WHOLE BODY VIBRATION AND NEUROMUSCULAR RESPONSE

By

Pradeep M. Abraham

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Sara Wilson, Chairperson

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Terry N. Faddis, Committee Member

_____

Elizabeth A. Friis, Committee Member

Date Defended: _____

The Thesis Committee for Pradeep M. Abraham certifies
That this is the approved Version of the following thesis

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_____

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Abstract

Whole body vibration is considered to be a significant risk factor for low back and other related musculoskeletal disorders, resulting in substantial financial costs to society and loss in quality of life. Both direct and indirect effects of vibration leading to pathology have been identified for the vibrating human. Vibration-induced neuromotor activation has been suggested as an indirect mechanism for increased injury risk by altering low back stabilization and dynamic response. The transmission of vibration through the neuromuscular system was investigated. Neuromotor transmission was defined as the contribution of vibration-induced lumbar motions to paraspinal muscle activity. A transmission function was quantified for a frequency range of 3-20 Hz at three different vibration magnitudes. A double peaked pattern was seen in this transmission with a peak between 4-6 Hz and another peak at 10 Hz. The latter peak may correspond with the internal resonance of the neuromuscular system.

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1. Introduction

1.1 Low back disorder on a global scale

Low back disorders (LBDs) are widespread, resulting in substantial financial costs to society and a loss in quality of life. LBDs and related musculoskeletal disorders (MSDs) constitute a major proportion of all registered and compensable work-related diseases in many countries, representing a third or more of all registered occupational diseases in North America, the Nordic countries, and Japan [Punnett *et al.* (2005)]. In Canada, Finland, and the United States, more people are disabled from working as a result of MSDs, especially back pain, than from any other group of diseases [Badley *et al.* (1994); Battie *et al.* (1997); Bernard (1997)]. The Pan American Health Organization identified LBD as one of the top three occupational health problems within the WHO Region of the Americas [Choi *et al.* (2001)]. It is predicted to be the leading cause of disability in the working population under the age of 45 and comprises approximately 45% of all compensation claims in the United States [Webster *et al.* (1990); Frymoyer *et al.* (1991); Guo *et al.* (1995); Maniadakis *et al.* (2000); Lu (2003)]. One third of the North American population (33 %) is estimated to be at risk for developing LBDs [Walsh *et al.* (1992); Papageorgiou *et al.* (1995)]. A higher prevalence of 38% is observed in Great Britain. A study conducted on Swedish citizens revealed a high risk factor (OR of 2.1 with a 95% confidence interval) for developing LBD in 2872 Swedish men and women [Saraste *et al.* (1987)].

Regional differences have been reported in the percentage of the population reporting LBDs [Reinecke *et al.* (2002.)]. Such regional differences are primarily driven by the labor force participation rate and the proportion of occupations that pose LBD risk factors in any specific part of the world. LBD has manifested as a more serious health hazard in industrialized nations affecting more than one quarter of the industrialized working population annually. Definitively, a large percentage of LBD claims persist for durations lasting more than 90 work days in industrialized regions [Murphy *et al.* (1999)]. The lifetime prevalence of LBD in industrial populations is estimated to be about 60-80% [Hartvigsen *et al.* (2000); Lee *et al.* (2001)]. A greater interaction with industrial equipment that facilitate awkward postures, prolonged seating, unfavorable equipment interaction and vibration exposures in technologically advanced nations has been suggested as a possible factor [Reinecke *et al.* (2002.)].

Significant gender differences were detected in personnel reporting LBD from occupational exposure. LBD risk for men was higher than for women, largely because of a higher participation rate in the labor force for men. Higher rates of participation in manual labor, occupations with heavy lifting and whole-body vibration were noted in men [Punnett *et al.* (2005)]. For instance, the number of cases of LBD attributable to whole body vibration exposure was estimated to be about 444,000 in men as compared to 95,000 in women in Great Britain alone [Palmer *et al.* (2003)].

1.2 Whole body vibration as a contributing factor to LBD

The major thrust of LBD research in the past two decades has been to identify specific occupational risk factors associated with its presence and occurrence [NIOSH (1997)]. The primary motivation stems from the significant impact it could possibly have on workman's compensation issues and the constant need to obtain information for devising better preventive measures. Whole body vibration (WBV) has been identified as such, a risk factor for LBDs. A number of researchers have investigated this risk factor through both epidemiological and biomechanical studies.

There is strong epidemiological evidence that occupational WBV that exceeds exposure limits can contribute to an increased risk of LBD and other related disorders such as sciatic pain, degenerative changes in the spinal system and intervertebral disc disorders [Hulshof *et al.* (1987); Bernard (1997); Bovenzi *et al.* (1999)]. A comprehensive review of studies by Bernard (1997) has suggested a positive relationship between low back disorders and WBV exposure with the incidence of LBDs to increase by 1.2 to 39.5 fold. Another epidemiological review has identified WBV doses encountered in most industrial vehicles in Europe to exceed exposure limits (8 hour vibration duration, 0.5 ms^{-2} vibration magnitude) proposed by the European Union Directive for physical agents [Council of the European Union (1994)]. Eight and a half million men and women are being exposed on a weekly basis to occupational WBV with 370,000 workers exceeding the proposed British Standard Action Level for the estimated vibration dose value [Palmer *et al.* (2003)].

Epidemiological studies have identified several confounding factors that affect the relation of WBV exposure to LBD development. Such studies have identified WBV as a primary LBD factor among others such as heavy or frequent lifting, heavy physical work, prolonged sitting, non-neutral postures (that include trunk rotation, flexion etc), pushing/pulling and impact loads [Burdorf *et al.* (1990); Liira *et al.* (1996); Levangie (1999); Johanning (2000); Lee *et al.* (2001); Hartvigsen *et al.* (2003); Kopec *et al.* (2004)]. Other confounding factors for WBV exposure that have been included are worker's age, duration of exposure, history of LBD and previous exposure [Seidel *et al.* (1986); Bongers *et al.* (1990); Boshuizen *et al.* (1992); Bovenzi *et al.* (1992); Ozkaya *et al.* (1994)]. Liira *et al.* (1996) found a higher risk factor for long term LBDs in blue-collar workers primarily from WBV, where sex and smoking history were identified as the confounding factors.

The contribution of WBV exposure to the onset and development of LBD is hard to separate from other confounding ergonomic risk factors mentioned above. Typical occupational settings present a combination of these factors to occupational workers. The effect of a single factor to LBD can be quite different than a combination with any other factor. For instance, a combination of prolonged sitting combined with vibration exposure can affect the worker differently than the presence of prolonged sitting or vibration exposure exclusively. The evidence for such variations is detected in several epidemiological studies that show higher LBD prevalence for combinatorial factors such as sitting and WBV exposure or sitting and awkward postures [Bongers *et al.* (1990); Burdorf *et al.* (1990); Boshuizen *et al.*

(1992); Hartvigsen *et al.* (2000); Lee *et al.* (2001); Chen *et al.* (2004)]. Definitively, higher risks of LBD and sciatica have been reported in occupations with WBV exposure in seated postures typical of occupations such as professional driving [Bongers *et al.* (1990); Burdorf *et al.* (1990); Boshuizen *et al.* (1992); Hartvigsen *et al.* (2000); Lee *et al.* (2001); Chen *et al.* (2004)]. Palmer *et al.* (2003) identified increased LBD risk in occupational activities that involved lifting and WBV exposure than the presence of lifting alone.

Identifying a standardized vibration dose measure is essential to quantify the increased susceptibility to developing LBDs from WBV exposure. The vibration dose value provides a convenient measure for assessing the total severity of vibration on human health [Griffin (1990)]. International Standard ISO 2631 has identified vibration dose as a parameter with multipronged dependence on vibration frequency, magnitude and duration to estimate the effects of vibration on comfort, performance and health. According to ISO 2631, vibration dose can be defined as the effect of a frequency weighted acceleration over specific durations [Griffin (1990)]. Weighting factors are used to dictate higher weighting values for the frequencies of higher importance (resonant frequencies) while calculating acceleration values. Mathematically, this is achieved by obtaining the integral of the fourth power of frequency-weighted acceleration over the time period of exposure and is given by the following expression.

Dose value = $\int_{t=0}^{t=T} a^4(t)dt \quad (Units: m^4 s^{-7})$ **Equation 1**

In this expression, a (t) represents the frequency weighted acceleration and T denotes the duration of vibration. This dose-response relationship can be non-linear depending on vibration magnitude, direction and several of the confounding factors indicated above.

Efforts have been made to quantify the effect of vibration magnitude, frequency, duration and other confounding factors on LBD in terms of the dose-response relationship. Some studies have examined the direct correlations of vibration magnitude and duration on vibration dose value and LBD prevalence [Boshuizen *et al.* (1990); Bovenzi *et al.* (1992); Bovenzi *et al.* (1994)]. In terms of dose value, duration of vibration exposure suggested a stronger association to LBD while vibration magnitude suggested a stronger association to sciatica. Robb *et al.* (2007) identified confounding factors such as manual handling and seat discomfort in truck drivers that affect the dose-response relationship. However, Palmer *et al.* (2003) identified modest excesses of LBD and sciatica with exposure to WBV in men after allowance for other confounding factors such as physical activity level, age and psychological risk factors with no consistent relation to vibration dose.

Epidemiological studies have identified specific occupations where high vibration exposure levels are encountered. Crane operators, bus drivers, tractor drivers, fork-lift truck drivers, helicopter pilots and taxi drivers were the most frequently investigated occupational groups in either cross-sectional or cohort studies [Bovenzi *et al.* (1999)]. The occupational group that was identified to have the

strongest association between LBD and vibration dose was the helicopter pilot (OR=6.6, 95% CI, 2.9–15.1) after adjusting for awkward postures [Bongers *et al.* (1990)]. Occupational groups that followed were tractor drivers (OR=2.8, 95% CI, 1.64-5), truck drivers (OR=1.96, 95% CI, 1.03-3.7), bus drivers (OR=1.76, 95% CI, 0.86-3.58) and taxi drivers for the same dose values. Bovenzi *et al.* (1994) have associated WBV exposure from prolonged tractor driving to chronic LBD and extended sick leave in tractor drivers. Robb *et al.* (2007) has shown a greater LBD prevalence exceeding 12 months from WBV exposure in a sample of truck drivers exposed to WBV as compared to controls.

Biomechanical studies have been conventionally used as an alternative approach to quantify WBV exposure, the dose-response relationship and its relation to LBD. While examining dose response is important, Lings *et al.* (2000) concluded that the strict isolation of a dose-response relationship to WBV is hard to achieve through epidemiological studies alone and might require data from biomechanical studies. In biomechanical studies, raw data is obtained in laboratory conditions using human subjects or animal models and the effects of vibration and injury risk on biological tissue are estimated with analysis of such data [Mansfield (2005)]. However, vibration exposures in such studies are not typical of lifetime vibration exposure and the experiments involve smaller populations that might not represent the population at risk very well.

1.3 Direct and Indirect effects:

In spite of strong epidemiological evidence for WBV induced LBD, the etiology of low back injuries and related MSDs has not yet been fully clarified [Lings *et al.* (2000)]. Biodynamic and physiological experiments have suggested that seated WBV exposure can lead to LBDs by several direct as well as indirect mechanisms. These mechanisms of injury could support the epidemiological findings of Bovenzi *et al.* (1999) by suggesting a possible causal role for WBV in the development of LBDs and are elaborated in this section.

1.3.1 Direct effects:

Mechanical creep: Spinal height losses have occurred from increased mechanical loading with WBV that forces fluid from the intervertebral discs [Magnusson *et al.* (1992)]. Pope *et al.* (1998) devised an experimental protocol that used a linear variable displacement transducer (LVDT) enabled stadiometer to clearly differentiate spinal shrinkage from posture change and in vivo creep. Vertebral shrinkage was observed for a 5 Hz, 0.1g RMS acceleration WBV setting ($p<0.03$) on 12 female subjects as compared to static sitting. Corrections for posture changes adopted by the subject during exposure still contributed significant shrinkage ($p<0.05$).

Mechanical fatigue: Vibration exposure has been associated with lumbar disc rupture from cyclic loading in addition to activities such as frequent bending and twisting

[Stokes *et al.* (2004)]. Adams *et al.* (1983) found distortions in the lamellae of the annulus fibrosus in lumbar vertebral discs subjected to cyclic loading. The cyclic loads that were simulated include compression loads and bending loads from spinal flexion-extension motions. Stokes *et al.* (2004) suggested that disc generation from cyclic loading could occur in two related pathways: 1) by direct microscopic damage that accumulates with repeated cycles 2) alterations (remodeling) in the material properties of the disc components that may weaken the disc. They further propose that relatively modest magnitudes of such loading patterns could be sufficient to cause disc herniations in the long run.

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Cellular and metabolic effects: Hirano *et al.* (1988) have identified vibration as a stimulus for proteoglycan and collagen production, but as an inhibitor for protein production in intervertebral disc tissue. This could impact cellular repair mechanisms of discs subject to fatigue loads. Buckwalter (1995) observed low protein synthesis rates and extracellular disc matrix degeneration at vibration frequencies (4-6 Hz) corresponding to the resonance in the human. Adams *et al.* (1983) suggests that such repair mechanisms may be effective over longer time periods (months, years) than shorter periods (days). Kamenskii Iu *et al.* (1988) examined the neuron-endocrine processes in 22 male subjects exposed to WBV magnitude of 0.6-1.4 m/s2 for a duration of 1 hour. Blood samples analyzed for lactic acid concentration showed an increase of 25.2 % immediately after exposure and 30.8 % after 30 minutes of rest. Higher lactic acid levels result from increased muscle activity and muscular fatigue.

Adenosine monophosphate levels which are indicative of energy production in the body showed a drop of 50.3 %. This nucleotide is involved in the overall energy production processes of the body and modifies many physiological reactions in response to vibration exposure.

1.3.2 Indirect effects:

Proprioception and spinal stability: Spinal instability can be stated as the loss of the spine's ability to maintain its patterns of displacement under physiologic loads [Li (2006)]. In a clinical sense, spinal stability incorporates the contributions of neural control and muscular dynamics in addition to the ability of the passive tissues of the spine and surrounding ligaments to maintain stability. Indahl *et al.* (1995) hypothesized that the motion and stabilization of the spine are based on a complex reflex activation system in which the proprioceptive nerve endings in the annulus fibrosus of the intervertebral disc, the facet joints, and paraspinal muscles initiate various reflex patterns. These reflexes are modulated by different interneurons, which receive input from muscle spindles, and by higher levels of the central nervous system. Consequently, accurate sensory input from the muscle spindles is key for proper stabilization of the spine.

Biomechanical studies have shown that vibration of the musculature can result in loss of proprioceptive information and increase spinal instability. Experimental measures of the upper extremity demonstrated that exposure to muscle vibration at frequencies between 10 Hz and 120 Hz can result in illusory movements and altered proprioception [Roll *et al.* (1982); Cordo *et al.* (1995)]. A perception of increased

length in a vibrated muscle can occur [Goodwin *et al.* (1972); Roll *et al.* (1982); Inglis *et al.* (1991)]. The specific role of paraspinal muscle spindles in lumbosacral position sense has been investigated [Brumagne *et al.* (2000)]. Brumagne suggested that vibration induced a lengthening illusion of the multifidus muscle, which was compensated for by a more lordotic spinal position. A significant increase in directional error was present during vibration of the paraspinal muscles. Wilson *et al.* (2006) investigated the effects of occupational vibration exposure on position sense and sudden loading dynamics. Position sense and sudden loading measures were performed before vibration exposure, and at selected time periods after vibration exposure. Increased absolute reposition errors were observed which indicated a loss of proprioceptive information and impaired ability in sensing torso position. Response time to a sudden impact load being applied to the upper torso was increased suggesting altered spinal instability. This was accompanied by an increase in torso flexion and lumbar curvature deflection. Increased response times and increases in torso flexion and lumbar curvature deflection are all indicative of a reduction in spinal stability.

Wilson *et al.* (2006) has described a model that represents the reflex response of the trunk musculature to torso motion (lumbar rotation) induced by any perturbations. The trunk was modeled as a simple linearized inverted pendulum (figure 1). The neuromotor response to perturbation was modeled as a gain that could be subdivided into a neuromotor gain, time delay and detection threshold. The neuromotor gain was defined as a parameter that quantifies the amount of muscle

activity elicited for a given trunk motion and a time delay was introduced to account for conduction delays. A detection threshold was defined further to represent a minimum value of detectable torso motion to which the reflex loops respond. For a perturbation input, it was found that an increased threshold (suggestive of losses in proprioception) led to greater delays in muscle response, as a greater deflection is needed to activate the neuromotor response. The researchers propose that such increased time delays for trunk muscle activation could impair trunk stiffness required for torso stabilization.

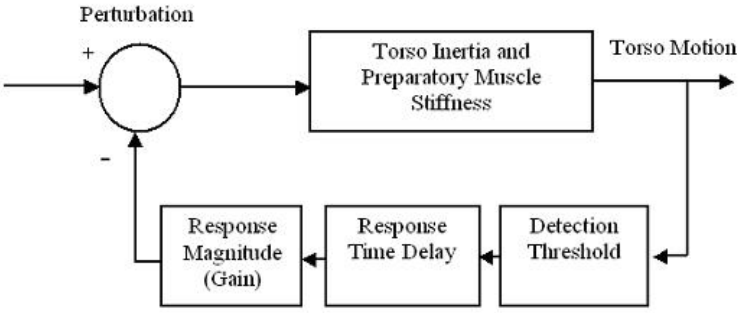

Figure 1: Wilson model

Muscular fatigue: Median frequencies of electromyographic signals (EMG) measured from the back muscle (Erector Spinae, Obliques) are an indicator of muscle fatigue [El Falou *et al.* (2003); Li *et al.* (2003); De Oliveira *et al.* (2004)]. A lower median frequency resulting from WBV exposure could indicate muscle fatigue. However, studies on fatigue of the paraspinal muscles exposed to WBV have had conflicting results. De Oliveira *et al.* (2004) observed no significant lower median frequencies of

EMG for 12 helicopter pilots subjected to WBV for flight duration of 2 hours. Li *et al.* (2003) observed median frequency shifts in subjects exposed to WBV in a simulated driving environment for an exposure time of 90 minutes at vibration frequencies of 1.8, 4 and 6 Hz. A higher shift in median frequency was observed at 4 Hz indicating that the muscle was more susceptible to fatigue due to a possible human resonance at this frequency. El Falou *et al.* (2003) exposed subjects to WBV for an extended duration (150 minutes) of seated driving for different types of seats. While using EMG median frequency as the fatigue index, no significance for median frequency change was noted, though subject discomfort increased significantly ($p<0.05$). Posture of the subject seems to play a major role in back fatigue where postures with backrest support fatiguing much less than upright postures without a backrest [Wilder *et al.* (1994)]. Zimmermann *et al.* (1993) noted that posture has a crucial effect on back muscle fatigue after a earlier onset of fatigue of ES muscle groups was noted in an anterior lean posture as compared to neutral or posterior lean postures. In this study, the mean EMG activity measured at different postures was used as a fatigue index. Postures exhibiting higher mean EMG activity suggested earlier onset of muscle fatigue.

1.4 WBV transmissibility

For the direct or indirect effects of WBV (mentioned above) to occur, vibration must be transmitted from a vibration source to the low back. Vibration in the workplace can have a varied effect depending on the delivery of the vibration, the transmissibility of the vibrating human and the neuromotor system susceptibility [Griffin (1990)]. The mode of vibration delivery can be through a vibrating seat pan (with or without backrest), a vibrating backrest, or a vibrating handgrip or a combination of the above. The direction of vibration could be vertical, horizontal, lateral, rotational or multidirectional and can vary in frequency and magnitude depending on the vehicle dynamics. Each combination of mode, frequency and direction may have different effects on the bony structures, low back musculature and sensory system.

Conventionally, vibration transmission measurements have been made by mounting accelerometers at bony locations at a site of interest on the seated human. Input accelerations from the seat are compared to the measured output accelerations to get an estimate of the transmissibility. This transmission is defined as the ratio of the output acceleration of the body segment of interest (head, trunk, limbs, etc) to the input acceleration from a vibrating seat or local vibrator and can be represented by the following transfer function.

$$Trans(f) = \frac{acceleration_{bodysegment}(f)}{acceleration_{input}(f)} \qquad \textbf{Equation 2}$$

The output to input ratio of the acceleration magnitude represents the magnitude of this transfer-function. Phase information associated with this function of transmissibility is simply the measured time lag between the output and input signals of interest.

Experimental studies have demonstrated consistency in the trends for the seated response of human body exposed to vertical WBV [Griffin (1990)]. Specific frequencies of resonance of the vibrating human have been identified. The resonant frequency is defined as the frequency at which an object will freely vibrate after it has been stuck mechanically. At resonant frequencies, the transmissibility function described in Equation 2 shows a distinct peak followed by decreases with increasing frequencies.

Transmission to the bony structures of the low back of the seated human has been studied extensively for vertical seatpan vibrations. A principal resonance has been found in the frequency range 4-6 Hz for the vibrating human exposed to WBV, resulting in large amplitude motions of the bony structures in the low back relative to the seat [Coermann (1962); Fairley *et al.* (1989)]. Different hypotheses have been proposed to explain the biomechanical mechanisms underlying the resonance phenomenon in humans. A bending motion of the lumbar spine at the principal resonance has been observed and attributed to the pitching motion of the pelvis [Sandover (1962)]. Later, Seidel *et al.* (1986) hypothesized that the principal resonance was a combination of vertical motion of the entire body and bending

motion of the lumbar spine. A drop in motion of approximately 2 amplitude decades per frequency decade is observed above 6 Hz with a secondary resonance often observed at 10-12 Hz [Griffin (1990)]. Other studies have reported a wider range for this second principal resonance between 8 Hz to 14 Hz [Coermann (1962); Fairley *et al.* (1989); Pope (1992)], but there exists large variability between different studies and subjects. This response may also correspond to vertical motion of the spinal column or represent a bending motion of the upper torso with repect to the lumbar spine [Hagen (1985); Pope *et al.* (1992)].

Investigators have shown a preference to measuring WBV transmission from the vibrating seat pan to the head for several reasons. The head, being a vital part of the body is susceptible to higher levels of vibration discomfort and movements that affect vision [Griffin (1990)]. In addition, head acceleration without skin motion artifacts is easy to measure by mounting accelerometers on a bite bar. Since the teeth are rigidly embedded in the skull, motion artifacts can be minimized in most frequency ranges that are measured Mansfield (2005), which is not necessarily the case for skin mounted accelerometers. Paddan *et al.* (1988) obtained a 5 Hz principal resonance peak in performing 12 repeated measures of the transmission of z-axis seat vibration to three axes of head vibration (roll, pitch, yaw) for 12 male subjects. The protocol used a vibration frequency range of 0.2-31.5 Hz and 1.75 m/s^2 RMS magnitude and the subjects assumed a comfortable upright posture, without a backrest and a moving footrest. For horizontal vibration mode, without the backrest, transmissibility for the fore-and-aft, vertical and pitch axes of the head were greatest

at about 2 Hz. The backrest greatly increased transmission at frequencies above 4 Hz and caused a second peak in the transmissibility curves at about 6 to 8 Hz. Lateral seat vibration mainly caused lateral head motion with a maximum transmissibility at about 2 Hz. The backrest had little effect on the transmission of lateral vibration to the head.

In certain studies, a 'softening effect' has been known to occur with increasing magnitudes of vibration for the same range of vibration frequencies [Griffin (1990)]. This 'softening effect' is observed as a nonlinear shift in the resonant peak towards lower frequencies as the magnitude is increased [Mansfield *et al.* (2000)]. Mansfield *et al.* 2000 measured this non-linear shift in twelve subjects exposed to vertical random vibration with for a frequency range of 0.2-20 Hz. A reduction in the resonant frequency from 5.4 to 4.2 Hz was observed as the vibration magnitude was increased from 0.25-2.5 m/s^2 for the same frequency range indicating a non-linear shift. However, this effect has not been observed in other studies [Panjabi *et al.* (1986); Broman *et al.* (1991); Pope *et al.* (1998)]. Recently, Mansfield *et al.* (2006) investigated this discrepancy further and measured trunk muscle pre-tension levels in 12 subjects exposed to random vibration at vibration magnitudes of 0.5, 1 and 1.5 RMS ms^{-2}. The softening effect was not apparent in cases where subjects pre-tensed the torso musculature as compared to subjects that exhibited less muscle pre-tension.

WBV transmission to the spine has been measured through invasive and non-invasive experimental protocols. However, a drawback to measuring transmission to

this region is the presence of skin motion artifacts when accelerometers are mounted on the skin above the spinous processes [Mansfield (2005)]. Other spinal transmission artifacts could include changes in the spine-accelerometer configuration such as *in vivo* creep [Magnusson *et al.* (1992)]. A few studies have attempted to eliminate such artifacts by attaching accelerometers directly to the spinous processes invasively [Panjabi *et al.* (1986); Pope *et al.* (1993)]. Zimmermann *et al.* (1997) have shown vertical transmission to the T-5 spinous process to have a principal resonance peak at 4.5-6 Hz. Accelerometers were mounted on the skin non-invasively for a frequency range of 4.5-16 Hz. Pope *et al*. 1993 conducted another invasive study with accelerometers placed at the L3 vertebra level observing a mechanical resonance from 4-5 Hz. Mansfield *et al.* (2000)observed seat to vertical motion at the spine with a primary resonant peak at around 4 Hz with a transmissibility magnitude of 1.5-1.8. A second resonant peak was observed at approximately 8-10 Hz. Panjabi *et al.* (1986) measured axial, horizontal and rotary accelerations in the sagittal plane for each vertebra and the sacrum. In this study, accelerometers directly attached to the spinous processes (*in vivo*) for a test frequency range of 2 to 15 Hz. A principal resonance in the vertical direction of an average of 4.4 Hz was recorded with no pronounced peaks observable in horizontal and rotational modes. The resonance frequencies tended to remain the same when measured from the first to the third lumbar vertebrae (L1-L3). However the resonance frequency of the sacrum was 16 to 18 percent higher than the lumbar vertebrae indicating that vibration transmission is variable along spinal vertebrae.

Other locations that transmissibility has been measured to include the upper and lower abdominal walls [Mansfield *et al.* (2000)]. For the lower abdominal wall, a resonance was observed for both horizontal and vertical vibration modes at approximately 6 Hz. For the upper abdominal wall (approx. 20 mm above the navel) a resonance was noted between 6-8 Hz. Researchers have investigated inter-subject variability, variations due to posture, backrest effects and foot-rest effects in measuring vibration transmission to the seated human with accelerometers mounted at various locations [Griffin (1990)]. Though most studies have been conducted in laboratory conditions, a few studies have measured transmissibility in real transport environments [Walsh (1966); Griffin (1972); Paddan (1985)]. Walsh (1966) measured vibration transmission in railroads. Griffin (1972) measured transmission in military helicopters and Paddan 1985 measured transmission to the head in military tanks.

In summary, the seated human's principal resonance response to vertical whole body vibration occurs around 5 Hz with the transmission value reaching the highest at this frequency. Panjabi *et al.* (1986) suggests that many operating motor vehicles have vibratory frequencies in this particular range are can serve as a potential source of injury to the spinal column. A secondary peak observed between 8-12 Hz might correlate to a bending motion (flexion-extension) of the spine. In design of machinery, it is advisable to avoid exposure at these resonance frequencies.

1.5 WBV and back motion

Vertical vibration of the seat pan has also been shown to result in a rotation (in the mid-sagital plane) and fore-aft translation of the head [Griffin (1990)]. Seidel *et al.* (1988) has measured accelerations in two dimensions of lumbar spinous processes have shown that spinal motion in response to vertical sinusoidal input acceleration includes both vertical with angular motions. On examining the motion of the spinal motion response to vertical sinusoidal acceleration at 4.5 Hz and 8 Hz, a flexion motion of the spine coinciding with the upward seat acceleration and an extension motion corresponding with the downward seat acceleration is clearly evident.

These cyclic flexion-extension motions (angular motions) of the spine have been observed to decrease with increasing frequency. Smaller magnitudes of back rotations were observed at 8 Hz than at 4.5 Hz. This frequency dependence has also been noted by Zimmermann *et al.* (1997), who examined whole body vibration induced pelvic and back motion for a frequencies of 4.5, 5, 6, 8, 10, 12 and 16 Hz at an magnitude of 1 RMS ms^{-2}. When group mean pelvic motion ensemble averages were inspected, much greater pelvic motions were observed at frequencies of 6 Hz and lower than at the 6-14 Hz range. The greater pelvic motion observed at the frequency range below 6 Hz directly correlates with a greater trunk acceleration transmissibility observed in that frequency range indicating that the principle resonance of the human exposed to WBV also results in an increase in back flexion-

extension motions. This motion has been identified with the second resonant peak at 8-12 Hz as mentioned above [Pope *et al.* (1992)].

Torso and head rotation may be affected by subject posture and trunk stiffness. Keegan (1953) has described certain seated postures where the trunk's line of gravity falls posterior to the ischial tuberosities and anterior to the flexed lumbar spine. This results in a trunk mass moment arm that produces increased posterior pelvic rotation and lumbar spine flexion. Zimmerman *et al*. 1997 examined pelvic and back motions for three different postures, neutral upright, anterior and posterior pelvic tilt with respect to neutral. Higher back motion was noticed in postures where the trunk is posterior with respect to a neutral upright posture as compared to anterior trunk postures.

1.6 WBV and muscle activity

Muscle activity has been measured in biomechanical studies primarily through electromyographic (EMG) techniques [De Luca (2003)]. Seroussi *et al.* (1989) have hypothesized that the back musculature exhibits cyclic muscle activity that is synchronous with vibration exposure. Zimmermann *et al.* (1997) describes this vibration synchronous response (VSR) to comprise of 1) tonic and 2) phasic activities. Tonic activity refers to the mean baseline EMG activity that represents the overall trunk muscle activation required to maintain a constant posture. Phasic activity represents the peak-to-peak variation of EMG activity in response to a sinusoidal vibration input. In response to WBV exposure, tonic activity is shown to increase with and without the phasic component. Both tonic and phasic muscle response are also affected by muscle preloading and posture [Zimmermann *et al.* (1993)]. Neutral and anterior lean postures present a greater baseline EMG activity and a more prominent VSR.

Sandover *et al*. 1981, Siedel *et al*. 1986 and Serrousi *et al*. 1989 have described simple models to explain VSR response to WBV. Seroussi *et al.* (1989) has proposed a mechanical lever system model with the disc at L3 level acting as a fulcrum (figure 2). In this model, the upper body mass is at the end of the anterior lever arm at a distance (l) from the fulcrum and the Erector Spinae (ES) muscle group acts as a tension element, F_{ES} at the end of the posterior lever arm at a distance d from the fulcrum. To a sinusoidal acceleration applied at the fulcrum [A sin (wt)], the

moment (T_{ES}) about the fulcrum generated by the muscles, due to the inertial forces of the upper body mass is given by the following expression.

$$T_{ES} = dF_{ES} = Ml[g + A\sin(\omega t)] \quad \textbf{Equation 3}$$

This model does not account for antagonistic muscle activity, ligamentous or facet forces, stabilizing effects of abdominal pressurization and additional inertia forces due to rotational responses.

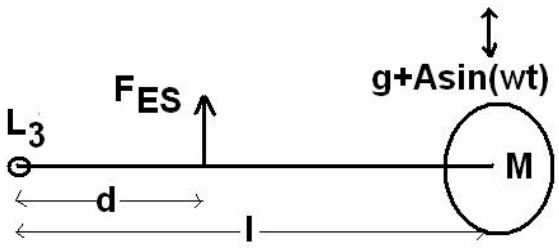

Figure 2: Seroussi Model

Several investigators have studied the effect of vibration frequency on phasic EMG response in vibratory and static settings [Seidel *et al.* (1986); Griffin *et al.* (1989); Seroussi *et al.* (1989)]. Isometric torque calibrations have been used to get an estimate of the tonic (average) and phasic (peak-peak) torque imposed on the spine. Seroussi *et al.* (1989) reported significantly higher paraspinal activity and torque was observed in the vibratory setting as compared to a static setting ($p<0.05$) except at 4 and 10 Hz ($p<0.1$). Phasic torque demonstrated a peak at 4 Hz and declined with increasing frequency for a frequency range of 3-10Hz at 0.1g RMS vibration magnitude. Griffin *et al.* (1989) reported a similar decreased phasic EMG response

with increasing frequency above 4 Hz. Seidel *et al*.1986 observed a similar trend describing a peak phasic EMG from 4-6 Hz and a decline with increasing frequency. A peak torque and EMG activity observed in this studies observed at about ~4-6 Hz is indicative of primary resonance of the human at that frequency range. This peak EMG observed at these frequencies represents the required higher muscle activity needed for meeting the demands for upper body stabilization as proposed by these researchers. Peak to peak muscle torque predicted by Seroussi's model described above was significantly lower than the measured experimental EMG.

Variations in tonic and phasic EMG activity have been investigated for different postures and duration of WBV exposure [Hosea *et al.* (1986); Tarkka (1986)]. Hosea *et al.* (1986) evaluated tonic EMG response of the back musculature when subjects to WBV for a period of 3.5 hours. Duration of exposure posed no change in the magnitude as well as median frequency of the acquired tonic EMG. Further, this measure was found to be a minimum for postures that were posterior with respect to neutral upright. Tarkka (1986) evaluated baseline EMG for three different postures: anterior lean; neutral upright sitting and posterior lean. The anterior lean posture showed the highest ES activity, followed by the neutral upright posture. The posterior lean posture showed minimal ES activity and the abdominal muscles (RA) were active and primarily stabilizing the trunk.

Time delays between the acceleration input and muscle activation has been quantified and a drop in this measure with increasing frequency has been noted [Seidel *et al.* (1986); Seroussi *et al.* (1989); Bluthner *et al.* (2001)]. However,

differences have been noted in these studies in the magnitude of this delay. Seroussi *et al.* (1989) observed a drop in magnitude of delay time (~230 ms to ~150 ms) with increasing frequency from 3-10 Hz. A drop in delay time of 81(+-19) ms (with increasing frequency) was observed over the tested frequency range between peak acceleration and peak EMG. The researchers suggest that the resulting imbalances that occur when inertial forces and muscle force are out of phase can excessively strain the spine and the associated stabilizing elements. Bluthner *et al*. 2001 developed a transfer function to quantify time lag between random WBV input and electromyographic activity of back musculature for 38 healthy male subjects. The targeted muscle groups of significance were the ileocostalis lumborum, pars thoracis, pars lumborum and the lumbar multifidus. For a frequency range of 1 to 9 Hz and 1.4 m/s^2 RMS acceleration, a higher mean lag time of ~65 ms was observed in the frequency range of 1-4 Hz. At higher frequencies (5-9 Hz) the mean response time lag was lower (~20 ms). This study has proposed different reflex mechanisms for lumbar muscle activation to account for the differences in delay times

1.7 Relation between Back flexion extension and EMG

Reflex and voluntary response loops are crucial in the trunk's response to perturbations that may include vibration. However, there is a question of paraspinal muscle being activated by different elements in the neuromuscular system such as the vestibular system and the central nervous system or from voluntary responses. Seidel (1988) claims that muscle response to WBV is influenced more by the stretch reflex than the vestibular system at frequencies above 1.25 Hz.

Vibration induced, lumbar flexion-extension motions could act to stretch the paraspinal musculature by stimulating the spindle organs and result in stretch reflexes. Seroussi *et al.* (1989) have suggested that vibration induced cyclic muscle activity induces a train of stretch reflexes in the paraspinal muscles as a response to muscle vibration. Stretch reflexes are facilitated by stretch receptors called spindle organs located deep within the muscle belly that are sensitive to changes in muscle length [McMahon (1984)]. These receptors experience the same relative length change as the overall muscle. When an activated muscle is stretched by an external agency, the stretch reflex contracts it in a manner such that the original length is regained. This is termed a stretch reflex and a cycle of stretch reflexes could be observed as basic EMG.

Certain WBV frequencies and subject postures could result in a higher magnitude of external stretch of the lumbar musculature (from vibration induced back rotations) and result in a higher stretch reflex magnitude. Zimmerman *et al*. 1997 observed increased ES peak-peak EMG activity in postures adopted by the subject

that resulted in increased pelvic motion when exposed to WBV. This increased EMG activity could be occurring as a result of a greater stretch of the lumbar ES muscle group in such postures that contributes to greater muscle activity. Seroussi *et al.* (1989) and Zimmerman *et al*. 1997 have both observed higher phasic EMG magnitude at frequencies less than 6 Hz where pelvic and back motion was higher.

Trunk muscle activation through reflex activation from lumbar rotations appears to be a component of vibration transmission that occurs exclusively within the neuromuscular system and requires further investigation. As mentioned above, vibration transmission studies have focused extensively on the mechanical transmission of input vibration to different body segments of interest. A few studies have examined paraspinal muscle activity as a response to input vibration from a seat pan. However, no study has isolated and described the transmission of vibration induced back rotations to back muscle activity.

As such, a set of vibration transmission functions can be defined namely,

$TMF1 = \frac{acceleration_{spine}}{acceleration_{seat}}$, $TMF2 = \frac{lumbar\_rotations}{acceleration_{seat}}$, $TMF3 = \frac{muscle\_activity}{acceleration_{seat}}$

and $TMF4 = \frac{lumbar\_rotations}{muscle\_activity}$. A schematic of these transmission functions is shown in figure 3.

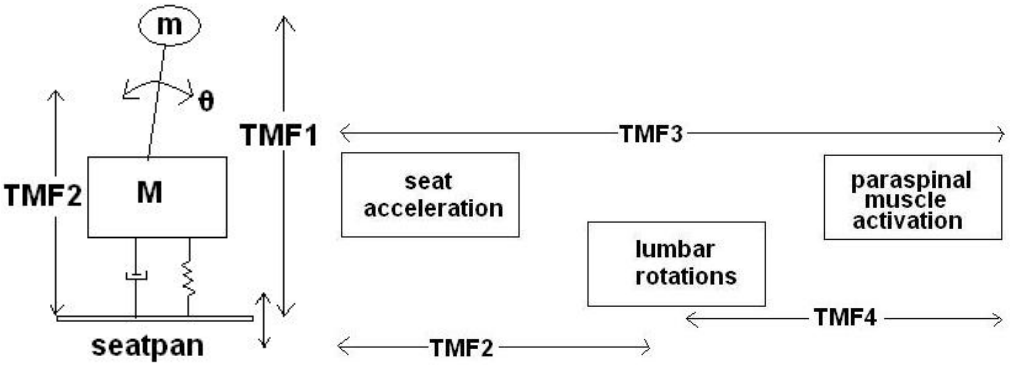

Figure 3: Schematic of transmission functions

1.7.1 Specific aims

The specific aims of this study were to describe the transmission of WBV translated as lumbar rotations through the neuromuscular system to activate paraspinal response (TMF4). Magnitude and phase measures of this transmission function were investigated. In addition, the response of trunk acceleration, lumbar rotations and paraspinal muscle activation to the input acceleration (TMFs 1-3) was measured. Using this data, the characteristic trend observed in TMF4 and the reflex responses that contribute to paraspinal muscle activation in this transmission function were described.

2. Methods

Informed consent (Appendix A) was obtained from 16 healthy adult subjects (8 male, 8 female, mean age 22 years ± 3 (SD), weight 65 ± 6 kg (SD), height 1.62 ± 0.04 m (SD). The experimental protocol for this study was approved by the Human Subjects Committee of the University of Kansas. The subjects were screened for a history of low back pain and other musculoskeletal disorders. To assess physical activity levels that may affect muscle response, the subjects were asked to complete a questionnaire to determine medical history and work experience. Subjects who reported a recent history of back pains were advised not to participate for safety reasons and informed consent was obtained only from subjects who qualified.

2.1 Data acquisition

In this study, measurement of muscle activity was restricted to the use non-invasive surface electromyography (EMG) and a 20-450 Hz (± 10%) EMG bandwidth non-invasive system was used (Bagnoli 8 EMG system, Delsys, Boston, MA). Eight single differential electrodes (preamplifier gain of 10 V/V ±1%) were attached on the skin to the left and right side of four muscle groups of interest, namely the Erector Spinae (ES), Rectus Abdominus (RA), External Obliques (EO) and the Internal Obliques (IO). Placement of electrodes on the muscle groups was based on protocols established by Mirka (1991). In targeting ES muscle activity, electrodes were placed lateral to the L3 spinous process with an inter electrode

spacing of ~ 3-4 cm. Electrodes were placed 1-2 cm superior to the umbilicus with an inter-electrode spacing of ~ 3-4 cm for the RA muscle group. For EO, electrodes were placed lateral to the umbilicus with an inter-electrode spacing of ~ 8-10 cm and oriented at 45 degrees with respect to vertical. For IO, orientation was at 45 degrees with respect to vertical, lateral to the midline of the lumbar triangle with an inter-electrode spacing of 8-10 cm [Mirka (1991)]. Minor differences in electrode spacing between subjects were attributed to differences in subject size. Pre-amplified signals from the electrodes were fed through two 4-channel input modules to a signal amplifier and further amplified at a gain of 1000 prior to acquisition. Usable energy of EMG signals is limited to 0-500 Hz range and is most dominant in the 50-150 Hz range [De Luca (2002)]. For the current application, software enabled reverse Butterworth filters were set up to capture the EMG bandwidth between 30-250 Hz. Notch filters were set up to eliminate contaminations from 60 Hz noise and concurrent multiples. The filtered EMG signal was demeaned by subtracting the mean from the signal (to remove possible DC offsets) and full-wave rectified by determining the absolute magnitude. The signal was further integrated with the use of a 100 pt Hanning window.

Processed EMG signals were normalized (nEMG) with respect to maximal activity exhibited by the subjects for each corresponding muscle group to eliminate inter-subject variability. Three maximal, voluntary, isometric muscular contractions were performed by the subjects in 5-second bursts with the pelvis fixed to a stationary platform. While lying prone, the subjects performed torso lifts against shoulder

restraint to exhibit maximum ES activity. Torso lifts to the sides were performed to target IO and EO muscle groups. For the RA, the subject performed abdominal crunches against a shoulder restraint. The average of the integrated EMG during these three maximal exertions was used to normalize the integrated EMG during the remainder of the experiment.

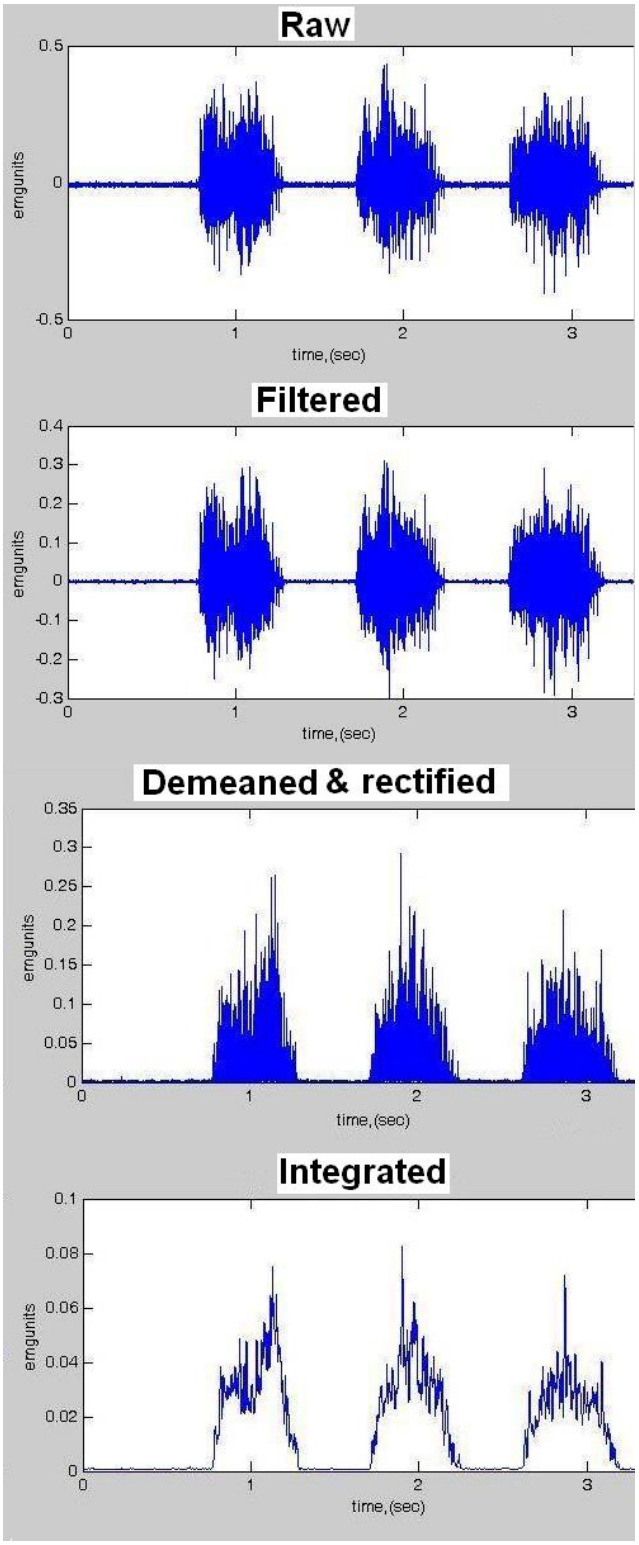

Figure 4 Raw ES EMG for subject 2 was filtered, demeaned, rectified and integrated

Back flexion-extension motions were monitored using a SG 150B twin axis lumbar electrogoniometer (Biometrics Ltd, Gwent, UK). Goniometer placement was such that the two goniometer ends coincided with the T-12 and S1 spinous processes. The subjects were asked to maintain a comfortable upright lumbar posture throughout the experiment by constant visual feedback from a ADU301 angle display unit interfaced with the goniometer. A potential difference of 2.5 V was subtracted from the measured output voltages and multiplied by 90 degrees to get the corresponding angles as per calibration specifications ($4.5V \Leftrightarrow +180^o, 0.5V \Leftrightarrow -180^o, 2.5V \Leftrightarrow 0^o$).

Accelerations from the seat pan and spine were measured with two 356 A17 triaxial accelerometers (PCB Piezotronics Inc., Depew, NY) attached to the seat pan and to the skin over the T10 spinous process. Accelerometer output voltage was converted to acceleration with units in m/s^2 using the sensitivities mentioned in the calibration specifications (Sensitivities, $\left(\frac{mV}{m/s^2}\right)$: x-axis: 52.7, 50.6 y-axis: 53.4, 50.1 z-axis: 51.5, 51.8). Acceleration signals were converted to units in m/s^2 by multiplying the output signal (in millivolts) by the sensitivity factors mentioned above. The raw goniometer and acceleration signals were filtered with a 220 Hz low pass filter and 60 Hz notch filter.

Sinusoidal vertical vibration was provided by a Ling 1512 electrodynamic shaker powered by a DMA 2/X solid-state power amplifier (Anaheim, CA). Adaptive control for the shaker was provided by a DAKTRON shaker control system (Fremont, CA). For shaker operation, each frequency and magnitude required in the

experimental protocol was specified in the DAKTRON control software. The software profile was setup for a constant acceleration sine vibration test. Throughout the experimental run, the match by the control software between demanded acceleration magnitude and real-time acceleration magnitude was monitored by the operator (figure 4). A screen shot of the interface window used to setup test vibration frequencies and magnitudes for any sine vibration protocol on the software is shown in figure 6. A schematic of the shaker setup is shown in figure 5. All data was collected at 1500 Hz on a 16 channel A/D board equipped with data acquisition software (Innsport, Chicago, IL).

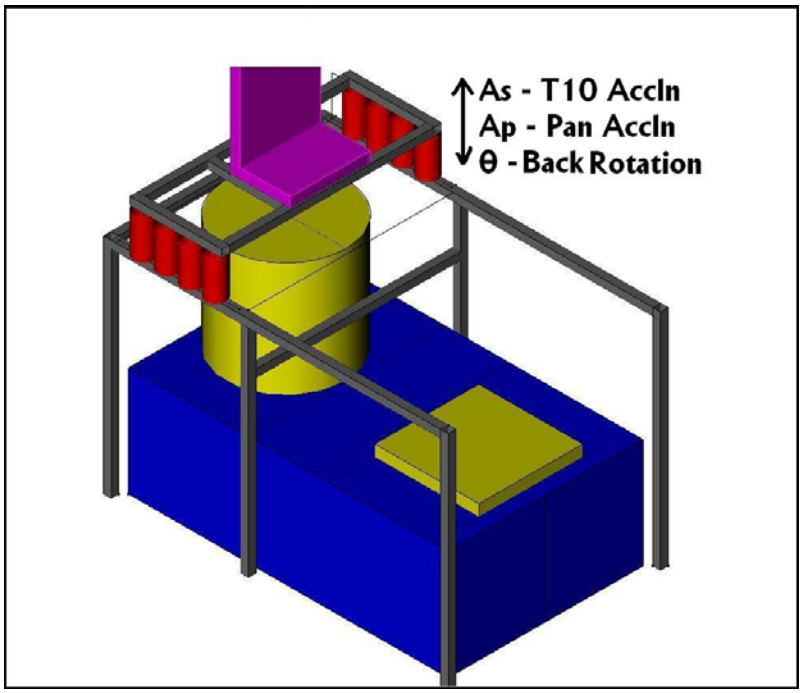

Figure 5: Schematic of shaker setup

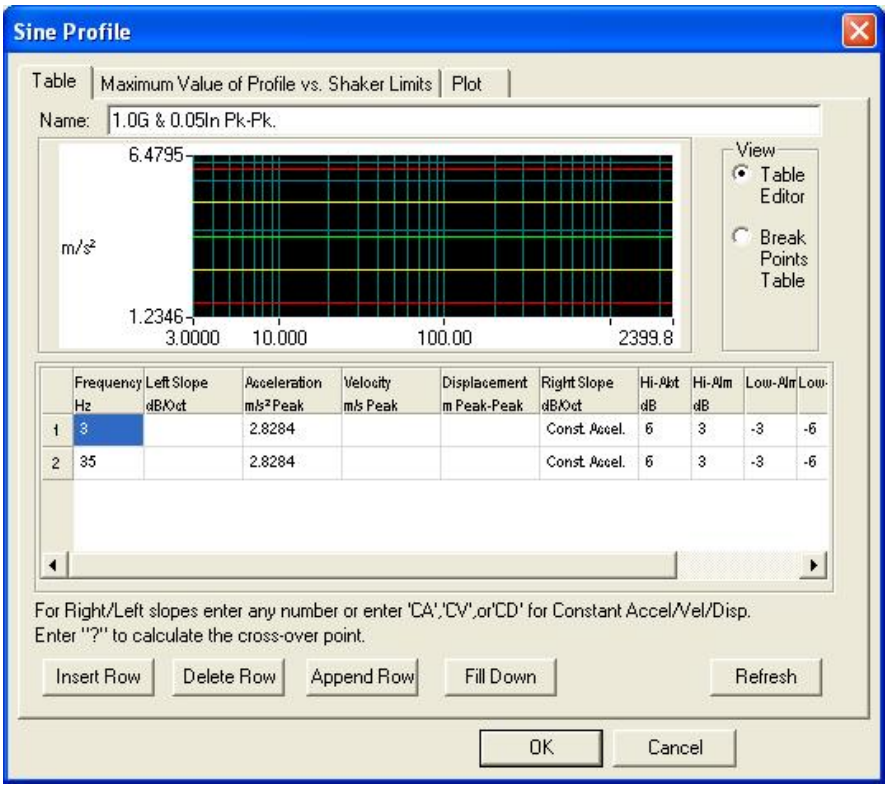

Figure 6: Magnitude and frequency setup window on DAKTRON

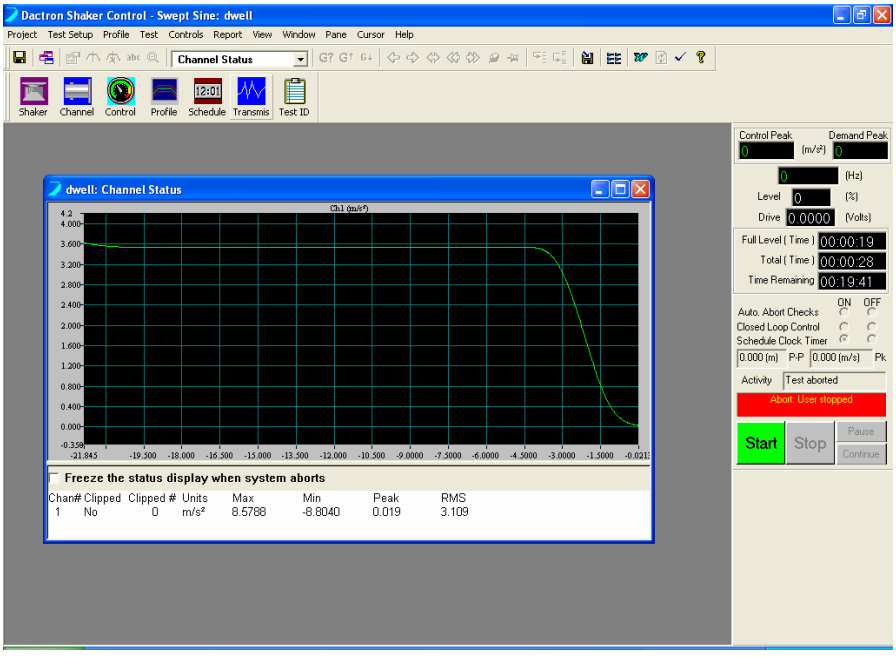

Figure 7: Monitoring a match between control and demand peak acceleration

2.2 Experimental protocol

Subjects were asked to fill out a medical history questionnaire to screen for any recent history of back injury prior to the start of the experiment. Subjects who reported a frequent history of back pain were advised not to participate for safety reasons and informed consent was obtained from subjects who qualified. EMG electrodes were placed as per established protocols on the muscle groups of interest. Prior to electrode placement, the skin was conditioned and a conductive gel was used for optimal conduction. To collect EMG maxes for the muscle groups, the subjects were instructed to perform abdominal crunches and torso lifts to the front and sides with the pelvis restrained on the stationary platform.

The subjects were further instrumented with the goniometer and the accelerometers and asked to sit carefully on the unpadded seatpan of the electrodynamic shaker. The subjects were instructed to adopt a comfortable upright posture and the angle display unit on the goniometer was zeroed after the posture was adopted. Visual feedback from the goniometer angle display unit was used by the subjects to maintain a consistent posture through each trial run. Vibration frequency (3, 4, 6, 8, 10, 12, 14, 16, 18, 20 Hz) and magnitude (1, 1.5 and 2 RMS m/s^2) were the independent input variables and a total of 30 (10×3) frequency-magnitude combinations (FMCs) were provided as inputs for the shaker software to initiate vibration test runs. EMG, accelerations and back-rotations were collected simultaneously at 1500 Hz for each vibration FMC for a time period of 40 seconds. A total of 30 individual sets of data were collected for the 30 FMCs. Rest times were

allowed between trials to prevent fatigue and the subjects were instructed to resume initial posture before each successive trial. Total setup and testing time was approximately 2 hours. The order of presentation of vibration frequency was randomized within each magnitude condition and vibration magnitude was block randomized.

2.3 Transmission functions

Processed EMG, seat acceleration, pan acceleration and lumbar rotation data were used in determining four transmission functions (TMFs). Transmission of input acceleration (seat) to the spine (TMF1) was quantified as the ratio in magnitude between acceleration measured at the spine to the acceleration measured at the seat. Translation of vertical vibration into lumbar rotations was measured as the magnitude ratio of lumbar rotation and seat acceleration (TMF2). TMF3 represents vibration induced muscle activity and is described as the ratio of nEMG magnitude to the seat acceleration magnitude. Mechano-neuromotor transmission ($TMF4_{magnitude}$) was defined as the ratio in magnitude between neuromuscular activation measured as nEMG magnitude and lumbar flexion-extension rotations measured by the electrogoniometer.

$$TMF1_{magnitude} = \frac{acceleration_{spine}}{acceleration_{seat}} \quad \textbf{Equation 4}$$

$$TMF2_{magnitude} = \frac{nEMG}{lumbarrotation} \quad \textbf{Equation 5}$$

$$\text{TMF3}_{\text{magnitude}} = \frac{nEMG}{acceleration_{seat}}$$ **Equation 6**

$$\text{TMF4}_{\text{magnitude}} = \frac{lumbarrotation}{acceleration_{seat}}$$ **Equation 7**

In each TMF described above, the denominator term and the numerator term represent the input and output variables respectively. Delay times were defined as the difference in time between the peak occurrence of output and input variables and were calculated for TMF2, TMF3, and TMF4. Two different methods were used in the calculation of the described TMFs.

2.3.1 Cross spectral density method (CPSD)

In this method, a transfer function conveying magnitude and phase information is calculated from the input and output signals. A conventional way of obtaining a transfer function is through the simple ratio of the power spectral density (PSD) of the output and input signals. However, in this study, transfer functions were calculated as the quotient of the cross power spectral density, $CSD_{input-output}(f)$ of the input and output signals and the power spectral density input, $PSD_{input}(f)$.

$$TF_{CSD}(f) = \frac{CSD_{input-output}(f)}{PSD_{input}(f)}$$ **Equation 8**

The advantage of using CSD in estimating transfer functions is retention of the phase information pertaining to the output response. Noise in the measurement system is reduced because this estimate includes TF data only at regions of higher

correlation between the input and output [Mansfield (2005)]. The power spectral density represents the power content of a signal in an infinitesimal frequency band. Mathematically, it can be expressed by the following expression.

$$\phi(\omega) = \lim_{N\to\infty} E\left\{\frac{1}{N}\left|\sum_{t=1}^{N} y(t)e^{-i\omega t}\right|^2\right\} \qquad \textbf{Equation 9}$$

In this expression, y (t) represents the signal, ω is the angular frequency and N corresponds to the length of the fast Fourier transform (FFT) of the signal [Stoica *et al.* (1997)].

A Welch's averaged periodogram (CSD) was used to calculate the cross spectral density of the input-output signals and the spectral densities of the input and output signals separately. The built-in scheme for the Welch periodogram in MATLAB 7.1 analysis software was used. This method employs Fast Fourier Transforms (FFT) to calculate CSD and PSD. A cross correlation sequence between the input-output signals and an autocorrelation sequence of the input signal alone is estimated before FFT algorithms are employed. The output and input signals are segmented into a finite integer number of overlapping segments to determine FFT length. A hamming window of length corresponding to each segment length is used in calculating the averaged periodogram described in Stoica *et al.* (1997).

In the current application, all the signals were collected for 40 seconds at 1500 Hz leading to data arrays consisting of 60,000 data points each. Input and output signals were split into eight segments with a 50 % overlap between each segment. A Hamming window of length corresponding to each segment length was applied and

the FFT length was set at the first power of 2 larger than the length of each segment. $CSD_{input-output}(f)$ and $PSD_{input}(f)$ length was set at half the length of the FFT (nFFT/2 +1). Transfer functions calculated using the Welch periodogram are complex variables and the magnitude and phase measures were obtained by the following expressions.

$$TF_{magnitude} = \sqrt{(TF_{real})^2 + (TF_{imag})^2} \quad \textbf{Equation 10}$$

$$TF_{phase} = \tan^{-1}\left(\frac{TF_{imag}}{TF_{real}}\right) \quad \textbf{Equation 11}$$

In equations 11 and 12, TF_{real} and TF_{imag} represent the real and imaginary parts of the complex TF variable. A magnitude coherence estimate was performed to quantify the degree of correlation extent between the input and output signals. A coherence value of 1 indicates 100 % correlation and a coherence value of 0 indicates no correlation. A higher value of this estimate dictates higher precision and lower noise contamination in the measurement system.

$$coherence(f^2) = \frac{\left|CSD_{input-output}(f)\right|^2}{PSD_{input}(f) \times PSD_{output}(f)} \quad \textbf{Equation 12}$$

2.3.2 Running Average Method

A second method used in this study for analyzing cyclic signals was the running average method. In this method, ensemble averages to a single vibration cycle were produced for all the processed signals (accelerations, EMG and back rotations) in each FMC described in the experimental protocol. For 40 seconds of data

on a 20 Hz FMC, this would correspond to an averaging of 20×40 cycles to a single cycle as compared to 3×40 cycles for a 3 Hz FMC. A schematic of the averaging process is shown in figure 8.

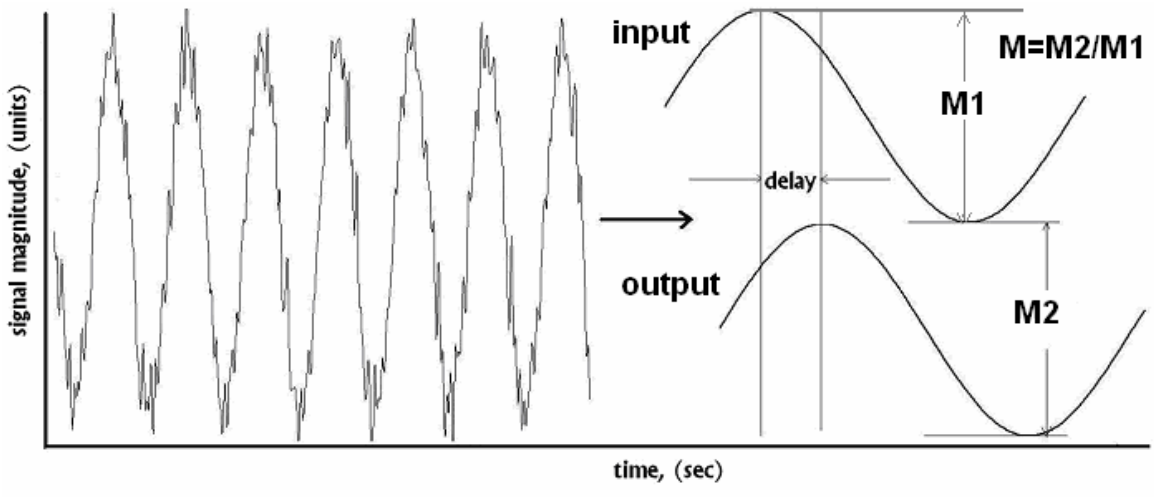

Figure 8: Schematic of running average method

The averaging process was initiated at the same time instant for the input and output signals to avoid contamination of phase information. Starting from the first minimum of the sinusoidal input signal as a base point, the entire signal was split into cycles. Cycle length was determined by the frequency of data acquisition divided by the vibration test frequency. An ensemble average was obtained by calculating the mean of the signal split into several cycles. Average magnitude of each signal is determined by noting the peak-peak difference of the time-averaged signal and TMF magnitude is produced by the ratio of the average magnitudes of the output and input signals. Output delay is determined as the difference in time between the input and

output maximums. All data processing for the individual signals and TMF estimations was performed on MATLAB software.

3. Results

Average values for all subjects for TMFs 1-4 calculated through the cross-spectral density method ($method_1$) and the running average method ($method_2$) are presented and compared (figures 9-13). All the calculated TMFs exhibited vast inter-subject variability and are presented in figures 21-24 for both methods at all vibration magnitudes. (Note: Inter-subject variability is not shown in the mean plots).

3.1 Transmission function magnitudes

Trunk acceleration transmissibility (TMF1) was found to have a gradual decline with increasing frequency exhibiting a sharp peak at 4 Hz and a smaller peak at 10 Hz (figure 9). Both methods exhibited a similar trend, while $method_1$ showed higher magnitudes than $method_2$ at all frequencies. $Method_2$ exhibited higher variability in transmission across vibration magnitudes than $method_1$. For $method_2$, average transmission for the entire frequency range was found to differ by 18.6 % between the highest (2 RMS ms^{-2}) and lowest (1 RMS ms^{-2}) vibration magnitudes. Variability across magnitudes was highest in the peak regions at 4 and 10 Hz. For $method_1$, variability across magnitudes was lower from 3-10 Hz than from 10-20 Hz. Average transmission for the frequency range differed by 5.1 % between the highest and lowest vibration magnitudes. Phase response of TMF1 calculated by $method_1$ declined with increasing frequency (figure 10).

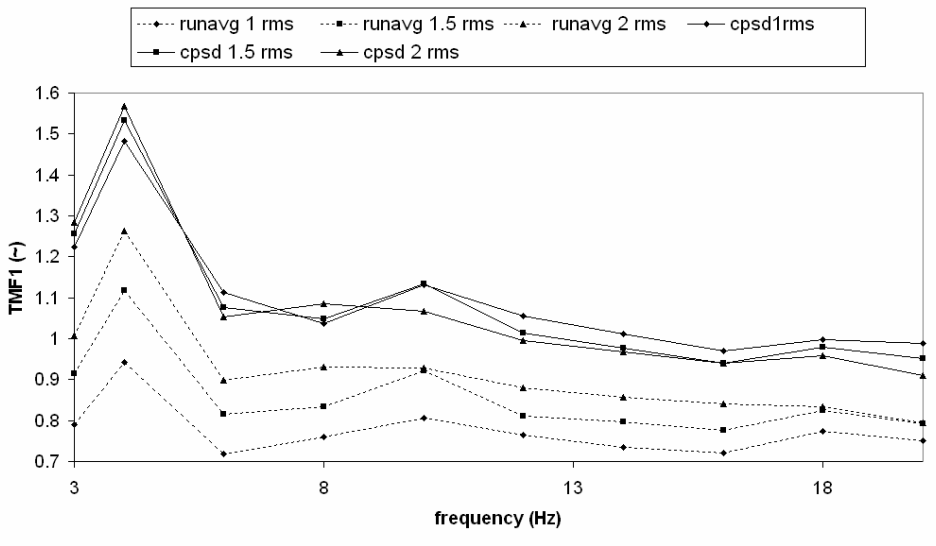

Figure 9: TMF1. Transmission of acceleration to the spine (T-10 spinous process) exhibited a sharp peak at 4 Hz and a smaller peak at 10 Hz.

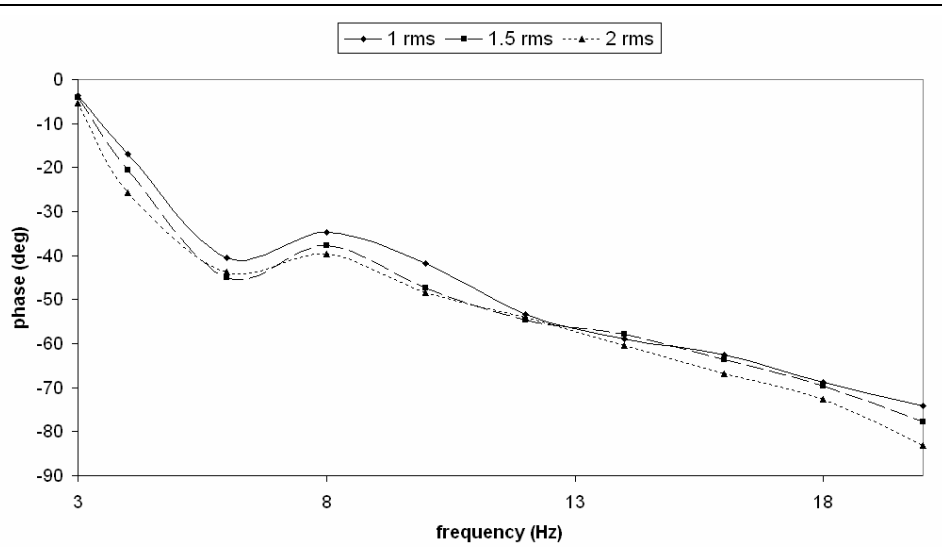

Figure 10: TMF1. Phase response for TMF1 was calculated by method$_1$.

TMF2 (indicative of vibration induced lumbar rotations) was found to decline with increasing frequency after peaking at 4 Hz (figure 11). Both methods exhibited a

similar trend, but higher values of TMF2 was observed for $method_1$ than $method_2$ from 3-10 Hz. $Method_1$ also demonstrated a higher variation across vibration magnitude in this frequency range. No large variations were seen between methods from 10-20 Hz. Average transmission for the frequency range differed by 8.4 % between the highest and lowest vibration magnitudes for $method_1$ and 6.4 % for $method_2$.

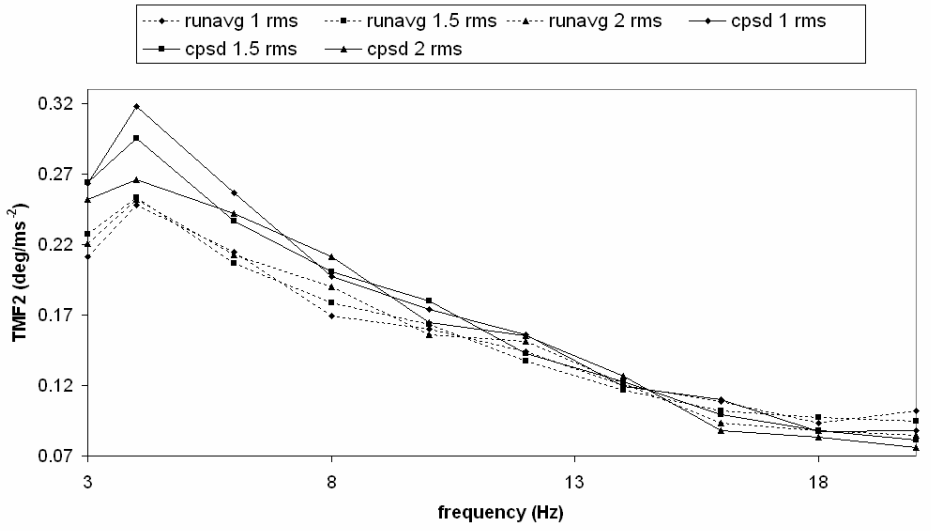

Figure 11: TMF2 magnitude showed a decline with increasing frequency after peaking at 4 Hz.

nEMG as a function of input acceleration (TMF3) was found to have a gradual decline with increasing frequency exhibiting a peak at 4Hz for 2 RMS (ms^{-2}) magnitude and a peak at 6 Hz for the two lower magnitudes (figure 12). Both methods exhibited a similar trend and had a smaller peak at 10 Hz. For $method_2$, average transmission for the entire frequency range was found to differ by 38.5 % between the highest (2 RMS ms^{-2}) and lowest (1 RMS ms^{-2}) vibration magnitudes.

This estimate was 31.5 % for $method_1$. Variability across magnitudes was higher from 3-14 Hz and highest in the peak regions for both methods. From 14-20 Hz, no variation across magnitude was seen and TMF3 magnitude was minimal.

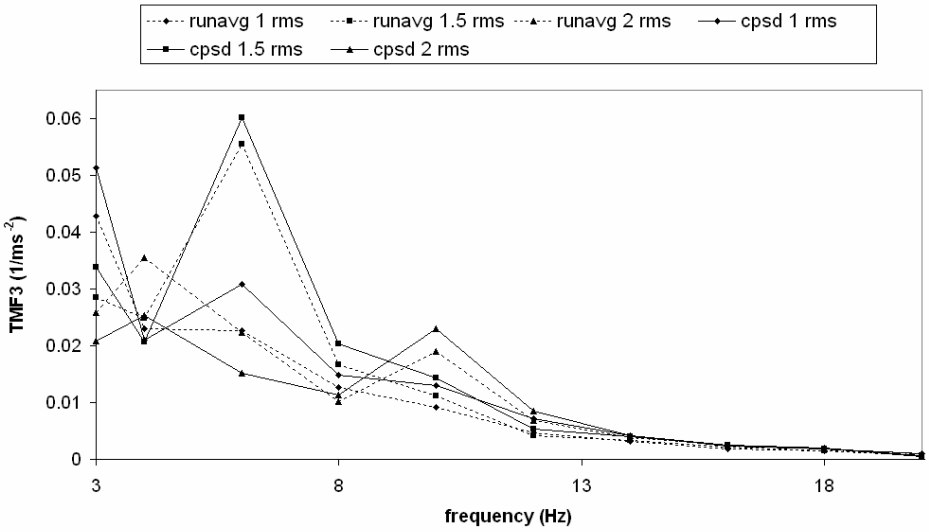

Figure 12: TMF3 showed a gradual decline with increasing frequency showing a small peak at 10 Hz for all vibration magnitudes. A higher peak at 4 Hz was seen for 2RMS and at 6 Hz for the two lower vibration magnitudes.

Mechano-neuromotor transmission (TMF4) was found to have a relatively constant transmission with increasing frequency exhibiting a peak at 4 Hz for 2 RMS, (ms^{-2}) magnitude and a peak at 6 Hz for the two lower magnitudes in both methods (figure 13). Both methods exhibited a similar trend and showed a smaller peak was seen at 10 Hz. For $method_2$, average transmission for the entire frequency range was found to differ by 34 % between the highest (2 RMS ms^{-2}) and lowest (1 RMS ms^{-2}) vibration magnitudes. This estimate was slightly lower at 33.1 % for $method_1$.

Variability across magnitudes was higher from 3-14 Hz and highest in the peak regions for both methods.

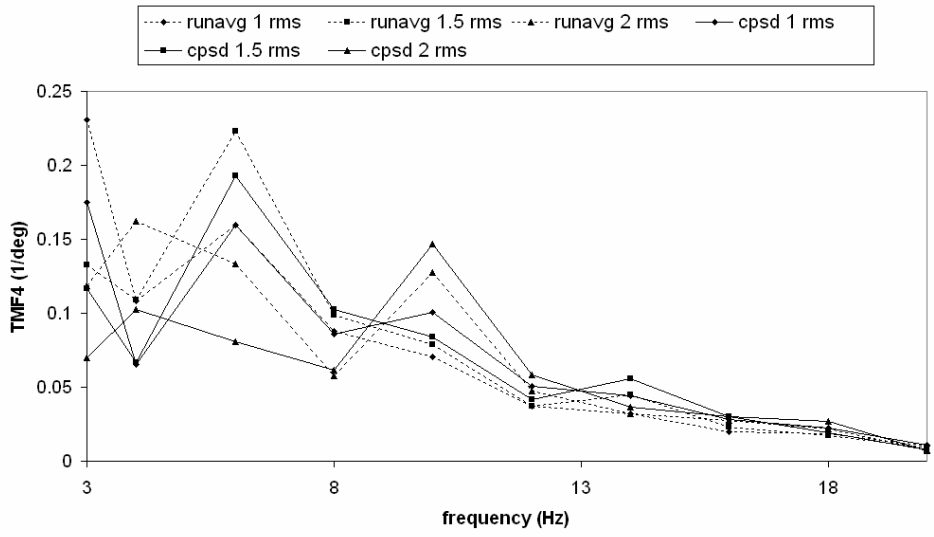

Figure 13: MNT remained relatively constant with increasing frequency showing a small peak at 10 Hz for all vibration magnitudes. A higher peak at 4 Hz was seen for 2RMS and at 6 Hz for the two lower vibration magnitudes.

3.2 Delay times

Average time delay for all subjects between input acceleration and vibration induced muscle activity ($delay_1$) decreased with increasing frequency (figure 14). nEMG lagged behind input acceleration by ~200-230 ms at 3 Hz to ~20-30 ms at 20 Hz. Average time delay for all subjects between vibration induced back rotations and nEMG ($delay_2$) showed a similar trend and decreased gradually with increasing

frequency (figure 15). nEMG lagged behind vibration induced back rotations by ~150 ms at 3 Hz to ~30 ms at 20 Hz. Both delay measures demonstrated a slightly higher delay time at the lowest vibration magnitude (1RMS ms^{-2}) as compared to the two higher vibration magnitudes. $delay_1$ averaged over the frequency range dropped from ~82 ms at 1RMS (ms^{-2}) vibration magnitude to 65 ms at 2RMS (ms^{-2}). $Delay_2$ averaged over the frequency range dropped from ~70 ms at 1RMS (ms^{-2}) vibration magnitude to 60 ms at 2RMS (ms^{-2}).

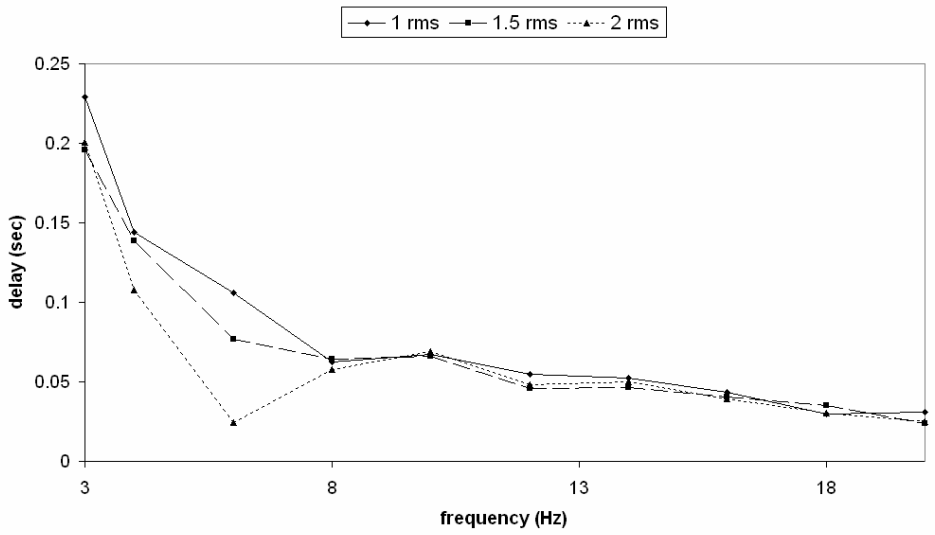

Figure 14: Time delay measured between peak input acceleration and peak nEMG activation showed a gradual decrease with increasing frequency from ~ 230ms at 3 Hz to ~30ms at 20 Hz.

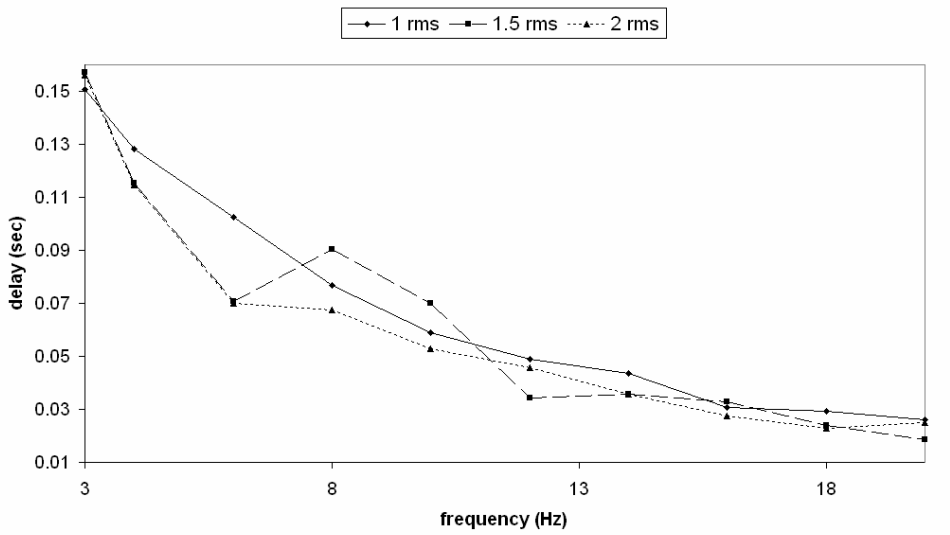

Figure 15: Time delay measured between peak vibration induced back rotations and peak nEMG activation showed a gradual decrease with increasing frequency from ~ 150ms at 3 Hz to ~30ms at 20 Hz.

3.3 Coherence measure for CPSD method

Coherence measures were averaged for the magnitude range and presented as a function of frequency for the four calculated TMFs (figure 16). At all frequencies, TMF1 shows highest coherence, followed by TMF2, TMF3 and TMF4. Magnitude variations over the whole frequency range are represented as standard vertical error bars. Standard deviations (indicative of magnitude variations for all frequencies) were highest for TMF4 (SD = 0.039) and lowest for TMF1 (SD = 0.0023). Coherence measures averaged over the whole frequency range are presented as a function of magnitude for the four calculated TMFs in figure 17. For all magnitudes, TMF1 shows the highest coherence followed by TMF2, TMF3 and TMF4 ($TMF1_{coh}$> $TMF2_{coh}$ > $TMF2_{coh}$ > $TMF4_{coh}$). When individual frequencies were examined

(figures 18-20), TMF3 and TMF4 exhibited much low signal coherence at 12 Hz and 20 Hz.

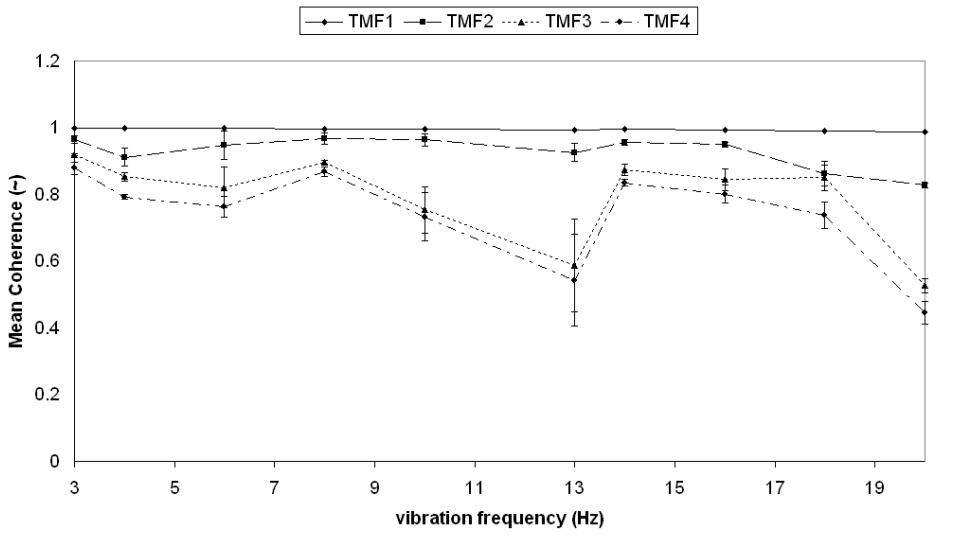

Figure 16: Magnitude averaged coherence estimate is presented for the whole frequency range for TMFS 1-4. Standard vertical error bars display variability with magnitude.

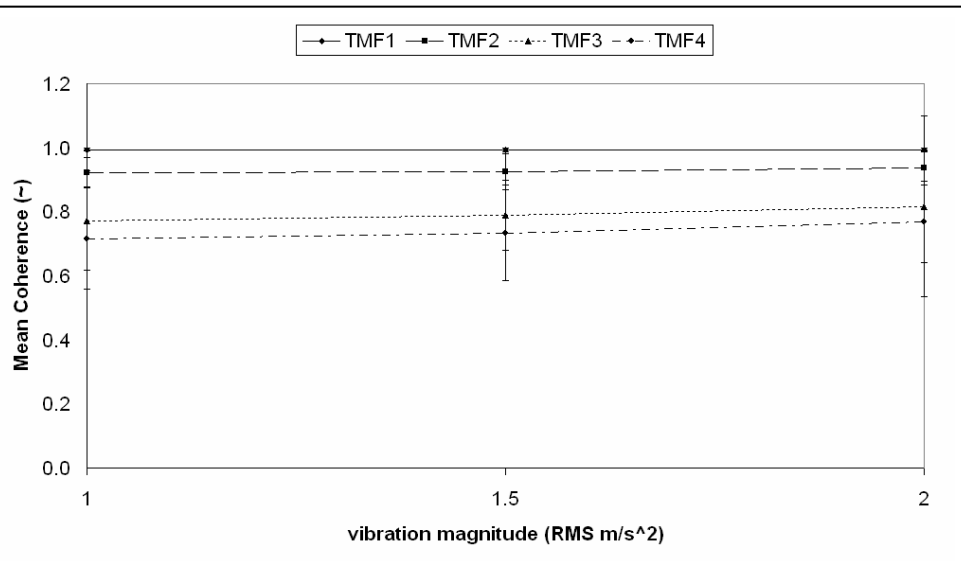

Figure 17: Frequency averaged coherence estimate is presented for the three different magnitudes for TMFS 1-4. Standard vertical error bars display variability with frequency.

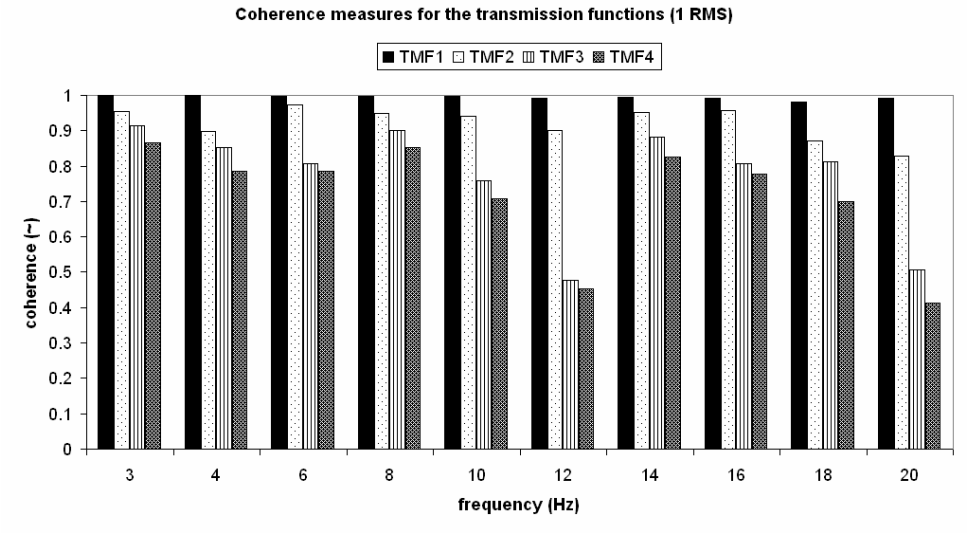

Figure 18: Coherence estimate for TMFs 1-4 at 1 RMS vibration magnitude show highest coherence for TMF1 and progressively lower measures for TMF 2-4. Specifically, lower measures at 12 Hz and 20 Hz are indicative of excessive noise in the measurement system at these frequencies.

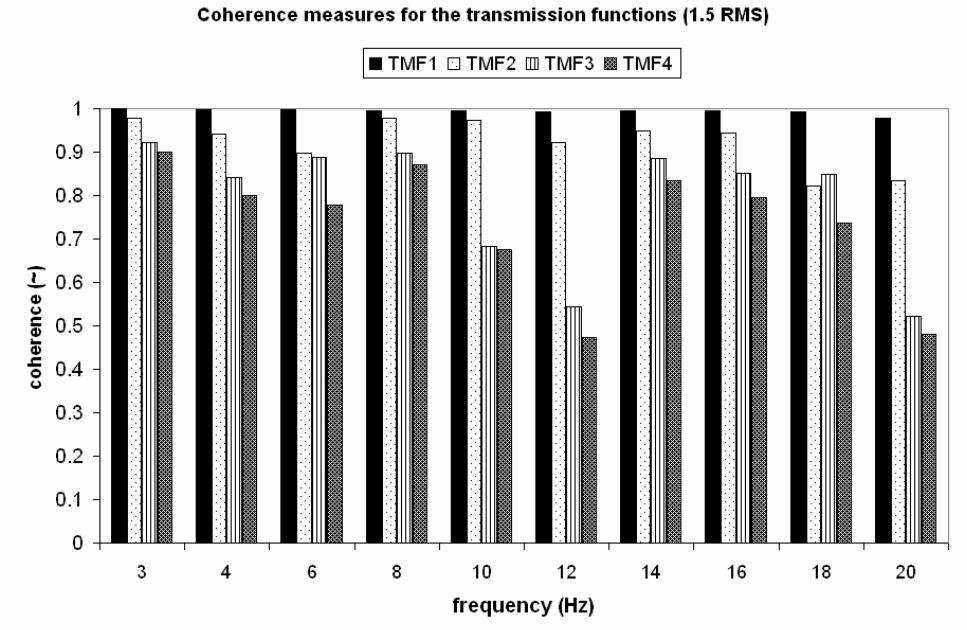

Figure 19: Coherence estimate for TMFs 1-4 at 1.5 RMS vibration magnitude show highest coherence for TMF1 and progressively lower measures for TMF 2-4.

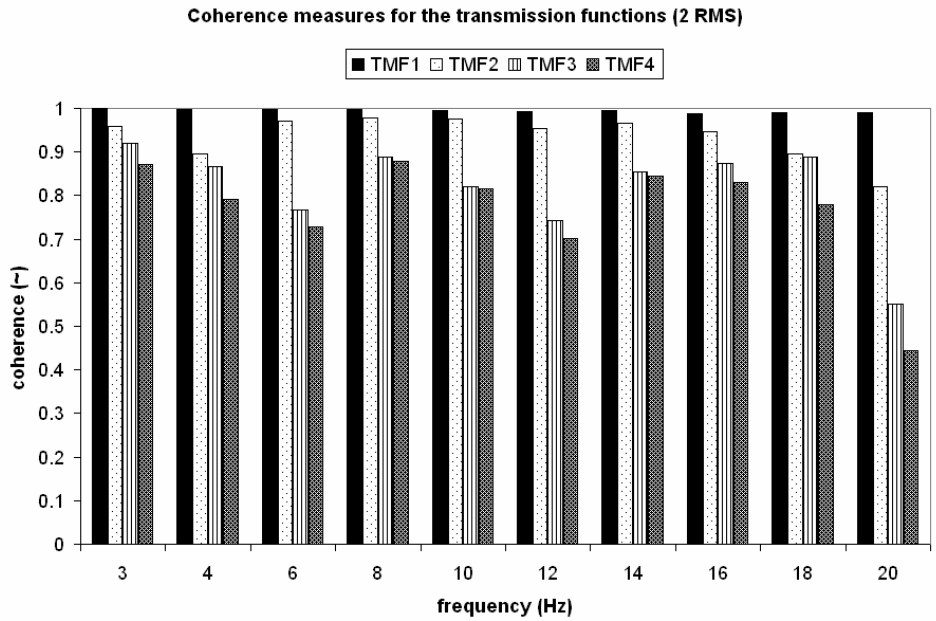

Figure 20: Coherence estimate for TMFs 1-4 at 1 RMS vibration magnitude show highest coherence for TMF1 and progressively lower measures for TMFs 2-4.

3.4 Inter-subject variability

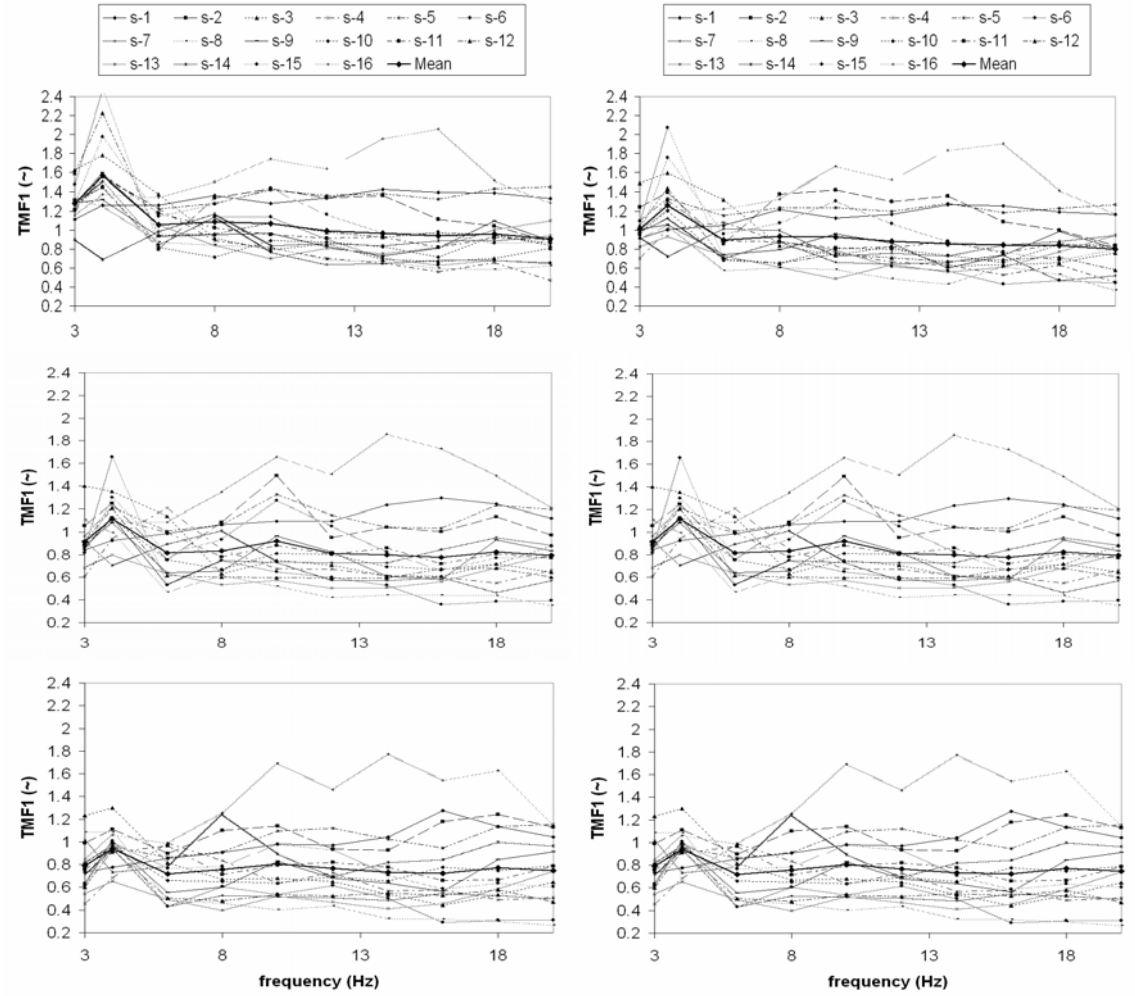

Figure 21: Inter-subject variability for TMF1 as a function of frequency is shown for both methods at all vibration magnitudes. Top left: $method_1$ **2RMS. Middle left:** $method_1$ **1.5RMS. Bottom left:** $method_1$ **1RMS. Top right:** $method_2$ **2RMS. Middle right:** $method_2$ **1.5RMS. Bottom right:** $method_2$ **1RMS**

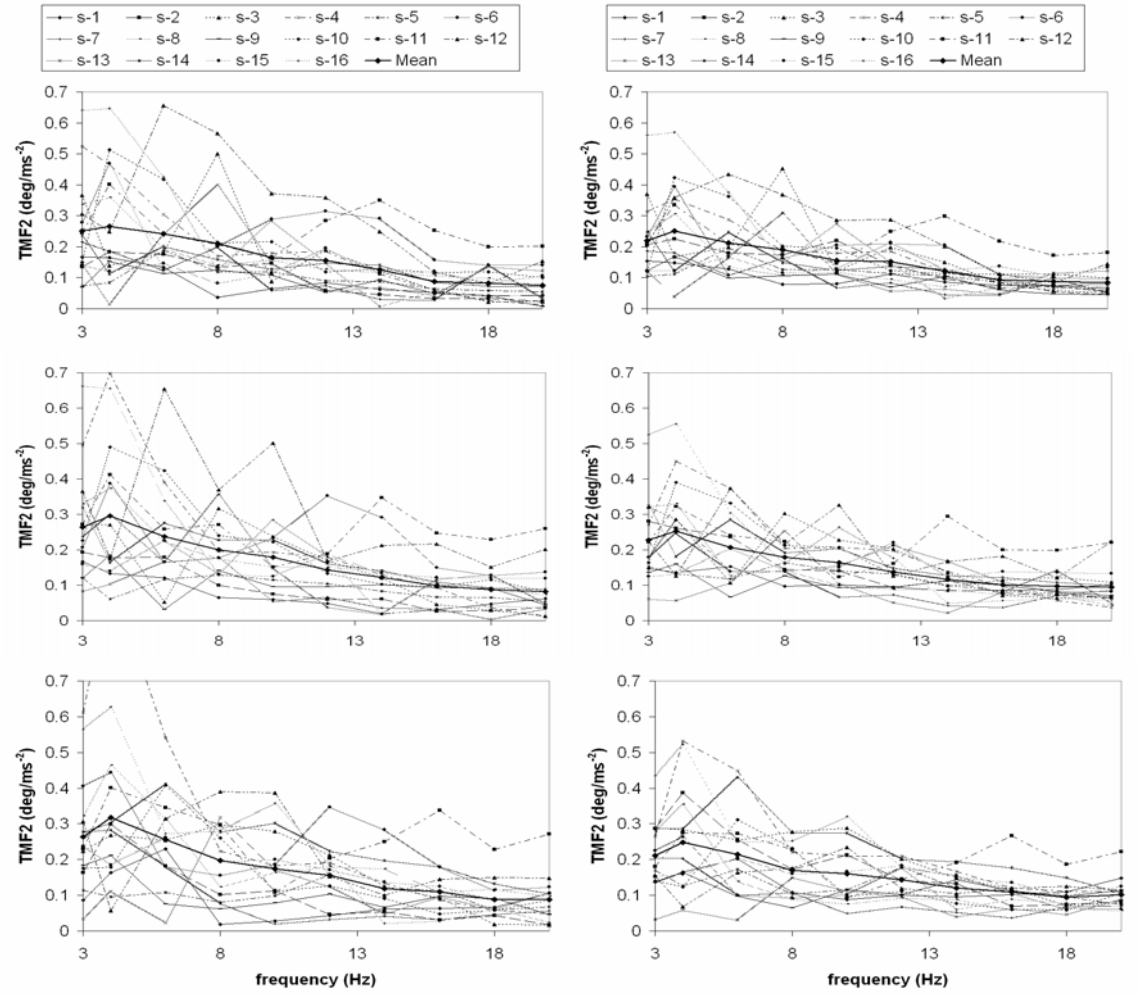

Figure 22: Inter-subject variability for TMF2 as a function of frequency is shown for both methods at all vibration magnitudes. Top left: $method_1$ **2RMS. Middle left:** $method_1$ **1.5RMS. Bottom left:** $method_1$ **1RMS. Top right:** $method_2$ **2RMS. Middle right:** $method_2$ **1.5RMS. Bottom right:** $method_2$ **1RMS**

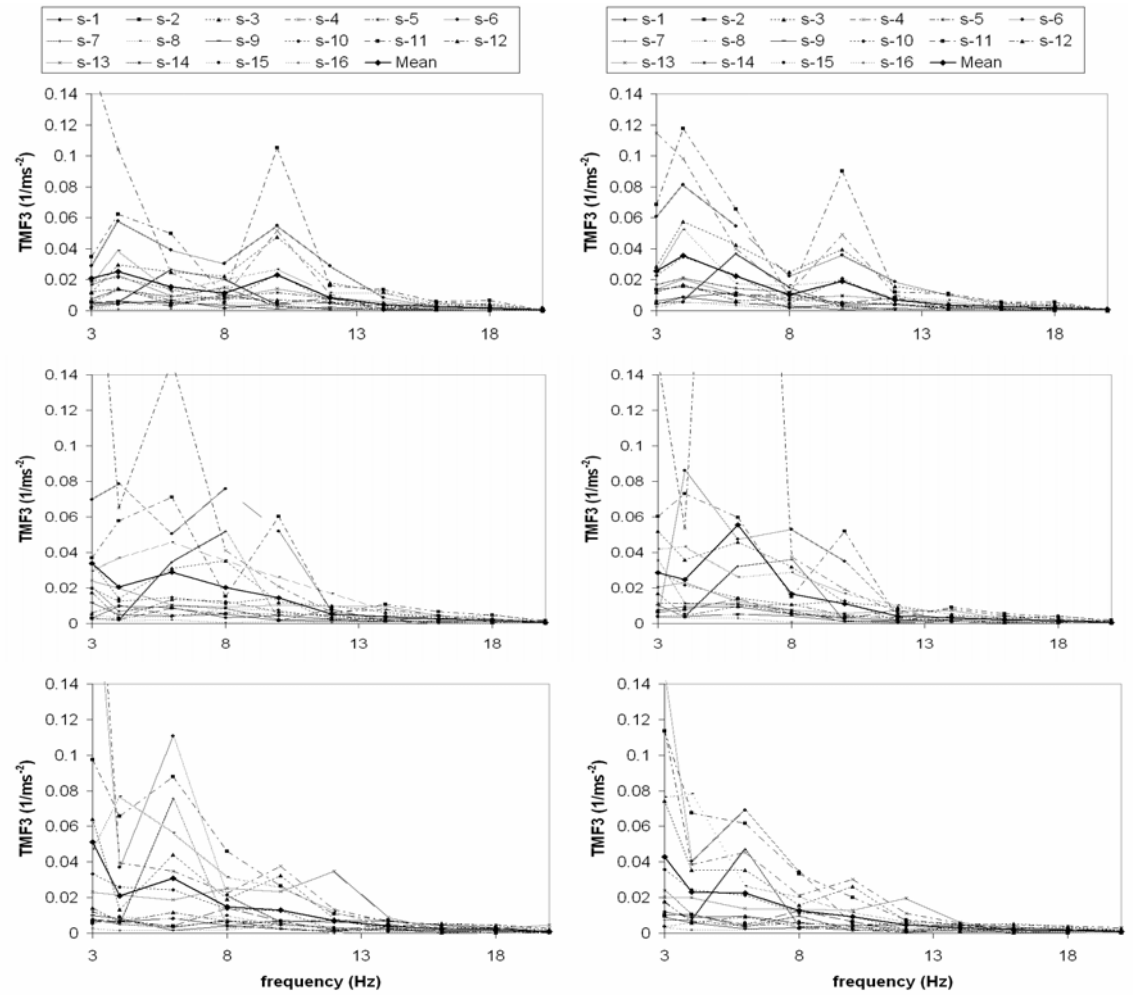

Figure 23: Inter-subject variability for TMF3 as a function of frequency is shown for both methods at all vibration magnitudes. Top left: $method_1$ **2RMS. Middle left:** $method_1$ **1.5RMS. Bottom left:** $method_1$ **1RMS. Top right:** $method_2$ **2RMS. Middle right:** $method_2$ **1.5RMS. Bottom right:** $method_2$ **1RMS**

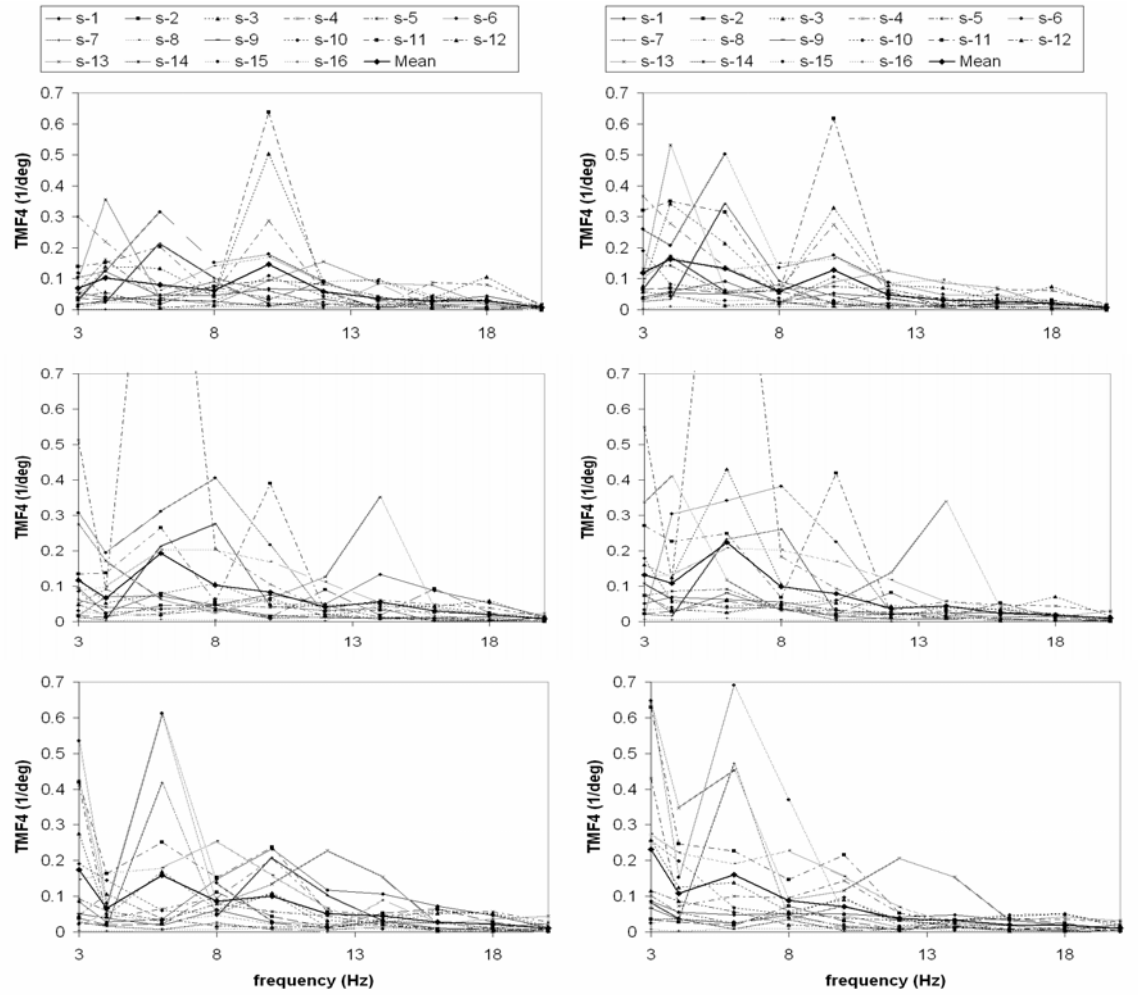

Figure 24: Inter-subject variability for TMF4 as a function of frequency is shown for both methods at all vibration magnitudes. Top left: $method_1$ **2RMS. Middle left:** $method_1$ **1.5RMS. Bottom left:** $method_1$ **1RMS. Top right:** $method_2$ **2RMS. Middle right:** $method_2$ **1.5RMS. Bottom right:** $method_2$ **1RMS**

4. Discussion

Four different transmission functions (TMF1-4) were assessed for a frequency range of 3-20 Hz for three different vibration magnitudes in this study. The results of this study indicate that the human response to WBV in an unsupported seated posture is complex and dependent on both vibration frequency and magnitude. These results can be used to better understand and quantify the neuromotor transmission of WBV to the paraspinal musculature.

4.1 Trunk acceleration transmissibility (TMF1)

Trunk acceleration transmissibility (TMF1) was measured as the ratio of accelerations measured at the T-10 spinous process to the input seat acceleration. TMF1 exhibited a response that was both vibration frequency and magnitude dependent. TMF1 magnitude decreased with increasing frequency after peaking at 4 Hz when accelerations at the trunk were measured through a accelerometer mounted on the skin at the T-10 spinous process. The cross-spectral density method ($method_1$) and the running average method ($method_2$) showed peak transmissions of 1.48 and 0.94 respectively, at 4 Hz. The obtained resonance frequency was comparable to Panjabi *et al.* (1986) who reported a resonance at 4.4 Hz and a peak transmission of 1.6 for the lumbar vertebrae (L1-L3) for vertical vibration (1 RMS ms^{-2} vibration magnitude). Zimmermann *et al.* (1997) measured this trunk transmission for accelerometers mounted at the T-5 level and reported a resonant peak between 4.5-6 Hz with a corresponding transmission of1.76. Although Panjabi *et al.* (1986) reduced

skin artifacts largely by attaching accelerometers directly to the spinous processes (in vivo) at the L2, L3 and sacral level, the results were similar to those found in the study. Transmission was measured at T-5 by Zimmermann *et al.* (1997) and was similar to the transmission at T-10 measured in this study. In this study, acceleration was measured at the T-10 spinous process to examine motion just above the lumbar spine for comparison with lumbar rotation. Matsumoto *et al.* (2002) measured transmission to the trunk at the T-10 process and reported a peak between 4.5-6 Hz. However, the input vibration used in that study was random rather than sinusoidal. The dynamic response of the body exposed to random vibration might differ from that of sinusoidal vibration because of an ability to predict future motion in the latter. Such differences in the experimental protocols could account for the differences in transmission to the spine while comparing the results from these studies.

Mansfield *et al.* (2000) has reported a nonlinear shift of the resonant peak towards lower vibration frequencies with increasing magnitude. This 'softening effect' that occurs with increasing vibration magnitude was not apparent in the current study as was also the case with Panjabi *et al.* (1986), Pope *et al.* (1989) and Broman *et al.* (1991). Mansfield *et al.* (2006) investigated this nonlinear further and observed that this softening effect was not apparent in cases where subjects pre-tensed the torso musculature prior to vibration. This was achieved by using two groups: a group where the subjects adopted a 'tense' posture and a controls group where the subjects assumed a relaxed posture. The subjects in this study could have stiffened (pre-tensed) their torso more while adopting the neutral upright posture.

However, torso “stiffness” or co-contraction was not controlled, limiting the discussion on the nonlinear effects of vibration magnitude.

4.2 Vibration induced back rotations (TMF2)

Seidel *et al.* (1988) has shown that vertical seat acceleration during WBV leads to both vertical and angular motions of the spine. In particular, spinal flexion corresponding with upward seat acceleration and spinal extension motion corresponding to downward seat acceleration has been documented. These cyclic flexion-extension motions (angular motions) of the spine have been observed to decrease with increasing frequency. Smaller magnitudes of back rotations were observed at 8 Hz than at 4.5 Hz. Zimmermann *et al.* (1997) measured a decrease in vibration induced pelvic and back motions over a frequency range of 4-16 Hz and noted maximum rotations in the frequency range of 4-6 Hz. Vibration induced lumbar rotations (TMF2) measured in this study exhibited a peak at 4 Hz and a gradual decrease with increasing frequency at all vibration magnitudes. Such higher lumbar rotations observed at 4 Hz are reminiscent of trunk acceleration transmissibility showing a resonant peak at the same frequency in this study. Resonance of the seated human at this frequency would have resulted in a higher magnitude of lumbar (or spine) flexion-extension motions at the same frequency. The result of decreasing lumbar rotations with increasing vibration frequency may also be related to the use of a constant magnitude of vibration RMS acceleration with frequency. When constant vibration acceleration is maintained over a frequency range, there is an inverse

relation between frequency and peak-peak displacement of the seat pan according to the following expressions.

$$x = A\sin\omega t \qquad \textbf{Equation 13}$$

$$\ddot{x} = -\omega^2 A\sin\omega t \qquad \textbf{Equation 14}$$

where ω is the angular frequency of vibration, A is the peak amplitude of the seat and $\ddot{x}$ represents the seat acceleration. There wan an option to use constant displacement of the shaker armature over the frequency range. However, the accelerations and force resulting from a constant displacement protocol at higher frequencies exceeded the human comfort contours mentioned in Griffin (1990) above 12 Hz and would have uncomfortable for the subjects. Consequently, a constant acceleration magnitude was used in this protocol over the whole frequency range for reasons of safety.

4.3 Vibration induced muscle activity (TMF3)

Peak to peak nEMG exhibited a peak at 4 Hz for 2 RMS vibration magnitude and at 6 Hz for the two lower vibration magnitudes used. A smaller peak was seen at 10 Hz. The initial peak between 4-6 Hz is similar to phasic EMG reported by Seroussi *et al.* (1989) and Zimmermann *et al.* (1997). The higher nEMG measured at these frequencies is indicative of higher muscle activity required at regions of trunk resonance (4-6 and 10 Hz) and peak lumbar rotation observed in this study to stabilize the upper body. Such maximum peak-peak EMG coinciding with TMF1 and back motion between 4.5-6 Hz has also been reported by Zimmermann *et al.* (1997). The muscle could be acting as a biomechanical feedback element and opposing inertial

trunk forces as suggested in the model described by Seroussi *et al.* (1989). These studies have noted that peak-peak EMG was highest at frequencies below 6 Hz and declining at frequencies above 6 Hz. Griffin *et al.* (1989) discuss lower peak-peak EMG values exhibiting a peak at 4 Hz and decreasing above and below this frequency. Similar results were observed for TMF3 in this study.

4.4 Neuromotor transmission (TMF4)

The neuromotor transmission of input sinusoidal vibration (TMF4) describes the response of the erector spinae muscle group to vibration induced lumbar rotations. TMF4 was seen to exhibit a similar trend as TMF3 exhibiting a double peaked (4-6 Hz and 10 Hz) transmission mode. Cyclic paraspinal muscle activity likely has its basis in a feedback response to muscle length changes. Stretch reflexes are known to respond to cyclic length changes in the muscle [McMahon (1984)]. Stretch reflexes are facilitated by stretch receptors called spindle organs located deep within the muscle belly that are sensitive to changes in muscle length. These receptors are attached at both ends of the main muscle mass and experience the same relative length as the overall muscle. When an activated muscle is stretched by an external agency and experiences a length change, it contracts to retain its original length. This is termed a stretch reflex and a cycle of stretch reflexes can be observed as with EMG. Seroussi *et al.* (1989) suggested the occurrence of cyclic stretch reflexes as a possible mechanism for vibration induced muscle activity. In this study, the

correspondence between TMF2 and TMF3 and the relatively constant TMF4 with frequency support this relationship.

If lumbar rotations (TMF2) directly influence nEMG (TMF3) during vibration, then TMF2 and TMF3 would be essentially the same and TMF4 would have shown a flat line characteristic over the measured frequency range. The characteristic double peaked trend seen in TMF4 suggests that additional factors may influence this relationship. The other factors may include other modes of reflex activation outside of the lumbar rotation, internal resonance of the neuromotor feedback loop due to delay in the circuit timing, or non-linearity in the neuromotor response with frequency. The peak in TMF4 at 4-6 Hz may be evidence of other factors such as the effect of axial vibration or the effect of other response feedback loops such as voluntary control. The second peak at 10 Hz is intriguing as this may correspond to a resonance of the neuromuscular system. The time delay between lumbar rotation and nEMG was measured to be ~ 50 ms at this frequency. Such a time delay would result in a system that resonates at 10 Hz. A SIMULINK model (MATLAB 7.1) with a transport delay element of 50 ms was created to represent the generic response of a sinusoidal sweep to time delay in a closed loop system (figure 25). Higher displacements (indicative of a resonance) were seen to occur at ~ 10 Hz. Seroussi's model underestimated trunk muscle activity at 3-10 Hz because the measured peak-peak muscle activity was significantly higher than the magnitude required to balance vibration induced upper trunk inertial forces. This excess muscle activity beyond the mechanical demands imposed on the muscle during vibration

could very well be substantiated by over excitation of stretch reflex component of nEMG.

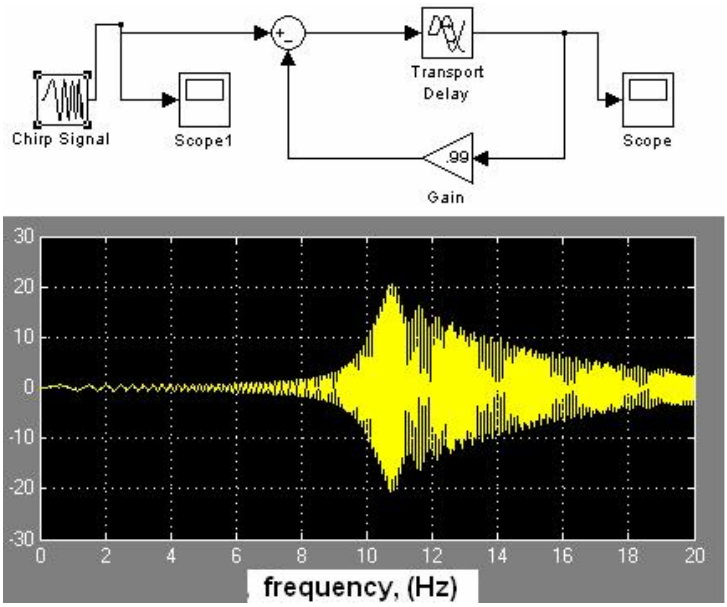

Figure 25: Simulink model

A greater influence of head, trunk and pelvic motion (serving as a stimulus) for peak-peak EMG activity at frequencies under 6 Hz, irrespective of subject posture, has been suggested by Zimmermann *et al.* (1997), where EMG was measured for different postures from 4-16 Hz. In this study, the influence of lumbar rotational motion on peak-peak EMG activity was assessed (as seen in TMF4). At frequencies higher than 14 Hz, magnitudes of both the lumbar rotation and nEMG were small, making the data at these frequencies susceptible to noise and more difficult to interpret.

4.5 Delay times

Delay times observed between nEMG and input acceleration in this study were calculated using the running average method. A drop in this delay time was observed with increasing frequency from ~230 ms at 3 Hz and starts to plateau off at ~70ms beyond 8 Hz. TMF3 delays measured in this study showed a steeper drop (with increasing frequency) than Seroussi *et al.* (1989) who measured a time delay that measured ~250 ms at 3 Hz and dropped to ~ 130 ms at 10 Hz. At frequencies higher than 14 Hz, magnitudes of both the lumbar rotation and nEMG were small, making the data at these frequencies susceptible to noise and more difficult to interpret. Consequently, delay calculations within this range could be prone to error

Time delay for TMF4 measured between peak lumbar rotation and peak nEMG activation measured ~150 ms at 3Hz and decreased with increasing frequency. Assuming lumbar rotation initiates the stretch reflex, this could suggest a transition from more complex polysnaptic reflex and voluntary feedback systems that are associated with longer time delays to faster monosynaptic reflex feedback systems. Monosynaptic reflexes are fast because they involve a single synapse connecting the neuron that transmits information from the muscle spindle and the neuron innervating the muscle [McMahon (1984)]. A synapse is a region where nerve impulses are transmitted from one nerve ending to another. More synapses are suggestive of higher delay times in the neural loop. Polysynaptic circuits involve target motor neurons as well as interneurons in the central nervous system (more synapses). A signal would

take more time to get transmitted through several interneurons and arrive at the proper motor neurons on either side of the spinal cord in these reflex types.

4.6 Variations in results

Differences were observed in TMFs 1-4 calculated by the CPSD method and the running average method used in this study. Lower magnitudes were observed in all four TMFs for the running average method than the CPSD method. Though the same software enabled filters were used for all the acquired signals in both methods, the CPSD method is less sensitive to noise in the measurement system. At every frequency run, the CPSD method isolates the transfer function at the exact test frequency, eliminating noise artifacts embedded in other frequencies. The running average method could be more sensitive to measurement system noise and depend more on software enabled filtering as compared to the CPSD method. The averaging scheme used here could reduce smooth out a portion of the noise, when ensemble averages are produced. A higher peak-to-peak magnitude of the input acceleration resulting from noise artifacts could have reduced the magnitudes of TMFs 1-3. Magnitude for the CPSD method is calculated from the whole wave form, but only from the ensemble averaged peaks for the running average method, potentially leading to different results. In addition, input-to-output coherences were generally lower in TMF3 and TMF4 at all frequencies. At 12 and 20 Hz, the coherence estimate was drastically lower for these TMFs indicating a lower precision for the transfer function calculated at these frequencies.

4.7 Future work

Vibration exposures in this study were conducted for a period of 40 seconds for each trial. In occupational vibration exposure, longer durations are typically encountered. Posture, trunk muscle coactivation and muscle pretension (prior to vibration) were also not included as controlled variables in this study. Future work should include exposure to longer durations typical of real time occupational exposure. Such longer durations of exposure could help analyze the effects of proprioceptive loss (from extended exposure) and possible muscular fatigue. The effects of posture and trunk muscle co-activation on the transmission of vibration to the neuromotor system should also be examined. EMG data collected on the Rectus Abdominus and internal and external obliques can also be processed to examine for neuromotor transmission through these muscle groups.

In general, formulation of preventive measures against vibration transmission has resulted in redesign/manufacture of machinery, limiting work hours for personnel, adaptive seating or design of vibration isolation devices. Operation of machinery at human resonant frequencies has been avoided. The results presented in this study describe another mode of transmission through the neuromuscular system with an internal resonance at ~ 10 Hz. These results offer another method of transmission that should be investigated further for possible prevention. The ability to alter neuromotor transmission independently could also be used in the future to better understand the role of indirect mechanisms that promote back injury. Other studies should include methods to reduce the neuromotor transmission of WBV with cheap alternative

solutions. In addition, the effects of posture and trunk muscle co-activation on the transmission of vibration to the neuromotor system should be examined.

5. Conclusions

In conclusion, whole body vibration is considered to be a significant factor in contributing to LBD and related MSDs. Vibration transmission studies have focused extensively on the mechanical transmission of input (seat) vibration to different body segments of interest and identified primary resonant peaks at 4.5-6 Hz. Other studies have found secondary resonant peaks from 8-14 Hz with large inter-subject variability and contradictions between studies. A few studies have examined response of the torso musculature to vibration. Both direct and indirect effects of vibration leading to pathology have been identified for the vibrating human. Vibration-induced neuromotor activation has been suggested in the literature as a possible indirect mechanism for altered low back stabilization and dynamic response, which may in turn increase injury risk. This study describes the transmission of WBV through the neuromuscular system. Neuromotor transmission was defined as the contribution of vibration induced lumbar motions to paraspinal (Erector Spinae) muscle activity and a transmission function was quantified for a frequency range of 3-20 Hz at three different vibration magnitudes. A double peaked trend was seen in this transmission showing a peak between 4-6 Hz and at 10 Hz. The 10 Hz peak may correspond to the internal resonance of the neuromuscular system. Future work should include exposure to longer durations typical of real time occupational exposure.

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Appendix A: Consent Form

Approved by the Human Subjects Committee University of Kansas, Lawrence Campus (HSCL). Approval expires one year from 12/17/2006.

Transmissibility of seated vibration to the nervous system

INTRODUCTION

The Department of Mechanical Engineering at the University of Kansas supports the practice of protection for human subjects participating in research. The following information is provided for you to decide whether you wish to participate in the present study. You may refuse to sign this form and not participate in this study. You should be aware that even if you agree to participate, you are free to withdraw at any time. If you do withdraw from this study, it will not affect your relationship with this unit, the services it may provide to you, or the University of Kansas.

PURPOSE OF THE STUDY

We are interested in evaluating how truck driver and other workers who are exposed to vibration move and how their reflexes change based on understanding how the vibration is transmitted through their muscular and nervous system.

PROCEDURES

If you choose to participate, we will first give you a health questionnaire to make sure you do not have any heart problems or back injuries that might make it difficult to do the experiment.

You will be seated on a chair that vibrates at different frequencies. It will vibrate less than 5mm. When the vibration is slow the experience might be similar to that when you sit on your dryer. When it is fast it will be similar to the speed on some vibrating massage chairs. We will put electromyographic sensors on the front and back of your torso that will measure what your muscles are doing. Further, we'll use devices to monitor the posture and body motion as you are seated on the chair.

The vibration will last about 20 minutes or less as we collect our data. Your participation is strictly voluntary and you can stop at anytime. You can indicate to us to shut off the vibration or get up off the seat at any time if you feel any discomfort. We assure that your name will not be associated in any way with the research findings.

RISKS

Truckers and similar workers who are exposed to vibrations all day in their trucks are known to experience higher rates of back injuries (about 2 or 3 times other workers). We believe that these increased risks are due to how the vibration numbs their back so they cannot lift heavy weights properly. We believe that this effect lasts about 20 minutes. In this experiment we will request you to limit strenuous activity for 20 minutes after the vibration to minimize these risks. Some people have allergies to adhesives such as in band-aids or in the tape we are using to attach the markers. In rare cases, some subjects may experience dizziness or motion sickness after extended vibration. If you feel dizzy or motion sick, please inform the investigator so he/she can stop the vibration.

BENEFITS

With this research we hope to be able to understand what happens to truck drivers and similar workers. It will help us understand how the vibration affects the muscular and nervous system of the body and how a person changes how they move after vibration will tell us something about why these workers get injured more often. There is, however, no direct benefit for the subject of this study.

PAYMENT TO PARTICIPANTS

Subjects will receive $10 per hour for participation in the study.

INFORMATION TO BE COLLECTED

To perform this study, researchers will collect information about you. This information will be obtained from a questionnaire that will assess if you have heart or back problems that might make exercise inadvisable. Also, information will be collected from the study activities that are listed in the Procedures section of this consent form. This includes information about how you walk, your height and your weight.

Your name will not be associated in any way with the information collected about you or with the research findings from this study. The researcher(s) will use a study number instead of your name.

In addition, Dr. Wilson and her team may share the information gathered in this study, including your information, with the Whitaker Foundation that is funding the study. Again, your name would not be associated with the information disclosed to these individuals. Some persons or groups that receive your information may not be required to comply with the Health Insurance Portability and Accountability Act's

privacy regulations, and your information may lose this federal protection if those persons or groups disclose it.

The researchers will not share information about you with anyone not specified above unless required by law or unless you give written permission.

Permission granted on this date to use and disclose your information remains in effect indefinitely. By signing this form you give permission for the use and disclosure of your information for purposes of this study at any time in the future.

INSTITUTIONAL DISCLAIMER STATEMENT

In the event of injury, the Kansas Tort Claims Act provides for compensation if it can be demonstrated that the injury was caused by the negligent or wrongful act or omission of a state employee acting within the scope of his/her employment.

REFUSAL TO SIGN CONSENT AND AUTHORIZATION

You are not required to sign this Consent and Authorization form and you may refuse to do so without affecting your right to any services you are receiving or may receive from the University of Kansas or to participate in any programs or events of the University of Kansas. However, if you refuse to sign, you cannot participate in this study.

CANCELLING THIS CONSENT AND AUTHORIZATION

You may withdraw your consent to participate in this study at any time. You also have the right to cancel your permission to use and disclose information collected about you, in writing, at any time, by sending your written request to: Dr. Sara Wilson, Mechanical Engineering, University of Kansas, Lawrence, KS 66045. If you cancel permission to use your information, the researchers will stop collecting additional information about you. However, the research team may use and disclose information that was gathered before they received your cancellation, as described above.

PARTICIPANT CERTIFICATION:

I have read this Consent and Authorization form. I have had the opportunity to ask, and I have received answers to, any questions I had regarding the study and the use and disclosure of information about me for the study. I understand that if I have any additional questions about my rights as a research participant, I may call (785) 864-7429 or write the Human Subjects Committee Lawrence Campus (HSCL),

University of Kansas, 2385 Irving Hill Road, Lawrence, Kansas 66045-7563, email dhann@ku.edu.

I agree to take part in this study as a research participant. I further agree to the uses and disclosures of my information as described above. By my signature I affirm that I am at least 18 years old and that I have received a copy of this Consent and Authorization form.

_____ _____

Type/Print Participant's Name Date

_____

Participant's Signature

Researcher Contact Information

Sara E. Wilson
Principal Investigator
Mechanical Engineering
3013 Learned Hall
University of Kansas
Lawrence, KS 66045
785 864-2103