

Effects of Seated Whole-Body Vibration on Spinal Stability Control

Gregory P Slota

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Kevin P. Granata, Chair
Michael L. Madigan, Co-Chair
John W. Grant
Maury A Nussbaum
Thurmon E. Lockhart
Sara E Wilson, University of Kansas

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ABSTRACT

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Low back disorders and their prevention is of great importance for companies and their employees. Whole-body vibration is a risk factor for low back disorders, but the neuromuscular, biomechanical, and/or physiological mechanisms responsible for this increased risk are unclear. These studies investigated changes in the biomechanics and control of the trunk in order to further the understanding of the mechanisms responsible for this increased risk.

The purpose of the first study was to measure the acute effect of seated whole-body vibration on the postural control of the trunk during unstable seated balance. The findings show that whole-body vibration impaired the postural control of the trunk as evidenced by increased kinematic variance and non-linear stability control measures during unstable sitting. These findings imply an impairment in spinal stability control

The purpose of the second study was to measure the effect of seated whole-body vibration on the parameters of spinal stability control: passive stiffness, active stiffness, and neuromuscular reflexes. The findings show that whole-body vibration altered trunk stiffness (passive stiffness and equivalent reflex stiffness) as well as reflex dynamics. There was no evidence of compensation by active muscle co-contraction recruitment for the decreased trunk stiffness and reflex gain.

The purpose of the third study was to measure the changes in the natural frequency characteristics of the trunk (which can be related to trunk stiffness and damping) during exposure to seated whole-body vibration. The findings show that whole-body vibration caused a decrease in natural frequency suggesting a decrease in the trunk stiffness, and also an increase in the peak amplitude of the frequency response functions suggesting a decrease in overall trunk damping. The rate of change of the natural frequency characteristics suggest that the majority of effects happen within the first 10 minutes of vibration exposure.

These findings reveal changes in the biomechanical properties of the trunk with exposure to seated whole body vibration, and a mechanism by which vibration may increase the risk of low back injury.

Dedication

This work is dedicated to
DR. KEVIN P. GRANATA



I first started to work for Dr. Granata back in the spring semester of 2000 at the University of Virginia in the Kluge Children's Rehabilitation Center. First working for free to show my worth, then being picked up and funded that summer. In December of 2002 I defended my master's thesis and prepared for the moving of the lab.

Starting January 1st, 2003, the lab of Dr. Granata, along with Shawn Russell and myself, was now a part of Virginia Tech in the ESM and emerging Biomedical Engineering departments. During the start-up of the lab, I was hired on as a research associate faculty member to assist with the grant work and working with the new students of the lab group. By that August I was enrolled as a PhD student with Dr. Granata as my advisor again. Throughout the years, I have worked with Dr. Granata and the lab on many projects. The final project I was in charge of was a new frontier of the lab, whole-body vibration. After completing a pilot study, we worked on a grant proposal to fund and continue the project further. While midway through data collection on my first main study, the tragedy of April 16th, 2007 came to be.

In memory, and with the dedication that he had for his own work, I pushed on to complete the first study, as well as finish the line of work on WBV.

In his honor, the lab has been renamed

The Kevin P. Granata Musculoskeletal Biomechanics Laboratory

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For his assistance in creating the vibration platform and greatly needed assistance with creating the computer control of the shaker. For the countless effort wrestling Simulink, though it may look like Labview, and it may be a Matlab program, it behaves like neither.

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Jul Davis

For being a sounding board for problem solving. For his laughter that can be heard though out Norris Hall. Not for poking his head in, seeing me acting as the subject, all connected to the equipment, asking "Are his hands tied?"

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For all the help with design / building of the wobble chair, all three of them. For the assistance in the lab with equipment, purchasing, and building. And for trips to the gym, to work out the body when the mind has had enough.

ESM Dept, et al

For all the assistance to continue and complete my studies following the events of April 16th, even though my project was not directly funded. May the Low Back Lab be able to continue on and not just pass out of sight.

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For being a home away from home. For still standing even after all the equipment that we have attached to the walls or sat on the floor, shaking and perturbing people as well as being heard though out the building.

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Chapter 1 :

Literature Review

(1.1) Low Back Pain

Low back pain (LBP) is defined as pain, muscle tension, or stiffness of the lumbar spine (Manek and MacGregor, 2005). Low back disorders (LBDs) are anatomical or neuromuscular changes that in some cases can include the sources of LBP. Examples of LBDs are intervertebral disc herniations, spinal stenosis, osteoporosis, osteoarthritis, ankylosing spondylitis, and spondylolisthesis.

LBP affects many people, disrupts daily life and work, and presents a great financial burden to society. It is estimated that between 60% and 80% of adults experience an episode of LBP at least once during their lifetime (van Tulder et al., 2001). Within a single year, 15% to 20% of adults experience LBP (Andersson, 1998). In people under the age of 45, LBDs are the most frequent cause of physical activity limitation (Kelsey et al., 1984), and the most frequent cause of absence from work (Andersson, 1998, Rubin, 2007). In addition, the evaluation and treatment of LBP are two of the most common reasons for patients to seek medical attention and apply for workers' compensation (Rubin, 2007). In terms of financial burden, LBP and spinal disorders are ranked in the top ten 'health and productivity costs' in America (Goetzel et al., 2003). The total health care expenditures for people with LBP in the United States exceeds \$90 billion (Luo et al., 2004).

(1.1.1) Risk Factors

According to Manek and MacGregor (2005), approximately 90% of LBP cases have no identifiable physiological cause, and are typically designated as nonspecific by health care professionals. LBP is typically classified as either acute (lasting up to 3 months) or chronic (lasting greater than 3 months). Current research continues to advance the understanding as to which individuals are susceptible to LBP, and the etiology of LBP. Risk factors are conditions and/or characteristics that can lead to an increased probability of LBP. A number of epidemiological studies have identified possible risk factors for LBP (Andersson, 1981, Andersson, 1998, Andersson, 1991, Rubin, 2007). The identification of these risk factors is important in the prevention of LBP. The different factors that have been related to LBP are grouped into three main categories: individual, occupational, and psychological/psychosocial. In most cases of LBP, multiple risk factors are likely involved.

(1.1.2) Individual Risk Factors

Individual risk factors are characteristics specific to a person that have been correlated to incidents of LBP. These risk factors include age, gender, anthropometrics, overall health, spinal anatomy, socioeconomic status, and education level. Initial cases of LBP are highest during the first 3-4 decades of life (Reigo et al., 1999). Incidence rates of LBP increase with age with the greatest frequency for LBP occurring between the ages

of 35 – 55 years (Andersson, 1981). In the older population (age > 65 years), the risk for LBP tends to be increased compared to a younger population due to the prevalence for women having osteoporosis of the spine (Bressler et al., 1999).

LBP is a significant problem for both genders (Rubin, 2007) and there is no clear consensus signifying one gender or another having an overall higher risk for LBP. Some studies present evidence suggesting that women are more predisposed to LBP (Kopec et al., 2004, Linton et al., 1998), while others report that men are more likely to undergo surgery for the same degree of symptoms (Kelsey and Ostfeld, 1975). Perhaps adding to the uncertainty is that gender tends to be coupled with other LBP risk factors. For example, males have more occurrence of disc herniations (individual risk factor) than females (Brown, 1975). Additionally, there may be more frequent mismatches between physical strength of female workers than males and the job requirements (occupational risk factor) (Brown, 1973).

There are correlations between anthropometric measures of height, weight, and body build with occurrence of LBP (Mellin, 1986). Rates increase with taller and heavier individuals, possibly due to increases in moment-loads on the spinal column. Additionally, BMI (body mass index) has been associated with herniated discs and LBP (Battie et al., 1990).

The health status of an individual is an important risk factor of development of LBP. Obesity (BMI >30) as a measure of health status is an independent predictor of the

development of LBP (Webb et al., 2003). Smoking habits have an impact on the musculoskeletal system, increasing the risk of osteoporosis, fractures, decreasing bone density, and increasing degenerative changes in the spine (Rubin, 2007) which can lead to LBDs and LBP. Self-rated health status and the perception of poorer health is also predictor for the development of LBP (Hartvigsen et al., 2004). In addition, the dietary habits and physical fitness level of an individual can be considered. Someone with a good state of physical fitness will have a lesser risk of chronic LBP and more rapid recovery after a LBP episode (Nachemson, 1989).

Degenerative changes in the spinal anatomy and supportive tissues can lead to LBP (Taskaynatan et al., 2005). Also, additional anatomical conditions of osteoporosis, naturally occurring spinal fusions, and disc herniations can cause pain at the affected vertebral joint (vertebrae-disc-vertebrae) or adjacent vertebral joints (Jonsson et al., 1989). Spinal deformities can contribute to an earlier and more severe development of degenerative disc changes (Noren et al., 1991) and mechanical stress to the spine accelerates the process (Biering-Sorensen et al., 1985). The disc acts as a spacer and cushion for the vertebrae, but damaged discs can alter the normal motions of the vertebrae and or cause spinal stenosis and nerve pinching. When a disc is damaged, the viscous nucleus can extend from the annulus resulting in protrusions, extrusions, and herniations. Intervertebral disc space narrowing is strongly associated with LBP (Pye et al., 2004) and disc degeneration is more frequent in individuals who do heavy lifting (Andersson, 1981). Spinal anatomy deformities may also be risk factors for LBP. It has

been reported that deformities such as scoliosis and kyphosis are related to incidents of LBP (Gremeaux et al., 2008, Kang et al., 2007).

Education and socioeconomic status have also been mentioned as risk factors for LBP (Dionne et al., 2001). It has been suggested that less education is correlated to lower socioeconomic status, and lower socioeconomic status is related to the job type, typically manual labor. It is also suggested that lower socioeconomic status may be correlated to other individual factors (smoking, obesity, dietary habits), psychological factors (stress, depression), and physical occupations (Rubin, 2007) each of which increase the risk for LBP.

(1.1.3) Occupational Risk Factors

Occupational risk factors are elements of a job task that have been correlated to incidence of LBP. These risk factors include aspects of physical/repetitive activities (lifting, pushing, pulling) and the postures and motions (static postures, bending, twisting) associated with manual material handling (Eriksen et al., 2004).

Physical activity (lifting, pushing, or pulling) applies loads and moments on the spine that can lead to development of LBP (Rubin, 2007). The highest rates of back muscle strains and ligament sprains are amongst workers in industries with heavy, physically demanding occupations (Klein et al., 1984). Occupations that consist of

manual material handling apply more stress on the structures of the spine (Andersson, 1998, Hoogendoorn et al., 1999). The risk of LBP is related to the degree of physical load, cumulative / long term load, type, and duration (Hoogendoorn et al., 1999). The repetitive nature of manual material handling increases the risk of LBP depending on the duty cycle of work and rest (Solomonow et al., 2000). Repetitive physical activity may consist of smaller loads and apply lower levels of stress on the spine, but the buildup over time can lead to LBP (Rubin, 2007). Additionally, one theory is that vibrations during load handling are associated with repetitive small trauma that can give rise to permanent damage to the structures of the spine which can increase LBP incidents (Andersson, 1981, Bernard, 1997).

Occupational risk factors for LBP include a worker's posture and motions required to complete required job tasks. Static work postures of standing and sitting can be correlated to incidents of LBP (Magora, 1972). In addition, postures of trunk flexion are a known risk for LBP (Punnett et al., 1991) as well as asymmetric postures (Marras et al., 1993). Bending and twisting of the trunk has an established connection with LBP (Brown, 1975, Magora, 1973, Eriksen et al., 2004) as well as the compounded effect when lifting loads (Kelsey et al., 1984).

Lifting is a risk factor of LBP, with LBP incidence increasing when lifting in excess of what is considered acceptable. When modeling the loading effects of lifting on the low back, disc compression forces are one measure of concern (Marras and Granata, 1995). Musculoskeletal and over-exertion medical problems are twice as common if

predicted lumbosacral disc compression forces exceed the recommended limit of 6800 N (Herrin et al., 1986). Other measures that have been examined include measures of spinal stability and the control parameters associated with stability control. Examples risk factors for LBP that have been modeled with spinal stability include load height (Granata and Orishimo, 2001), flexed postures (Granata and Rogers, 2007), prolonged flex postures (Granata et al., 2005), and muscle fatigue (Granata et al., 2004). Delayed trunk muscle responses have also been shown to be predictive of low back injury (Cholewicki et al., 2005).

(1.1.4) Psychological & Psychosocial Risk Factors

Psychological and psychosocial risk factors for LBP consist of person's emotional state and interactions with other people. Psychological risk factors associated with LBP including stress, distress, anxiety, and mood and can have more impact than biomechanical factors on LBP disability (Linton, 2000). There is an especially strong association between LBP and depression (Rubin, 2007). A person's emotional state can also affect their ability to cope with pain. Job satisfaction is also associated with LBP with a reported higher incidence of LBP in people with lower job satisfaction.

Psychosocial risk factors associated with LBP patients are problems establishing emotional contacts, sociophobic attitudes, and poorer intellectual capacity (Andersson, 1981). Social factors of LBP patients also include drug and alcohol abuse, divorces, and family problems.

(1.1.5) Differences between individuals with and without LBP

Several studies have reported differences between individuals with and without LBP to help understand the possible causes of the LBP. While these studies are helpful in identifying important biomechanical and/or neurological factors in LBP, they are limited by the fact that it is difficult to discern if any differences contributed to LBP, or resulted from LBP. Nevertheless, these studies have provided a rich source of information on the occurrence of LBP. There are suggested diminished strength of the trunk muscles (Takemasa et al., 1995), reduced reflex response times (Radebold et al., 2000), and vertebral instability (Weiler et al., 1990). All of these factors can cause impaired spinal stability and increase the risk of LBP (Leone et al., 2007) but it is not known if this is a cause or effect (Andersson, 1981). Currently there are no reported studies where data collections were made before, and then after an incident of LBP.

(1.2) Vibration

Whole-body vibration (WBV) is another risk factor for LBP and is the focus of this dissertation. Long-term exposure to WBV can have detrimental effects on the musculoskeletal system, and extensive repeated exposure can lead to the development of pathological changes to the spinal anatomy leading to LBDs. There is epidemiologic evidence linking occupational WBV exposure and reports of LBP. Presented here will be a theory of how WBV exposure can lead to LBP, and current methods used to measure vibration exposure and human dynamics during vibration.

(1.2.1) Epidemiological Evidence

WBV commonly occurs in job tasks that include the seated operation of vehicles and other heavy machines. Six million workers are exposed to seated WBV (Griffin, 2006) including delivery vehicles drivers (Rosegger and Rosegger, 1960), forklift operators (Boshuizen et al., 1992), helicopters pilots (De Oliveira and Nadal, 2005), and construction equipment operators (Spear et al., 1976). WBV exposure has been correlated to incidence of acute and chronic LBP in industrial workers. For example, tractor drivers have a reported 61-94% prevalence of LBP and pathological changes in the spine (Dupris, 1966) as well as heavy-equipment drivers reported 70% prevalence of LBP (Cremona, 1972). Additionally, there is also the compounding risk for LBP when WBV exposure is followed by other physical activities. This can include delivery drivers

unloading their payload immediately when reaching their destination (Pope and Hansson, 1992).

(1.2.2) Vibration and Low Back Pain

Although a link between WBV and LBP is generally accepted, there is no consensus as to the mechanism of injury (Mansfield and Maeda, 2005a). Moreover, methods of predicting which individuals are most susceptible to the effects of WBV remain elusive (Mansfield, 2005b). One mechanism by which WBV may lead to LBP is through changes in the system of spinal stability control. Such changes in the spine dynamics could lead to increased dynamic loads of the spine, increasing out-of-range compressive loads. The neuromusculoskeletal system that controls spinal stability is comprised of the three subsystems: passive tissue stiffness, active muscle stiffness, and neuromuscular reflexes (Panjabi, 1992). The physiologic systems involved in these subsystems include the nervous system (sensory information, motor control), muscular system (force production, reflex) and system of passive tissues (connective, surrounding). WBV can alter a single element of a subsystem, or combination of multiple elements, that can then lead to a disruption of spinal stability control and LBP. It has been reported that “if the [control] system does not perform well, then it is more prone to injury” (Reeves et al., 2007).

(1.2.2.1) Nervous System

The nervous system has a role in both active muscle stiffness and muscle reflexes, which in turn, contribute to spinal stability. Thus alterations in the nervous system may affect spinal stability. Studies have shown that vibration of the human body, either localized or whole-body exposure, causes changes to the nervous system sensory information and motor control. Vibration can affect the perception of the position and motion of a joint (Goodwin et al., 1972). Additionally, the perception of the intensity of contraction and tension in muscles can be distorted (Carlsoo, 1982). More specifically, muscles are perceived as being longer than their actual length (Eklund, 1972). There is also a change in the sensitivity and impairment of movement precision. The severity of the influence of vibration on a muscle depends on the level of receptor activity and the state of the muscle (length, change in length, tension) (Carlsoo, 1982). Vibration can also cause the neural pathways to become saturated with stimulation rendering additional stimuli ineffective (Brown et al., 1967, Granit and Henatsch, 1956). It was concluded that sustained Ia sensory activity elicited by vibration also has an after effect on the motor system producing a long lasting dynamical modification of posture (Wierzbicka et al., 1998).

There are changes to the static and dynamic control of the musculoskeletal system due to vibration. WBV causes changes to the perceived orientation of the body (Ceyte et al., 2006). In the case of vibration of the muscle groups of pretibial, hamstring, and paraspinal, there is an induced forward tipping of the body (Carlsoo, 1982). When

vibrations were applied to the multifidus muscles, an error in target positioning of the trunk was induced, potentially due to error in proprioception feed back used in muscle movement control (Brumagne et al., 1999). The effects of muscle vibration on postural control were concluded to originate from changes in sensory information (Slijper and Latash, 2004). Vibration exposure has also been shown to produce deviations in motion during straight-ahead walking (Schmid et al., 2005).

(1.2.2.2) Muscular System

The muscular system has a role in both the active stiffness and reflex subsystems of spinal stability control. Thus alterations in the muscular system may affect spinal stability. The force generation capability of muscle can be disturbed during vibration exposure causing excessive muscular contractions and the onset of fatigue. Vibration exposure can elicit contractions produced by motion of muscle spindles at low frequencies as phasic muscular activity and at higher frequencies as ‘tonic vibration reflexes’. Practical uses include physiotherapy via tonic vibration reflexes (Rittweger et al., 2002) as well as athletic training. In addition to standard weight training, WBV exercise has been reported to increase activation of muscles during the workout (Roelants et al., 2006) as well as increase vertical jump height (Cormie et al., 2006). However, an implication of tonic vibration reflexes is an undesired excessive muscle force production. In the case of vibrating hand tools, the hand’s grip force on a handle increases in intensity when the handle starts vibrating (Farkkila et al., 1979). These involuntary contractions in

the hand and arms can also become so severe that there is difficulty releasing of the hand's grip (Rood, 1860). Additionally, in trunk perturbation testing, it was reported that there was an increased reflex amplitude from 55% to 67% maximum voluntary contraction (Wilder et al., 1996). Vibration induced muscle contraction and increased reflex responses can lead to an earlier onset of muscle fatigue with either a reduced maximum force production or shorter duration of sustained sub-maximal contractions (Vollestad, 1997). WBV vibration can cause muscle fatigue and WBV has been used as a fatigue intervention for the trunk (Wilder et al., 1996).

Muscle reflex delay can also be affected by vibration exposure. More specifically, the short-latency and medium-latency reflex delays increase due to WBV exposure (Bove et al., 2003). This has been reported by studies of trunk muscle reflexes where reflex delays increased. In one study, trunk perturbations applied by the sudden loading of a hand-held box showed an increased paraspinal reflex delay from 87 to 108 msec after exposure to WBV (Wilder et al., 1996). In another study, WBV exposure increased reflex delay from 205 to 228 msec with perturbations applied directly to the trunk (Li et al., 2008). Vibration exposure can also suppress muscle reflexes. As described earlier, vibration of muscles can produce a saturation of the neural pathways blocking any additional stimulus from causing a reflex response (Brown et al., 1967, Granit and Hennatsch, 1956). This in turn can lead to an inhibition of muscle reflexes which normally assist in the injury protection of preventing joints from twisting or ligaments from rupturing (Carlsoo, 1982).

(1.2.2.3) System of Passive Tissues

The system of passive tissues has a role in the passive tissue stiffness subsystem of spinal stability control. Thus alterations in the passive tissues may affect spinal stability. These tissues include the ligaments and tendons that surround the spine, as well as the vertebrae and intervertebral discs. These tissues provide a basic structure for the spine and generate resistance to forces and moments. WBV exposure has been shown to cause creep deformation in various tissue types and can possibly lead to tissue failure (Wilder et al., 1988). For example, it has been shown that low amplitude WBV can stretch the hamstrings in a similar manner as standard physiotherapy stretching protocols (van den Tillaar, 2006). In the spine, the intervertebral discs act as spacers between the vertebral bodies. Seated WBV has been shown to result in a greater loss of height of the spine beyond the normal diurnal changes (Klingenstierna and Pope, 1987, Sullivan and McGill, 1990). The majority of this loss of height is a direct result of creep of the intervertebral disc (Keller and Nathan, 1999). Vibration damage to bones and joints has also been reported where exposure to vibration leads to pathological changes in joints (Freund and Dupuis, 1974). In addition to the tissues that provide support of the spine, there are reports of vibration exposure damage to vascular tissue (Curry et al., 2002) and nerve tissue (Govindaraju et al., 2006).

(1.2.3) Measurements of Vibration Exposure

In order to study the effects of WBV exposure, there is the need to quantify vibration characteristics of the human body. The two primary characteristics of vibration are frequency and amplitude, and both have been shown to affect the human body differently. Two common approaches to measure the human body's response to vibration include using frequency response functions of apparent mass and transmissibility. Parameters from these functions can be related to the human dynamics and how the body reacts during vibration exposure.

(1.2.3.1) Vibration Characteristics

When describing vibrations, it is important to fully describe the vibration characteristics involved. For WBV exposure, this includes the vibration frequency content, amplitude, direction, and exposure duration. A vibration wave can consist of a single frequency sine wave, or be as complex as a broad-band waveform consisting of multiple frequencies. Typical vibrations (in industrial job settings) may have one main frequency component and about that frequency a broad-band of random vibration. The frequency of a vibration wave describes how fast the vibrations are moving, while the vibration characteristic of the amplitude of the signal describes the strength of the movement. The amplitude can be reported in units of length (displacement) or acceleration.

While vibration has characteristics of frequency and amplitude, the application of the vibration to the human body has two more characteristics. The direction of the vibration's force to the body needs to be considered. WBV can be applied vertically, horizontally anterior/posterior, horizontally lateral, and any combination there of. Additionally, the duration of the vibration exposure needs to be considered.

(1.2.3.2) Apparent Mass

There are two common approaches used for studying the human biomechanical response during WBV, one of which is the impedance method to quantify apparent mass (Mansfield and Maeda, 2005a). The apparent mass represents the kinetic response of the body to vibration and is a measure of the driving-point frequency response function determined at the seat-person interface (Fairley and Griffin, 1989). The apparent mass ($AM(f)$) is calculated as the ratio of the applied force ($F(f)$) transmitted to the person from the seat and the subsequent acceleration ($Acc(f)$) in the frequency domain (Equation 1) (Mansfield, 2005b).

$$AM(f) = \frac{F(f)}{Acc(f)} \quad (1)$$

The process of calculating the apparent mass of the human body requires the measurement in the time domain of the force applied to the body ($F(t)$) and the subsequent acceleration ($Acc(t)$) at the seat-person interface (Figure 1). Using the Fast Fourier

Transformation (FFT), the time domain data is translated into the force ($F(f)$) and acceleration ($Acc(f)$) in the frequency domain (Figure 2). Following equation 1 above, the apparent mass ($AM(f)$) is calculated as the ratio of force and acceleration at each individual frequency sample (Figure 3).

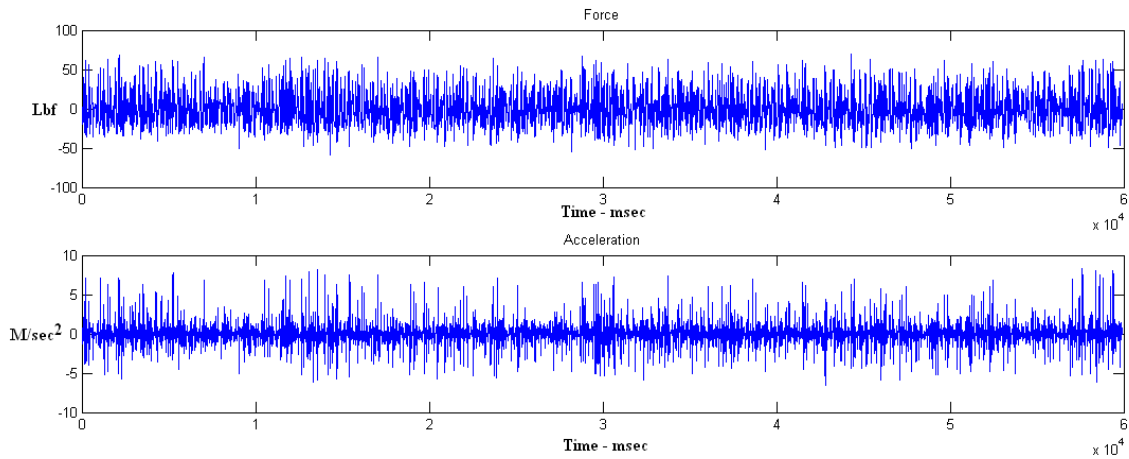


Figure 1: Example of time domain data of force recorded from a load cell (above) and acceleration from an accelerometer (below) located at the seat-person interface.

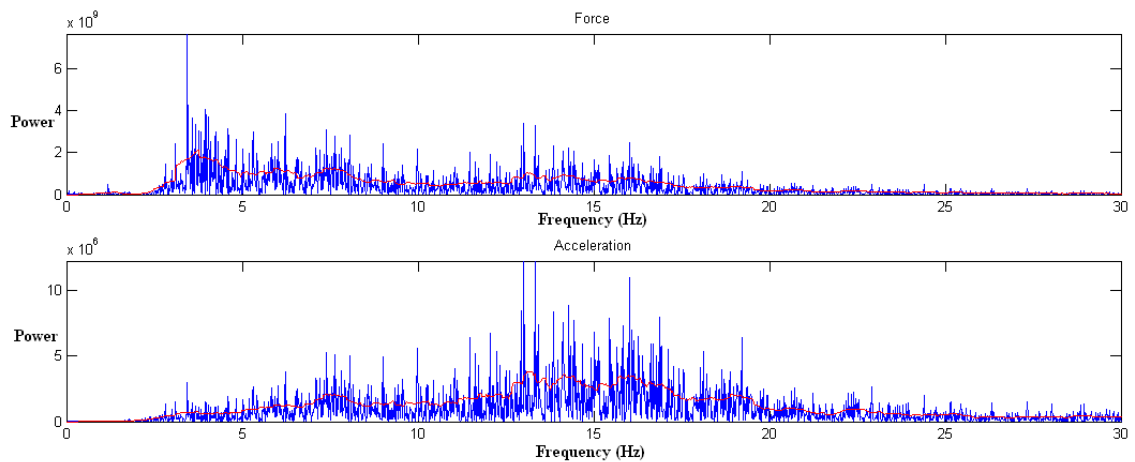


Figure 2: Example of frequency domain data of force (above) and acceleration (below). Data is the Fast Fourier Transform of the data in figure 1.

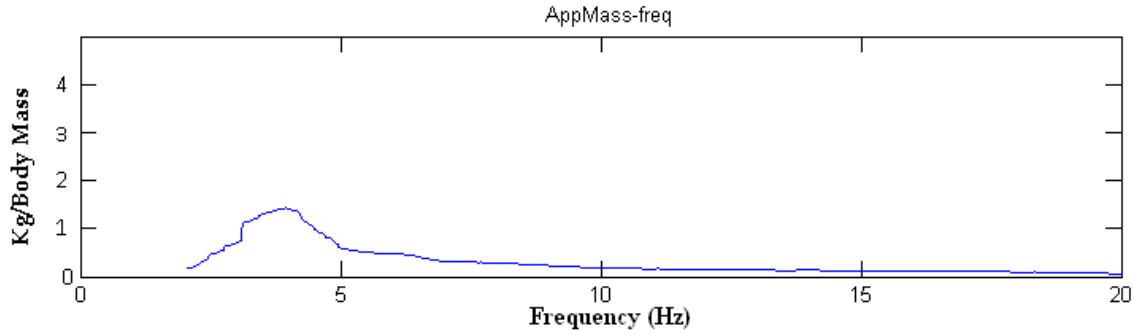


Figure 3: Example apparent mass frequency response function in the frequency domain (2-20 Hz). The graph is the results of the calculation using equation 1 and from the data shown in figures 1 and 2 and is normalized to the static mass.

For the vibration of a rigid mass, the apparent mass at all frequencies is equal to the actual mass. The apparent mass of a dynamic system that has compliance will have an apparent mass greater than its actual mass at frequencies where it resonates and less than its actual mass at frequencies where movement is attenuated. The frequency response function of apparent mass is relative to the actual mass and therefore can be normalized. For vertical WBV, the human body acts as a second order, low-pass system with unity (equal to actual mass) for low frequencies (≤ 1 Hz, not shown in figure 4), a resonance frequency near 5 Hz, and attenuation at higher frequencies (Figure 4).

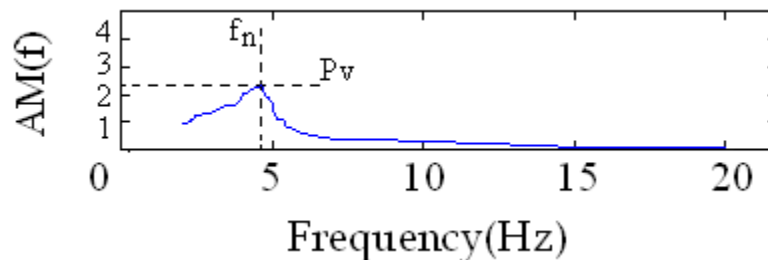


Figure 4: Example apparent mass frequency response curve (2-20 Hz) normalized to static mass. The peak (>1) near 5 Hz signifies the natural frequency of the system. The natural frequency of the system is related to the system stiffness, and the peak value is related to the system damping.

The apparent mass is used to measure the dynamic response of the human body to vibration. The use of apparent mass has been shown to be an effective laboratory tool for testing the dynamics of vehicle seats (Boileau et al., 2002), seat vibration transmission (Wei and Griffin, 1998), and the effects of different seat backrests (Mansfield and Maeda, 2005b). In addition, understanding the apparent mass of the human body assists in the improved design characteristics of dynamic test dummies (Lewis and Griffin, 2002) as well as to further the understanding of human biomechanics (Mansfield and Griffin, 2002).

(1.2.3.3) Transmissibility

Transmissibility methods are another common approach used for studying the human biomechanical response during WBV. This method studies the kinematic assessment of the human body by measuring the transmission of vibration from one point to another. Transmissibility functions indicate the transmission of vibration between the point at which the vibrations are applied the body and at a second point at which the vibration is measured on the body (Hinz et al., 2001). Transmissibility ($TR(f)$) is expressed in the frequency domain as the ratio of two accelerations ($Acc_{head}(f)$, $Acc_{seat}(f)$) measured at two different locations (Equation 2).

$$TR(f) = \frac{Acc_{head}(f)}{Acc_{seat}(f)} \quad (2)$$

The process of calculating the transmissibility of the human body requires the measurement in the time domain of the acceleration at the seat-person interface ($Acc_{seat}(t)$) and a second acceleration on the subject's body, in the example the head ($Acc_{head}(t)$) (Figure 5). Using the Fast Fourier Transformation (FFT), the time domain data is translated into accelerations ($Acc_{seat}(f)$, $Acc_{head}(f)$) in the frequency domain (Figure 6). Following equation 2 above, the transmissibility ($TR(f)$) is calculated as the ratio of the accelerations at each individual frequency sample (Figure 7).

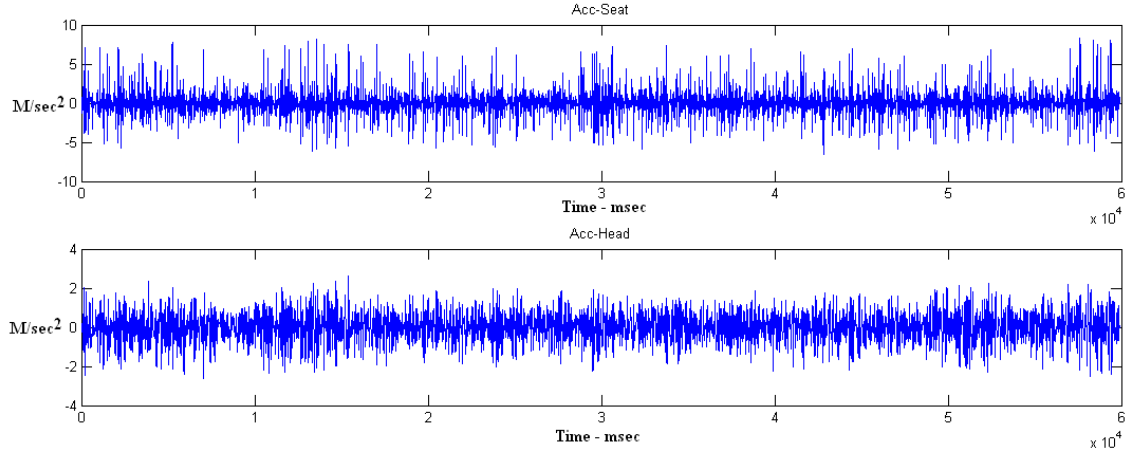


Figure 5: Example of time domain data of acceleration from an accelerometer located at the seat-person interface (above) and acceleration at the subject's head from an accelerometer held in the mouth with a bite-bar (below).

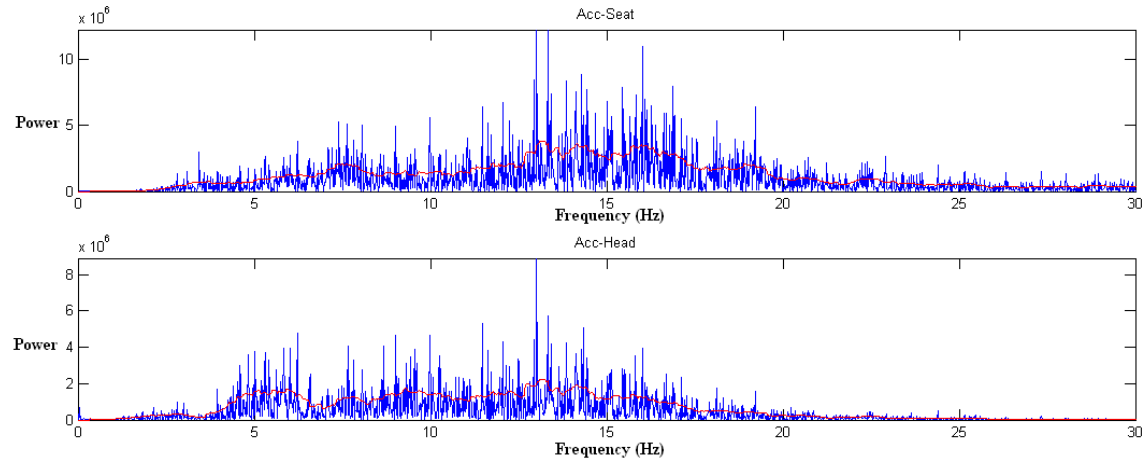


Figure 6: Example of frequency domain data of acceleration at the seat (above) and acceleration at the subject's head (below). Data is the Fast Fourier Transform of the data in figure 5.

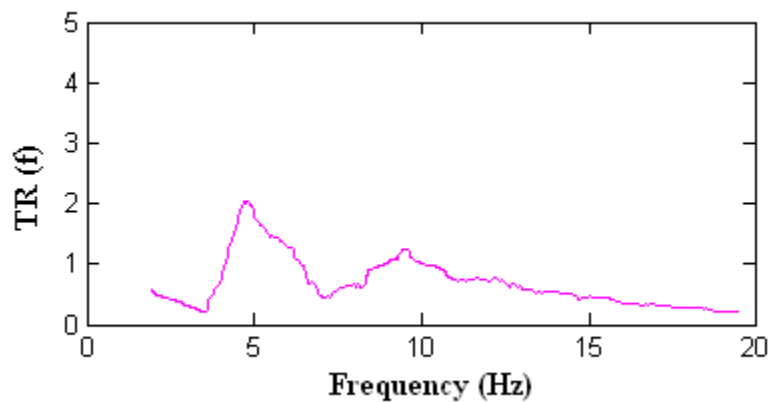


Figure 7: Example transmissibility frequency response function in the frequency domain (2-20 Hz). The graph is the results of the calculation using equation 2 and from the data shown in figures 5 and 6.

The data collection for the calculation of transmissibility can include sensors placed at the seat interface, at anatomical landmarks (typically applied on the skin), and bite-bars held in the mouth. Sensors located at anatomical landmarks require either knowledge of the skin/tissue localized movement (Kitazaki and Griffin, 1995) or fixation

to bony structures (Rubin et al., 2003). For ease of testing, a bite-bar can be used to measure the seat-to-head transfer function of the human body along the spinal column. Similar to apparent mass measures, transmissibility frequency response curves can be used to measure the characteristic frequencies and response of the human body (Figure 8).

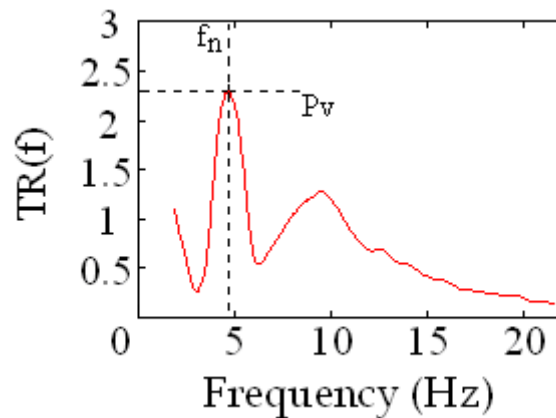


Figure 8: Example transmissibility frequency response curve (2-20 Hz). The frequency peak near 5 Hz signifies the natural frequency of transmissibility. The natural frequency of the system is related to the system stiffness, and the peak value is related to the system damping. The second peak in transmissibility near 10 Hz represents an additional characteristic frequency of the system. (Transmissibility is a unit less measure)

Transmissibility can be used to measure how the human body reacts to vibration exposure. It has been used to assess vibration on work vehicle drivers and helicopter pilots (De Oliveira and Nadal, 2005) as well as for designing wheelchairs (Hostens et al., 2003). Also, the understanding of the transmissibility of vibrations has been used to improve models of the human body (Boileau et al., 2002, Hinz et al., 2001).

Apparent mass and transmissibility are two common approaches used for studying the human biomechanical response during WBV. Apparent mass can be measured in laboratory tests requiring straightforward instrumentation measuring applied forces and resultant accelerations. For seated WBV, the measure of apparent mass is influenced primarily by the torso and contributions of the limbs can be controlled and limited. Measures of apparent mass can also be influenced by the effects of postural movements (Mansfield and Griffin, 2002, Mansfield et al., 2006). Transmissibility can be measured in field testing with unobtrusive measurements of accelerations. In the case of measuring seat to head transmissibility, attenuation along the entire spine contributes to the transmissibility. However, the bite-bar typically used in this assessment will also be influenced by movements of the head and neck. Though the transfer functions of apparent mass and transmissibility are measured differently, it was concluded that similar mechanisms influence their frequency response curves (Mansfield and Griffin, 2000).

(1.2.3.4) Human Vibration Dynamics

The human body's perception of WBV is a function of frequency (Mansfield, 2005a), with greatest sensitivity to seated vertical vibration at frequencies between 4 and 8 Hz (Parsons and Griffin, 1988). Investigators have found that these subjective ratings for the perception of vibration are related to the biomechanical response of the body to WBV (Mansfield and Maeda, 2005c). The study of the frequency response of the human trunk has been investigated initially using a series of vertical sinusoidal WBV inputs and

has progressed to random, broad-band vibrations (Fairley and Griffin, 1989). The results were similar between sinusoidal vibration and random, broad-band vibration, the latter of which has a better frequency resolution as well as a better definition of any resultant peaks (Mansfield and Maeda, 2005a). The frequency response of the trunk is non-linear (second order system) for both vertical WBV (Mansfield and Griffin, 2000) and different directions of horizontal WBV (Mansfield and Lundstrom, 1999). The results of initial studies that have quantified the natural frequency for vertical WBV of the human trunk found a major resonance in the region of 5 Hz and in some cases a second mode in the region of 10 Hz (Fairley and Griffin, 1989).

Using the frequency response functions of apparent mass and transmissibility, studies have investigated some effects of WBV on the human body. Increasing the magnitude of acceleration of WBV led to a ‘softening’ of the body with increase in magnitude (Mansfield and Griffin, 2000). This softening was reported as the decrease of the resonance frequency from about 6 Hz to 4 Hz when the magnitude of the vibration was increased from 0.25 to 2.0 m/s² root-mean-squared (Fairley and Griffin, 1989). In addition to studies where the magnitude of vibration was varied, one study measured the apparent mass as the direction of horizontal WBV vibration was changed. Similar to that of increasing vibration magnitude, the results for the resonance frequency decreased as the direction of horizontal WBV was changed incrementally from 0° (anterior/posterior) to 90° (medial/lateral) (Mansfield and Lundstrom, 1999). However, it has been reported that the frequency and peak in apparent mass in one direction are not related to that of another (Mansfield and Maeda, 2007).

In order to simulate work environments and study the effects of WBV on the human body, additional factors need to be taken into account. These can be considered in two parts, 1) the human component, and 2) the human/environment interfaces. Current standards for the human body's frequency response functions are based on average values of apparent mass or transmissibility and the existence of significant inter-subject differences and different test conditions can lead to misinterpretations (Hinz et al., 2001). In the case of the human component, the subject's body mass, height, posture, and state of muscular contraction needs to be considered (Fairley and Griffin, 1989). Body mass can be accounted for when calculating apparent mass by normalizing to the static mass, however transmissibility is not normalized to static mass. Also, a study concluded that the ISO 5982 guidelines are insufficient for comparison with Japanese people who have higher natural frequencies and peak values than the guidelines' range (Maeda and Mansfield, 2005). The study concluded that further research is needed to improve the standards when considering anthropometrics and the biodynamics of WBV. Possibilities for the variance from the standards mentioned in the study included controlling for factors of age and height. Additionally, a subject's muscle tension should also be taken into account (Mansfield et al., 2006) as WBV can induce a vibration-synchronous response from the trunk muscles when they are preloaded (Zimmermann et al., 1993) and increase the effective trunk stiffness (Lee et al., 2006).

The human/environmental interfaces can also have an influence on the human body's response to WBV and include the support of the feet and legs, hands on a steering

wheel, and the use of seat backrests. Compared to the static mass of the subject, a static foot rest (independent of vibrating seat) as it is lowered away from the seat will decrease the apparent mass results. There is an increase above static mass for free hanging feet and legs, and measurements closer to static mass when using a moving foot rest (moving with the seat) (Fairley and Griffin, 1989). In the case of accounting for hands on a steering wheel, there is the suppression of horizontal body rocking and changes to model parameters of stiffness and damping (Stein et al., 2007). The use of a back rest will increase the natural frequency and median normalized apparent mass of a subject (Mansfield and Maeda, 2005b).

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Chapter 2 :

Preface to Studies

Preface to Studies

Although WBV is a risk factor for LBP, there is no clear agreement for the mechanism as to how WBV exposure leads to LBDs. The three studies performed here aim to clarify how WBV may contribute to LBP. The first study investigated the after effects of WBV exposure on the postural control of the trunk. The results found an increase in trunk dynamics that suggest a loss in postural and spinal stability control, potentially a risk factor for LBP due to increased variability in trunk dynamics(Granata et al., 1999). The second study further investigated the change in spinal stability control by quantifying the effects of WBV on parameters of a control model. From the results of this study, there is a better understanding as to which subsystems (passive stiffness, active stiffness, neuromuscular reflex) are affected by WBV exposure. The third study investigated the progression of changes during the exposure to WBV. These three studies used spinal stability control as a mechanism that links WBV exposure and the risk for LBP. The results from these studies can be helpful in the understanding of how WBV leads to LBDs and aid in the development of guidelines for WBV exposure duration.

Does WBV exposure change spinal stability control?

Previous studies have reported some information that would suggest a possible change in spinal stability control due to WBV including sensory error and changes in muscular reflex response. These studies were limited to single frequency vibration and

different methods of applying trunk perturbations. The first study was designed to continue from these other studies and to assess changes to spinal stability control via measures of postural control. The postural control of the trunk was measured by a wobble chair before and after a period of controlled, broad-band random WBV. The measures of the wobble chair movement relate to the postural control of the trunk and infers to spinal stability control.

Study 1:

*Effects of Seated Whole-Body Vibration on Postural Control of the Trunk
During Unstable Seated Balance*

From this study, it was shown that spinal stability control, as measured by postural control, decreased with vibration exposure.

How is spinal stability control affected after WBV exposure?

From the results of the first study, we know that spinal stability control, as measured from postural control, was reduced due to WBV exposure. The first study was limited to reporting only if there is a change in spinal stability control. This second study further addresses the issue by assessing the changes in parameters of a spinal stability control model. Data was collected from trunk perturbations before and after vibration

exposure and system identification was used to quantify trunk stiffness, co-contraction recruitment, muscle reflex delay and gain.

Study 2:

Effects of Seated Whole-Body Vibration on Spinal Stability Control:

Stiffness and Muscle Reflex

From this study, it was shown that after seated WBV, trunk stiffness and muscle reflexes were decreased. There were no significant changes in reflex delays or recruitment of muscle co-contraction.

What changes in the human body during WBV exposure?

From the results from the first two studies, the change in spinal stability control after exposure to WBV was addressed. The third study was performed to assess the progressive changes in trunk dynamics during the WBV exposure leading to the reduction in spinal stability control. Data was collected from the seated subject during the WBV exposure. Frequency response functions of apparent mass and transmissibility were calculated in the frequency domain and natural frequencies and peak values were quantified. Changes in these two parameters infers to the biomechanical properties of trunk stiffness and damping respectively. Previous studies that have used apparent mass and transmissibility to study the spine have been limited to short duration (<1 min)

vibration exposure, and were performed to test the vibration characteristics of frequency, amplitude, and direction. The purpose of this study was to assess the vibration characteristic of exposure duration on the human trunk.

Study 3:

Changes in the Natural Frequency of the Trunk

During Seated Whole-Body Vibration

From this study, it was shown that trunk stiffness and damping decreased over time of WBV exposure with a trend for a rapid initial decrease in parameters.

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Chapter 3

Effects of Seated Whole-Body Vibration on Postural Control of the Trunk During Unstable Seated Balance

Gregory P Slota, MS

SLOTA, G. P., GRANATA, K. P. & MADIGAN, M. L. (2008). Effects of seated whole-body vibration on postural control of the trunk during unstable seated balance. *Clinical Biomechanics (Bristol, Avon)*,**23**(4): 318-6.

**Virginia Polytechnic Institute & State University
School of Biomedical Engineering & Sciences
The Kevin P. Granata Musculoskeletal Biomechanics Laboratory**

(3.2) Abstract

Background: Low back disorders and their prevention is of great importance for companies and their employees. Whole-body vibration is thought to be a risk factor for low back disorders, but the neuromuscular, biomechanical, and/or physiological mechanisms responsible for this increased risk are unclear. The purpose of this study was to measure the acute effect of seated whole-body vibration on the postural control of the trunk during unstable seated balance.

Methods: Twenty-one healthy subjects (age: 23 years (SD 4 years)) were tested on a wobble chair designed to measure trunk postural control. Measurements of kinematic variance and non-linear stability control were based on seat angle before and after 30 minutes of seated whole-body vibration (bandwidth = 2 – 20 Hz, root-mean-squared amplitude = 1.15 m/s²).

Findings: All measures of kinematic variance of unstable seated balance increased ($P < 0.05$) after vibration including: ellipse area (35.5%), root-mean-squared radial lean angle (17.9%), and path length (12.2%). Measures of non-linear stability control also increased ($P < 0.05$) including Lyapunov exponent (8.78%), stability diffusion analysis (1.95%), and Hurst rescaled range analysis (5.2%).

Interpretation: Whole-body vibration impaired postural control of the trunk as evidenced by the increase in kinematic variance and non-linear stability control measures during unstable sitting. These findings imply an impairment in spinal stability and a mechanism by which vibration may increase low back injury risk. Future work should investigate the effects of whole-body vibration on the anatomical and neuromuscular components that contribute to spinal stability.

(3.3) Introduction

Low back disorders (LBDs) are common, costly, and debilitating. For Example, 26.4% of adults of the United States reported having low back pain within the last three months (Deyo et al., 2006) and 60% to 80% experience low back pain (LBP) at some point in their lives (van Tulder et al., 2001, Kelsey et al., 1984). The total health care expenditures for people with LBP in the United States is over \$90 billion (Luo et al., 2004, Pope, 1996), while back pain and spine disorders were ranked in the top ten health and productivity expenditures (Goetzel et al., 2003). Numerous risk factors for LBDs have been identified including excessive spinal loading (Fathallah et al., 1998, Lee et al., 1991, Marras et al., 1993), atypical postures (Marras and Granata, 1995, Granata and Rogers, 2007, Keyserling, 2000), and spinal instability (Granata et al., 2004). Whole body vibration (WBV) is another risk factor for LBDs. The National Research Council (2001) reported that there is evidence of a “clear relationship between back disorders and ... whole-body vibration”. Six million workers are exposed to WBV (Griffin, 2006) typically while in a seated position including delivery vehicles drivers (Rosegger and Rosegger, 1960), forklift operators (Boshuizen et al., 1992), helicopters pilots (De Oliveira and Nadal, 2005), and construction equipment operators (Spear et al., 1976). Tractor drivers have reported 61-94% prevalence of LBP and pathological changes in the spine (Dupris, 1966), and heavy-equipment drivers report 70% prevalence of LBP (Cremona, 1972).

Despite the categorization of WBV as a risk factor for LBDs and its prevalence in the workplace, the neuromuscular, biomechanical, and/or physiological mechanisms by which WBV can contribute to low back injuries are not well understood (Dong et al., 2006, Griffin, 2006). Panjabi (1992a) decomposed the neuromusculoskeletal system that controls spinal stability into three subsystems: passive tissue stiffness, active muscular stiffness, and neuromuscular reflexes. Several studies have identified effects of WBV on some of these subsystems. In regard to the passive tissue stiffness subsystem, seated WBV increased the loss of height of the spine beyond the normal diurnal changes (Klingenstierna and Pope, 1987, Sullivan and McGill, 1990), and the majority of this loss of height is a direct result of creep of the intervertebral disc (Keller and Nathan, 1999). Creep deformation of the connective tissue can disrupt the passive stability of the spine and sensory organs contained within. In regard to the neuromuscular reflex subsystem, WBV has been reported to decrease reflex activity (Roll et al., 1980). The effects were reported to be due to alterations in the peripheral sensory motor system performance affecting the senses of position and effort (Gauthier et al., 1981). Although these studies illustrate the effects of WBV on the subsystems of passive tissue stiffness and neuromuscular reflexes, the effect of WBV on overall spinal stability and postural control is not known.

Measures of seated postural sway during unstable seated balance have been used as surrogate measures of trunk postural control (Cholewicki et al., 2000, Van Daele et al., 2007) and have been related to spinal stability (Reeves et al., 2006, Silfies et al., 2003). In addition, differences in seated postural sway have been identified between patients

with LBP and healthy individuals (Radebold et al., 2001). The goal of this study was to investigate the acute effects of seated WBV on the postural control of the trunk. Seated WBV was investigated because machine operators are typically exposed to vibration while seated. Measures of kinematic variance and non-linear stability control were recorded during unstable seated balance. It was hypothesized that WBV would impair postural control of the trunk, suggesting a loss of spinal stability and perhaps an increased risk for low back injury.

(3.4) Materials & Methods

A wobble chair was designed and constructed to measure unstable seated balance, and a platform was created to allow exposure to vertical seated WBV. The experimental design involved repeated measurements of unstable seated balance both before and after controlled exposure to seated WBV.

(3.4.1) Apparatus Design

The wobble chair design (Figure 1) was adapted from the concept by Cholewicki et al. (2000). The seat pivoted on a low friction ball-and-socket joint located under the center of the seat. Supplemental seat support was also provided by four springs (stiffness = 99.82 N/cm) spaced every 90° circumferentially that could be moved radially from the ball-and-socket to modulate task difficulty (as the springs are moved radially outward, resistance to rotation is increased and maintaining a stable seat becomes easier). The range of motion of the seat allowed approximately twelve degrees of rotation in any direction from the initial level position before the seat would impact the physical limits of the wobble chair. Affixed to the seat was an adjustable foot rest to limit the motion of the lower limbs during unstable seated balance testing. Two seat belts were used to restrict the movement of the pelvis with respect to the seat, thus limiting motion to only the spine. The anterior/posterior (A/P) position of the seat with respect to the ball-and-socket

joint was adjustable so that the equilibrium balance position of an upright trunk posture would coincide with a level seat platform.

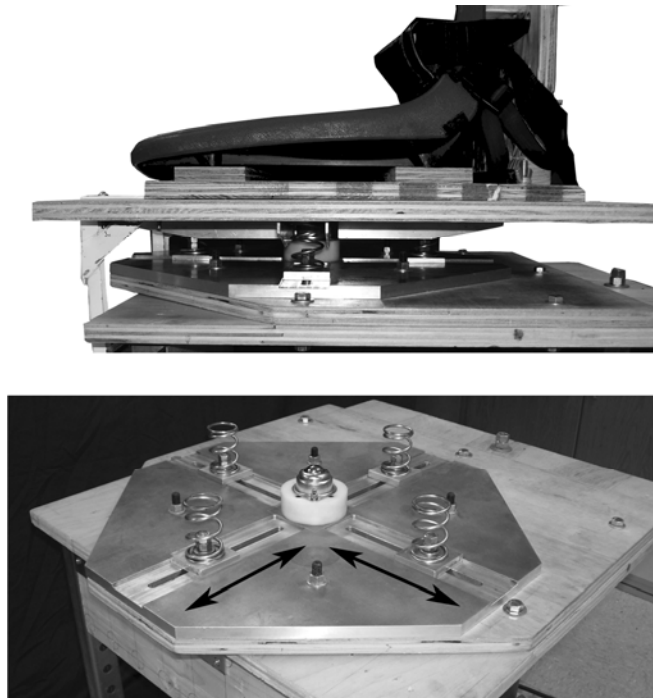


Figure 1: Wobble chair (above) and spring layout (below). The spring position was adjustable radially from the central ball-and-socket pivot point.

The wobble chair was designed (Figure 2) such that a tilt of the seat platform (θ_2) would result in a restorative force from the springs ($KL^2\sin(\theta_2)$) in proportion to the square of the radial position from the center (L). The springs resisted the gravitational moment of the subject's center of mass ($MGH\sin(\theta_1)$) (Equation 1). The springs could be positioned 7-22 cm from the center. This distance was converted to percent MGH to correspond to the ratio of spring force to gravitational force. MGH was calculated from two measurements of moment on a force plate (Equation 2) during static tilted postures at two different angles. More specifically, it was the ratio of the difference in moments over the differences in the angle ($\sin(\theta_a)-\sin(\theta_b)$) (Equation 3). A spring position of

$L=100\%_{MGH}$ (i.e. $KL^2 = MGH$) was considered neutrally stable (as a rigid mass would reach equilibrium at any angle). A greater percentage of MGH would create a more robustly stable seating condition approaching that of fixed seat. At a lower percent MGH, the seat was unstable without active control to balance. The adjustability of the springs and conversion to percent MGH allowed for testing at comparable levels of relative stability robustness regardless of individual subject's anthropometrics (differences in mass or center of mass location) as seen in previous studies (Cholewicki et al., 2000).

$$MGH \sin(\theta_1) = KL^2 \sin(\theta_2) \quad (1)$$

$$\begin{aligned} MGH \sin(\theta_a) &= My_a + e \\ MGH \sin(\theta_b) &= My_b + e \end{aligned} \quad (2)$$

$$MGH = \frac{(My_a + e) - (My_b + e)}{\sin(\theta_a) - \sin(\theta_b)} \quad (3)$$

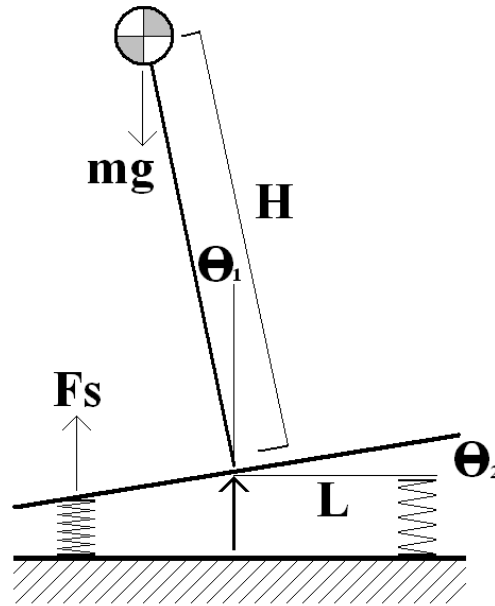


Figure 2: Model diagram of the wobble chair functionality. Springs used only in compression and are not attached to top surface.

The wobble chair was fixed to the top of a vibration platform so that each subject could be exposed to WBV or unstable seated balance trials without leaving the chair (Figure 3). This also allowed for the continued use of the seat belts and foot rest to restrict the pelvic and lower limb motions during WBV. The vibrating platform consisted of an electro-mechanical shaker (MB Dynamics PM-100, Cleveland, OH, USA) on the back of the platform, a spring in the middle to offset the subject's weight, and a hinge in the front to allow vertical movement of the chair. Using this configuration, the maximum vertical excursion of the subject was approximately 1.27cm above and below the neutral position during WBV.

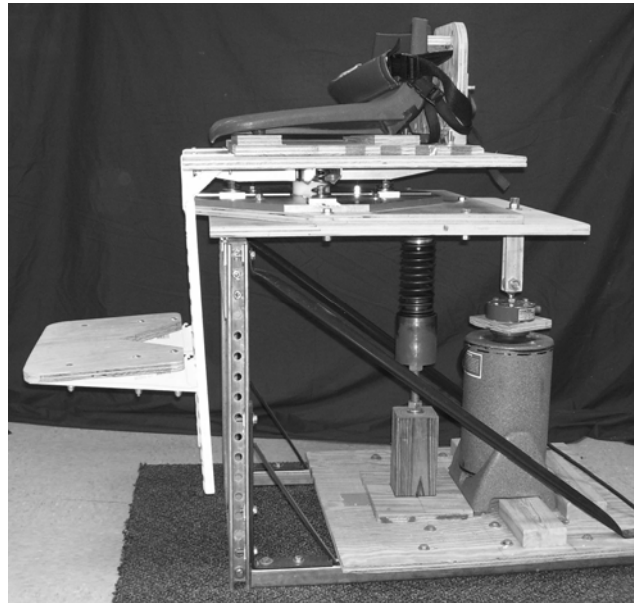


Figure 3: Vibration apparatus set-up with wobble chair on top.

An accelerometer (CXL04, Crossbow Inertial Systems, San Jose, CA, USA) was attached to the seat of the wobble chair inline with the base of the spine to monitor the

vibration characteristics. The shaker was driven by a Quanser Q8-I/O card (Quanser Consulting Inc., Markham, Ontario, Canada) connected to a SS530 Amplifier (MB Dynamics). Simulink (The MathWorks, Natick, MA, USA) code was used to generate in real-time a signal and control the shaker. The signal was created from a random number generator passed through discrete high-pass and low-pass filters set to 2 Hz and 20 Hz, respectively (transmissibility band of the torso (Griffin et al., 1982)). The filtered signal was adjusted by controlling the proportional gain based on the difference between a five second window of root-mean-squared (RMS) amplitude from the accelerometer and the desired acceleration for this test of 1.15 m/s^2 (International Organization for Standardization exposure limit (International Organization for Standardization, 1997)).

(3.4.2) Experimental Protocol

Testing was performed on 21 healthy adults (age: 23 years (SD 4 years) , height: 170.8 cm (SD 12.2 cm), mass: 73.89 kg (SD 13.97 kg), 13 males and 8 females) who had no previous history of low back pain. Fourteen of the 21 subjects completed a second experiment during which WBV was replaced with quiet sitting. The goal of this second control experiment was to identify any effects of prolonged sitting on postural control of the trunk. This study was approved by the Virginia Polytechnic Institute & State University Institutional Review Board, and all subjects provided informed consent prior to participation.

The experiment began by determining the desired spring position on the wobble chair. The desired spring position would be one where balance could be maintained without hitting the physical limits of the wobble chair, but still be sufficiently challenging. In a previous study measuring the differences in unstable seated balance, between LBP patients and healthy individuals, the differences were more apparent when sitting is more challenging (Radebold et al., 2001). Starting with a spring position of 13 cm (near $L=100\%_{\text{MGH}}$), unstable seated trials were performed. After each balance trial of the wobble chair, the spring positions were moved in 1-2 cm. This process continued until the range of motion of the seat tilt was constantly crossing a 7° radial tilt threshold and or the subject was unable to balance without hitting the physical limits. In the case of the latter, the springs were then moved out 0.5 cm between trials until the ability to balance was restored and the threshold was still being crossed. This final spring position was then used for all subsequent trials for that subject. This process typically required 6-8 trials. These trials also served as practice trials to 1) minimize any learning effects in the data collection trials, 2) allow the subject to acclimatize to the task. The mean spring position during all testing was $43.55\%_{\text{MGH}}$ (SD $8.4\%_{\text{MGH}}$).

Four unstable seated balance trials were collected while the subject sat on the wobble chair. Each subject was instructed to sit with an upright posture, eyes open, and their arms folded across their chest. Each trial lasted 60 seconds, and a minimum of one minute rest was allowed between trials to minimize fatigue. After these trials, WBV was applied for 30 minutes while the subject sat on the wobble chair after locking it in place. Immediately following WBV, three more trials of unstable seated balance were

performed as before WBV. Rest periods of less than ten seconds between trials were used to minimize any potential recovery from WBV.

The control group completed an additional experimental session within one week of the WBV test session. During this session, each subject performed the same protocol as described, but the 30 minutes of WBV was replaced with quiet sitting between the measurements of unstable seated balance on the wobble chair. The same spring settings as in the vibration session were used.

Wobble chair angular position was sampled at 100 Hz during all trials using a tri-axial angle sensor (X-sens Motion Technologies, Netherlands). The sensor was oriented such that rotation in the pitch and roll directions were aligned with anatomical directions of A/P and medial/lateral (M/L) respectively. Calculations were also performed in the resultant radial (RR); resultant lean angle. Kinematic variance of the angle data were determined using common measures of postural sway during quiet standing including the 95% ellipse area, RMS (A/P, M/L, and RR), and path length (Prieto et al., 1996). Non-linear stability control measures of the data consisted of Lyapunov exponent, stability diffusion analysis, and Hurst rescaled range analysis. The Lyapunov exponent looks at the rhythmic activity of the system in state space as a limit cycle. The process quantifies the exponential separation of trajectories of nearest neighbors with time (Harbourne and Stergiou, 2003). The analysis used the 4 dimensional state dynamics (two angles, two angular velocities) to calculate the nearest neighbors (Kang and Dingwell, 2006). Stability diffusion analysis allows for correlation between past and future displacements

and summarizes the mean square displacement as a function of the time interval between comparisons (Collins and De Luca, 1993). The analysis allows for the system to be broken down into two parts, short-term open-loop control (reported here as HS-A/P, -M/L, -RR) and long-term closed-loop control. The long-term stability diffusion analysis parameter is not representative of a bounded system (Delignieres et al., 2003) and therefore was not used in this study. The Hurst rescaled range analysis describes the fractal properties of fluctuations in measurements during postural control (Duarte and Zatsiorsky, 2000) (reported here as HR-A/P, -ML, -RR). All calculation of non-linear stability control measures were made with custom-written code in Matlab (The MathWorks, Natick, MA, USA) by our lab.

Initial analysis resulted in no significance due to learning effects, therefore all four trials from each subject before vibration were averaged, and all three trials after vibration were averaged. One subject had to be excluded from the analysis because they were unable to balance on the wobble chair after vibration. Data from the two experimental sessions were analyzed separately due to the differing number of subjects. The effects of vibration on kinematic variance and non-linear stability control measures of unstable seated balance were investigated using a one-way repeated measures ANCOVA. The independent variable was vibration (before and after), and the covariate was normalized spring position (%_{MGH}) because it was shown to influence some measures of kinematic variance and non-linear stability control. The effects of 30 minutes of sitting were investigated using the same statistical test. All statistical analysis

was performed using Stastica (StatSoft, Inc., Tulsa, OK, USA) with $\alpha=0.05$ indicating statistical significance.

(3.5) Results

WBV increased all measures of kinematic variance (Table 1). For example, ellipse area increased 35.5% (SD 50.5%) ($P < 0.01$), RMS-RR increased 17.9% (SD 21.0%) ($P < 0.01$), and path length increased 12.2% (SD 15.1%) ($P < 0.01$). WBV also increased all non-linear stability control measures (Table 2). Lyapunov exponent increased 8.78% (SD 10.9%) ($P < 0.01$), and all three directions of stability diffusion analysis and Hurst rescaled range analysis increased including HS-RR increasing 1.95% (SD 2.8%) ($P < 0.01$), and HR-RR increasing 5.2% (SD 4.0%) ($P < 0.01$). Thirty minutes of sitting did not affect most measures of kinematic variance and non-linear stability control measures. Only RMS-M/L decreased 7.6% (SD 12.0%) ($P = 0.027$), and HS-M/L decreased 1.0% (SD 1.3%) ($P = 0.019$) after prolonged sitting.

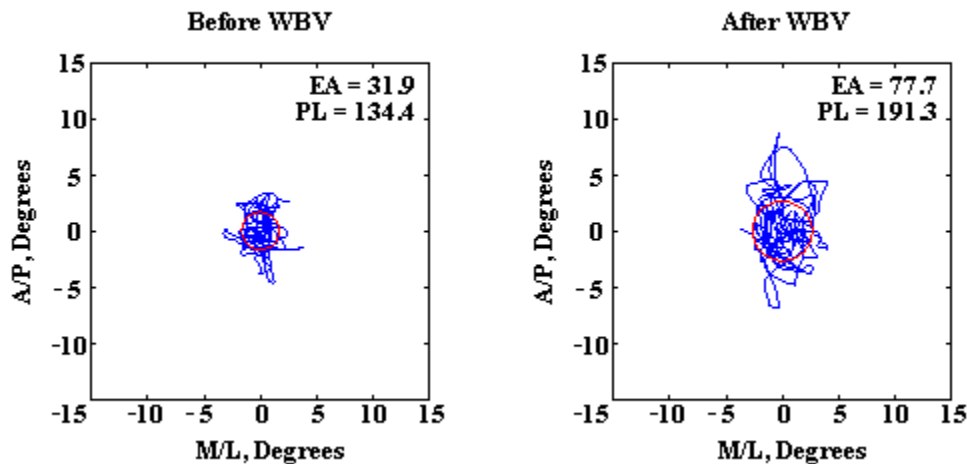


Figure 4: Example stabilogram data of before and after WBV exposure. The circle represents the area of the average distance from equilibrium. Also labeled are ellipse area (EA) and path length (PL). Units are degrees of seat tilt.

Table 1: Kinematic variance measures (Mean (SD)) of unstable seated balance both before and after vibration exposure.

Variable	Before	After	<i>P</i> -value
Ellipse Area (degrees)	58.600 (40.468)	79.390 (40.864)	0.005*
RMS-A/P (degrees)	1.461 (0.614)	1.801 (0.626)	0.003*
RMS-M/L (degrees)	1.835 (0.437)	2.283 (0.569)	p<0.001*
RMS-RR (degrees)	2.448 (0.778)	2.887 (0.767)	0.001*
Path Length (degrees)	173.179 (60.275)	194.338 (60.134)	0.002*

(* indicates p<0.05)

Table 2: Stability assessment measures of unstable seated balance (Mean (SD)) both before and after vibration exposure.

Variable	Before	After	<i>P</i> -value
Lyapunov Exponent	0.613 (0.082)	0.667 (0.074)	0.002*
HS-A/P	0.643 (0.028)	0.652 (0.029)	0.039*
HS-M/L	0.659 (0.015)	0.668 (0.019)	0.041*
HS-RR	0.608 (0.022)	0.620 (0.025)	0.006*
HR-A/P	0.886 (0.030)	0.929 (0.021)	p<0.001*
HR-M/L	0.905 (0.024)	0.930 (0.021)	p<0.001*
HR-RR	0.853 (0.024)	0.897 (0.026)	p<0.001*

(* indicates p<0.05)

(3.6) Discussion

The goal of this study was to investigate the effects of seated WBV on the postural control of the trunk. All measures of kinematic variance and non-linear stability control during unstable seated balance increased following WBV, suggesting that the postural control of the trunk was impaired. It is commonly believed that greater variability indicates greater instability (Dickstein and Laufer, 2004, Dingwell and Marin, 2006). Therefore, the results of this study imply an impairment of spinal stability with WBV. The results of the control group analysis suggest that the changes in postural control were not due to sitting alone.

The changes in postural control of the trunk during unstable seated balance reported here imply an impairment of spinal stability. Spinal stability is maintained through contributions from passive tissue stiffness, active muscular stiffness, and neuromuscular reflexes. The effect of WBV on any of these subsystems could conceivably alter postural control of the trunk (Reeves et al., 2007), and explain the changes found in the present study. Wilder et al. (1996) investigated the effects of seated, sinusoidal WBV on neuromuscular reflexes elicited from a sudden trunk flexion loading from a hand-held box. Erector spinae reaction times increased from 87 msec to 108 msec, and reflex amplitudes from 55% to 67% maximum voluntary contraction. In a similar study, Li et al. (2007) investigated the effects of seated WBV on trunk kinematics and neuromuscular reflexes in response to a sudden trunk flexion loading applied to the trunk. Maximum trunk flexion angle increased from 11.8 to 13.2 degrees, and time to

peak muscle response increased from 205 msec to 228 msec. In addition, the position sense error of the torso increased 1.58-fold. The increase in peak muscle response time was therefore attributed to an increase in the movement threshold to elicit a reflex. These studies indicate that seated WBV impairs neuromuscular reflexes, which can impair postural control of the trunk, and spinal stability (Franklin and Granata, 2007). Perhaps related to this increase in movement threshold, Panjabi (1992b) described a change in the passive tissue stiffness due to an increase in the range of the neutral zone (i.e. the range of motion where spinal motion is produced with a minimal internal resistance). Seated WBV has also been shown to affect the passive stiffness components by causing creep deformation in intervertebral discs (Klingenstierna and Pope, 1987), which could also increase the neutral zone and affect postural control of the trunk and spinal stability (Courville et al., 2005, Panjabi, 1992b).

A few limitations to this study are worth noting. First, this study investigated WBV with specific characteristics (RMS amplitude, frequency content, duration). This amplitude was selected to match the exposure limit as set by the International Organization for Standardization. Vibration with different characteristics may have different effects on unstable seated balance. Second, this study only investigated the acute effects of WBV. It is unclear how long these effects persist. Third, although all dependent variables exhibited statically significant changes after WBV, it is unclear if these changes are clinically meaningful. Effect sizes ranged from 0.31 to 2.07 (average 1.19), which we feel would likely reveal meaningful changes, but follow up studies are required to validate. Fourth, this study implicitly attributed the changes in the postural

control of the trunk with WBV to aspects directly-related to the control of spinal stability (i.e. passive tissue stiffness, active muscular stiffness, and neuromuscular reflex control). However, WBV could have influenced other systems or factors such as the vestibular (Gauthier et al., 1981, Roll et al., 1980) and visual systems (Ishitake et al., 1998), which in turn influenced our dependent measures.

(3.7) Conclusion

In conclusion, WBV impaired postural control of the trunk, which may indicate impaired spinal stability. Future studies are required to further explore the affects of WBV on the subsystems of the neuromusculoskeletal system that controls spinal stability, as well as to understand the effects of different vibration characteristics, exposure durations, and recovery time-line.

Acknowledgements

This work is dedicated to Dr. Kevin P. Granata, whose contributions started this study, but was tragically killed on April 16, 2007 at Virginia Tech, and was not able to see it through to completion. Also to be acknowledged are Duncan Hall for his contributions on the vibration platform construction and software control, and Emmanuel Ekwueme for his assistance with data collections.

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Chapter 4

Effects of Seated Whole-Body Vibration on Spinal Stability Control: Stiffness & Muscle Reflex

Gregory P Slota, MS

**Virginia Polytechnic Institute & State University
School of Biomedical Engineering & Sciences
The Kevin P. Granata Musculoskeletal Biomechanics Laboratory**

(4.2) Abstract

Background: Whole-body vibration is a risk factor for low back disorders, but the neuromuscular, biomechanical, and/or physiological mechanisms responsible for this increased risk are unclear. The purpose of this study was to measure the effect of seated whole-body vibration on spinal stability control parameters.

Methods: Twenty healthy subjects were exposed to a series of pseudorandom force perturbations applied to the trunk both before and after 30 minutes of seated whole-body vibration (2 – 20 Hz and 1.15 m/s² root-mean-squared amplitude). System identification techniques were performed to quantify total trunk stiffness, co-contraction recruitment, and erector spinae reflex delay and gain.

Findings: Total trunk stiffness and erector spinae reflex gain decreased significantly after exposure to seated whole-body vibration. Vibration did not change erector spinae reflex delay. Active muscle co-contraction recruitment was also not found to change due to vibration exposure.

Interpretation: Seated whole-body vibration altered trunk stiffness (passive stiffness and effective reflex stiffness) as well as reflex dynamics. All of which contribute to spinal stability. There was no evidence of compensation for decreases in trunk stiffness and reflex gain by active muscle co-contraction recruitment. This research helps to elucidate a biomechanical mechanism by which vibration may increase low back injury risk.

(4.3) Introduction

Low back disorders (LBDs) afflict a large percentage of workers every day, and enact a large economic toll in terms of medical costs and lost work time. They affect 59% to 80% of U.S. workers (Kelsey et al., 1984), cost approximately \$100 billion a year in total compensation (Pope, 1996), and account for 25% of all lost work days in industry (Guo et al., 1995). Numerous risk factors for LBDs have been identified including excessive spinal loading (Fathallah et al., 1998, Lee et al., 1991, Marras et al., 1993), atypical postures (Marras and Granata, 1995, Keyserling, 2000), and spinal instability (Granata et al., 2004). Whole-body vibration (WBV) has also been identified as a risk factor for LBDs (Waters et al., 2007). In fact, the National Research Council (2001) reported that there is evidence of a “clear relationship between back disorders and ... whole-body vibration”. In support of this, tractor and heavy-equipment drivers have reported 61-94% and 70% prevalence of low back pain (LBP), respectively (Dupris, 1966, Cremona, 1972).

Despite the characterization of WBV as a risk factor for LBDs, the neuromuscular, biomechanical, and/or physiological mechanisms by which WBV contributes to low back injuries are not well understood (Dong et al., 2006, Griffin, 2006). The neuromusculoskeletal system that contributes to the control of spinal stability can be thought of as three subsystems: passive tissue stiffness, active muscular stiffness, and neuromuscular reflexes (Panjabi, 1992a). Passive tissue stiffness, which is attributed to bony and ligamentous structures, is only able to support minimal compressive loads up

to 88 N without buckling (Crisco, 1989), and is insufficient to independently provide stability. Active muscle stiffness, which is attributed to muscle co-contraction, is able to support larger loads, but requires an elevated energy cost due to maintaining constant muscular activity. Neuromuscular reflexes provide a reactive component that responds to disruptions in spinal alignment. Up to 40% of the total stiffness from all three subsystems has been attributed to the contribution of neuromuscular reflexes which the authors called effective reflex stiffness (Moorhouse and Granata, 2007).

Several studies have identified the effects of WBV on these subsystems. In regard to the passive tissue stiffness, seated WBV increased the loss of height of the spine beyond the normal diurnal changes (Klingenshierna and Pope, 1987, Sullivan and McGill, 1990). The majority of this height loss was a direct result of intervertebral disc deformation (Keller and Nathan, 1999). In terms of the neuromuscular reflexes, WBV has been reported to decrease reflex activity. Seated WBV, leg vibration, and head-trunk vibration each resulted in reflex inhibition for tendon-stretch, tonic vibration, and Hoffmann reflexes in the soleus muscle (Roll et al., 1980). Studies of trunk perturbations found increased erector spinae reaction times (i.e. reflex delay) after exposure to seated WBV in addition to increased torso angular displacement and muscle reflex amplitudes (Wilder et al., 1996, Li et al., 2008). These effects have been attributed to an increase in the movement threshold necessary to elicit a reflex (Li et al., 2008), or a disturbance to the proprioceptive system creating errors in position, velocity, and force control (Gauthier et al., 1981). These changes can decrease spinal stability and increase the risk of LBP. However, another study that used a protocol of 60 minutes random WBV found

conflicting results. It was reported that there were no significant effects of WBV on balance or EMG reflex gain or delay (Santos et al., 2008). These conflicting results may be due to differences in vibration protocols and data analysis methods. All three studies used sudden loading protocols to elicit reflexes. Wilder et al. (1996) used a hand-held crate and Li et al. (2008) used trunk perturbations, both measuring from force onset to reflex peak while Santos et al. (2008) used trunk perturbations and reflex onset time. Additionally, the studies that found effects of WBV used 5 Hz sinusoidal vibration (0.223 and 0.315 m/s² RMS) targeting the natural frequency of the trunk while the other used a seated posture and random WBV exposure (0.5-20 Hz, 0.86 m/s² RMS) to simulate a mining trunk.

This lab has also been investigating the effects of seated WBV on the neuromusculoskeletal system. A previous study reported increased seated postural sway during unstable seated balance after exposure to seated WBV (Slota et al., 2008). These results suggest changes in seated postural control, and potentially spinal stability, which conflicts with the balance tests of Santos et al. (2008). Based on the change in seated balance and using the same vibration protocol, the goal of this current study was to measure the effects on the three subsystems of the neuromusculoskeletal system that contribute to seated postural control and spinal stability. Understanding the effects of WBV on these subsystems will help elucidate how WBV influences the control of the spine, and may contribute to the development of interventions to help prevent LBDs.

(4.4) Methods

This experiment was performed on 20 healthy adults who had no history of LBP. Subjects included ten males and ten females with a mean (standard deviation) age of 24.8 years (SD 6.2 years), height of 171.5 cm (SD 8.8 cm), and mass of 69.0 kg (SD 10.6 kg). This study was approved by the Virginia Polytechnic Institute & State University Institutional Review Board, and each subject provided informed consent prior to participation.

Each subject completed two experimental sessions separated by approximately one week in an order that was counter-balanced across all subjects. During both sessions, measurements to determine total trunk stiffness, muscle reflex gain and delay, and muscle activity were collected before and after an intervention. The interventions were either 30 minutes of seated WBV or 30 minutes of quiet sitting. Quiet sitting was investigated to separate the effects of seated WBV from any effects due to simply sitting or sequential measures.

(4.4.1) Experimental Protocol

The subject was assisted into the testing apparatus seat (Figure 1) and secured to the seat with two seatbelts to restrict pelvic movement. A footrest limited lower limb movement. Four maximum voluntary contractions (MVC) in trunk flexion and extension

were performed. Flexion exertions were performed by having the subject press their chest against a rigid board wedged between the subject and the wall they were facing. Extension exertions were performed by having the subject extend their back against a rigid board that was attached to a load cell. The force during these MVCs was collected from the load cell and used to scale the baseline force during trunk perturbation trials.



Figure 1: Experimental set-up for anteriorly-directed forces applied to the trunk. A servo motor with torque cell and pulley provided controlled force levels applied to the subject via a chest harness and cable system.

To quantify total trunk stiffness and reflex response, three 40-second trials of pseudorandom force perturbations applied to the trunk were performed using an apparatus described elsewhere (Granata et al., 2005, Moorhouse and Granata, 2005). A chest harness was attached with a cable to a pulley on a servo motor (Pacific Scientific, Rockford, IL, USA). The motor was computer controlled to apply an anteriorly-directed baseline force to the trunk that required a $20\%_{MVC}$ trunk extensor torque to remain

upright. Trunk perturbations consisted of pseudorandom, anteriorly-directed, force perturbations of 33% of the subject's combined head, arms, and trunk weight (58% of total weight (Granata and Rogers, 2007)) ranging 50-150 msec in duration and rest periods. Each trial consisted of: 5 seconds of ramp loading from zero torque to baseline, 3 seconds of sustained baseline, 30 seconds of perturbations superimposed on the baseline (Figure 2), and 2 seconds of ramping down to zero torque. The subject was instructed to not co-contract their trunk flexors, and this was monitored using EMG. Rest periods were provided between trials to avoid neuromuscular fatigue.

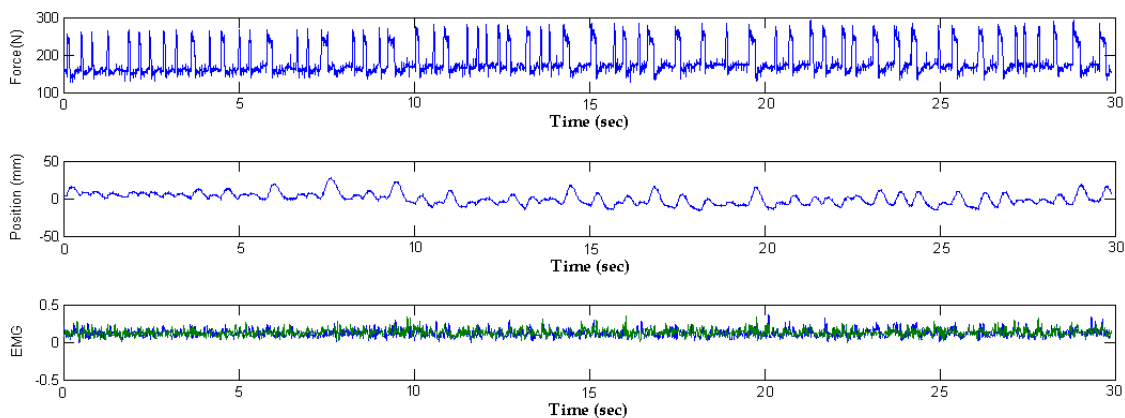


Figure 2: Example data of a) forces (N) applied to the trunk, b) position displacement (mm) of the trunk, and c) ES muscle activity (normalized left and right signals).

After these measurements, seated WBV was applied for 30 minutes. The subject sat on a vibrating platform (Figure 3) that consisted of an electro-mechanical shaker (MB Dynamics PM-100, Cleveland, OH, USA) on the back of the platform, a spring in the middle to offset the subject's weight, and a hinge in the front to allow for primarily vertical movement of the seat. Using this configuration, the maximum vertical excursion of the seat observed during testing was approximately +/- 1.27cm during vibration. The

shaker was driven by a Quanser Q8-I/O card (Quanser Consulting Inc., Markham, Ontario, Canada) connected to a SS530 Amplifier (MB Dynamics, Cleveland, OH, USA). Simulink (The MathWorks, Novi, MI, USA) code was used to generate a signal and control the shaker. The input signal to the shaker was white noise with a frequency range of 2-20 Hz (transmissibility band of the torso (Griffin et al., 1982)) using low-pass and high-pass filters and maintaining an root-mean squared (RMS) amplitude of acceleration of 1.15 m/s^2 (ISO 2631-1 (1997) exposure limit for 30 minutes of WBV) applied to the seat pan at the base of the spine. An accelerometer (CXL04, Crossbow Inertial Systems, San Jose, CA, USA) was attached to the seat platform of the chair in-line with the base of the spine. Feedback control in the software used the accelerometer signal to maintain the desired level of vibration in terms of RMS amplitude of acceleration.

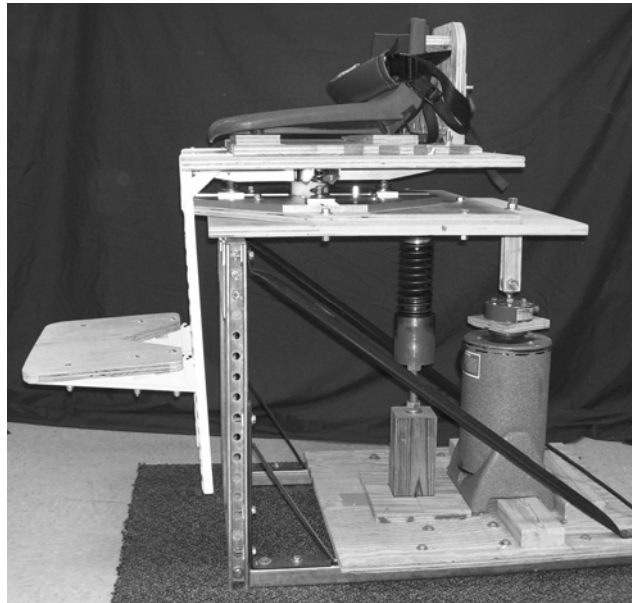


Figure 3: Vibration apparatus set-up with chair on top.

Immediately following the vibration, the vibration platform was locked in place and three more 40 second trials of trunk perturbations were performed. Time between trials was kept at less than ten seconds to minimize potential recovery from WBV.

During the experimental session with quiet sitting, each subject performed the same protocol as described, but was not exposed to vibration during the 30 minutes between the measurement sets of trunk perturbations. The same seated posture as well as preload and perturbation settings were used for both test sessions, scheduled one week apart in a counter-balanced design.

(4.4.2) Data Collection / Analysis

During all trunk perturbation trials, trunk position was sampled at 60 Hz using a three degree of freedom rotational position sensor (X-sens Motion Technologies, Netherlands) placed on the subject's manubrium. Motor torque, position, and velocity were sampled at 1000 Hz from a torque cell (Omega, Stamford, CT, USA) and motor encoder. Electromyogram (EMG, Measurement Systems Inc, Ann Harbor , MI) data was sampled at 1000 Hz from the right and left erector spinae (ES) and rectus abdominus (RA) muscles using electrode placement reported by Marras and Mirka (1992). EMG data were band-pass filtered from 20-450 Hz, band-stop filtered at 60 Hz, rectified, and integrated with a 25 Hz low pass, zero phase shift, seventh-order Butterworth filter.

Total trunk stiffness and muscle reflex delay and gain were quantified using system identification methods (Moorhouse and Granata, 2005). By using pseudorandom trunk perturbations and system identifications, results are based on an average of numerous (greater than 50) trunk loading events. The reaction of the trunk to the applied force, be it changes in position or muscle response, is accounted for within the transfer function for stiffness or muscle reflex. Total trunk stiffness, which includes potential contributions from all three subsystems contributing to the control of spinal stability, was calculated from the transfer function of the torque applied by the motor and the trunk angle displacement. Muscle reflex parameters of gain and delay were quantified from the transfer function of the ES muscle activity and the trunk angle displacement.

Agonist/antagonist co-contraction of trunk flexor and extensor muscles was quantified using recordings of the RA muscle activity averaged across the perturbation trials. The average RMS value of muscle activity during each trunk perturbation trial signified the baseline recruitment level. Values were compared from trials before WBV to those measured after.

Initial analysis verified that there were no trends due to learning effects so that all three trials from each subject before vibration were averaged, and all three trials after vibration were averaged. Additionally, reflex and muscle activity parameters were averaged across the left and right sides since the task was symmetric about the sagittal plane. A two-way repeated measures ANOVA was used to investigate the effects of intervention (WBV or quiet sitting), time (before after the intervention), and their interaction on total trunk stiffness, muscle reflex gain and delay, and RA muscle activity.

Post-hoc comparisons following a significant interaction were performed using Tukey's HSD. All statistical analysis were performed using Stastica (StatSoft, Inc., Tulsa, OK, USA) with $\alpha=0.05$ indicating statistical significance.

(4.5) Results

Total trunk stiffness (Figure 4a) exhibited a main effect of intervention ($P=0.025$), time ($P=0.007$), and an interaction between time and intervention ($P=0.041$). Post-hoc analysis showed a 12.5% (SD 15.4%) decrease in trunk stiffness with WBV ($P=0.003$), but no changes with quiet sitting ($P=0.765$). There was also a difference in pre-intervention measures of trunk stiffness ($P=0.009$). RA muscle activity (Figure 4b) exhibited no main effect of intervention ($P=0.348$), time ($P=0.325$) or their interaction ($P=0.484$). Reflex gain (Figure 4c) exhibited no effect of intervention ($P=0.118$), an 8.7% (SD 22.0%) decrease with time ($P=0.007$), and no significant interaction between intervention and time ($P=0.262$). Finally, ES reflex delay (Figure 4d) exhibited no main effect of intervention ($P=0.303$), time ($P=0.148$), or their interaction ($P=0.416$).

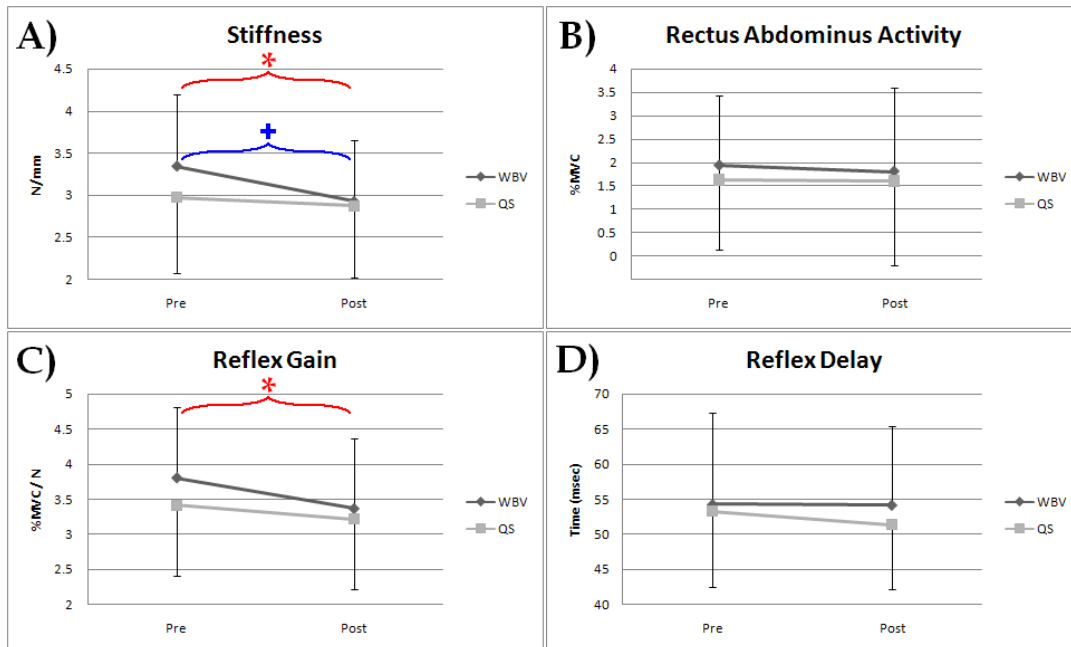


Figure 4: Results of the dependant measures with respect to exposure to WBV and quiet sitting for a) total trunk stiffness, b) RA activity, c) reflex gain, d) reflex delay. (* time as a main effect $P<0.05$, + post-hoc WBV $P<0.05$)

(4.6) Discussion

The goal of this study was to measure the effects of seated WBV on the three subsystems of the neuromusculoskeletal system that contribute to seated postural control and spinal stability. Results demonstrated that seated WBV significantly reduced total trunk stiffness, and that both interventions of seated WBV and quiet sitting reduced reflex gain when exposed to trunk flexion perturbations. Neither seated WBV nor quiet sitting affected trunk flexor activity or reflex delay. The decrease in total trunk stiffness and reflex gain with seated WBV suggest that spinal stability control was impaired.

Trunk stiffness decreased with seated WBV. This total trunk stiffness incorporates passive stiffness, active stiffness, and effective reflex stiffness. Because it included contributions from all three subsystems, it is difficult to determine which subsystem is responsible for the change in total trunk stiffness. However, other variables also collected in this study can help to begin to isolate changes to the individual subsystems. Results showed that ES reflex gain was decreased 8.7% due to 30 minutes of maintaining a seated posture for both interventions. Though there was not a significant interaction of time and intervention, post-hoc analysis showed that the 11.5% decrease for WBV was significant ($P=0.008$) while the 5.61% in quiet sitting was not ($P=0.114$), however these results are ambiguous. The decrease in reflex gain can reduce the trunk stiffness because ES reflexes has been reported to account for up to 42% of trunk stiffness (Moorhouse and Granata, 2007). However, the decrease in reflex gain was significant as a result of maintain the seated posture while the decrease in total trunk

stiffness was only significant for seated WBV and not the quiet sitting. Thus, it appears that the decrease in reflex gain due to maintaining a seated posture was not sufficient by itself to cause the decrease in total trunk stiffness due to seated WBV. The decrease in total trunk stiffness would therefore have to be due in part by the decrease in reflex gain and by changes in the active and or passive stiffness subsystems. Active stiffness is controlled through co-contraction of the trunk flexor and extensor muscles, and was monitored by the RA muscle activity. Results showed that seated WBV did not affect the average RA muscle activity, which indicates that the active stiffness subsystem did not change with WBV. This indicates that the decrease in total trunk stiffness may be due, in part, by a decrease in passive stiffness. Although passive stiffness was not directly measured in this study, it has been shown that vibration can lead to compression of the intervertebral discs (Klingenstierna and Pope, 1987) and creep deformation of the connective tissue due to cyclic loading (Solomonow et al., 2000). These changes can result in a decrease in stiffness and increase in joint laxity (Panjabi, 1992b). This leads to the conclusion that the decrease in total trunk stiffness due to seated WBV was possibly due to the combination of reduced passive stiffness and reflex gain. This decrease in trunk stiffness will impair spinal stability control.

Of the three neuromusculoskeletal subsystems that contribute to spinal stability, active stiffness and reflexes have demonstrated the ability to compensate when one subsystem is compromised. For example, external conditions of increases in the height a load is held (Granata and Orishimo, 2001) or decreases in person-object interface stability (Lee and Granata, 2006) increase the overall stability requirements for the spine.

In both studies, the increased stability requirements were compensated for by increased trunk flexor/extensor muscle co-contraction which increased active trunk stiffness (Lee et al., 2006). Other factors that can impair spinal stability include prolonged trunk flexion (Granata et al., 2005) and trunk muscle fatigue (Granata et al., 2004). Prolonged flexion, which reduced passive trunk stiffness, resulted in increased muscle reflex gain as compensation (Granata et al., 2005). Trunk muscle fatigue results in reduced force generation capabilities (Vollestad, 1997) and increased reflex delays (Wilder et al., 1996). These changes can impair stability (Franklin and Granata, 2007) and it has been shown to result in compensation events including increased co-contraction recruitment (Granata et al., 2001, Granata et al., 2004) as well as increased muscular reflex gain (Herrmann et al., 2006).

Including the current study, four different reports have been made on the effects of WBV on the neuromuscular control of the trunk (Li et al., 2008, Santos et al., 2008, Wilder et al., 1996). Two studies have used 5 Hz sinusoidal vibration, targeting the natural frequency of the trunk, and different neuromuscular methods to measure changes in reflex responses. These studies reported significant effects of WBV on the delay of reflexes. The third study used random vibration in a simulation of a mining vehicle. Their vibration characteristics consisted of a 0.5-20 Hz frequency bandwidth with the peak frequency centered around 2.7 Hz, below the natural frequency of the trunk. Unlike the other studies, the environment included a suspension-type seat with arm rests and a steering wheel. Their results showed no effects on balance performance or muscle reflex gain or delay. A previous study performed in our lab, and the current study reported

here, used a 2-20 Hz bandwidth with even distributed frequency content. It was demonstrated that seated postural balance was impaired and further that shown that reflex gain and trunk stiffness were decreased due to WBV exposure. Additionally, the current study differs on how reflex dynamics were assessed. Two of the previous studies used reflex onset time and found conflicting results. The other study used time to reflex peak, while the current used the transfer function of numerous hits, both finding significant changes due to WBV exposure.

The results of the current seated WBV study suggest that passive trunk stiffness was decreased in addition to decreased reflex gain, both of which can impair spinal stability control. This study also did not provide any evidence of the neuromuscular systems compensating for a loss in spinal stability by either increased co-contraction recruitment or increased reflex gain. One possibility that explains the lack of compensation would be if the stability control systems did not perceive that there was impairment in control due to errors in the sensory information. This is suggestive that WBV exposure can disrupt the proprioceptive system creating errors in position, velocity, and force control (Gauthier et al., 1981). WBV exposure resulted in changes in trunk proprioceptive acuity increasing maximum torso flexion angle from 11.8° to 13.2° in response to sudden loadings to a chest harness (Li et al., 2008). This result was coupled with a 1.58-fold increase in the position sense error of the torso after WBV exposure. Errors in the sensory information can possibly explain the impairment of compensation mechanisms in response to impaired spinal stability control.

Several limitations of this study warrant discussion. Trunk stiffness was only measured in terms of the total trunk stiffness. The elements of passive stiffness, active stiffness and effective reflex stiffness were not independently measured. Assessment of active stiffness and effective reflex stiffness were inferred from measures of muscular activity with EMGs. Future studies should consider directly measuring each component in order to attribute changes to each subsystem. Secondly, there is a potential problem with the measurement of trunk stiffness in that the values before WBV were greater than that before quiet sitting. The same protocol was used for each lab visit and the intervention presentation order was randomized and scheduled in a balance design in order to control for this.

(4.7) Conclusion

In conclusion, 30 minutes of seated WBV reduced the stiffness of the trunk and decreased reflex gain without compensation from increased reflex gain or co-contraction recruitment. A reduction of total trunk stiffness and reflexes following seated WBV can impair spinal stability and increase the risk of a low back injury. By understanding the effects of WBV on the subsystems of spinal stability control, interventions can be developed to help prevent possible LBDs.

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Chapter 5

Changes in the Natural Frequency of the Trunk During Seated Whole-Body Vibration

Gregory P Slota, MS

**Virginia Polytechnic Institute & State University
School of Biomedical Engineering & Sciences
The Kevin P. Granata Musculoskeletal Biomechanics Laboratory**

(5.2) Abstract

Background: In the interest of low back pain prevention, research has been testing the effects of whole-body vibration exposure. Studies investigating changes in the frequency response functions of apparent mass and transmissibility of the trunk during vibration strive to further understand the mechanisms responsible for the increased risk of low back pain. The purpose of this study was to measure the changes in the natural frequency characteristics of the trunk (which can be related to trunk stiffness and damping) over time with exposure to seated whole-body vibration.

Methods: 41 healthy subjects were tested during 30 minutes of seated whole-body vibration (bandwidth = 2 – 20 Hz, root-mean-squared amplitude = 1.15 m/s^2). The force applied to the seat and the accelerations of the seat and trunk were recorded at 5 minute intervals. These data were used to determine the natural frequency characteristics of the trunk using the frequency response functions of apparent mass and transmissibility.

Findings: For frequency response functions of both apparent mass and transmissibility, the natural frequency decreased ($P < 0.01$), and the peak magnitude of the frequency response functions increased ($P < 0.01$) with exposure to whole-body vibration.

Interpretation: Changes in the natural frequency characteristics of the trunk are indicative of changes in overall axial trunk stiffness and damping. The decrease in natural frequency suggested a decrease in the axial trunk stiffness. The increase in the peak magnitude of the frequency response function suggested a decrease in overall trunk damping. These findings reveal changes in the biomechanical properties of the trunk with exposure to seated whole body vibration as a possible mechanism for an increase risk of low back injury.

(5.3) Introduction

Whole-body vibration (WBV) is a risk factor for LBP (Bovenzi et al., 2006). Approximately 6 million workers are exposed to seated WBV, including, but not limited to, professional drivers (e.g. fork-lift, truck, earth movers, bus, tractor) and helicopter pilots (De Oliveira and Nadal, 2005, Griffin, 2006). Although a link between WBV and LBP is generally accepted, there is no consensus as to the mechanism of injury (Mansfield and Maeda, 2005, Damkot et al., 1984, Frymoyer et al., 1983). In addition, the methods of identifying individuals who are most susceptible to the effects of WBV remain elusive (Mansfield, 2005). By investigating the frequencies of WBV delivered to the spine and the resultant effects, further understanding can be achieved as to the possible causes of discomfort and injury

There are two common approaches for studying the human biomechanical response to seated WBV: apparent mass and transmissibility (Mansfield and Maeda, 2005). The apparent mass approach first involves converting into the frequency domain the force applied to the individual relative to the acceleration at the driving-point where the force is applied. Based on Newton's second law, the quotient of these two quantities results in the "apparent" mass of the individual (Mansfield, 2005). Calculating this quotient for all frequencies will result in a frequency response function that illustrates the apparent mass of the individual as a function of driving-point frequency. The transmissibility approach first involves converting into the frequency domain the acceleration measured at two locations (Hinz et al., 2001). Calculating the quotient of

these two accelerations for all frequencies will result in a frequency response function that illustrates how well the acceleration is transmitted between the two measurement sites as a function of driving-point frequency. The magnitude of the apparent mass and transmissibility frequency response functions is non-linear with frequency, and can be used to identify the dynamics of the system (such as the natural frequency defined as the frequency at the peak magnitude of the frequency response function) (Mansfield and Griffin, 2000).

Several studies have used these approaches to quantify the biomechanical response of the human body to seated WBV. The human body's natural frequency when exposed to seated vertical WBV has been demonstrated to be in the region of 5 Hz and with a second resonance mode in the region of 10 Hz (Fairley and Griffin, 1989, Mansfield and Griffin, 2000). The natural frequency appears to decrease when the magnitude of vibration is increased. Fairley and Griffin (1989) reported a decrease in natural frequency from about 6 Hz to 4 Hz when the magnitude of the vibration was increased from 0.25 to 2.0 m/s² RMS. This has been interpreted as a 'softening' (i.e. decrease in lumped vertical stiffness) of the body with increase in vibration magnitude (Mansfield and Griffin, 2000). The peak magnitude of the frequency response function has also been shown to increase as the magnitude of vibration increases (Mansfield and Griffin, 2000, Mansfield and Lundstrom, 1999), but the authors of these studies made no conclusions based upon these findings. Finally, the orientation of WBV also influences the biomechanical response of the human body. Mansfield and Maeda (2007) reported that the natural frequency and the peak magnitude of the apparent mass frequency

response function are dependent upon the direction that WBV is applied (Mansfield and Lundstrom, 1999).

The goal of these previous studies was to quantify the biomechanical response of the human body to seated WBV. As such, the duration of WBV exposure was only long enough to obtain sufficient data for the analysis (typically < 1 minute). In the workplace, individuals are exposed to WBV over extended periods of time (minutes to hours). It is unclear how the biomechanical response of the human body to WBV changes over extended exposure to WBV. Knowing this would advance our understanding of how WBV affects the body, and potentially contribute to the improvement of safety guidelines and work schedules. Therefore, the goal of this study was to investigate changes in the biomechanical response of the human body to WBV. Both the apparent mass and transmissibility approach were used to quantify changes in the natural frequency characteristics of the body. Subjects were exposed to 30 minutes of seated WBV to simulate the environment during driving. It was hypothesized that extended exposure to WBV would decrease the trunk natural frequency and increase the peak magnitude of the apparent mass and transmissibility frequency response functions. This would imply a decrease in both total trunk stiffness and damping that may contribute to the reported increase in risk for low-back injury associated with WBV.

(5.4) Methods

Testing was performed on 41 healthy adults (23 males and 18 females) with a mean (standard deviation) age of 24 (SD 5.2) years, height of 171.2 (SD 10.5) cm, mass of 71.5 (SD 12.6) kg, and no self-reported history of LBP. Twenty of the 41 subjects also completed a control experiment during which WBV was replaced with quiet sitting. This study was approved by the Virginia Polytechnic Institute & State University Institutional Review Board, and all subjects provided informed consent prior to participation.

Subjects were exposed to WBV for 30 minutes while sitting in an upright posture with their hands resting in their lap. Measurements of frequency response dynamics to determine apparent mass and transmissibility were collected for 1 minute at 5 minute intervals throughout exposure. The control testing on 20 subjects was performed at least one week before or after the experimental session involving WBV. The order of presentation of these experimental sessions was counter-balanced. The purpose of the control testing was to determine the effect of 30 minutes of sitting itself (without WBV) on apparent mass and transmissibility. It consisted of 1 minute of WBV before and after 30 minutes of quiet sitting. This amount of WBV was required to collect the data necessary for our analysis.

(5.4.1) Experimental Protocol

The testing apparatus (Figure 1) consisted of a seat affixed to the top of a vibration platform so that each subject could be exposed to WBV while sitting (Slota et al., 2008). Attached to the seat were an adjustable foot rest and two seat belts used to limit the motion of the lower limbs and the pelvis, respectively, with respect to the seat during vibration. The vibration platform consisted of an electro-mechanical shaker (MB Dynamics PM-100, Cleveland, OH, USA), a counter-balancing spring in the middle to offset the subject weight, and a hinge in the front to allow vertical movement of the chair. Using this configuration, the maximum vertical excursion of the seat was approximately +/- 1.27cm during WBV. The shaker was driven by a Quanser Q8-I/O card (Quanser Consulting Inc., Markham, Ontario, Canada) connected to a SS530 Amplifier (MB Dynamics, Cleveland, OH, USA). Simulink (The MathWorks, Natick, MA, USA) code was used to generate a white noise signal with a frequency range of 2-20 Hz, which is the transmissibility frequency bandwidth of the torso (Griffin et al., 1982, Huang and Griffin, 2008), using low-pass and high-pass filters. The root-mean squared (RMS) amplitude of acceleration was 1.15 m/s^2 , which is the ISO 2631-1 (1997) exposure amplitude limit for 30 minutes of WBV. A tri-axial piezoelectric accelerometer (CXL04, Crossbow Inertial Systems, San Jose, CA, USA) was attached to the chair in-line with the base of the spine. Feedback control in the software used the accelerometer signal as input to maintain the desired level of seat vibration in terms of RMS amplitude of acceleration.

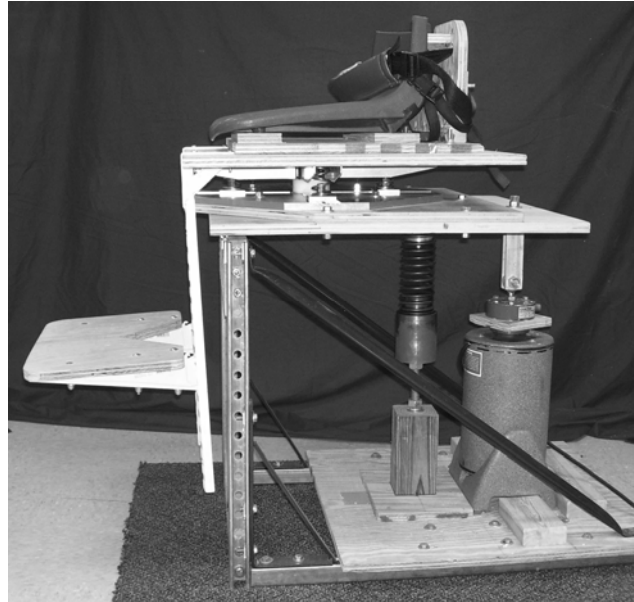


Figure 1: Photograph of testing apparatus. On top of the vibration platform was the seat. Under the vibration platform, from right to left was the electro-mechanical shaker with integrated load cell, spring to offset subject body weight, and hinge. The seat was equipped with seatbelts and foot rest to control pelvic and lower limb movement.

Prior to testing, each subject was briefed on the testing protocol and assisted into the seat. Adjustments were made to account for the subject's height (foot rest) and weight (counter-balancing spring), and the subject was secured with seatbelts. During the 30 minute WBV exposure, each subject was asked to sit upright with their hands resting on their lap. The data necessary for the analyses (see below) was sampled for one minute every five minutes. During this time, each subject was required to hold an instrumented bite-bar in their mouth and hold their arms folded across their chest. After the data was sampled, the bite-bar was removed and the subject could relax their arms with their hands resting on their lap until the next recording.

(5.4.2) Data Collection / Analysis

During each one minute of data collection, force applied to the vibration platform by the shaker was sampled at 1000 Hz from an in-line load cell (Interface Force, Scottsdale, AZ, USA) positioned between the shaker and platform. Seat acceleration was sampled at 1000 Hz using the tri-axial accelerometer attached to the seat in-line with the base of the spine (Crossbow Inertial Systems, San Jose, CA, USA). Body acceleration at the head was sampled at 1000 Hz using an accelerometer on a bite-bar held between the teeth.

The calculation of the apparent mass frequency response function involved determining the transfer function of input force to output acceleration (Mansfield, 2005). Using the Fast Fourier Transformation (FFT), the force applied to the vibration platform and acceleration measured at the seat were converted to the frequency domain. The magnitude of the frequency response function of apparent mass at each frequency was then determined by dividing the amplitude of the force and acceleration at each frequency. The apparent mass frequency response function was then normalized by dividing all magnitudes by the mass of the individual.

The calculation of the transmissibility frequency response function involved determining the transfer function of applied acceleration to the acceleration measured elsewhere on the body (Mansfield, 2005). Using the FFT, the acceleration measured at the seat and the acceleration measured at the head were converted to the frequency

domain. The magnitude of the transmissibility frequency response function at each frequency was then determined by dividing the amplitude of the head acceleration by the seat acceleration at each frequency.

The magnitudes of the frequency response function indicate frequencies where vibrations are attenuated (magnitudes less than one) as well as amplified (magnitudes greater than one). Peak magnitudes, typically greater than one, signify characteristic resonant frequencies of the system. From each of the frequency response functions, the natural frequency (f_n) was identified as the first resonance frequency (Figure 2), and the peak magnitude (P_v) was calculated as the magnitude of the frequency response function at that local maximum.

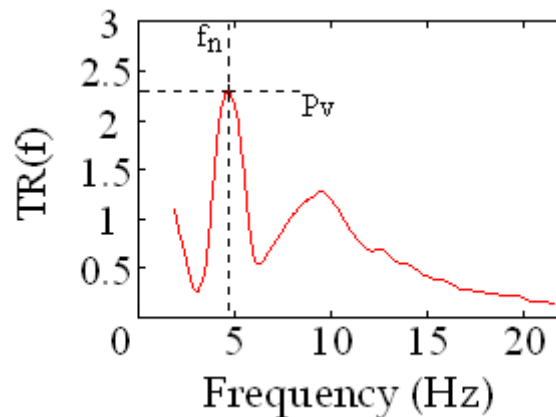


Figure 2: Example transmissibility frequency response function. Note the function’s peak magnitude at about 5 Hz signifying the natural frequency of transmissibility as well as that the magnitudes of the peaks are greater than one signifying vibration amplification.

A one-way repeated measures ANOVA was used to determine the effect of time on apparent mass natural frequency (AM_{f_n}), apparent mass peak magnitude (AM_{P_v}),

transmissibility natural frequency (TR_{fn}), and transmissibility peak magnitude (TR_{pv}). A two-way repeated measures ANOVA was used to determine if the effects of WBV differed from the effects of quiet sitting. This separate analysis was necessary because not all of the subjects participated in the control testing, and because control testing only involved collecting data at 0 min and 30 mins. Post hoc analysis was performed using the Tukey's HSD. All statistical analysis were performed using Stastica (StatSoft, Inc., Tulsa, OK, USA) with level for significance for all tests set to an α -level of 0.05.

(5.5) Results

Exposure to seated WBV decreased both measures of natural frequency (Figure 3). More specifically, on average AM_{fn} decreased 6.65% (SD 13.2%, $P<0.01$) and TR_{fn} decreased 4.74% (SD 7.56%, $P<0.01$) after the first 5 minutes of WBV. Visualizing the changes over time revealed a general trend whereby the natural frequency decreased over the first 5 minutes of exposure to WBV followed by little change over the remaining 25 minutes. Control testing revealed no changes in natural frequency during 30 minutes of quiet sitting (AM_{fn} : $P=0.512$, TR_{fn} : $P=0.761$).

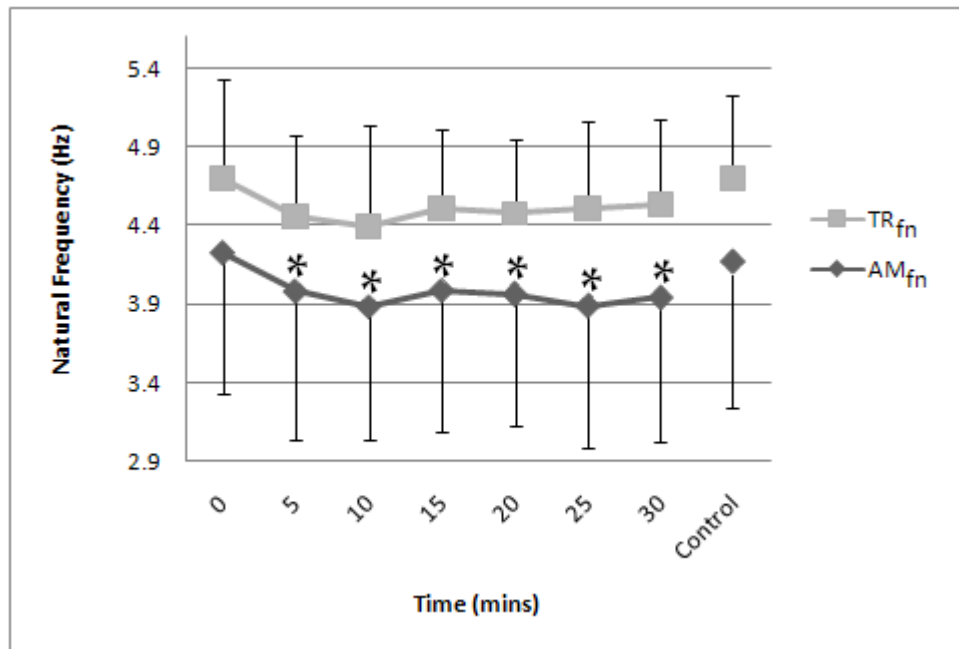


Figure 3: Changes in natural frequency during 30 minutes of seated WBV as measured by apparent mass and transmissibility. Results of control testing of 30 mins quiet sitting are also included for reference. * indicate $P<0.05$ with respect to 0 min.

Exposure to seated WBV increased the peak magnitudes of the frequency response functions (Figure 4). More specifically, on average AM_{PV} increased 4.59 % (SD 17.4%, $P<0.01$) and TR_{PV} increased 31.8% (SD 45.6%, $P<0.01$) after the first 5 minutes of WBV. Visualizing the changes over time revealed a general trend whereby AM_{PV} increased over the first five minutes of exposure to WBV followed by little change over the remaining 25 minutes. For TR_{PV} , in addition to a general increases after the first 10 minutes, there were differences between measurements pairs of 5-20,5-25,5-30, and 10-20 minutes suggesting a more prolonged rate of increase as compared to AM_{PV} . Control testing revealed no changes in peak magnitude of either the frequency response function during quiet sitting (AM_{PV} : $P=0.999$, TR_{PV} : $P=0.962$).

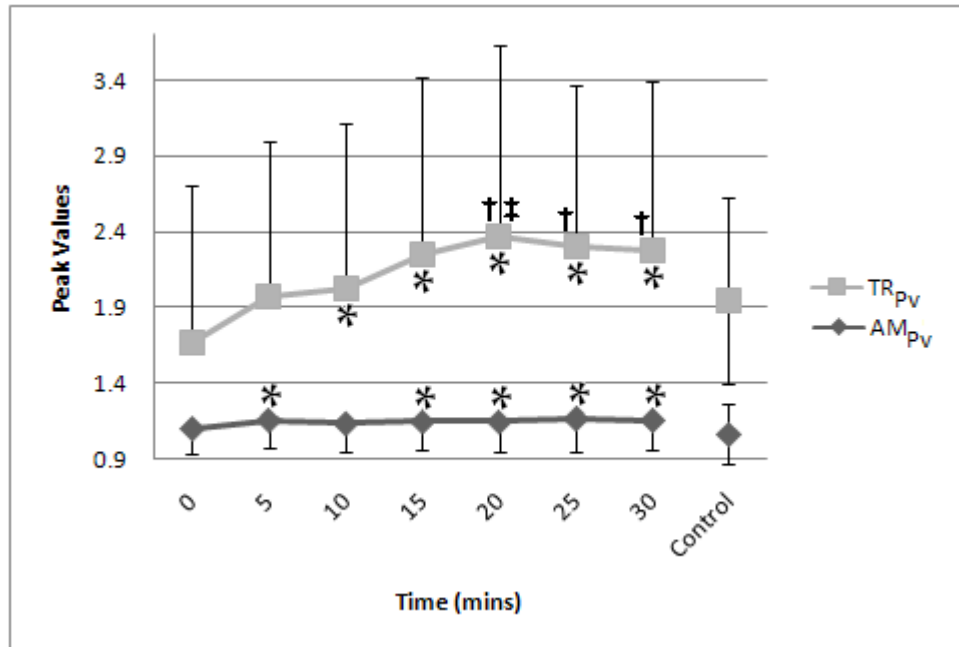


Figure 4: Changes in the peak magnitudes of the frequency response functions during 30 minutes of seated WBV. Results of control testing of 30 mins quiet sitting are also included for reference.

(* , †, ‡ indicate $P<0.05$ with respect to measures at 0, 5, 10 mins respectively)

(5.6) Discussion

The goal of this study was to measure changes in the frequency response characteristics of the human body during 30 minutes of seated WBV. The results demonstrated that maximal effects of exposure to seated WBV decreased the natural frequency 6.61-7.96% and increased the peak magnitude of the frequency response functions 5.90-42.0% while quiet sitting resulted in 3.84-5.03% and 2.08-6.01% respectively. Moreover, the time course of these changes began as a decrease in natural frequency over the first five minutes, with less change over the remaining 25 minutes. The peak magnitude of the frequency response functions increased for 10-20 minutes before leveling off.

The natural frequency of a simple mass-spring system is related to the system stiffness, and can be calculated as the square-root of the ratio of stiffness to mass (Equation 1a). Thus, an increase (or decrease) in natural frequency indicates an overall increase (or decrease) in stiffness or a decrease (or increase) in mass. Since mass is constant, the change in stiffness is equal to the change in natural frequency squared (Equations 2). Thus the result of a 4.74% average decrease in TR_{fn} implies a decrease of 9.26% in stiffness and a decrease of 6.68% in AM_{fn} to a 12.9% decrease in stiffness. The cause for the difference in stiffness assessment may be a function of pelvis and abdominal soft tissues increasing the apparent mass measure while having limited impact on transmissibility.

$$1) \quad f_n = \sqrt{\frac{k}{m}}$$

$$2) \quad k = m(f_n)^2$$

The parameter of trunk damping has been provided less attention than trunk stiffness in the literature even though it can be of great importance. Damping resists the velocity of movement, dissipates energy, and controls the magnitude of the frequency response functions. As vibrations are transmitted through the body, the tissues exhibit a damping effect which in effect, decreases the magnitude of vibration (Jovanovic, 2003). In this study, the peak magnitudes of the frequency response functions were all greater than unity, indicating that the vibrations at the natural frequency were being amplified. Additionally, the peak magnitudes exhibited an increase of 5.1% as measured by AM_{P_v} and 36.4% as measured by TR_{P_v} after 30 minutes of seated WBV. This indicates that the system damping was decreased, dissipating less vibration energy and increasing motion, and may result in greater strains on the spinal column (Wilder et al., 1988). This can be due to various factors that can contribute to the results including changes in trunk posture, passive tissue properties as well as the neuromuscular response. More studies are required in understanding the changes in the damping properties of the trunk as the importance of this property calls for more attention. Testing of the damping properties of the various trunk systems (passive elements, muscles) along with modeling of the internal loading changes due to reduction of damping can further elucidate the effects over time of WBV exposure.

Trunk muscle fatigue and prolonged trunk flexion are two risk factors for LBP that have also been shown to influence rotational trunk stiffness, which may be related to the observed changes in axial trunk stiffness. Granata et al. (2004) used sudden loading perturbations applied to the trunk along with a biomechanical model to quantify changes in rotational trunk stiffness after a lumbar extensor fatiguing protocol. The model simulated fatigue-induced reduction in active muscle stiffness and force generating capacity (reduced reflex gain) resulting in diminished spinal stability. Empirical data supported the model predictions with increased active stiffness due to increased agonist-antagonist co-contraction. As a result total trunk stiffness did not change with fatigue. In a different study, Granata et al. (2005) used pseudo-random force perturbations of the trunk along with a system identification analytical approach to quantify changes in rotational trunk stiffness after prolonged trunk flexion. Trunk flexion for 15 minutes reduced the passive stiffness of the trunk. Additionally, prolonged flexion resulted in increased reflex response (hyper-excitability). As a result, total rotational trunk stiffness was maintained. With both of these risk factors for LBP, there appeared to be a compensatory mechanism between the different neuromuscular systems that contribute to total trunk stiffness. In the current study, axial trunk stiffness was found to be decreased, suggesting that no compensation mechanism was evident. One potential reason as to why there was no compensation may be due to a disruption of the sensory pathways of the trunk. WBV has been shown to alter the proprioceptive acuity of the trunk (Li et al., 2008).

Measuring changes in the frequency response dynamics during 30 minutes of WBV reveals trends in these variables over time that had not been previously examined. The characteristics provide valuable information on the anatomical structures and/or mechanisms involved. Changes in natural frequency (or stiffness) happen within the first five minutes of exposure while the peak magnitudes (or damping) change within the first five to fifteen minutes. This can be the result of different tissues that are changing at different rates or during different time periods. For instance, intervertebral discs have been shown deform with WBV exposure (Klingenstierna and Pope, 1987) where two thirds of the total height loss occurs in a initial time period of axial loading of the spine (Keller and Nathan, 1999). Additionally, other tissues including ligaments and tendon-muscle systems can possibly experience creep deformations at different rates. These deformations are coupled with changes in neuromuscular control. Measuring changes in the natural frequency characteristics during 30 minutes of seated WBV can also help us to begin to understand possible exposure limits. During the second half of the vibration period, there were nominal changes to the natural frequency characteristics suggesting that no additional changes will occur with WBV exposure beyond 30 minutes. However, it is not evident from this current study if any additional changes to the physiology of the trunk will occur over larger time periods. Future research is needed to explore the effects of WBV for time periods greater than 30 minutes, including the equivalent of an entire work shift. Differences were seen between the measures of peak magnitude for apparent mass and transmissibility. Apparent mass measures were lower overall than that of transmissibility while also resulting in less of an increase over time of WBV exposure. The main difference in factors that affect apparent mass more than transmissibility are the

pelvic and abdominal soft tissues. The difference in results would suggest that these tissues may act as a buffer to the measurement of vibration. These elements may dissipate the energy of vibration applied to the whole body. Transmissibility along the more rigid structure of the spine would not be as susceptible to these effects.

Several limitations of this study warrant discussion. The apparent mass frequency response function measures the dynamic properties and relative movement of bodies of mass. This measure can be influenced by not only the properties and movement of the trunk mass, but also the properties and movement of the head and limbs masses (Mansfield and Griffin, 2002, Mansfield et al., 2006). By restricting the relative movement of the arms with respect to the trunk and the legs with respect to the seat (Fairley and Griffin, 1989), the measures of apparent mass can be concluded to be caused along a transmission path common to the spine and abdomen (Mansfield and Griffin, 2000). Similarly, the transmissibility frequency response function measured by the bite-bar is influenced by motions along the lumbar spine (area of interest) as well as the neck, head and buttocks. Additionally, rotational movement of the head and trunk in the sagittal plane influences the measures made in the vertical direction. Though these effects could not be controlled directly, they could be accounted for by comparing the trends in transmissibility with that of apparent mass. Matching trends in the frequency response functions would be conclusive of having come from similar main effects on the trunk (Mansfield and Griffin, 2000). Finally, although there were statistically significance for the decreased natural frequency and increased peak magnitudes which relate to trunk stiffness and damping, it is not clear if the effect size is clinically relevant.

However, these results are for decreased total effective trunk stiffness and damping which suggests there was no compensation (example co-contraction recruitment) and implies that the control of spinal stability was affected.

(5.7) Conclusion

In conclusion, 30 minutes of seated WBV decreased the natural frequency of the trunk and increased the peak magnitudes of the frequency response functions. These results suggest decreased trunk stiffness and damping and changes in pelvic soft tissue properties. The trends suggest that WBV effects are greatest during the first 5-15 minutes of exposure. The overall effect of decreased stiffness and damping would result in increased tissue strain with the potential of leading to LBP. Further studies are still required to assess the physiologic harm due to extended WBV exposure.

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Chapter 6 :

Closing Statements

Closing Statements

The goal of the three studies was to investigate the effects of seated WBV on spinal stability control in order to understand the risk for developing LBP. These studies were designed to investigate a series of questions:

Does WBV exposure change spinal stability control?

How is spinal stability control affected after WBV exposure?

What changes in the human body during WBV exposure?

With the results of the first study, it was shown that spinal stability control decreased with vibration exposure. With the second study, it was shown that after WBV, trunk stiffness decreased and the magnitude of muscle reflexes was reduced. In the second study, there was no additional recruitment of muscle co-contraction to compensate for the impaired spinal stability control. In the third study, it was shown that axial trunk stiffness and damping decreased over time of WBV exposure with a pattern of a rapid initial decrease, followed by diminishing effects.

The possible options for physiological explanations for loss in trunk stiffness include: muscle fatigue, passive tissue creep or damage, and sensory changes. Passive tissues are the basis for trunk stiffness and the damage that accumulates could impair spinal stability control. Passive tissue damage could contribute to decreases in spinal stability control (Study 1), decrease in trunk stiffness (Study 2), shifts in natural frequency characteristics (Study 3). In addition to passive tissue changes, and perhaps

due to it, are changes to sensory information. If the sensory information relaying the position of the trunk, velocities of moments, as well as the tension of the muscle are impaired or misinterpreted, then the feedback control of spinal stability will be impaired. This would explain the changes in muscle reflex gain (Study 2), impairment of active muscle recruitment (Study 2), and leading to impairment of spinal stability control (Study 1). Active muscle involvement may also be a part of the shifts in natural frequency characteristics (Study 3). Muscle fatigue was not assessed in the studies, but could possibly be a factor that decreased stability control (Study 1), caused the decreased in effective trunk stiffness (Study 2), and contributed to the changes in the natural frequency characteristics (Study3).

These 3 studies suggested spinal stability control as a mechanism that links WBV exposure and the risk for LBP. The results from these studies can be helpful in the understanding of how WBV could lead to LBDs and aid in the development of guidelines for WBV exposure duration. These studies have shown impairment in spinal stability control (via mechanical and sensory information) with WBV exposure and give a biomechanical possibility for the increased risk for LBP.

Future work in the field of WBV exposure following these studies should progress where questions are unanswered or limitations were encountered. Improvements on testing protocols need to be able to measure each component of trunk stiffness (passive stiffness, active stiffness, effective stiffness due to reflexes) directly and not just infer from other measures (i.e. EMG). Studies also need to address WBV

exposure consisting different levels of the vibration characteristics including RMS amplitude, frequency content, and duration. In order to understand the effects of WBV on industry workers, different vibration characteristics need to be investigated to match that of the machinery being used and to the work shifts. Different vibration characteristics may result in various effects on the biomechanics of the trunk, involving different degrees of the effect as well as the timing (rate of change, time period of change, recovery period). Understanding how different elements are affected by WBV, which frequencies are involved, and the post vibration after effects will aid in the design of proper guidelines and protective measures to prevent lasting complications such as the development of LBP.

The studies presented suggested decreases in spinal stability control subsystems without evidence of compensation mechanisms. The summary effect suggests that WBV exposure impaired spinal stability control which can possibly increase the risk for low back injury. Recommendations would include the basic understanding that WBV exposure should be controlled through limited the duration of expose, attenuation of vibration magnitudes especially near 5 and 10 Hz, as well as accounting for recovery time periods. Implications of impaired spinal stability control following WBV exposure suggests that precautions should be imposed limited physically demanding tasks following exposure.

Chapter 7 :

Appendix

(7.1) Equilibrium & Stability

The definition of equilibrium is the state of a body or physical system that is at rest or in constant and unchanging motion. A system is in stable equilibrium if small disturbances to the system cause only temporary changes before it returns to its original state.

(7.1.1) Ball & Bowl

The example that will be used to describe stability terms is a ball in a bowl. The equilibrium state is for the ball to be at rest at the bottom of the bowl (Figure 1a). Perturbations up the side of the bowl (Figure 1b) will displace the ball away from its point of equilibrium. The result will be the movement of the ball back towards equilibrium, returning to rest at the bottom again (Figure 1c).

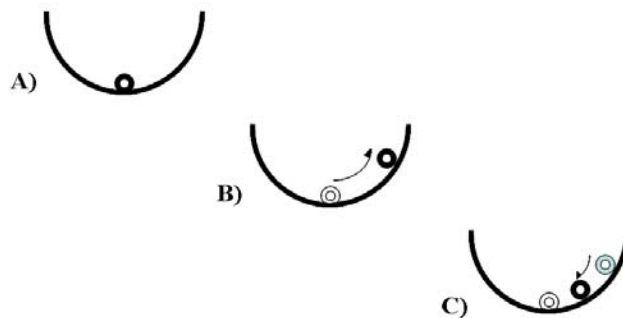


Figure 1: Ball & Bowl: Stable system
A) Equilibrium position, B) Perturbation displacement,
C) Resultant movement, toward equilibrium

An example of an unstable system is an overturned bowl. The ball can rest at equilibrium on top of the bowl (Figure 2a). Any perturbations will displace the ball away from the equilibrium point (Figure 2b). The subsequent movement will consist of the ball rolling further away from the equilibrium (Figure 2c).

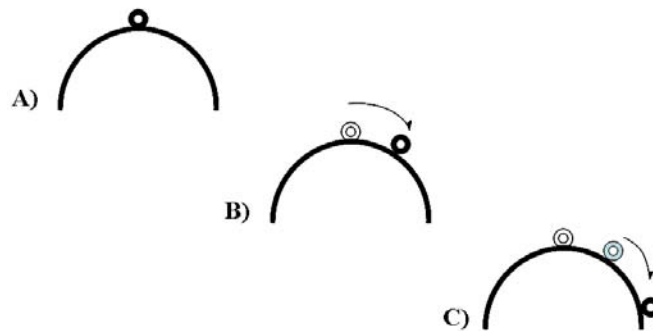


Figure 2: Ball & Bowl: Unstable system
A) Equilibrium position, B) Perturbation displacement,
C) Resultant movement, away from equilibrium

Stability also has its limits; the robustness of stability (Reeves et al., 2007). Any perturbation that pushes the ball within the limits of the bowl will result in the ball returning to the equilibrium point (Figure 3a). However, if the perturbation displaces the ball to a point outside of the bowl's limits, the result will be the ball rolling away from the bowl (Figure 3b).

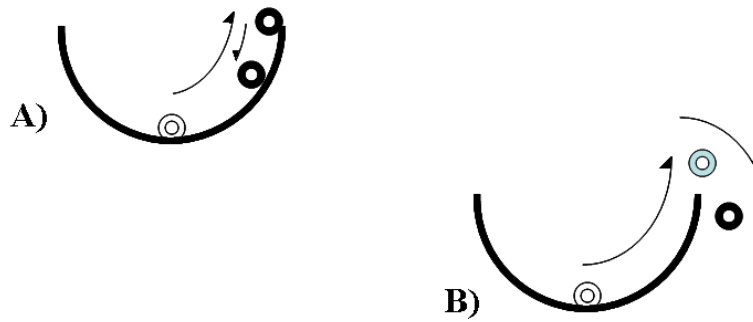


Figure 3: Ball & Bowl: System stability robustness
A) Displacements within the limits of the bowl, stable
B) Displacements outside the limits of the bowl, unstable

(7.1.2) Spine Model

The spinal column can be modeled as a single pendulum (Figure 4), similar to that of the ball and bowl, with stable and unstable configurations. On its own, the inverted pendulum is unstable and any perturbations will result in the pendulum falling over. There is nothing to resist the potential energy of the mass on top from falling to the ground (Figure 5a).

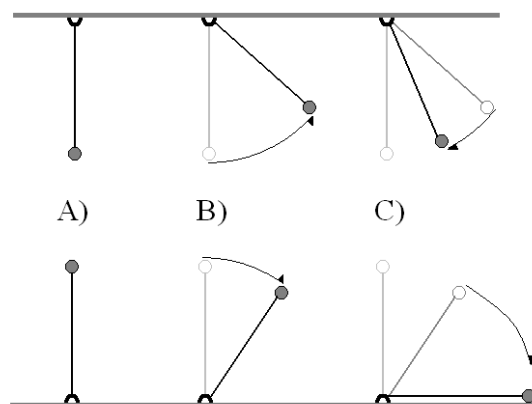


Figure 4: Pendulum: Stable system (above) and unstable system (below)
 A) Equilibrium position, B) Perturbation displacement,
 C) Resultant movement, toward/away equilibrium

The system of the spinal column consists of three components: the passive musculoskeletal subsystem, the active musculoskeletal subsystem, and the neural and feedback subsystem (Panjabi, 1992). Each of these subsystems contributes to the mechanical stability of the spine and controls the motion of each segment. Excessive motion at any point along the spine can cause tissue strain or tears leading to LBP and LBDs.

The passive musculoskeletal subsystem consists of the structural elements of the spine. The vertebrae are the building blocks of the spinal column supported in between by the intervertebral discs. The facet joints, spinal ligaments, joint capsules, and the passive properties of the muscles aid in the alignment and moments of the spine. All together, these components contribute to the passive stiffness (K_p) of the spine (Figure 5b).

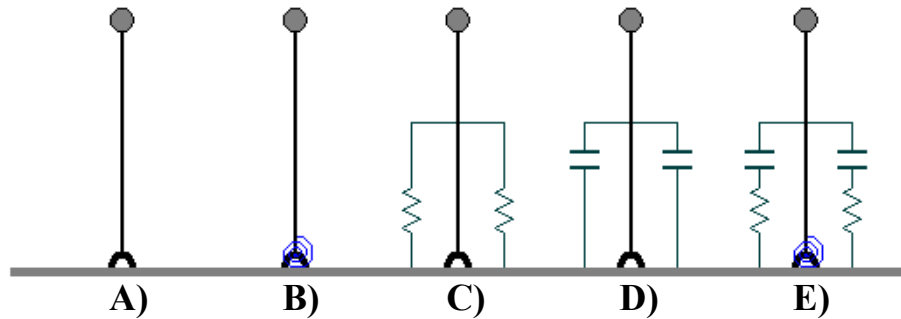


Figure 5: Model of spinal stability control
 A) Spine, B) Passive stiffness (torsion spring),
 C) Active stiffness (external linear springs),
 D) Neuromuscular reflex (contractile elements), E) Complete System

The active musculoskeletal subsystem components are the muscles and tendons that are associated with the trunk. This included muscles that are attached to the spine directly (ex. erector spinae) along with muscle that connect from adjacent structures (ex. rectus abdominus connecting the pelvis to the ribcage). Muscle contractions provide additional stiffness to the spinal column. Contraction from the agonist muscles (providing force in the desired direction) along with contraction from the antagonist muscles (working against the agonist) is call co-contraction. The opposing force generation works to stiffen the spinal column (Lee et al., 2006). This is considered the active stiffness (K_a) of the spine as seen in the model above (Figure 5c).

The neural and feedback subsystem is comprised of three components. Sensory organs perceive the position and velocity of the motion segments as well as the tension of connective tissues and muscles. The control system processes the information. And the final output is muscular control (contraction or relaxation). Some actions will elicit a muscular reflex while others will inhibit a muscular contraction. Muscle reflexes are

modeled as having a proportional response gain (G) with respect to their input as well as a delay (τ) in response. Different reflexes are attributed to having different delay times. Muscle reflexes are modeled above as external contractile elements (Figure 5d).

Passive stiffness, active stiffness, and muscle reflexes each support the spinal column. Alone, each subsystem is insufficient at supporting the spinal column under normal loading conditions. The passive stiffness of the spine has been measured at a load-carrying capacity of 2-9 kg (Crisco, 1989) as compared to the loading of body mass at 140-210 kg (Nachemson and Evans, 1968). The combination of the three subsystems (Figure 5e) provides the spinal stability control needed to support the spinal column. Reflexes have been measured to account for 42% of the total stabilizing trunk stiffness (Moorhouse and Granata, 2007) noting that passive stiffness, without active stiffness, was not sufficient at stabilizing the spine. Each subsystem is susceptible to deterioration from use including: tissue creep, muscle fatigue, and sensory overload. Compensation from one subsystem to another works to maintain the total system stability (Granata and Orishimo, 2001). If muscle reflexes were to have increase delay and or decrease gain, there must be an increase in co-contraction to maintain stability (Franklin and Granata, 2007). However, no pattern of muscle contraction will maintain spinal stability when exposed to an external load of 200 N or greater; reflexes are required (Le et al., 2007).

(7.1.3) Stability Control

The human spine is subjected to external disturbance forces resulting in displacements. The system of spinal stability control functions to maintain equilibrium within physiological limits. The system consists of the intrinsic properties: mass, damping and stiffness. With the addition of feedback control, muscle reflexes contribute to the system stability (Figure 6).

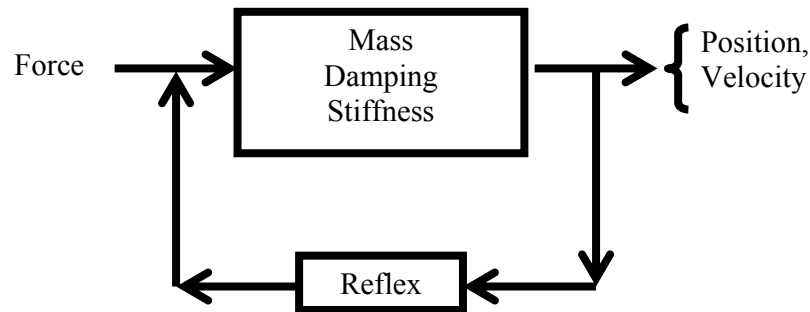


Figure 6: Spinal stability control diagram

The control diagram of spinal stability control can be represented in equation form (Figure 7). When the system is reduced into a single equation (Equation 1), there can be seen the control of stability. Holding inertia (I) constant and with damping (B) being a dissipative element, the final collection of terms holds the key to stability. In order for the system to be stable (all poles in the left hand plane), the final set of terms must be greater or equal to zero (Equation 2). An inverted pendulum alone is unstable with the potential energy (V) of the mass resulting in the pendulum falling down. The restorative elements of stiffness and muscle reflex counteract the potential for the pendulum falling over (Equation 3). The contributions from each component (passive

stiffness, active stiffness, and muscle reflex) work together to maintain stability. In the event that one component's normal contribution is diminished, the other components can compensate for the loss.

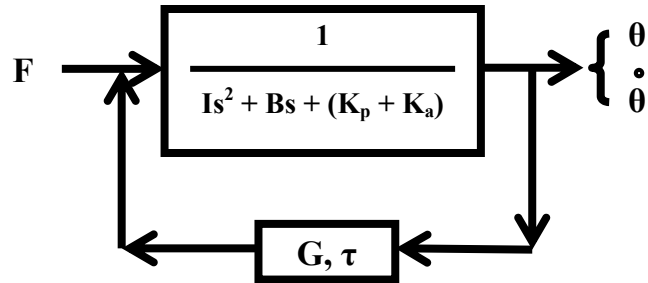


Figure 7: Spinal stability control: Equation

$$1 / (s^2 + Bs + [(K_p + K_a) + (\text{Slota et al.}) - V]) \quad (1)$$

$$[(K_p + K_a) + (\text{Slota et al.}) - V] \geq 0 \quad (2)$$

$$[(K_p + K_a) + (\text{Slota et al.})] \geq V \quad (3)$$

(7.1.4) Factors Affecting Stability

Spinal stability is composed of passive stiffness, active stiffness, and neuromuscular reflexes. These subsystems are composed of ligaments, tendons, muscles, intervertebral discs, and other surrounding soft tissues. Different factors affect these tissues and can disrupt the subsystems of spinal stability. Muscle fatigue, caused by sustained, maximal, or repeated exertions, weakens the muscles' force production capabilities. Trunk posture, when working at different degrees of flexion, lateral bending, or twisting, increases the loading of the passive tissues. Prolonged work in flexed postures, repetitive movements and tissue loading can then lead to tissue stretch disrupting passive stiffness and sensory feedback. Task demands (lifting, pushing, and pulling) can affect the spinal stability requirements. In studies to understand how changes in spinal stability control can lead to LBP, researchers have also compared measurements between LBP patients and healthy people.

(7.1.4.1) Muscle Fatigue

Trunk stability control is dependent on the muscles for support both passively and actively. Muscle fatigue affects the power output of motor groups. The neuromuscular system is able to adjust to changes in the system. Muscles provide active trunk stiffness through co-contractions and assist stability with feedback reflexes. Following muscle fatiguing exertions, it has been shown that subjects have an increased state of co-

contraction (Granata et al., 2001). This has been reported as a means for compensation for fatigue to maintain stability when exposed to sudden loading (Granata et al., 2004b). While in a fatigued state, muscle reflexes have been measured to have increased reflex amplitudes (Herrmann et al., 2006). Further, differences have been seen between genders, with increased electromechanical delay in females and decreased EMG and force amplitude in males (Moore et al., 2002).

(7.1.4.2) Trunk Posture

Work performed in a flexed posture is a known risk factor for LBP (Marras et al., 1993). Flexed postures have been shown to increase the reliance on passive tissue support of the trunk extension moment as flexion increases. This results in greater trunk stiffness with flexion angle as well as a decrease in the reflex gain (Granata and Rogers, 2007). With the increase in passive tissue stiffness, the need for muscle reflex is reduced in order to maintain stability. However, a study has also shown that there is an increase in reposition error with flexed postures (Wilson and Granata, 2003) suggesting that the sensory information of the trunk is disturbed and may suggest an inhibition of muscle reflex response.

(7.1.4.3) Tissue Stretch

Working in a sustained flexed posture or the repetitive movement to the limits of the trunk range of motion results in immediate and residual laxity of the joint (Little and Khalsa, 2005). Joint laxity of the trunk is the result of a decrease in the passive stiffness without compensation or with reduction of active stiffness (Courville et al., 2005). Studies of trunk flexion and tissue creep have reported two effects on the neuromuscular reflex system. In studies of direct spinal stretch loading (Solomonow et al., 2000) as well as with whole-body trunk flexion (Rogers and Granata, 2006), tissue creep and joint laxity suppress reflexive muscular activity and decrease the reflex gain. Tissue creep reduces the sensory information along the spine. This change in reflex behavior of the muscles has been shown to recover with a delayed state of hyperexcitability (Hoops et al., 2007). This increased reflex gain after tissue stretching (Granata et al., 2005b) has been reported as the recovery of fast adapting receptors in tendons and ligaments with rest (Courville et al., 2005). The ligamento-muscular reflex provides the lumbar spine with a mechanism in which increased musculature activity compensates for the laxity developing in the lumbar viscoelastic tissue (Le et al., 2007).

(7.1.4.4) Task Demands

The control system for trunk stability utilizes and adapts the three subsystems to the demands of the tasks to be performed in order to maintain spinal stability. Voluntary

increase in trunk muscle co-contraction will increase trunk stiffness (Lee et al., 2006). With the expectation of a sudden load, there is a reported trend of preparatory EMG increase (Granata et al., 2001). This would suggest that the trend was for an increased trunk stiffness to handle the loading and stabilize the spine. Co-contraction has also been recorded to increase as a task demands more stability control. As a barbell was maintained at a constant moment arm (no change in equilibrium demand) and raised to higher levels (increase in potential energy and therefore and increase in stability demand) there were increasing levels of co-contraction of the trunk muscles (Granata and Orishimo, 2001). In the case of a study of different handles used in pushing tasks, there was an increase in co-contraction from the stable handle (1-DoF) to the unstable handle (2-DoF) (Lee and Granata, 2006). Comparing pushing and pulling tasks, the flexion exertion associated with the pushing task resulted in increased co-contractions from that of extension exertions, with additional increases with exertion level (Lee et al., 2007). Flexion exertions had average co-contraction approximately twice the value of extension tasks with co-contraction; accounted for up to 47% of total spinal load (Granata et al., 2005a).

Trunk muscles and their adaptation to different tasks has been an area of active research. Pre-loading the trunk muscles with an external flexion force was associated with a significant increase in preparatory EMG activity (Granata et al., 2001). With the increase in muscle extension exertion, the results were of increased system damping and effective stiffness (Moorhouse and Granata, 2005). Muscle reflex amplitude has been reported to decrease with preload activity and to increase with perturbation force

(Granata et al., 2004a). Muscle reflexes are important to the stability of the spine and account for 42% of the total stabilizing trunk stiffness (Moorhouse and Granata, 2007).

(7.1.4.5) People with LBP

Studies of spinal stability are concerned with preventing LBP. Of interest are studies that investigate the differences between people who have a history of low back pain and those that are healthy. Studies by Radebold (Radebold et al., 2000, Radebold et al., 2001) have reported that LBP patients demonstrate poorer postural control of the lumbar spine. They exhibit patterns of higher levels of co-contraction and had longer muscle reaction times. The consensus is for people with LBP to adopt a stiff spine strategy, possibly in adaptation to compromised quality of trunk control (Mok et al., 2007). However, there are no studies that confirm if the effects are preexisting for LBP patients or if it is due to the pain.

(7.2) Wobble Chair

The wobble chair is a tool designed in the Musculoskeletal Biomechanics Lab at Virginia Tech. The purpose of the wobble chair is to create an unstable seat for testing the stability control of the human trunk/spine. Under the supervision of Dr. Kevin Granata, the project was initiated with Steve Desso creating the first version. This original design however was flawed and prevented the practical use. When the design was passed on to Greg Slota and Mike Diersing, a series of wobble chairs were designed and made. Currently there are three wobble chairs, one each at Ohio University, Ohio State, and Virginia Tech. While there have been slight improvements with the creation of each wobble chair, the basic design is still the same.

(7.2.1) Background

Published in 2000, Cholewicki's lab group at Yale authored a paper titled 'Postural control of trunk during unstable sitting' (Cholewicki et al., 2000). In this study, the use of an unstable seat was implemented to challenge the postural control of the trunk. This seat was designed with a flat board for the subjects to sit on while underneath various diameter hemispheres could be attached (Figure 8). When the seat is placed on a force plate, motions of the subject balancing can be measured in terms of the center of pressure location.

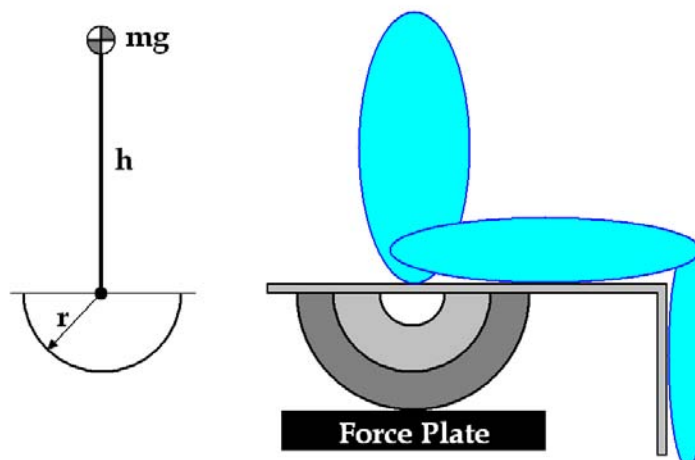


Figure 8: Unstable seat as designed by Cholewicki (2000) using hemisphere seat bottom and measuring a force plate CoP
 (Model: where task difficulty can be changed by adjusting radius- r , or by different subject's height- h or mass- m)

By changing the diameter size of the hemisphere, the task difficulty could be changed. The smaller the hemisphere diameter is, the harder the task becomes. Without the hemisphere attached, the flat board is equivalent to a hemisphere of infinite diameter.

This first study presented the unstable seated balance method and showed how postural control decreased with increased task difficulty. From there, two more studies were published. Using the unstable seat, postural control of low back pain patients was tested (Radebold et al., 2001) as well as the effects of trunk stiffness (Reeves et al., 2006). Another lab has since used a similar unstable seat to check the reproducibility of postural control measures during unstable sitting in low back pain patients (Van Daele et al., 2007).

(7.2.2) New Approach

In the study by Cholewicki where the unstable seat was presented, there was reported a high correlation of subject anthropometrics and ability to balance. With the hemisphere approach, four diameters were used: Infinite (flat), 50 cm, 44 cm, 22cm. It was reported that balance ability was correlated to the subject's mass and height. In the cases of subjects that were unable to balance on the smaller hemispheres, there were the tallest or heaviest people. To account for the differences in subjects, a new approach to the unstable seat was designed. The wobble chair was created with the idea of having unlimited adjustability (within bounds). The basis behind the new approach was to use a small pivot point that would create an unstable seat that is impossible to balance on. In comparison to the unstable seat of Cholewicki, this would be the equivalent to a hemisphere of about 2 cm. To adjust the stability of the seat, a series of springs are positioned under the seat. By varying the spring position, the unstable seat can be controlled in a manner similar to that of changing the diameter of the hemisphere.

(7.2.3) Design

The basis of the wobble chair design is still based off of the original concept by Cholewicki. The wobble chair is designed to balance on a single pivot point created by using a ball-in-socket ball bearing of about 2 cm in diameter (Figure 9a). The wobble chair is created in two parts, a seat and a base. The base has a central post with the ball-

bearing pivot attached on top. The seat platform has attached to the bottom a plastic piece with a small divot (Figure 9b) in the center which rests atop the pivot.

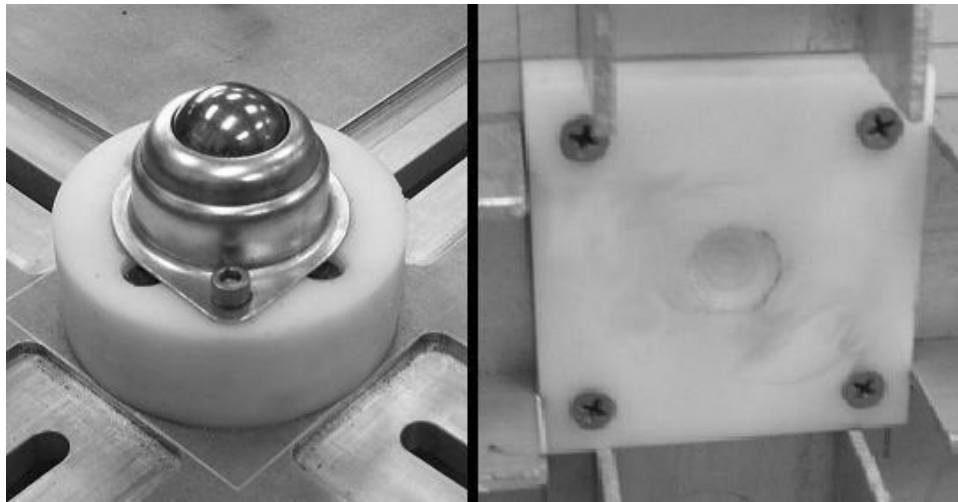


Figure 9: Pivot point for the wobble chair (left) and the seat contact point (right)

In this current state, balance of a seated subject is unlikely. To provide support and allow for the adjustability of the wobble chair, four spring assemblies are positioned between the seat platform and the base (Figure 10). Each spring assembly holds the bottom end of the spring and is attached to the wobble chair base in one of four grooves and bolted into place (Figure 11). The grooves run radially out from the central pivot point in front, behind, left, and right. The spring assembly can be positioned anywhere from 7 cm to 22 cm away from the central pivot. The closer the springs are to the center, the harder the task of balancing on the seat becomes.

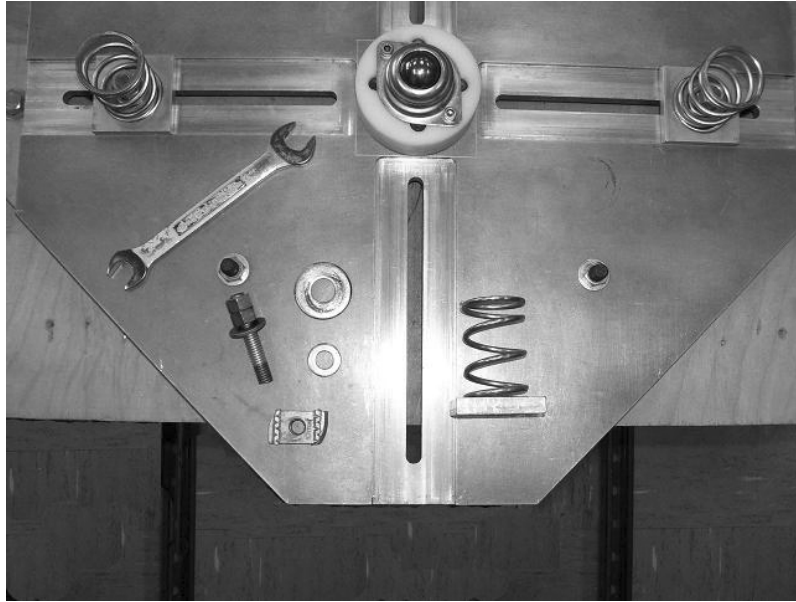


Figure 10: Wobble chair base with central pivot point, radial grooves, and spring assemblies.

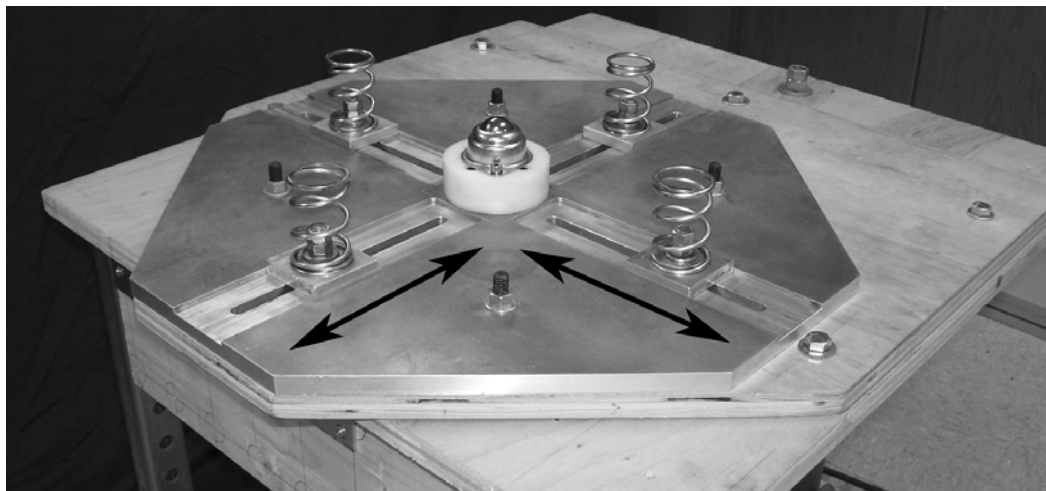


Figure 11: Spring Base with central pivot point. The spring position can be adjustable radially from the central ball-and-socket pivot point.

The seat platform is designed to sit atop the ball bearing and tilt in any direction around it. The bottom of the seat platform, in addition to the plastic pivot interface, has four metal channels (Figure 12). Each channel is aligned with a groove in the base where

the springs are attached. The channels act as the interface point for the tops of the springs. The springs are not attached to the seat platform and only work in compression. As the seat tilts, the space between the seat platform and base decreases and compresses the spring. The channels also functions to limit the rotation of the seat about the third axis (Z).

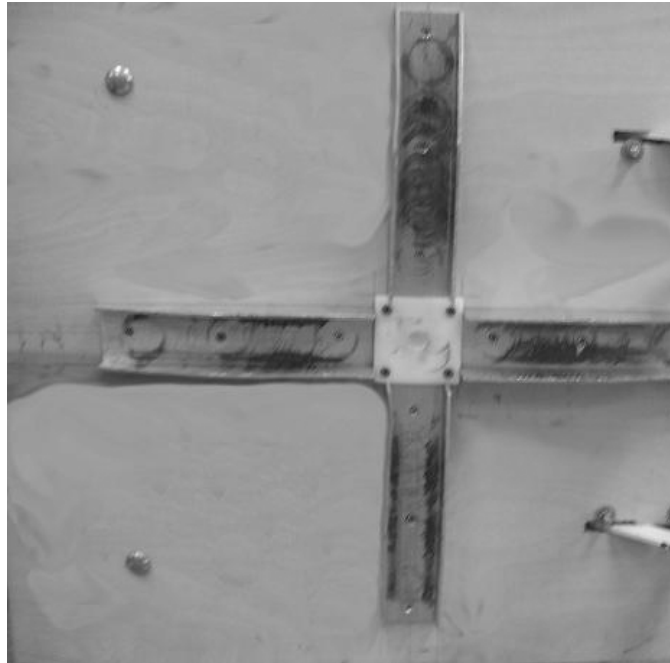


Figure 12: Metal channel on top section of the wobble chair. The springs of the base make contact within the metal channel.

To assist in the measurement of postural control, the movements of the pelvis and lower limbs are restricted. The seat is equipped with two seat belts to assist in holding the pelvis still and allowing movement from the spine (Figure 13). An adjustable foot rest is attached to the front of the seat platform to restrict lower limb movement with respect to the pelvis and seat.

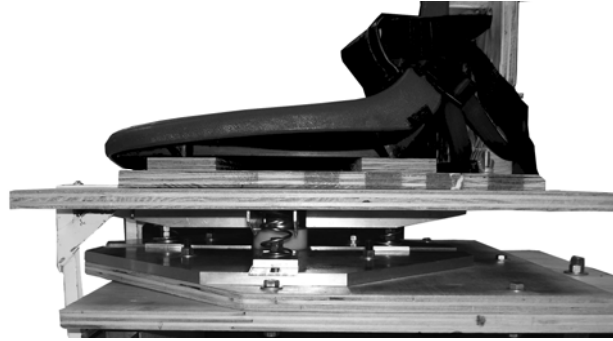


Figure 13: Complete wobble chair. The seat is equipped with two seatbelts and an adjustable footrest (off picture).

The seat that is mounted on the top platform of the wobble chair is also adjustable. The neutral position of the wobble chair is to have a subject sitting upright with the seat platform level. To accommodate for differences in subjects' anthropometrics (height, weight, mass distribution) the seat can be moved forward or backwards. With the use of a level, the seat position is adjusted to achieve equilibrium in the neutral position.

(7.2.4) Method of Use

Initially, measurements of the wobble chair were performed by using center of pressure on a force plate located below the wobble chair. The premise is that as the subject tilts the wobble chair in any direction, the force of the body will be distributed between the center pivot point and any springs that are compressed. In this manner, the movement of the wobble chair can be traced similar to that of standing postural sway.

Currently, the measurements of the wobble chair have changed from forces and moments on a force plate, to that of measuring the angle of tilt of the seat and trunk. X-sens motion sensors are currently used in the Virginia Tech lab. Each sensor provides rotational measurements in both planes of movement. Additionally, measures of linear accelerometers in all three axes are collected. This information can be used in detection of “bottoming out” when the physical limits of the wobble chair are reached.

(7.2.5) Calibration

It was noted in one of Cholewicki’s studies that people of greater body height and great mass had more difficulty in balancing on the smaller hemispheres. With the current design of the wobble chair, the springs can be positioned at a greater variable range than that of pre-sized hemispheres. With the adjustability, two different scales can be used to describe the spring positions. There is the distance from the center of the spring to the center of pivot point. This measurement is made in centimeters. The other method to position the springs is terms of the *MGH* of the subject (Equation 4, Figure 14). This method scales the spring positioning based on the anthropometrics of the subject. To do this, a calibration of the wobble chair is required.

$$MGH \sin(\theta_1) = KL^2 \sin(\theta_2) \quad (4)$$

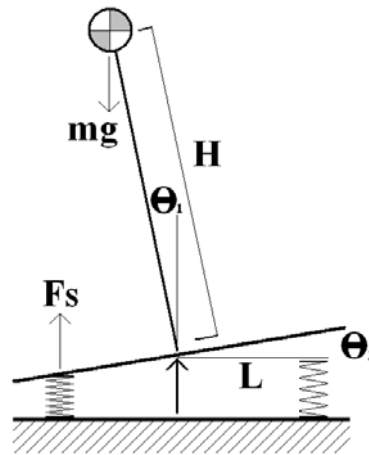


Figure 14: Model diagram of the wobble chair functionality. Springs used only in compression and are not attached to top surface.

Initially, the wobble chair needs to be adjusted for the individual subject. The height of the foot rest is positioned to allow for a 90° knee flexion. Next, the seat position is set for the subject to be able to balance in the neutral position for the wobble chair. Once the wobble chair is set, two sets of measurements are required (Equation 5). On a force plate, the wobble chair is set in a fixed 10° backwards tilt. With the addition of a back positioning bar on the chair, the subject is required to sit still with their arms crossed their chest and their back just touching the bar. This test is then repeated in a similar fashion with a 10° forwards tilt. Measuring the tilt of the wobble chair and the changes in the moment about the pivot point, the MGH for that subject can be calculated (Equation 6).

$$\begin{aligned} MGH \sin(\theta_a) &= My_a + e \\ MGH \sin(\theta_b) &= My_b + e \end{aligned} \quad (5)$$

$$MGH = \frac{(My_a + e) - (My_b + e)}{\sin(\theta_a) - \sin(\theta_b)} \quad (6)$$

Two strategies have been employed with this design and task difficulty control. The basic method is this set difficulty based on %-MGH. This will account for differences in subjects' height and body mass (i.e. center of mass location and value). Typical usage is to use a spring setting of 100%_{MGH} and other settings below that. Previous lab testing used 75%_{MGH} and 50%_{MGH}. It has also been noted in other research as well as from collaborators at Ohio University, that for testing the differences between low back pain patients and pain free people, that more challenging settings are required. Based off of this is the need for higher task difficulty.

The other method, which is used in the first study with WBV, is to account for and match a subject's ability to balance. While a subject's height and weight play a factor in the difficulty to balance on the wobble chair, the subject's 'skill' to balance can be considered. This method requires a series of trials and spring adjustments to locate a desired level of difficulty. Limits of stability can be test specific as well as the criteria for adjusting the springs. One example is finding the threshold for balancing without reaching the physical limits of the wobble chair.

(7.2.6) Data Processing

With either the measurement of force plate center of pressure, or with the tilt of the seat platform (Figure 15), the data processing is similar and only the units are

different. Measures of kinematic variance, similar to that of standing postural sway (Prieto et al., 1996), can be used to quantify the control of seated balance. This includes ellipse area, path length, and root mean squared (RMS) movement. The ellipse area represents the 95% range of motion area during the balance trial where a larger area is attributed with a decrease in balance control. The ellipse area is greatly affected by the general ‘waling’ trend (low frequency signal content). The path length is simply the linear length of movement regardless of direction traveled and is greatly effected by ‘trembling’ or noise (high frequency signal content). RMS movement is the average distance from the point of equilibrium. The RMS value can be broken into components of anterior/posterior, medial/lateral, and also the resultant radial directions (Figure 15, circles).

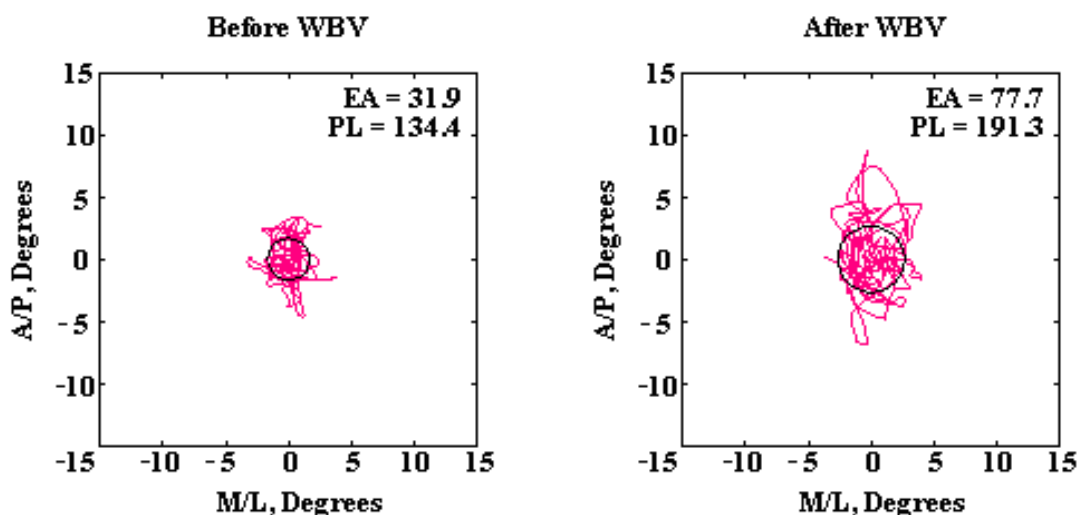


Figure 15: Example plots of data from the wobble chair. Shown are plots for before and after WBV exposure. Parameters of ellipse area (EA), path length (PL) are labeled, as well as circles representing RMS-RR.

Non-linear stability control measures include Lyapunov exponent, stability diffusion analysis (SDA), Hurst rescaled range analysis (HRRA). The Lyapunov exponent looks at the rhythmic activity of the system in state space as a limit cycle. The process quantifies the exponential separation of trajectories of nearest neighbors with time (Harbourne and Stergiou, 2003). The analysis uses the 4 dimensional state dynamics (two angles, two angular velocities) to calculate the nearest neighbors (Kang and Dingwell, 2006). SDA allows for correlation between past and future displacements and summarizes the mean square displacement as a function of the time interval between comparisons (Collins and De Luca, 1993). The analysis allows for the system to be broken down into two parts, short-term open-loop control and long-term closed-loop control. The long-term stability diffusion analysis parameter is not representative of a bounded system (Delignieres et al., 2003) and therefore cannot be used with the wobble chair. The HRRA describes the fractal properties of fluctuations in measurements during postural control (Duarte and Zatsiorsky, 2000). Similar to that of the kinematic measure of RMS movement, SDA and HRRA can be analyzed for each direction (-A/P, -ML, -RR).

(7.3) Chapter 3 Supplemental

Wobble Chair Spring Settings – Location, Location, Location...

The purpose of the wobble chair is to provide a “stability-controlled” seating condition. This means that the springs can be moved and positioned to desired setting, thusly controlling the “stability” of the seat. As mentioned earlier, the position of the springs can create a neutrally-stable seat when the subject can achieve equilibrium at any angle of seat tilt. In this case the restorative force of the springs is equal to the moment created by a tilted torso. The springs can also be moved further out from this position (with respect to the pivot point) and create a “stable” seating condition where equilibrium is held in the upright posture. Of typical interest is when the springs are moved closer to the pivot point and an unstable seated condition is created. In this case, only with the inclusion of muscle reflexes to trunk stiffness can balance be maintained.

For the purpose of testing, there are four methods that have been used for setting the springs. A simple approach is to use an absolute spring position for all subjects; that is to say that the springs are set at 11 cm away from the pivot point. This is similar to the Cholewicki hemisphere approach in which all subjects were exposed to the exact same seat condition.

The next evolution in control of the spring position is the basis of the current wobble chair design. With infinite (yet bounded) spring position control, the springs can

be fine-tuned to different scaling methods. The initial method account for the differences in subject anthropometrics. By measuring a subject's MGH, the springs can now be positioned in terms of percent MGH instead of just length away from the pivot. With $100\%_{\text{MGH}}$ as the neutrally stable condition, subjects can now be tested at similar seat stability levels accounting for the limitations in the Cholewicki design. Past testing protocols have used the spring settings of $100\%_{\text{MGH}}$, $75\%_{\text{MGH}}$ and $50\%_{\text{MGH}}$.

Even though the scale of $\%_{\text{MGH}}$ can account for anthropometrics, people still perform differently. The third method of setting the springs is to account for the subject's personal ability to balance. In method, the springs are adjusted to result in trials that have similar performance outcomes. As used in chapter 3, the springs were adjusted till the subject's balance performance was within bounds of challenging enough, but not too difficult. In this case, the subject's performance was monitored such that the tilt of the chair stayed primarily greater than 4° but less than 7° of seat tilt. The purpose of this method was to use an increased difficulty level where changes in the postural stability of the trunk would be more pronounced after the applied factor (in this case WBV).

The fourth method that has been investigated for spring positioning would be the location of the subject's threshold of stability. Though not complete in process, this method has been explored by progressively moving the springs away from the pivot point. In addition, analysis of the data collected can be used to locate the basin of stability for parameters of tilt angle and angular velocity. With this it can be shown that while it looks like one person can balance better (smaller ellipse area) than another, the

person with the larger ellipse area may also in fact have a larger basin of stability and can handle larger disturbances to their trunk posture.

(7.4) Chapter 4 Supplemental

Maximum Voluntary Co-Contraction (MCC)

In addition to the protocol used in the second study (chapter 4), there were also trials pre-exposure that had subjects providing maximum voluntary co-contraction during the pseudo-random trunk perturbations. For three of the trials, the subjects were instructed to maximally co-contract their trunk flexors while maintaining the 20%_{MVE} baseline extensor torque. For the other three trials, the subject will be instructed to not co-contrast. During the six trials, EMG activity was monitored of all four muscle groups (L-ES, R-ES, L-RA, R-RA), with importance on the activity of the rectus abdominus. Trials without the requirement of co-contraction were seen to be maintained on average below 5% MVC. Trials where co-contraction was required were seen to be greater than 10% MVC and were off the visible scale. Verbal encouragement was provided along with time updates so that subjects could maintain a maximal effort through the trials.

The initial purpose of having MCC trials were to use to measure the effects of WBV and then scale them based on the non-MCC (0%) and MCC (100%) values of muscle activity. However there was 1) no significant changes in rectus abdominus activity due to WBV exposure, and 2) the results of the MCC trials are counter intuitive. To expand on this complication there were three expected outcomes of the MCC trials versus the non-MCC. First, that there would be an increase in activity of the rectus abdominus muscles, as this is the muscle group that you can ‘consciously’ recruit when

thinking about co-contracting. Second, that there would be an increase in the activity of the erector spinae muscles to counter the bending moment generated by the rectus abdominus. This is the definition of co-contraction. The third expectation would be for an increase in trunk stiffness due to the increase in co-contraction. The ‘bracing’ effect increases the stiffness of the spine at the costs of increase muscle usage and compression loading of the spine.

For the MCC trials, subjects’ rectus abdominus activity was monitored for increased activity, and the post processing analysis revealed these results (on average from 4%_{MVC} non-MCC to 11%_{MVC} MCC). However, the activity of the erector spinae and trunk stiffness were not seen to increase. The lack of noticeable changes in erector spinae activity can possibly be due to effect size. The force increase in the rectus abdominus may have been minimal compared to the 20%_{MVE} force level that was required to be sustained as well as the perturbation forces. Noise levels of the EMG recordings may have also occluded any differences.

Trunk stiffness, as measured by the IRF method yielded no significant increased in trunk stiffness with co-contraction. This is counter intuitive. Additional analysis does show that the average force level during a trial of perturbations was increased as well as average trunk angle displacement was decreased. The combination of these results would suggest that the stiffness of the trunk increased with co-contraction, however, the IRF method uses de-trended data series and would not see these differences in trials.

Recovery After WBV

The effects of WBV vibration were tested and reported for effects from before to after exposure. Additionally, for the study of trunk perturbations, there was an additional series of three trials after 30 minutes of quiet sitting (serving as recovery) after the WBV exposure. The two main effects seen for WBV exposure, a decrease in total trunk stiffness and decrease in reflex gain, were not seen to have any significant change with recovery. This follows the trends for creep exposure of tissues to exhibit longer recovery periods compared to the time of effect causing the creep deformation.

Post-Hoc: Gender

Some results were seen between genders that are worth noting. The mean forces for trunk perturbations were significantly higher for males (185 N) than females (145 N) ($P=0.0360$). This has a two part explanation. First is that the males had higher MVE torques and second is that the males were heavier than the females. These two factors add together to have an increased mean force level even though all subjects were exposed to the same perturbation sequence. (Note: the sequence is composed of 20% MVE baseline torque and 33% BW perturbations.) Now it is of interest that the purpose of scaling the perturbations to the subjects' trunk mass was successful, and that there were no significant differences in the mean trunk displacements. Keeping this in mind, then it is significant that results also show that females had a higher reflex gain as compared to

males ($P=0.0339$). Based off of the current information presented here, this would mean that the reflex response is either higher in females in general, or that the difference in force is more accountable. Finally, most measures of rectus abdominus activity was found to be higher in females than males for base-line pre-trial activity ($P=0.0106$), during trials ($P=0.0046$), and in terms of MCC effort ($P=0.0406$).

(7.5) Chapter 5 Supplemental

Matlab Modeling of Stiffness & Damping

A model of a second order system (mass, spring, damper) was used to illustrate the results for Chapter 5. Holding other parameters constant, the stiffness was reduced. The resultant frequency response curves (Figure 16) can be seen to have decreasing natural frequencies. This model therefore shows how the measured decrease in natural frequency can be hypothesized to be in part due to a change in total trunk stiffness.

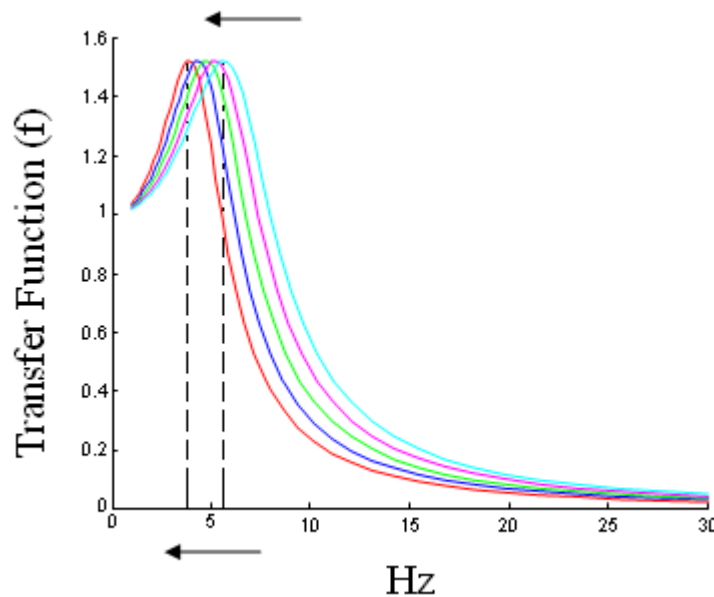


Figure 16: Example of decrease in natural frequency due to a decrease in the stiffness of a second order system.

Continuing on with the model, this time all factors were kept constant and only changing the damping. A reduction of the damping resulted in an increase in the peak

value of the frequency response function (Figure 17). This model demonstrates how a change in the peak value is an implication of a decrease in the system damping.

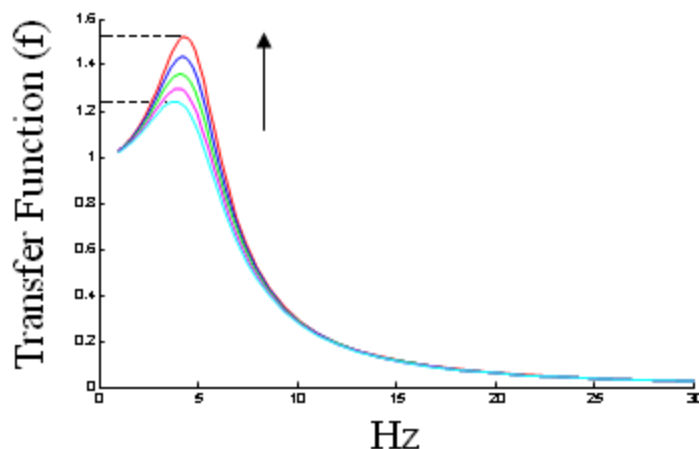


Figure 17: Example of increase in peak value at the natural frequency as seen with the increase in vibration exposure duration. Modeled here as a decrease in damping of a second order system.

Transmissibility Second Characteristic Frequency

The transmissibility frequency response function, in addition to the natural frequency near 5 Hz, also has a secondary characteristic frequency. Similar to that of the natural frequency (Figure 18), there was a significant overall drop of 6.40% (SD 16.0%) for TR_{f2} ($P < 0.01$). Visualizing the changes over time indicate an initial rapid decline with a gradual leveling off. The peak value measured at the secondary frequency was lower than that of the natural frequency and also exhibited the same increase with exposure to WBV (Figure 19). The overall increase was 22.8% (SD 69.6%) for TR_{p2} ($P < 0.01$). Similar to rate of change of the natural frequency, the increase in the frequency response functions' peak value had an initial increase which gradually levels (Figure 19). Control

group testing revealed no significant changes in peak values for either of the frequency response functions.

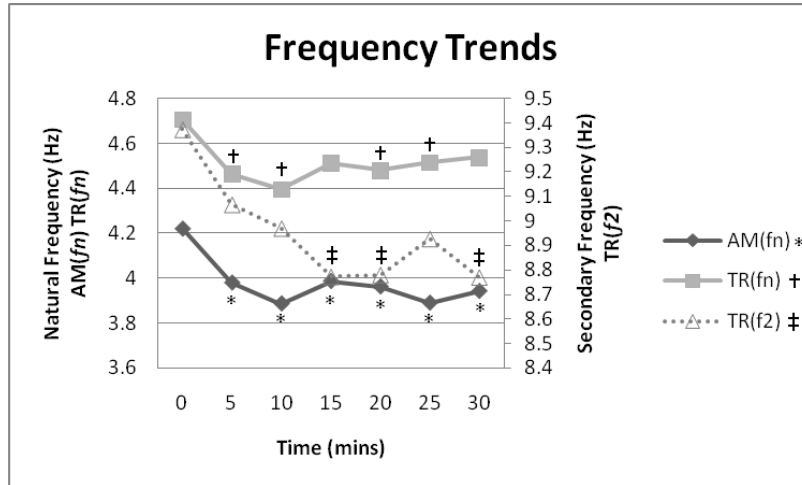


Figure 18: Frequency trend for the secondary characteristic frequency of transmissibility with respect to the natural frequencies of transmissibility and apparent mass.

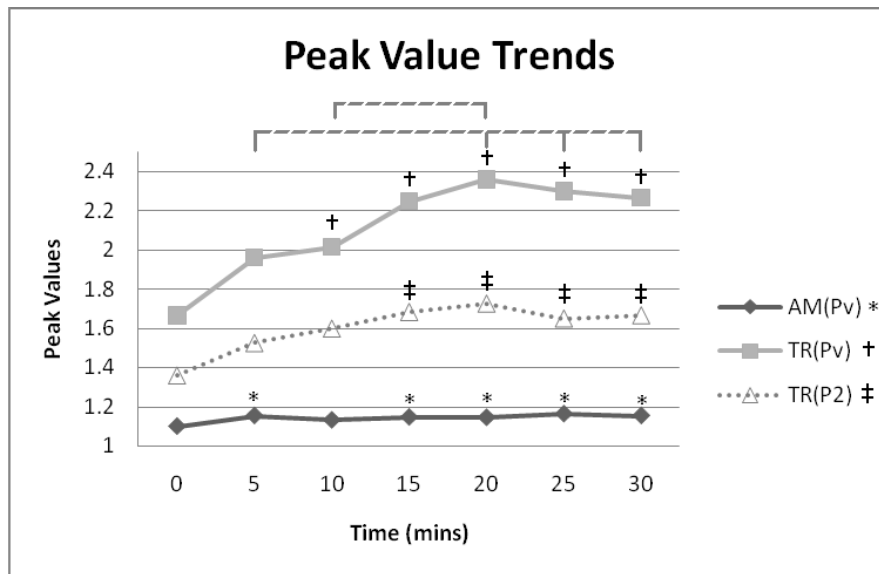


Figure 19: Peak value trend for the second characteristic frequency of transmissibility with respect to the natural frequency of transmissibility and apparent mass.

Post-Hoc: Gender

Some significance between genders and interactions with time were seen. There was a significant trend for males to have a lower secondary frequency for transmissibility ($P=0.0324$). This was also evident in the interaction of gender and time ($P=0.0347$) where the males had lower values than females ($P<0.05$) for all samples after the initial (Figure 20). Additionally, only the males had differences ($P<0.05$) from the initial value to recordings at later times of WBV exposure. In contrast, in the control group testing, the natural frequency for both frequency response functions showed males to have a higher natural frequency than females ($AM(fn) P=0.0069$, $TR(fn) P=0.0468$).

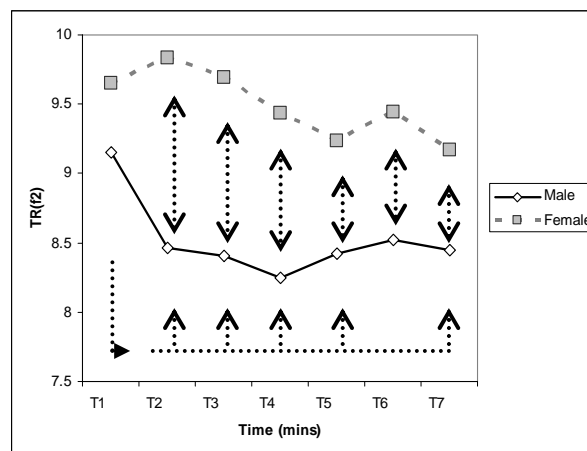


Figure 20: Gender and time interaction for $TR(f_2)$. Arrows mark post-hoc significance between genders and per-gender differences from the initial measurement.

Significance was found for the interaction of gender and time for the peak values for both apparent mass ($P=0.0495$) and transmissibility ($P=0.0457$). Differences were seen for males to have lower peak values than females for apparent mass at some time

samples (Figure 21a). For transmissibility, initially there was a lower peak value for females than males, where the difference between genders is reduced due to the rapid increase in the females' parameter (Figure 21b).

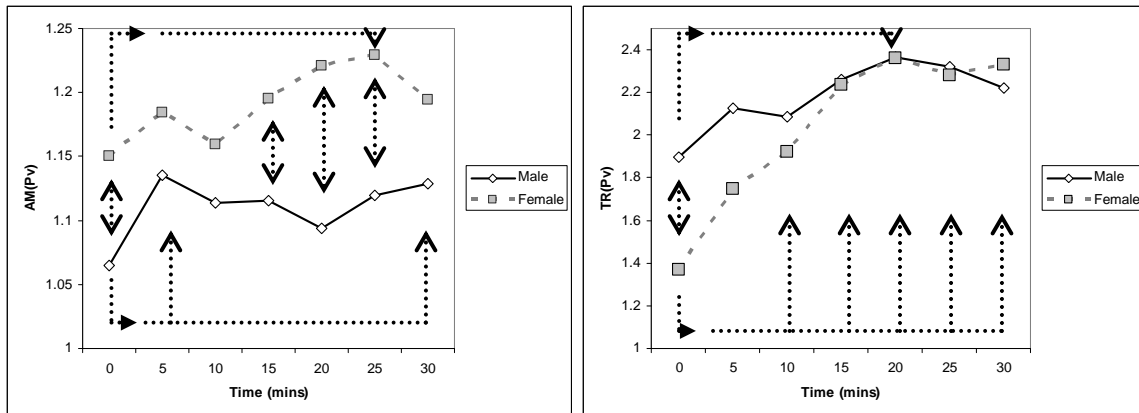


Figure 21: $AM(P_v)$ and $TR(P_v)$ gender by time interaction. Arrows mark post-hoc significance between genders and per-gender differences from the initial measurement.

Alternate Movement: Anterior/Posterior Frequency Function

Measures of frequency response functions can be prone to the effects of postural movements (Mansfield and Griffin, 2002, Mansfield et al., 2006). This applies to changes in posture during vibration as well as possible effects of anterior/posterior pivoting. In the case of postural changes, subjects were monitored to limit any changes throughout testing. As for any anterior/posterior complications, additional analysis was performed to address this issue.

In the case of transmissibility, there are questions as to where the 10 Hz secondary characteristic frequency originates from. One hypothesis is that the rocking motion in the anterior/posterior direction would cause it. Another possibility is that the effect is of the cervical spine and the head. This second hypothesis can also explain why the second characteristic frequency is primarily seen in transmissibility and not in apparent mass. The agreement between the two methods at the natural frequency near 5 Hz is and not at 10 Hz is the proportion of mass that is involved. With the head being a smaller mass with respect to the entire HAT (head, arms, trunk) mass, any effects on apparent mass would be negligible. With the measure of transmissibility with a bite bar, segment mass is less of an issue.

To address the concept of for/aft rocking, transmissibility in the for/aft direction was process and compared to the vertical direction (Figure 22). In the four examples, the darker line represents the Z-axis (vertical) transmissibility while the lighter line is the Y-axis (anterior/posterior). In each case the peaks of one axis does not line up with the peaks of the other. If the for/aft pivoting moment was responsible for peaks in the vertical direction, it would be expected that the transmissibility curves would share some characteristics. In fact it has been stated that the frequency and peak in one direction are not related to that of another (Mansfield and Maeda, 2007). Of interest to this investigation is the noticeable occurrence of a peak in one signal matching up with a local minimum in another (Figure 23). As shown, the natural frequency (F_n) in the Z-axis lines up with a local minimum in the Y-axis. Likewise, the peak value in the Y-axis (F_Y) is aligned with a local minimum of the Z-axis in between the natural frequency and the secondary frequency (F_2).

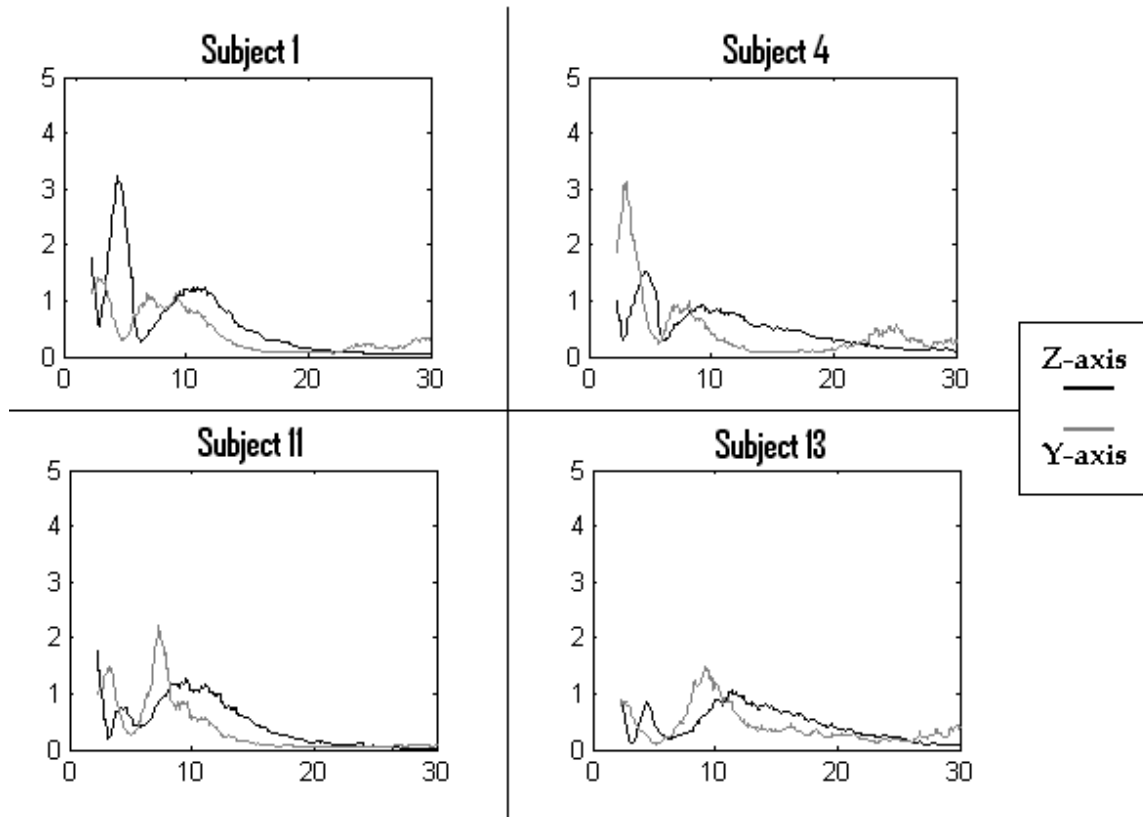


Figure 22: Examples of frequency response curves for the Z (Superior/inferior) and Y (anterior/posterior) directions. The darker line represents the Z-axis while the lighter line is the Y-axis transmissibility functions.

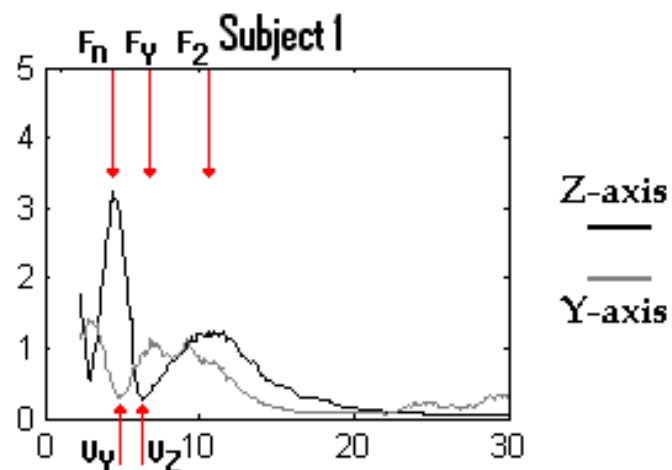


Figure 23: Example of frequency local maximums and minimums for the frequency response functions in the Z-axis (dark line) and Y-axis (light line). Note how the natural frequency (F_n) for the Z-axis lines up with a valley in the Y-axis and vice versa.

(7.6) Appendix References

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