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Cervical electromyographic activity during low-speed rear impact

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Abstract Whiplash motion of the neck is characterized by having an extension-flexion motion of the neck. It has been previously assumed that muscles do not play a role in the injury. Eight healthy males were seated in a car seat mounted on a sled. The sled was accelerated by a spring mechanism. Muscle electromyographic (EMG) activity was measured by wire electrodes in semispinalis capitis, splenius capitis, and levator scapulae. Surface EMG activity was measured over trapezius and sternocleidomastoideus. Wavelet analysis was used to establish the onset of muscle activity with respect to sled movement. Shorter reaction times were found to be as low as 13.2 ms from head acceleration and 65.6 ms from sled acceleration. Thus the muscles could influence the injury pattern. It is of interest that clinical symptoms are often attributed to muscle tendon injuries.

Key words Whiplash · Muscle activity · Electromyography Acceleration

Introduction

Rear-end collisions typically occur in dense traffic at very low speeds. The vehicle is subjected to a forward acceleration during which the occupants are pushed forward by the seatbacks. The head lags behind forcing the neck into extension. This head motion continues until the neck hits the headrest or reaches its maximum range of motion, or is counteracted by the muscles. The head then reacts by moving forward into a flexed neck posture. This is the typically described injurious extension-flexion motion of the neck called "whiplash motion" [12]. Hypertranslation of the head was suggested by Penning as being the primary mechanism of whiplash injury [16]. The translatory motion takes place in the upper cervical spine followed by an angular extension in the lower part, forming as S-shape of the cervical spine. This means that the upper cervical spine is undergoing a flexion motion while the lower cervical spine is undergoing an extension motion. When the upper cervical spine reaches its limit for maximum flexion, the lower cervical spine reaches its limit for full extension. The cervical spine then goes into full extension and stops before the motion is reversed [22].

The hyperextension of the cervical spine results in compressive forces on the posterior structures, such as the

facet joints, and tensile forces on the anterior structures, especially the anterior longitudinal ligament, muscles, and the intervertebral discs. During the flexion phase of the motion, hyperflexion causes compressive forces anteriorly and tensile forces posteriorly. The anterior structures at risk are the intervertebral discs and vertebral bodies, and the posterior structures at risk are the facet joint capsules and the posterior neck muscles. MacNab found in his experiments with monkeys a predominance of anterior element injuries and hypothesized that the hyperextension of the cervical spine caused the injuries [7–9].

Hypertranslation produces horizontal shear between the vertebrae, resulting in compression of the facet joints and stretching of the annular fibers at the anterior part of the disc [2]. McConnell et al. found in their experiments of rear-end collisions that even when the extension angle of the neck did not exceed 45° and the subjects were not exposed to hyperextension, mild but clinically classical neck symptoms were experienced, indicating that injuries occurred even when the neck moved within its normal range of motion [12].

The introduction of head restraints in cars was based on the role of hyperextension as the causative mechanism in whiplash injury, and thus was designed to stop the extension before it reached hyperextension (i.e., beyond the physiological limit). Head restraints have been standard in most cars in Sweden for two decades. The decrease in whiplash injury due to rear impact collisions during this time period was only 20% [14]. The risk of injury to rear seat passengers was only 50% of the risk of injury for front seat passengers in rear-end collisions [6]. This indicates that the prevention of hyperextension by head restraints does not alone solve the problem of reducing the whiplash injury risk. One of the most important findings in whiplash studies was the one by Severy et al., who found that in low-impact rear-end collisions the head and neck were exposed to 2.5 times higher acceleration forces than the vehicle itself, and at higher speeds 4–10 times higher [19]. The head restraint may not only be insufficient in preventing the accelerated extension but also, according to some studies, may even intensify the injury [22]. There are also suggestions that seat belts may increase the incidence of neck injuries, especially when they are not properly used [21].

The role of the muscles has not until recently been studied or been incorporated in models of the mechanics of injury [23]. The basic assumption behind this is that muscles do not play a significant role during the injury-causing phase, as muscle reaction times necessary to develop sufficient muscle forces to brace the spine (100–200 ms) are much longer than the rise times of the loads causing injury [24]. Szabo and Welcher studied the muscle activity of cervical flexors and extensors and the lumbar paraspinal musculature, using surface electromyograph (EMG) electrodes, and found that the muscle reaction time was 100–125 ms after the moment of bumper contact [23]. In our previous studies we found that the reaction time of the lumbar muscles to sudden load was about 100 ms, and when the load was unexpected the response was significantly delayed. However, some of the fastest responses were about 40 ms [1, 10]. It could be expected that the cervical muscles react faster than the lumbar ones due to shorter neurological pathways. In addition, it can be questioned what should be defined as the reaction time in the case of a rear-end impact: the time lag between seat acceleration and EMG response, or the time lag between head acceleration and EMG response. There is a time lag of about 50 ms between detection of vehicle and detection of occupant motion [13]. This is important, as the vehicle reaches its final velocity before the occupant starts to move.

Preliminary results, using surface electrodes unilaterally over the sternocleidomastoid and trapezius muscles, showed contraction times of less than 50 ms with respect to head acceleration [17]. It is hypothesized that the contraction is initiated by the sled impact, and is a "bottom-up" response, consistent with our findings in the lumbar muscle response to sudden loads. In those experiments, a perturbation of the chest led to a reaction that was initiated at the feet [10].

The aim of this study was to further study the reaction time of different muscles in rear-end impacts using both surface and wire EMG electrodes. The muscles chosen for study were representative muscles of different levels (the sternocleidomastoideus, trapezius, levator scapulae, splenius capitis, and semispinalis capitis).

Hypotheses:

1. The cervical muscles do have an influence in the mechanism of "whiplash injury".
2. The motor control recruitment strategy is different for expected and unexpected acceleration.
3. Muscles at different depths will respond with different reaction times.

Materials and methods

Eight male subjects with no history of whiplash or other neck problems were tested. Their average age was 40.4 years with a range of 24–56 years. They all gave full informed consent before participating. The protocol was approved by the Human Ethics Committee.

The subjects were seated on a car seat mounted on a sled. The legs and feet were positioned as in a driving position, no steering wheel was used and hands were resting in the lap (Fig. 1). Neither a seat belt nor head restraint was used. A head restraint was present on the seat, but the accelerations were never sufficient to cause any subject's head to contact it. The seat was propelled forward by a spring under tension, much like an arrow propelled by a bowstring. The spring was attached to the front of the sled. A gate latch was attached to the back of the sled, serving as a release mechanism for the sled. This posterior-anterior gate latch and spring construction allowed for rear collision simulation. The tension of the spring was used to provide acceleration and was set not to exceed 0.5 g (4.9 m/s^2). Pre-trial settings for spring tension were made for each

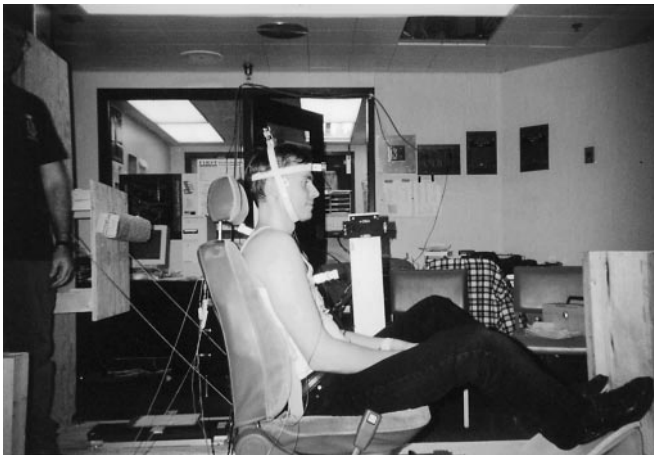

Fig. 1 The experimental set-up with a subject in a car seat on the sled. A head restraint was present but subjects never contracted it. Selspot markers and accelerometers parallel to the direction of motion were located on the sled, chest, and head

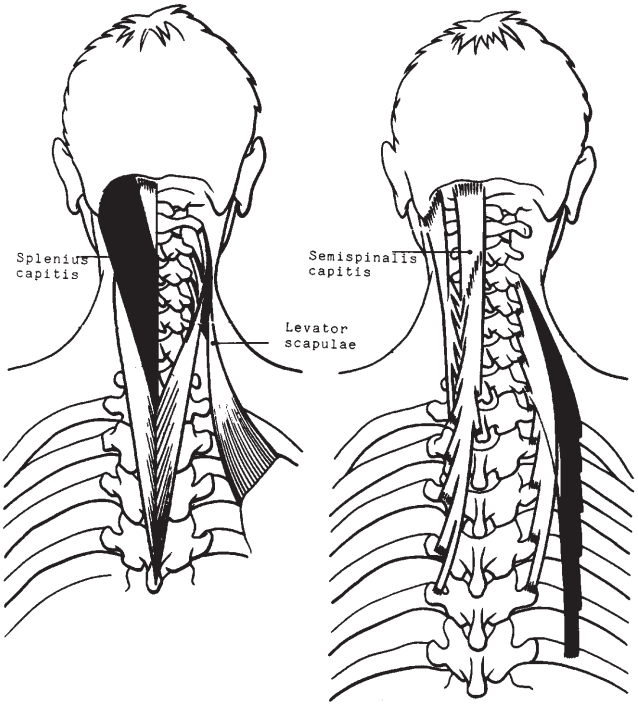

Fig. 2 Wire electrodes were placed in splenius capitis, semispinalis capitis, and levator scapulae at the C3 level

subject, and these settings were duplicated for each trial for that subject. This ensured reproducibility between trials for spring tension.

Wire EMG placement was determined using X, Y, Z dimensions from MRI scans for accurately placing the electrodes into the levator scapulae, splenius capitis, and semispinalis capitis muscles. With C3 as reference, the lateral and sagittal dimensions for each muscle was determined. Thus, the wires were placed as follows: 21 mm lateral C3 at 34 mm depth (semispinalis capitis), 26 mm lateral C3 at 27 mm depth (splenius capitis), and posterior of the sternocleidomastoid muscle at 18 mm depth (levator scapulae) (Fig. 2). As the wire electrodes were placed, the fascial sheats were appreciated by the clinician as he went from one level to another. These locations were verified in cadaver studies, in which the same dimensions were used to place markers in the muscles, after which dissection was done and the positions confirmed. Surface EMG electrodes were placed over the trapezius upper portion and the sternocleidomastoideus. Thus, four extensor muscles and one flexor muscle were studied.

The deep neck muscle activity was monitored using woven platinum wire electrode pairs. Wire electrodes were implanted by a neurologist after local anesthesia and sterilization of the insertion areas. The output ends of the wire electrodes were connected to the Therapeutics Unlimited (Iowa City, Iowa) EMG amplifier system. Acceleration data were monitored using piezoelectric accelerometers made by Endevco (San Juan Capistrano, Calif.). Accelerometers were oriented parallel to the anticipated vector of travel on the base of the sled, on the head just anterior to the ear, and on the mid-sternum, for the determination of acceleration between sled, upper body, and head. A two camera Selspot system (Qualysis Inc., Gothenburg) with three markers placed on each of the sled, chest, and the head was used to calculate the angular and translatory displacements and velocities between sled, body, and head.

The subjects were exposed to rear-end impacts, applied expectedly and "unexpectedly". In the expected case a countdown was done to notify the subject. They were free to prepare any way they liked. In the "unexpected" case the impacts were applied at irregular times, the subjects' eyes were closed and earplugs were used to hide any clue of when the impact was to come. Thus, the subjects were not completely surprised in the "unexpected" case. They knew an impact would come but not when. This is also often the case in real life situations for low-impact collisions. Even "unexpected" cases, may not be totally unexpected, due to visual or audio cues and images in mirrors before impact. Two impacts in each condition were made.

Data acquisition

Data from eight channels (five EMG, three accelerometers) were acquired at a rate of 1000 Hz per channel for a period of 4 s. Data acquisition started at least 1 s before stimulus in order to record the system's resting conditions. The Asyst (Keithley Metrabite/Asyst/dac, Taunton, Mass.) data acquisition software was used to acquire and view the data prior to storage in an Ascii format. Data were acquired using a Data Translation (Marlboro, Mass.) model 2821 data acquisition board installed in a Gateway 2000, 66 mHz computer with a 486 central processor (Gateway, North Sioux City, S.D.).

Wavelet analysis

The EMG signals were analyzed using wavelet techniques [3]. Wavelet transform (WT) is a relatively new mathematical technique that has been applied in analyzing complex non-stationary signals in other fields. The technique provides the best resolution in both time and frequency domains. This is particularly useful in the analysis of EMG signals obtained during dynamic conditions in which the characteristics of the signals are of unequal scales. Wavelet can separate a signal into different frequency components and handle each component with a resolution matched to its scale, known as the multi-resolution analysis (MRA). The main difference between WT and the traditionally used Fourier Transform methods is that WT decomposes the signal into both time and frequency domains. In this study, the EMG signals were first decomposed by WT. The MRA decomposition provided levels of EMG signals from high-frequency to low-frequency components. Move-

ment artifacts have different charcteristics in the time-frequency domain than EMG signals and can therefore be ruled out. A proper thresholding technique was applied to each level of decomposed signal to eliminate unnecessary noise signal components and movement artifacts. The threshold parameter was set to select only 2.5% of the WT parameters in MRA decomposition in order to eliminate high-frequency noise and low-frequency movement artifacts. This filtered transform parameter was used to reconstruct a clearer signal and to provide better temporal information.

Statistical analysis

Muscle reaction times from five sites were gathered twice (two trials) for each subject with respect to motion beginning at three separate locations (sled, torso, head), in two conditions (expected and unexpected). Thus, since each subject experienced all levels of each factor, the experiment was conceptualized as a 3wLocation × 5wMuscle × 2wExpectancy × 2wTrial factorial design. In addition, an overall measure of acceleration onset for each trial from two locations (head, torso) were analyzed as a 2wLocation × 2wExpectancy × 2wTrial factorial experiment.

Initial analyses were conducted to determine the nature and magnitude of the repeated trials effect. If the trial effect was not significant and if the internal consistency estimates (Cronbach's alpha) were not particularly high, it was planned to average the scores from the two trials to increase reliability of the measures and to then drop the trials factor from the design.

All outcomes were evaluated using the general linear models (GLM) procedure in the SAS System version 6.12 running under OS/2 version 4.0. Nominal type I error rates (α) was set at 0.05, and contrasts to investigate anticipated interactions among the expectancy, muscle group, and location factors were planned to control family-wise type I error rates by using Tukey's adjustment procedures.

Results

Reaction times of all muscles and motions of trunk and head were referred to the onset of sled acceleration. Chest and head angular motion was computed from the Selspot and accelerometer data. As there was no significant difference between trials, the scores from the two trials were averaged. Figure 3 shows an example of the acceleration of the sled, trunk, and head with its typical pattern of sequence. The average time lag between sled acceleration and trunk acceleration was 18.6 ms (SD 9.8), and between sled and head acceleration 52.9 ms (SD 14.0). There was a significant and large difference between trunk and head accelerations, consistent over all subjects ($P < 0.001$). The acceleration of the head was of the order of twice the magnitude of that of the sled. Due to the time lag, the peak acceleration of the head was reached when the sled had reached peak acceleration in the opposite direction. This diverse direction of acceleration led to an increased second acceleration peak of the trunk and head (Fig. 3). The trunk and head continued to oscillate at approximately 2 Hz for roughly 1 s after the sled acceleration had fully ceased. There was no significant difference of reaction time between expected and unexpected acceleration of the torso and head.

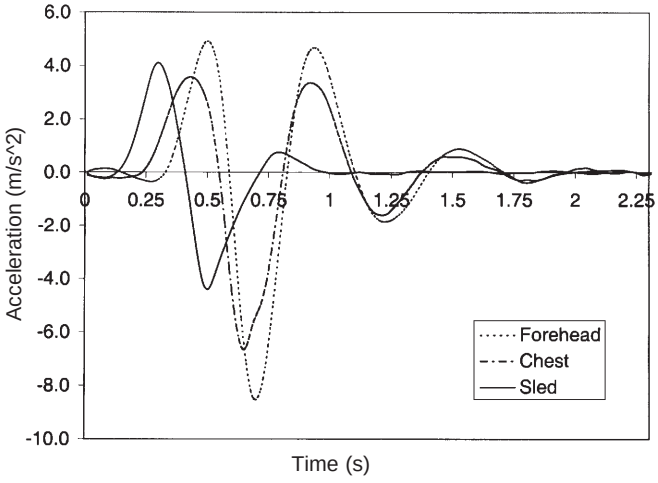

Fig. 3 *Typical* accelerations of the sled, trunk (chest), and head

The angular motion in the sagittal plane was analyzed with respect to sled acceleration. The head and trunk began to move about the time when the sled reached peak acceleration and continued until the sled reached its second acceleration peak (bouncing). The angular motion of the trunk and head is shown in Fig. 4.

The average muscle response time, for both expected and unexpected impacts, from *sled* acceleration to onset of EMG activity of all muscles was 112.4 ms (SD 58.4), from *trunk* acceleration to EMG muscle activity 93.6 ms (SD 58.9), and from *head* acceleration to EMG onset 59.1 ms (SD 60.5) ($P < 0.05$). The sequence of events of accelerations and general muscle activity is illustrated in Fig. 5. These onset times were significantly different ($P < 0.05$). Reaction times for all muscles in the expected and unexpected conditions are given in Table 1. There was a significant difference in reaction time between muscles ($P < 0.001$). Reaction times for each muscle with respect to sled, trunk, and head acceleration are summarized in Tables 2–4. There was no overall significant difference between expected and unexpected impacts, except for splenius capitis ($P = 0.0001$) and a tendency ($P = 0.0706$) for semispinalis capitis. The average muscle response time of each muscle can be seen in Fig. 6. The first responses occurred in the levator scapulae, sternocleidomastoid, and trapezius muscles at average response times of 73.2 ms (SD 15.2), 73.3 ms (SD 14.7), and 83.0 ms (SD 22.4) respectively. There were no significant differences between the reaction times of these muscles. The reaction times of EMG activity of the semispinalis and the splenius muscles were significantly longer ($P < 0.05$), at 169.4 ms (SD 39.6) and 174.9 ms (SD 67.1) respectively. Reaction times computed from head acceleration were thus as low as between 13.2 ms and 22 ms for the fastest muscles, i.e., fast enough for the muscles to be active before the head had reached peak acceleration at about 50 ms (Fig. 7).

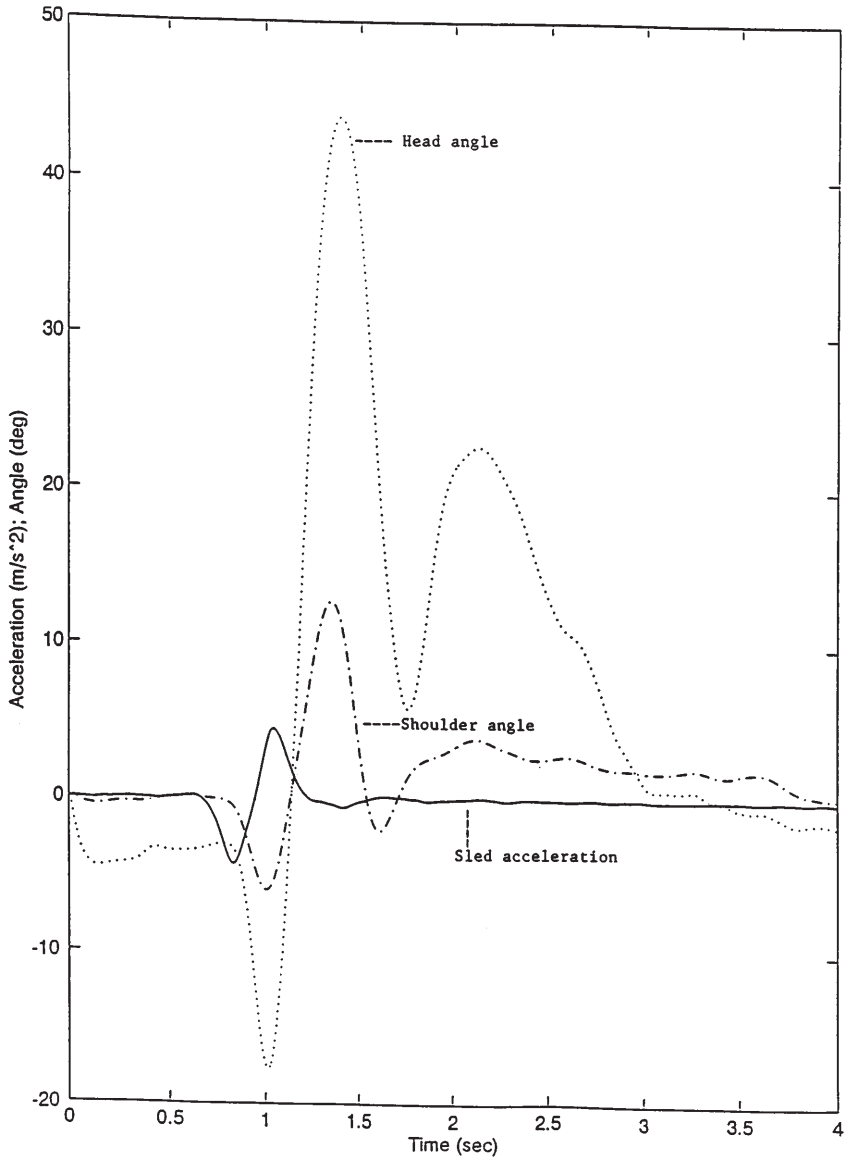

Fig. 4 The acceleration of the sled (——) and angular positions of the trunk (– · – ·) and head (·····) with respect to time

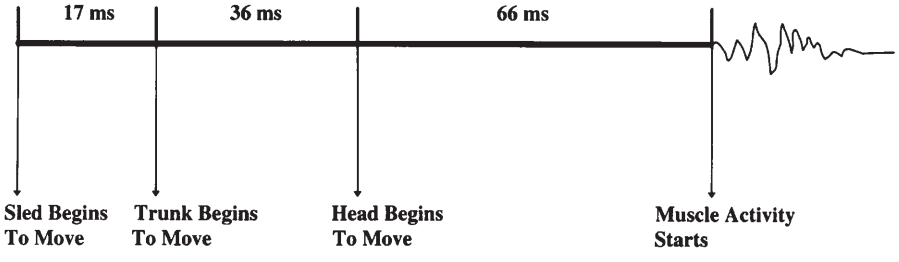

Fig. 5 The sequence and time lag (ms) of regions beginning to move and muscle activity in the expected case

The EMG peak amplitude was on average 4.8 mV, with no difference between expectancies. There was a significant difference between muscles, the highest amplitude occurring in the semispinalis muscle with a peak of 7.9 mV (SD 3.0), which differed significantly from the lowest peak amplitude of 1.8 (SD 1.9) found in trapezius ($P < 0.05$). This, however, must be considered with cau-

Table 1 Average (SD) muscle reaction time (all muscles) from sled, trunk, and head acceleration (ms)

	Sled	Trunk	Head
Average	112.4 (58.4)	93.6 (58.9)	59.7 (60.5)
Expected	118.9 (67.1)	102.4 (66.7)	66.5 (69.7)
Unexpected	105.9 (47.7)	84.8 (48.7)	52.9 (49.1)

Table 2 Muscle reaction time (SD) from sled acceleration (ms) (*st. cleido* sternocleidomastoideus, *lev. scap* levator scapulae, *trap* trapezius, *spl. cap* splenius capitis, *sem. cap* semispinalis capitis)

	St. cleido	Lev. scap	Trap	Spl. cap	Sem. cap
Average	73.3 (14.7)	73.2 (15.8)	83.0 (22.4)	174.9 (67.1)	169.4 (39.6)
Expected	74.4 (17.2)	65.6 (13.2)	90.5 (27.1)	201.2 (67.9)	178.3 (47.9)
Unexpected	72.2 (12.2)	80.8 (13.4)	75.4 (13.5)	148.5 (57.2)	160.6 (28.1)

Table 3 Muscle reaction time (SD) from trunk acceleration (ms)

	St. cleido	Lev. scap	Trap	Spl. cap	Sem. cap
Average	54.7 (16.8)	54.6 (16.7)	64.3 (23.3)	154.9 (68.2)	150.8 (41.2)
Expected	58.1 (18.8)	49.3 (16.0)	74.2 (25.2)	183.4 (66.2)	161.9 (48.4)
Unexpected	51.3 (14.2)	59.9 (16.1)	54.5 (16.7)	126.4 (59.6)	139.6 (30.1)

Table 4 Muscle reaction time (SD) from head acceleration (ms)

	St. cleido	Lev. scap	Trap	Spl. cap	Sem. Cap
Average	20.4 (19.6)	20.34 (22.9)	30.1 (26.7)	122.8 (69.4)	116.5 (42.8)
Expected	22.0 (22.8)	13.2 (20.9)	38.1 (31.5)	148.8 (72.0)	125.8 (54.0)
Unexpected	18.9 (16.4)	27.5 (21.7)	22.1 (18.5)	96.8 (58.3)	107.3 (26.1)

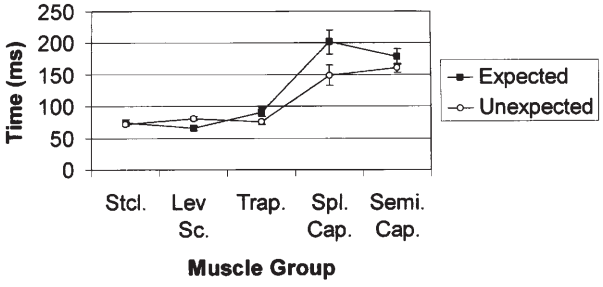

Fig. 6 The reaction times of each muscle with respect to sled acceleration

tion, because of the difference between surface and wire electrodes, placement of wire electrodes, and impedance.

The duration from onset of muscle activity to peak activity was 128.3 ms (SD 134.8), with no difference between expectancies. There was a significant difference between muscles ($P < 0.005$), with the longest duration in trapezius, at 183.6 ms (SD 212.1), and the shortest in the sternocleidomastoid muscle, at 42.8 ms (SD 29.9) ($P < 0.05$).

There was no significant difference in the number of peaks with regard to expectancy, but there was between muscles ($P < 0.0001$). The duration of total muscle activ-

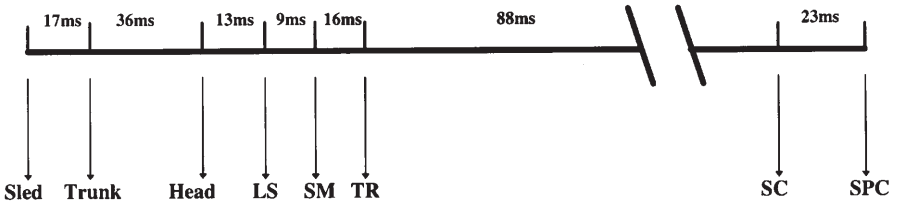

Fig. 7 The typical pattern of motion and muscular activity events (*LS* levator scapulae, *SM* sternocleidomastoid, *TR* trapezius, *SC* semispinalis capitis, *SPC* splenius capitis)

Table 5 Peak amplitude (SD) of the first muscle response burst (mV)

	St. cleido	Lev. scap	Trap	Spl. cap	Sem. cap
Average	3.6 (2.6)	5.2 (3.8)	1.8 (1.9)	7.9 (3.0)	6.0 (2.9)
Expected	3.2 (2.8)	5.2 (3.7)	1.0 (0.8)	8.0 (3.0)	5.7 (2.5)
Unexpected	4.0 (2.3)	5.3 (3.9)	2.5 (2.3)	7.8 (3.0)	6.2 (3.4)

Table 6 Duration (SD) from onset to peak of first EMG burst (ms)

	St. cleido	Lev. scap	Trap	Spl. cap	Sem. cap
Average	42.8 (29.9)	134.8 (114.4)	183.6 (212.1)	161.7 (111.6)	127.2 (90.5)
Expected	44.1 (38.1)	149.8 (114.8)	161.1 (212.6)	148.6 (121.1)	130.1 (72.2)
Unexpected	41.4 (19.9)	119.9 (115.8)	206.1 (216.1)	174.8 (104.9)	124.3 (108.1)

Table 7 Number of peaks (SD) during the duration of muscle activity

	St. cleido	Lev. scap	Trap	Spl. cap	Sem. cap
Average	1.8 (0.7)	3.9 (1.5)	2.1 (0.9)	4.1 (1.9)	5.2 (1.2)
Expected	1.8 (0.8)	3.8 (1.5)	1.9 (0.8)	3.8 (1.9)	5.4 (1.0)
Unexpected	1.9 (0.6)	4.0 (1.5)	2.4 (1.0)	4.5 (1.9)	4.9 (1.4)

Table 8 Duration of muscle activity (SD) (ms)

	St. cleido	Lev. scap	Trap	Spl. cap	Sem. cap
Average	350.0 (226.1)	839.7 (280.5)	511.2 (362.2)	798.7 (390.7)	996.0 (290.0)
Expected	312.6 (226.8)	837.8 (278.5)	358.3 (137.5)	756.0 (371.8)	1049.0 (272.9)
Unexpected	387.4 (226.4)	841.7 (291.6)	664.1 (449.8)	841.3 (420.7)	943.0 (305.5)

ity, i.e., from start to end of trial, was on average 693.9 ms. There was a significant difference in duration with respect to expectancy ($P < 0.04$). The duration varied from 350 ms to 996 ms in the different muscles ($P = 0.0001$).

Discussion

Only a few studies have directly measured EMG activity during cervical spine acceleration associated with low-speed rear-end impacts. EMG activity was recorded in sled tests with simulated rear-end impacts in the studies of Matsushita et al. and Ono and Kanno; however, in neither study were EMG results presented [11, 15]. Reflex times were defined as the time lag between the onset of head acceleration and a distinct increase in muscle activity in response to sudden unexpected stimulus in the sagittal direction of the head [20]. Average reflex time for the sternocleidomastoid muscle was 77 ms and for splenius and semispinalis capiti muscles 66 ms. The study also reported the muscle activation time (i.e., the time from onset of muscle activity to the time of peak head deceleration) for the respective muscles as 61 and 69 ms. In a similar study the reflex time was approximately 90 ms for an unexpected stimulus [18]. The important difference between these studies and the one presented here may lie in the different application of stimuli or impact. In our study the impacts were caused by the acceleration of the sled, from a rear-end impact, which generated a centrally triggered response, whereas in their studies the stimulus was directly applied to the head. The reaction times for the sternocleidomastoid muscle as measured from the sled (73 ms) is very close to the 77 ms that Snyder et al. found during rearward head acceleration, but shorter than the 90 ms in Reid's study. The large difference between the reaction times of the splenius and semispinalis muscles in Snyder's and those in this study is also explained by the difference in impact applications. In testing these muscles, Snyder applied the stimulus in the forward direction of the head, whereas in our study this direction was represented as the recoil, occurring after the extension acceleration.

In our study, there was a significant difference between the first responding "muscle group" and a second "muscle group". It is possible that the reaction times are related to the location of muscles with respect to the spinal axis. Muscles with larger moment arms had shorter reaction times. Since muscles with the larger moment arms are more effective in stabilizing the spine, shorter reaction times for these muscles are beneficial for the subject. Interestingly, the slower muscle group responded with more muscle bursts than the faster group. These muscles are the deep muscles located very close to the spine, and mainly provide fine movements and adjustments. One can speculate that when the larger muscles acting effectively with longer moment arms have repositioned the spine, the deeper muscles continue to adjust and stabilize.

A central response, involving the central nervous system, as opposed to a stretch reflex, was suggested in a study monitoring the activity of lumbar extensors and neck extensors and flexors [4]. Later, Szabo and Welcher presented a study that supported the idea of a central response, and concluded that muscle activities in different parts of the body occurred at approximately the same time and were not dependent on acceleration or movement of that area of the body [23]. It was suggested that the trigger mechanism for the centrally generated response could come from three sources: the somato-sensory system, the vestibular system, and the vision [5]. In our study, we did not find a difference in the expected impacts and the unexpected ones (i.e., excluding the vestibular and visual systems) and believe the trigger in this methodology of impact application was of a somato-sensory feature, when the backrest hit the back. Szabo and Welcher reported latency times of neck flexors and extensors as low as 20–30 ms from head acceleration, and inferred that the stimulus occurred before the onset of head acceleration, triggered by the lumbar spine acceleration that occurred 90–120 ms before the onset of muscle activity [23]. This is in excellent agreement with our results of 20 ms (13 ms in the expected case) from head acceleration and 73 ms from sled acceleration. The centrally triggered response generated by a somato-sensory feature may explain the finding of no difference between the expected and unexpected impacts.

In whiplash, due to a rear-end collision, the vehicle is subject to a sudden forward acceleration causing the back of the seat to push the torso forward and the neck to (hyper) extend. It can be argued that the set-up for these experiments was not ideally realistic, in that the sled acceleration continued in a deceleration, indicating a simulation of a rear-end followed by front-end impact. However, in the very low-impact conditions reported here, we have shown that the muscles fire as quickly as 13 ms after head acceleration. Although there is an additional latency before the muscle reaches peak force, it is self-evident that the muscles will influence the injury pattern (Hypothesis 1). In fact, the flexor muscle (sternocleidomastoid) reached peak magnitude fast enough to be within the time (52.9 ms) of head acceleration. It is noteworthy that clinically symptoms are often attributed to muscle tendon injuries. It can be speculated that these injuries occur from negative or eccentric muscle contractions due to the lag between motion and peak muscle activity. Thus, the muscle involvement can as well be a disadvantage in the whiplash injury mechanism. There were no differences between expected and unexpected conditions (Hypothesis 2). This speaks against the voluntary nature of the contraction. In a previous study, however, it was shown that angular movement of the head decreased if the subject was told to brace their shoulder and neck muscles before the impact was applied. There were some differences in firing between the muscles (Hypothesis 3), with splenius capitis and semispinalis firing last. Future work will concentrate on attempting to model these muscles contractions to ascertain how they affect the force distribution in the neck.

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