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Research Article

Adaptation of Postural Reactions in Seated Positions and Influence of Head Posture when Exposed to a Single Sideway Perturbation: Relevance for Driving on Irregular Terrain

Abstract

Background and objectives: Mechanical perturbations in seated positions caused by driving on irregular terrain destabilize the driver which, combined with the drivers' posture, may cause musculoskeletal disorders. The aim of this study was to investigate adaptation and the effect of different head postures on seated postural reactions caused by perturbations.

Materials and Methods: Twenty healthy male participants, aged 18-43 years, were tested on a movable platform delivering 15 sideways perturbations (peak acceleration 13.3 m/s^2) while the participants held their head in a neutral or a laterally flexed posture. Surface electromyography (EMG) signals were recorded bilaterally in upper neck, trapezius, erector spinae and external oblique, while kinematics were recorded with inertial sensors for the head, trunk and pelvis. EMG amplitudes, muscle onset latencies and angular displacements in the frontal plane were analyzed.

Results: In the neutral position, the EMG amplitudes and neck angular displacements significantly decreased by 0.2% and more than 1.6° respectively after repeated perturbations. Muscle onset latencies remained unchanged. During lateral flexion of the head, the EMG amplitudes decreased by 0.5% but the muscular onset latencies increased by more than 9 ms.

Conclusion: The developed neuromuscular strategy seem to prefer a reduced EMG amplitude. The modest size of the postural reactions during the conditions presented here do not by themselves explain the musculoskeletal disorders found in drivers.

Abbreviations

EMG: Electromyography; IMUs: Inertial Measurement Units; MVC: Maximum Voluntary Contractions; UN: Upper Posterior Neck Muscles; UT: Upper Trapezius; ES: Erector Spinae Lumbar Level; EO: External Oblique; E0: (Epoch 0) Time Window 150-0 ms before the Perturbation; E1: (Epoch 1) Time Window 0-150 ms after the Perturbation; E2: (Epoch 2) Time Window 150-300 ms after the Perturbation

Introduction

Mechanical perturbations in vehicles, caused by driving on irregular terrain, are transmitted to the body of the seated driver. For drivers of certain vehicles, such as forest machines and quad bikes, there can be a substantial exposure to perturbations in the sideway directions [1,2]. Perturbations are suggested to be hazardous to the spine [3,4] even though few studies have analyzed and reported adverse consequences.

The spine has to be stable to counteract for mechanical perturbations and in order to maintain equilibrium. The stabilization is achieved by the postural control system which is dependent upon intact sensory information to generate compensatory muscle reactions. If the postural control system does not adapt properly there may be potential hazards for musculoskeletal tissues. Low back pain is frequently reported among professional drivers exposed to perturbations [3,4].

Postural reactions in the neck are more sparsely studied. The head-neck system is a complex biomechanical linkage with at least 20 pairs of muscles rendering a range of opportunities in stabilizing the head [5]. The control mechanism for head stabilization depends on voluntary muscle mechanisms, postural reflexes, and passive mechanical (i.e. inertial, viscous, and elastic) properties [6,7]. The initial detectable movement caused by a perturbation occurs closest to the contact point and propagates further on to more distal body parts [7,8]. Therefore, the movements start in the pelvis segment followed by the trunk and head. It has been suggested that the head and trunk reactions initially rely on passive mechanical properties and signals from segmental proprioceptors [9].

The muscle reaction from a perturbation has been shown to be direction dependent in the trunk [10-12] and neck [8,13]. The muscle

reaction to a sideways perturbation has been suggested to have a reciprocal activation pattern in the neck, starting in the contralateral muscle that stretches first [8,14]. The EMG amplitude in the neck region has been reported to be high, especially in the contralateral splenius capitis and is therefore most likely to be injured [14]. Further, the initial posture has been reported to influence the nature of the postural reactions, e.g. a head rotation reduces the EMG amplitude in the upper neck [15]. A head posture divergent from neutral is common as a result of work demands [16], with large variations in time spent in that position assumed depending on the work, driver and vehicle [17]. We have not found any study that has investigated the influence of lateral flexion of the head on postural muscles or kinematic reactions from sideways perturbations.

Seated postural reactions, other than those caused by perturbations in fore and aft directions, are scarcely studied. However, sideways perturbations have been suggested to cause two types of reaction strategies, stiff and sloppy. Both reactions were found to be stereotypical, using either muscle co-contractions (stiff strategy) or a reciprocal more relaxed muscle activity (sloppy strategy) [8]. A strict stiff strategy might cause muscle fatigue or myalgia while a relaxed strategy, depending on passive structures, might increase the risk for injury in joint structures and tissues. Contrary to Vibert et al. (2001), who found no or little adaptation in sideways reactions, seated postural reactions in the forward direction have been found to adapt after the first perturbation with decreased EMG amplitude over time [18,19]. If seated postural reactions in sideways directions adapts or not is still an unanswered question. Based on this background we hypothesized that EMG amplitudes would be reduced after repeated perturbations and that different head postures would affect onset latencies and EMG amplitudes.

Therefore the aims of this study were to explore if seated postural neck and trunk reactions in healthy men adapt during repeated sideways perturbations and whether different head postures influence the results.

Materials and Methods

Participants

Twenty healthy males, age 27.5 ± 4.1 years, height 1.81 ± 0.07 m, body mass index (BMI) 24.5 ± 4.1 kg/m^2, participated. They were recruited among staff and students at Umea University, Sweden. Young male participants were targeted as they are representative of the majority of professional drivers [20] and to decrease the risk for degeneration and rigidity of the spine. Exclusion criteria were any reported neurological conditions or reduced ability to perform daily routines during the last 12 months because of back or neck problems. Written informed consent was obtained from each participant and the study was approved from Regional Ethical Review Board in Umea (Dnr 2014-228-32M).

Experimental protocol

This study used a repeated-measurement design with the participants exposed to 15 sideways perturbations in total (Figure 1). All perturbations were delivered from the participants' right side while the participant sat with the neck either in a neutral position or approximately 15° laterally flexed to the right or to the left.

A fixed experimental chair was mounted on top of a motion simulator (Micro Motion System, Bosch Rexroth, Netherlands) that delivered a sideways perturbation (Figure 2). The perturbations parameters were peak acceleration 13.3 (±1.6) m/s^2 with a total sideways translational stroke distance of 0.24 m. The acceleration level was approximately the same as the level used in the study by Kumar et al. [15]. The participants were seated facing forward, in a self-selected, but still relaxed posture (Figure 2). To reduce recovery strategies from other parts than the trunk and neck, several precautions were taken. The feet, without shoes, were placed together on a footrest. The height of the footrest was adjusted to a level where the participant's thighs were horizontal, while the feet still had contact with the support. The thighs were separated with a small cushion and tied together with a belt. The hands were resting on the thighs with palms upwards. Participants were instructed to retain their initial upper body and neck posture until the perturbation started and to resume a neutral neck posture following the perturbation.

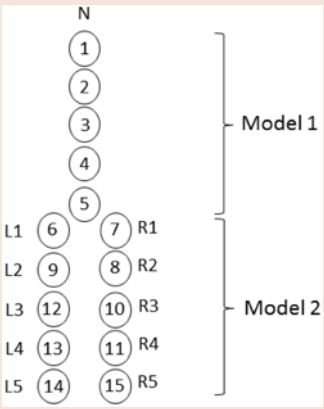

Figure 1: Scheme of perturbations. Totally 15 sideways perturbations, all delivered from the participant's right side. The first 5 perturbations, model 1, were delivered with the participant's neck in neutral position (N). This was followed by 10 perturbations, model 2, with the head laterally flexed 15° in left (L) or right (R) direction.

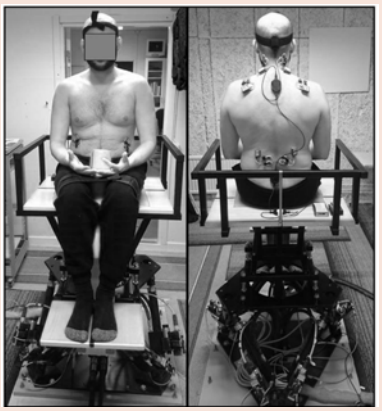

Figure 2: The movable platform, with the experimental flat chair from the front and back with a participant seated.

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Data acquisition

All motions from body segments and seat were detected by an analysis system from AnyMo AB, Sweden. The system, in this setting, sampled with a rate of 128 Hz and was synchronized with the EMG system. The motion system consisted of four inertial measurement units (IMUs) and one data acquisition unit. Each IMU detects acceleration and angular velocity of its mounted body segment. Customized software, using the collected accelerations and angular velocities, then calculated relative (between adjacent segments) and absolute angles (for each segment) and visualized the real-time orientation of the IMUs. The IMUs were placed on the back of the head and at spinal processes at level Th2 and S2 using either adhesive tape on the skin or a Velcro strap. Further, one IMU was mounted on the back of the seat. Movements were described as relative angles between two adjacent IMUs. There were three segments with relative joints; Neck (Head to Th2), Trunk (Th2 to S2) and Pelvis (S2 to seat). The IMUs, were aligned with the participant's selected posture and recalibrated just before the perturbation started. Motion data were collected in all three planes but only presented for the frontal plane where the major movements occurred.

The muscle activity was recorded with surface electromyography (S-EMG) from four locations in the neck and trunk, with a sampling frequency of 1500 Hz, using the TeleMyo Direct Transmission System and model 542 DTS EMG Sensor (Noraxon USA Inc., US). Prior to attaching the electrodes, the skin area was shaved and cleaned with a 70% alcohol solution. Bipolar circular surface Ag/AgCl electrodes (Ambu De) were placed with an inter-electrode distance of 2 cm, bilaterally on the upper posterior neck muscles (UN), upper trapezius (UT), erector spinae lumbar level (ES) and external oblique (EO) in accordance with our previous study [21].

Prior to measurements, the participants were asked to perform isometric maximum voluntary contractions (MVC). This was done for five seconds in static conditions using manual or rigid resistance. The UN and UT were tested in an erect seated position. The UN muscle was tested while the participants held their head in a neutral position and pressed the head in extension or lateral flexion against a headband providing rigid resistance. The UT muscle was tested with a height-adjusted arm sling over the shoulder to provide rigid vertical resistance immediately upon shoulder elevation. For the remaining muscles the participant were asked to lie on a bench that was tilted down approximately 30° for the upper body. The ES was tested in prone position with hands held beside the head and EO in sideway position with arms crossed. In both positions the participant's pelvis was buckled to the bench by a belt. For the ES the participants were asked to do a back extension during which they were given manual resistance at the horizontal level from the experimenter [22]. For the EO the participants were asked to perform a lateral flexion of the upper body and at the horizontal level were given manual resistance [22]. All tests were repeated twice with a minimum rest of one minute between tests. The EMG signals from the MVC test were normalized to a peak value during a 500 ms window. The highest amplitude of the EMG from the MVC tests for each participant and muscle was used for normalization of the EMG signals from perturbations when calculating the mean amplitudes.

Data analysis

Body motion data were low pass filtered at 10 Hz with a second order Butterworth filter. The segment angles are presented in the frontal plane and the algorithms used for calculating the angles is described in detail in Öhberg et al. (2013). Data are presented as relative angles (each IMU relative to the other) where the participant's first peak angle was used for statistical analysis. The IMU placed on the chair registered chair acceleration, which was used to calculate time events for the EMG analysis.

The EMG signals were amplified 500 times and low pass filtered at a cut-off frequency of 500 Hz. EMG signals, except muscle onset latencies were all processed in MyoResearch 1.07.63 XP™, (Noraxon USA Inc., US). EMG signals were digitally high pass filtered using a Butterworth filter, with a cut-off frequency set at 10 Hz. Electrocardiogram activity contaminating the EMG signals was automatically identified and removed. The EMG signals were further rectified and smoothed using root mean square (RMS) during a running 25 ms window [23]. Amplitudes were calculated during three epochs E0; 150-0 ms before the perturbation, E1; 0-150 ms after the perturbation and E2; 150-300 ms after the perturbation.

For muscle onset calculation the contamination of electrocardiogram was first identified and removed. Further a computer algorithm, Matlab, (R2013B, the MathWorks, Inc., USA), automatically determined the onset of EMG activity relative the start of perturbation for each muscle. The calculation of onset was based on linear trends and were in accordance with the description by Stensdotter et al. 2007 [24]. Subsequent to the automatic determination, muscle onsets were checked manually to avoid obvious misplacements or artifacts [25]. The investigator was blinded to the study while doing the muscle onset analysis and in cases of doubt a second blinded investigator gave an opinion. Trials were excluded according to criterion by Granata et.al (2004), i.e. if the onset of EMG activity occurred after more than 200 ms after the perturbation [26]. The time constraint was imposed to avoid confounding between automatic and voluntary reactions since voluntary reactions in standing has been suggested to begin 180 ms after perturbation onset [27,28].

The UT was removed from further analysis since UT had very little activity (< 2% MVC) regarding side and head postures. For the muscle onset analysis of the left side muscles, i.e. UN, ES, EO, there was a detectable muscle onset in over 90% of the cases, while the right side muscles only had 60% detectable onsets. Thirty cases (3%) of the left side muscles and 48 cases (8%) of the right side muscles were further excluded due to the 200 ms time window constraint.

Statistical analysis

Dependent variables (muscle amplitude, muscle onset latencies and peak angle) were normally distributed after log transformation and used for analyses. For comparisons, the muscle activity and the first peak angle displacements were analyzed in two steps using linear mixed models. The linear mixed model used here is preferred compared to a repeated measurements ANOVA because it deals better with missing values. Model 1 compared postural reactions using the first five repetitions. Model 2 compared the effect of different head postures and the order of repetition defined according to Figure 1.

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The neutral head posture was not included when comparing different head postures since it was not randomized in the order.

The fixed factors considered in the models for the muscle activity were: Side (left or right); Muscles (EO, ES and UN); Repetitions (1-5); Epochs (E0, E1, E2 (only for model 1)); Head posture (only for model 2). Random effects considered in the model, using a diagonal covariance structure model, were for EMG amplitudes random slopes Muscles + Epochs and for muscle onset latencies random slopes Muscle + Side. The decisive random factors were based on which factor was contributing with the largest variations on the dependent factor. All two-way interactions were tested but only those significantly improving the model were included. Models were compared using information criteria, – *2 Restricted Log likelihood* (maximum likelihood estimated) and the number of parameters included in the models.

Fixed effects considered for the peak angle displacements were the segments (pelvis, trunk, head) and order of repetitions. The segments were further used as a random effect with random slopes using a diagonal covariance structure model which fitted the data best. The alpha-value was set to 0.05 for all analysis and a Bonferroni correction was implemented when multiple comparisons were made for the repetitions. Analyses were performed using IBM SPSS version 22 (IBM Corp. IBM SPSS Statistics for Windows, Armonk, NY: IBM Corp.)

Results

Postural reactions

The final model, including all main factors and the significant interaction effects, presented in Tables 1,2 show that the EMG amplitude was (regardless of model) increasing for each epoch. The interaction *Side* * *Epoch* showed that the increase was less in the right side muscles. The interaction *Muscle* * *Epoch* showed a larger increase in the EO. There were no differences between the muscle sides other than in the conditions for Model 2. However the interaction *Side* * *Muscle* revealed a higher EMG amplitude in the right EO. The kinematics in Figure 3 displays an initially positive angle in pelvis. This is caused when the seat accelerates and the pelvis reacts later making the pelvis tilt to the right. The Trunk and Head have initially negative angles caused by inertia since the trunk and head reacts later than the pelvis.

Adaptation

The EMG amplitude in Model 1 was significantly reduced with 0.2% at the fourth ($p = .005$) and fifth ($p < .001$) perturbation compared the first. In Model 2, the EMG amplitude was reduced 0.2% at the third ($p = .006$) and fifth ($p < .001$) perturbation while the muscle onset latencies were increased at the third ($p = .006$) and at the fifth ($p < .001$) perturbation with 9 and 12 ms respectively.

The effect on the first peak angle displacement in Model 1 using a model including Segments and Repetitions, are presented in Table 3. The interaction *Segment* * *Repetition* showed that the neck peak angle displacement was significantly reduced with more than 1.6° after the first perturbation ($p < .001$). This is also displayed in Figure 3, where the deviation between the first and the following four perturbations was most prominent for the head.

Head postures

With a right laterally flexed head, there was a lower EMG amplitude (-0.5%) in the right side muscles compared to the left, a result that was not present in Model 1. There was generally a lower EMG amplitude in the muscles when laterally flexing the head to the left (-0.5%) compared to the right. The interaction *Side* * *Head posture* further showed that the EMG amplitude at the right side muscles were increased with 1.5% when the head was laterally flexed to the left. There was a general decrease in the muscle onset latencies (-3 ms) when laterally flexing the head to the left compared to the right, but no further interaction effects. There were differences between muscle sides with 15 ms higher muscle onset latencies in the right side muscles ($p = .011$), a result not present in Model 1.

Discussion

This study´s main objective was to investigate adaptation and the effect of different head postures on seated postural reactions in healthy male adults exposed to repeated sideways perturbations. The postural reactions in later repetitions in the session were initially decreased with regards to EMG amplitudes and peak angle displacements i.e. a short-term adaptation occurred. The influence of different head postures showed a side dependent change in EMG amplitude. The muscle onset latencies were reduced for a left-flexed head compared to a right. Differences in muscle onset latencies between the left and right side muscles were only present during the laterally flexed head condition. Thus, the hypotheses were confirmed except that the muscle onset latencies were only slightly affected.

There were no general side differences in EMG amplitude other than during the laterally flexed head condition. The EMG amplitudes increased for each epoch and the increases were stronger in the left side muscles. However, the interaction *Side* * *Muscle* showed that the amplitude in the right side EO were increasing. Therefore a lower EMG amplitude in the right side muscles was primarily in the UN and the ES. Higher EMG amplitude in the left side UN was expected and conforms with other studies investigating asymmetrically used muscles [14].

Our results show that the EMG amplitudes were significantly decreased after the third perturbation when the participants held their heads in a neutral position. The order of repetitions continued to have an effect during different head postures. These results conform to the results of Blouin et al., and Siegmund et al., who found decreased EMG amplitudes in the neck muscles (sternocleidomastoid, cervical paraspinal, scalenes and trapezius) following repeated forward perturbations [19,29]. The kinematics showed that a significantly decreased peak angle displacement in the neck occurred after the first perturbation. Previous studies of forward perturbations have reported both increased head angles and no change in head angles [18,19]. An increased segment angle should be possible as the EMG amplitudes was reduced. However, deeper muscles might be more active [8,30].

For muscle onset latencies no differences between the muscle sides were found in Model 1, but an increased muscle onset latencies were presented in the interaction *Side* * *Muscle* for the right side ES.

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Table 1: Mixed model for EMG amplitude (%MVC)): Estimates of fixed effects shows deviations from Intercept. Base Line is set to left side muscles (Side), upper neck muscle (Muscle) and Epoch 0 (E0). For model 1 and 2 the baseline includes repetition 1 (Repetition), for model 2 the head posture laterally flexed at right (Head posture). Two-ways interaction are included in the model when deviation is significant (′ p<.05, ″ p<.01, ‴ p<.001). Model 1; Adaptation for the first five repetitions, Model 2; Laterally flexing the head. Random effects were Muscle + Epoch for EMG amplitude and Muscle + Side for muscle onset latencies using a diagonal covariance structure model. Statistics is based on the logarithmic values and the back log estimate is presented to inform about the approximately linear values.

EMG amplitude (%MVC)		Model 1 Log Estimate (CI 95%)	*Back log Estimate*		Model 2 Log Estimate (CI 95%)	*Back log Estimate*
Intercept		0.08 (-0.05 - 0.21)	1.2		0.24 **(0.12 - 0.37)″**	**1.8**
Side	R	-0.01 (-0.05 - 0.21)	0.0		**-0.14 (-0.18 - -0.1)‴**	**-0.5**
Epoch	E1	**0.39 (0.29 - 0.49)‴**	**1.8**		**0.28 (0.22 - 0.34)‴**	**1.5**
	E2	**0.76 (0.66 - 0.85)‴**	**5.6**		**0.68 (0.61 - 0.74)‴**	**6.5**
Muscle	ES	0.15 (-0.01 - 0.32)	0.5		0.09 (-0.07 - 0.26)	0.4
	EO	-0.06 (-0.2 - 0.08)	-0.2		-0.08 (-0.22 - 0.07)	-0.3
Repetition	2	-0.01 (-0.04 - 0.03)	0.0		-0.01 (-0.04 - 0.01)	-0.1
	3	**-0.01 (-0.04 - 0.03)″**	**0.0**		-0.04 **(-0.07 - -0.02)″**	**-0.2**
	4	**-0.06 (-0.09 - -0.03)″**	**-0.2**		-0.03 **(-0.06 - -0.01)′**	**-0.2**
	5	**-.07 (-0.1 - -0.04)‴**	**-0.2**		**-0.05 (-0.08 - -0.03)‴**	**-0.2**
Head posture	L	n/a			**-0.13 (-0.16 - -0.11)‴**	**-0.5**
Side*Muscle	R*ES	0.01 (-0.05 - 0.06)	0.0		**-0.13 (-0.17 - -0.1)‴**	**-0.5**
	R*EO	**0.25 (0.2 - 0.3)‴**	**0.9**		**0.19 (0.16 - 0.23)‴**	**0.9**
Side*Epoch	R*E1	**-0.19 (-0.24 - -0.13)‴**	**-0.4**		**-0.16 (-0.2 - -0.12)‴**	**-0.6**
	R*E2	**-0.34 (-0.4 - -0.29)‴**	**-0.7**		**-0.34 (-0.38 - -0.3)‴**	**-1.0**
Muscle*Epoch	ES*E1	-0.05 **(-0.11 - 0.02)**	-0.1		0.03(-0.02 - 0.08)	0.1
	ES*E2	**-0.13 (-0.19 - -0.07)‴**	**-0.3**		-0.03 (-0.07 - 0.02)	-0.1
	EO*E1	**0.20 (0.14 - 0.27)‴**	**0.7**		**0.23 (0.18 - 0.28)‴**	**1.2**
	EO*E2	**0.22 (0.15 - 0.28)‴**	**0.8**		**0.22(0.18 - 0.27)‴**	**1.1**
Side* Head posture	R*L	n/a	n/a		**0.27 (0.24 - 0.3)‴**	**1.5**

Table 2: Mixed model for muscle onset latencies (ms): Estimates of fixed effects shows deviations from Intercept. Base Line is set to left side muscles (Side), upper neck muscle (Muscle). For model 1 and 2 the baseline includes repetition 1 (Repetition), for model 2 the head posture laterally flexed at right (Head posture). Two-ways interaction are included in the model when deviation is significant (′ p<.05, ″ p<.01, ‴ p<.001). Model 1; Adaptation for the first five repetitions, Model 2; Laterally flexing the head. Random effects were Muscle + Epoch for EMG amplitude and Muscle + Side for muscle onset latencies using a diagonal covariance structure model. Statistics is based on the logarithmic values and the back log estimate is presented to inform about the approximately linear values.

Muscle onset (ms)		Model 1			Model 2	
		Log Estimate (CI 95%)	*Back log Estimate*		Log Estimate (CI 95%)	*Back log Estimate*
Intercept		**2.01 (1,98 - 2.05)‴**	**103.4**		**2.00 (1.96 - 2.04)‴**	**100.6**
Side	R	0.03 (-0.02 - 0.09)	8.5		**0.06 (0.02 - 0.11)′**	**15.4**
Muscle	ES	-0.01 (-0.06 - 0.04)	-2.6		-0.02 (-0.07 - 0.02)	-5.2
	EO	-0.02 (-0.06 - 0.02)	-4.9		**-0.07 (-0.1 - -0.03)″**	**-14.1**
Repetition	2	-0.03 (-0.05 - 0.01)	-6.3		0.01 (-0.01 - 0.03)	3.0
	3	-0.03 (-0.06 - -0.01)	-8.0		**0.04 (0.02 - 0.06)″**	**9.3**
	4	-0.01 -0.04 - 0.01)	-3.4		0.02 (-0.01 - 0.04)	5.0
	5	-0.02 (-0.05 - 0.01)	-5.3		**0.05 (0.03 - 0.07)‴**	**12.1**
Head posture	L	n/a	n/a		**-0.01(-0.03 - 0.01)′**	**-3.2**
Side*Muscle	R*ES	**0.08 (0.03 - 0.14)″**	**22.1**		n/a	n/a
	R*EO	-0.02 (-0.06 - 0.03))	-3.6		n/a	n/a

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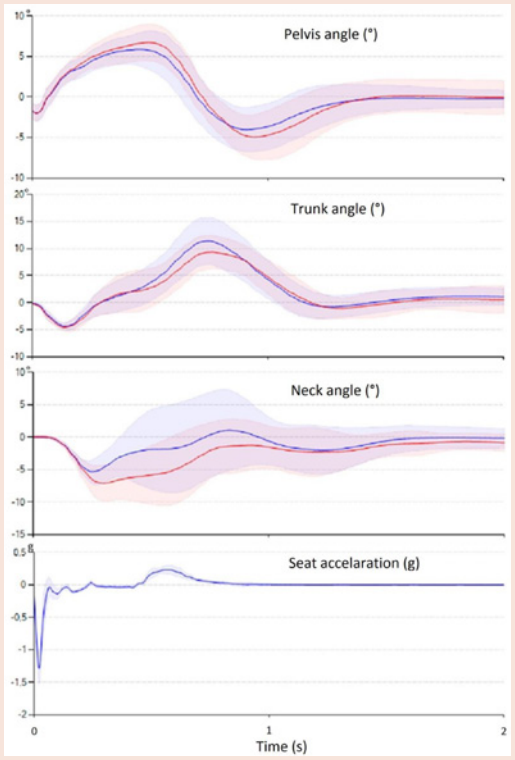

Figure 3: Angle displacements in frontal plane for the pelvis, trunk and head segments. Presented together with the seat acceleration in g (9.81 m/s^2) for 2 seconds following the start of perturbation. The angle displacements and standard deviation are presented at group level for the first perturbation, red, and the following perturbations, blue.

Table 3: Mixed model: Estimates of fixed effects shows deviations from Intercept. Mixed model with the base line pelvis (Segment), repetition 1 (Repetition) and interactions (Segment*Repetition) Trunk*1 and Neck*1 for adaptation for the first five repetition with head in neutral position. Segments were included in the model as random effect using an Diagonal covariance structure model.

Angle deviation (°)		Log Estimate (CI 95%)	Back log Estimate
Intercept		**0.99 (0.94 – 1.03)‴**	**9.7**
Segment	Trunk	**-0.33 (-0.38 - -0.27)‴**	**-5.1**
	Neck	**-0.14 (-0.22 - -0.06)″**	**-2.6**
Repetition	2	**-0.04 (-0.07 - -0.01)′**	**-0.8**
	3	-0.03 (-0.06 - 0.01)	-0.6
	4	-0.02 (-0.06 - 0.01))	-0.5
	5	-0.02 (-0.05 - 0.01)	-0.4
Segment*Repetition	Trunk*2	0.03 (-0.02 - 0.07)	0.7
	Trunk*3	0.01 (-0.03 - 0.06)	0.3
	Trunk*4	0.01 (-0.04 - 0.05)	0.1
	Trunk*5	0.01 ((-0.04 - 0.05)	0.2
	Neck*2	**-0.08 (-0.13 - -0.04)‴**	**-1.6**
	Neck*3	**-0.09 (-0.13 - -0.04)‴**	**-1.7**
	Neck*4	**-0.09 (-0.13 - -0.04)‴**	**-1.7**
	Neck*5	**-0.11 (-0.15 - -0.06)‴**	**-2.1**

′p<0.05. ″ p<0.01. ‴ p<0.001

In Model 2 there were general side differences with higher muscle onset latencies in the right side muscles but no interaction effects explaining the results. A slower onset in the right side splenius capite has previously been shown [14]. Their muscle onset latencies in left splenius capite conforms to our estimated onset latencies in UN while their right onset were much slower.

No adaptation was found for the muscle onset latencies when the head was in a neutral position. This is in agreement with Siegmund et al., who tested sternocleidomastoid and cervical paraspinal muscles [18]. In contrast to those result, we observed an adaptation effect towards increased muscle onset latencies when the participants flexed their heads. Further, a left laterally flexed head showed a decreased muscle onset latency as compared with a right laterally flexed head. There were no interactions explaining this difference so the mechanism behind this finding is hard to explain. However, one possible explanation could be that the muscles in the antagonist side, i.e. right side muscles, responded more to a prestretch compared with the agonist muscles. This may have reduced the muscle onset latency when the head was laterally flexed to the left. Prestretching the muscles before a perturbation may initially reduce the muscle onset latency slightly, but the effect is washed out or reversed in later repetitions which is why such a strategy should not be recomended.

A laterally flexed head posture affects the EMG amplitude. At baseline, a right laterally flexed head reduced the EMG amplitude in the right side muscles. The condition seems reversed with a left flexed head when including the head posture main effect and the interaction *side * head*. The hypothesis was that the muscle spindles would be prestretched due to the laterally flexed head and therefore affect the EMG amplitude, which seems to be confirmed. The estimated EMG amplitudes with a laterally flexed head seems higher compared to a neutral head posture. This is in contrast to Kumar et al. who reported a reduced EMG amplitudes e.g. splenius capite, trapezii, for a paticipant seated with a rotated neck compared a neutral head posture [15]. The different outcomes may be explained by biomechanical differences between a rotated and a laterally flexed neck.

Perturbations are suggested to be hazardous to the spine where muscles may overcompensate in their reactions [31] or be passive relying on intrinsic mechanism [8]. In this study the UT was excluded from further analyses since the level was considered too low (< 2% of an MVC), reducing the possibility to detect clear muscle onset. The remaining muscles EMG amplitudes were higher, but still modest since the estimates never exceeded 15% of an MVC. Previously reported EMG amplitudes of 80-90% of an MVC in splenius capitis may be found in temporary peaks but not in a 150 ms time window. The level might depend on cross-talk from other muscles and the performance of MVC is also very important and may greatly impact the result.

In this study a transient perturbation in one translational direction was used. An authentic perturbation when driving on irregular terrain would probably spread in multiple directions with abrupt changes and high accelerations affecting the postural reactions. Further, there may be repeated perturbations in field conditions. We excluded the influence of using the arms against a steering wheel although that would be the natural position for a driver. This procedure was

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done in order to solely rely on the spine kinematics which should have the highest relevance for musculoskeletal disorders in this body region. The participants were all young males which may influence the generalizability of the results, for example with respect to age and gender. The perturbation was chosen to always be delivered from the participant's right side. The postural reaction from a perturbation from the left side would probably be inversed from our results. However, muscle side dominance could affect the outcome. No power analysis was made so insignificant results should be interpreted with caution. However most of the outcomes showed a statistical significance.

Electrical muscle activity was collected with surface EMG on superficial muscles and not with intramuscular electrodes inserted in deeper muscles. Deeper muscles have been suggested to be of importance for stabilizing the spine in seated postural reactions during external perturbations [8,30]. However, the needle insertion causes discomfort, could be difficult to place and, given the sensitive structures in the neck, and requires special ethical considerations.

The dropout of detectable muscle onsets may have several explanations such as signal noise, artefacts from cables, small muscle reactions relative the baseline or a combination of any of these. For the right side muscles the missing muscle onsets was of importance since only 55% of the cases have a clear muscle onset below 200 ms and even less in the ES and EO. The ES is activated in sitting so the lack of clear muscle onset in the right ES may be caused by a small discrepancy between reaction and the baseline level. Difficulties in finding muscle onsets in abdominal muscles in the contra lateral directions of the perturbation has been reported in an earlier study [11].

Small activities in superficial muscles combined with small peak angular displacements seem not to imply a high risk for musculoskeletal problems from these perturbations. A clear short-term adaptation exists with reduced EMG amplitudes for healthy male adults with no experience of driving vehicles on rough terrain. The neuromuscular system seems to use feedback from previous perturbations to reduce the muscular activation which may lower the risk for myalgia and fatigue. However, the first unexpected perturbation should be the most severe and thus the driver should be aware of the terrain and be prepared to respond accordingly. It appears that a laterally flexed head should be neither worse nor better than a neutral head posture according to the results in this study. The low EMG amplitudes found in the present study for tested muscles supports the theory that deeper muscles or passive structures are involved and their role in stabilization of the spine during external perturbations should therefore be studied further. Future studies should also investigate whether the modest postural reactions measured experimentally complies with authentic field measurements. Ideal head and neck postures, optimal spine stabilizing strategy as well as vehicle ergonomic design should be further studied.

Conclusions

Seated postural reactions after repeated sideways perturbations with the head in neutral position adapted with decreased EMG amplitudes in the upper neck, erector spinae and external oblique and decreased neck angle displacement. Muscle onset latencies were unaffected. With the head laterally flexed to the left, the EMG amplitude in the right side muscles increased. The muscle onset latencies also started to adapt with increased muscle onset latencies at later repetitions when the head postures were flexed. The size of the peak angular displacements and muscle activities by themselves do not imply a high risk for musculoskeletal overload. The developed neuromuscular strategy due to seated perturbations appear to prefer a reduced EMG amplitude.

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