Rapid neck muscle adaptation alters the head kinematics of aware and unaware subjects undergoing multiple whiplash-like perturbations

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Accepted 2 December 2002

Abstract

To examine whether habituation confounds the study of whiplash injury using human subjects, we quantified changes in the magnitude and temporal development of the neck muscle electromyogram and peak linear and angular head/torso kinematics of subjects exposed to sequential whiplash-like perturbations. Forty-four seated subjects (23F, 21M) underwent 11 consecutive forward horizontal perturbations (peak sled acceleration = 1.5 g). Electromyographic (EMG) activity was recorded over the sternocleidomastoid (SCM) and cervical paraspinal (PARA) muscles with surface electrodes, and head and torso kinematics were measured using linear and angular accelerometers and a 3D motion analysis system. EMG onset occurred at reflex latencies (67–75 ms in SCM) and did not vary with repeated perturbations. EMG amplitude was significantly attenuated by the second perturbation in PARA muscles and by the third perturbation in SCM muscles. The mean decrement in EMG amplitude between the first trial and the mean of the last five trials was between 41% and 64%. Related kinematic changes ranged from a 21% increase in head extension angle to a 29% decrease in forward acceleration at the forehead, and were also significantly different by the second exposure in some variables. Although a wider range of perturbation intensities and inter-perturbation intervals need to be studied, the significant changes observed in both muscle and kinematic variables by the second perturbation indicated that habituation was a potential confounder of whiplash injury studies using repeated perturbations of human subjects.

Keywords: Whiplash; Habituation; Neck muscle; Reflex; Awareness

1. Introduction

Rapid attenuation of reflex responses to sequentially presented stimuli has been observed in postural reflexes (Nashner, 1976; Keshner et al., 1987; Hansen et al., 1988; Woollacott et al., 1988; Allum et al., 1992; Bisoffi et al., 1994; Timmann and Horak, 1997) and startle reflexes in humans (Landis and Hunt, 1939; Davis, 1984; Brown et al., 1991). This attenuation, called habituation, is a centrally generated process (Harris, 1943) and decrements in electromyographic (EMG) activity of 30–50% have been observed by the second or third stimulus in both postural and startle experiments despite the different sensory systems involved (Bisoffi et al., 1994; Valls-Solé et al., 1997).

The perturbations used to study whiplash injury also evoke reflex muscle responses. In the sternocleidomastoid muscle, for instance, the onset latencies of 73–81 ms observed in whiplash studies (Ono et al., 1997; Magnusson et al., 1999; Brault et al., 2000) are shorter than the voluntary latencies observed during forewarned reaction-time experiments (107 ± 21 ms, Mazzini and Schieppati, 1992; 107 ± 28 ms, Siegmund et al., 2001b). This comparison demonstrates that neck muscles are activated reflexively rather than voluntarily during whiplash perturbations. Evidence of whether these neck muscle reflexes also habituate is less definitive. In one early study, a lower peak head acceleration in the second
exposure of one subject was attributed to a “conditioned muscle reflex…acquired from his initial experience” (Severy et al., 1955). In addition, a comparison between the minimal or absent cervical paraspinous muscle activity observed in experienced subjects (Gutierrez, 1978; Ono et al., 1997) and the presence of this activity in novice subjects (Brault et al., 2000) also suggests habituation may be present. If the reflex muscle responses evoked by repeated whiplash perturbations habituate as rapidly as those in previous startle and postural studies, then the muscle activity and head kinematics evoked by single real-world collision exposures may be different from those reported in whiplash studies using repeated exposures (Mertz and Patrick, 1967; Gutierrez, 1978; McConnell et al., 1993, 1995; Geigl et al., 1994; Matsushita et al., 1994; Szabo et al., 1994; Ono and Kanno, 1996; Szabo and Welcher, 1996; Castro et al., 1997; Ono et al., 1997; Siegmund et al., 1997; Meyer et al., 1998; Davidsson et al., 1998; Pope et al., 1998; van den Kroonenberg et al., 1998; Kaneoka et al., 1999; Magnusson et al., 1999; Brault et al., 2000; Vibert et al., 2001).

Awareness regarding perturbation timing has also been varied between whiplash studies. In some studies, subjects could accurately predict perturbation onset (Mertz and Patrick, 1967; Ono et al., 1997), whereas in many other studies subjects could not. Reduced muscle activity and lower peak head accelerations have been attributed to subject awareness by one group of researchers (Kumar et al., 2000, 2002), though these awareness-related reductions have not been universally observed (Magnusson et al., 1999). Given these disparate findings, it remains unclear if awareness of perturbation timing should be controlled in whiplash studies.

In this study, changes in the amplitude of neck muscle activity between sequential whiplash-like perturbations were quantified for subjects aware and unaware of the perturbation’s exact timing. Concomitant changes in the peak linear and angular acceleration and displacement of the head were also quantified. We hypothesized that the amplitude of the neck muscle activity would decrease with the number of perturbations, and that this habituation would generate changes in the head kinematics consistent with a more flexible neck.

2. Methods

Forty-four subjects (23F, 21M) with no history of whiplash injury, sensorimotor dysfunction, or neck/back pain participated in the experiment. Female subjects were 23 ± 4 years old, 165 ± 6 cm tall and 60 ± 9 kg; male subjects were 28 ± 6 years old, 174 ± 8 cm tall and 78 ± 18 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 with the Declaration of Helsinki.

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-skinly 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 linear accelerometers (Kistler 8302B20S1, Amherst, NY) to measure horizontal (x') and vertical (z') head acceleration and an uniaxial angular rate sensor (ATA Sensors ARS-04E, Albuquerque, NM) to measure flexion and extension velocity were strapped to the forehead immediately above the glabella (Fig. 1). Torso acceleration was measured in some subjects using a similar uniaxial linear accelerometer located 2 cm below the superior margin of the manubrium. Horizontal sled acceleration was measured using a uniaxial 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 3200, 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 (Fig. 1). A force transducer (Artech SS20210, Riverside, CA) was used to measure reaction loads during normalizing contractions of the SCM and PARA muscles. EMG signals were bandpass filtered (10–1000 Hz) and transducer signals were low-pass filtered (1 kHz) before being simultaneously sampled at 2 kHz. Optotrak data were acquired at 200 Hz, which based on power analyses of the accelerometer data, was sufficient to accurately capture the motion.

The front passenger seat (without its head restraint) of a 1991 Honda Accord was mounted to a feedback-controlled linear sled that produced a horizontal (+x) speed change of 0.5 m/s over 60 ms with a peak acceleration of 1.51 g (Fig. 2). The sled generated no pre-perturbation signals—audible or mechanical—from which subjects could predict perturbation onset. Subjects were given about 15 min to adopt a comfortable seated posture before their first perturbation. Prior to testing, subjects were instructed to sit normally, face forward, rest their forearms on their lap and to relax their face and neck muscles.

Each subject underwent 11 perturbations. Half of the subjects (temporally aware subjects) received a countdown for each perturbation; the other half (temporally
unaware subjects) did not. The countdown consisted of two auditory tones (1000 Hz, 80 dB, 40 ms duration) spaced 1 s apart followed 1 s later by a perturbation. Unaware subjects were told to expect randomly spaced perturbations beginning within 60 s of the start of the experiment. Both groups received their first perturbation about 15 s after the start of the experiment. The median time between the first and second perturbations of all subjects was 2.2 min (range 1.2–9.7). The time between the remaining 10 perturbations varied randomly (uniform distribution) between 20 and 30 s. No practice or demonstration perturbations were given.

Prior to testing, seated subjects performed submaximal isometric contractions in flexion and extension from the neutral position to generate normalizing data for the SCM and PARA muscles, respectively (Winter, 1991; De Luca, 1997). A strap attached to the load cell was placed around the head immediately above the glabella for flexion contractions and at the external occipital protuberance for extension contractions. Subjects generated a 50 N force with visual feedback and EMG data were acquired for 5 s for each contraction.

The ability of subjects to reproduce their initial head and torso positions and angles was assessed from the Optotrak data (RMS accuracy of 0.1 mm and 0.1°). The onset of head and torso movement was determined from the accelerometer data and reported relative to perturbation onset. Forehead accelerations were reported in the head-fixed coordinate system ($x'$, $z'$) and mastoid accelerations were reported in the lab reference frame ($X$, $Z$). Retraction, defined as the horizontal rearward translation between the top and bottom of the cervical spine, was estimated using the Optotrak markers on the mastoid and manubrium. Angular rate data were digitally compensated (Laughlin, 1998) and angular acceleration was then computed by finite differences (5 ms window). Head angular displacement was determined from Optotrak markers on the forehead and vertex.

EMG onset was defined as the time when the root-mean-squared (RMS) amplitude (20 ms window) reached 10% of its maximum value, and was then confirmed visually. The RMS amplitude of the each EMG signal was calculated for the interval between EMG onset and peak head extension, and then adjusted for pre-stimulus noise present in the 100 ms preceding the perturbation. The SCM and PARA data were then normalized by the RMS amplitudes calculated from the normalizing contractions.

For each dependent variable, a three-way analysis of variance (ANOVA) was used to assess differences related to awareness (temporally aware, temporally unaware), gender (female, male) and trial number (1–11). Trial number was the only repeated-measures variable. Since no differences were observed between the left and right muscles, the average value for each muscle pair was used for the analysis. All tests were performed using Statistica (v.5.1, Statsoft Inc., Tulsa, OK) and a significance level of $\alpha = 0.05$. Post hoc comparisons amongst the 11 trials were performed using a Tukey’s honest significant difference test. Post hoc testing of interaction terms that included the repeated-measures trial variable was performed using separate two-way ANOVAs for each trial to identify the trials responsible for the interaction effect.

3. Results

Subjects made only minor alterations to their pre-perturbation head and torso posture over the 11 perturbations (Table 1). The torso and head dropped...
an average of 2 and 3 mm respectively by the third trial
and the torso shifted rearward an average of 3 mm by
the sixth trial ($p_{0.0001}$). Torso and head angles varied
less than 1° and no one trial was significantly different
from the first.

All subjects rapidly restored their upright head
position during their first trial; however, not all subjects
did so in subsequent trials (middle panel, Fig. 2). For six
subjects, peak head angle ($\theta$) and retraction ($r_x$) in these
delayed responses could not be identified in all trials
(Fig. 3), and these subjects were thus removed from the
remaining analyses.

Multiple perturbations produced large decrements in
the EMG amplitude but no changes in the onset latency
of both neck muscles (Fig. 2, Table 1). The average
SCM EMG amplitude decreased by 41–54% and the
average PARA EMG amplitude decreased by 48–64%
(Fig. 4). Post hoc analyses of the interaction terms
showed that a gender difference in SCM amplitude
(male $>$ female, $p_{0.036}$) and an awareness difference in

![Fig. 2. Sample data from the first, third and 11th trial of a single subject who exhibited habituation of their muscle response and corresponding changes in their kinematic response. Labeled hollow circles in the first trial represent kinematic peaks used for subsequent analysis. Identically located circles in the other trials highlight changes due to habituation. The vertical scale bars are aligned with perturbation onset and are equal to 1 g, 25 mm, 100 rad/s², 5 rad/s and 10 deg. OO, orbicularis oculi; MAS, masseter; SCM, sternocleidomastoid; PARA, cervical paraspinal, l, left; r, right; $a$, linear acceleration, subscripts $x$ and $z$ refers to the $x$- and $z$-directions; $\alpha$, head angular acceleration; $\omega$, head angular velocity; $\theta$, head angle.](image-url)
Table 1
Mean (SD) of the initial head and torso positions, peak linear and angular kinematics, EMG onset times and normalized RMS magnitude for the first and last five trials

<table>
<thead>
<tr>
<th>Variables</th>
<th>First trial</th>
<th>Average of last five trials</th>
<th>ANOVA results</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unaware</td>
<td>Aware</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>N</td>
<td></td>
<td></td>
<td>Female</td>
</tr>
<tr>
<td>Head</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(x) (mm)</td>
<td>11 (16)</td>
<td>28 (12)</td>
<td>7 (19)</td>
</tr>
<tr>
<td>(z) (mm)</td>
<td>−642 (23)</td>
<td>−661 (36)</td>
<td>−641 (35)</td>
</tr>
<tr>
<td>(\theta) (deg)</td>
<td>7.1 (1.9)</td>
<td>9.4 (4.0)</td>
<td>7.9 (3.4)</td>
</tr>
<tr>
<td>Torso</td>
<td>21 (14)</td>
<td>38 (6)</td>
<td>21 (11)</td>
</tr>
<tr>
<td>(x) (mm)</td>
<td>−552 (19)</td>
<td>−572 (35)</td>
<td>−559 (28)</td>
</tr>
<tr>
<td>(z) (mm)</td>
<td>−159 (4.1)</td>
<td>−153 (3.3)</td>
<td>−176 (4.0)</td>
</tr>
<tr>
<td>(\theta) (deg)</td>
<td>15.9 (4.1)</td>
<td>17.2 (5.3)</td>
<td>17.6 (4.0)</td>
</tr>
<tr>
<td>Linear kinematics</td>
<td></td>
<td></td>
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<tr>
<td>Forehead</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>(x_1) (g)</td>
<td>1.20 (0.22)</td>
<td>1.30 (0.37)</td>
<td>1.11 (0.17)</td>
</tr>
<tr>
<td>(z_1) (g)</td>
<td>−0.60 (0.13)</td>
<td>−0.70 (0.22)</td>
<td>−0.64 (0.21)</td>
</tr>
<tr>
<td>(z_2) (g)</td>
<td>1.63 (0.62)</td>
<td>1.64 (0.25)</td>
<td>1.51 (0.41)</td>
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<tr>
<td>Mastoid</td>
<td></td>
<td></td>
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<tr>
<td>(x_3) (g)</td>
<td>1.35 (0.41)</td>
<td>1.17 (0.27)</td>
<td>1.30 (0.40)</td>
</tr>
<tr>
<td>(z_3) (g)</td>
<td>−0.19 (0.10)</td>
<td>−0.22 (0.08)</td>
<td>−0.19 (0.06)</td>
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<td>(z_4) (g)</td>
<td>0.66 (0.35)</td>
<td>0.55 (0.24)</td>
<td>0.51 (0.19)</td>
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<tr>
<td>Retraction</td>
<td></td>
<td></td>
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<tr>
<td>(r_x) (mm)</td>
<td>−20.4 (3.8)</td>
<td>−20.4 (3.9)</td>
<td>−21.5 (3.1)</td>
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<tr>
<td>Angular kinematics</td>
<td></td>
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<tr>
<td>Head</td>
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<tr>
<td>(x_1) (rad/s²)</td>
<td>75 (23)</td>
<td>68 (26)</td>
<td>79 (31)</td>
</tr>
<tr>
<td>(x_2) (rad/s²)</td>
<td>−145 (53)</td>
<td>−112 (24)</td>
<td>−133 (45)</td>
</tr>
<tr>
<td>(\omega) (rad/s)</td>
<td>3.2 (1.3)</td>
<td>3.1 (0.6)</td>
<td>3.5 (0.9)</td>
</tr>
<tr>
<td>(\theta) (deg)</td>
<td>12 (5)</td>
<td>11 (2)</td>
<td>12 (2)</td>
</tr>
<tr>
<td>Electromyography</td>
<td></td>
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<tr>
<td>Onset (ms)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCM</td>
<td>68 (5)</td>
<td>74 (6)</td>
<td>69 (5)</td>
</tr>
<tr>
<td>PARA</td>
<td>78 (8)</td>
<td>81 (7)</td>
<td>76 (7)</td>
</tr>
<tr>
<td>RMS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCM</td>
<td>0.21 (0.09)</td>
<td>0.29 (0.11)</td>
<td>0.17 (0.04)</td>
</tr>
<tr>
<td>PARA</td>
<td>0.17 (0.09)</td>
<td>0.19 (0.13)</td>
<td>0.12 (0.08)</td>
</tr>
</tbody>
</table>

Note: significantly different at \(*p<0.05; \, **p<0.01; \, ***p<0.001; \, ****p<0.0001.\)

Data also grouped as a function of temporal awareness (unaware, aware) and gender (female, male). F-statistics summarized at right for separate three-way ANOVAs using awareness (A), gender (G) and trial number (T) as independent variables. The 11 trials were considered separately in the ANOVA. Initial positions reported relative to the seat hinge. Kinematic peaks are labeled with hollow circles in the left panel of Fig. 2. \(N\), number of subjects in analysis; SCM, sternocleidomastoid muscle; PARA, cervical paraspinal muscles.
PARA amplitude (unaware > aware, \( p < 0.031 \)) present in the first two trials were not present in subsequent trials. Similarly, an initial gender difference in SCM onset latency (females < males, \( p = 0.001 \)) was no longer significant by the sixth perturbation.

Multiple perturbations also produced large changes in the peak magnitude of the head kinematics, but no changes in the onset times of torso or head acceleration (Fig. 2, Table 1). Compared to the first trial, repeated perturbations resulted in a lower peak horizontal forehead acceleration (\( x_1 \)), horizontal mastoid acceleration (\( x_2 \)), downward mastoid acceleration (\( z_4 \)) and the angular head acceleration in flexion (Fig. 4). In contrast, peak amplitude increased in the vertical forehead acceleration (\( z_1 \)), retraction (\( r_x \)), head angular acceleration in extension (\( z_1 \)), head angular velocity in extension (\( \omega \)) and peak head extension angle (\( \theta \)). A variable pattern, which consisted of increased amplitude by the third trial followed by a return to baseline, was observed in the initial upward acceleration of the mastoid process (\( z_1 \)). Awareness \( \times \) trial interaction terms in the horizontal forehead acceleration (\( x_1 \)) and retraction (\( r_x \)) resulted from slower adaptations in aware subjects than in unaware subjects over the first three trials (Fig. 4).

4. Discussion

Although the rapid habituation of reflex muscle responses has been quantified for repetitive startle stimuli and postural perturbations in standing, the magnitude and temporal development of this habituation have not been quantified for sequential whiplash-like perturbations. The results of this study showed that the neck muscle EMG amplitude decreased significantly by the second or third whiplash-like perturbation and subsequently decreased to about half its initial amplitude. The peak amplitude of some kinematic variables also changed significantly by the second or third perturbation, though these changes were smaller than those observed in the neck muscles. Based on these observations, habituation appears to confound the response of human subjects who are sequentially exposed to whiplash-like perturbations of the magnitude used in this study.

Although many previous whiplash studies repeatedly perturbed their subjects, each of these previous studies used a slightly different protocol (e.g., perturbation timing, magnitude, direction and presentation order). These protocol differences make it difficult to directly apply the current results to all of these previous experiments; however, the magnitude of the habituation observed here suggests that these previous whiplash experiments should be scrutinized for potential problems related to habituation. In particular, low or absent cervical paraspinal muscle activity may be a positive indicator of subject habituation (Gutierrez, 1978; Ono et al., 1997; Vibert et al., 2001). Two previous studies limited subjects to a single exposure (Matsushita et al., 1994; Castro et al., 1997) and another study, in which subjects were tested only twice with at least 1 week between tests, specifically tested for habituation and showed it was absent (Brault et al., 2000). Data from these latter studies may better reflect the muscle, kinematic and kinetic responses relevant to whiplash injury biomechanics.

The large and rapid changes in neck muscle EMG amplitude were consistent with previous reports of head, neck and shoulder muscle habituation using supine free-fall (Bisdorff et al., 1994), acoustic startle (Valls-Solé et al., 1997) and standing postural perturbations (Woollacott et al., 1988; Allum et al., 1992). The slightly larger reduction observed in PARA than SCM was consistent with its antagonist role in the current
perturbation, and was similar to the reduced activity seen in antagonist lower limb muscles during multiple standing perturbations (Woollacott et al., 1988; Horak et al., 1989). Large adaptations, similar to those observed in the six subjects excluded from the current analysis have also been observed in standing subjects and were thought to represent changes in postural strategy (Horak et al., 1989).

The initial co-activation of the SCM and PARA muscles (Fig. 2) appeared to be a protective attempt to stiffen the connection between the torso and head in response to a novel stimulus, and was similar to a “strap down” strategy described by Nashner (1985). As subjects became more comfortable with the perturbation, the co-contraction diminished and neck stiffness was presumably reduced. Larger rearward head displacements (e.g., $r_x$ and $\theta$ in Fig. 2) and smaller peak responses related to restoring upright head posture (e.g., $x_1$ and $z_2$ in Fig. 2) were biomechanically consistent with reduced neck stiffness after habituation. In addition, the delayed kinematic peaks observed in later trials (Fig. 2) suggested a lower natural frequency in the head-neck system—also consistent with reduced neck stiffness after habituation. Although changes in initial posture may have also contributed to these kinematic changes, they were likely too small to be solely responsible.

Fig. 4. Mean (SD) of some normalized dependent variables over the eleven exposures. Subject data were normalized to the peak amplitude or the time of peak amplitude observed in their first trial before the normalized means (SD) depicted in this figure were calculated. Interactions with gender and temporal awareness are shown only where the effect was significant. Hollow markers indicate trials significantly different from the first trial. Number at right indicates the percentage change of the last 5 trials relative to the first trial. F, female; M, male; A, aware; U, unaware.
Unlike habituation, awareness of perturbation timing affected only the rate at which a few variables changed over the first three or four perturbations. This minor role for awareness was more consistent with Magnusson et al. (1999), who reported no response differences between temporally aware and unaware subjects seated in an automobile seat, than with Kumar et al. (2000), who reported differences in both the time and amplitude of peak head acceleration between temporally aware and unaware subjects seated in a low-back, molded-plastic seat. The peak kinematics in this latter study were delayed compared to those reported both here and by Magnusson et al. (1999) and therefore may not be directly applicable to automobile collisions.

A role for reflexively activated neck muscles in whiplash injury has been previously discounted on the premise that substantial levels of muscle force are generated too late to alter injury potential (Foust et al., 1973; Snyder et al., 1975; Panjabi et al., 1998; Yoganandan et al., 1999). The large changes in peak kinematics that occurred with neck muscle habituation in the current study showed that muscles make a measurable contribution to head and neck motion, even for muscles that were relaxed prior to the perturbation and reflexively activated by the perturbation. Moreover, Bogduk and Teasell (2000) have postulated that some facet joint injuries are caused by intervertebral motion, which peaks about 150–200 ms after perturbation onset (Kaneoka et al., 1999). Peak SCM muscle activation in the current study occurred 104 ± 10 ms after perturbation onset, and all of the peak kinematic variables with habituation-related changes occurred within 200 ms of perturbation onset. These findings demonstrate that reflexively activated muscles should be considered in the study of whiplash injury.

In addition to altering head/neck kinematics, muscles may also contribute directly to whiplash injury. Winkelstein et al. (2001) have shown that posterior neck muscles insert directly on the cervical facet joint capsular ligament—a structure that has been linked to whiplash injury in both clinical and biomechanical studies (Lord et al., 1996; Winkelstein et al., 2000; Siegmund et al., 2001a). The larger posterior neck muscle contractions observed during the first perturbation may generate higher strains in the capsular ligament than would develop in habituated subjects. As a result, loads and strains in tissues relevant to whiplash injury may only be valid during a test subject’s first perturbation.

The time interval between perturbations in the current study was less than in most previous whiplash studies and may have hastened habituation. In other work, inter-stimulus intervals of 20 min have produced habituation of the acoustic startle response within 2–6 exposures (Brown et al., 1991). Moreover, repeated exposures to startling acoustic stimuli over sequential days have produced long-term habituation effects (Maschke et al., 2000), though partial recovery has been observed within a week (Foss et al., 1989). In whiplash studies, Brault et al. (2000) reported no test order effect in subjects tested only twice with a minimum 1 week between tests. Although more work is needed to quantify the rate of habituation to perturbations spaced further apart, this latter study suggests that a week between tests may be sufficient to minimize the confounding effect of habituation.

The perturbation amplitude (0.5 m/s, 1.5 g, 60 ms) used in the current study was at the low end of the range used in most previous whiplash experiments (0.14–4.0 m/s, 0.4–7.6 g, 55–270 ms) (Mertz and Patrick, 1967; Matsushita et al., 1994; Magnusson et al., 1999; Vibert et al., 2001). A low intensity was chosen because subjects were exposed to 11 sequential perturbations and did not have head restraint protection. Previously observed gradations of muscle and kinematic responses about both the current intensity (Siegmund et al., 2001) and at higher intensities (Ono et al., 1997; Siegmund et al., 1997; Brault et al., 2000) suggest that the adaptations observed here would also be present at higher perturbation intensities; however, sensitization rather than habituation would presumably occur at some higher intensity. Since only a single perturbation direction was used in the current study, the effect that randomizing perturbation direction has on the magnitude and time course of any muscle habituation remains unclear. Further work is thus needed to quantify habituation using both higher perturbation intensities and random perturbation directions.

In summary, a large decrement in neck muscle activation and large corresponding changes in peak head kinematics occurred in subjects exposed to multiple whiplash-like perturbations. These changes occurred rapidly—by the second trial in some variables—and indicated that habituation was a confounder of whiplash injury studies using human subjects and repeated perturbations. The results of the current study suggest that human subjects should not be exposed to more than one whiplash-like perturbation if a muscle and kinematic response unaffected by habituation is important.

Acknowledgements

This research was partially funded by grants from the Physical Medicine Research Foundation, Natural Sciences and Engineering Research Council of Canada, and the Science Council of British Columbia.

References

surface movement the only factor influencing head stabilization.


