



Review Article

Muscle activity during low-speed rear impact

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ABSTRACT

Purpose: Whiplash associated disorders remain a major health problem in terms of impact on health care and on societal costs. Aetiology remains controversial including the old supposition that the cervical muscles do not play a significant role. This study examined the muscle activity from relevant muscles during rear-end impacts in an effort to gauge their influence on the aetiology of whiplash associated disorders.

Methods: Volunteers were subjected to a sub-injury level of rear impact. Surface electromyography (EMG) was used to record cervical muscle activity before, during and after impact. Muscle response time and EMG signal amplitude were analysed. Head, pelvis, and T1 acceleration data were recorded.

Results: The activities of the cervical muscles were found to be significant. The sternocleidomastoideus, trapezius and erector spinae were activated on average 59 ms, 73 ms and 84 ms after the impact stimulus, respectively, prior to peak head acceleration (113 ms).

Conclusion: The cervical muscles reacted prior to peak head acceleration, thus in time to influence whiplash biomechanics and possibly injury mechanisms. It is recommended therefore, that muscular influences be incorporated into the development of the new rear-impact crash test dummy in order to make the dummy as biofidelic as possible.

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Introduction

The purpose of this study was to question the old supposition that the cervical muscles do not play a significant role in the development of Whiplash associated disorders (WAD).¹ Past research has focused mainly on the kinematics and acceleration parameters governing whiplash motion, neglecting the role of cervical muscles in models describing the mechanism of injury.¹ Little attention had been focused on the cervical muscles as a possible injury site until it was discovered that the time delay between the onset of muscular activity and the generation of force, electromechanical delay (EMD) was far shorter than was previously believed.² Following this, interest in how the cervical muscles could possibly contribute to the mechanism of whiplash injury was reignited. Subsequent research revealed that the cervical spine muscles did indeed react fast enough to have an influence on head

and neck motion.^{3,4} Further, muscle response times recorded from human volunteers revealed that cervical muscle activity was not dependent on movement of the head and neck, and it was hypothesised that muscle activity was triggered by a centrally generated response originating from the lumbar region.^{3–5} Other somatosensory, vestibular and visual stimuli may have contributed to the overall muscle activity. This finding was additionally supported by Magnusson et al.³ because no significant differences in muscle reaction times were found between expected and unexpected impact conditions, which eliminated vestibular and vision as trigger mechanisms.

Although there has been a marked increase in the amount of research employed to investigate muscular involvement during the phases of whiplash motion, Magnusson et al.³ were the first to propose how injury to cervical musculature might occur. They speculated that injury might occur due to negative or eccentric muscle contraction. Muscle physiology has established that for a given strain, an increased level of muscle activation results in greater muscle damage, the severity of which can increase with impact severity.^{6,7} Hence, it was concluded that muscle activity

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during whiplash motion is likely to cause injurious muscular effects.^{8–10} The aim of the present study was to gauge the extent of cervical muscle involvement under realistic impact conditions.

Methods

Ten male volunteers aged between 20 and 29 years (mean 26.5 year, SD 3.34 years) participated in the study. Their body weight ranged from 60 kg to 89 kg (mean 77.5 kg, SD 9.51 kg) and their height from 168 cm to 188 cm (mean 179 cm, SD 5.93 cm). The subjects underwent a medical examination before and after the tests. This included full painless range of motion throughout the spine, reflexes, muscle power, blood pressure, and cardiovascular examination. The male volunteers were recruited from the pool of volunteers on file at the Transport Research Laboratory (TRL). The test protocol was accepted by the North West Surrey Ethics Committee. The subjects gave informed consent prior to the experiment.

The tests were conducted using the Dynamic Restraint Test Facility (DRTF) at the TRL in Berkshire in England. The DRTF has been utilised in many of tests of hybrid impact dummies. The DRTF is a sled mechanism, which allowed a bullet trolley to impact into a target trolley of similar mass (688 kg) at slow speed. Both trolleys were able to move freely along a pair of parallel guide rails. The subjects were seated in a rigid seat mounted on the target trolley (Fig. 1). The seat used was based on the UN/ECE Regulation No. 44 (1998) seat.¹¹ The seatback cushion was made from a 70 mm thick layer of closed cell polyethylene foam of density 30 kg/m³. For safety reasons, the seat was fitted with a head restraint to prevent hyperextension of the neck. The impacts were expected both to the event and time, i.e. the subjects knew exactly from a pre-test as well as a countdown what and when it was going to happen, thus they could prepare in any way they wanted.

The impact velocity used in the test programme was 7 km/h, i.e. the target trolley acquired a velocity 2.2 m/s from rest. The impact duration was at least 120 ms, which implies a mean acceleration for the target trolley of 2 g.

An EMG system (Bagnoli-8, Delsys Inc., USA), which had eight active double differential surface electrodes was used for recording the muscle activity during the 7 km/h test. The system used a bandpass filter of 20–450 Hz and the signals were sampled at a rate of 1024 Hz for 120 s or until the test was completed. The gain was set to a factor of 1 K or 10 K, which are nominal gains for typical and faint surface EMG signals ranging from 1 mV to 5 mV and from $\pm 100 \mu\text{V}$ and $\pm 500 \mu\text{V}$ respectively.

A Signal Acquisition and Analysis Software (EMGworks®, Delsys Inc., USA) was used to acquire and analyse all the EMG data. The software could display the raw EMG signal with the root mean square, power spectrum, and median frequency in real time. Each channel of the EMG signal was viewed separately and segments of data were magnified and exported to Excel for further analysis. Pre- and post-baseline amplitude was taken as the average amplitude recorded five seconds prior and post impact. Peak signal amplitude was used as an indication of muscle activity between right and left sides and individual muscles. The sternocleidomastoideus (SCM), erector spinae (ES), i.e. semispinalis capitis and upper trapezius (TR) muscles were located and electrodes were placed on mid-belly of SCM, mid-cervical (C4) spine and on the upper part of trapezius. These positions were chosen as they had been successfully used in a previous study.³ The electrodes were positioned bilaterally and perpendicular to the direction of the muscle fibres and a reference electrode was placed on the back of each volunteer's hand. A

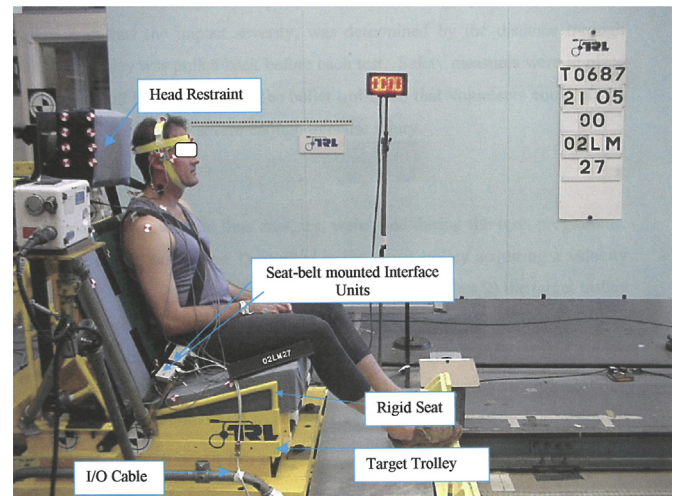


Fig. 1. Subject seated in the test seat on the target trolley.

series of pre-impact tests were performed to check signal viability. Each volunteer was instructed to perform a number of neck extension, flexion and shoulder raise exercises to elicit muscle activity in each muscle under investigation. The signals from each muscle group were examined for noise or presence of any other interfering artefacts. The electrode, which was placed on the hand, was sensitive to motion and was used as a trigger to record the exact time of impact.

Linear horizontal and vertical head accelerations were recorded by pairs of small piezoresistive accelerometers (Endevco 7264B, Meggitt Inc., USA) attached to either side of the volunteer's head, located as close as possible to the centre of gravity of the subject's head. The sensing axes of the accelerometers were parallel to and perpendicular to the Frankfort plane and these axes defined the local x and z-axes respectively for each volunteer. A biaxial arrangement of accelerometers was mounted at the T1 location with sensing direction normal and tangential to the surface of the volunteer's skin at T1. The initial angle of orientation of the accelerometers was measured relative to the vertical before each test. These accelerometers recorded acceleration in the x-z- direction. Another accelerometer was mounted at the base of the spine above the bony protrusion of the sacrum. This accelerometer recorded acceleration in the fore/aft x-direction. The recorded signals were processed in accordance with Society of Automotive Engineers standards (SAE J211c). Thus, head and pelvis accelerations were filtered at CFC 1000 while the T1 accelerations were filtered at channel frequency class filter (CFC 180). The displacements were recorded on the volunteers by use of visual markers and a high-speed camera. The mean and standard deviation of the displacements were calculated.

The following data were analysed and compared by paired *t*-test with level of significance set at $p = 0.05$.

- Time to onset of muscular activity was established by inspecting the EMG signals and identifying a distinct rise in muscle signal amplitude after impact.
- Time to peak muscle activity was defined as the time to maximum signal amplitude.
- Pre- and post-baseline amplitudes were defined as the average amplitude (mV) two seconds before and after impact.

Results

Peak head acceleration was reached approximately 113 ms after the impact (Fig. 2). The onset times of head, T1 and pelvic accelerations from the onset of sled acceleration were significantly different. The onset of both head and pelvic acceleration occurred significantly before the onset of T1 acceleration with $p < 0.05$ (Table 1).

The mean time to onset of muscular activity for the three muscles relative to the mean resultant head acceleration is shown in Fig. 3. As can be seen, all three showed the relative timing between peak muscle activity and the mean resultant head acceleration. The SCM and TR muscles reached peak activity before peak head acceleration. The impact tests carried out at 7 km/h indicated that the ES, TR and SCM were activated on average (73 ± 27) ms, (84 ± 41) ms and (59 ± 9) ms after the impact stimulus, respectively (Fig. 4). The difference in reaction times between the three muscle groups was statistically significant with $p < 0.05$. It was clear that the SCM was activated significantly prior to the ES. SCM also reacted significantly faster than the TR. There was no significant difference between the reaction times of right and left ER and TR, whereas right SCM reacted faster than the left. The line chart of Fig. 4 is an illustrative summary of the analyses that were performed in an effort to gauge if the volunteers initiated motion prior to the onset of muscular contraction. The line chart summarises the relative timings between muscle activity and head acceleration as shown in Figs. 2 and 3. Also shown are the mean times of the start and peak acceleration for T1 and the pelvis.

The onset of head, T1 and pelvic acceleration occurred significantly prior to the onset of any muscular activity ($p = 0.001$). Peak acceleration of head, T1 and pelvis occurred significantly before the ES muscles reached peak activity ($p = 0.003$, 0.03, 0.01 respectively). No significant difference existed between time to peak TR activity and time to peak acceleration of head, T1 or pelvis. The SCM muscle achieved peak levels of activity significantly before the head ($p < 0.001$), pelvis ($p < 0.001$) before T1, and ($p = 0.006$) before pelvis had attained maximum acceleration. Baseline amplitude was examined in an effort to establish whether the volunteers were

Table 1

Approximate timing of onset of head, T1 and pelvis acceleration from the onset of sled acceleration.

Subject	Head (ms)	T1 (ms)	Pelvis (ms)
1	17	21	8.8
2	17	18	8.5
3	20	20	9.5
4	20	17	9.5
5	17	22	9.5
6	20	25	8.5
7	18	22	7.0
8	17	25	9.5
9	17	25	12
10	17	20	8.5
Mean	17.8	22.0	9.1
SD	1.26	2.47	1.32

more relaxed before or after impact. Pre-impact levels of baseline muscle activity were significantly greater than post-impact levels ($p < 0.001$). Wide variations, in terms of muscle response times, were also noted between subjects. It was found that 81% of the SCM activity occurred 50–80 ms post impact, indicating that this muscle was involuntary active during neck extension. This was not true for TR and ES.

None of the volunteers reported muscle soreness post test or at 48 h later. Volunteers had been subjected to the impact 7 days earlier and only participated if they had had 7 symptom-free days thereafter.

Discussion

It is possible that there is a causal relationship between cervical muscle activity and the aetiology of WAD but further work would be needed to confirm this. The TR and ES muscles reached their peak activity level an average of 94 ms and 150 ms post impact, respectively. Although the extensor muscles TR and ES reacted with a more delayed response than did the flexor SCM, the overall speed of the responses was rapid enough for them to be active when the

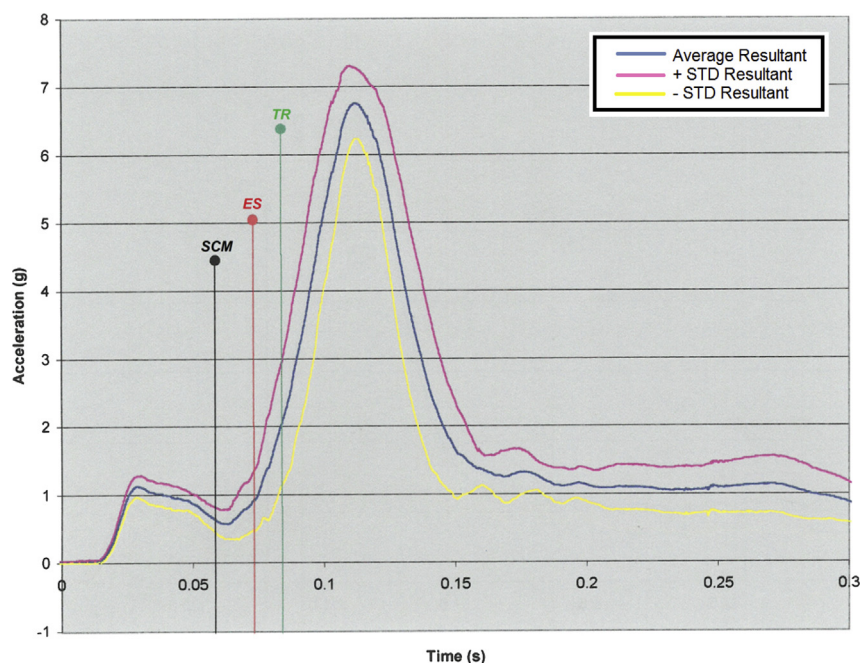


Fig. 2. Resultant head acceleration and time to onset of muscular activity. SCM: sternocleidomastoides; ES: erector spinae; TR: trapezius.

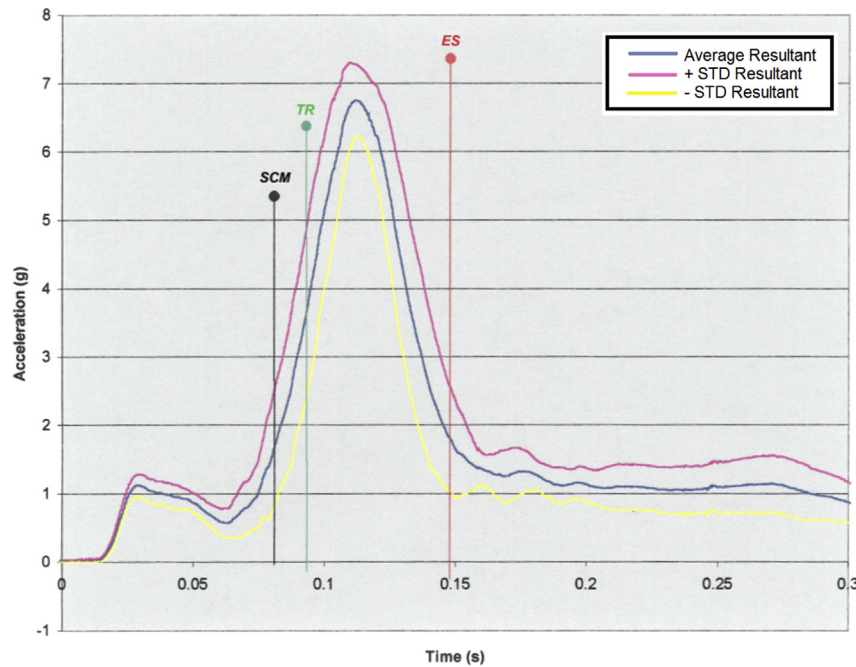


Fig. 3. Resultant head acceleration and time to peak muscle activity. SCM: sternocleidomastoideus; ES: erector spinae; TR: trapezius.

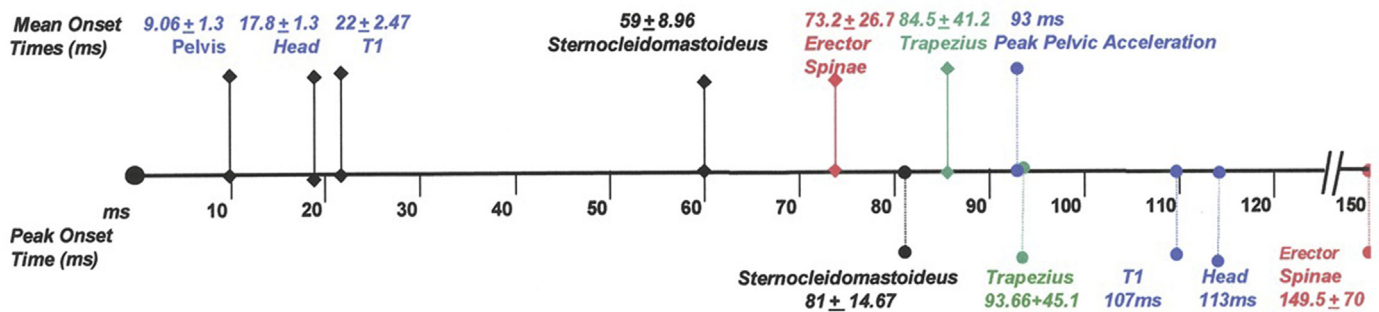


Fig. 4. Line chart summarising average times to onset and peak muscular activity and acceleration.

car occupant underwent whiplash motion, i.e. <200 ms. These results agree quite well with those of Siegmund et al.,¹² although our subjects were warned about the pending impact. Siegmund et al.¹² found no significant difference of reaction time between expected and unexpected impacts although the amplitude of response was greater for unexpected.¹³ These data implied that the neck flexors contracted eccentrically, as they contracted during the rearward acceleration of the head. This was shortly followed by contraction of the extensor muscles during head flexion, again indicating eccentric contraction. This was, however, not confirmed by measurement of the length of the muscles. With this assumption, it seems probable that muscle activation might amplify the risk of soft tissue injury development through negative or eccentric muscle contraction.

Occupants involved in rear-end collisions often complain of muscle tenderness in the neck and shoulder region and reduction of range of motion. The symptoms described by patients with such strain injuries have been attributed to primary sarcomere injury, which is thought to lead to an inflammatory response peaking 1–5 days later. Negative or eccentric muscle work is more likely to produce muscle soreness than concentric work,¹⁴ which may explain pain following rear impact in whiplash victims. It may be

that the acute injury may lead to degenerative changes, but further work will be needed to confirm this.

The most vulnerable site for these strain injuries appears to be near the myotendonous junction or the tendon bone junction.¹⁰ This may implicate the soft tissue proximal to the mastoid process of the temporal bone (proximal attachment of the SCM) and the inner third of the superior sternum (distal attachment of the SCM) as potential sites for strain injury to manifest itself. Given the advances in imaging technology (i.e. MRI), a closer look should be taken at these sites to look for damage and thus as a potential cause of pain.

The lumbar spine area was the first part of each volunteer to make contact with the seatback during impact, which explains why pelvic acceleration registered before T1 and head acceleration.

Since muscle activity was greater before than after impact, suggests that the subjects were bracing themselves for impact. The volunteers were aware that an impact was imminent and knew exactly when and what was going to happen. Some variations, in terms of muscle response times, were also noted between subjects. Such preparation may also account for this as failure to eliminate the audio and visual cues during this work may have allowed volunteers to subconsciously organise muscle action in advance. This

was a limitation of the current study, which had to be imposed for ethical reasons. However, as noted above, this probably changed the amplitude of the response, but not the latency.

It should be noted that these data were restricted to males whereas proportionately more women suffer from WAD.¹⁵ Perhaps the strength of the musculature is an explanation for these differences.

The EMG results suggest that observations based on cadaveric testing, for neck behaviour may be misleading and that any criteria and biofidelic crash dummies should be adopted to evaluate whiplash injury may have to incorporate more than simple neck based measurements.¹⁶

The cervical muscles reacted prior to peak head acceleration, thus in time to influence whiplash biomechanics and possibly injury mechanisms. It is recommended that muscular influences be incorporated into the development of the new rear-impact crash test dummy in order to make the dummy as biofidelic as possible.

Funding

None.

Ethical statement

The project was reviewed and approved by the Research Ethics Committee of the University of Aberdeen.

Conflicts of interest

There are no conflicts of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.cjtee.2018.10.006>.

References

1. Panjabi M, Grauer J, Cholewicki J, et al. Whiplash trauma injury mechanism: a biomechanical viewpoint. In: Gunzburg R, Szpalski M, eds. *Whiplash Injuries: Concepts in Prevention, Diagnosis, and Treatment of the Cervical Whiplash Syndrome*. Philadelphia: Pa: Lippincott-Raven; 1998:79–88.
2. Corcos DM, Gottlieb GL, Latash ML, et al. Electromechanical delay: an experimental artifact. *J Electromyogr Kinesiol*. 1992;2:59–68. [https://doi.org/10.1016/1050-6411\(92\)90017-D](https://doi.org/10.1016/1050-6411(92)90017-D).
3. Magnusson ML, Pope MH, Hasselquist L, et al. Cervical electromyographic activity during low-speed rear impact. *Eur Spine J*. 1998;8:118–125.
4. Szabo T, Welcher J. *Human Subject Kinematics and Electromyographic Activity During Low Speed Rear Impacts*. SAE Technical Paper; 1996, 962432 <https://doi.org/10.4271/962432>.
5. Forssberg H, Hirschfeld H. Postural adjustments in sitting humans following external perturbations: muscle activity and kinematics. *Exp Brain Res*. 1994;97:515–527.
6. Peterson L, Renstrom P. *Preventative Measures*. Illinois: Year Book Medical Publishers; 1986.
7. Renstrom PA. *Sports Injuries: Basic Principles of Prevention and Care*. London: Oxford Blackwell Scientific Publications; 1995.
8. Brault JR, Siegmund GP, Wheeler JB. Cervical muscle response during whiplash: evidence of a lengthening muscle contraction. *Clin Biomech (Bristol, Avon)*. 2000;15:426–435.
9. Brault JR, Wheeler JB, Siegmund GP, et al. Clinical response of human subjects to rear-end automobile collisions. *Arch Phys Med Rehabil*. 1998;79:72–80.
10. Garret Jr WE, Tidball J. Myotendinous junction: structure, function and failure. In: Woo SLY, Buckwater JA, eds. *Injury and Repair of the Musculoskeletal Soft Tissues*. Illinois: American Academy of Orthopaedic Surgeons; 1987:171–201.
11. UN/ECE Regulation No 44. *Restraining Devices for Child Occupants of Power-Driven Vehicles (Child Restraint System)*. 1998.
12. Siegmund GP, Sanderson DJ, Inglis JT. The effect of perturbation acceleration and advance warning on the neck postural responses of seated subjects. *Exp Brain Res*. 2002;144:314–321. <https://doi.org/10.1007/s00221-002-1048-2>.
13. Siegmund GP, Sanderson DJ, Myers BS, et al. Awareness effects the response of human subjects to a single whiplash-like perturbation. *Spine (Phila Pa 1976)*. 2003;28:671–692. <https://doi.org/10.1097/01.BRS.0000051911.45505.D3>.
14. Asmussen E. Observations on experimental muscular soreness. *Acta Rheumatol Scand*. 1956;2:109–116.
15. Spitzer W, Skovron ML, Salmi LR. Scientific monograph of quebec task force on whiplash-associated disorders: redefining “whiplash” and its management. *Spine (Phila Pa 1976)*. 1995;20:1S–75S.
16. Roberts AK, Hynd D, Dixon PR, et al. Kinematics of the human spine and the biofidelity of current dummies. In: *Proceedings of the International Conference on Vehicle Safety 2002*. London, UK: Imech Headquarters; 2002.