such as a large cutaneous stimulus due to interaction with the seat, or muscle or joints receptors in the pelvis or trunk, are therefore a more likely source of the afferent signal triggering the neck muscle response in these perturbations.

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DISCUSSION

PAPER: **Sensory Pathways Mediating the Neck Muscle Response in Rear-end Impacts**

PRESENTER: ***Gunther Siegmund, MEA Forensic Engineers & Scientists, School of Human Kinetics – University of British Columbia***

QUESTION: *James Funk, BRC*

That's really interesting work, Gunter. I have a question about whether you think, with this set-up, you really have enough information to look at that stretch reflex. And I'm wondering, really from the standpoint of collision severity because not only is there a low-level stimulus to begin with, but I would think it would be hard to pick up on the subtleties of that stimulus because my back-of-the-envelope calculation is that your pulse is over within about 2" of motion of the sled; and so, the peak is probably about ½" or less. And then, you're really measuring acceleration as opposed to actually displacement, which is going to lag that for muscles. Are these hurdles you think you can overcome without going to higher severities?

ANSWER: I don't know yet. You raise valid points. The data set we analyzed was not perfect for this analysis, but we figured we'd start there before we wondered down the site of addressing some of the issues that you're raising. I don't have a good answer for your question.

Q: It's a lot of innovative stuff and some of your other approaches—neat work.

A: Thank you.

Q: *Guy Nusholtz, Daimler Chrysler*

How are you defining onset in the acceleration? The reason for that question sort of comes back to the first question. If you look at acceleration and then you look at the integrals of acceleration, and then you choose a level from the other two—from the two integrals, you may find that there's a relationship because you will say, "Okay. Once you move this certain distance, then we have a higher correlation to response." So the onset may be—You may have data there that you can still milk for more information. Have you thought about that?

A: Yes. You're absolutely right. The whole experiment turns on detecting onset and the algorithm you choose to detect onset will affect the results, both for the accelerometers and the EMG. What we did was choose a very simple algorithm for both because what we didn't want to do was to Mickey-Mouse around with different algorithms until we got a correlation. Because if we didn't see a pattern with a simple onset for both, there wasn't much point in wandering down the path much further.

Q: Well, those are two different numerical methods that you would use for that.

A: Yes.

Q: You can easily do a statistical approach for the EMG and then you have a certain confidence. But then when you're talking about onset, it becomes tricky because you probably want to look at either velocity or displacement and then you want to look at a level as to what you're calling onset. So that would take some investigation to see what makes sense.

A: Absolutely. And we need to match our thresholds to what the stumbles—or the GTOs in the neck—have as a threshold as well.

Q: Thank you.

Q: *Erik Takhounts, NHTSA*

I have a question, Gunter, which sort of expands on Jim's questions a little bit. I was wondering if you could explain, in simple terms, whether the muscle response that you presented is a voluntary or involuntary muscle response. And, how is it going to change with increased severity?

A: Okay. The muscle response is involuntary. It's what we call a postural response. A reflex response: Most of us think of that as a monosynaptic simple-arch stretch reflex. That's the one we think of when we think of the reflex. Reflexes actually consist of short, medium, and long latency responses and it's these long latency responses, which are still shorter than voluntary—or the medium, at least, are still shorter than voluntary, that we're seeing here. Voluntary activation of sternocleidomastoid is in the region of a 110 milliseconds. We're seeing onsets of 60-80 milliseconds so they're not critical.

Q: So there's some kind of extrapolation towards a severe test?

A: Yes.

Q: Okay. Thanks.