

**AN EXPERIMENTAL AND SIMULATION BASED
APPROACH TOWARD UNDERSTANDING THE EFFECTS
OF OBESITY ON BALANCE RECOVERY FROM A
POSTURAL PERTURBATION**

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An experimental and simulation based approach toward understanding the effects of obesity on balance recovery from a postural perturbation

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ABSTRACT

Obesity is associated with an increased risk of falls and subsequent injury. Most falls result from some type of postural perturbation. As such, it is important to understand how obesity influences balance recovery from a postural perturbation. There is limited information on the effects of obesity on balance recovery, and the limited available information is ambiguous. Therefore, the purpose of the research within this dissertation was to investigate the effects of obesity on balance recovery after a postural perturbation in young adults to better understand how obesity contributes to fall risk.

Four separate studies make up this dissertation. The purpose of the first study was to investigate the effects of obesity on balance recovery ability using an ankle strategy in young adults. Normal-weight and obese participants recovered balance using an ankle strategy after three types of postural perturbations: an initial angular displacement, an initial angular velocity from the natural stance, and an initial angular velocity from a prescribed position. Obese participants were unable to recover balance using an ankle strategy as well as normal-weight participants when perturbations involved an initial angular velocity. However, no differences between obese and normal-weight participants were found when perturbations only involved an initial angular displacement. The effect of obesity on balance recovery in young adults was dependent on the perturbation characteristics, and may be explained by a possible beneficial effect of increased inertia on balance recovery after perturbations with little or no initial angular velocity.

The purpose of the second study was to examine the effects of obesity on balance recovery by stepping in young adults. The ankle strategy has the benefit of simplifying the mechanics of balance recovery, but limits generalizability to more realistic fall scenarios where stepping to extend the base of support and recover balance is desired. Similar to the first study, participants attempted to recover balance following two types of postural perturbations: an initial angular displacement from an upright stance (by releasing participants from a static forward lean), and an initial angular velocity while in an upright stance (using a translating platform). In contrast to the first study, the ability to recover balance with a single-step did not differ between young normal-weight and obese adults. These results suggest that the reported increase in

fall risk in obese adults is not a result of impaired balance recovery ability (at least among young adults that were tested here).

The third study examined the effects of obesity on body kinematics immediately following a trip-like perturbation in young adults. Obesity was found to increase body angular velocity the perturbation, and that increases in body angular velocity were associated with an increased probability of a failed recovery. These results suggest that when a young obese and young normal-weight individual trip while walking at similar speeds, the young obese individual may be at a greater risk of falling following a trip because the young obese individual will experience a greater body angular velocity. This detrimental effect of obesity on the difficulty of recovering from a trip-like perturbation in young adults is most likely due to how mass is distributed throughout the body and not the amount of mass itself.

The final study examined the relationship between relative strength and functional capability in young adults, and how obesity influences this relationship. To compare relative strength used during a functional task (i.e. balance recovery from a forward fall), the obese and normal-weight individual should complete the task with identical kinematics. Forward dynamic simulations were used to address this research question, instead of human subjects testing, to achieve identical kinematics. Differences in peak relative torques were found between the normal-weight and obese model, with the largest differences seen at the hip. These findings suggest that young obese individuals use greater relative strength at some joints than young normal-weight individuals to perform the time-critical task of balance recovery, and that these differences in relative strength demands may limit functional capability in young individuals who are obese.

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Chapter 1 – Overview

1.1 Research problem

Obesity is a major health concern in the United States (US) and around the world. For example, more than one-third of adults, or over 72 million people, in the US are considered obese (Ogden et al. 2007). One of the many concerns with the high prevalence of obesity is its association with an increased risk of falls and subsequent injury. Individuals who are obese fall almost twice as often (27% vs. 15%) compared to non-obese individuals (Fjeldstad et al. 2008), and falls have been identified as the most common (~36%) cause of injuries in individuals who are the obese (Matter et al. 2007).

Based upon this increased risk, it is important to understand the biomechanical mechanisms by which obesity contributes to fall risk in order to develop effective solutions. Several biomechanical studies have reported that increased body weight is associated with increased postural sway during quiet standing, and increased postural sway is associated with an increased risk of falling (Fernie et al. 1982; Maki et al. 1994). However, most falls do not occur during quiet standing but instead result from some type of postural perturbation (Horak et al. 1997). Therefore, it can be hypothesized that obesity may impair the ability to recover balance after a postural perturbation and avoid a fall. However, there is limited knowledge on balance recovery ability in the obese, and such information is important to develop fall prevention interventions to reduce the risk of falls.

The purpose of the research within this dissertation was to investigate the effects of obesity on balance recovery after postural perturbations in young adults to better understand how obesity contributes to fall risk. Both experimental testing on human subjects and computer modeling and simulation were used to address this topic. This research will provide valuable information towards understanding fall risk in individuals who are obese, and hopefully contribute to the development of interventions to improve balance recovery ability, and ultimately reduce the risk of falls.

1.2 Document organization

This document is organized into eight chapters. Chapter 2 presents a literature review of the problem of falls and injury in individuals who are obese, the effects of obesity on the musculoskeletal system that could contribute to this risk, and the effects of obesity on balance and balance recovery. Chapter 3 presents an overview of forward dynamic simulations and modeling. Forward dynamic simulations will be used to address the goals of this research: 1) to better understand the relationship between obesity and balance recovery, and 2) to improve balance recovery ability and possibly reduce the risk of falls in obese individuals through fall-prevention interventions. The first study of this research is presented in Chapter 4, and is one of the first studies to investigate the effects of obesity on balance recovery ability. This chapter, entitled "*The effects of obesity on balance recovery using an ankle strategy in young adults*", will give insight on the effects of obesity on balance recovery and the fundamental mechanics involved in order to improve our understanding as to how obesity contributes to falls. This study used a combination of human subject experiments and forward dynamic simulations of balance recovery. The second study is presented in Chapter 5: "*The effects of obesity on single-step balance recovery from a forward fall in young adults*". Stepping is a common reaction to external perturbations, and the only reaction to successfully recover balance from large postural perturbations (Maki and McIlroy 1997). Therefore, to understand the effects of obesity on balance recovery from a more challenging task, and to increase the generalizability of our results to more realistic fall scenarios, balance recovery using a single step was investigated through human subject experiments. This leads into the study presented in Chapter 6: "*The effects of obesity on body kinematics immediately following a trip-like perturbation in young adults*". Obesity is associated with alterations in body mass and body segment inertial parameters. These alterations may increase fall risk in obese individuals by increasing the difficulty of recovering balance. Chapter 7 examines the relationship between relative strength (i.e. strength normalized to body mass) and functional capability, both of which are reduced in the obese (Lakdawalla et al. 2004; Handigan et al. 2010). This chapter, entitled "*The effects of obesity on relative strength used during balance recovery from a forward fall in young adults*", utilized the knowledge gained in previous chapters on the effects of

obesity on balance recovery and forward dynamic simulations to examine how obesity influences the relative strength employed during a functional task (balance recovery from a forward fall) in young adults. Finally, Chapter 8 summarizes the major findings from these studies, and proposes future directions of this research.

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Chapter 2 – Obesity and balance literature

2.1 Obesity: a global health concern

Obesity is a major health concern in the United States (US) and around the world. Over 1 billion people worldwide are considered overweight (Body Mass Index – $30 > \text{BMI} > 25 \text{ kg/m}^2$), and of those, 300 million are considered obese ($\text{BMI} > 30 \text{ kg/m}^2$) (Puska et al. 2003). In the US, more than 68% of adults in the US are overweight and over 33% are obese (Flegal et al. 2010). Not only is the prevalence of obesity high, it is continually increasing. From 1980 to 2000, the prevalence of obesity among US adults more than doubled from 15% to 31% (Flegal et al. 2002; National Center for Health Statistics 2008). If these trends continue, it is estimated that more than 50% of adults will be obese by 2030 (Wang et al. 2008).

A grave concern with this high and increasing prevalence of obesity is its association with numerous health conditions. For example, obesity is associated with hypertension, type 2 diabetes mellitus, cardiovascular disease, osteoarthritis, and respiratory disease (Puska et al. 2003; Bray 2004). Obesity is also associated with lower life expectancies, and is responsible for approximately 95 million of years-of-life-lost among US adults (Finkelstein et al. 2009). Not only is obesity debilitating and hazardous, but it is also costly. Obese individuals have 40% higher on medical costs compared to normal-weight ($25 > \text{BMI} > 18.5 \text{ kg/m}^2$) individuals (Finkelstein et al. 2009b). In 2008, over \$147 billion of total medical expenditures in the US were attributed to obesity (Finkelstein et al. 2009b).

2.2 Functional capability in the obese

In addition to these medical conditions, obesity is associated with reduced functional capability. Functional capability is the capability to perform tasks that are necessary or desirable in life. These functional tasks are typically performed repeatedly each day. For example, adults are estimated to rise from a chair on average 90 times a day (McLeod et al. 1975). Other examples include walking, ascending/descending stairs, picking up an object from the floor, and sitting in a chair from a standing

position. Among older adults, obesity is associated with impaired functional capability (Lang et al. 2008). In particular, the odds of having impaired functional capability increase as BMI increases (Houston et al. 2005), and with obese older adults having a ~five times increased risk of impaired functional capability compared to their non-obese counterparts (Zoico et al. 2004). Although most of this literature focuses on obese older adults, evidence suggests that obesity's effect on functional capability is independent of age (Lakdawalla et al. 2004). For example, rising from a chair was more difficult for obese children compared to non-obese children (Riddiford 2000). Additionally, Evers Larsson and Mattsson (2001) found obese middle-aged women took longer and had more difficulty performing 13 of 16 tasks including balancing, squatting, stair ascent/descent, walking while carrying external weight, and rising from a seated position.

2.3 Obesity, falls, and injury

Balance is another commonly examined task when assessing functional capability. This is of particular concern among obese individuals because, in addition to medical conditions and reduced functional capability, obesity is also associated with an increased risk of falls and subsequent injury. Fjeldstad et al. (2008) found obese individuals to fall or stumble twice as often as normal-weight individuals. Additionally, in the occupational setting, individuals who are overweight or obese are more likely to experience a fall, as well as multiple falls (Wallace et al. 2002; Chau et al. 2004). Falls by themselves are not problematic, but an increased risk of falls increases the risk of injury. Falls have been identified as the most common cause of hospitalized injuries in individuals who are obese (~36% of all injuries) and the cause of a higher proportion of injury-related hospitalization compared to non-obese individuals (Matter et al. 2007). Similarly, Finkelstein et al. (2007) found overweight and obese individuals are more likely to sustain a fall injury than normal-weight individuals. In particular, fractures, sprains/strains, and dislocations have been reported to be the most common injuries and occur more frequently among obese individuals compared to non-obese individuals (Finkelstein et al. 2007; Matter et al. 2007).

2.4 Effects of obesity on the musculoskeletal system

Although the mechanism by which obesity contributes to falls has not been determined, it would seem that the negative impact of obesity on musculoskeletal function is at least partly responsible for the increased risk of falls in individuals who are obese. Obesity has been associated with impaired musculoskeletal function, particularly in the lower limbs (James 1995). It is thought that excess weight impairs gait, muscle function, muscle strength, sensory function, and posture. These impairments have been identified as strong risk factors for falls (Close et al. 2005).

Obesity causes alterations in gait that are associated with an increased risk of falls. Several studies have found preferred walking speed, step length, and step frequency to be lower in obese compared to the non-obese individuals (Spyropoulos et al. 1991; DeVita and Hortobagyi 2003; Fabris de Souza et al. 2005). Additionally, individuals who are obese have a longer stance phase (Spyropoulos et al. 1991; DeVita and Hortobagyi 2003) and greater period of double support (Spyropoulos et al. 1991). Reduced walking speed, cadence, and step length as well as increased stance duration are associated with an increased risk for falls (Close et al. 2005). Although Spyropoulos et al. (1991) have suggested that an obese individual walks slowly, takes smaller strides, and remains in double support longer in order to maintain balance, and deviations from this gait pattern (i.e. walking faster when in a hurry) would result in instability and loss of balance. DeVita and Hortobagyi (2003) found that obese adults tend to have a more erect posture compared to non-obese adults while walking at a set speed due to reduced knee and hip flexion. It is possible this posture provides stability by counteracting an anterior displacement of the center of mass (COM) from the longitudinal axis of the body associated with obesity, reducing the amount of corrective torque needed to maintain balance. This suggests that obesity reduces the range of anterior displacement of the COM for a stable posture, and similarly any motions or tasks that result in anterior displacement outside of this range would increase the risk of falls.

Obesity has also been shown to affect muscle composition and fatigability. In particular, obese individuals tend to have a lower percentage of type I (slow-twitch) muscle fibers and a higher percentage of type IIb (fast-twitch glycolytic) muscle fibers (Hickey et al. 1995; Tanner et al. 2002). These findings have several implications. Type I muscle fibers are oxidative, highly vascularized, fatigue resistant, and found in higher quantities in postural muscles (Peachey et al. 1983). Type IIb muscle fibers are glycolytic, provide rapid force production, and are highly fatigable (Peachey et al. 1983). These fibers are typically used for short, anaerobic, high force production activities, such as sprinting and jumping (Peachey et al. 1983). The lower percentage of type I fibers in the obese implies that obese individuals have a decreased ability to generate small magnitude forces over a long period of time for maintaining balance and fatigue more quickly. As such, obese individuals may be more susceptible to increases in postural sway during quiet standing and use large quick forces to maintain balance. The higher percentage of type IIb fibers in obese individuals has confounding implications. It is possible that the higher proportion of type IIb fibers makes the obese less susceptible to falls by being able to generate large forces quickly in response to a perturbation. However, a greater proportion of these fibers also indicates that the obese are more easily fatigued. Muscle fatigue negatively affects balance (Davidson et al. 2004; Davidson et al. 2009), thereby increasing the risk of falls. In support of this, Maffiuletti et al. (2007) found obese individuals to have lower fatigue resistance compared to their non-obese counterparts. In addition, Sartorio et al. (2003) found BMI to be correlated with fatigability, with individuals with a higher BMI associated with lower fatigue resistance.

Obesity also alters muscle strength, and specifically impairs muscle strength per body weight. Impaired muscle strength is associated with an increased risk of falls (Close et al. 2005). In particular, knee and ankle strength are important contributors to balance ability, and impaired strength in these joints can indicate an increased risk of falls (Whipple et al. 1987; Takazawa et al. 2003). Obese adults are able to generate higher absolute strength and power with the lower extremity compared to non-obese adults. However, strength and power are lower in obese adults when normalized to body weight. For example,

Lafortuna et al. (2005) investigated the effects of obesity on strength and power of several lower limb muscle groups (quadriceps, gluteus, gastrocnemius, and soleus). Absolute lower limb strength was higher in the obese subjects. However, while performing a jumping exercise, the obese subjects had similar absolute lower limb power output and decreased lower limb power output per unit body weight compared to the non-obese subjects. Other studies have also shown absolute knee strength to be higher and knee strength per body weight to be lower in obese compared to non-obese individuals (Hulens et al. 2001; Maffiuletti et al. 2007; Handrigan et al. 2010). These findings imply that although a greater absolute muscle force is used by individuals who are obese to perform daily activities, they cannot produce equivalent muscle force per weight compared to non-obese individuals. It is possible that this deficit in muscle strength per body weight limits balance recovery ability because balance recovery after a postural perturbation commonly requires one to generate and maintain high levels of joint torques in order to successfully recover balance (Robinovitch et al. 2002).

The high incidence of foot pain (Riddle et al. 2003) and impaired foot structure and function (Prichasuk 1994; Hills et al. 2002) are also possible contributors to impaired balance and increased risk of falls. In particular, plantar heel pain, characterized by a pain involving the insertion of the plantar fascia into the calcaneus, is five times more likely to develop in an obese individual than a non-obese individual (Riddle et al. 2003). Pain in the feet has been shown to increase postural oscillations, indicating poor balance (Corbeil et al. 2004). Studies have considered the effects of obesity on foot structure to be a pathological source for heel pain. For example, individuals who are obese have a significantly higher contact area compared to non-obese individuals (Riddiford-Harland et al. 2000; Fabris et al. 2006). A greater contact area suggests that obesity may inflict structural dysfunction such as a collapse of the longitudinal arch of the foot. It is also possible that increases in foot pressure alter balance. Foot mechanoreceptors and cutaneous sensation are contributors to balance control (Kavounoudias et al. 2001; Meyer et al. 2004). Therefore, greater pressure and contact area may lead to continuous over stimulation and eventual reduced sensitivity of the foot mechanoreceptors and, consequently, impaired balance. Nass et al. (1999)

found plantar peak pressures to be significantly higher at the heel in overweight compared to normal-weight individuals. Hennig et al. (1993) have suggested that changes in foot structure to increase foot dimensions allowed a redistribution of plantar loads from areas of high pressure to areas of low pressure, and that obese individuals will have higher plantar pressures under select portions of the foot when compared to non-obese individuals. As such, the mid foot and fore foot plantar pressures were found to be significantly higher with obesity (Messier et al. 1994; Dowling and Steel 2001; Fabris et al. 2006). Though the exact mechanism by which obesity impairs foot function remains unclear, a combination of reduced sensitivity and heel pain may compound to impair balance and increase the risk of falls.

2.5 Effects of obesity on balance during quiet standing

Epidemiological evidence linking obesity with an increased risk of falls suggests that obesity negatively affects balance. In support of this, numerous studies have reported increased postural sway during quiet standing. Balance during quiet standing is typically quantified using several center of pressure (COP) measures, and can be defined as the neuromuscular response to the imbalances of the body's COM (Winter 2005). These measures, including mean COP displacement and mean COP velocity, have repeatedly been related to risk of falling (Fernie et al. 1982; Maki et al. 1990; Maki et al. 1994; Prieto et al. 1996). Mean COP displacement represents the “degree” of postural stability, or the excursion of the COP towards the boundaries of the base of support (Maki et al. 1990), and indicates the effectiveness of postural control (Maki et al. 1990; Prieto et al. 1996). Mean COP velocity represents the amount of activity needed to regulate body sway oscillations and maintain an upright posture (Maki et al. 1994). Though falls typically occur from some type of postural perturbation (Horak et al. 1997), and not during quiet standing, the factors that increase postural sway most likely also increase the risk of falls.

Several studies have investigated the effects of obesity on balance in young populations (McGraw et al. 2000; Bernard et al. 2003; Goulding et al. 2003; Colne et al. 2008). Goulding et al. (2003) used several clinical balance tests, including the Bruininks-Oseretsky test, to observe static and dynamic balance in

overweight and lean boys of age 10 to 21 years. The Bruininks-Oseretsky test of balance consisted of three static balance tasks (standing on one leg on the floor, and standing on one leg on balance beam with and without eyes open) and five dynamic balance tests (walking on a line and balance beam, walking heel-to-toe on a line and balance beam, and stepping over a stick on the balance beam). Their results showed lower Bruininks-Oseretsky test scores in overweight boys, indicating poorer balance. McGraw et al. (2000) observed obese and non-obese boys of age 8 to 10 years during quiet standing with a normal (heels together) and tandem (dominant foot forward, heel-to-toe) stance and several visual conditions (full vision, dark, and visual conflict). Obese boys had greater maximum COP displacement, root mean square COP, and sway areas than non-obese boys in the anterior-posterior and medial-lateral direction. These results suggest that obese children have poorer balance than non-obese children, particularly in the medial-lateral direction. In contrast, Bernard et al. (2003) found obese adolescents of age 13 to 17 to have a greater COP path length during quiet standing only when standing on a foam surface, a more challenging balance task than standing on a hard surface. Similarly, Colne et al. (2008) found no differences in COP displacement in the anterior-posterior direction between obese and non-obese adolescents (average age of 17 years) while standing on a hard surface. Colne et al. (2008) also assessed balance during a forward leaning posture in which subjects were to rotate forward about the ankles as far as possible without lifting the heels or toes from the ground. During a forward lean, obese adolescents had decreased COP displacements and similar sway areas compared to non-obese adolescents. However, measurements during a sustained forward lean were taken at each subject's maximum lean angle and these angles were not reported. It is possible that the smaller COP displacements in obese adolescents compared to lean adolescents could be due to a smaller maximum lean angle in obese individuals. These findings suggest that more challenging balance tasks are needed to observe differences in balance during quiet standing between obese and non-obese adolescents.

While these studies investigated the effect of obesity on balance in children up to 21 years, the effect of obesity on postural sway has also been investigated in adults. Some studies have found an association of

adult obesity and postural sway parameters. For example, Hue et al. (2007) found that as mass increased, mean COP displacement and mean COP velocity increased. These results suggest that an increase in body weight causes an individual to be less sensitive in the regulation of body sway oscillations. Other studies have reported similar results of increased mean COP speed with obesity (Chiari et al. 2002; Teasdale et al. 2007; Handrigan et al. 2010). Singh et al. (2009) also found obese individuals to have poorer balance compared to non-obese individuals in several measures of postural sway during prolonged (>18 minutes) quiet standing. In addition, as time increased, performance decreased among obese individuals, but not among non-obese individuals. These results indicate that obesity may impair postural control, and that this effect becomes more apparent the longer the task is performed.

Although measures of postural sway are have been related to risk of falling (Fernie et al. 1982; Maki et al. 1990; Maki et al. 1994; Prieto et al. 1996), they do not indicate how or why risk of falling increases with obesity. Most falls are caused by a slip or a trip (Horak et al. 1997; Courtney et al. 2001). The risk of falling by slipping or tripping is dependent on: 1) the likelihood of losing balance by slipping or tripping, and 2) the ability to recover balance after slipping or tripping to avoid a fall (Robinovitch et al. 2002). Therefore, in order to understand the specific reasons why individuals who are obese have an increased risk of falling, it is necessary to investigate the effects of obesity on the likelihood of losing balance and the ability to recover balance.

2.6 Effects of obesity on the likelihood of losing balance

It is possible that the risk for falling among obese individuals could be increased due to an increased likelihood of slipping or tripping (Robinovitch et al. 2002). Slipping occurs when the shoe/floor interface is unable to support the shear force necessary to keep the foot in place. The required coefficient of friction (RCOF), or utilized coefficient of friction, is an indicator of the risk of slipping during gait (Redfern et al. 2001). RCOF is calculated as the ratio of shear ground reaction force (GRF) to vertical GRF. A higher RCOF value (e.g., 0.5–0.6) indicates a larger shear force is needed to prevent a slip, and

therefore the risk for slipping is much higher. Tripping occurs when the foot is obstructed during the swing phase of gait. As such, the minimum ground clearance of the foot during this phase has been used as an indicator of tripping risk (Rietdyk et al. 2005). A smaller toe clearance value implies an increased risk of tripping.

Obesity can influence several gait characteristics that also influence both RCOF and the minimum ground clearance (Corbeil et al. 2001; DeVita and Hortobagyi 2003; Browning and Kram 2007; Hue et al. 2007; Lai et al. 2008). As a result, obesity likely influences the risk of slipping and tripping. Unfortunately, the actual effect of obesity on the risk of slipping or tripping is unclear due to a lack of studies directly investigating these gait characteristics. Several studies that have reported the effects of obesity on other related gait characteristics have conflicting implications. Obese individuals exhibit significantly lower preferred walking speed, step length, and step frequency compared to non-obese individuals (Spyropoulos et al. 1991; DeVita and Hortobagyi 2003; Fabris de Souza et al. 2005; Lai et al. 2008). Additionally, obese individuals have a longer stance phase and greater period of double support (Spyropoulos et al. 1991; DeVita and Hortobagyi 2003; Lai et al. 2008). These gait characteristics would imply a decreased risk for slipping. In contrast, Browning & Kram (2007) found peak anterior-posterior (AP) ground reaction force (GRF) to be 63% higher in obese compared to non-obese individuals, while the vertical GRF was only 60% greater. This indicates that individuals who are obese use greater shear forces at the shoe/floor interface than non-obese individuals during gait, and therefore would have an increased risk of slipping. Similar conflicting evidence exists for tripping risk. Individuals who are obese walk with less hip and knee flexion, suggesting a smaller clearance of the foot from the ground and greater risk of tripping (Spyropoulos et al. 1991). However, obese individuals also exhibit greater ankle dorsiflexion (Spyropoulos et al. 1991), possibly increasing toe clearance, and thereby decreasing the risk of tripping.

To summarize, it is unclear whether obesity affects the likelihood of losing balance. Gait studies in the literature imply both an increased and decreased risk of slipping or tripping in individuals who are obese. Therefore, studies are needed to directly assess the effects of obesity on the likelihood of losing balance.

2.7 Effects of obesity on balance recovery

The increased risk of falls among obese individuals could also be due to a decreased ability to recover balance after a perturbation and avoid a fall (Robinovitch et al. 2002). However, only two studies to our knowledge have investigated the effects of obesity on balance recovery. In the first study, Miller (2008) investigated the effects of obesity on balance recovery after small postural perturbations. A ballistic pendulum was used to apply small anteriorly-directed perturbations just inferior to the scapula. These perturbations were small enough so that subjects could recover without moving their feet or taking a step. Obese subjects exhibited reduced peak COP displacement and velocity and peak COM displacement and velocity when subjects were exposed to the same impulse perturbation. This could imply that the obese subjects had better balance recovery capability than the non-obese subjects. However, when exposed to the same perturbations relative to body weight, there were no differences in COP or COM displacement and velocity between the obese and non-obese subjects. In the second study, Berrigan et al. (2006) examined the effect of obesity on the postural response to a perturbation resulting from a sudden arm movement. Subjects, in a standing posture, were to point to a target as fast and as precisely as possible after an auditory signal. This rapid acceleration of the arm was considered a small postural perturbation due to the momentum the movement exerted on the body. Obese subjects were found to have greater COP speed and displacement than their non-obese counterparts. During this study, the obese subjects moved their whole body forward while aiming at the target, as can be seen in a greater COP displacement. The authors suggested that by moving their COM closer to the target, the obese subjects were better positioned for correcting hand movements. However, a forward displacement of the COM can result in a greater risk for a forward fall due to a larger corrective torque needed to maintain balance. Additionally, Berrigan et al. (2008) examined the effects of weight loss on this task. It was found that

balance and control of the upper limb during the aiming task in obese subjects improved with weight-loss via hypocaloric diet. The subjects' forward displacement during the aiming task was reduced after weight-loss. These findings suggest that obesity negatively influenced balance control.

The effects of obesity on balance recovery have also been investigated using non-obese subjects with external weight to simulate obesity. In one such study, Li and Aruin (2005) investigated the effects of simulated obesity on feed-forward postural control. Feed-forward postural control is based on a prediction of the effect of an impending perturbation (the previous studies that have been discussed would be considered feed-back postural control, which is the restoration of equilibrium after a perturbation through sensory feedback signals). A perturbation was induced by having subjects stand with their arms extended forward and a 5-lb load placed 10 cm above the subjects' hands. Subjects were required to catch the load while wearing external weight to simulate obesity. This study found that as the amount of external weight increased, so did anticipatory activation of postural muscles. In addition, following onset of the perturbation, COP displacement increased as the amount of external weight increased. These results imply that although there is increased muscle tension in preparation for the perturbation, balance is impaired with increased weight as seen by increases in COP displacement. It should be noted that a limitation to the use of non-obese subjects with external weight to simulate obesity is that there may be adaptations in motor and postural control if the increased mass was worn day to day as in the obese. Experiments using non-obese subjects with external weight may not account for such adaptations.

Studies are needed to investigate the effects of obesity on balance recovery after large scale perturbations in order to identify the biomechanical mechanisms by which obesity contributes to fall risk. Fall prevention interventions can then be developed that target these mechanisms.

In addition to these studies, fundamental mechanics suggests a potentially ambiguous relationship between obesity and balance recovery. To illustrate this, consider an inverted pendulum model of the

human body (Figure 2.1). This is a reasonable model of the body during quiet standing and recovery from small postural perturbations because movements are typically limited to the ankle (i.e. the so-called ankle strategy). First, obese individuals have increased body weight, which leads to an increased gravitational moment about the ankles. This results in an increased angular acceleration about the ankles, and would require larger ankle plantar flexor torque to recover from anteriorly-directed postural perturbations, making balance recovery more physically demanding (Corbeil et al. 2001). Second, obese individuals also have an increased mass moment of inertia about the ankles. Inertia can be defined as the resistance to rate of change in velocity (Meriam and Kraige 2002). Therefore, the increased inertia in obese individuals may be beneficial in resisting an increase in angular velocity. This may help to limit how quickly the body angular velocity increases as an individual falls forward, making balance recovery less physically-demanding after selected types of perturbations. These potentially offsetting fundamental mechanical considerations make it difficult to predict the effects of obesity on balance recovery using an ankle strategy. It should also be noted that while weight and inertia are closely related, they are not necessarily directly proportional to one another because inertia is dependent upon the distribution of mass over the body, which can vary between individuals.

Overall, there is very limited information as to the effects of obesity on balance recovery. Although obesity has been shown to impair balance during quiet standing, balance recovery ability is not associated with postural sway (Owings et al. 2000). Therefore, impaired balance during quiet standing does not necessarily mean obese individuals also have an impaired ability to recover balance. Additionally, previous studies on balance recovery in obese individuals did not challenge subjects to their maximum capabilities. Moreover, fundamental mechanics suggest a highly ambiguous relationship between obesity and balance recovery. It is possible that if differences in balance recovery exist between obese and non-obese individuals, more challenging conditions are needed to identify these differences (Alexander et al. 1992).

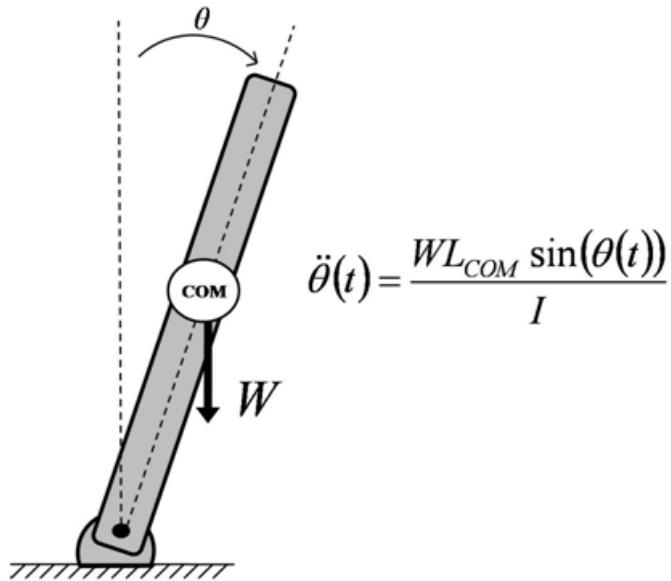


Figure 2.1. Inverted pendulum of the human body. The COM is located a distance L_{COM} from the ankle joint. From the equation of motion of the pendulum (angular acceleration, $\ddot{\theta}$), one can see if weight (W) is increased, angular acceleration increases. However, if inertia (I) is increased, angular acceleration decreases. Since both weight and inertia are increased in obese individuals, the overall effect on angular acceleration is unclear.

2.8 Fall interventions for the obese

The high and increasing prevalence of obesity and its association with an increased risk of falls point to a substantial need for fall interventions aimed at obese individuals. Weight loss is critical for improving the overall health in obese individuals (Pi-Sunyer 2002). However, because the mechanisms by which obesity increases fall risk are not well understood, development of an intervention may not be as simple as losing weight. For example, when considering the inverted pendulum problem (Figure 2.1), decreasing weight would decrease the gravitational torque, and would reduce the effort to recover balance. Weight loss would also decrease the subject's inertia, thereby increasing the subject's sensitivity to changes in velocity and potentially increasing the effort to recover balance. Additionally, weight loss is commonly accompanied by several negative side effects that could increase the risk of fall and subsequent injury. These include decreases in bone mineral density (Ensrud et al. 2003) and decreases in muscle mass and strength (Weiss et al. 2007; Hue et al. 2008). Therefore, it is unclear if and to what degree weight loss

interventions would be beneficial for balance recovery, or if other interventions are necessary to supplement weight loss.

Weight loss alone has can reduce postural sway during quiet standing. Teasdale et al. (2007) imposed a hypocaloric diet and bariatric surgery on obese and morbidly obese subjects, respectively. Mean COP speed decreased linearly with weight-loss in both groups. Additionally, there were no differences in mean COP speed between the non-obese and either the obese or morbidly obese group after weight loss. Decreases in COP displacement in the anterior-posterior and medial-lateral directions were also seen with weight loss. These results indicate that balance during quiet standing improves with weight loss, and that improvement is linearly related to the magnitude of weight loss. Similarly, Handrigan et al. (2010) investigated the effects of weight loss on relative strength and balance. A hypocaloric diet and bariatric surgery were used for obese and morbidly obese subjects, respectively. In agreement with previous studies, COP speed and absolute lower limb strength decreased with weight loss. However, in agreement with a previous study (Hue et al. 2008), relative lower limb strength (to body weight) increased with weight loss. This study suggests that even with the reduction in absolute lower limb strength from weight loss, the increase in relative lower limb strength is substantial enough to meet the very low strength demands of quiet standing. Maffiuletti et al. (2005) also investigated the effects of weight loss on balance during single limb stance. Obese subjects participated in a body weight reduction (BWR) program that included an energy-restricted diet, moderate exercise, and nutritional education. A portion of the obese subjects also received balance training in addition to the BWR program. Balance training consisted of repeated exposures to the balance task, i.e. single limb stance. It was found that trunk sway decreased and time of balance maintenance increased with weight-loss. However, these changes were found only with the combination of BWR and balance training. This study suggests that weight loss by itself may not be effective for improving balance. Overall, these studies suggest that weight loss interventions have potential to improve balance. However, muscle mass and strength can decrease with weight loss (Weiss et al. 2007). These decreases may not be problematic for balance during quiet standing because this task

has relatively low strength demands. However, it is unclear if the decreases in strength would negatively affect balance recovery from more challenging tasks with greater strength demands (i.e. a trip).

Strength training is a commonly used fall-prevention intervention for older adults (Seguin and Nelson 2003), and could also be beneficial obese individuals as well. Balance recovery after a postural perturbation has been shown to improve with increased strength (Corbeil et al. 2001; Robinovitch et al. 2002). In particular, impaired ankle and knee strength are significant contributors to falls (Whipple et al. 1987). However, lower limb function in obese individuals is impaired and obese individuals have reduced lower limb strength and power per body weight compared to non-obese individuals. Therefore, improving lower limb strength could help to provide more adequate force production and improve balance recovery ability. Additionally, strength training can be used in conjunction with weight loss interventions to attenuate decreases in muscle mass and strength from losing weight (Sartorio et al. 2003). Although strength training has been encouraged as a fall prevention intervention among obese individuals (Clark 2004), no studies have investigated the effects of strength training on balance recovery in obese individuals.

A study was completed to understand the relative effects of weight loss and strength training on balance recovery using an ankle strategy (Matrangola and Madigan 2009). This study used a combination of laboratory experiments and mathematical modeling. Independent manipulation of weight and strength in human subjects is difficult and time-consuming. Therefore, laboratory experiments were used to determine balance recovery ability in the obese and inputs for a mathematical model of the balance recovery task. Weight loss and strength gain were then applied to the mathematical model. In the laboratory experiments, nine male subjects (aged 23.3 ± 4.5 years) with BMI 30.1 to 36.9 kg/m^2 were released from a forward lean and attempted to recover balance using an ankle strategy. Repeated trials were performed while increasing the initial lean angle until subjects could no longer recover their balance. The maximum initial angular position (θ_{max}) from which the subject could recover was used as the

measure of balance recovery capability. Subjects' mass, height, and ankle torque during recovery were used as inputs to an inverted pendulum model of balance recovery. The 2-D model included a slender rod rotating about a hinge joint to model the body rotating about the ankle joints with a torque actuator to represent the effect of the ankle muscles. Multiple simulations were used to determine the effects of ankle strength and weight loss on θ_{max} . Results showed that less weight loss was needed compared to increased strength for a unit improvement in θ_{max} (improved balance recovery ability). For example, an $8.6 \pm 0.8\%$ decrease in weight or $15.3 \pm 1.1\%$ increase in strength would be required to improve θ_{max} by 1 degree (Figure 2.2). This suggests that weight loss may be a more potent intervention than strength training in improving balance recovery using an ankle strategy. Several limitations of this study are important to consider when interpreting results. First, these results do not give insight on which intervention may be easier for individuals to achieve. For example, it may be easier for some individuals to achieve a 15% increase in strength rather than a 15% decrease in weight. It is likely that the difficulty of achieving these changes would be highly dependent upon the specific interventions employed and differences between individuals. Second, this study focused on balance recovery using an ankle strategy and it is uncertain how the relationship between balance recovery ability and weight loss or strength training may change with more challenging strategies (i.e. hip, combined ankle and hip, stepping) or tasks (i.e. trip, slip).

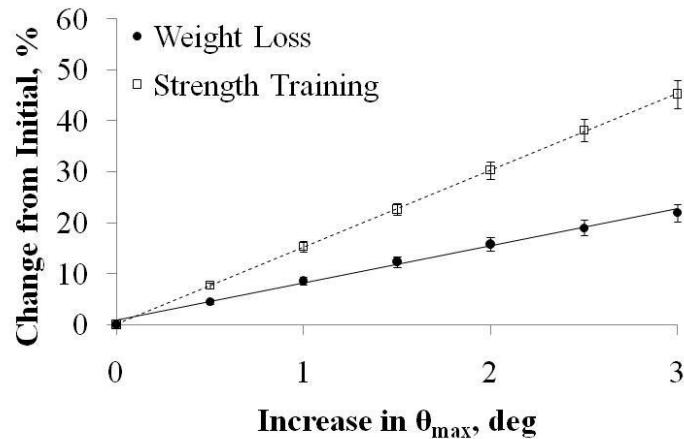


Figure 2.2. Average changes in weight and strength to improve θ_{max} . Smaller changes in weight loss were needed than increases in strength for a given improvement in θ_{max} .

2.9 Summary

The prevalence of obesity is high and increasing. A grave concern with this high and increasing prevalence is the association of obesity with an increased risk of falls and subsequent injury.

Epidemiological evidence linking obesity with an increased risk of falls suggests that obesity negatively affects balance. In support of this, numerous studies have reported increased postural sway during quiet standing (Chiari et al. 2002; Hue et al. 2007; Teasdale et al. 2007; Handigan et al. 2010). The risk of falling is dependent on: 1) the likelihood of losing balance, and 2) the ability to recover balance to avoid a fall (Robinovitch et al. 2002). Due to conflicting evidence in literature, it is unclear how obesity affects the likelihood of losing balance. Based upon the increased risk of falls among obese individuals and the fact that most falls result from some type of postural perturbation (Horak et al. 1997), it can be hypothesized that obese individuals have an impaired ability to recover their balance after a postural perturbation. Unfortunately, there is limited knowledge on the effects of obesity on balance recovery and fundamental mechanics suggest a highly ambiguous relationship between obesity and balance recovery. This makes it difficult to predict how obesity influences balance recovery. Studies are therefore needed to better understand how obesity increases risk of falls.

By understanding the mechanisms by which obesity contributes to fall risk, interventions can be developed to reduce that risk. Weight loss is critical for improving the overall health in obese individuals (Pi-Sunyer 2002). However, the mechanisms by which obesity increases fall risk are not well understood and there are several negative consequences associated with weight loss, such as decreases in muscle mass and strength (Weiss et al. 2007). Therefore, development of an intervention may not be as simple as losing weight. Both weight loss and strength training have the potential to improve balance recovery ability, but the relationship between each of these interventions and balance recovery remains unclear.

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Chapter 3– Forward dynamic simulations of movement

3.1 Introduction

There are two general types of analyses for rigid-link models of the human body. The first type is inverse dynamics, which uses measured kinematics (inputs, ex. joint position, velocity, acceleration) to determine kinetics (outputs, ex. muscle forces, joint torques) (Winter 2005). Inverse dynamics is widely used for determining the forces and moments that generated a measured movement. However, limitations include the motion to be known and an inability to alter the model inputs (kinematics) to investigate its effects on model outputs (kinetics). The second type of analysis is forward dynamics, which uses forces to determine motion (Winter 2005). Unlike inverse dynamics, this type of analyses does not require experimental kinematics as input, and can be used to explore how changes in model input (kinetics) affect model output (kinematics). This allows for the investigation of a large number of research questions related to human movement, and makes forward dynamics a powerful research tool in the biomechanics field.

3.2 Benefits and limitations of forward dynamic simulations

There are several benefits to using forward dynamics simulations for studying human movement. First, forward dynamics simulations allow researchers to investigate variables that could not otherwise be manipulated in human subjects. For example, one could manipulate specific muscle parameters, such as maximum isometric force or optimal fiber length of the muscle. Second, and closely related to the first benefit, is increased experimental control. For example, one could increase maximum isometric force in a human through training, but other variables likely change too. Forward dynamics allows the researcher to change only the maximum isometric force to examine its specific effect. The third benefit is also closely related to the first and second, and is that forward dynamics simulations help to establish cause and effect relationships within complex dynamical systems. For example, simulations have been developed to examine the contribution of specific muscles to bicycle pedaling (Neptune et al. 2000) and

gait (Neptune et al. 2001). Researchers determined the contribution of a muscle by comparing the model predictions with and without the muscle of interest. A fourth benefit to forward dynamics simulations is that muscle forces can be quantified. Generally, it is difficult to accurately determine muscle forces because of the redundancy of the musculoskeletal system. Typically, more muscles cross a joint than there are degrees of freedom for that joint, resulting in an underdetermined system of equations for solving muscle forces (i.e. more unknowns than equations). Finally, forward dynamics simulations can be cost effective. For example, weight-loss programs in human subjects can last several months (Paquette et al. 2000; Teasdale et al. 2007). A forward dynamics simulation would require initial model building time and computational time, and can be far less costly than implementing the intervention experimentally. Therefore, forward dynamics simulations can be used to gain insight on potential interventions to implement on human subjects for a longitudinal study.

Forward dynamic simulations also pose several limitations. First, it can be challenging to determine the level of model complexity needed to answer a given research question. The simpler a model is, the easier it is to understand the parameter effects (Alexander 2003). Therefore, it is desirable to make the model as simple as possible. However, a model that is too simple may fail to capture the true nature of the problem. For example, when the mass of the legs are ignored in a model of human jumping, there was no advantage to activating the muscles in a sequential order (Alexander 1989). Pandy et al. (1990) developed a more complex model that included leg mass, and were able to find an optimal sequence of muscle activation that also agreed well with patterns used by athletes. Second, it can be difficult to determine specific but essential model parameters. For example, Bobbert (2001) assumed certain properties of muscles in order to be able to model several lower limb muscles because the necessary experimental data was not available. Third, most systems used in forward dynamics require complex mathematics to determine the equations of motion. As a result of these complex mathematics, numerical approaches are used that require large computation times. For example, it is not uncommon for some

simulations to require more than 1,000 hours of central processing unit (CPU) time (Anderson and Pandy 2001).

3.3 Forward dynamic simulation framework

The framework of forward dynamic simulations is as follows (Figure 3.1). First, a control signal (input) prescribes an activation level for each actuator in the model. The magnitude of these activations, in combination with the current state of the system, determines the amount of muscle force or joint torque by the model actuators at that instant in time. These muscle forces and/or joint torques, along with any external forces, are then entered into the body segment dynamics (equations of motion), which are then used to determine body segment accelerations. These accelerations are then integrated to determine the new state of the system (i.e. body segment velocities and positions). This new state of the system is then used to determine body motion at the next time step.

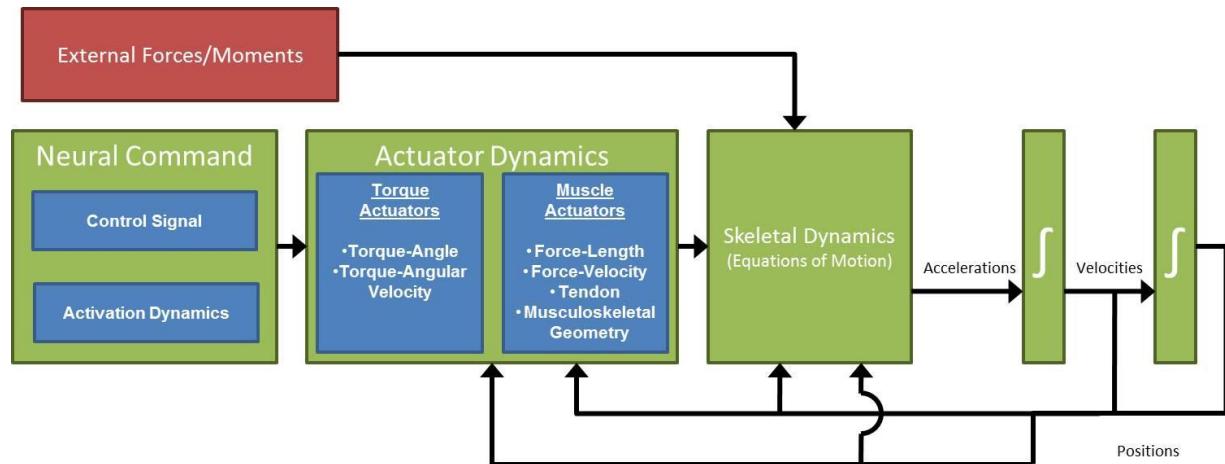


Figure 3.1. Forward dynamics simulation diagram.

3.3.1 Activation dynamics

The input to forward dynamics as shown in Figure 3.1 is a control signal (neural excitation), which prescribes an activation level for each actuator in a model. When using torque actuators, the control signal is represented by a joint activation that scales the torque at a given joint. When using muscle actuators, motor control is represented by a muscle activation that scales the force in a given muscle.

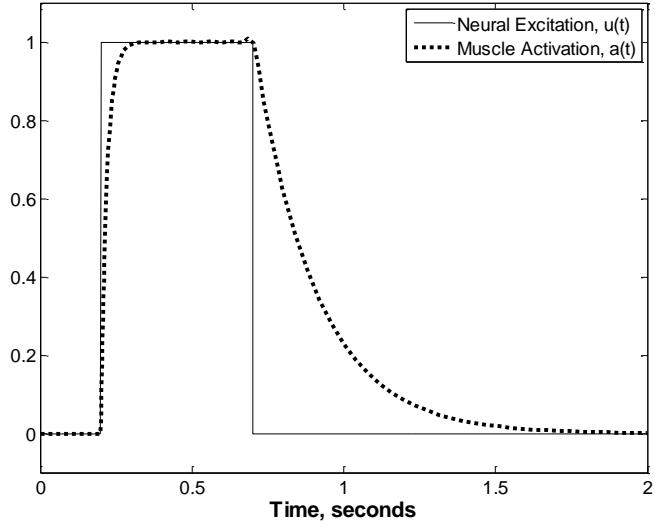


Figure 3.2. Neural excitation, $u(t)$, can be modeled using a step function. Given τ_{act} and τ_{deact} , muscle activation, $a(t)$, can be determined. For the plot presented here, time constants were estimated to be $\tau_{act}=20\text{ms}$ and $\tau_{deact}=200\text{ms}$.

A muscle cannot activate or relax from the control signal instantaneously (Figure 3.2). There is a delay from activation to force production due mainly to the time it takes calcium to leave the sarcoplasmic reticulum and bind to troponin (Pandy 2001). One can account for these delays in muscle force or joint torque from the control signal by modeling muscle activation with a first-order differential equation (Zajac 1989) :

$$\frac{da(t)}{dt} + \left[\frac{1}{\tau_{act}} (\beta + [1 - \beta] \cdot u(t)) \right] \cdot a(t) = \left(\frac{1}{\tau_{act}} \right) \cdot u(t) \quad (3-1)$$

$$\beta = \frac{\tau_{act}}{\tau_{deact}} \quad (3-2)$$

where $a(t)$ is the muscle activation , τ_{act} is the time constant for activation, τ_{deact} is the time constant for deactivation, β is the ratio of the activation and deactivation constants, and $u(t)$ is the neural excitation. Time constant values can range from 12-20 ms for τ_{act} and from 24-200 ms for τ_{deact} (Pandy 2001). Similar equations can be used for torque actuators with parameters that represent the whole joint rather than specific muscles.

3.3.2 Actuators

Rigid-link models of the human body represent the mass and inertial characteristics of the body segments.

In order to get the model to move, some type of actuator is needed. There are two types of actuators that are commonly used when creating a musculoskeletal model for forward dynamics simulations: joint torque actuators or muscle actuators. Both types of actuators can account for well-known characteristics of muscle function such as force-length and force-velocity relations. The selection of the type of actuator depends highly on the research question and the desired model complexity. Torque actuators are the simpler of the two actuators and require fewer parameters to define the actuator model as they represent the net effect of multiple muscles that cross a joint. Muscle actuators represent individual muscles or groups of closely related muscles. Muscle actuators allow researchers to manipulate specific muscle groups or muscle activation patterns and to determine their effect on the outcome of the simulation.

3.3.2.1 Torque actuators

Torque actuators can be implemented in varying levels of complexity. One of the simplest torque actuators is a function of activation level and maximum isometric torque (Yang et al. 2007; Yang et al. 2008). Yang et al. (2008) used such a torque actuator for a seven-link model of slipping during gait to determine the threshold of center of mass (COM) velocity required to prevent a backward loss of balance. This model accurately predicted balance thresholds, and all human subjects whose COM velocity was below the predicted threshold resulted in a backward step instead of a recovery step forward.

More commonly, a torque actuator is a function of activation level, maximum isometric torque, joint angle, and joint angular velocity (Selbie and Caldwell 1996; Ashby and Delp 2006; Cheng 2008; Cheng et al. 2008). Similar to the force-length and force-velocity properties of muscle in which the maximum voluntary force generated in the muscle is dependent on the current length and shortening velocity of that muscle, joint torque is modulated by joint angle and joint angular velocity:

$$T = T_{max} * f(\theta) * h(\omega) * A(t) \quad (3-3)$$

where T_{max} is the maximum isometric torque (units: Nm), and three scaling factors that are dimensionless and vary from 0 to 1: $f(\theta)$ is the joint angle factor, $h(\omega)$ is the joint angular velocity factor, and $A(t)$ is the joint activation level. These scaling factors are used to scale the value of T_{max} based on the current joint angle and angular velocity. For numerous biomechanical models involving the lower limb (Selbie and Caldwell 1996; Ashby and Delp 2006; Cheng 2008; Cheng et al. 2008), the torque-angle relationship is typically described from previous experimental data (Hoy et al. 1990; Pandy et al. 1990). In particular, Pandy et al. (1990) generated torque-angle data for the ankle, knee, and hip from several experimental studies. Selbie and Caldwell (1996) utilized this data and generated equations to describe the torque-angle relationship of these joints as:

$$f_A = 0.65 - 0.25(\tan([8.0 * \theta_A] - 6\pi)) \quad (3-4)$$

$$f_K = 1.0 - 0.4(\theta_K - 2.18)^2 \quad (3-5)$$

$$f_H = 0.80 - 0.16(\tan([4.0 * \theta_H] - 8.78)) \quad (3-6)$$

where the subscripts A , K , and H represent the ankle, knee, and hip, respectively. The torque-velocity relation was first described by Alexander (1989) as:

$$\begin{cases} h(\omega) = \frac{(\omega_0 - \omega)}{(\omega_0 - \Gamma\omega)} & , \omega/\omega_0 < 1 \\ h(\omega) = 0 & , \omega/\omega_0 \geq 1 \end{cases} \quad (3-7)$$

where ω is the angular velocity, ω_0 is the maximum angular velocity (20 rad/sec), and Γ is the shape factor describing the torque-angular velocity curve ($\Gamma=2.5$). When an eccentric muscle contraction takes place, the torque-angular velocity factor is increased to a maximum value of 1.5. Joint activation typically varies from 0, representing no activation, to 1, representing full activation.

3.3.2.2 Muscle actuators

Muscle actuators are the more complex of the two types of actuators and require more parameters to define the actuator model than the torque actuator. For example, a muscle model requires insertion points, lines of action, maximum isometric force, pennation angle, maximum shortening velocity, and various other parameters to define the actuator (Anderson and Pandy 1999). However, torque actuators cannot account for co-contraction of muscles (i.e. simultaneous contraction of agonist and antagonist muscles) or the coupled effects of bi-articular muscles. Bi-articular muscles provide a mechanical coupling that can facilitate movement control and efficiency.

Muscle actuators allow researchers to manipulate specific muscle groups or muscle activation patterns and to determine their effect on the outcome of the simulation. Such simulations have been developed to examine the contribution of specific muscles to bicycle pedaling (Neptune et al. 2000), contribution of specific muscles to gait (Neptune et al. 2001), and the effects of muscle control and strength on maximum jump height (Bobbert and Van Soest 1994).

The most common muscle actuator (i.e. muscle model) used in forward dynamics simulations is the Hill-type muscle model (Figure 3.3). The muscle is represented by three elements: a contractile element (CE), parallel elastic element (PEE) and series elastic element (SEE) (Winter 2005). The CE represents the sarcomere and is governed by the active force-length and force-velocity properties of muscle. The PEE is thought to represent the connective tissue (epimysium, perimysium, and endomysium) that surrounds the CE and is governed by the passive force-length property of muscle. The SEE typically represents the tendon and is governed by the passive properties of the tendon. This muscle model is frequently used because it captures the nonlinearity of the muscle dynamics, can be scaled based on anatomical dimensions, and is computationally simple (Winters and Crago 2000).

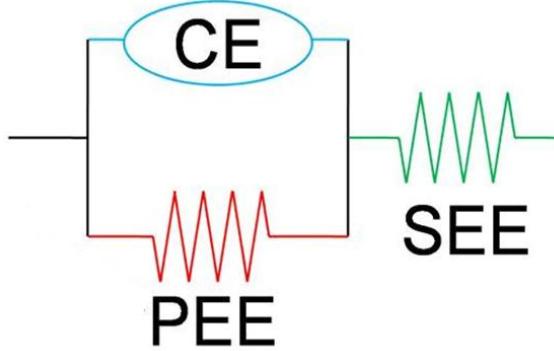


Figure 3.3. The Hill-type muscle model with three elements: the contractile element (CE) governed by the active force-length and force-velocity properties of muscle, the parallel elastic element (PEE) governed by the passive force-length property of muscle, and the series elastic element (SEE) governed by the passive properties of the tendon.

Because the model assumes that the muscle elements (CE and PEE) are in series with the tendon (SEE), the force in the tendon is equal to the force in the muscle. Using this information, the musculotendon contraction dynamics can be expressed dimensionally as (Zajac 1989):

$$\frac{d\tilde{F}^T}{d\tau} = \tilde{k}^T \cdot [\tilde{v}^{MT} - \tilde{v}^M] \quad (3-8)$$

where \tilde{F}^T is the tendon force (normalized to peak isometric active force in the muscle, F_0^M), \tilde{k}^T is the tendon stiffness (normalized to the ratio of optimal muscle fiber length, L_0^M , to F_0^M), \tilde{v}^{MT} is the musculotendon velocity (normalized to the maximum shortening velocity of the muscle, v_M), and \tilde{v}^M is the muscle fiber velocity (normalized to v_M). Muscle fiber velocity can be expressed as a function of actuator (musculotendon) length (\tilde{L}^{MT} , normalized to L_0^M) and muscle activation ($a(\tau)$, with time normalized to the time-scaling parameter τ_c) (Zajac 1989):

$$\tilde{v}^M = f(\tilde{L}^{MT}, \tilde{F}^T, a(\tau)) \quad (3-9)$$

$$\tau_c = \frac{L_0^M}{v_m} \quad (3-10)$$

Using dimensionless curves for the force-length of passive and active muscle, force-velocity, and activations dynamics in combination with muscle specific parameters (F_0^M , L_0^M , fiber pennation angle, v_M) and tendon slack length, the Hill-type model can be scaled for any muscle. After the muscle model is scaled by the muscle specific parameters, these equations, the muscle length, the muscle shortening velocity, and activation level at a given time instant are then used to determine the force output of the muscle actuator at that time instant.

3.3.3 Body Segment Dynamics

Both torque actuators and muscle actuators generate torques or forces that are applied to a system of interconnected rigid bodies that serve as a model of the body segments. The equations of motion (EOM) relating forces and torques applied to the rigid bodies and the resulting body segment motion can be expressed as:

$$M(\underline{q})\ddot{\underline{q}} + C(\underline{q})\dot{\underline{q}}^2 + G(\underline{q}) + R(\underline{q})F^{MT} + E(\underline{q}, \dot{\underline{q}}) = 0 \quad (3-11)$$

where \underline{q} , $\dot{\underline{q}}$, $\ddot{\underline{q}}$ are vectors of the generalized coordinates, velocities, and accelerations, respectively; $M(\underline{q})$ is the system mass matrix and $M(\underline{q})\ddot{\underline{q}}$ is a vector of inertial forces and torques; $C(\underline{q})\dot{\underline{q}}^2$ is a vector of centrifugal and Coriolis forces and torques; $G(\underline{q})$ is a vector of gravitational forces and torques; $R(\underline{q})$ is the matrix of muscle moment arms; F^{MT} is a vector of musculotendon forces; the product $R(\underline{q})F^{MT}$ is therefore a vector of musculotendon torques; and $E(\underline{q}, \dot{\underline{q}})$ is a vector of external forces and torques applied to the body (Pandy 2001).

Lagrange mechanics is one of the most common methods for determining the EOM of a system. Lagrange mechanics is considered an analytical approach and often allows one to determine all EOM without solving explicitly for constraint forces acting on the system because it is based on the principle of

virtual work (Greenwood 1977). This is useful in that it reduces the number of equations describing the system and the number of unknown variables. The Lagrangian function, L , is first calculated from the kinetic (T) and potential (V) energy of the system as:

$$L = T - V \quad (3-12)$$

The Lagrange equations are then determined by:

$$\frac{d}{dt} \left(\frac{\partial L}{\partial \dot{q}_i} \right) - \frac{\partial L}{\partial q_i} = Q'_i \quad (3-13)$$

where \dot{q}_i and q_i are generalized coordinates and Q'_i are generalized applied forces (Greenwood 1977).

Examples of generalized forces include muscle forces, joint torques, and ground reaction forces.

Software packages are available to determine the EOM, and are a practical necessity for complex biomechanical models. SD/FAST (Symbolic Dynamics, Inc., Mountain View, CA) and Autolev (OnLine Dynamics, Inc., Sunnyvale, CA) are two of the most common dynamic engines used in the biomechanics field.

3.4 Motor Control

A time history of torque (or muscle) activation is needed as input to a forward dynamic simulation. It can be very challenging to determine these activations that generate realistic and smooth movements of the desired task due to the high level of complexity and redundancy of the musculoskeletal system. Two methods are commonly used to determine the activation signals, and both use optimization techniques to solve for the activation levels that minimize (or maximize) an objective function. For the so-called “tracking method”, the objective function is a calculation of the differences between experimentally collected and simulated kinematics and kinetics. This is done by an optimization algorithm that systematically varies the activation signals to find the specific signals that minimize the difference

between simulation and experimental data. The tracking method is useful for estimating motor control while preserving the experimental movement. However, a drawback to this approach is that experimental data is needed and is not always available. The other method, optimization of a performance based objective function, is used when the task has a readily defined goal and the objective function is designed to meet this goal. For example, during walking, metabolic energy consumption is minimized, and therefore an objective function that solves for activation signals that minimize metabolic energy was used to simulate walking (Anderson and Pandy 2001). This method can produce realistic musculoskeletal movement without requiring experimental data. However, there is a trade-off in that it can be difficult to define an objective function.

3.5.1 Tracking method

The tracking method determines activation signals so that the simulated body motion matches the experimental body motion (Neptune 1999). The tracking method has been utilized for numerous biomechanical applications including examining differences in muscle excitations between normal and pathological gait (Delp et al. 1998; Delp et al. 2007), muscle function and contribution towards movement (Neptune et al. 2001; Neptune et al. 2004), and injury mechanisms (Gerritsen et al. 1996; Neptune et al. 2000). Examples of studies that used forward dynamic simulations and the tracking method are presented in this section.

The goal of a study conducted by Delp et al. (2007) was to identify the sources of pathological movement in a spastic cerebral palsy patient and explore the effects of possible treatments. Forward dynamic simulations were used to determine cause and effect relationships between muscle excitations and the resulting movement. Because the researchers desired to understand the mechanisms behind the pathological gait seen experimentally, the tracking method was used to determine a set of muscle activations that produce a muscle-driven simulation of the experimental movement. A 12-year-old patient with spastic cerebral palsy was selected as a case study on stiff-knee gait, a condition in which

swing-phase knee flexion is substantially diminished. The reduced knee flexion is often attributed to excessive excitation of the rectus femoris during the swing phase of gait (Perry 1987). Stiff knee gait is commonly treated with: 1) botulinum toxin injections, which reduce muscle activation and therefore hip and knee moment-generating capacity, or 2) rectus femoris transfer, which moves the rectus femoris tendon from the patellar tendon to the iliotibial band and reduces knee extension moment while maintaining hip flexion moment (Delp et al. 2007). A 3-D musculoskeletal model was scaled to the patient and consisted of 7 segments, 21 degrees-of-freedom and 92 muscle actuators. The tracking method was then used to determine the muscle activations that produce the pathological gait. After determining the excitation signals that reproduced the stiff-knee gait, it was found that the excessive activity of the knee extensors in pre-swing was the major cause of stiff-knee gait in the patient. Subsequently, the musculoskeletal model was used to simulate the botulinum toxin injection and rectus femoris transfer treatments and found that the model increased peak knee flexion the most from a rectus femoris transfer. The patient underwent a rectus femoris transfer and showed improvement in knee flexion similar to the model predictions.

Neptune et al. (2000) aimed to investigate how individual muscles contribute to forward and backward pedaling to better understand the coupling between segments and muscle synergies to accomplish different motor tasks. A forward dynamic approach was necessary because it is extremely difficult to manipulate muscles and muscle activations in humans, let alone to manipulate them independently to determine muscle specific contributions to movement. A two-legged sagittal plane model of a bicycle rider was created and included three segments per leg (foot, shank, thigh) and nine Hill-type muscle actuators. The tracking method was used to determine the muscle activation patterns (including muscle onset, offset, and magnitude of stimulation) to match human subject kinetic, kinematic, and electromyography (EMG) data. A mechanical energy analysis was performed to identify how the muscles generate, absorb, or transfer energy to perform the forward and backward pedaling task (Fregly and Zajac 1996). Results showed that the muscles contribute in similar ways to both forward and

backward pedaling. In addition, complex biomechanical mechanisms of the rectus femoris were identified, such as the negative muscle work to accelerate the crank, which could otherwise not be explained through human experimentation alone.

Gerritsen et al. (1996) aimed to investigate possible anterior cruciate ligament (ACL) injury mechanisms during a landing movement high-speed downhill skiing. As mentioned previously, it is very difficult to measure and manipulate muscle activations in human subjects. Therefore, a forward dynamics approach was used in order to examine motor control of a baseline landing, and then to determine the effects of an “injurious” motor control on ACL loading. A five-segment sagittal plane model (skis, boots, shanks, thighs, and trunk) with eight Hill-type muscle actuators (glutei, hamstrings, iliopsoas, rectus femoris, vasti, gastrocnemius, soleus, tibialis anterior) was used for this study. The tracking method was used to first find the muscle activations for a baseline landing movement. This baseline muscle activation pattern was then used to investigate an injury condition by over-stimulating the gastrocnemius muscle, making the model fall slightly backwards. To recover from the disturbance, the quadriceps and iliopsoas muscles were maximally stimulated. The peak ACL force during the injury simulation was found to be within range of failure loads for the ACL. In addition, external forces (i.e. the landing) were responsible for 75% of the shear loading of the ACL, and that only 25% was due to the fully activated quadriceps muscle. This suggested that external forces are a major contributor to ACL injuries during a landing movement in downhill skiing.

3.5.3 Performance based objective function

Using a performance-based objective function to determine activation signals is common when the task under investigation has a readily defined goal. Previous studies have utilized optimization of a performance based objective function to minimize metabolic energy during walking (Anderson and Pandy 2001), maximize jump height (Pandy et al. 1990; Bobbert and Van Soest 1994; Anderson and Pandy 1999; Cheng 2008), maximize pedaling speed (Raasch et al. 1997), and maximize jump length for

standing long jump (Ashby and Delp 2006). A benefit to this method is that experimental data is not needed. However, there is a trade-off in that it can be difficult to define an objective function depending upon the task of interest. Examples of studies that used forward dynamic simulations and a performance-based objective function are presented in this section.

Anderson and Pandy (2001) investigated a physiological, time-dependent performance criterion that produce a forward dynamic solution for normal walking. The goal of this study was to produce realistic gait by using an objective function and not the tracking method. This was done by minimizing a performance-based cost function based upon metabolic energy expenditure and a penalty function to limit joint hyperextension. The model consisted of 10 segments with 23 degrees-of-freedom and 54 muscle actuators. The simulation reproduced normal walking fairly accurately. The majority of joint kinematics predicted by the model were within one standard deviation of human subject data. Additionally, the ground reaction forces predicted by the model were similar to human subject data. One notable difference included an additional peak in the fore-aft component of the ground reaction force, but was attributed to the foot being modeled as a single segment. Overall, minimizing metabolic energy proved to be a valid objective function to produce simulations of normal walking.

Multiple studies have aimed to identify key factors to maximize jump height, and ultimately improve athletic performance (Pandy et al. 1990; Bobbert and Van Soest 1994; Anderson and Pandy 1999; Cheng 2008). Optimization of a performance based objective function is used for such studies because jumping has a clearly defined goal for a cost function: maximizing jump height. For example, Pandy et al. (1990) aimed to determine the “optimal” muscle activations to maximize jump height. The human body was modeled as a four segment (foot, shank, thigh, HAT) planar linkage with eight Hill-type muscle actuators. Muscle activations were optimized to maximize the model jump height. Qualitatively, the model produced similar kinematics, ground reaction forces, muscular activity sequence, and overall jump height compared to human experiment data. Bobbert and Van Soest (1994) used a similar model of jumping to

investigate the effects of muscle strengthening on jump height. In contrast to Pandy et al. (1990), Bobbert and Van Soest (1994) used only six Hill-type muscle actuators. Muscle activation was optimized to maximize jump height for the baseline model. The maximum isometric force for the knee extensor muscles was increased by 5%, 10%, and 20%, and then all muscles were strengthened by 5%, 10%, and 20%. Finally, muscle activations were re-optimized with the increased strength in the muscles. If the muscles were strengthened without re-optimizing the muscle stimulations, jump height decreases from baseline. When only the knee extensor muscles were increased by 20% and muscle activations re-optimized, jump height increased by 3cm. When all muscles were increased by 20% in strength, and muscle activations re-optimized, jump height increased by almost 8cm. Overall, these findings suggest that motor control needs to adapt to increases in strength to gain the maximal benefit from muscle strengthening. Cheng (2008) also examined the effects of strength on vertical jump performance. Similarly, Cheng (2008) examined the effects of joint strength on maximum jump height. A five segment (feet, shanks, thighs, head/trunk, and arms) model driven by joint torque actuators was used. The maximum isometric torque of one joint was varied by $\pm 20\%$ while keeping all other joints unchanged. Joint torque activations were re-optimized for each strengthening condition. It was found that jump height was most sensitive to knee and ankle joint strength, with a 20% increase in strength corresponding to $\sim 2.4\%$ increase in jump height (3cm).

Forward dynamic simulations and a performance-based objective function have also been used to understand muscle coordination in bicycle pedaling that maximized pedaling speed (Raasch et al. 1997). Maximum pedaling speed was chosen because it is a clearly defined goal for cycling and could be easily used as an objective function. A two-legged model was used, with three segments (foot, shank, and thigh) and nine Hill-type muscle groups per leg. The foot of each leg was rigidly attached to the pedal, and the two pedals were connected by the crank. These simulations suggested that pedaling can be performed, and produce similar kinematics to human experiments, by portioning all the muscles in a leg into two pairs of phase-controlled alternating functional groups. The extensor/flexor pairs generate

energy to both the limb and the crank, while the top/bottom pairs transfer energy from the limb to the crank and provide propulsion through stroke transitions.

Asbhy and Delp (2006) utilized forward dynamic simulations and a performance-based objective function to investigate the mechanisms of arm motion that enhances performance from a standing long jump. The objective function was used to maximize horizontal jump length. The model consisted of five segments (foot, shank, thigh, head-neck-trunk, and arm) and four joint torque actuators (ankle, knee, hip, and shoulder). The magnitude of torque was scaled by activation, joint angle, and joint angular velocity to represent the torque-angle and torque-angular velocity relationship. Torque activations were optimized in order to maximize jump length with and without restricted arms. It was found that the simulated jump distance was 40 cm greater when arm movement was free. The majority of this improvement was due to an increase in take-off velocity from work done by the shoulder actuator.

As noted earlier, if the goal of the movement is not easily defined, the selection of a performance-based objective function is more ambiguous. For example, Pandy et al. (1995) evaluated several different objective functions to determine the optimal muscle activations for rising from a chair. A three-segment sagittal plane model consisting of the shanks, thighs, and head/arms/trunk (HAT) with eight Hill-type muscle actuators was used. Five different performance criterion were evaluated: movement time (TIME), normalized muscle force integrated over time (IMPULSE), normalized muscle force squared and integrated over time (STRESS), total metabolic energy (ENERGY) and the time derivative of muscle force normalized , squared, and integrated over the movement time (FDOT). Minimizing TIME, IMPULSE, STRESS, and ENERGY produced similar solutions for rising from a squat. Therefore, only STRESS and FDOT were evaluated for rising from a chair. Neither criterion was able to reproduce the movement seen in human experimental data. However, when these performance criterions were combined into one objective function (STRESSFDOT), there was good agreement between the simulated and experimental movement. The authors cautioned that even though the STRESSFDOT criterion

reproduced the major components of the movement, one cannot conclude that it uniquely defined the goal of rising from a chair.

3.6 Summary

Forward dynamics simulations are a powerful research tool in the biomechanics field. The musculoskeletal system has a high level of complexity and redundancy, making it extremely difficult to isolate or manipulate some variables (such as muscle forces and muscle activations) within humans. Forward dynamics simulations allow researchers more experimental control, and these variables can be investigated independently and simultaneously. In addition, forward dynamic simulations allow researchers to determine cause and effect relationships between these variables and movement or goals of a task. Without forward dynamic simulations as a research tool may research questions simply could not be addressed.

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Chapter 4 – The effects of obesity on balance recovery using an ankle strategy in young adults

4.1 Abstract

Obesity is associated with an increased risk of falls. The purpose of this study was to investigate the effects of obesity on balance recovery using an ankle strategy. In addition, computer simulations to understand how increased inertia and weight associated with obesity independently influence balance recovery. Ten normal weight (BMI Range: 22.0 to 23.7 kg/m²) and ten obese (BMI Range: 30.0 to 36.1 kg/m²) young adult male subjects were used in this study. Subjects recovered balance using an ankle strategy after three types of postural perturbations: an initial angular displacement, an initial angular velocity from the natural stance, and an initial angular velocity from a prescribed position. Balance recovery was quantified by the largest initial angular displacement or angular velocity from which balance could be recovered. Obesity impaired balance recovery from perturbations involving an initial angular velocity, but not from an initial angular displacement. Similarly, computer simulations determined that increased inertia is beneficial to balance recovery when there is little to no initial angular velocity. These findings indicate that the effects of obesity on balance recovery are dependent on the type of perturbation, and that increased inertia associated with obesity can be beneficial for perturbations that involve little to no initial angular velocity.

4.2 Introduction

Obesity is a major health concern in the United States (US) and around the world. Over one billion people worldwide are considered overweight, and of those, 300 million are considered obese (Puska et al. 2003). In the US, more than one-third of adults, or over 72 million people, are obese (Ogden et al. 2007). The prevalence of obesity is not only high, but it continues to increase. From 1980 to 2000, the prevalence of obesity among adults more than doubled from 15% to 31% (Flegal et al. 2002; National Center for Health Statistics 2008). One of the many concerns with the high and increasing prevalence of obesity is its association with an increased risk of falls and subsequent injury. Obese individuals fall

almost twice as often (27% vs. 15%) compared to non-obese individuals (Fjeldstad et al. 2008), and falls have been identified as the most common (~36%) cause of injuries among obese individuals (Matter et al. 2007).

Consistent with this epidemiological evidence, several biomechanical studies have reported that increased body weight is associated with increased postural sway during quiet standing (McGraw et al. 2000; Chiari et al. 2002; Hue et al. 2007; Menegoni et al. 2009). For example, Hue et al. (2007) found that as weight increased, mean center of pressure (COP) displacement and speed increased. Similarly, Menegoni et al. (2009) found obese adults to have higher mean COP speeds compared to their normal-weight counterparts. Increased postural sway is associated with an increased risk of falling (Fernie et al. 1982; Maki et al. 1994), so these studies also support the notion of an increased risk of falling in individuals who are obese. However, balance performance during quiet standing is not predictive of balance recovery after a postural perturbation.

Based upon the increased risk of falls among obese individuals and the fact that most falls result from some type of postural perturbation (Horak et al. 1997), it can be hypothesized that obese individuals have an impaired ability to recover their balance after a postural perturbation. However, only two studies to our knowledge have investigated the effects of obesity on balance recovery. Miller (2008) investigated the effects of obesity on balance recovery from small sub-maximal postural perturbations using an ankle strategy. Two types of perturbations were applied including force perturbations from a ballistic pendulum, and position perturbations by releasing subjects from a static forward lean. Contrary to expectations, there were no differences in center of mass (COM) kinematics between obese and non-obese individuals following release from a submaximal forward lean or perturbations of similar magnitude (normalized to body weight). Berrigan et al. (2006) examined the effect of a goal-oriented movements on postural stability in obese and non-obese subjects. Similar to studies that investigated the effects of obesity on balance during quiet standing, obese subjects exhibited greater COP speed and

displacement than their non-obese counterparts, suggesting poorer balance. In addition, the obese subjects moved their whole body forward while performing the task. This also suggests an increased risk of imbalance for obese subjects because leaning forward would move the COM closer to the boundary of support imposed by the feet.

In addition to these studies, fundamental mechanics suggests a potentially ambiguous relationship between obesity and balance recovery. To illustrate this, consider an inverted pendulum model of the human body (Figure 4.1). This is a reasonable model of the body during quiet standing and recovery from small postural perturbations because movements are typically limited to the ankle (i.e. the so-called ankle strategy). First, obese individuals have increased body weight and typically a more anterior COM than normal-weight individuals (Corbeil et al. 2001). These characteristics lead to an increased gravitational moment about the ankles, and therefore an increased angular acceleration about the ankles. This would require larger ankle plantar flexor torque to recover from anteriorly-directed postural perturbations, making balance recovery more physically demanding (Corbeil et al. 2001). Second, obese individuals also have an increased mass moment of inertia about the ankles. Inertia can be defined as the resistance to rate of change in velocity (Meriam and Kraige 2002). Therefore, the increased inertia in obese individuals may be beneficial in resisting an increase in angular velocity, which may help to limit how quickly the body angular velocity increases as an individual falls forward. As a result, balance recovery would be less physically-demanding after selected types of perturbations. These potentially offsetting fundamental mechanical considerations make it difficult to predict the effects of obesity on balance recovery using an ankle strategy. It should also be noted that while weight and inertia are closely related, they are not necessarily directly proportional to one another because inertia is dependent upon the distribution of mass over the body, which can vary between individuals.

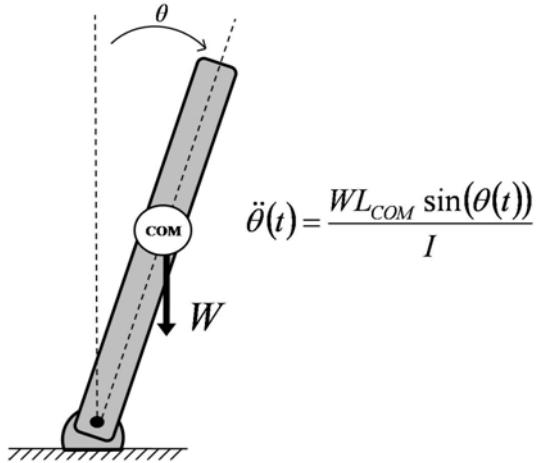


Figure 4.1. Inverted pendulum of the human body. The COM is located a distance L_{COM} from the ankle joint. When the equation of motion of the pendulum is solved for the angular acceleration ($\ddot{\theta}$), one can see if weight (W) is increased, angular acceleration increases. However, if inertia (I) is increased, angular acceleration decreases. Since both weight and inertia are increased in the obese, the overall effect on angular acceleration is unclear.

The purpose of this study was two-fold. First, we used human subjects testing to investigate the effects of obesity on balance recovery using an ankle strategy in young adults. Second, we used computer simulations to better understand how fundamental mechanics associated with obesity (increased inertia and weight) independently influence balance recovery. A better understanding of the effects of obesity on balance recovery and the fundamental mechanics involved may improve our understanding as to how obesity contributes to falls. Balance recovery was limited to the ankle strategy in order to interpret our results in context of an inverted pendulum. While this may limit the generalizability of our results to falls while performing functional activities, it will provide controlled experimental conditions that will facilitate understanding of the fundamental mechanics involved. One potential limitation of the study conducted by Miller (2008) that investigated the effects of obesity on balance recovery was that subjects were not challenged to their maximum capabilities. It is possible that if differences in balance recovery exist between obese and non-obese individuals, more challenging conditions are needed to identify these differences (Alexander et al. 1992). Also, Miller (2008) compared COM displacement and velocity at similar normalized (to body weight) perturbation levels. While this gives insight on differences in control

of the body from a perturbation, it does not address differences in balance recovery capability. As such, subjects were tested to their maximum capabilities.

4.3 Methods

Twenty adult male subjects were used in this study including ten normal-weight (mean \pm standard deviation, age: 21.3 ± 1.1 years, BMI Range: 22.0 to 23.7 kg/m²) and ten obese (age: 22.4 ± 3.6 years, BMI Range: 30.0 to 36.1 kg/m²). Normal-weight and obese subjects were matched by height, and all subjects were free of self-reported musculoskeletal impairments and injuries. This study was approved by the Virginia Tech Institutional Review Board, and written consent was obtained from all subjects prior to participation.

Balance recovery from three types of perturbations was investigated due to the potential for the effects of obesity to differ across perturbations. The three types of perturbations differed in the initial conditions imposed on the subjects. First, releasing subjects from a static forward lean (static lean trials) was used to impose an initial angular displacement from vertical with no initial angular velocity. Second, applying an abrupt anteriorly-directed impulse to the upper back with a ballistic pendulum while subjects stood comfortably (free perturbation trials) was used to impose an initial angular velocity at an unconstrained body angle. Third, applying a similar force impulse while subjects leaned backward against a rigid stop (lean perturbation trials) was used to impose an initial angular velocity at a controlled body angle. The presentation order of the three types of perturbations was randomized for each subject. Balance recovery after all three types of perturbations was limited to an ankle strategy. Ankle strategy was defined as limiting movement to plantar flexion/dorsiflexion at the ankle while keeping the body straight (not stepping). Subjects were harnessed to a backboard to enforce these constraints (Figures 4.2a and 4.2b). The heels were allowed to rise from the ground, and only slight heel raise was used during most trials. A safety rope was attached to the backboard to prevent a fall to the ground in the event of an unsuccessful recovery. A real-time display of ankle torque was provided to the subjects prior to all trials using a force

platform positioned under their feet. This real-time display was used to standardize the initial ankle torque, which was calculated using an inverse dynamics analysis. To calculate ankle torque, the ankle was lined up with a tape line at a prescribed location on the force platform. This position was monitored throughout all trials and was used to determine the COP relative to the ankle for the real-time display and subsequent analysis of ankle torque. Subjects were instructed to maintain their initial ankle torque at the pre-determined threshold ($0.35*M*H$; M = body mass, kg; H = height, m). This was done to eliminate confounding influences of initial ankle torque on balance recovery limits (Robinovitch et al. 2002). The threshold was selected based on pilot data, and was chosen because it was an intermediate value that most subjects were able to maintain easily prior to perturbations. Verbal feedback was given to contract or relax ankle muscles if their initial ankle torque fell below or rose above the pre-determined threshold, respectively. During all trials, body motion was quantified by the body angle relative to vertical (θ), and was measured from vertical to a line connecting the lateral malleolus and greater trochanter (Owings et al. 2000; Grabiner et al. 2005).

Static lean trials involved releasing subjects from a static forward lean without warning (Figure 4.2a). The effective initial conditions for these trials included an initial angular displacement of the body from vertical, and no initial angular velocity. For the first trial, the forward lean angle was set to 2° forward of the subjects' natural body angle during quiet standing. Subjects were held at this angle by a tether at waist level. After a random delay of 1-20 seconds, the tether was released without warning and subjects attempted to return to an upright standing position using an ankle strategy. After each successful recovery, the initial lean angle was increased by 0.5° and the test repeated. After each failed recovery (i.e. being "caught" by the safety rope), another attempt was performed at the same angle. This process was repeated until subjects failed three times at a given initial lean angle. After three failures, the lean angle was decreased by 1° and the process repeated. The maximum initial lean angle from which balance was successfully recovered (θ_{max}) was used to quantify balance recovery capability for this type of perturbation.

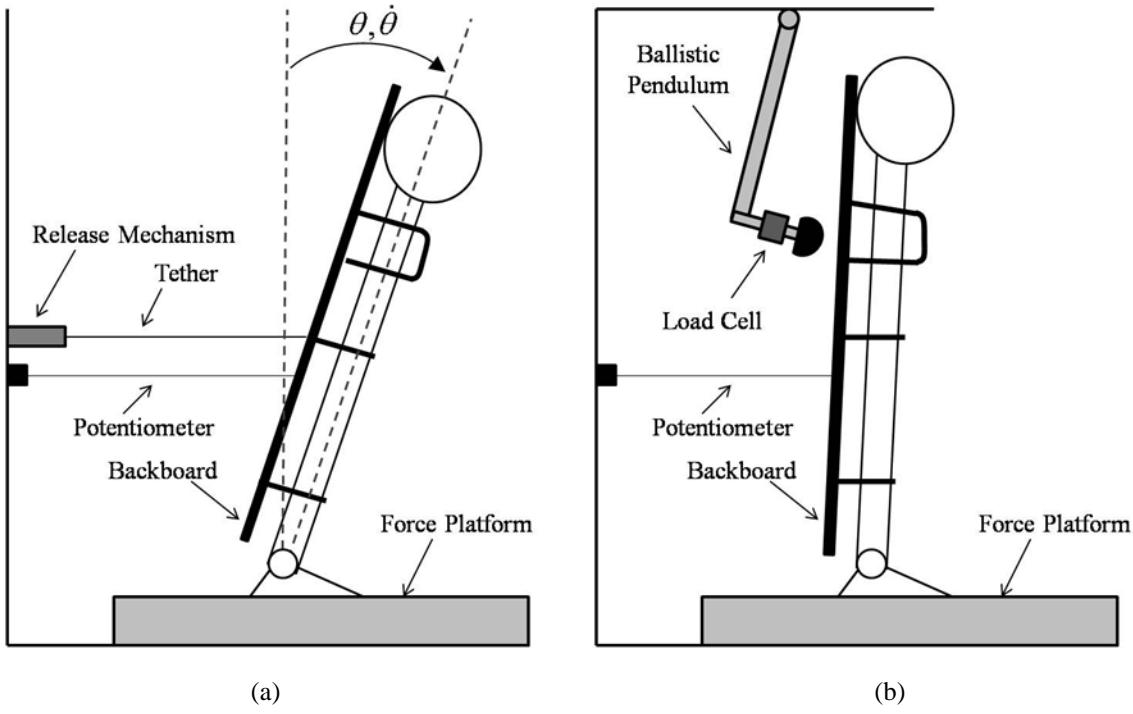


Figure 4.2. Experimental setup for the three types of perturbations investigated. The body angle (θ) was measured from vertical to a line connecting the lateral malleolus and greater trochanter by a linear potentiometer. A computer screen provided a real time display of initial ankle torque. In the lean trials (a), the tether, attached to the backboard at waist level, was released without warning after a random time delay. In the free and lean perturbation trials (b), a force impulse was applied to the backboard/body without warning using a padded ballistic pendulum that resulted in an abrupt increase in angular velocity.

Free perturbation trials involved applying abrupt anteriorly-directed force perturbations using a padded ballistic pendulum while the subjects stood naturally (Figure 4.2b). The effective initial conditions for these perturbations included an initial angular velocity due to the applied force and the impulse-momentum relation, yet minimal initial angular displacement from the free-standing body angle (the relatively short 120 ms time duration of the force impulse did not allow significant movement of the body prior to the end of the perturbation). Perturbations were applied by pulling the pendulum away from the subjects in the mid-sagittal plane and then releasing it without warning. Each trial consisted of three impulses at a specified pull-back angle of the pendulum, and results were averaged across these impulses. The pull-back angle was systematically increased to provide impulse increments of ~2 N·s until subjects failed to recover all three perturbations within the trial (i.e. being “caught” by the safety rope). After

three failures, the process was repeated after decreasing the impulse by ~ 4 N·s. The maximum angular velocity ($\dot{\theta}_{\max}$) at the body angle at the end of the force perturbation (θ_0) was used to quantify balance recovery capability for this type of perturbation. Through post-hoc analysis, the range of initial angular velocities was determined to be from 3 to 25 deg/s.

Lean perturbation trials were similar to the free perturbation trials, with the exception that subjects leaned backward against a stop so that the initial body angle was 2° forward from vertical (body angle during free standing averaged 4.7°). The protocol for applying the perturbations was identical to free perturbation trials, and the same measures were used to quantify balance recovery capability for this type of perturbation. Through post-hoc analysis, the range of initial angular velocities was determined to be from 5 to 30 deg/s.

During all trials, body angle, ground reaction force, voltage applied to the release mechanism (lean trials), and pendulum impact force (perturbation trials) were sampled at 1000 Hz. Body angle was measured using a linear potentiometer (Unimeasure, Corvallis, OR). Ground reaction force was sampled using a force platform (Bertec Corporation, Type 9090-15, Columbus, OH). Voltage applied to a solenoid that released subjects from the forward lean during lean trials was used to determine the time of release. Pendulum impact force was measured using an in-line load cell (Cooper Instruments and Systems, Warrenton, VA) attached to the end of the pendulum and oriented along the direction of the applied force. Body angle, pendulum impact force, and voltage applied to the release mechanism were low-pass filtered at 20 Hz, and ground reaction force data at 7 Hz (eighth order zero-phase-shift Butterworth filter for all filtering).

In addition to the human subjects testing, forward dynamic computer simulations were used to understand how increased inertia and weight in obese individuals independently affect balance recovery. An inverted

pendulum model similar to those used in previous studies (Robinovitch et al. 2002; Matrangola and Madigan 2009) was used and included a slender rod rotating about a hinge joint to represent the human body rotating about the ankle joints in the mid-sagittal plane with a torque actuator at the ankle (Figure 4.3a). Body segment inertial parameters were calculated for each group based on population-specific estimates of these parameters (Chandler et al. 1975; Matrangola et al. 2008). All model simulations were performed in MATLAB (The MathWorks, Natick, MA) using an ordinary differential equation solver with a variable time step (MATLAB command *ODE45*).

A linear approximation of ankle torque history collected during the human subjects testing was applied to the model (Figure 4.3b). Initial ankle torque (T_i), response time (Δt), maximum ankle torque (T_{max}), and ankle torque generation rate (C) were calculated using a similar approach as Robinovitch et al.(2002). Ankle torque decline rate (D) was defined as the slope of a straight line from T_{max} to 500ms later, and the response time was determined to be the time from release until ankle torque exceeded T_i by 2.5 Nm. Average ankle torque parameters for the normal-weight and obese group were determined from maximum trials (i.e. the trials in which the maximum lean angle or maximum angular velocity were achieved for a given type of perturbation), and were used to predict balance recovery limits for each group. For each subject, a single set of ankle torque parameters was determined by averaging T_i , D , and Δt across the maximum trials of the three different perturbation tasks and selecting the maximum T_{max} and C across the maximum trials of the three perturbation tasks. In addition, T_i , T_{max} , C, and D were normalized to the product of body mass (kg) and height (m).

A qualitative validation of the model was performed by determining the balance recovery limits for an average normal-weight and average obese subject (model inputs were set to mean values from human subjects testing). The average balance recovery limits for each group were determined in a similar fashion as our human subjects testing. First, the model was repeatedly released from progressively increasing angles until the predicted θ_{max} was found. The maximum angular velocity was then found for

each lean angle over the range of 0° to θ_{max} , incrementing by 0.01° . Both here and in all subsequent simulations, a successful recovery was defined by the occurrence of $\dot{\theta} < 0$ while $\theta < 90^\circ$ (Robinovitch et al. 2002). These limits were then compared with all successful and failed recoveries from human subjects testing. In addition, these limits were compared with the average θ_{max} and $\dot{\theta}_{max}$ values measured from each group.

Inertia and weight were independently manipulated within the model to better understand their independent effects on balance recovery limits. For each subject, body mass, height, and ankle torque parameters were entered into the model to determine θ_{max} and $\dot{\theta}_{max}$ in a similar fashion as mentioned previously. Inertia was varied in the equation of motion (Figure 4.3a) from a 50% decrease in inertia to a 50% increase in inertia in increments of 25%. Similarly, weight was varied in the equation of motion from a 50% decrease to a 50% increase in increments of 25%. Resulting changes in θ_{max} and $\dot{\theta}_{max}$ were used to quantify the independent effects of inertia and weight on balance recovery.

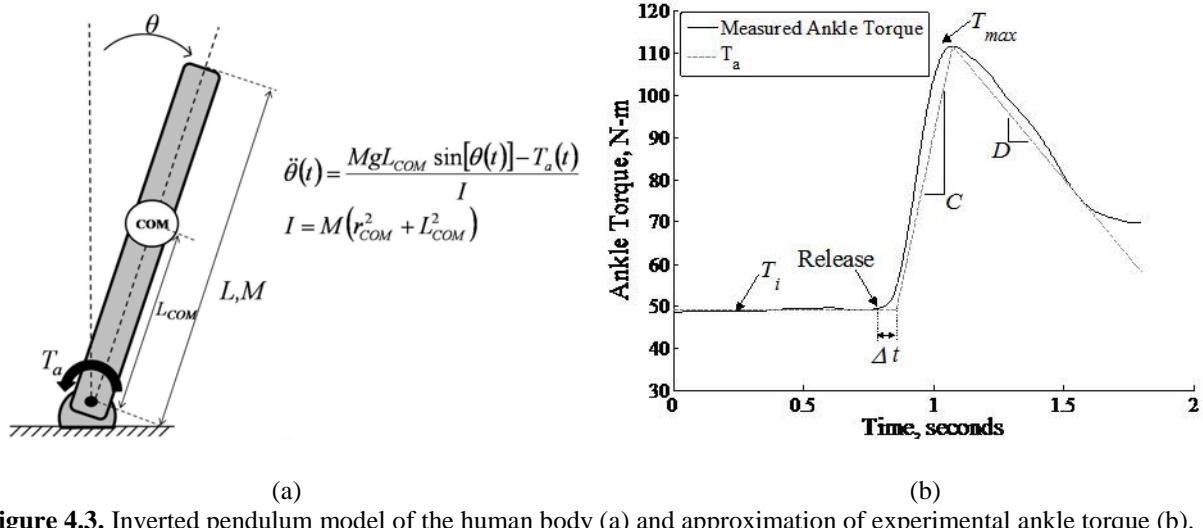


Figure 4.3. Inverted pendulum model of the human body (a) and approximation of experimental ankle torque (b). The variables M , L , and I are the mass, height, and moment of inertia about the ankle of the subject, respectively, g is the gravitational constant (9.81 m/s^2), and T_a is the torque generated about the ankle. L_{COM} is the location of the COM, and was assumed to be 58.6% and 61.5% of the subject's height for normal-weight and obese subjects, respectively. r_{COM} is the radius of gyration about the COM, and was assumed to be 24.7% and 18.9% of the subject's height for normal-weight and obese subjects, respectively (Chandler et al. 1975; Matrangola et al. 2008). T_a was linearly approximated from the experimental ankle torque history during recovery (b).

A one-way ANOVA was used to investigate the effects of obesity on balance recovery capability from human subjects testing. The dependent variables were maximum lean angle from static lean trials, and maximum angular velocity from free and lean perturbation trials. Body angle at the end of the perturbation was included as a covariate. A *t*-test was used to investigate the effect of obesity on body angular velocity at subject response time. A one-way repeated measures ANOVA was used to investigate differences between normal-weight and obese subjects in ankle torque parameters (T_i , T_{max} , C , D , Δt) across maximum trials (i.e. trials during which subjects successfully recovered from the largest achieved lean and angular velocity) of all three types of perturbations, as well as these variables normalized to body mass and height. All data distributions were visually inspected prior to the ANOVAs, and none significantly deviated from a normal distribution. One obese subject was excluded from the analysis of lean perturbation trials because this subject successfully recovered from all perturbations (further increases in perturbation were deemed too uncomfortable by the investigators), and therefore their balance recovery limits could not be determined. Also, another obese subject was excluded from the analysis of free perturbation trials due to an equipment malfunction. All ANOVAs were conducted using JMP 7 (Cary, North Carolina, USA) with a significance level of $p \leq 0.05$.

A three-way mixed factor ANOVA was used to investigate the effects of inertia and weight on balance recovery. The independent variables were group (normal-weight or obese), term (inertia or weight), and change in term (-50%, -25%, 25%, 50%). The dependent variables were percent change in θ_{max} and $\dot{\theta}_{max}$. Pairwise comparisons were evaluated using Tukey's Honestly Significant Difference (HSD).

4.4 Results and Discussion

Obesity impaired balance recovery, but only after certain types of perturbations (Table 4.1). For the static lean trials, θ_{max} was not significantly different between normal-weight and obese subjects ($p=0.497$). In

contrast, $\dot{\theta}_{\max}$ was 29.9% ($p=0.008$) and 20.1% ($p=0.035$) lower in obese subjects for free and lean perturbations, respectively, compared to normal-weight subjects.

Interestingly, during static lean trials the body angular velocity after subject response time (release time + Δt) was 26.1% lower in obese subjects compared to normal-weight subjects. Because initial torques were constant relative to body weight during all trials, this result indicates that the increased inertia in young obese subjects did tend to moderate the angular acceleration of the body and allowed young obese subjects to recover their balance from lower angular velocities after release from identical static lean angles as young normal-weight subjects. In addition, the change in body angular velocity from perturbation onset to subject response time from free and lean perturbations were similar between normal-weight and obese subjects ($p=0.372$ and $p=0.493$, respectively). The obese participants exhibited similar changes in angular velocity, but from smaller magnitude perturbations. This indicates that increased inertia moderated angular acceleration and made it difficult to decrease and reverse their angular velocity.

Table 4.1. Summary of balance recovery measures

Perturbation Type	Parameter	Normal-Weight	Obese	
Static Lean	θ_{\max}	7.57 ± 1.14	7.18 ± 1.36	
	$\dot{\theta}_{\Delta t}$	4.44 ± 0.61	3.28 ± 0.87	*
Free Perturbations	$\dot{\theta}_{\max}$	16.53 ± 1.51	11.93 ± 2.72	*
Lean Perturbations	$\dot{\theta}_{\max}$	21.06 ± 2.44	17.54 ± 3.92	*

Mean \pm S.D.

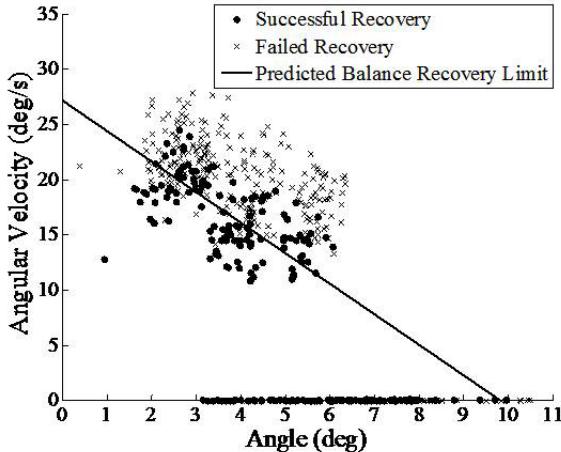
* indicates a significant difference between normal-weight and obese subjects ($p<0.05$)

Ankle strength (i.e. maximum isometric ankle torque) and torque development rate are critical for balance recovery with an ankle strategy after a postural perturbation (Whipple et al. 1987; Simoneau and Corbeil 2005; Bento et al. 2010). For example, Robinovitch et al. (2002) investigated the effects of ankle plantar flexor torque and torque development rate on balance recovery using an ankle strategy. They found that

the ability to recover balance after release from a static forward lean is dependent on the capability to quickly generate and maintain high magnitudes of ankle torque. Several studies have found that obese adults exhibit increased strength and power producing capability compared to normal-weight adults, but that strength and power are significantly lower in obese adults when normalized to body weight (Hulens et al. 2001; Lafortuna et al. 2005; Maffiuletti et al. 2007). This would suggest that when obese individuals are perturbed, smaller torques relative to their body weight may make it more difficult to maintain balance. Our results indicated that a subset of ankle torque parameters were significantly higher in young obese subjects compared to normal-weight subjects (Figures 4.4 and 4.5). In particular, absolute T_i was 33.7% higher ($p<0.001$), absolute T_{max} was 30.6% higher ($p<0.001$), and absolute D was 79.4% higher ($p=0.016$) in obese subjects compared to normal-weight subjects. However, no effects of obesity were found for the remaining ankle torque parameters (C : $p=0.853$, Δt : $p=0.073$, normalized T_i : $p=0.158$, normalized T_{max} : $p=0.159$, normalized C : $p=0.057$, normalized D : $p=0.273$). These results are consistent with the literature in that maximum absolute ankle torque was greater in obese subjects and tended to be lower in obese subjects when normalized to body weight. However, there were no differences in normalized maximum ankle torque or torque generation rate between young normal-weight and obese subjects.

The average ankle torque parameters for the normal-weight (Figure 4.4) and obese (Figure 4.5) subjects were used to predict balance recovery limits for each group with computer simulations. Balance recovery limits for both groups showed a tradeoff between body angle and angular velocity in that as body angle increased, the maximum angular velocity from which subjects could recover their balance decreased linearly. This in agreement with results from previous models (Pai and Patton 1997; Simoneau and Corbeil 2005). Balance recovery limits predicted by the computer simulations provided reasonable agreement with experimental data. A comparison of balance recovery limits between young obese and normal-weight subjects (Figure 4.6) showed similar slopes but overall lower limits in young obese individuals that was similar to trends from the experimental data. The simulations typically under-

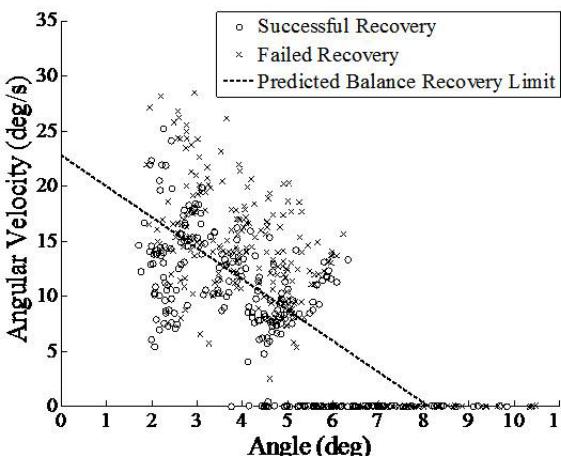
predicted the recovery limits, and this was attributed to 1) the absence of heel raising in the computer simulations, and 2) the fact that our linear approximation of ankle torque history tended to underpredict the ankle torque impulse over the entire trial (Figure 4.3b). Despite these under predictions, we think that the model provided reasonable agreement with the experimental data.



Parameter	Normal-Weight
T_i (Nm) [#]	$56.10 \pm 3.72^*$ (0.42 ± 0.05)
Δt (ms)	121 ± 18
C (Nm/s) ^{#†}	$1156.28 \pm$ 429.05 (8.76 ± 3.29)
D (Nm/s) [#]	$39.32 \pm 20.32^*$ (0.30 ± 0.16)
T_{max} (Nm) ^{#†}	$187.27 \pm 18.91^*$ (1.41 ± 0.14)

Mean \pm S.D., † Maximum values
* Values given in parentheses are normalized = parameter value / {body mass (kg) \times height (m)}

Figure 4.4. Summary of results for normal weight subjects including all successful (black circles) and failed (x's) trials, and balance recovery limits predicted from the simulations (solid line). The balance recovery limits predicted from the simulations indicate that any combination of angle and angular velocity below this line would result in a successful recovery, while any combination above this line would result in a failed recovery. This line was determined using the average torque parameters for the normal-weight group shown in this figure.
* indicates a significant difference between normal weight and obese subjects ($p < 0.05$)



Parameter	Obese
T_i (Nm) [#]	$75.03 \pm 7.88^*$ (0.40 ± 0.02)
Δt (ms)	143 ± 32
C (Nm/s) ^{#†}	$1189.70 \pm$ 365.17 (6.33 ± 1.87)
D (Nm/s) [#]	$70.54 \pm 30.96^*$ (0.38 ± 0.17)
T_{max} (Nm) ^{#†}	$244.60 \pm 32.33^*$ (1.31 ± 0.16)

Mean \pm S.D., † Maximum values
* Values given in parentheses are normalized = parameter value / {body mass (kg) \times height (m)}

Figure 4.5. Summary of results for obese subjects including all successful (white circles) and failed (x's) trials, and balance recovery limits predicted from the simulations (dashed line). Balance recovery limits were determined using average torque parameters for the obese group shown in this figure. * indicates a significant difference between normal weight and obese subjects ($p < 0.05$)

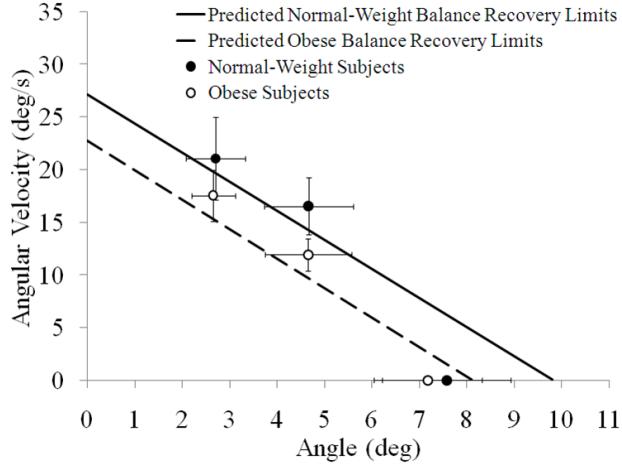


Figure 4.6. Comparison of mean experimental results and balance recovery limits predicted from the simulations. Experimental values at approximately 2.5 degrees are from the lean perturbation trials, values at approximately 4.5 degrees are from the free perturbation trials, and values along x-axis are from the static lean trials. This figure illustrates good qualitative agreement between experimental data and the predicted balance recovery limits for lean perturbation trials and free perturbation trials, but not the static lean trials. It also illustrates the perturbation-dependent effects of obesity on balance recovery.

Inertia and weight had different effects on balance recovery limits when determining the balance recovery limits a single obese subject. As inertia was increased (Figure 4.7a), the slope and y-intercept of the predicted balance recovery limit changed such that $\dot{\theta}_{max}$ decreased and θ_{max} increased. These results suggest increased inertia impairs balance recovery using an ankle strategy for initial conditions involving an angular velocity, but improves balance recovery following perturbations involving limited or no initial velocity. In contrast, as weight was increased (Figure 4.7b), the slope and y-intercept of the predicted balance recovery limit changed such that both $\dot{\theta}_{max}$ and θ_{max} decreased. These results suggest increased weight impairs balance recovery regardless of the initial conditions resulting from the perturbation. Similar effects were found when each individual subject was modeled, and inertia and weight were manipulated. Increases in inertia increased maximum lean angle and decreased maximum angular velocity (Figure 4.8a), while increases in weight decreased both maximum lean angle and maximum angular velocity (Figure 4.8b). There were no differences between normal-weight and obese models in how varying inertia and weight affected balance recovery (including all interaction terms involving group; $p>0.05$).

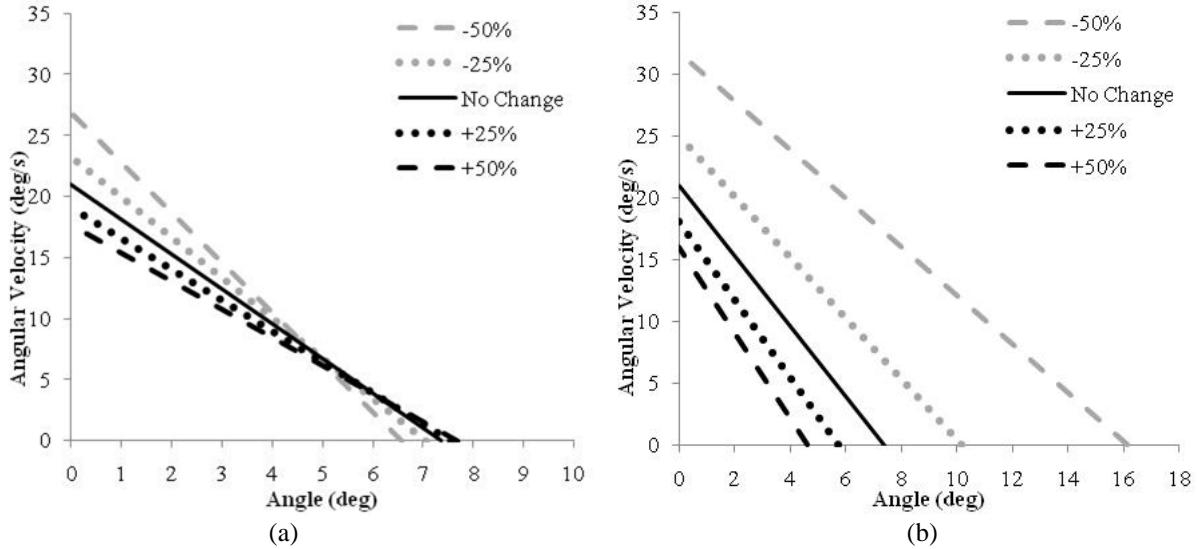


Figure 4.7. The independent effects of inertia (a) and weight (b) on balance recovery limits for a representative obese subject ($BMI = 32.2 \text{ kg/m}^2$).

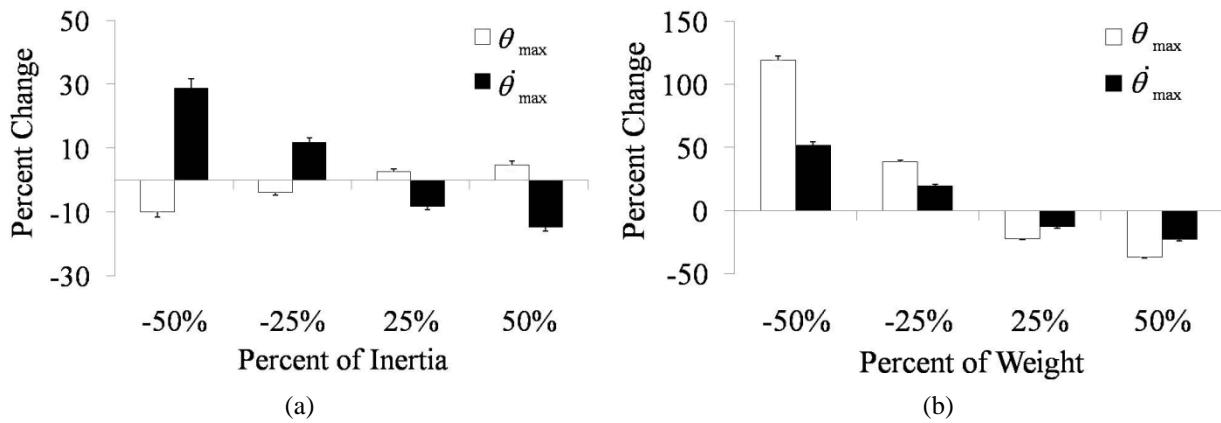


Figure 4.8. Percent change in θ_{\max} (white bars) and $\dot{\theta}_{\max}$ (black bars) for a given percent change in inertia only (a) and weight only (b) averaged across simulations for all subjects.

Results from the simulations appear to help explain why we found differing effects of obesity on balance recovery between the three types of perturbations we investigated. The two types of perturbations after which obese subjects exhibited decreased balance recovery ability compared to normal-weight subjects (free perturbation trials and lean perturbation trials) were the same two types of perturbations in which our simulations revealed that increased inertia and weight both negatively affected balance recovery limits. However, the type of perturbation after which no differences between normal-weight and obese subjects were found (static forward lean) was the same type of perturbation that our simulations revealed

that increased inertia provided a benefit to balance recovery. The results suggest that obesity impaired balance recovery following perturbations that imposed an initial angular velocity, but did not affect balance recovery following perturbations that imposed an initial angular displacement with little or no initial angular velocity due to beneficial effects of increased inertia after these types of perturbations.

Several limitations of this study warrant discussion. First, balance recovery was constrained to an ankle strategy. While this approach likely limits the generalizability of our results to balance recoveries involving stepping, generalization was not our goal. Our goal was to investigate the effects of obesity on a constrained balance recovery task to facilitate a better understanding of the fundamental mechanics variables involved. Follow up studies are needed to determine if the results found here apply to balance recovery movements involving stepping. Second, our balance recovery model assumed the COM to be located along the center line of the body. Obese individuals have a preferential accumulation of abdominal visceral fat resulting in an anterior displacement of the COM with respect to the ankle (Corbeil et al. 2001). Due to the lack of specific guidance on anterior-posterior displacement of the COM for individuals who are obese in the literature, the current study assumed the COM to be along the center line of the body. However, we feel that our results pertaining to the independent effects of inertia and weight were largely insensitive to COM location. Third, the inertia of the backboard was not included in the model. This would result in only slight systematic differences because the inertia of the backboard is constant. Therefore, the independent and relative effects of weight and inertia would not be affected by inclusion of inertia from the backboard. Fourth, trials within each perturbation type were presented in order of increasing difficulty instead of being randomized. Therefore, it is possible a learning effect could have occurred or perturbation amplitude could have been anticipated. However, this would only have affected our results if normal weight and obese subjects learned/anticipated differently, and we are aware of no evidence to support such a difference. Fifth, initial ankle torque was controlled prior the perturbation, not muscle activity. It is possible the subjects co-contracted ankle muscles. This would result in the required initial ankle torque, but could have aided in balance recovery. Finally, similar to all

cross-sectional studies, differences between the normal weight and obese groups other than those investigated here could have contributed to our results.

4.5 Conclusions

In conclusion, young obese subjects were unable to recover balance using an ankle strategy as well as young normal weight subjects when perturbations involved an initial angular velocity. However, no differences between young obese and normal weight subjects were found when perturbations only involved an initial angular displacement. The differing results between perturbation types may be explained by a possible beneficial effect of increased inertia on balance recovery after perturbations with little or no initial angular velocity. It can be hypothesized that increased inertia will resist changes in body angular velocity as subjects fall forward following release from a static lean, and therefore their body angular velocity after subject response time will be lower than if inertia was not increased. These findings improve our understanding of how increased body weight and inertia associated with obesity affect balance recovery in young adults. Additional research balance recoveries involving stepping, such as those typically seen after slipping and tripping, are warranted to further understand the source of the increased risk of falls among obese individuals.

4.6 Acknowledgements

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Chapter 5 – The effects of obesity on single-step balance recovery from a forward fall in young adults

5.1 Abstract

Obesity has been associated with an increased risk of falls. The purpose of this study was to investigate the effects of obesity on balance recovery by stepping in young adults. Ten normal weight (age: 21.4 ± 1.5 years, BMI: $22.0 \pm 1.6 \text{ kg/m}^2$) and ten obese (age: 21.5 ± 2.0 years, BMI: $32.9 \pm 2.2 \text{ kg/m}^2$) young male adults participated in this study. Participants attempted to recover balance by taking a single step following two types of postural perturbations: an initial angular displacement from an upright stance (by releasing participants from a static forward lean), and an initial angular velocity while in an upright stance (using a translating platform). Balance recovery ability was quantified by the largest initial angular displacement or angular velocity from which balance could successfully be recovered. The ability to recover balance with a single-step did not differ between normal-weight and obese young adults for either type of perturbation, suggesting that increased fall risk in the obese is not a result of impaired balance recovery ability. Future research is needed investigating the influence of obesity and aging as well as obesity and comorbidities (i.e., diabetes, osteoarthritis, etc) on balance recovery ability.

5.2 Introduction

Obesity is a major health concern in the United States (US). More than one-third of adults, or over 72 million people, are obese (Ogden et al. 2007). Not only is the prevalence of obesity high, but it continues to increase. For example, from 1980 to 2000, the prevalence of obesity among adults more than doubled from 15% to 31% (Flegal et al. 2002; National Center for Health Statistics 2008). One of the many concerns with the high and increasing prevalence of obesity is its association with an increased risk of falls and subsequent injury. Obese individuals fall almost twice as often (27% vs. 15%) compared to non-obese individuals (Fjeldstad et al. 2008), and falls have been identified as the most common (~36%) cause of hospitalized injuries among obese individuals (Matter et al. 2007).

Consistent with this epidemiological evidence, several biomechanical studies have reported that increased body weight is associated with increased postural sway during quiet standing (McGraw et al. 2000; Chiari et al. 2002; Hue et al. 2007; Menegoni et al. 2009). For example, Menegoni et al. (2009) found mean center of pressure (COP) distance and speed to be 37% greater in obese adults compared to their normal-weight counterparts. Similarly, Hue et al. (2007) found that a 1 kg increase in weight corresponded to a 1% increases in mean COP speed. Increased postural sway is associated with an increased risk of falling (Fernie et al. 1982; Maki et al. 1994), so these studies provide additional support for an increased risk of falling among individuals who are obese.

While these studies on quiet standing support an increased risk of falling in obese individuals, most falls do not occur during quiet standing. Instead, most falls occur as a result of some type of postural perturbation (Horak et al. 1997). It can thus be hypothesized that obese individuals have an impaired ability to recover their balance after a postural perturbation. However, there is very limited knowledge on the effects of obesity on balance recovery. Miller et al. (2009) investigated the effects of obesity on balance recovery from small sub-maximal postural perturbations using an ankle strategy. Two types of perturbations were applied: position perturbations by releasing participants from a static forward lean, and force perturbations from a ballistic pendulum. Contrary to expectations, there were no differences in center of mass (COM) kinematics between obese and non-obese individuals following perturbations. Matrangola and Madigan (2011) investigated the effects of obesity on balance recovery ability using an ankle strategy in young adults. Normal-weight (age: 21.3 ± 1.1 years) and obese (age: 22.4 ± 3.6 years) adults attempted to recover balance from two types of perturbations: release from a static forward lean (which provided an initial angular displacement from an upright standing position and no initial angular velocity), and a brief anteriorly-directed force impulses applied at the back (which provided an initial angular velocity with a negligible initial angular displacement from an upright standing position). Balance recovery ability was quantified by determining the largest initial angular displacement (θ_{max}) after

release from a static forward lean, and the largest initial angular velocity ($\dot{\theta}_{max}$) from which balance could be recovered after anteriorly-directed force impulses. Results showed that obesity did not affect $\dot{\theta}_{max}$ (obese = 7.18 deg, normal-weight = 7.57 deg; $p=0.497$), but did decrease $\dot{\theta}_{max}$ (obese = 12.76 deg/s, normal-weight = 16.53 deg/s; $p=0.008$). These results suggest that obesity impaired balance recovery, but only after certain types of perturbations.

Studies by Miller et al. (2009) and Matrangola and Madigan (2011) limited balance recovery to a non-stepping ankle strategy. While this has the benefit of simplifying the mechanics, it can limit the generalizability to more realistic fall scenarios where stepping to extend the base of support and recover balance is desired. Moreover, it is possible that obesity could have greater effects after larger postural perturbations that require stepping to maintain balance due to larger inertial forces associated with larger body segments. Therefore, the purpose of this study was to investigate the effects of obesity on balance recovery by stepping in young adults. It was hypothesized that, similar to balance recovery ability with an ankle strategy in young adults (Matrangola and Madigan 2011), obesity would negatively affect the ability to recover balance from a postural perturbation involving an initial velocity, and that these differences would be more exaggerated compared to those found with an ankle strategy.

5.3 Methods

Twenty adult male participants were recruited for this study including ten normal-weight (mean \pm standard deviation, age: 21.4 ± 1.5 years, height: 1.76 ± 0.06 m, mass: 65.9 ± 5.4 kg, BMI Range: 19.4 to 22.9 kg/m^2) and ten obese (age: 21.5 ± 2.0 years, height: 1.75 ± 0.04 m, mass: 102.3 ± 9.7 kg, BMI Range: 30.1 to 36.7 kg/m^2). Normal-weight and obese participants were matched by height, and all participants were required to be free of self-reported musculoskeletal impairments and injuries. This study was approved by the Virginia Tech Institutional Review Board, and written consent was obtained from all participants prior to participation.

Balance recovery from two types of perturbations were studied based upon previous work suggesting that the effects of obesity on balance recovery was dependent upon the characteristics of the perturbation (Matrangola and Madigan 2011). The first type of perturbation imposed an initial angular displacement on the body from an upright standing position with no initial angular velocity. This was accomplished by releasing participants from a static forward lean (i.e. forward lean trials) and having participants attempt to recover their balance with a single step. The second type of perturbation imposed an initial angular velocity on the body with no initial angular displacement from an upright standing position. This was accomplished by having participants stand on a custom-built TRanslatIng Platform (TRIP), and pushing it forward at a constant speed (i.e. TRIP trials). The TRIP then collided with the end of a walkway. Through conservation of momentum (Meriam and Kraige 2002), this imposed an angular velocity on the participant about an axis through the ankles, and participants attempted to recover their balance with a single step. Balance recovery ability was defined by the maximum lean angle and maximum angular velocity from which the participant could successfully recover their balance from forward lean and TRIP perturbations, respectively.

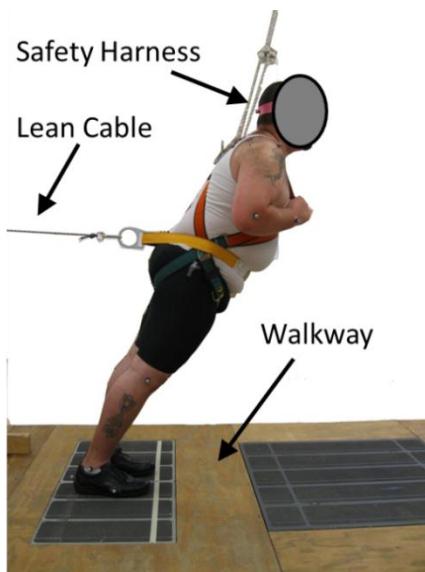


Figure 5.1. Photograph of lean trial prior to release of participant. Participants were held in a static forward lean by a lean cable and wore a safety harness in the event of a failed balance recovery. Participants began with arms folded across their chest, and stepped with their right foot after release.

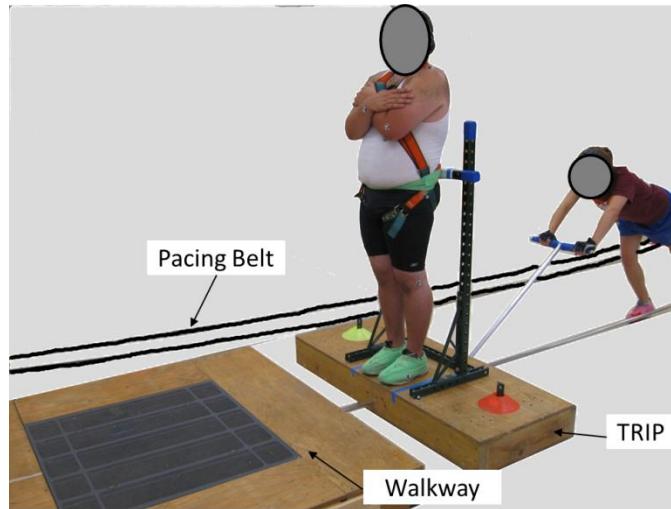
The protocol for the forward lean trials was adapted from Madigan and Lloyd (2005) and Thelen et al. (1997). Forward lean trials began with participants standing with their feet approximately 3 inches apart, arms folded across their chest, straight posture, and looking straight ahead. Participants were then placed into a forward lean position using a lean cable connecting a rigid support to the back of a belt (Figure 5.1). During testing, the forward lean was quantified by measuring force in the lean cable, as a percentage of participant weight. The forward lean was manipulated by having participants maintain a constant feet position and adjusting the length of the lean cable. After a random time delay between 1 and 30 seconds, participants were released from the forward lean and attempted to recover their balance with a single step of their right foot. Violation of any of the following criteria was used to define a failed balance recovery: 1) More than one step taken with the right foot, 2) force greater than 20% of body weight applied to the harness at any point during recovery, and 3) left foot not remaining in contact with the ground. The initial lean corresponded to 15% of body weight (BW) in the lean cable. Upon successful recovery, the lean was increased by 5% BW. Upon failed recovery, another trial was performed at the same lean. After three consecutive failures at a given lean, the lean was decreased by 2.5% BW. The process was repeated with increments of 2.5% BW until participant failed three consecutive attempts at a given lean. Participants wore a safety harness attached to the ceiling to prevent a fall in the event of a failed recovery.

TRIP trials began with participants standing on the TRIP with their feet approximately 3 inches apart and their eyes closed. Similar to the forward lean trials, participants were instructed to fold their arms across their chest, and to maintain a straight, upright posture (i.e. knees fully extended, no hip extension or flexion) before each trial. After a random time delay between 1 and 30 seconds, the TRIP was accelerated to a constant velocity by a research assistant. The velocity was controlled by a treadmill and belt system, with the assistant matching speed with markers on the belt. Once the TRIP impacted the end of the walkway, the participants opened their eyes and attempted to recover their balance with a single step of their right foot (Figure 5.2). During testing, perturbation magnitude was quantified by TRIP

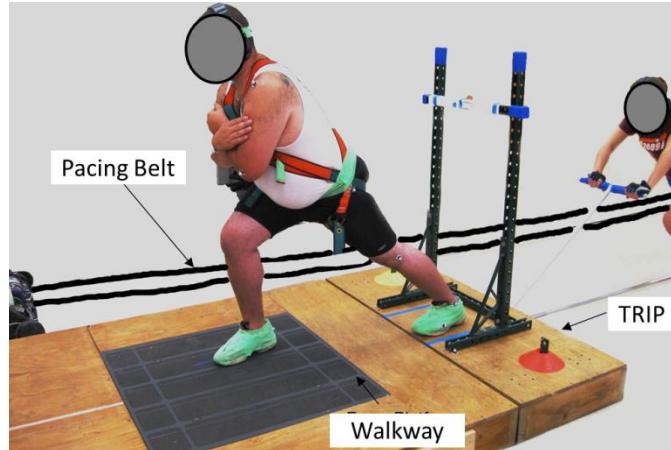
speed. Failed recovery was defined with the same criteria as the forward lean trials, and maximum angular velocity was determined in a similar manner as maximum lean angle. The initial TRIP speed was 2.0mph for all participants. Upon successful recovery, the TRIP speed was increased by 0.4mph. Upon failed recovery, another trial was performed at the same speed. After three consecutive failures at a given speed, TRIP speed was reduced by 0.2mph. This process was then repeated with increments of 0.2mph until three consecutive failures at a given speed. TRIP speed was then reduced by 0.1mph, and the process repeated with increments of 0.1mph to determine the largest speed from which participants could successfully recover balance. As in forward lean trials, participants wore a safety harness attached to the ceiling to prevent a fall in the event of a failed recovery.

During all trials, body segment position, force in the lean cable, and force in the safety harness were collected. Body segment positions were sampled at 100 Hz using a Vicon 460 motion analysis system (Vicon, Lake Forest, CA, USA) and reflective markers placed bilaterally on selected anatomical landmarks. Forces in the lean cable and safety harness were sampled at 1000 Hz using an in-line load cell (Cooper Instruments & Systems, Warrenton, VA, USA). Body segment positions and load cell data were low-passed filtered at 5 and 20 Hz, respectively (eighth order zero-phase-shift Butterworth filter used for all filtering) prior to analyses.

The whole body COM trajectory was approximated using inertial parameter estimate methods presented by Pavol et al. (2002). The body angle (θ) and body angular velocity ($\dot{\theta}$) were calculated from a line connecting the non-stepping (i.e. left) ankle to the body COM, and was measured from vertical. Balance recovery ability was defined as the maximum initial body angle for forward lean trials (θ_{max}) and maximum initial body angular velocity for TRIP trials ($\dot{\theta}_{max}$).



(a)



(b)

Figure 5.2. Photograph of TRIP trials before (a) and after (b) a successful recovery. Participants stood upright on the TRIP with eyes closed while an assistant pushed it forward at a constant speed. The assistant matched speed with a pacing belt on the side of the walkway (a). The TRIP had low-friction wheels and a guide pole to insure consistent impact location with the walkway. The TRIP impacted the end of the walkway and participants took a single step with their right leg to recover their balance (b).

Several measures of stepping characteristics and balance recovery kinematics were also investigated.

These included liftoff time (i.e. time from perturbation onset to liftoff of stepping toe), step time (i.e. time from perturbation onset to stepping toe contacted the ground), step height, step length, and time to stabilization (time from perturbation onset to reversal of COM velocity). Trunk flexion angle (measured from vertical) and trunk angular velocity were also determined at step time because reducing trunk flexion and trunk flexion velocity is crucial to recovering balance after a trip (Grabiner et al. 1993).

For each perturbation type, a *t*-test was used to determine the effects of obesity on balance recovery ability, and measures of stepping characteristics and balance recovery kinematics during maximum trials. A Wilcoxon Rank Sum test was used for variables with non-normal distributions (step height for forward lean trials; time to liftoff for TRIP trials; time to stabilization for both perturbation types). All statistical analysis was performed using JMP v7 (Cary, NC, USA) with a significance level of $p \leq 0.05$. Trends were also noted when $0.05 < p < 0.10$.

5.4 Results

For forward lean trials, no difference in θ_{max} was found between normal-weight and obese participants (Table 5.1). In addition, no differences in stepping characteristics during recovery from maximum forward lean trials were found including: time to liftoff ($p=0.155$), time from perturbation onset to foot contact ($p=0.196$), step length ($p=0.920$) and step height ($p=0.847$). There was also no difference between groups in time to stabilization ($p=0.423$). Trunk angular velocity at foot contact was 413.5% higher in obese participants compared to normal-weight participants ($p=0.020$), although no differences were found in trunk flexion angle at foot contact ($p=0.307$).

Table 5.1. Forward Lean Balance Recovery Measures and Stepping Characteristics

	<i>Normal-weight</i>	<i>Obese</i>	<i>p-value</i>
<i>Maximum initial lean angle (θ_{max}, deg)</i>	33.1 ± 2.1	31.5 ± 2.8	0.176
<i>Time to liftoff (ms)</i>	169 ± 29	192 ± 38	0.155
<i>Perturbation onset to foot contact (ms)</i>	495 ± 30	517 ± 39	0.196
<i>Step length (m)</i>	1.2 ± 0.1	1.2 ± 0.1	0.920
<i>Step height (mm)</i>	179.8 ± 24.5	178.4 ± 26.0	0.847
<i>Time to stabilization (ms)</i>	912 ± 134	1015 ± 236	0.423
<i>Trunk flexion angle at foot contact (deg)</i>	55.0 ± 7.0	50.6 ± 9.7	0.307
<i>Trunk angular velocity at foot contact (deg/s)</i>	-11.9 ± 32.2	37.4 ± 43.9	0.020*

* indicates $p < 0.05$

For TRIP trials, no difference in $\dot{\theta}_{max}$ was found between normal-weight and obese participants (Table 5.2). However, the TRIP speed at which $\dot{\theta}_{max}$ occurred was 13.3% lower in obese participants ($p=0.044$). Additionally, time to stabilization was 27.9% longer in obese participants ($p=0.037$). Step height was 11.7% lower in obese participants, but this did not reach statistical significance ($p=0.059$). No differences in trunk flexion angle ($p=0.420$) and angular velocity ($p=0.575$) at foot contact were found between obese and normal-weight participants.

Table 5.2. TRIP Balance Recovery Measures and Stepping Characteristics

	<i>Normal-weight</i>	<i>Obese</i>	<i>p-value</i>
<i>Maximum initial angular velocity ($\dot{\theta}_{max}$, deg/s)</i>	101.5 ± 14.0	98.7 ± 18.2	0.713
<i>TRIP speed (m/s)</i>	2.2 ± 0.3	1.9 ± 0.3	0.044*
<i>Time to liftoff (ms)</i>	92 ± 20	88 ± 35	0.999
<i>Perturbation onset to foot contact (ms)</i>	443 ± 32	431 ± 24	0.375
<i>Step length (m)</i>	1.3 ± 0.2	1.2 ± 0.2	0.456
<i>Step height (mm)</i>	242.1 ± 34.0	213.8 ± 25.0	0.059
<i>Time to stabilization (ms)</i>	1046 ± 227	1338 ± 475	0.037*
<i>Trunk flexion angle at foot contact (deg)</i>	29.4 ± 9.5	25.9 ± 8.8	0.420
<i>Trunk angular velocity at foot contact (deg/s)</i>	114.2 ± 32.7	121.3 ± 20.3	0.575

* indicates $p<0.05$

5.5 Discussion

The purpose of this study was to investigate the effects of obesity on single step balance recovery in young adults. There was no effect of obesity on the maximum static forward lean or the maximum forward angular velocity from which balance could be recovered with a single step. This implies that obesity does not affect balance recovery ability in young adults. Obese participants did exhibit increased trunk angular velocity at foot contact following release from a static forward lean, and increased time to stabilization following a TRIP perturbation. Typically, these differences would indicate deficiencies in balance recovery, but because no differences in θ_{max} and $\dot{\theta}_{max}$ were found between groups, our results suggest that these differences did not ultimately influence balance recovery ability.

No differences were found in balance recovery ability after release from a static forward lean. However, trunk angular velocity at foot contact was higher among obese participants. These results suggest that normal-weight participants may be able to reduce their body angular velocity more effectively than obese participants during the stepping phase of balance recovery (Pijnappels et al. 2005; Pijnappels et al. 2005b). This is notable because reducing trunk flexion and trunk flexion velocity is crucial to recovering balance after a trip (Grabiner et al. 1993). These results also suggest that although obese participants have a higher trunk angular velocity after completing a step, they were able to sufficiently arrest this motion to successfully recover balance. This may be due to the increased absolute lower extremity strength (Lafortuna et al. 2005; Maffiuletti et al. 2007) typically seen in obese individuals.

Our results did not support our hypothesis that obesity would negatively affect balance recovery ability in young adults when perturbations involved an initial body angular velocity. No differences in maximum initial body angular velocities were found between groups. Young obese individuals did exhibit an increased time to stabilization compared to young normal-weight individuals during TRIP trials. Longer time to stabilize the center of pressure (COP) or COM trajectory is associated with impaired balance (Brauer et al. 2001; Vearrier et al. 2005; Bieryla 2009). However, no statistically significant differences were found in trunk angle and trunk angular velocity at foot contact. With this in consideration, the young obese individuals had similar trunk movement at foot contact, but took longer to stabilize that movement. Ultimately, the increased time to stabilization among young obese participants did not lead to differences in balance recovery ability.

During TRIP trials, TRIP speed was directly controlled by the experimenters. Body angular velocity immediately after impact with the walkway was one of our dependent variables. Interestingly, the relationship between TRIP speed and body angular velocity immediately after impact differed between normal-weight and obese participants. The TRIP speed at which the maximum initial body angular

velocity was achieved was lower in obese participants. This suggests that the ratio of body angular velocity to TRIP speed is larger in young obese participants compared to normal-weight participants. This may be related to differences in body inertial parameters between the two participant groups, and will be further explored in Chapter 6.

Obesity has been associated with an increased rate of falls (Wallace et al. 2002; Chau et al. 2004; Fjeldstad et al. 2008). We found no differences in balance recovery ability between obese and normal-weight young adults. This suggests that the increased risk of falls among young obese adults is not due to an impaired ability to recover balance after a postural perturbation. It is possible that obesity may increase fall risk in young adults by increasing the likelihood of initiating a fall. For example, obese individuals walk with less hip and knee flexion (Spyropoulos et al. 1991), suggesting a smaller clearance of the foot from the ground. A smaller foot-ground clearance in obese individuals would make it more likely for the shoe to be obstructed by an elevated obstacle, indicating a higher risk of tripping (Rietdyk et al. 2005). Obesity may also increase fall risk by decreasing the ability to detect a loss of balance by impairing proprioception (i.e. the sense of position and orientation of parts of the body). Obesity is linked to increased sensory thresholds in mechanoreceptors (Miscio et al. 2005). This could make it more difficult for obese older adults to sense when postural or gait corrections are needed to maintain balance or when a loss of balance is initiated. However, our results didn't reflect this given that we found no difference in time to foot liftoff between normal-weight and obese participants, and this measure can be considered a surrogate measure of reaction time. It is also possible that obesity may impair the ability to recover balance among the obese when in combinations with other conditions commonly associated with obesity. For example, osteoarthritis and diabetes are both associated with obesity (Puska et al. 2003; Bray 2004) and an increased risk of falls (Nevitt et al. 1991; Gregg et al. 2000). In support of this, Fjeldstad et al. (2008) found a greater prevalence of comorbidities such as diabetes and arthritis among obese individuals. Wallace et al. (2002) also found that presence of comorbidities such as heart disease and respiratory disease increased risk of falls. Finally, muscle fatigue also negatively affects balance

(Davidson et al. 2004; Davidson et al. 2009) and could be another contributing factor towards increased fall risk in the obese. BMI has been correlated with fatigability, with higher BMIs being associated with lower fatigue resistance (Sartorio et al. 2003; Maffiuletti et al. 2007). In support of this, Singh et al. (2009) found postural sway to increase at a faster rate during a prolonged quiet standing task in obese compared to non-obese participants, suggesting muscle fatigue occurred and negatively affected balance more quickly in obese individuals.

Several limitations of this study warrant discussion. First, unlike most falls outside the laboratory, our participants were expecting a fall during testing. Additionally, trials within each perturbation type were presented in order of increasing difficulty instead of being randomized. The effects of expectation and experience would be seen during the participant's reaction time. However, reaction time is invariant with the initial lean angle (Wojcik et al. 1999; Hsiao-Wecksler 2008), suggesting that expectation and experience do not influence the initial motor response. Therefore, any differences in balance recovery response due to expectation or previous experience of a perturbation would be minimal. Furthermore, in comparison with literature that presented common perturbation amplitudes in a randomized order (Thelen et al. 1997) and those that presented perturbations in increasing difficulty (Madigan and Lloyd 2005), the average maximum lean angle for both normal-weight (33.1 ± 2.1 deg) and obese (31.5 ± 2.8 deg) participants are well within ranges previously reported for young males. Thelen et al. (1997) reported young males (age range 20 to 30 years) to have a maximum lean angle of 32 ± 4 deg. Madigan and Lloyd (2005) reported a similar maximum lean angle of 30 ± 4 deg. Second, similar to all cross-sectional studies, differences between the normal-weight and obese groups other than height/weight could have contributed to our results. In particular, strength measurements were not collected or compared between groups. It is possible that differences in strength exist between the normal-weight and obese participants in this study, and that this contributed to the lack of differences in balance recovery ability. Finally, the perturbations used in this study to simulate trip recovery have distinct differences from an actual trip recovery (e.g., static posture for initial angle perturbations, minimal angular displacement for initial

angular velocity perturbations, limiting recovery to a single step). This may have compromised the generalizability of our results to falls outside the laboratory.

5.6 Conclusions

In conclusion, the ability to recover balance with a single-step did not differ between normal-weight and obese young adults. These findings suggest that the increased fall risk in young obese adults reported in epidemiological studies is not a result of impaired balance recovery ability. Additional research investigating the influence of obesity and comorbidities (i.e. diabetes, osteoarthritis, muscle fatigue etc) on balance recovery ability and the effect of obesity on the likelihood of losing balance is needed in order to understand the source of the increased risk of falls among individuals who are obese.

5.7 Acknowledgements

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Chapter 6 – The effects of obesity on body kinematics immediately following a trip-like perturbation in young adults

6.1 Abstract

Obesity is reported to increase fall risk, and this may partially be due to a greater difficulty recovering balance following a trip. Therefore, the purpose of this study was to investigate the influence of obesity on body kinematics immediately after a trip-like perturbation that would relate to the difficulty of balance recovery. Ten normal weight (age: 21.4 ± 1.5 years, BMI: $22.0 \pm 1.6 \text{ kg/m}^2$) and ten obese (age: 21.5 ± 2.0 years, BMI: $32.9 \pm 2.2 \text{ kg/m}^2$) young male adults participated in this study. Participants recovered balance by taking a single step following a perturbation similar to an actual trip. Obese participants exhibited a higher body angular velocity than healthy weight participants when both experienced a trip-like perturbation at the same speed. Higher body angular velocity was associated with a higher probability of a failed balance recovery. These results suggest that, when walking at similar speeds, obese individuals may be at a greater risk of falling following a trip, and that this may be due to differences in how body mass is distributed.

6.2 Introduction

Obesity is a major health concern in the United States (US) both in terms of prevalence and severity. More than one-third of US adults, or over 72 million people, are obese, and the prevalence of obesity among adults has more than doubled from 1980 to 2000 (Flegal et al. 2002; Ogden et al. 2007; National Center for Health Statistics 2008). This is problematic because obesity is associated with numerous medical conditions including osteoarthritis, hypertension, type 2 diabetes mellitus, and cardiovascular disease (Puska et al. 2003; Bray 2004). In addition, obesity is associated with an increased risk of falls and subsequent injury. For example, fall rates are higher among obese individuals, with obese individuals falling almost twice as often (27% vs. 15%) as non-obese individuals (Fjeldstad et al. 2008). Additionally, falls are the most common (~36%) cause of injuries requiring hospitalization among obese individuals (Matter et al. 2007).

The increased risk of falls among individuals who are obese may be due, in part, to greater difficulty recovering balance without falling after a postural perturbation such as tripping or slipping. This greater difficulty could result from increased body mass and associated changes in body segment inertial parameters (BSIPs). A prior study in our laboratory induced postural perturbations similar to tripping by having participants stand upright on a platform and translating the platform forward until it impacted the end of a walkway (Chapter 5). This impact abruptly decelerated the platform to zero velocity, and participants subsequently fell forward and attempted to recover their balance by stepping. When normal-weight and obese participants were translated at the same speed, the body angular velocity immediately after impact was higher in obese participants. This would seem to make balance recovery more challenging for obese participants, and may be related to the aforementioned increased fall risk among obese individuals. The goal of this study was to more fully explore the reason for this difference in body angular velocity.

This study will focus on body kinematics when tripping. In particular, we will focus on the time interval from immediately before the swing foot impacts an obstacle to ~200ms later when this same foot is lowered to the ground without stepping over the obstacle. The body during this time interval can be reasonably modeled as a single segment (van den Bogert et al. 2002). Immediately before the swing foot impacts an obstacle, the body can be considered to be translating forward with a horizontal linear velocity (v_1) and no angular velocity (Figure 6.1a). Foot impact with the tripping obstacle can be approximated by a horizontal impulse (p) applied at the ankle (Figure 6.1b). Immediately after foot impact, the body can be considered to rotate forward about the stance ankle with angular velocity ω_2 (Figure 6.1c).

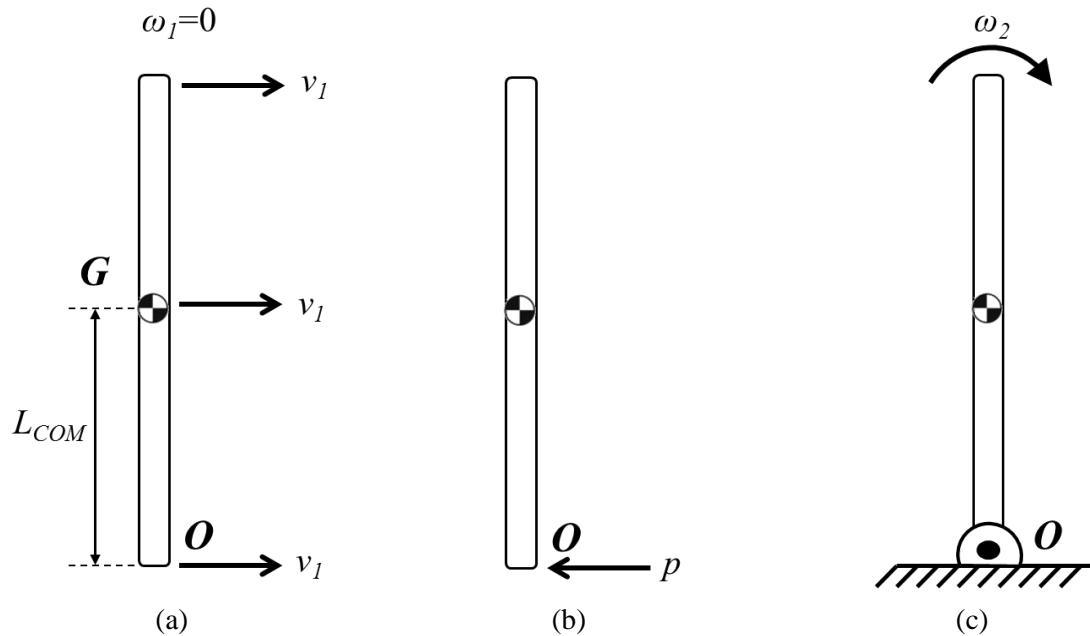


Figure 6.1. Single segment model of the body during a trip. (a) Immediately before the trip, the body translates with a horizontal linear velocity (v_I). (b) A horizontal impulse (p) is applied at O , simulating a trip. (c) After the trip, the body rotates at an angular velocity (ω_2) about a fixed pin support at the stance ankle. Subscripts of 1 and 2 denote before and after the trip, respectively.

The body angular momentum about O prior to foot impact can be expressed as:

$$(H_O)_1 = I_G \omega_1 + mv_1 L_{COM} = mv_1 L_{COM} \quad (6-1)$$

where I_G is the mass moment of inertia of the body about center of mass (COM), ω_1 is the body angular velocity prior to the trip (equal to zero), m is the body mass, v_I is the linear velocity of the body prior to the trip, and L_{COM} is COM distance from the ankle. The body angular momentum about O after foot impact can be expressed as:

$$(H_O)_2 = I_O \omega_2 \quad (6-2)$$

where I_O is the mass moment of inertia of the body about the ankle and ω_2 is the body angular velocity after the trip. We can consider angular momentum about point O to be conserved from immediately before the trip (Equation 6-1) to immediately after the trip (Equation 6-2) since the impulse is applied at O and thus does not influence angular momentum about O . Using conservation of angular momentum and setting $(H_O)_1$ equal to $(H_O)_2$, we obtain the expression:

$$mv_1 L_{COM} = I_O \omega_2 \quad (6-3)$$

which can be rearranged to:

$$\frac{\omega_2}{v_1} = \frac{mL_{COM}}{I_O} \quad (6-4)$$

Given from the parallel axis theorem:

$$I_O = m(r_{COM}^2 + L_{COM}^2) \quad (6-5)$$

where r_{COM} is the radius of gyration about the COM, Equation (6-4) can be expressed as:

$$\frac{\omega_2}{v_1} = \frac{L_{COM}}{r_{COM}^2 + L_{COM}^2} \quad (6-6)$$

This equation indicates the ratio of body angular velocity after foot impact to body velocity before foot impact is dependent on the distribution of body mass (and not body mass itself). Because obesity affects the distribution of body mass (Matrangola et al. 2008), this ratio may differ between normal-weight and obese individuals and lead to differences in body angular velocity after tripping. The specific goal of this study was to investigate the effect of obesity on this ratio.

6.3 Methods

Twenty young adult male participants were recruited for this study including ten normal-weight (mean \pm standard deviation, age: 21.4 ± 1.5 years, height: 1.76 ± 0.06 m, mass: 65.9 ± 5.4 kg, BMI Range: 19.4 to 22.9 kg/m^2) and ten height-matched obese (age: 21.5 ± 2.0 years, height: 1.75 ± 0.04 m, mass: 102.3 ± 9.7 kg, BMI Range: 30.1 to 36.7 kg/m^2). All participants were required to currently be free of self-reported musculoskeletal impairments and injuries. This study was approved by the Virginia Tech Institutional Review Board, and written consent was obtained from all participants prior to participation.

The effects of obesity on the ratio of body angular velocity after foot impact to body velocity before foot impact was determined by direct measurement of body kinematics during a trip-like perturbation. A trip-like perturbation was used, rather than an actual trip, because controlling body kinematics prior to an

actual trip can be challenging. A custom-built TRanslatIng Platform (TRIP) was used to impose a trip-like perturbation (Figure 6.2). The TRIP was a wooden platform with low-friction wheels and moved along a guide pole connected to a walkway. A back support was rigidly attached to the TRIP to prevent a backward loss of balance. Participants stood upright on the TRIP, and the TRIP was pushed forward at a constant velocity while the participants faced forward. The TRIP then collided with the end of a walkway with the same height as the TRIP, and participants fell forward similar to a forward fall after tripping.

Trials began with participants standing on the TRIP with their feet approximately 3 inches apart and their eyes closed. Participants were instructed to fold their arms across their chest, and to maintain a straight, upright posture (i.e. knees fully extended and no hip extension or flexion). The TRIP was then accelerated to a constant velocity by a research assistant. The velocity was controlled by a treadmill and belt system, with the assistant matching speed with markers on the belt. Upon impact with the walkway, participants attempted to recover their balance with a single step of the right foot and hold their recovery position after the step (Figure 6.3). Participants were allowed to open their eyes following TRIP impact. A safety harness worn by all participants was attached to a ceiling-mounted track to prevent a fall in the event of a failed recovery. Violation of any of the following criteria was used to define a failed recovery:

- 1) More than one step taken with the right foot, 2) a force greater than 20% of body weight being applied to the harness at any point during recovery, and 3) the left foot not remaining in contact with the ground during recovery.

The first trial for all participants was at an initial TRIP speed of 2.0mph. Additional trials were performed on each participant using progressively higher TRIP speeds to determine the highest TRIP speed from which each participant could recover balance (reported elsewhere).

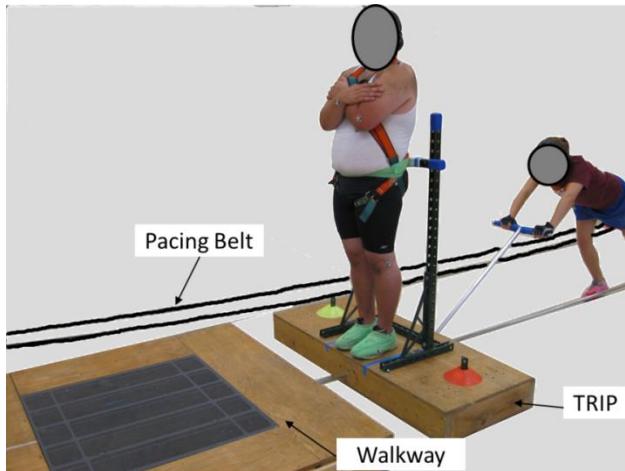


Figure 6.2. Photograph of a TRIP prior to impact with the walkway. In this photo, the TRIP was being pushed at a constant velocity by an assistant matching speed with a pacing belt on the side of the walkway.

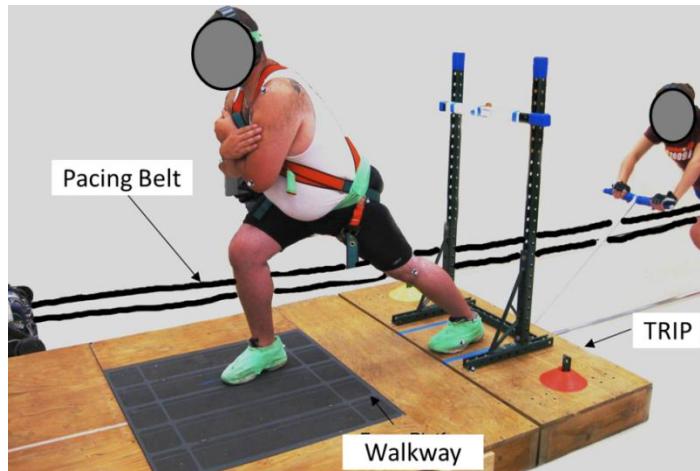


Figure 6.3. Photograph of a TRIP perturbation after a successful recovery. Once the TRIP impacted the walkway, participants took a single step with their right foot in an attempt to recover their balance.

During all trials, body segment position was sampled at 100 Hz using a Vicon 460 motion analysis system (Vicon, Lake Forest, CA, USA) and reflective markers placed bilaterally on the head, arms, trunk, and lower extremities. Force applied to the safety harness was sampled at 1000 Hz using a S-shape load cell (Cooper Instruments & Systems, Warrenton, VA, USA). Body segment and load cell data were low-passed filtered at 5 and 20 Hz, respectively (eighth order zero-phase-shift Butterworth filter used for all filtering). TRIP speed prior to impact (v_I) was calculated using a reflective marker on the TRIP and a central-difference method (Winter 2005). The whole body COM trajectory was approximated using

inertial parameter estimate methods presented by Pavol et al. (2002). Body angle (θ) was calculated from a line connecting the non-stepping (i.e. left) ankle to the body COM, and was measured from vertical. Body angular velocity after impact (ω_2) was calculated from θ using a central-difference method (Winter 2005).

The ratio of body angular velocity after TRIP impact with the walkway to TRIP speed prior to impact was determined for all trials. One obese participant was excluded from analysis due to incomplete marker data. A one-way analysis of variance (ANOVA) on the ranks (due to non-normal distributions) was used to determine the effects of obesity on this ratio. Additionally, a two-way mixed-model ANOVA was used to investigate the relationship between TRIP speed before impact and body angular velocity after impact. The independent variables for this analysis were group (normal-weight or obese), TRIP speed, and their interaction. The dependent variable for this analysis was the ranks of body angular velocity (due to non-normal distributions). Both of these analyses were performed using JMP v8 (Cary, NC, USA) with a significance level of $p \leq 0.05$. Additionally, a logistic regression model was used to investigate the relationship between body angular velocity after impact and balance recovery outcome. The independent variables for this analysis were group (normal-weight or obese), body angular velocity after impact, and their interaction. The dependent variable for this analysis was balance recovery outcome (successful or failed recovery). This analysis was completed using SAS v9.2 (Cary, NC, USA) with a significance level of $p \leq 0.05$.

6.4 Results

The ratio of body angular velocity after impact to TRIP speed was 8.8% higher in the obese compared to normal-weight participants (Obese: $0.889 \pm 0.107 \text{ m}^{-1}$, Normal-weight: $0.817 \pm 0.110 \text{ m}^{-1}$; $p=0.049$).

Body angular velocity after impact increased as TRIP speed increased ($p < 0.001$; Figure 6.4). In addition, a TRIP speed \times group interaction ($p=0.031$) indicated that as TRIP speed increased, body angular velocity

after impact increased at a higher rate in obese participants compared to normal-weight participants. The main effect of group did not reach but was approaching significance ($p=0.101$).

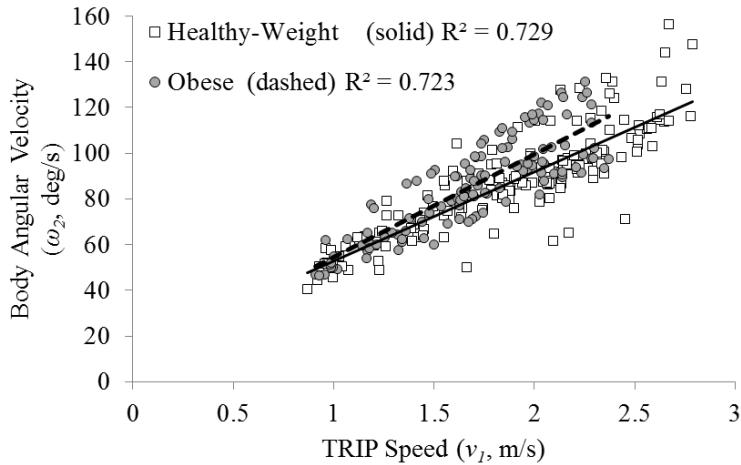


Figure 6.4. Body angular velocity (ω_2) as a function of TRIP speed (v_1) for normal-weight (white square) and obese (grey circle) participants. Regression lines were included for normal-weight (solid) and obese (dashed) participants for clarity of the relationship between body angular velocity and TRIP speed.

Balance recovery outcome was affected by body angular velocity after impact. As body angular velocity increased, the probability of a failed recovery also increased ($p<0.001$; Figure 6.5). There was a borderline significant effect of group ($p=0.080$) on balance recover outcome. The $\omega_2 \times$ group interaction did not reach but was approaching significance ($p=0.109$). Obese participants were at a greater odds of a failed recovery compared to healthy-weight participants, and this odds of a failed recovery decreased as ω_2 increased. For example, at an ω_2 of 40 °/s, an obese participant was 7.58 (95% Confidence Interval (CI): [0.84 66.67]) times more likely to have a failed recovery. At an ω_2 of 60 °/s, an obese participant was 4.18 (95% CI: [0.86 20.00]) times more likely to have a failed recovery. Finally, at an ω_2 of 80 °/s, an obese participant was 2.30 (95% CI: [0.73 7.30]) times more likely to have a failed recovery.

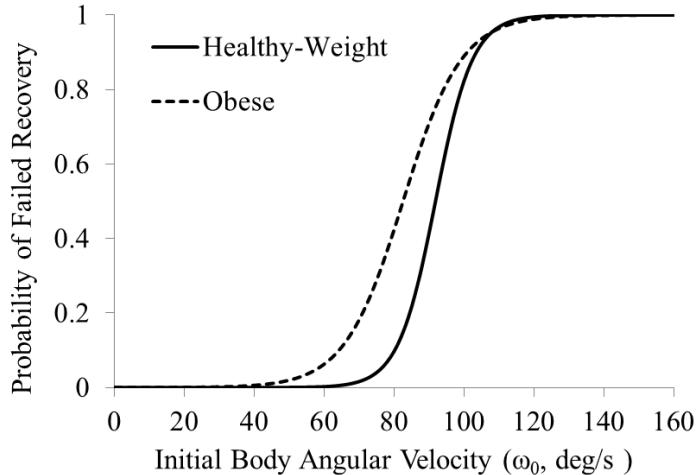


Figure 6.5. Probability of a failed recovery for normal-weight (solid line) and obese (dashed line) participants as a function of body angular velocity after impact with the walkway (ω_2).

Combining the relationship between TRIP speed and body angular velocity after impact with the relationship between body angular velocity after impact and probability of a failed balance recovery allowed the likelihood of failed balance recovery to be predicted based upon TRIP speed. For example, at a TRIP speed of 1 m/s, a typical normal-weight and obese participant were 0.1% and 3.4% likely to have a failed recovery, respectively. When TRIP speed was increased to 1.5 m/s, a normal-weight participant's probability of a failed recovery increased to 2.5% while an obese participant's likelihood increased to 34.3%. If TRIP speed was further increased to 2.0 m/s, a normal-weight participant's probability of a failed recovery increased to 50.0% while an obese participant's probability increased to 88.5%.

6.5 Discussion

The goal of this study was to investigate how body angular velocity immediately after a trip-like perturbation was related to the body linear velocity immediately before the perturbation. A larger ω_2/v_I ratio among obese participants would seem to make balance recovery more challenging for the obese, and may contribute to the reported increased fall risk among obese individuals. The ratio was found to be, on average, 8.8% higher among young obese compared to young normal-weight participants. In addition, the probability of a failed balance recovery increased as body angular velocity after the trip-like

perturbation increased. Together, the results suggest that if a young normal-weight and obese individual tripped while walking at identical speeds, the obese individual would experience a larger body angular velocity about the ankle, and this larger angular velocity would make balance recovery more challenging. For example, if a young normal-weight and obese participant were walking at 2.1 m/s, the obese participant would have an 8.8% higher body angular velocity immediately after tripping. This increase in angular velocity would result in a 22.9% higher risk of a failed balance recovery.

We have demonstrated that the ratio ω_2/v_I differs between young normal-weight and obese individuals. Based upon Equation (6-6), this difference is due to differences in BSIPs that appear on the right-hand-side of this equation. The right-hand-side can be approximated with BSIPs from Matrangola et al. (2008) for an obese individual and BSIPs from Chandler et al. (1975) for a normal-weight individual. Using these estimates, the right-hand-side of this equation predicts an obese individual to have a 2.5% greater ω_2/v_I ratio. This is smaller than the 8.8% difference that we measured experimentally after a trip-like perturbation, and there are several possible explanations as to why these do not agree more closely. Previous work has noted inaccuracies with predicting segment inertial parameters of the obese (Matrangola et al. 2008; Chambers et al. 2010). For example, Matrangola et al. (2008), estimated BSIPs of obese individuals while lying in a supine position (Matrangola et al. 2008), and this likely contributed to error in these estimates due to soft tissue deformation. Also, two separate BSIPs estimates were used to predict the right-hand-side, and this could have contributed to the smaller difference estimate between groups.

Our results provide a biomechanical mechanism by which obesity may contribute to falls after tripping. Following laboratory-induced trips, 46.2% of obese versus 25.0% of normal-weight women fell, though this difference did not reach statistical significance ($p=0.44$) (Rosenblatt and Grabiner 2011). While this may appear to be inconsistent with this current study, it is interesting to note that participants were tripped while walking at a self-selected speed, and obese participants walked more slowly than their normal-

weight counterparts (obese: 1.26 ± 0.15 m/s, normal-weight: 1.40 ± 0.21 m/s, $p=0.07$) (Rosenblatt 2011).

Preferred walking speed is typically lower in obese individuals compared to the non-obese individuals (Spyropoulos et al. 1991; DeVita and Hortobagyi 2003; Fabris de Souza et al. 2005). The lack of difference in fall rate reported by Rosenblatt et al (2010) could be explained by a slower walking speed in the obese, which would result in similar or smaller body angular velocities in individuals who are obese at onset of the trip. Using average gait speeds reported by Spyropoulos et al. (1991) for normal-weight (1.64 m/s) and obese (1.09 m/s) adults, our results indicate that obese participants would have a body angular velocity of approximately 60 deg/s following a trip at a self-selected speed, whereas a normal-weight individual would have a body angular velocity of approximately 80 deg/s. This would translate to a 5.4% and 6.6% probability of a failed balance recovery in the obese and normal-weight participants, respectively. If obese participants were required to walk at a similar speed as normal-weight participants, their probability of a failed recovery would increase to 52.6%. This illustrates the importance of walking speed on fall risk among individuals who are obese. With this in mind, it is also important to acknowledge that some of the TRIP speeds obtained in this study are typically higher than preferred walking speed of either normal-weight or obese individuals. It is not known how often normal-weight and obese individuals walk at these higher speeds. Balance recovery was also limited to a single step for simplicity of analysis and comparison across groups. During a real trip, individuals are able to take multiple steps if they are unable to arrest forward momentum after the initial step. These two limitations may have compromised the generalizability of our results pertaining to prediction of balance recovery outcome to falls outside the laboratory.

The derivation of Equation (6-6) was based upon modeling the body as a single rigid segment. This has been shown to be reasonable for a short time interval surrounding an actual trip (van den Bogert et al. 2002) and is also reasonable here for trip-like perturbations on the TRIP for two reasons. First, body segments were lined up reasonably vertically at the instant the TRIP impacted the walkway. Second, the typical time interval from time “1” to time “2” in our analysis was 130 ms. Typical changes in joint

angles over this time interval for the TRIP perturbation were approximately 2, 10, and 3 degrees at the hip, knee, and ankle, respectively.

Additional limitations warrant discussion. First, analysis was limited to the sagittal plane. While non-sagittal plane movements can occur during balance recovery after tripping, movement was primarily in the sagittal plane for the time interval of interest of this study. Second, ten participants were recruited for each group, and this small sample size may have contributed to the borderline significance of some of our results by being underpowered. Third, differences between the normal weight and obese groups other than those investigated here could have contributed to our results. For example, it is possible that there were differences in baseline muscle activity to stiffen the joints immediately before impact with the walkway. However, we found no differences in the change in body angle from time “1” to time “2” between groups (Normal-weight: $5.0 \pm 1.5^\circ$, Obese: $5.5 \pm 1.3^\circ$, $p=0.537$). Finally, BMI was used to classify participants as normal-weight or obese. While BMI is a frequently used standard and is highly correlated to the relative amount of body fat (Wilmore and Costill 1994), it is still possible for an individual to be classified as obese but have a normal or lower-than-normal body fat content. This would suggest that a greater portion of the individual’s mass is lean tissue (i.e. muscle), which would contribute differently to the distribution of mass compared to fat.

6.6 Conclusions

In conclusion, young obese participants exhibited a higher body angular velocity than young healthy weight participants when both experienced a trip-like perturbation at the same speed. Higher body angular velocity was associated with a higher probability of a failed balance recovery. These results suggest that, when walking at similar speeds, young obese individuals may be at a greater risk of falling following a trip, and that this may be due to differences in how body mass is distributed. The results of the present study may provide a biomechanical mechanism to help explain the increased risk of falls among young obese individuals.

6.7 Acknowledgements

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Chapter 7 – The effects of obesity on relative strength used during balance recovery from a forward fall in young adults

7.1 Abstract

Obesity is a major health concern around the world and in the United States. Obesity is associated with reduced functional capability, and this may be related to the documented lower strength relative to body weight among individuals who are obese. The purpose of this study was to determine if an obese and normal-weight individual employ the same relative strength when performing the same functional task (balance recovery from a forward fall). In order to compare relative strength used during balance recovery, the obese and normal-weight individual should complete the task with identical kinematics. To accomplish identical kinematics between groups, forward dynamic simulations were used because doing so with human subjects would be difficult. Differences in peak relative torques were found between the normal-weight and obese model, with the largest differences seen at the hip. These results indicate that the obese model required a greater relative hip strength than the normal-weight model to move with similar kinematics, and this is likely due to the greater proportion of mass in the trunk of the obese model. Overall, these findings suggest that obese individuals use greater relative strength at some joints than normal-weight individuals to perform the time-critical task of balance recovery, and that these differences in relative strength demands may limit functional capability in obese individuals.

7.2 Introduction

Obesity is a major health concern around the world and the United States (US). The World Health Organization (WHO) estimated over 1.5 billion adults world-wide were overweight in 2008, and of those, over 500 million were obese (WHO 2011). In the same year, over one-third of US adults were obese (Flegal et al. 2010). This high prevalence is a major concern due to the multitude of medical conditions associated with obesity including type 2 diabetes mellitus, cardiovascular disease, osteoarthritis, and respiratory disease (Puska et al. 2003; Bray 2004). In fact, obesity has been estimated to be responsible for approximately 95 million of years-of-life-lost among US adults in 2008 (Finkelstein et al. 2009).

Obesity is also costly to society, accounting for over \$147 billion of medical expenditures in the US in 2008 (Finkelstein et al. 2009b).

In addition to these medical conditions, obesity reduces functional capability, regardless of age (Lakdawalla et al. 2004). For example, rising from a chair was more difficult for obese children compared to non-obese children (Riddiford 2000). Evers Larsson and Mattsson (2001) found obese middle-aged women took longer and had more difficulty performing 13 of 16 functional tasks including balancing, squatting, stair ascent/descent, walking while carrying external weight, and rising from a seated position. Among older adults, the odds of having impaired functional capability increase as BMI increases (Houston et al. 2005), and with obese older adults having a ~5 times increased risk of impaired functional capability compared to their non-obese counterparts (Zoico et al. 2004). Because the reduced functional capability in the obese is typically associated with the lower-body (Ferraro et al. 2002), it is thought the reduced functional capability is a result of increased lower extremity strength demands during various activities (Capodaglio et al. 2010).

Obese individuals exhibit greater lower limb strength, in an absolute sense, compared to non-obese individuals (Hulens et al. 2001; Lafortuna et al. 2005; Capodaglio et al. 2009). At the same time, obese individuals exhibit less strength, compared to non-obese individuals, when strength is expressed relative to body weight (Hulens et al. 2001; Lafortuna et al. 2005; Maffiuletti et al. 2007; Capodaglio et al. 2009; Handrigan et al. 2010). For example, compared to normal-weight individuals, obese individuals were ~20% stronger when considering absolute knee extensor strength, but were 30% weaker when knee extensor strength was normalized to body weight (Capodaglio et al. 2009). These strength differences, along with the limited functional capability of the obese, suggest that strength relative to body weight is a more important indicator of functional capability than strength in an absolute sense. For example, the correlation between muscle strength and performance during a maximal lift task and one leg chair rise task improved after accounting for body mass (Aasa et al. 2003). Similarly, when muscle strength values

of the hip extensors and knee extensors in older adults were summed and normalized to body weight, the correlation of this “leg strength” variable to gait speed and completion of an obstacle increased (Brown et al. 1995).

The relationship between absolute strength and functional capability is straightforward. In general, greater absolute strength is related to better functional capability (Wolfson et al. 1995; Landers et al. 2001; Avers and Brown 2009). However, the relationship between relative strength and functional capability remains less clear. This relationship is important to understand the significance of differences in relative strength between obese and non-obese individuals reported in the literature. Clearly, an increase in relative strength would be related to better functional capability. But how much relative strength is necessary for an obese individual to maintain the same level of functional capability as a non-obese individual? It is tempting to assume that an obese individual and a non-obese individual with the same relative strength would exhibit the same level of functional capability. However, it is unclear whether this is true. The purpose of this study is to improve our understanding of the relationship between relative strength and functional capability. In particular, this study will determine if an obese individual and a non-obese individual employ the same relative strength when performing the same task. Forward dynamic simulations will be used to address this research question, instead of human subjects testing, due to difficulties in achieving identical kinematics across multiple subjects. The task we will investigate is balance recovery from a forward fall. This task was chosen because 1) it is a time-critical task that involves high strength demands, 2) it is related to a significant source of injuries among the obese (falls).

7.3 Methods

A combination of human subjects testing and forward dynamic simulations of balance recovery were used for this study. Human subject testing was first used to determine joint kinematics during balance recovery in normal-weight participants. Joint kinematics were then used as inputs to a mathematical

model of balance recovery by stepping to determine joint torques necessary for a representative normal-weight and obese participant to recover balance.

Ten normal-weight adult male participants were recruited for the human subject testing portion of this study (mean \pm standard deviation, age: 21.4 ± 1.5 years, height: 1.76 ± 0.06 m, weight: 65.9 ± 5.4 kg, BMI Range: 19.4 to 22.9 kg/m^2). This study was approved by the Virginia Tech Institutional Review Board, and written consent was obtained from all participants prior to participation.

Participants were subjected to perturbations that imposed an initial angular velocity of the body with no initial angular displacement from the natural standing position, and used a single step to recover balance. This was accomplished by having participants stand on a custom-built TRanslatIng Platform (TRIP), and the TRIP was pushed forward at a constant velocity while the participants faced forward (i.e. TRIP trials). The TRIP then collided with a rigid stop (Figure 7.1). Through conservation of momentum (Meriam and Kraige 2002), this imposed an angular velocity on the participant about an axis through the ankles. More detailed methodology of TRIP trials is available in Chapter 5.

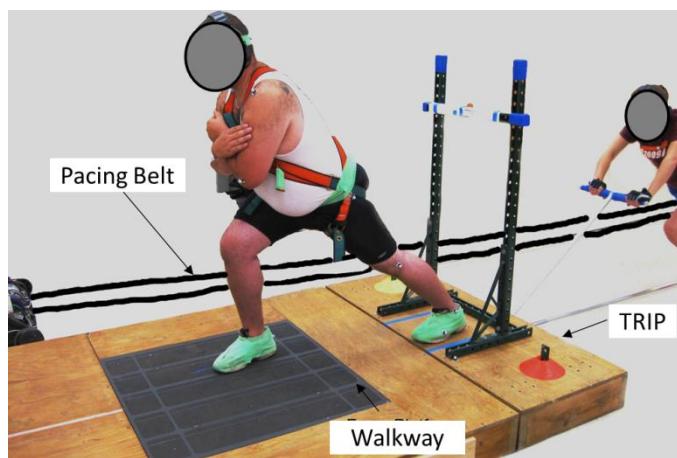


Figure 7.1. Photograph of cart trials after a successful recovery. Participants stood upright on a cart with eyes closed while an assistant pushed the cart forward at a constant velocity. The assistant matched speed with a pacing belt on the side of the walkway. The cart hit a rigid stop, where participants took a single step with their right leg to recover their balance.

During all trials, body segment positions were sampled at 100 Hz using a Vicon 460 motion analysis system (Vicon, Lake Forest, CA, USA) and reflective markers placed bilaterally on selected anatomical landmarks. Body segment positions were low-passed filtered at 5 Hz (eighth order zero-phase-shift Butterworth filter). A time series of joint angles (ankle, knee, hip) of the non-stepping and stepping limb were then determined for each participant. Time was normalized from 0 to 100% of time to stabilization (defined as reversal of the center of mass velocity). Joint angles were then averaged across participants to determine an average time series of joint kinematics for a representative normal-weight subject. This average time series of joint kinematics was determined from trials in which normal-weight participants successfully recovered balance from their maximum TRIP speed (Chapter 5).

A two dimensional (sagittal plane) model with seven segments and six torque actuators was developed to investigate the effects of obesity on relative strength demands during balance recovery. The seven segments represent the feet, shanks, thighs, and a head, arms and trunk (HAT) segment. The six torque actuators representing ankle, knee, and hip joint torques of each lower limb (Figure 7.2). A dynamics engine (Autolev, OnLine Dynamics, Inc., Sunnyvale, CA) was used to determine the equations of motion for the system.

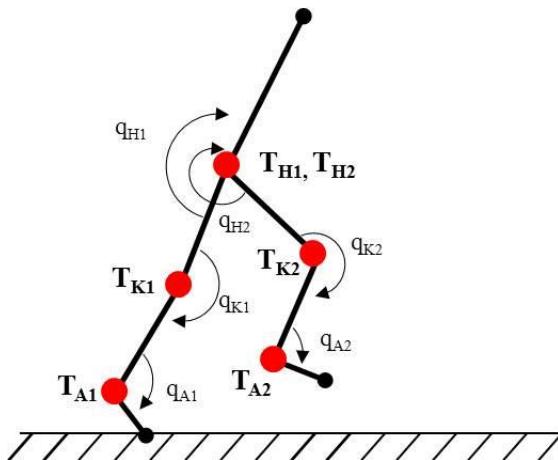


Figure 7.2. Seven link model (feet, shanks, thighs, HAT) with six torque actuators (ankles, knees, hips). “1” subscript denotes the stance leg and “2” subscript denotes the stepping leg.

The foot-ground interface was modeled using three spring-damper units located at the stance limb heel, stepping limb heel, and stepping limb toe (Anderson and Pandy 1999). The vertical force varied exponentially with the height of foot from the ground and was expressed as:

$$F_{vert,i} = 0.5336e^{-1150(p_{y_i}-y_0)} - 1000v_{y_i}g(p_{y_i}) \quad (7-1)$$

$$g(p_{y_i}) = \frac{1}{1 + 10e^{500(p_{y_i}-g_0)}} \quad (7-2)$$

where p_{y_i} is the vertical position of the i th point of application of the spring force ($i=3$), y_0 is a parameter that determines when the magnitude of force becomes significant ($y_0 = 0.0065905$ m), v_{y_i} is the vertical velocity of the i th point of application of the spring force, $g(p_{y_i})$ is a function that gradually adds damping as the foot approaches the ground, and g_0 is the parameter that determines when the damping effect is applied ($g_0 = 0.02$) (Anderson and Pandy 1999). Friction between the foot and ground was determined from a Coulomb friction model as:

$$F_{shear,i} = \gamma F_{vert,i} \frac{\dot{x}_i}{|\dot{x}_i|} \quad (7-3)$$

where γ is the coefficient of friction and \dot{x}_i is the horizontal (anterior-posterior) velocity of the i th point of application of the spring force (van den Bogert et al. 1989; Gerritsen et al. 1995). For numerical stability, this friction model was approximated as:

$$F_{shear,i} = -\frac{2\gamma F_{vert,i}}{\pi} \arctan\left(\frac{\lambda_h \pi}{2\gamma F_{vert,i}} \dot{x}_i\right) \quad (7-4)$$

where γ is the coefficient of friction ($\gamma = 0.7$, unitless) and λ_h is the slope of the function approximation ($\lambda_h = 3000$ N s m⁻¹) (Gerritsen et al. 1995; Anderson and Pandy 1999).

Torque actuators were used to represent the effects of muscle activation on model movement, and are a sum of passive and active joint torques. Passive joint torques were used to limit the joint angles from reaching values that are physically impossible, and are computed as a sum of exponential terms (Audu 1985; Anderson 1999; Anderson and Pandy 1999) :

$$T_{passive,j} = k_{0,j} + k_{1,j}e^{k_{2,j}(q_j - \theta_j)} + k_{3,j}e^{k_{4,j}(q_j - \varphi_j)} \quad (7-5)$$

where q_j is the angular displacement of the j th joint, and $k_{0,j}$, $k_{1,j}$, $k_{2,j}$, $k_{3,j}$, θ_j , and φ_j are constants that determine the shape of the torque-angle curve of the j th joint. Constants were defined by Anderson (1999).

The active joint torque (T_{act}) was a function of maximum isometric torque, joint angle, joint angular velocity, and activation level (Selbie and Caldwell 1996; Ashby and Delp 2006; Cheng 2008; Cheng et al. 2008):

$$T_{act} = T_{max} * f(\theta) * h(\omega) * A(t) \quad (7-6)$$

where T_{max} is the maximum isometric torque (units: Nm), two scaling factors that are dimensionless and vary from 0 to 1: $f(\theta)$ is the joint angle factor and $h(\omega)$ is the joint angular velocity factor, and $A(t)$ is the joint activation level that varies from -1 to 1 to represent flexion and extension of the joint. Maximum isometric torque was approximated from previous experimental data with participant height and mass (Anderson et al. 2007). The scaling factors $f(\theta)$ and $h(\omega)$ were used to scale the value of T_{max} based on the current joint angle and angular velocity. The torque-angle relationship ($f(\theta)$) was obtained from a previous study that determined polynomial fits (Bieryla 2009) from previous experimental data (Hoy et al. 1990). The torque-velocity relation is expressed as:

$$\begin{cases} h(\omega) = \frac{(\omega_0 - \omega)}{(\omega_0 - \Gamma\omega)} & , \omega/\omega_0 < 1 \\ h(\omega) = 0 & , \omega/\omega_0 \geq 1 \end{cases} \quad (7-7)$$

where ω is the angular velocity, ω_0 is the maximum angular velocity (20 rad/sec), and Γ is the shape factor describing the torque-angular velocity curve ($\Gamma=2.5$) (Alexander 1989; Selbie and Caldwell 1996). When an eccentric muscle contraction occurred, the torque-angular velocity factor was increased to a

maximum value of 1.5. A time series of joint activation for each joint was determined from activation nodes equally spaced at 100ms increments and linear interpolation between consecutive nodes.

A simulated annealing algorithm was used to determine joint activations, and ultimately joint torque, for each representative subject. Simulated annealing is beneficial when optimizing a large set of variables sets and has a random component that allows the algorithm to escape from local optimums to find a global optimum (Corana et al. 1987; Goffe et al. 1994). The algorithm was used to track experimental data by minimizing the error between experimental and simulated joint angles.

Participant height and mass were set to the average experimental values (Chapter 5) to define a representative subject for each group (normal-weight and obese). Additionally, segment inertial parameters were estimated using de Leva (1996) for the representative normal-weight participant and Matrangola et al. (2008) for the representative obese participant. The stepping phase (i.e. time from perturbation onset until the stepping foot contacts the ground after stepping) and support phase (i.e. time from foot contact after stepping until reversal of the COM velocity) were modeled separately for each representative subject. This was done because the different phases of single-step recovery have different goals and functional requirements (Madigan and Lloyd 2005). By modeling these phases separately, the requirements of one phase will not influence model performance in the other. The duration of the stepping phase was set to the average time to foot contact from experimental trials (0 to 500ms). Due to estimates of segment length, the joint kinematics immediately after foot contact time resulted in a foot location below ground. To reduce large accelerations due to the foot-ground interface, the modeling of the support phase began 100ms prior to foot contact. Model output was then truncated to the support phase (i.e. foot contact to time to stabilization; 500 to 1100 ms). This resulted in initial joint configurations that were different from the experimental mean, but within one standard deviation for nearly all joints. Optimal joint activations determined from the simulated annealing algorithm were then used to calculate relative joint torques (joint torque normalized by body mass, Nm/kg).

7.4 Results

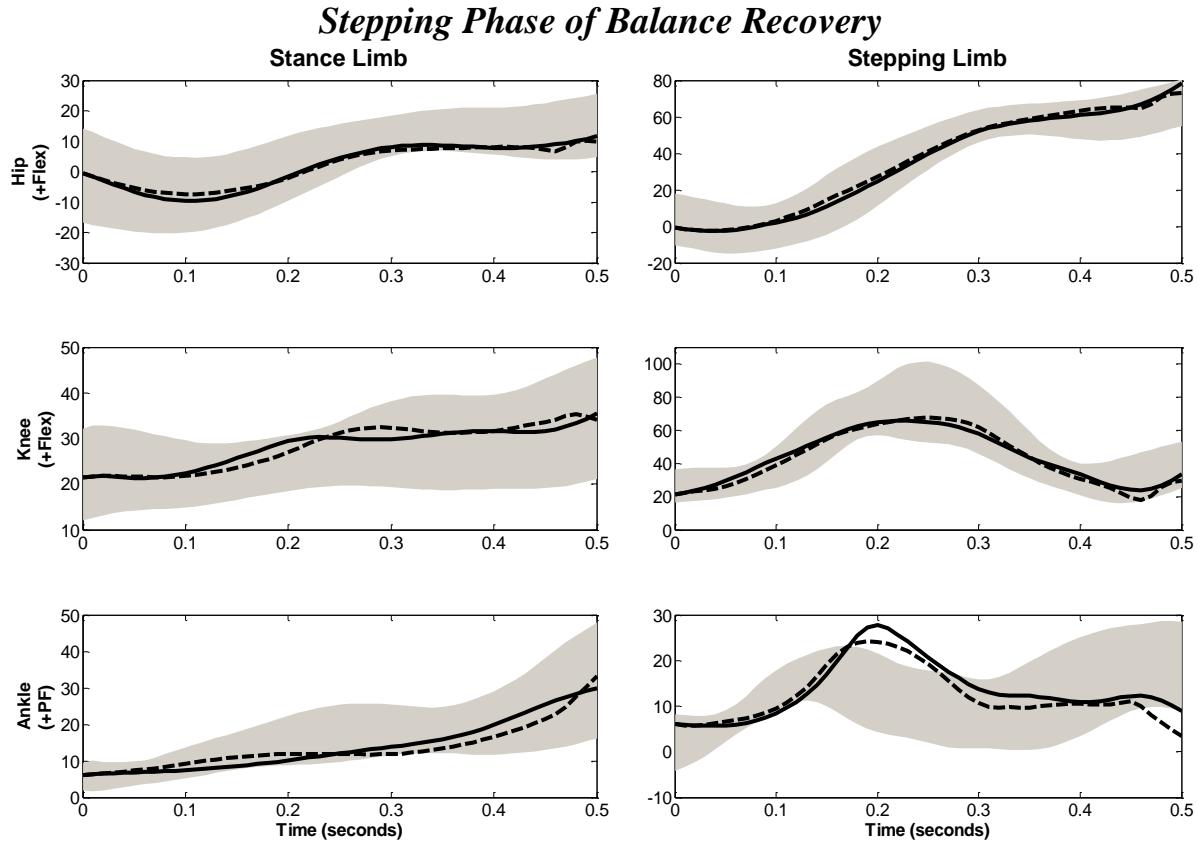


Figure 7.3. Joint angles during the stepping phase. Experimental data of normal-weight young adults with ± 1 s.d. (gray area) is plotted with normal-weight (solid) and obese (dashed) model. Simulated joint angles were within one standard deviation of the experimental angles for most of the simulations. Positive angles correspond to ankle plantarflexion, knee flexion, and hip flexion.

The models reproduced the normal-weight experimental joint kinematics fairly well, and normal-weight and obese model kinematics exhibited only minor differences from each other. For the stepping phase (Figure 7.3), the average root mean square (RMS) difference between simulated and mean experimental joint angles was 4.1° and 4.0° for the normal-weight and obese model, respectively. In addition, simulated joint angles were within one standard deviation of the experimental mean for 95% of the simulation. The average RMS difference in joint angles between the normal-weight and obese model simulations was 1.5° . For the support phase (Figure 7.4), the average difference between simulated and

mean experimental joint angles was 6.1° for both the normal-weight and obese model, and simulated joint angles were within one standard deviation of the experimental mean for 75% of the simulation. The average RMS difference in joint angles between the normal-weight and obese model was 1.7° .

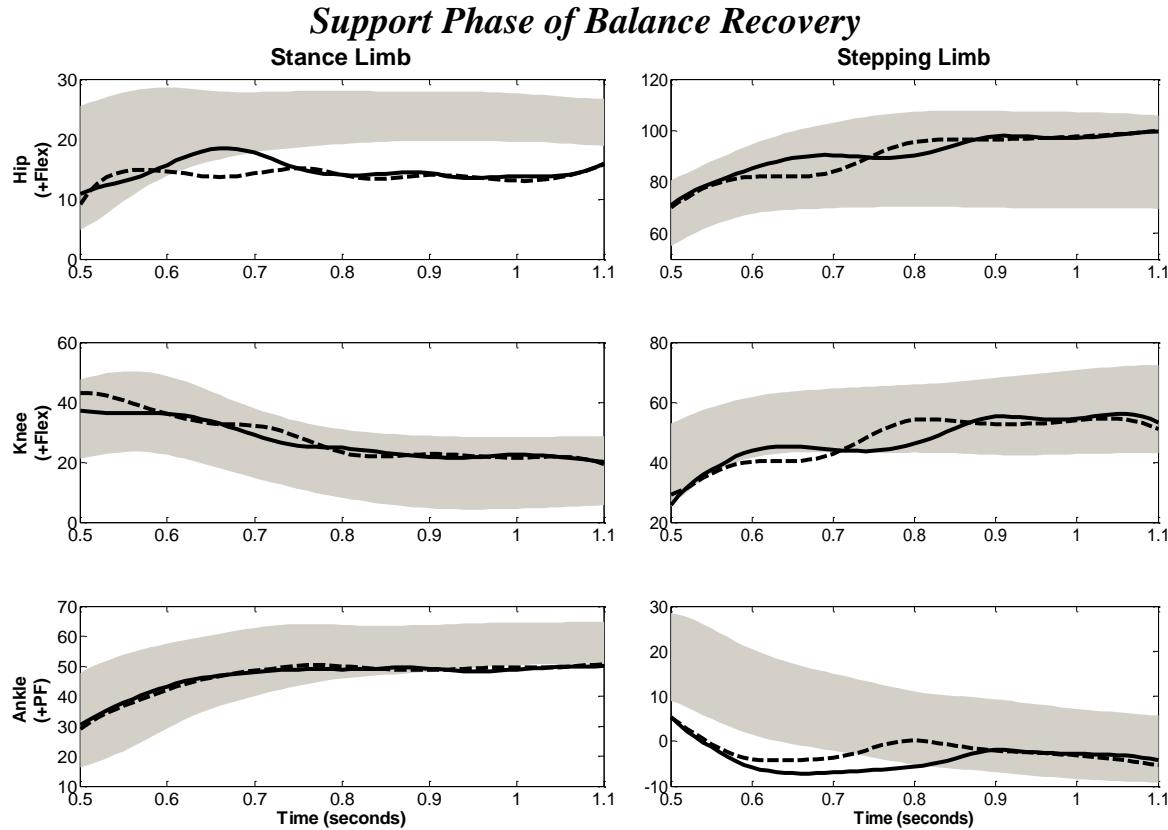


Figure 7.4. Joint angles during the support phase. Experimental data of normal-weight young adults with ± 1 s.d. (gray area) is plotted with normal-weight (solid) and obese (dashed) model. Simulated joint angles were within one standard deviation of the experimental angles for most of the simulations, with the exception of the stance hip and stepping ankle. Positive angles correspond to ankle plantarflexion, knee flexion, and hip flexion.

Differences in peak relative torques were apparent between normal-weight and obese simulations. The largest differences in peak relative joint torque were found at the hip. During the stepping phase (Figure 7.5), the peak relative hip extensor torque was 1.07 and 0.95 Nm/kg higher in the obese model stance and stepping limb, respectively, compared to the normal-weight model. This difference corresponds to an increase in absolute hip extensor torque of 95-110 Nm in the obese model. During the support phase (Figure 7.6), peak relative hip extensor torque was 0.60 Nm/kg higher in the obese model stepping limb

compared to the normal-weight model. This difference corresponds to an increase in absolute hip extensor torque of 60 Nm in the obese model.

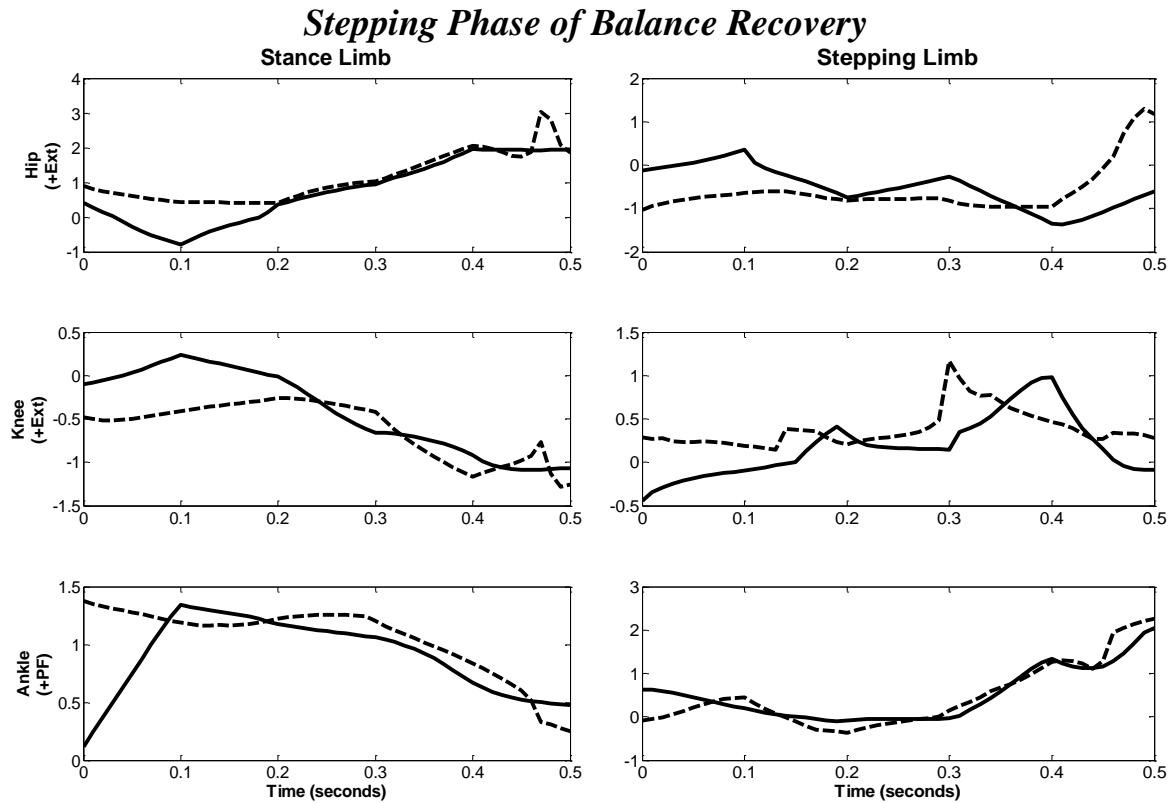


Figure 7.5. Relative joint torques in the stance (left graphs) and stepping (right graphs) limb for the stepping phase for the normal-weight (solid) and obese (dashed) model. Positive values indicate extensor (or plantarflexor) torques.

Other differences in peak relative torques between normal-weight and obese models were smaller in magnitude. Only differences greater than 0.20 Nm/kg were deemed to be functionally important. During the stepping phase, the peak relative dorsiflexor and plantarflexor torque were 0.26 and 0.23 Nm/kg higher in the obese model stepping limb, respectively, compared to the normal-weight model. During the support phase, the peak relative plantarflexor torque in the stepping limb was 0.48 Nm/kg lower in the obese model. Also during the support phase, peak relative knee extensor torque in the stance limb was 0.23 Nm/kg higher in the obese model. These differences correspond to a 23-49 Nm difference in absolute torque for the obese model.

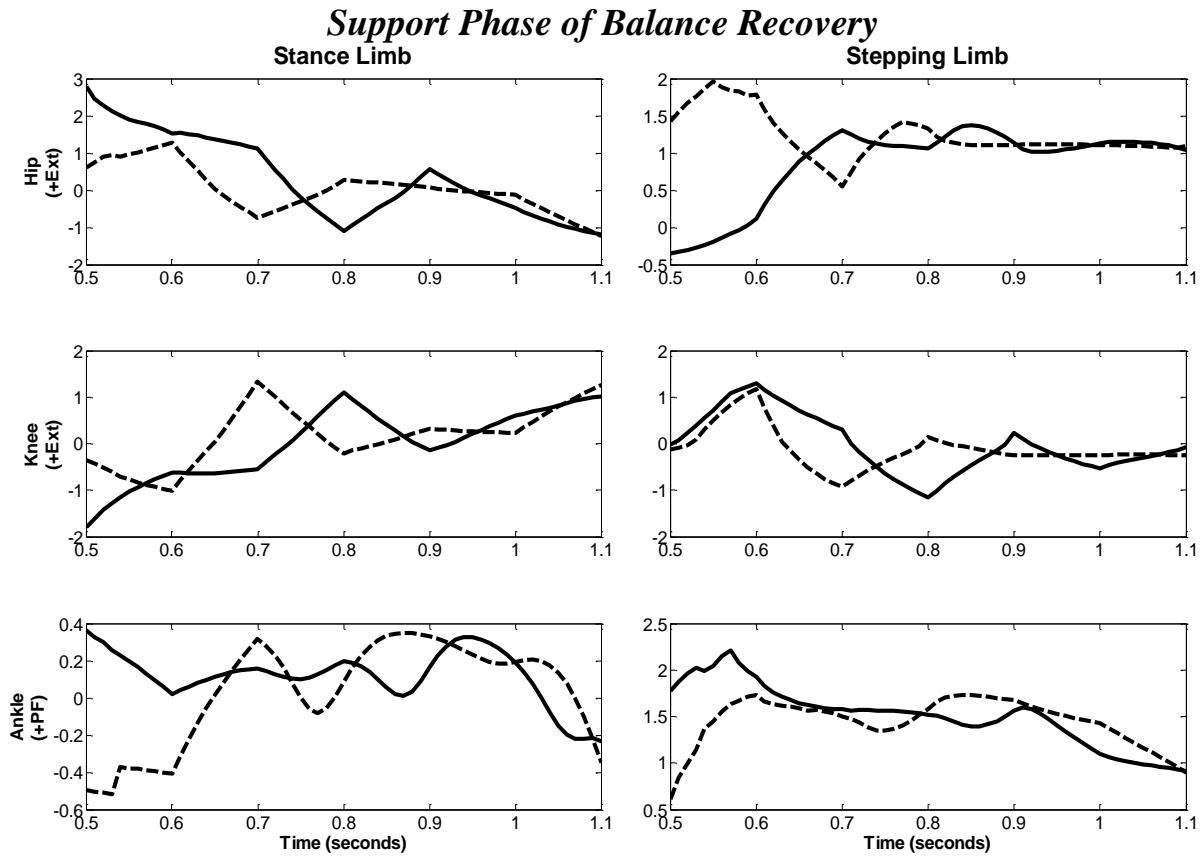


Figure 7.6. Relative joint torques in the stance (left graphs) and stepping (right graphs) limb for the support phase for the normal-weight (solid) and obese (dashed) model. Positive values indicate extensor (or plantarflexor) torques.

In addition to these differences in peak relative joint torques, differences were seen in the overall relative joint torque patterns. During initiation of the step (time = 0.00 to ~0.20 s), the obese model used more hip flexor torque to initiate the step, as seen by a higher relative hip flexor torque and no knee flexor torque during this time. In contrast, the normal-weight model primarily used a combination of knee flexor and plantarflexor torque. Also during step initiation, the obese model used a higher relative hip extensor and knee flexor torque in the stance limb, while the normal-weight model primarily used knee extensor torque in the stance limb. Prior to foot contact (time = 0.45 to 0.50 s), the obese model used a relative hip extensor torque in the stepping limb while the normal-weight model was still producing a hip flexor torque. At the beginning of the support phase, the obese model used greater hip extensor torque to stabilize, while the normal-weight model used a combination of knee extensor and ankle plantarflexor.

7.5 Discussion

The goal of this study was to determine differences between an obese and non-obese individual in relative strength necessary to recover balance from forward fall with nearly identical kinematics. Obese individuals are reportedly weaker than normal-weight individuals when comparing relative strength and this deficit may be indicative of functional capability. The largest difference was that the obese model utilized significantly higher peak relative hip extensor torque (approximately 1 Nm/kg) during weight acceptance after stepping. Smaller differences in relative torques were also identified at the ankle, with the obese model utilizing greater relative dorsiflexor and plantarflexor torque during the step, and decreased relative plantarflexor torque during weight acceptance after stepping. These findings suggest that young obese individuals require more relative strength than young normal-weight individuals to perform the time-critical task of balance recovery by stepping with nearly-identical kinematics.

The difference in experimental and simulated joint angles is comparable to other previously published forward dynamic simulations that attempted to match simulated kinematics of a multi-link model with experimental kinematics. For example, two studies using a nine-segment model of the body to simulate walking reported mean differences in joint angles of 3.9° and 2.6° between simulated and mean experimental kinematics with all simulated joint angles falling within two standard deviations of the mean experimental data (Sasaki and Neptune 2010; Neptune and McGowan 2011). While our average difference values are slightly higher ($\sim 5^\circ$ averaged across the stepping and support phase), the majority of our simulated joint angles were within one standard deviation of the experimental data. Additionally, a five-segment model of gymnastics tumbling using torque actuators reported similar mean differences in simulated and experimental kinematics of $5\text{--}6^\circ$ (Yeadon and King 2002). There was also good agreement in joint torque trends and magnitudes between our joint torque profiles and those in literature for similar balance tasks (Madigan and Lloyd 2005; Pijnappels et al. 2005b; Madigan 2006). One exception was that the model used approximately twice the hip extensor torque in the stance limb during the step compared to published experimental joint torque profiles.

The increased relative hip extensor torque and utilization of the hip for step initiation and COM stabilization in the obese model suggests that hip strength is an important contributor to balance related tasks among young individuals who are obese. These results also suggest that controlling motion of the trunk requires greater relative strength in a young obese individual compared to a young normal-weight individual, which is related to the increased proportion of mass and preferential accumulation of fat in the trunk of obese individuals (Bjorntorp 1991; Corbeil et al. 2001; Matrangola et al. 2008). In contrast to our findings, previous work suggests that knee and ankle strength are important contributors to balance ability, and impaired strength in these joints was indicative of an increased risk of falls (Whipple et al. 1987; Takazawa et al. 2003). Similarly, knee extensor strength has been suggested to be important to functional capability (Landers et al. 2001; Ploutz-Snyder et al. 2002). However, it is possible that hip extensor strength may be more critical for some functional activities. In support of the importance of hip extensor strength, Hasegawa et al. (2008) reported that the relative strength of the hip extensors was the best discriminator between older adults who could or could not perform functional tasks independently. While the ankle and knee may still be important contributors to balance ability and functional capability among individuals who are obese, the higher relative torques at the hip compared to normal-weight adults suggest that hip extensor strength is more critical towards successful recovery among young obese individuals.

Differences in the trends of the joint torque profiles were also seen between the normal-weight and obese model. The obese model used a higher relative hip flexor torque during initiation of the step while the normal-weight model had a larger ankle plantarflexor and knee flexor torque. The larger relative hip flexor torque in the obese model was most likely needed to flex the hip due to the increased proportion of mass in the trunk and lower limb. This increased hip flexor torque can also contribute to knee flexion and ankle plantarflexion (Madigan 2006), thereby decreasing the relative strength demands at these joints. The opposite effect was seen during weight acceptance, with the normal-weight model using a higher

relative knee extensor and ankle plantarflexor torque and the obese model using a higher relative hip extensor torque. The larger relative hip extensor torque to control trunk movement in the obese model contributed to knee extension and ankle plantarflexion and resulted in lower relative torques at the knee and ankle. Similar to our obese model, older adults have been shown to use greater hip torques during balance recovery from a release from a static forward lean (Wojcik et al. 2001; Madigan and Lloyd 2005; Madigan 2006). It is hypothesized that the larger hip torques seen in older adults is a neuromuscular adaptation in order to perform this task and has been noted in other functional activities (DeVita and Hortobagyi 2000; Madigan and Lloyd 2005). Similarly, it is possible that obesity may require a neuromuscular adaptation in order to perform functional tasks in young adults. In particular, adaptations are necessary to control the trunk, and may cascade down to reduce strength demands at other joints. In support of this, DeVita and Hortobagyi (2003) have previously suggested that neuromuscular function is reorganized in obese individuals to result in reduced knee joint loading during gait.

In addition to differences in the stepping limb, there were also differences in relative joint torque patterns in the stance limb. The obese model used a higher relative hip extensor and knee flexor torque in the stance limb during step initiation. A large knee flexor and hip extensor torque in the stance limb is necessary for the push-off reaction to complete the step in the contralateral limb and is also important for restraining forward angular momentum of the body (Pijnappels et al. 2005b). Our results suggest that obesity may require a greater relative strength in order to provide a similar contribution of the stance limb to balance recovery as a normal-weight individual in young adults.

There are some limitations to our forward dynamics model that warrant discussion. First, torque actuators were used instead of muscle actuators. Muscle actuators allow for consideration of bi-articular muscles and co-contraction of muscles. However, previous studies have shown reasonable agreement between experimental and model kinematics when using torque actuators (Yeadon and King 2002; Ashby and Delp 2006). Second, maximum isometric torques were estimated for the normal-weight and obese model

based on experimental data from a previous study (Anderson et al. 2007). While this may have limited the available joint torque to track experimental data, differences between the normal-weight and obese model joint kinematics were small. Third, the stepping and support phase were modeled separately. In particular, by starting the support phase prior to foot contact, there was approximately 5° difference in the stance knee flexion angle at foot contact between the normal-weight and obese model. However, both models were within one standard deviation of the experimental mean. Additionally, all other initial joint configurations of the normal-weight and obese model were within one degree of each other. It is important to note that even with these limitations, we achieved similar differences between simulated and experimental data as a more complex two-legged walking model, with thirteen degrees of freedom in the sagittal plane and 25 muscle actuators per leg (Sasaki and Neptune 2010; Neptune and McGowan 2011).

It is also important to note limitations in generalizing our modeling results to falls in young adults outside the laboratory. First, the model investigated balance recovery using a single step. During a real trip, individuals are able to take multiple steps if they are unable to arrest forward momentum after the initial step. Second, the normal-weight and obese model used identical kinematics, and this ignores fall scenarios in which strategies and kinematics differ between normal-weight and obese individuals. Finally, individual differences exist in the distribution of mass among individuals who are normal-weight and who are obese. Therefore, the results from this study are limited to those with similar distribution as those imposed on the representative normal-weight and obese models.

7.6 Conclusions

In conclusion, differences in peak relative torques were found between the normal-weight and obese model during single-step balance recovery with the largest differences seen at the hip. Our results suggest that the obese model required a greater relative hip strength than the normal-weight model to move with similar kinematics. This also suggests controlling motion of the trunk requires greater relative strength in young individuals who are obese compared to normal-weight, and this is likely due to the greater

proportion of mass in the trunk of the obese model. Overall, these findings suggest that young obese individuals use greater relative strength at some joints than young normal-weight individuals to perform the time-critical task of balance recovery, and that these differences in relative strength demands may limit functional capability among young individuals who are obese.

7.7 Acknowledgements

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Chapter 8 – Conclusions

8.1 Summary and contributions

The studies presented here have explored the effects of obesity on balance recovery from postural perturbations in young adults. In the first study (Chapter 4), we used laboratory experiments to examine the effects of obesity on balance recovery using an ankle strategy in young adults. Obese participants were unable to recover balance using an ankle strategy as well as normal-weight participants when perturbations involved an initial angular velocity. However, no differences in balance recovery were found between young obese and normal-weight participants when perturbations only involved an initial angular displacement. It was hypothesized that the dependence of our results on perturbation characteristics may be explained by a possible beneficial effect of increased inertia on balance recovery after perturbations with little or no initial angular velocity.

In the second study, (Chapter 5) we also used laboratory experiments to examine the effects of obesity on balance recovery using a single step in young adults. In contrast to the first study on balance recovery using an ankle strategy, the ability to recover balance with a single-step did not differ between young normal-weight and young obese adults. These results suggest that the reported increase in fall risk in young obese adults is not a result of impaired balance recovery ability.

The third study (Chapter 6) examined the effects of obesity on body kinematics immediately following a trip-like perturbation. Obesity was found to increase body angular velocity following a trip-like perturbation, and that increases in body angular velocity increased the probability of a failed recovery. Put another way, the results of this study suggest that when an obese and normal-weight individual are walking at similar speeds, the obese individual may be at a greater risk of falling following a trip because the obese individual will experience a greater body angular velocity. In contrast to the previous study on the effects of obesity on balance recovery using an ankle strategy, the detrimental effect of obesity on the

difficulty of recovering from a trip-like perturbation is most likely due to how mass is distributed throughout the body and not the amount of mass itself.

Finally, the fourth study (Chapter 7) utilized forward dynamic simulations to examine how obesity influences the relative strength employed during a functional task (balance recovery from a forward fall) in young adults. The obese model required greater relative hip strength than the normal-weight model to move with similar kinematics, and this is likely due to the greater proportion of mass in the trunk of the obese model. Overall, these findings suggest that young obese individuals may require greater relative strength at some joints than young normal-weight individuals to perform functional tasks, and that these differences in relative strength demands may limit functional capability among individuals who are obese.

This research has two main contributions. First, this research is one of the first to investigate balance recovery in young obese individuals. This research has provided information as to the biomechanical mechanisms that contribute to impaired balance recovery ability when using ankle strategy in young individuals who are obese. Furthermore, the results from this work also suggest that increased fall risk reported elsewhere in young obese individuals is not due to a decreased ability to recover balance by stepping. Second, this research provides valuable information towards the development of fall prevention interventions to ultimately reduce the risk of falls in the obese. For example, the results from Chapter 7 suggest that greater relative hip strength may be necessary for obese individuals to successfully recover balance. Therefore, strength training interventions can be targeted at the hip musculature to improve relative strength at this joint.

8.2 Expected publications

Chapter	Title	Targeted Journal
4	The effects of obesity on balance recovery using an ankle strategy in young adults	<i>Human Movement Science</i> *published, Vol. 30, Issue 3, p. 584-595*
5	The effects of obesity on single-step balance recovery from a forward fall in young adults	<i>Gait and Posture</i>
6	The effects of obesity on body kinematics immediately following a trip-like perturbation in young adults	<i>Journal of Biomechanics</i> (short communication)
7	The effects of obesity on relative strength used during balance recovery from a forward fall in young adults	<i>Journal of Applied Biomechanics</i>

8.3 Future Research

Further research is needed to examine other mechanisms by which obesity contributes to fall risk in young adults. It is possible that obesity may increase fall risk in young adults by increasing the likelihood of initiating a fall. For example, obese individuals walk with less hip and knee flexion (Spyropoulos et al. 1991), suggesting a smaller clearance of the foot from the ground. A smaller foot-ground clearance in obese individuals would make it more likely for the shoe to be obstructed by an elevated obstacle, indicating a higher risk of tripping (Rietdyk et al. 2005). Additionally, muscle fatigue negatively affects balance (Davidson et al. 2004; Davidson et al. 2009) and could be another contributing factor towards increased fall risk among individuals who are obese. BMI has been correlated with fatigability, with individuals with a higher BMI being associated with lower fatigue resistance (Sartorio et al. 2003; Maffiuletti et al. 2007).

Future research should also be directed towards understanding fall risk in obese older adults.

Falls account for over 60% of non-fatal injuries (~1.92 million older adults) and 45% of injury-related deaths among older adults (CDC 2007). Obese older adults would seem to be at an even greater risk of falls due to their decreased strength from sarcopenia (i.e. loss of skeletal muscle mass with age) (Janssen et al. 2002) and increased body mass from obesity. Because balance recovery is a time-critical task that involves high strength demands, future studies should examine the effects of obesity and aging on balance

recovery using a single step and balance recovery from a trip. Future research should also be directed towards understanding the relationship between walking speed and initial body angular velocity following a trip, how this relationship compares to that seen with a trip-like perturbation (Chapter 6), and how age-related changes in mass distribution also affect this relationship.

Finally, the model developed in the fourth study (Chapter 7), has the potential to explore numerous research questions. As an extension of the fourth study, the effects of obesity and perturbation magnitude on strength demands during balance recovery should be investigated. Additionally, this model can also be used to investigate the effects of decreasing mass and increasing strength on balance recovery ability in older adults to gain information towards development of fall prevention interventions for obese older adults (i.e. weight-loss, strength training).

8.4 References

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Appendix A – Institutional Review Board Approval



Office of Research Compliance
Institutional Review Board
2000 Kraft Drive, Suite 2000 (0497)
Blacksburg, Virginia 24061
540/231-4991 Fax 540/231-0959
e-mail moored@vt.edu
www.irb.vt.edu

DATE: August 3, 2007

FWA00000572 (expires 1/20/2010)
IRB # is IRB00000667

MEMORANDUM

TO: Michael L. Madigan
Sara Matrangola

Approval date: 8/3/2007
Continuing Review Due Date: 7/19/2008
Expiration Date: 8/2/2008

FROM: David M. Moore

SUBJECT: **IRB Expedited Approval:** "Effects of Body Mass Index on Balance Recovery", IRB # 07-379

This memo is regarding the above-mentioned protocol. The proposed research is eligible for expedited review according to the specifications authorized by 45 CFR 46.110 and 21 CFR 56.110. As Chair of the Virginia Tech Institutional Review Board, I have granted approval to the study for a period of 12 months, effective August 3, 2007.

As an investigator of human subjects, your responsibilities include the following:

1. Report promptly proposed changes in previously approved human subject research activities to the IRB, including changes to your study forms, procedures and investigators, regardless of how minor. The proposed changes must not be initiated without IRB review and approval, except where necessary to eliminate apparent immediate hazards to the subjects.
2. Report promptly to the IRB any injuries or other unanticipated or adverse events involving risks or harms to human research subjects or others.
3. Report promptly to the IRB of the study's closing (i.e., data collecting and data analysis complete at Virginia Tech). If the study is to continue past the expiration date (listed above), investigators must submit a request for continuing review prior to the continuing review due date (listed above). It is the researcher's responsibility to obtain re-approval from the IRB before the study's expiration date.
4. If re-approval is not obtained (unless the study has been reported to the IRB as closed) prior to the expiration date, all activities involving human subjects and data analysis must cease immediately, except where necessary to eliminate apparent immediate hazards to the subjects.

Important:

If you are conducting **federally funded non-exempt research**, this approval letter must state that the IRB has compared the OSP grant application and IRB application and found the documents to be consistent. Otherwise, this approval letter is invalid for OSP to release funds. Visit our website at <http://www.irb.vt.edu/pages/newstudy.htm#OSP> for further information.

cc: File

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VirginiaTech

Office of Research Compliance
Institutional Review Board
2000 Kraft Drive, Suite 2000 (0497)
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DATE: June 3, 2009

FWA00000572(expires 1/20/2010)

IRB # is IRB00000867

MEMORANDUM

TO: Michael L. Madigan
Sara Matrangola

Approval date: 8/3/2008
Continuing Review Due Date: 7/19/2009
Expiration Date: 8/2/2009

FROM: David M. Moore

SUBJECT: **IRB Amendment 2 Approval:** "Effects of Body Mass Index on Balance Recovery",
IRB # 07-379

This memo is regarding the above referenced protocol which was previously granted approval by the IRB on August 3, 2008. You subsequently requested permission to amend your IRB application. Since the requested amendment is nonsubstantive in nature, I, as Chair of the Virginia Tech Institutional Review Board, have granted approval for requested protocol amendment, effective as of June 3, 2009. The anniversary date will remain the same as the original approval date.

As an investigator of human subjects, your responsibilities include the following:

1. Report promptly proposed changes in previously approved human subject research activities to the IRB, including changes to your study forms, procedures and investigators, regardless of how minor. The proposed changes must not be initiated without IRB review and approval, except where necessary to eliminate apparent immediate hazards to the subjects.
2. Report promptly to the IRB any injuries or other unanticipated or adverse events involving risks or harms to human research subjects or others.
3. Report promptly to the IRB of the study's closing (i.e., data collecting and data analysis complete at Virginia Tech). If the study is to continue past the expiration date (listed above), investigators must submit a request for continuing review prior to the continuing review due date (listed above). It is the researcher's responsibility to obtain re-approval from the IRB before the study's expiration date.
4. If re-approval is not obtained (unless the study has been reported to the IRB as closed) prior to the expiration date, all activities involving human subjects and data analysis must cease immediately, except where necessary to eliminate apparent immediate hazards to the subjects.

cc: File

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MEMORANDUM

DATE: December 1, 2010

TO: Michael L. Madigan, Sara Matrangola, Kerry Costello

FROM: Virginia Tech Institutional Review Board (FWA00000572, expires October 26, 2013)

PROTOCOL TITLE: Biomechanics of Walking, Balance, and Balance Recovery

IRB NUMBER: 09-971

Effective December 18, 2010, the Virginia Tech IRB Chair, Dr. David M. Moore, approved the continuation request for the above-mentioned research protocol.

This approval provides permission to begin the human subject activities outlined in the IRB-approved protocol and supporting documents.

Plans to deviate from the approved protocol and/or supporting documents must be submitted to the IRB as an amendment request and approved by the IRB prior to the implementation of any changes, regardless of how minor, except where necessary to eliminate apparent immediate hazards to the subjects. Report promptly to the IRB any injuries or other unanticipated or adverse events involving risks or harms to human research subjects or others.

All investigators (listed above) are required to comply with the researcher requirements outlined at <http://www.irb.vt.edu/pages/responsibilities.htm> (please review before the commencement of your research).

PROTOCOL INFORMATION:

Approved as: **Expedited, under 45 CFR 46.110 category(ies) 4, 6, 7**

Protocol Approval Date: **12/18/2010** (protocol's initial approval date: **12/18/2009**)

Protocol Expiration Date: **12/17/2011**

Continuing Review Due Date*: **12/3/2011**

*Date a Continuing Review application is due to the IRB office if human subject activities covered under this protocol, including data analysis, are to continue beyond the Protocol Expiration Date.

FEDERALLY FUNDED RESEARCH REQUIREMENTS:

Per federally regulations, 45 CFR 46.103(f), the IRB is required to compare all federally funded grant proposals / work statements to the IRB protocol(s) which cover the human research activities included in the proposal / work statement before funds are released. Note that this requirement does not apply to Exempt and Interim IRB protocols, or grants for which VT is not the primary awardee.

The table on the following page indicates whether grant proposals are related to this IRB protocol, and which of the listed proposals, if any, have been compared to this IRB protocol, if required.

*Date this proposal number was compared, assessed as not requiring comparison, or comparison information was revised.

If this IRB protocol is to cover any other grant proposals, please contact the IRB office (irbadmin@vt.edu) immediately.

cc: File

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Appendix B – Informed Consent

VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY

Informed Consent for Participants In Research Projects Involving Human Subjects

Title of the Research Study

Biomechanics of balance recovery

Investigators

Michael L. Madigan, Ph.D. 231-1215

Department of Engineering Science and Mechanics

Sara Matrangola, B.S.

VT-WF School of Biomedical Engineering and Sciences

I. Purpose of this Study

The purpose of this research study is to investigate selected biomechanical factors during recovery from an induced forward fall. Fall-related injuries are a major medical problem that costs billions of dollars annually in treatment and care. The findings from this research study will contribute to the pool of fall-related knowledge that can be used to develop intervention techniques to prevent falls.

II. Procedures

Sixty subjects between the ages of 21-65 will be used.

The study will take place in the Musculoskeletal Biomechanics Lab of the Department of Engineering Science and Mechanics (111 Norris Hall). You will be asked to attend one or two sessions in the lab. If you are asked to participate in a second session, you will repeat this same procedure with extra weight added to your body.

Upon arriving, you will be briefed of the study protocol, asked if you have any questions, and asked to sign this informed consent form. Three different laboratory-based measures of balance will be performed. These will include: 1) standing as still as possible, 2) maintaining balance after being softly bumped, and 3) maintaining balance after being released from a small forward lean. At no time throughout these tests will you fall to the ground. In fact, you will not even need to take a step to maintain your balance during most trials. During a few of the trials, you might need to take a single step to maintain your balance. You will be asked to perform each of these tests several times.

During testing, you will be asked to wear several non-invasive position sensors placed on your body using double-sided tape by Ms. Matrangola. Markers will be placed on both the sides of your body at your toes, ankles, knees, hips, shoulders, elbows, wrists, and head. These sensors allow the researchers to analyze your movement patterns during testing.

These tests will take approximately 2 hours to complete.

III. Risks

The risks involved in this study are minimal because the tasks will require little effort to maintain balance.

6/2/2009

Virginia Tech Institutional Review Board: Project No. 07-379

Approved June 3, 2009 to August 2, 2009

IV. Benefits

The scientific community will benefit through the additional information that is expected to result from the completion of this study. This information will contribute to the foundation of fall-related biomechanical knowledge that will eventually be used to develop intervention techniques to prevent falls and fall-related injuries.

No promise or guarantee of benefits has been made to encourage you to participate.

V. Extent of Anonymity and Confidentiality

The results of this research study may be presented at meetings or in publications. Your identity will not be disclosed in those presentations. All subjects will be identified based only on their gender and a unique identifying number. Only the investigator will have access to these identifying numbers.

VI. Compensation

Subjects will be paid \$10/hour for their participation.

VII. Freedom to Withdraw

Your participation in this research study is voluntary. Refusal to participate will involve no penalty or loss of benefits to which you are otherwise entitled. You are free to withdraw from the study at any time without penalty.

VIII. Approval of Research

This research project has been approved, as required, by the Institutional Review Board for Research Involving Human Subjects at Virginia Polytechnic Institute and State University, by the Department of Engineering Science and Mechanics.

IX. Subject Responsibilities

I voluntarily agree to participate in this study.

X. Subject's Permission

I have read and understand the Informed Consent and conditions of this project. I have had all my questions answered. I hereby acknowledge the above and give my voluntary consent:

Subject signature

Date

Witness

Date

Should I have any pertinent questions about this research or its conduct, and research subjects' rights, and whom to contact in the event of a research related injury to the subject, I may contact:

<u>Investigator:</u>	Michael Madigan, PhD	231-1215	mlmadigan@vt.edu
<u>ESM Head:</u>	Ishwar Puri, PhD	231-6651	ikpuri@vt.edu
<u>Chair, IRB:</u>	David M. Moore, DVM	231-4991	moored@vt.edu

6/2/2009

Virginia Tech Institutional Review Board: Project No. 07-379

Approved June 3, 2009 to August 2, 2009

VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY

Informed Consent for Participants In Research Projects Involving Human Subjects

Title of the Research Study

Biomechanics of Walking, Balance, and Balance Recovery

Principal Investigators

Michael L. Madigan, Ph.D. 231-1215
Engineering Science and Mechanics

Sara Matrangola, M.S.
VT-WF School of Biomedical Engineering and Sciences

Kerry Costello, B.S.
VT-WF School of Biomedical Engineering and Sciences

I. Purpose of this Study

The purpose of this research study is to investigate biomechanical relationships between walking, balance during quiet standing, and balance recovery from a postural perturbation. Fall-related injuries are a major medical problem that costs billions of dollars annually in treatment and care. The findings from this research study will contribute to the pool of fall-related knowledge that can be used to development intervention techniques to prevent falls.

Forty subjects aged 18-28 will be recruited. Individuals with prior medical history of neck and spine injury, osteoporosis, or pinched nerves in the cervicle spine (neck), will be excluded from the study.

II. Procedures

The study will take place in the Kevin P. Granata Biomechanics Lab (208 Norris Hall). You will be asked to complete three experimental sessions, each of which will take approximately 1.5 hours to complete. Upon arriving in the lab, you will be briefed of the study protocol, asked if you have any questions, and asked to sign this informed consent form.

First experimental session:

You will first will be asked to change your clothing into a tank-top and biker shorts (provide by the lab), and to don a torso harness and belt. Reflective markers will then be placed on your body in specific anatomical locations for the researchers to measure body movements during walking and balance recovery.

The first experimental session will focus on balance recovery and walking. Trials will start by having you lean forward while a rope holds you in this position. After a random delay, the rope will be released, you will fall forward and attempt to recover your balance with a single step of your right foot. Trials will begin by leaning you at a small lean angle. After a successful balance recovery, another trial will be performed at a slightly larger angle. After a failed recovery, you will be provided a second attempt. This process will be repeated until you fail twice at the same lean angle.

After the lean trials, you will then be asked to simply walk along a walkway several times as described by the investigator.

Second experimental session:

The second experimental session will focus on balance during quiet standing. After strapping you to a board so that you can only move at your ankles, you will be asked to try to stand as still as possible for 30 seconds. This will be repeated under four different experimental conditions as described by the investigator.

Third experimental session:

You will be asked to change your clothing into a tank-top and biker shorts (provide by the lab), and to don a torso harness and belt. Reflective markers will then be placed on your body in specific anatomical locations for the researcher to measure body movements during walking and balance recovery.

The third experimental session will use a different type of postural perturbation as that used in the lean trials of session one. Trials will start by having you stand upright on a platform that can translate forward. Trials will begin with the experimenter pushing the platform forward smoothly at a slow walking speed while you maintain an upright standing posture on the platform. After pushing the platform for about 15 feet, it will impact an stopper anchored in the floor. Your momentum will carry you forward, and you will attempt to recover your balance with a single step. After a successful balance recovery with a single step, another trial will be performed while the platform is pushed slightly faster. After a failed recovery, another attempt will be performed while the platform is pushed at the same speed. This process will be repeated until you fail to recover your balance with a single step three times at the same speed.

III. Risks

The risks involved in this study are minimal because 1) you will wear a harness suspended from the ceiling during balance recovery trials of session one, 2) safety straps will prevent a fall while you are strapped to the backboard, and 3) a spotter will provide assistance to you during the moving platform trials of session three. These safety mechanisms will prevent a fall to the ground in the event that you lose your balance or are unable to regain your balance. The other task we are investigating (walking) provides effectively no risk for injury.

IV. Benefits

The scientific community will benefit through the additional information that is expected to result from the completion of this study. This information will contribute to the foundation of fall-related biomechanical knowledge that will eventually be used to development intervention techniques to prevent falls and fall-related injuries.

No promise or guarantee of benefits has been made to encourage you to participate.

V. Extent of Anonymity and Confidentiality

The results of this research study may be presented in reports, publications, and presentations. Subject identity will not be disclosed in any situation. Subjects will only be identified using a unique identifying number assigned during your experiment.

Experiments will be video taped to assist with our analysis, and possibly to show in a report, publication, or presentation. The tapes will be maintained under the supervision of the project PI's, stored in a laboratory with restricted access, and kept for the foreseeable future for documentation purposes.

It is possible that the Institutional Review Board (IRB) may view this study's collected data for auditing purposes. The IRB is responsible for the oversight of the protection of human subjects involved in research.

VI. Compensation

You will be paid \$10/hour for your participation. Any expenses accrued for medical treatment following participation in this experimental will be your responsibility and not that of the research project, research team, or Virginia Tech.

VII. Freedom to Withdraw

You are free to withdraw from the study at any time without penalty. If you choose to withdraw, you will be compensated for the portion of the study that you completed.

VIII. Subject's Responsibilities

I voluntarily agree to participate in this study. I have the following responsibilities: accurately report my age, gender, and history of musculoskeletal injuries.

IX. Subject's Permission

I have read the Consent Form and conditions of this project. I have had all my questions answered. I hereby acknowledge the above and give my voluntary consent:

Subject signature

Date

Witness

Date

Should I have any pertinent questions about this research or its conduct, and research subjects' rights, and whom to contact in the event of a research related injury to the subject, I may contact:

<u>Investigator:</u>	Michael Madigan, PhD	231-1215	mlmadigan@vt.edu
<u>ESM Head:</u>	Ishwar Puri, PhD	231-6651	ikpuri@vt.edu
<u>Chair, IRB:</u>	David M. Moore, DVM	231-4991	moored@vt.edu