

NIOSH RO3 Final Progress Report

Project:

Vibration, Proprioception, and Low Back Stability

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List of Terms and Abbreviations

LBD – Low Back Disorder

HAT – Head, Arms and Trunk

WBV – Whole Body Vibration

Position Sense Test Measures:

aRSE – absolute reposition sense error

dRSE – directional reposition sense error

Sudden Loading Test Measures:

timePM – time to peak muscle activity

preEMG – preparatory muscle activity

deltaEMG – peak muscular response

TF – peak torso flexion

preTF – preparatory torso flexion

Other Experimental Abbreviations:

EMG – electromyography

iEMG – integrated EMG

RMS – root mean square error

C7 – the 7th cervical vertebra

T10 – the 10th thoracic vertebra

T12 – the 12th thoracic vertebra

L2 – the 2nd lumbar vertebra

L3 – the 3rd lumbar vertebra

S1 – the 1st sacral vertebra

ANOVA – analysis of variance

SD – standard deviation

Model Parameters:

Td – time delay

m – mass

I – rotational inertia

B – damping constant

K – preparatory trunk stiffness

Mp – perturbation moment

l – height of center of mass of the trunk

θ – trunk flexion angle

ζ – damping ratio

G – neuromotor gain

r – ratio of neuromotor gain to trunk stiffness

Th – threshold of the neuromotor response

Abstract

The National Institute of Occupational Safety and Health report “Musculoskeletal disorders and workplace factors” lists vibration as one of the five physical workplace factors associated with low back disorders with whole body vibration increasing risk from 1.2 to 39.5 times. However, little had been done to examine the effect of vibration on low back neuromotor control. Such neuromotor control can be a factor in stabilization and injury avoidance in the lumbar spine. The goals of this research project were to assess the effects of muscle vibration on low back proprioception (the ability to sense posture) and dynamic response both during and after vibration exposure in order to examine this possible effect as well as to examine the role of sensory elements within the muscle in dynamic low back stabilization. This research investigated this using both a model of dynamic spine motion and an experimental study examining proprioception as a function of muscle vibration.

In the model, changes in neuromotor response variables including decreased response gain, increased response threshold and increased response delay were all found to decrease the effective trunk stiffness, increasing the amount of trunk flexion that might be observed and possibly increasing the potential for injury. Experimentally, such an increased trunk flexion was observed both during and after exposure. Subjects were found to have a 16.1% greater torso flexion during exposure to paraspinal muscle vibration. Torso flexion remained significantly increased after vibration exposure relative to before exposure. Subjects were also found to have a 19.5% slower time to peak muscle activity supporting the neuromotor role in this change in dynamic response.

For the worker exposed to vibration, this research, as well as additional investigations by our laboratory and others, suggests that vibration may alter dynamic response to vibration. For such workers, dynamic events such as shock exposure in off-road driving or an unstable footing stepping out of a truck may pose a problem. A driver going to unload his or her truck after a day of vibration exposure may find himself or herself unable to appropriately to control trunk motion in the face of unexpected perturbations.

Highlights and Significant Findings

The key finding of this study were that vibration applied to the paraspinal musculature resulted in altered dynamic control of trunk motion both during and after exposure to 20 minutes of 20 Hz vibration including increased torso flexion in response to a perturbation and delayed peak muscle activity in response to a perturbation. These experimental results were supported by a model which suggested such findings could be a result of altered sensory and reflex behavior. Applying vibration directly to the muscle has been known to affect a sensory unit within the muscles called the muscle spindle organ. These results suggest that this sensory unit within the paraspinal musculature plays a role in stabilizing the lumbar spine and protecting against injury. In addition to elucidating the role of this sensory unit, this research suggests that occupation exposure to vibration such as in vehicular whole body vibration exposure may alter low back dynamics and that this mechanism could explain the increased incidence of low back injuries that has been observed in such occupations.

Translation of Findings

This research suggests that vibration exposure may alter one's sense of posture and control of low back motion. Both during and after exposure to whole body or low back vibration, workers should consider limiting strenuous or jostling manual materials handling tasks as they may have less motion control and be more susceptible to injury. Our preliminary results from this study and other studies suggest that during a period of 15-30 minutes after exposure, workers should limit lifting and other manual tasks.

Efforts should also be made to minimize shock exposure in combination with vibration exposure and to reduce overall vibration exposure.

Outcomes/Relevance/Impact

The potential impacts of this research are two fold. First, epidemiological evidence has long suggested that vibration exposure is associated with increased incidence of low back injuries. However, the causal mechanism by which this association occurs was not understood. This research suggests that the mechanism may be an indirect mechanism, namely altered sensory behavior in the spindle organs of the paraspinal musculature could alter lumbar motion control predisposing one to injury. Understanding this mechanism is important because it can provide additional methods for injury prevention. While removing the vibration exposure should remain the gold standard, rest periods after exposure may be useful to prevent injury. Future research should investigate whether there may also be unique methods that involve avoiding or recovering more quickly from the altered sensory behavior that might be of use in injury prevention.

The second potential impact is in improving our understanding of how the lumbar spine motion is controlled and the role of the sensory system in this control. Deficits in sensory ability, such as were created here by local muscle vibration but could also result from disease, previous injury, or other factors, might be a mechanism for low back injury in some populations and should be examined further.

Scientific Report

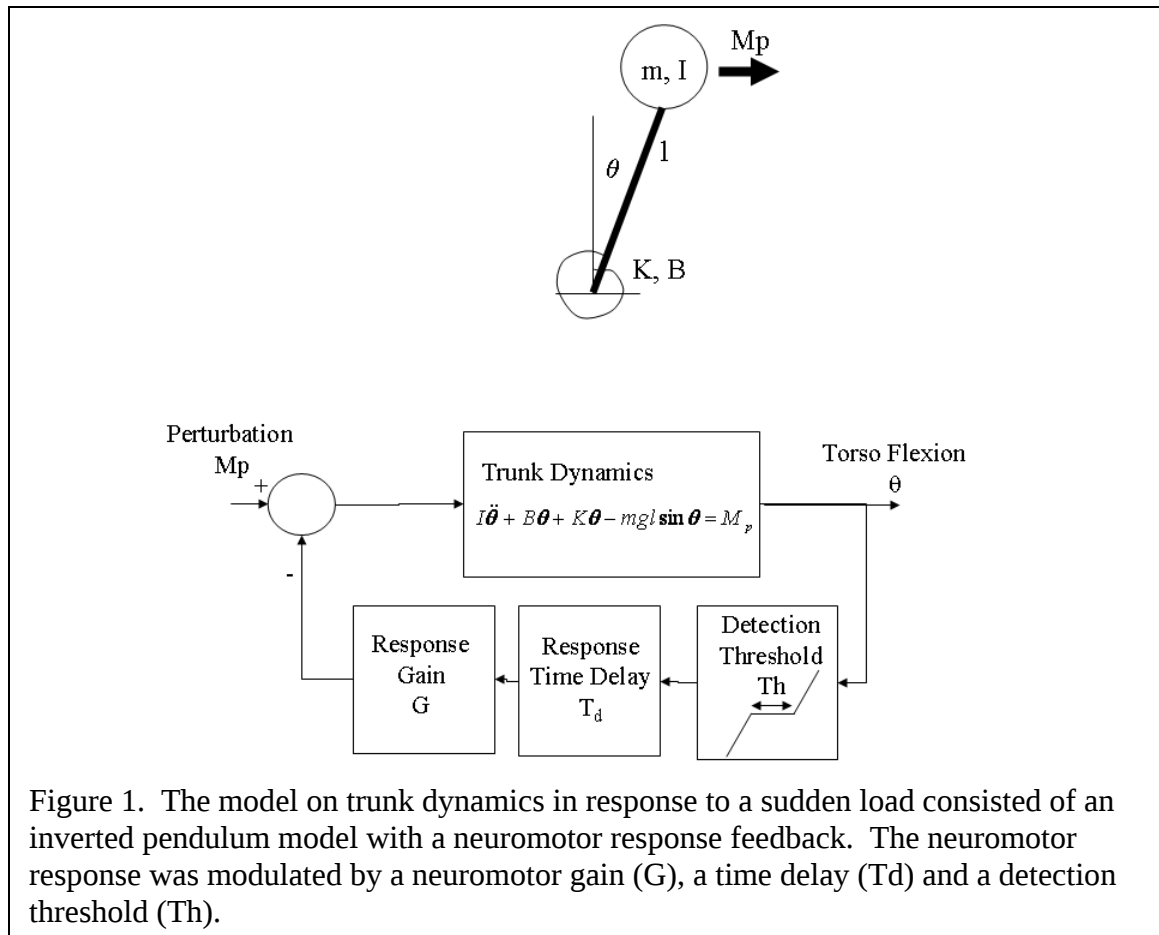
Background

Occupationally-related low back disorders (LBDs) are a leading cause of worker disability affecting up to 47% of workers and costing society from \$25 to \$100 billion annually [1, 2]. Loss of stability of the lumbar spine has been identified as a potential factor in the etiology of these injuries [3-7]. In particular, sudden loading events including slips and falls have been found to be associated with low back injuries in studies of injury rates in industry [8, 9]. Factors influencing spine stability such as reflex response time, lumbar co-contraction, and proprioception have been found to be altered in subjects with a history of low back pain [10-13].

A number of factors can contribute to the dynamic performance of the spine including the preparatory stiffening of the trunk through initial muscular activation and the timing and magnitude of neuromotor (reflex) response [3, 14]. Neuromotor response has been shown to be delayed in subjects with a history of low back pain, in subjects with exposure to whole body vibration, and in subjects with muscular fatigue [11, 15-17]. Delayed reflex response times have also been shown to be a risk factor for low back injury in athletes [6]. While a number of factors contribute to the dynamic motion of the spine, the neuromotor response has been shown to be necessary for dynamic performance of the trunk accounting for 42% of the total effective trunk stiffness [18].

A component of the neuromotor response is the ability to sense perturbations in joint position. Sensory systems in trunk dynamic stabilization include primarily the muscle spindle organs, but also ligament, intervertebral disc, and facet joint elements [19-22]. The muscle spindle organs are sensitive to local muscle vibration making local

muscle vibration a useful tool in evaluating the role of the muscle spindle organs. Such vibration induces increased errors in proprioceptive measures such as position sense and kinesthesia as well as a perception of muscle lengthening [23-26]. In addition to the kinesthetic illusions (perception of muscle lengthening) and increased error observed during muscle vibration, researchers have observed persistent proprioceptive errors after vibration exposure due possibly to neuromotor habituation or adaptation [27, 28]. In the low back, researchers have demonstrated that vibration of the paraspinal muscles of the low back results in increased proprioceptive errors both during and after exposure to vibration [19, 28]. Brumagne et al. [19], for example, found an increase in proprioceptive error from 2.16 degrees before vibration to 4.51 degrees after vibration.



In this work, the objective was to examine the role of the sensory system in dynamic motion of the trunk both with a simple dynamic model and experimentally. Using the dynamic model, the effect of increasing sensory error (modeled as an increase in sensory threshold) on dynamic system performance of the trunk was assessed (Aim 2 of the original proposal). The effects observed in the model were then tested experimentally by applying vibration to the paraspinal musculature and observing a subject's response to a sudden unexpected load applied to the trunk (Aim 1 of the original proposal).

Aim 2: Procedures and Methodology

To understand the role of the sensory system in trunk dynamic performance, a lumped parameter model of the trunk was created using Simulink (MATLAB, Natick MA) and a variable step, Runge-Kutta differential equation solver. In this model, trunk dynamics were modeled as a second order, inverted pendulum system (Figure 1):

$$I\ddot{\theta} + B\dot{\theta} + K\theta - mgl \sin \theta = M_p \quad \text{eq.1}$$

where θ is the angle of trunk flexion, I is the rotation inertia of the head-arms-trunk (HAT), B is the damping constant, K is the preparatory trunk stiffness, m is the HAT mass, l is the height of the center of mass and M is a perturbation moment. Torso inertia and torso geometry were estimated using anthropometric data from Winter [29]. For this analysis, a 78 kg, 1.755 m male was used. The rotational inertia of the HAT (I) was estimated as 12.47 kgm^2 , the HAT mass (m) as 52.9 kg, and the height of the center of mass (l) as 0.381 m. The damping constant (B) was calculated based on maintaining a damping ratio (ζ) of approximately 0.2 for the combined stiffness and neuromotor response gain:

$$B = 2\zeta \sqrt{(K - mgl + G)I} \quad \text{eq. 2}$$

Similar simple models of trunk dynamics have been shown to be effective in modeling sudden loading dynamics in the trunk [16, 30-32].

To this model, a neuromotor response to angular deflection was added (Figure 1). This response contained three components. First, a proportional gain (G) in the response represented the moment generated as a response to a unit change in angular deflection. Second, a time delay (T_d) of 70 ms was included to account for conduction and electromechanical delays in the neuromotor systems. This corresponds to the average

reflex latencies of 67 to 71 ms reported by Granata et al. [33]. Finally, a detection threshold (Th) was included to model the ability of the sensory system to detect an angular deflection.

Cholewicki et al. found trunk stiffnesses of 1253 ± 760 Nm/rad (21.88 ± 13.26 Nm/deg) [31]. This trunk stiffness is the effective stiffness of the trunk dynamic response system and would include both the preparatory trunk stiffness (K) as well as the gain (G) from the neuromotor response system. In a simple, proportional feedback system, the effective stiffness would be the sum of the preparatory stiffness (K) and the gain (G). To examine the relative roles of the preparatory trunk stiffness and the response gain on the effect of the detection threshold, the effective stiffness of 1253 Nm/rad was split between the trunk stiffness and the response gain. The ratio of response gain to trunk stiffness was then increased from 0 (no neuromotor response) to 1 (equal contributions of neuromotor gain and trunk stiffness).

To simulate the experimental setup and the load cell pattern, a 115 Nm perturbation moment was applied to the system for 0.10 seconds. The detection threshold was then varied from 0 to 5 degrees. The detection threshold is the minimum angular deflection required before a neuromotor response is elicited. Increasing the detection threshold represents a loss in the ability to sense trunk posture. This range was selected to represent the increase in proprioceptive errors with local muscle vibration (from 2.16 to 4.51 degrees) observed by Brumagne et al.[19].

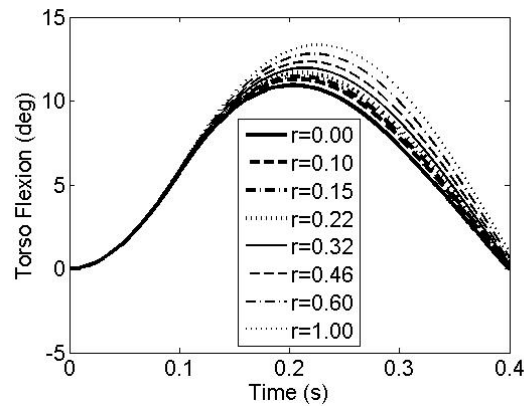


Figure 2. Increasing the ratio of neuromotor gain over preparatory stiffness ($r=G/K$) resulted in an increase in torso flexion and in time to peak torso flexion. Although the effective overall trunk stiffness of a simple proportional feedback system should be the sum of neuromotor gain (G) plus intrinsic trunk stiffness (K , which was held constant), the time delay in the neuromotor gain led to a decrease in effective overall trunk stiffness and an increase in trunk flexion.

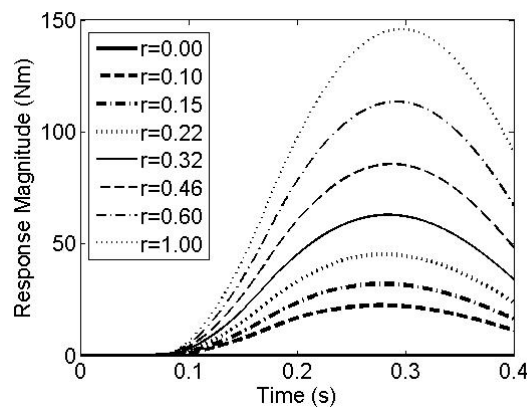


Figure 3. Increasing the ratio of neuromotor gain over preparatory stiffness ($r=G/K$) increases the magnitude of the neuromotor response.

Aim 2: Results

Both increases in the ratio of response gain to preparatory stiffness and increases in the detection threshold were examined using the computational model. The ratio of response gain to preparatory stiffness represents the relative contribution of the neuromotor response versus the preparatory stiffness in the sudden loading dynamics. While some have argued the neuromotor response is not necessary in dynamic trunk dynamics response [34], others have found the neuromotor response to be important for

trunk dynamics response [18]. By maintaining the sum of preparatory stiffness (K) and neuromotor response gain (G) at 1253 Nm/rad, the overall effective stiffness of the system should remain relatively constant. However, due to the time delays in the neuromotor response, an increase in torso flexion and time to peak flexion is observed with increase in the ratio (Figure 2). As the ratio increases, the neuromotor response gain increases which also results in an increased magnitude of the neuromotor response (Figure 3).

From these results, two values of the ratio of gain to stiffness were selected for further analysis: $r=0.1$, representing a system where the neuromotor response has very little effect and $r=0.7$, representing a system with a strong neuromotor response component. The ratio of 0.7 corresponds to the neuromotor gain contributing 41% of the total effective stiffness, close to the 42% of the total effective trunk stiffness reported by Moorhouse and Granata [18]. The detection threshold was then varied from 0 to 5 degrees. With the weak neuromotor response ($r=0.1$), increasing the detection threshold was found to increase the time to initiation of the neuromotor response. However, the time to both peak neuromotor response and the magnitude of the peak torso flexion were unchanged (Figures 4a and 5a). With a stronger neuromotor response ($r=0.7$), increasing the detection threshold resulted in increases in the time to initiation of the neuromotor response, the time to peak neuromotor response, and the magnitude of the peak torso flexion (Figure 4b, 5b, 6 and 7).

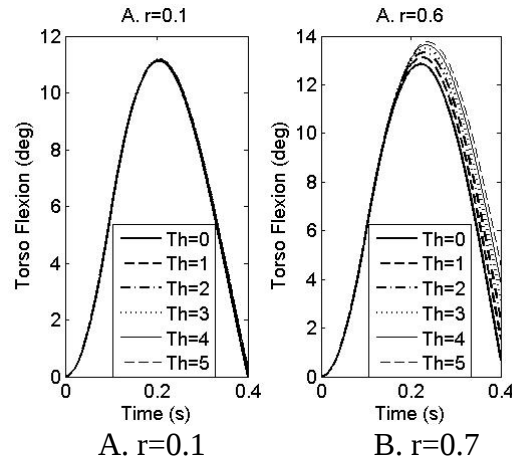


Figure 4. Increasing the detection threshold of the neuromotor response (Th) was assessed at both a low neuromotor response (A. $r=0.1$) and a high neuromotor response (B. $r=0.7$). With a low neuromotor response, little change was observed in the torso flexion. With a high neuromotor response, the magnitude of torso flexion and the time to peak torso flexion were increased with increases in the detection threshold.

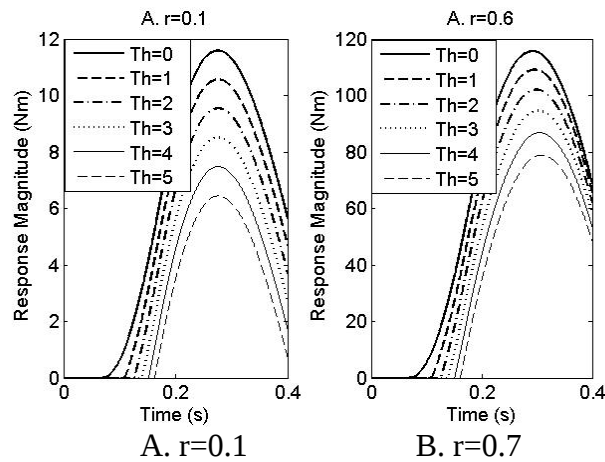


Figure 5. Increasing the detection threshold of the neuromotor response (Th) was assessed at both a low neuromotor response ($r=0.1$) and a high neuromotor response ($r=0.7$). Increased delays in initiation of the response were observed at both response levels. An increased time to peak flexion was observed with a high neuromotor response. With the neuromotor response gain held constant, a decreased magnitude of the neuromotor response was observed.

With the strong neuromotor response, increasing the detection threshold, in conjunction with a constant, linear neuromotor gain, results in a decrease in magnitude of neuromotor response (Figure 5). It could be argued that this decrease might be compensated by an increase in response gain. To investigate this possibility, while

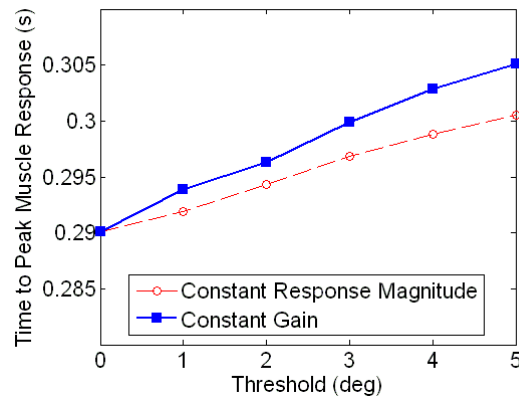


Figure 6. Increasing the detection threshold of the neuromotor response (Th) was assessed both while maintaining a constant gain (as in Figures 4 and 5) and while maintaining a constant response magnitude. With both, the time to peak muscle response was found to increase with increases in the detection threshold. The ratio (r) of gain over stiffness for this analysis was maintained at 0.7.

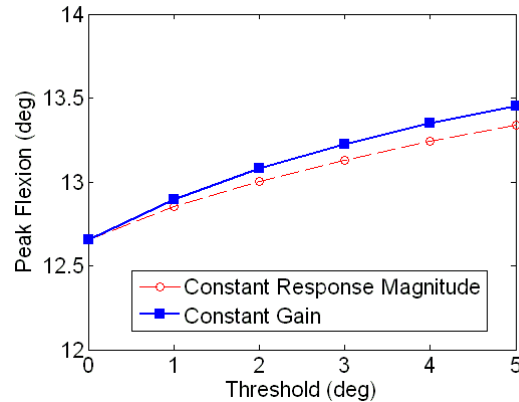
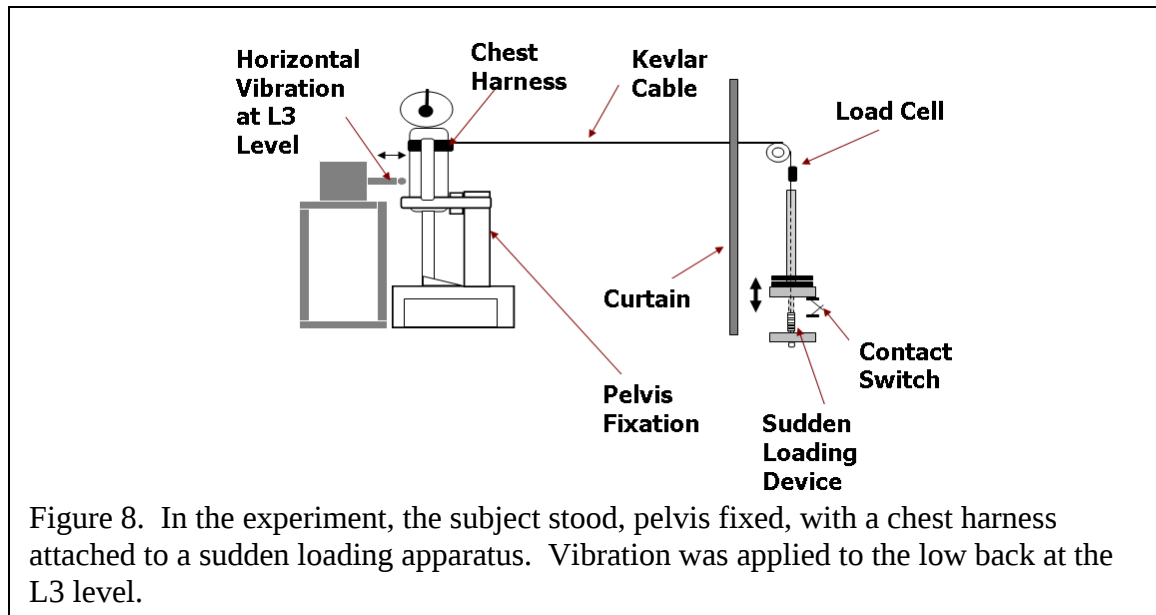


Figure 7. Increasing the detection threshold of the neuromotor response (Th) was assessed both while maintaining a constant gain and while maintaining a constant response magnitude. With both, the torso flexion was found to increase with increases in the detection threshold. The ratio (r) of gain over stiffness for this analysis was maintained at 0.7.

increasing the detection threshold, the gain was increased to maintain a constant magnitude of the neuromotor response. The time to peak neuromotor response and the magnitude of peak torso flexion were still found to increase with increasing detection threshold (Figure 6 and 7).

These results suggest that if the neuromotor response is a factor in trunk dynamic response (r is large), altering the threshold of the sensory system should result in increased time to peak neuromotor response and increased peak torso flexion. Based on



these results, it was hypothesized that paraspinal muscle vibration would result in increased time to peak neuromotor response and increased peak torso flexion.

Aim 1: Procedures and Methodology

21 adult subjects (13 male, 8 female) with ages ranging from 21 to 33 years consented for this study, which was approved by the Human Subjects Committee, University of Kansas, Lawrence, KS. Subjects with low back pain for more than a week within the last year or with any other musculoskeletal disorder that would interfere in performing painless trunk flexion and extension were excluded from the study. In this experiment, the response to sudden loading and lumbar position sense was assessed in three conditions: before, during, and immediately after vibration exposure.

A constant amplitude (2 mm) vibration of 20 Hz frequency was applied bilaterally over the paraspinal muscle groups at the L3 level of the lumbar spine for 20 minutes via a mechanical vibrating device. In studies of vibration induced proprioceptive errors, frequencies ranging from 20 to 120 Hz have been found to result altered proprioception

[23, 26, 28]. In a pilot study of proprioceptive errors with paraspinal muscle vibration, 20 Hz was found to have the most pronounced effects on positioning errors both during and after muscle vibration [28].

A 3-D electromagnetic motion analysis system (Ascension Tech., Burlington, VT and Motion Monitor, Chicago, IL) was used to collect kinematic data. This system has a resolution of 0.08 cm and 0.1 degrees and an RMS accuracy of 0.76 cm and 0.5 degrees. The three sensors were attached to the skin with double-sided tape over the C7, T10, T12, and S1 spinous processes, and over the manubrium. Using the position of the T10 and S1 sensors, torso flexion angle was determined as the angle between a line connecting these sensors and vertical. The difference in angular orientation between the T10 and S1 sensors in the anterior-posterior plane was defined as the lumbar angle. The manubrium marker allowed detection of trunk rotation and asymmetry of motion. This configuration is consistent with previous literature on lumbar position sense and lumbar-pelvic coordination [35, 36]. The electromagnetic sensor data was collected at 40 Hz.

Surface electromyographic electrodes (Delsys, Boston, MA) were attached to the skin over the Erector Spinae muscle groups at L2/L3 level of the spine with 4 cm inter-electrode spacing. The electromyographic data (EMG) were collected at 1500 Hz. Raw EMG data were band-pass filtered between 20 Hz and 500 Hz with several notch filters (40, 60, 80, 120, 180 and 240 Hz) to remove electrical and electromagnetic noise. The EMG data were rectified and integrated using a 100-point Hanning window. Prior to the experiment, isometric, maximum, voluntary exertions were performed. The subjects were instructed to do extension exertions three times for 5 seconds against a fixed frame while standing with their pelvis fixed to a stationary support. The average of the

integrated EMG (iEMG) for these maximal exertions was determined and used to normalize all subsequent iEMG.

During the position sense test, three electromagnetic sensors (T10, S1, and C7) were used in defining torso inclination (flexion) and lumbar angle. Torso inclination was defined as the angle between a line connecting the T10 and S1 sensors and vertical. Lumbar angle was defined as the angle between the T10 and S1 sensors. The C7 sensor was used to adjust for any axial rotational motion. The subject's target lumbar angle was determined before starting the trials as the average of the minimum and maximum ranges of motion (measured three times each). The target torso inclination (flexion) angle was kept at zero throughout the experiment. During the test, the subject was instructed to match the target lumbar angle, keeping their target torso inclination at zero. When the subjects reached their target posture, they were instructed to hold at that posture for 5 seconds while the data was collected. Reposition sense error was defined as the difference between the target lumbar angle and the assumed lumbar angle. The reposition sense protocol consisted of two types of trials. In the training trials, continuous visual feedback of both the torso inclination and lumbar angle was made available to the subject. The subject was instructed to match the target posture and was asked to remember that posture. In the assessment trials, the torso inclination feedback was still available, but the lumbar angle feedback was removed. The subject was instructed to reproduce the target posture from memory. Between every trial the subject was asked to perform a torso flexion to assure that he/she didn't hold the same posture.

The reposition sense test consisted of eight trials. First, the subject completed three training trials where they were given visual feedback of lumbar angle. After the

training trials the subject was given an assessment trial, where the subject did not have lumbar angle feedback (but torso inclination feed back was available) and the target posture was reproduced from memory. Training trials and assessment trials were alternated for a total of three assessment trials. Absolute reposition sense error (aRSE) was defined as an absolute value of the difference between the target lumbar angle and the actual lumbar angle (in degrees) during the assessment trials. Directional reposition sense error (dRSE) was defined as the difference between the target lumbar angle and the angle lumbar angle (in degrees) in the assessment trials.

During the sudden loading test, the subject stood with their hands to their sides, their legs straight and their pelvis fixed in place by an anterior support and belt. The subject wore a chest harness to which a weight was attached by a Kevlar cable via a load cell and a pulley (Figure 8). Both audio and visual cues were eliminated using headphones with music and a curtain blocking the subject's view of the drop mechanism. A load cell recorded the tension in the cable (Transducer Techniques, Temecula, CA). A preload (0.5 kg) was applied to maintain tension in the cable. To create a sudden impulse load a weight equal to 10% body weight was dropped a height of 5 cm. This weight was allowed to bounce against springs attached to the weight holder to create an impulse load. A contact switch was used to indicate the instant the dropped weight made contact with the springs. The subject was instructed to match the target lumbar angle and target torso flexion (to a visual biofeedback) prior to the sudden load to ensure the same pre-perturbation posture throughout the experiment.

During the sudden loading protocol, the electromyographic data, the load cell data and the contact switch data were simultaneously collected at 1500 Hz for 5 seconds.

Trunk motion was collected from the electromagnetic markers at 40 Hz simultaneously.

The sudden loading protocol was repeated 3 times for each condition.

EMG data from the right side Erector Spinae muscle group were analyzed to find the time to peak muscular activity (timePM). This timePM was defined as the time between the onset of the contact switch and the peak muscular response measured by iEMG. The preparatory muscle activity (preEMG) was calculated as the average iEMG for the 750ms prior to the onset of the contact switch. The peak muscular response (deltaEMG) was calculated as the difference between the maximum of the iEMG and the preparatory muscle activity. The torso flexion resulting from the sudden perturbation (TF) was defined as the difference between the peak torso flexion and the average of the torso flexion 750ms before the perturbation.

Huynh-Feldt adjusted, repeated measures analyses of variance (ANOVA) were performed with absolute reposition sense error (aRSE), direction reposition sense error (dRSE), the time to peak muscle activity (timePM), peak muscular activity (deltaEMG) and the torso flexion magnitude (TF) as dependent variables. The independent variables for these analyses were time (before, during and after vibration) and trial number (1st, 2nd or 3rd repeat). Multiple comparisons of the significant effects were further analyzed with simple within-subjects contrasts. These analyses were performed to test the hypotheses that aRSE, timePM, deltaEMG and TF would increase either during or after exposure to whole body vibration relative to before exposure. Additional repeated measures ANOVA were performed on the preparatory muscular activity (preEMG) and preparatory torso flexion (preTF) to ascertain if changes occurred in the pre-sudden load

Table 1. In the experiment, the sudden loading protocol was repeated three times before, during, and after vibration exposure. Time to peak muscle activity (timePM), peak muscular activity (deltaEMG), torso flexion (TF), preparatory muscular activity (preEMG) and preparatory torso flexion (preTF) were assessed.

	Before Vibration		During Vibration		After Vibration		
	Mean	SD	Mean	SD	Mean	SD	
timePM	213.4	57.0	255.0	62.9	237.4	70.6	ms
deltaEMG	41.5	48.1	46.8	59.5	49.0	52.5	%max
TF	8.77	3.39	10.18	5.19	9.38	3.70	deg
preEMG	18.4	12.8	20.1	16.5	19.1	14.3	%max
preTF	-0.37	3.51	-0.47	4.19	-0.59	2.90	deg

state of the subject with vibration exposure. These measures were assessed to examine other potential factors in the dynamic performance of the low back.

Aim 1: Results

The computational model predicted that if neuromotor response was large, increases in the detection threshold (representing loss in sensitivity of the sensory system) would result in increased time to peak neuromotor response, decreased magnitude of neuromotor response, and increased torso flexion magnitude. Experimentally, the time to peak neuromotor response can be observed by the time to peak Erector Spinae muscle activity (timePM). The timePM was found to increase from 213.4 (SD 57.0) ms to 255.0 ms (SD 62.9) during vibration and 237.4 (SD 70.6) ms after vibration (Table 1). A Huynh-Feldt adjusted, repeated measures ANOVA found the effect of the vibration condition to be significant ($p < 0.01$, Table 2). Further within-subjects contrasts found significant increases in timePM between the before and during conditions ($p < 0.01$, Table 2), but only a trend between the before and after vibration conditions ($p = 0.09$, Table 2). The magnitude of the ES muscle activity (deltaEMG) was also found to increase from 41.5 (SD 48.1) %max to 46.8 (SD 59.5) %max during vibration and 49.0 (SD 52.5)

%max after vibration (Table 1). However, this increase was not found to be statistically significant ($p=0.22$, Table 2).

The torso flexion magnitude (TF) was found to increase both during and after vibration relative to before vibration exposure (Table 1). The TF increased from 8.77 (SD 3.39) degrees to 10.18 (SD 5.19) degrees during vibration and 9.38 (SD 3.70) degrees after vibration. A Huynh-Feldt adjusted, repeated measures ANOVA found a significant effect of the vibration condition ($p<0.05$, Table 2). Further, within-subjects, contrasts revealed significance between the before and during vibration conditions ($p<0.05$, Table 2) and the before and after conditions ($p<0.05$, Table 2).

To assess if preparatory stiffness (K) behavior may have changed, the preparatory muscle activity (preEMG) and preparatory torso flexion (preTF) were also examined (Table 1). Neither the preparatory stiffness nor the preparatory torso flexion were found to change significantly with vibration condition ($p=0.60$ and $p = 0.91$, Table 2).

Table 2. Repeated measures ANOVA were performed for the five sudden loading variables. Multiple comparisons of the significant effects were made using within-subjects contrasts to compare before vibration to both during and after vibration separately. Significant results ($p < 0.05$) are highlighted.

A. Time to Peak Muscle Activity (timePM)

ANOVA	epsilon	df	F	P
Vibration	1.000	2.000	5.270	0.009
Trial	1.000	2.000	0.816	0.449
Vibration*Trial	1.000	4.000	0.402	0.807

Within-Subjects Contrasts	Levels	F	P
Vibration	Before-During	10.400	0.004
Vibration	Before-After	3.168	0.090

B. Peak Muscular Activity (deltaEMG)

ANOVA	epsilon	df	F	P
Vibration	0.961	1.922	1.561	0.223
Trial	1.000	2.000	0.165	0.849
Vibration*Trial	0.350	1.400	0.769	0.430

C. Torso Flexion Magnitude (TF)

ANOVA	epsilon	df	F	P
Vibration	0.673	1.346	4.251	0.038
Trial	1.000	2.000	0.033	0.968
Vibration*Trial	0.651	2.606	0.307	0.792

Within-Subjects Contrasts	Levels	F	P
Vibration	Before-During	5.612	0.028
Vibration	Before-After	5.581	0.028

D. Preparatory Muscular Activity (preEMG)

ANOVA	epsilon	df	F	P
Vibration	1.000	2.000	0.515	0.601
Trial	1.000	2.000	2.479	0.097
Vibration*Trial	0.823	3.291	1.767	0.157

E. Preparatory Torso Flexion (preTF)

ANOVA	epsilon	df	F	P
Vibration	1.000	2.000	0.094	0.911
Trial	0.704	1.408	0.703	0.456
Vibration*Trial	0.435	1.739	1.976	0.159

Error	Before Vibration	During Vibration	Immediately after vibration	After 10 minutes rest	After 20 minutes rest
aRSE (degrees)	1.79 $\pm$ 0.82	2.23 $\pm$ 1.56	1.92 $\pm$ 1.28	1.86 $\pm$ 1.57	1.94 $\pm$ 1.26
dRSE (degrees)	-0.43 $\pm$ 1.65	-1.13 $\pm$ 1.72	-0.53 $\pm$ 1.63	-0.56 $\pm$ 2.24	0.17 $\pm$ 1.92

Table 3. Mean $\pm$ S.D. for absolute reposition sense error and directional error in degrees for all conditions.

For the position sense test, the results were less clear. The aRSE did increased by 24.6% during vibration compared to before vibration condition and was increased by 7.3% after vibration relative to before vibration. However, the ANOVA did not show significance for pre, during and after vibration ($p=0.404$). Within-subjects contrasts showed a trend between before and during vibration ($p=0.202$) and did not show significance between before and after vibration exposure ($p=0.67$).

The dRSE increased negatively during vibration (from -0.43 ± 1.65 deg to -2.23 ± 1.56 deg) and returned close to zero after vibration (Table 2.2). The ANOVA did not show significance ($p=0.203$) for pre, during and after vibration. Within-subjects contrasts showed a strong trend between pre and during vibration ($p=0.077$) and did not show significance between before and after vibration exposure ($p=0.760$). While these results for reposition sense errors followed the patterns one might expect for a system with losses in proprioception and with kinesthetic illusions during vibration, as had been seen in previous studies of ankle and arm proprioception with vibration, the variability of this data makes it difficult to make any firm conclusions.

Discussion

In the dynamic model of the trunk, increases in the sensory detection threshold (a model of increases in proprioceptive error) were predicted to result in increases in peak torso flexion (TF) and time to peak neuromotor response (timePM). Experimentally, this was confirmed with increases in both TF and timePM during vibration and smaller increases in TF and timePM after vibration exposure relative to before vibration. For both TF and timePM, the increases observed during vibration were statistically significant. After vibration exposure, although there was a significant increase in TF relative to before vibration, only a trend ($p=0.09$) was found of increase in the timePM. These increase in TF and timePM during vibration exposure confirm the model predictions. These results also suggest that the neuromotor response does play a role in the dynamic performance of the spine. If the neuromotor response was not a major component of the dynamic response, one would not expect to see changes in the torso flexion (TF) with vibration.

In the model, the effect of an increasing threshold was assessed both while maintaining a constant, linear gain (G) and while maintaining a constant magnitude of neuromotor response. With a constant, linear gain, the magnitude of the neuromotor response would be expected to decrease with increasing detection threshold. Experimentally the magnitude of the neuromotor response was found to increase slightly but not significantly. This suggests that rather than a constant, linear gain, a non-linear or compensatory gain is a more appropriate model of the neuromotor response in this experiment.

In the experiment preparatory values such as the preparatory muscle activity and the preparatory torso flexion were not found to change significantly with vibration.

These preparatory values represent elements of the initial trunk stiffness (K). While a subject might increase the preparatory muscle activity in order to stiffen the trunk and compensate for changes in the neuromotor response, such a compensation was not observed in this experiment.

While the model created for this study is anatomically simple, such models have proven successful in the past for modeling unexpected sudden loading and trunk dynamics [18, 37, 38]. Maintaining simplicity in the trunk model allows for a simpler representation of the neuromotor response. This neuromotor response can be seen as a composite of a number of possible reflex loops and sensory feedbacks. While it is impossible to identify specific neuromotor pathways with this model, the experimental results emphasize the role of muscle spindle organs of the paraspinal musculature in the sensory feedback.

This research demonstrates not only the role of the neuromotor system in dynamic response of the trunk and lumbar spine, but also the role of the paraspinal muscle spindle organs. The muscle spindle organs are generally thought to be primary proprioceptive elements in joints within the normal range of motion and loading [39, 40]. In the spine, the paraspinal musculature is believed to be important in generating extension moments around the spine as well as in stabilizing the lumbar spine. This group of muscles would include a number of muscles including the erector spinae and multifidus muscles.

Vibration applied to the paraspinal musculature should affect all of these muscles. Future work should examine the individual roles of these muscles. A number of other muscle groups may also contribute to low back stability including the rectus abdominus, the internal oblique, the external oblique and the transversus abdominus muscles. These

muscles may also contribute to proprioceptive sensing in the trunk and should also be investigated.

The effect of vibration on dynamic trunk motion is also intriguing. While this experiment focused on vibration applied locally to the paraspinal musculature, it may have implications for occupational vibration exposure such as whole body vibration in truck drivers. Whole body vibration has been documented as a risk factor for low back injuries in a number of occupations. The observed changes in dynamic performance of the spine in this experiment are similar to those observed in whole body vibration by Wilder et al. and by Li and Wilson [15, 17]. This suggests that vibration-induced effects in the muscle spindle organs may play a role in the mechanism of low back injuries. Future work should examine more closely the role proprioception may play in vibration-induced low back injuries.

Direct measurement of proprioception in the low back can be challenging as most of these measures have high variability [24, 41-45]. In subjects with low back pain, these measurements can be confounded by the sensory feedback of pain. A number of studies have identified increased proprioceptive errors in subjects with a history of low back pain [24, 44, 45]. Other studies, however, have not observed these increases [41-43]. Studies examining sudden loading response times have observed increased delays in subjects with a history of low back pain [11, 46]. Increases in time to peak neuromotor response have also been identified as a predictor of low back injury [6]. If proprioceptive errors are indeed increased in subjects with a history of low back pain, the model presented here provides a possible reason why increases in neuromotor response delays are also been observed in similar subjects. It also suggests a mechanism for injury, namely that such

increased errors lead to delayed responses and increased deflection in response to an unexpected load. Further research is necessary to demonstrate this potential link.

In conclusion, modeling of the dynamic response of the human torso to sudden loading suggests that changes in the sensory threshold for neuromotor response may have deleterious effects on dynamic trunk stability including increased delays in neuromotor response and increased torso flexion in response to a sudden, unexpected perturbation. These changes were also observed experimentally by applying local vibration to the paraspinal musculature. These results indicate that the neuromotor response and, in particular, the muscle spindle organs of the paraspinal musculature play a role in maintaining trunk stability and in responding to unexpected perturbations.

Publications as a direct result of NIOSH grant

Arashanapalli M, Wilson SE: [2008] “Paraspinal Muscle Vibration Alters Dynamic Stabilization in the Trunk”, Journal of Biomechanical Engineering, 130(2): 021001.

Wilson SE, Soltys JS: [2008] “Vibration and the Sensory System”, Chapter 7 in “Biomedical Applications of Vibration and Acoustics in Therapy, Bioeffects and Modeling”, (eds. Al-Jumaily A), ASME Press, pp 153-163.

Wilson SE, Li L and Arashanapalli M: [2006] “Neuromotor Habituation as a Mechanism for Vibration Induced Low Back Pain”, National Institute of Occupational Safety and Health, 1st American Conference on Human Vibration, Morgantown, WV.

Wilson SE: [2006] “Occupational Vibration Alters Neuromuscular Response in the Low Back”, National Occupational Research Agenda (NORA) Symposium 2006: Research Makes a Difference, Washington, DC.

Wilson SE, Li L: [2005] “The Sensorimotor System in Dynamic Feedback Models of Trunk Dynamics”, ASME Summer Bioengineering Conference, Vail, CO.

Arashanapalli M, Wilson SE: [2005] “Vibration Alters Proprioception and Dynamic Low Back Stability”, XXth Congress of the International Society of Biomechanics and 29th Annual Meeting of the American Society of Biomechanics, Cleveland, OH, p. 254.

Inclusion of Gender and Minority Subjects

Overall we had a fair distribution across race and gender. Women were slightly under-represented at 38% of the subject population. However, no gender differences were observed in the final results. Across race, Caucasians, Hispanics and African-Americans were under-represented and Asians were over-represented relative to local population demographics. We were able to recruit a Native American and a Bi-racial subject. No effects were observed in the data as a function of race, but the numbers are probably too small for any race to get statistically significant results for racial differences. In future studies we will need to examine better methods for recruiting Hispanic and African-American subjects.

TOTAL ENROLLMENT REPORT: Number of Subjects Enrolled to Date (Cumulative) by Ethnicity and Race				
Ethnic Category	Sex/Gender			
	Females	Males	Unknown or Not Reported	Total
Hispanic or Latino	0	0	0	0 **
Not Hispanic or Latino	8	13	0	21
Unknown (individuals not reporting ethnicity)	0	0	0	0
Ethnic Category: Total of All Subjects*	8	13	0	21 *
Racial Categories				
American Indian/Alaska Native	0	1	0	1
Asian	3	6	0	9
Native Hawaiian or Other Pacific Islander	0	0	0	0
Black or African American	0	0	0	0
White	5	5	0	10
More Than One Race	0	1	0	1
Unknown or Not Reported	0	0	0	0
Racial Categories: Total of All Subjects*	8	13	0	21 *

HISPANIC ENROLLMENT REPORT: Number of Hispanics or Latinos Enrolled to Date (Cumulative)				
Racial Categories	Females	Males	Unknown or Not Reported	Total
American Indian or Alaska Native	0	0	0	0
Asian	0	0	0	0
Native Hawaiian or Other Pacific Islander	0	0	0	0
Black or African American	0	0	0	0
White	0	0	0	0
More Than One Race	0	0	0	0
Unknown or Not Reported	0	0	0	0
Racial Categories: Total of Hispanics or Latinos**	0	0	0	0 **

Inclusion of Children

Since this study examined the effects of a workplace exposure, which would affect working aged populations (18-65 years), subjects under the age of 18 were not recruited for this study.

Materials Available for Other Investigators

The model created in MATLAB for this work and the raw experimental data are available upon request within the limitations of HIPAA regulations.

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