# MUSCLE ADAPTATION AND CHALLENGES TO MOTOR STABILITY DURING WALKING IN YOUNG HEALTHY INDIVIDUALS

by

Jason Marc Schoenfeld

A thesis submitted to the Faculty of the University of Delaware in partial fulfillment of the requirements for the degree of Honors Bachelor of Arts in Biological Sciences with Distinction.

Spring 2009

**MUSCLE ADAPTATION AND CHALLENGES TO MOTOR STABILITY DURING WALKING IN YOUNG HEALTHY INDIVIDUALS**

by

Jason Marc Schoenfeld

Approved: _____
Katherine Rudolph, Ph.D.
Professor in charge of thesis on behalf of the Advisory Committee

Approved: _____
Darcy Reisman, Ph.D.
Committee member from the Department of Physical Therapy

Approved: _____
James Glancey, P.E.
Committee member from the Board of Senior Thesis Readers

Approved: _____
Alan Fox, Ph.D.
Director, University Honors Program

## ACKNOWLEDGMENTS

I would like to thank my advisor, Dr. Katherine Rudolph, for her guidance, patience, and understanding during my time as an undergraduate researcher.

I would not have completed this project without her support, and I am truly grateful for the countless hours that she spent working with me.

Deepak Kumar, a Ph. D candidate, is an invaluable mentor. Deepak taught me so much about science, research, and life in general, and I will never forget his truly wise advice.

I thank Ph. D student Josh Winters and graduate students John Battles and Steve Thomas for helping me with collecting and processing data.

I greatly appreciate Dr. Buzz Swanik's and Matt Hinsey's help with stiffness and proprioception testing.

Dr. James Glancey and Dr. Darcy Reisman committed valuable time to serve on my thesis committee. I thank them for their insightful comments and questions.

The Undergraduate Research Program also provided essential support for my project. I was able to obtain funding for my research and honed my presentation skills through their services.

Last but certainly not least, I would like to thank my family for their love, support, and encouragement throughout the years. I would not be where I am today without them.

## TABLE OF CONTENTS

## LIST OF TABLES

## LIST OF FIGURES

## ABSTRACT

It is well established that humans adapt to sudden or unexpected perturbations through a posture control system that has vestibular, visual, proprioceptive, and sensory components. Older adults, however, are not able to generate postural responses as quickly as young adults when environmental conditions or sensory cues change rapidly. The slower postural responses may arise from progressive degeneration of the nervous system as a result of normal aging. To investigate the neuromuscular responses at the knee to perturbations during walking, ten "young healthy" (mean= 21.9 years; range= 20-23; sex= 4 males) and ten healthy "older adults" (mean= 63 years; range= 52-73; sex= 7 males) walked for 10 level trials across a 13 m walkway. Next, they walked for 30 trials across a custom-built, movable platform (NSK Ltd, Tokyo, Japan) that translated laterally when stepped upon. EMG and knee kinematic data were collected. Subjects also underwent knee proprioception and stiffness testing on a custom built Stiffness and Proprioception Assessment Device (SPAD). There were no group differences in the magnitude of muscle activation in response to the first perturbation, or in the rate of adaptation over the first five trials. However, there were differences in the strategies used by the young subjects and the older adults. The younger subjects walked more quickly and moved their knees faster during the perturbation, whereas the older subjects did not. In younger subjects, the quadriceps and hamstring muscle activation during all three phases of stance were related to the knee kinematics, whereas in older subjects, the gastrocnemius muscle activation during the preparation phase was the only muscle

response related to knee kinematics. These data suggest that normal aging is associated with changes in knee control strategies. Whether those strategies are related to other conditions related to age, such as knee osteoarthritis, warrants further investigation.

# Chapter 1

# INTRODUCTION

In humans, balance, the equilibrium condition whereby net external forces and moments on the body approach zero, is transient even during quiet standing (Pai et al. 2007). As a result, stability, a person's ability to restore balance following an external perturbation (such as a lateral translation during walking), poses a difficult challenge to humans (Pai et al. 2007). Stability during locomotion-dynamic stability- is the control of the center of mass within a changing base of support, and requires effective proactive and reactive recovery strategies when the base of support is altered (Marigold et al. 2002). It is well established that humans adapt to sudden or unexpected external perturbations through a posture-control system that regulates body position in order to maintain orientation and balance (Mcilroy et al. 1995; Horak et al. 1997; Marigold et al. 2002). The posture-control system is based on the integration of vestibular, visual, proprioceptive, and sensory information by the central nervous system (CNS) (Lacour et al. 2008).

The CNS appears to control the reaction to an unexpected external perturbation with early muscle onset latencies from the perturbed leg. Several investigators have reported that the shorter muscle latencies are in the range of 70-180 msec post-perturbation. The 70-180 msec onset latency suggests a polysynaptic (long-loop) reflex that involves the brain (Horak et al. 1986; Marigold et al. 2002; Nielson et al. 2002). The initial muscular response, the purpose of which is to correct the ongoing movement and ultimately prevent a fall, arises from afferent neural signaling

and subsequent efferent motor activation (Nielson et al. 2002). Recent studies have shown that muscle onset latencies typically lessen as an adaptation to maintain postural stability following repeated exposures of the same perturbation (Horak et al. 1997; Marigold et al. 2002; Pai et al. 2003).

Over time, a newly acquired, predominantly predictive form of adaptive control emerges. Such control exhibits feed-forward behavior and relies on acquired strategies to proactively counter the perturbation (Schiedt et al. 2001). Afferent feedback, however, continues to play a role in fine-tuning the movement. In light of this aforementioned mechanism, the rate of adaptation to external perturbations has been used to evaluate how sensory and motor systems respond to challenges to stability (Horak et al. 1997; McGibbon et al. 2004; Pai et al. 2007)

Older adults are not able to integrate sensory information and generate an appropriate postural response as quickly as young adults when environmental conditions or sensory cues change rapidly (Woolacott et al. 1986; McGibbon et al. 2004; O'Connor et al. 2007). This slower rate of adaptation may arise from progressive degeneration of the nervous system as a result of normal aging. For example, people over age 60 have been found to have a decreased number of motor neurons (Tomlinson et al. 1977). Kawamura et al. (1977) also found a clear age-related reduction in the numbers of large and intermediate myelinated ventral root fibers (Kawamura et al. 1977).

A decline in sensory and proprioceptive ability during the course of normative aging has also been reported (Bergman et al. 1999; Li et al. 2002). The resulting deficits in afferent pathways lead to ineffective efferent neuromuscular strategies and joint instability in the elderly (Johansson et al. 1991; Dietz 1992;

Gardsden et al. 1999). Older people respond to a perturbation with greater co-contraction of the lower extremity muscles, which may be a compensation for a reduced stretch sensitivity of the postural response (Dietz 1992; Woollacott et al. 1998). Proprioceptive input has also been found to regulate muscle activation and muscle stiffness during walking (Dietz 1992). A correlation between proprioception and stiffness at the ankle has been found in young, healthy individuals, which suggests that mechanoreceptors play a role in the regulation of stiffness (Sjolander et al. 2002).

Muscle stiffness, which is composed of instrinsic stiffness and reflex mediated stiffness, has been traditionally quantified in the laboratory using high velocity, small amplitude perturbations under highly controlled conditions (Zhang et al. 1998; Zhang et al. 2001; McHugh et al. 2004). Intrinsic stiffness is mediated by the viscoelastic properties of the muscle, passive joint structures, and existing actin-myosin cross-bridges (Mirbagheri et al. 2000). Reflex mediated stiffness, on the other hand, is a time-delayed, restorative force that is determined by the excitability of alpha motorneurons (Moorehouse et al. 2006). Intrinsic muscle stiffness, the first line of defense following a perturbation, depends on the level of muscle activation. Reflexive stiffness does not intervene in time to protect the joint from injury, but plays a role in programming the stiffness through afferent information from previous experience (Johansson et al 1991). Diminished afferent input could therefore cause an alteration in the regulation of muscle stiffness, and possibly lead to the failure of protective neuromuscular reflexes and injury.

Our lab has developed a perturbation paradigm in which the support surface translates laterally during walking. Knee kinematic and electromyographic data are collected to compare the neuromuscular response of healthy older adults with

young subjects. Proprioception and short range active muscle stiffness data are also collected. This study investigates the effect of age on neuromuscular responses in healthy young versus older adult individuals. We hypothesize that the muscle responses of older adults will adapt more slowly to repeated perturbations during walking and will display greater gait deviations than young subjects, and that the rate of adaptation will be related to the threshold to detect passive motion and short range active muscle stiffness. Such findings would provide evidence that healthy aging is associated with alterations in neuromuscular control that may contribute to the development of osteoarthritis in the adult population.

# Chapter 2

# MATERIALS AND METHODS

## 2.1 Subjects

Ten "young healthy" (mean= 21.9 years; range= 20-23; sex= 4 males) and ten healthy "older adults" (mean= 63 years; range= 52-73; sex= 7 males) were recruited from the local community. To be characterized as "healthy" for this study, subjects could not have been diagnosed with knee OA, undergone a knee operation, or been suffering from chronic knee pain. Subjects also had to be free of any other significant health problems. All subjects signed an informed consent approved by the Institutional Review Board.

## 2.2 Motion Analysis

The position of reflective markers placed on the pelvis and both legs was tracked by an 8-camera, passive, three dimensional motion analysis system (VICON 512, Vicon Peak, Oxford, UK) at 120 Hz. The retroflective (15.5 mm) markers were placed on the pelvis and posterior aspects of the thigh and shank, and individually placed over the bilateral greater trochanters, the medial and lateral knee joint, the medial and lateral malleoli, and the top and bottom heel. Marker data were filtered at 6 Hz with a fourth order, phase-corrected Butterworth filter.

## 2.3 Electromyograpgy (EMG)

EMG data were sampled at 1080 Hz and collected simultaneously with preamplified surface electrodes (MA-16, Motion Lab Systems, Baton Rouge, LA) with an 18-mm interelectrode distance. Ground reaction force data were sampled from a force platform (Bertec, Washington, Ohio). EMG signals were recorded from the medial quadriceps (VM), lateral quadriceps (VL) medial hamstrings (MH), lateral hamstrings (LH), medial gastrocnemii (MG), and lateral gastrocnemii (LG).

## 2.4 Testing Procedure- Level Walking

Subjects walked at a self-selected speed along a 13-m walkway. The starting position of the subject was adjusted appropriately such that the foot contacted the center of the force plate by the test limb without adjusting the stride to do so. Walking velocity was monitored with 2 photoelectric beams to ensure that speed did not vary more than 5 percent from the self-selected speed. Subjects completed 10 trials.

## 2.5 Testing Procedure- Disturbed Walking Paradigm

Subjects walked across a custom-built, movable platform (NSK Ltd, Tokyo, Japan) that was located mid-way through the 13-m walkway. When unlocked, as the subject stepped on a switch mat on the surface of the platform, a signal was sent to the platform which translated laterally (5.8 cm at 40 cm/s). Subjects completed 30 of these lateral walking trials. Subjects observed the translation before walking over it, and were aware that the translation would occur.

## 2.6 Testing Procedure- Knee Joint Proprioception and Knee Stifness

Knee joint proprioception and stiffness were measured using a custom built Stiffness and Proprioception Assessment Device (SPAD). The subjects were seated with their back supported and hip flexed 80°, and the trunk and thigh were securely stabilized with straps. The mechanical axis of the SPAD was aligned with the anatomic knee axis, and the adapter arm was adjusted so that the distal pad was positioned approximately two inches proximal to lateral malleolus on the shin. The lower leg was secured in a pneumatic sleeve to provide constant pressure on the skin, which minimized cutaneous cues. The sleeve was attached to the drive shaft of a brushless servomotor. The SPAD device was operated using a personal computer with a customized LabVIEW (LabVIEW 8.2, National Instruments, Austin, TX) virtual instrument and motor control software program. The SPAD recorded torque and position data at 2000 Hz.

Threshold to detect passive motion (TTDPM) was tested at 15 degrees and 45 degrees from the end of the subject's available knee extension range of motion. Both flexion and extension movements were measured at each angle. Headphones and a blindfold were used to eliminate auditory and visual cues. In each trial, the subject was tapped on the shoulder to notify him/her that the device would start moving in the next 10 seconds. At a random interval within the 10 seconds, the SPAD passively rotated the lower leg of the subject at a velocity of 0.5°/sec and acceleration of $100°/sec^2$. When the subject perceived knee movement, he or she pressed a hand-held switch to disengage the motor. The angle at which the subject detected motion was recorded. This procedure was repeated 3 times for each condition and the TTDPM was averaged across the 3 trials. A greater angle indicated worse proprioception.

With the subject in the same position in the SPAD, Joint Repositioning Error (JRE) was measured at 15 degrees and 45 degrees from the end of the subject's available knee extension range. With head phones removed and blindfold in place, the subject's leg was moved to a baseline position of 15 or 45 degrees from the end of the available range of motion. Next, the SPAD passively rotated the lower leg of the subject at a velocity of 0.5°/sec and acceleration of 100°/sec$^2$ to a target angle, and held there for several seconds. The subject's leg was then returned to the baseline position. The SPAD then moved the subject's leg toward the target angle, and the subject pressed a hand-held switch to disengage the motor when the subject perceived that his or her knee had reached the target angle. The difference between the actual and perceived target angles was recorded. The procedure was repeated at both baseline angles for both flexion and extension movements.

Knee stiffness was measured with the subject in the same position in the SPAD, but the pneumatic sleeve was replaced by a vacuum splint that created rigid interface between the limb and the SPAD to prevent any absorption of the torque by soft tissues of leg during testing. The weight of the relaxed limb with the subject's knee flexed 30 degrees was used for gravity correction. Stiffness was measured during a relaxed condition (passive stiffness condition) and with muscles contracted (active stiffness condition). After several sub-maximal warm-up contractions, three maximum voluntary isometric contractions (MVIC) for quadriceps were performed with the knee flexed 30 degrees. Subjects were encouraged verbally to produce a maximum effort, and the measured torque was shown on a digital display for visual feedback. The highest recorded torque was used to calculate the 30% MVIC, which was the target torque that the subjects were asked to maintain during the active

stiffness condition. Perturbations were applied to the knee using a 20 degree movement into flexion (angular velocity of 100°/sec; acceleration of 3000°/sec$^2$) for quadriceps stiffness. For the active stiffness tests, the subject was asked to hold a force level equivalent to 30% MVIC. Each condition was repeated 3 times, and the average of the 3 trials was used in the analysis.

### 2.7 Data Management

Sagittal and frontal plane knee joint angles were calculated (Visual3D, C-Motion, Rockville, MD) over three intervals: pre-heel strike (100 msec before heel contact), weight acceptance (from initial contact through peak knee flexion), and midstance (from peak knee flexion angle through peak knee extension). Data were time normalized and averaged across the 10 lateral trials.

EMG data were also processed using custom-written software. A linear envelope was created with full wave rectification and filtering with a 60 Hz low pass, phase-corrected, eighth-order Butterworth filter. The linear envelope was normalized by peak EMG activity that had been recorded during maximum voluntary contractions. EMG activity was averaged across the 3 intervals. The difference between the magnitude of muscle activation recorded during the first perturbation trial and level walking (Δ P1-Level) was calculated and used in the analysis. The rate of change in the magnitude of muscle activation across the first 5 perturbation trials was operationally defined as rate of adaptation.

Joint stiffness was calculated as the slope of the torque versus position curve. Two stiffness values were determined for each condition. The first stiffness value, called the short range stiffness, was calculated as the slope of the curve from movement onset to the point where the adapter and shank reached a constant velocity.

The short range stiffness indicated the intrinsic component of total stiffness. The second stiffness value, called the total stiffness, was the slope from movement onset until the end of the movement. All stiffness values were corrected for gravity and the stiffness of the adapter arm, so that the final value represented stiffness generated only by the limb of the subject.

### 2.8 Statistical Analysis

Groups means and standard deviations were compared between the groups with independent samples t-tests (SPSS 16.0, SPSS Inc., Chicago, IL). Significance was established when $p \leq 0.05$. Relationships among variables were determined by calculating the correlation of TTDPM, JRE and Stiffness values with kinematic and EMG variables. Correlations that were statistically significant were entered into regression models to determine which variables predicted the responses to the successive perturbation trials. In a secondary analysis, the frequency of responses to the first perturbation trial were evaluated using $\chi^2$.

## Chapter 3

## RESULTS

No group differences were observed in BMI (p= 0.399), short range quadriceps stiffness (p= 0.065), quadriceps strength (p= 0.223), or quadriceps strength normalized to BMI (p= 0.053) (Table 1). There were no group differences in level walking speed (p= 0.521) or walking speed during the first perturbed trial (p= 0.963) (Table 1). There were also no group differences in kinematics during walking (Table 2).

**Table 1 Results of Independent Samples T-Tests for BMI, short range quadriceps stiffness, quadriceps strength, level walking speed, and first perturbation walking speed between young and older subjects. Mean (SD) are reported**

| Variable | Young | Old | p-value |
|---|---|---|---|
| BMI | 25.09 (3.38) | 26.48 (3.82) | 0.399 |
| Short Range Active Quadriceps Stiffness | 7.19 N·M/º (2.17) | 5.47 N·M/º (1.53) | 0.065 |
| Quadriceps Strength | 347.82 N (112.43) | 288.94 N (86.86) | 0.223 |
| Quadriceps Strength Normalized to BMI | 13.76 N (3.79) | 10.72 N (2.31) | 0.053 |
| Level Walking Speed | 1.45 m/s (0.12) | 1.49 m/s (0.17) | 0.521 |
| First Perturbation Walking Speed | 1.51 m/s (0.11) | 1.51 m/s (0.18) | 0.963 |

**Table 2 Results of Independent Samples T-Tests for kinematics during walking between young and older subjects. Mean (SD) are reported**

| Variable | Young | Old | p-value |
|---|---|---|---|
| **PREP PHASE** | | | |
| Knee Flexion Angle at Initial Contact (Level) | - 8.15º (3.83) | - 6.27º (3.80) | 0.285 |
| Knee Flexion Angle at Initial Contact (Δ P1-Level) | 1.58º (2.57) | 0.89º (1.81) | 0.493 |
| Knee Flexion Angle at Initial Contact (Adaptation Over First Five Trials) | 0.32º (0.58) | 0.003º (0.64) | 0.267 |
| | | | |
| **WEIGHT ACCEPTANCE** | | | |
| Knee Flexion Angle at Loading Response (Level) | -20.79º (4.72) | -20.79º (4.70) | 1.000 |
| Knee Angular Velocity During Loading Response (Level) | -144.48º/sec (30.88) | -159.81º/sec (23.43) | 0.227 |
| Knee Angular Velocity During Loading Response (Δ P1-Level) | -24.48º/sec (23.41) | -7.09º/sec (23.02) | 0.111 |
| | | | |
| **MID-STANCE** | | | |
| Knee Flexion Angle at Midstance (Level) | -2.06º (4.02) | -2.14 º (3.94) | 0.967 |
| Knee Angular Velocity During Midstance (Level) | 110.60º/sec (32.81) | 105.16 º/sec (23.93) | 0.677 |
| Knee Angular Velocity During Midstance (Δ P1-Level) | - 4.44º/sec (23.01) | - 4.75 º/sec (35.90) | 0.982 |

No group differences were observed in muscle activation in response to the first perturbation (Δ P1-Level), or in the rate of adaptation over the first five trials (Table 3). The only significant difference in proprioception between the groups was that at 15 degrees of extension, the young subjects had a lower Joint Repositioning Error ($p= 0.049$) (Table 4).

**Table 3** **Results of Independent Samples T-Tests for Δ P1-Level and adaptation to the perturbation over the first five trials between young and older subjects. Values are for the midstance interval of the gait cycle. Mean (SD) are reported**

| **Δ P1-Level** | | | | | **Adaptation** | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | | |
| **Muscle** | **Young** | **Old** | **p-value** | | **Muscle** | **Young** | **Old** | **p-value** |
| LG | 0.14 (0.18) | 0.10 (0.12) | 0.649 | | LG | -0.02 (0.03) | -0.01 (0.02) | 0.504 |
| MG | 0.03 (0.07) | 0.08 (0.11) | 0.257 | | MG | 0.00 (0.02) | -0.01 (0.03) | 0.373 |
| LH | 0.10 (0.12) | 0.12 (0.13) | 0.742 | | LH | -0.01 (0.02) | -0.02 (0.03) | 0.430 |
| MH | 0.07 (0.09) | 0.11 (0.13) | 0.404 | | MH | -0.01 (0.01) | -0.02 (0.03) | 0.285 |
| VL | 0.15 (0.13) | 0.12 (0.10) | 0.536 | | VL | -0.03 (0.03) | -0.02 (0.01) | 0.433 |
| VM | 0.13 (0.15) | 0.17 (0.16) | 0.546 | | VM | -0.02 (0.03) | -0.03 (0.03) | 0.511 |

**Table 4 Results of Independent Samples T-Tests for proprioception data between young and older subjects. Mean (SD) are reported**

| Variable | Young | Old | p-value |
|---|---|---|---|
| TTDPM- 15º Flexion | 0.68 (0.14) | 0.81 (0.27) | 0.200 |
| TTDPM- 15º Extension | 0.71 (0.11) | 0.73 (0.24) | 0.822 |
| TTDPM- 45º Flexion | 0.73 (0.21) | 0.79 (0.14) | 0.489 |
| TTDPM- 45º Extension | 0.81 (0.27) | 1.45 (1.18) | 0.111 |
| JRE- 15º Flexion | 2.35 (1.41) | 3.03 (1.49) | 0.309 |
| JRE- 15º Extension | 1.16 (0.88) | 1.91 (0.69) | ***0.049** |
| JRE- 45º Flexion | 2.60 (1.75) | 2.44 (1.83) | 0.845 |
| JRE- 45º Extension | 2.01 (1.38) | 2.29 (1.51) | 0.674 |

Amongst the young subjects, only one person slowed his/her walking speed during the first perturbation trial as compared to level walking ($X^2$= 6.4, p= 0.011). Amongst the older subjects, however, four people slowed their walking speed during the first perturbation ($X^2$=0.400, p= 0.527). Amongst the younger subjects, two people flexed their knees faster during loading response of the first perturbation trial than during level walking ($X^2$= 3.600, p= 0.058). Amongst the older subjects, however, four people flexed their knees more quickly during loading response of the first perturbation trial than during level walking ($X^2$= 0.400, p= 0.527). Amongst the

younger subjects, three people extended more quickly during midstance of the first perturbation trial than during level walking ($X^2$= 1.600, p= 0.206). Amongst the older subjects, five people extended more quickly during midstance of the first perturbation trial than during level walking ($X^2$=0.000, p=1.000) (Figure 1).

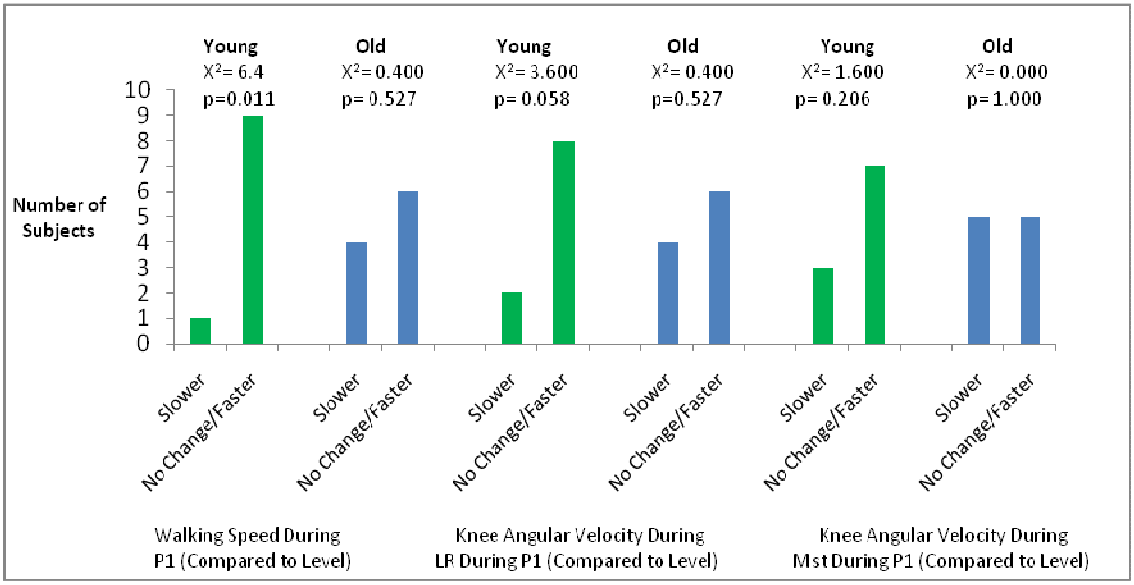


**Figure 1** **Comparison of the walking speed, knee angular velocity during loading response, and knee angular velocity during midstance between level walking and the first perturbation trial (P1). Subjects were categorized as either moving "slower" or "no change/faster" during P1**

The knee flexion angle amongst young and older subjects as an initial response to the perturbation was then correlated with several proprioceptive, stiffness, and EMG variables. These variables included Joint Motion Angle (JMA) in 15 and 45 degrees of flexion, Joint Repositioning Error (JRE) in 15 and 45 degrees of flexion,

short range quadriceps stiffness, and the initial response of LG, LH, MH, MH, VL, and VM during each respective gait interval (Pre-HS, loading response, and midstance) (Table 5).

**Table 5** **Young and older healthy subjects' knee flexion angle Δ P1-Level correlated with Threshold to Detect Passive Motion (TTDPM) in 15 and 45 degrees of flexion, Joint Repositioning Error (JRE) in 15 and 45 degrees of flexion, short range quadriceps stiffness, quadriceps strength normalized to BMI, and the initial response of LG, LH, MH, MH, VL, and VM during each respective gait interval (Pre-HS, loading response, and midstance). Correlations approaching significance (p≤ 0.10) are shown below (M= Medial; L= Lateral; G= Gastrocnemius; H= Hamstring; TTDPM= threshold to detect passive motion)**

| Knee Flexion Angle-Initial Response at: | Young | Old |
|---|---|---|
| Initial Contact (IC) | *VM_Pre-Hs_(Δ P1-Level) **r= - 0.711, p = 0.021** | *MG_Pre-Hs__(ΔP1-Level) **r= - 0.711, p= 0.032**<br><br>*LG_Pre-Hs__(ΔP1-Level) **r= - 0.666, p= 0.05** |
| Loading Response (LR) | *VM_LR_(Δ P1-Level) **r= 0.595, p= 0.070**<br><br>*LG_LR__(Δ P1-Level) **r= - 0.576, p= 0.082**<br><br>*TTDPM_45 Flexion **r= 0.574, p= 0.082** | No Correlations |
| Midstance (Mst) | *LH_Mst__(Δ P1-Level) **r= - 0.720, p= 0.019**<br><br>*MH_Mst__(Δ P1-Level) **r= - 0.645, p= 0.044**<br><br>*VL_MSt__(Δ P1-Level) **r= - 0.671, p=0.034**<br><br>*VM_MSt__(Δ P1-Level) **r= - 0.710, p= 0.022** | No Correlations |

Significant correlation values or those approaching significance were then entered into a regression analysis. In young subjects, 50.5% of the variance in knee flexion angle at initial contact in the initial response to the perturbation is explained by the initial response of VM during pre-heel strike ($r^2$= 0.505, p=0.021). In older subjects, 64.7 % of the variance in knee flexion angle at initial contact is explained by the initial response to the perturbation of MG and LG ($r^2$= 0.647, p= 0.026). During loading response in young subjects, 86.8% of the variance in the initial response to the perturbation of the peak knee flexion angle is explained by the initial response of LG and VM, and by TTDPM at 45 degrees flexion ($r^2$= 0.868, p=0.005). During midstance in young subjects, 51.8% of the variance in peak knee flexion during is explained by the initial response of LH ($r^2$= 0.518, p= 0.019) (Table 6).

**Table 6 Regression analysis for significant or approaching significant correlation values of proprioception, neuromuscular, and joint position responses to the perturbation**

| Knee Flexion Angle-ΔP1-Level at: | Young | Old |
|---|---|---|
| Initial Contact | *VM_Pre-Hs_(ΔP1-Level) **$r^2$= 0.505, p= 0.021** | *MG_Pre-Hs_ (ΔP1-Level) *LG_Pre-Hs _(ΔP1-Level) **$r^2$= 0.647, p= 0.026** |
| Loading Response | *VM_LR_ (ΔP1-Level) *LG_LR_ (ΔP1-Level) *TTDPM_45 Flexion **$r^2$= 0.868, p= 0.005** | None |
| Midstance | *LH_Mst_ (ΔP1-Level) **$r^2$= 0.518, p= 0.019** | None |

# Chapter 4

# DISCUSSION

The results of this study showed few differences between young and older groups in their knee joint proprioception, knee stiffness, initial muscle and knee kinematic responses to the perturbation, or in their rate of adaptations. The only group difference in proprioception was observed in the lower joint repositioning error in the young subjects when they repositioned the joint into extension close to the end of the range of knee motion. No differences were observed when the subjects repositioned the joint into extension from a 45 degree flexed position or when repositioning the knee into flexion from the same starting positions. The lack of group differences was somewhat surprising, given that neuromuscular changes are known to occur at ages as young at 40 years. In particular, previous work has shown that older adults are not able to generate an appropriate postural response as quickly as young adults when their environment is altered (Woolacott et al. 1986; McGibbon et al. 2004; O'Connor et al. 2007). However, it is possible that the age of our older subjects was not advanced enough to detect age related differences. The average age of members in the older group was 63 years, and their ages ranged from 52-73 years. Tomlinson et al. (1977) found changes in the number of motor units in people over age 60, but pronounced changes may not occur in people younger than 60 years of age. Including subjects under 60 may have masked true differences in the groups in this study. The range of ages of the older adults included in this study related to some inclusion criteria of a related study on neuromuscular patterns in people with knee osteoarthritis, for which

the older adults were healthy controls. The age range of the healthy subjects however, may have limited the ability to detect differences in neuromuscular responses related to age.

Despite the lack of group differences in the variables in the primary analysis, several observations were made that comprised a secondary data analysis in which some group differences were observed.

Although there was no difference in the mean walking speed between groups either during level walking or the perturbation trials, nearly all of the young subjects walked more quickly during the first perturbation trial. Young subjects also flexed the knee more quickly during loading response and extended the knee more quickly during midstance. However, there did not appear to be a preference in the older adults to walk more quickly or move the knee faster, which suggests that younger subjects adopt a strategy to complete the perturbation as quickly as possible, whereas older subjects do not have a common strategy. The slower knee angular velocity in some of the older subjects in the absence of proprioceptive deficits is consistent with the studies that suggest that older adults are not able to integrate sensory information to generate rapid postural responses (Woolacott et al. 1986; McGibbon et al. 2004; O'Connor et al. 2007). This relationship warrants further study.

In addition to differences in the speed related strategies, differences were also observed in the relationships between proprioception, neuromuscular, and joint position responses to the perturbations. In young subjects, 50.5% of the variance in knee flexion angle at initial contact in the initial response to the perturbation is explained by the initial response of VM during pre-heel strike. In these subjects,

greater VM activity during pre-heel strike may be a strategy that is used to brace the knee at heel strike in response to the perturbation. In older subjects, 64.7 % of the variance in knee flexion angle at initial contact in the initial response to the perturbation is explained by the initial response of MG and LG during pre-heel strike. Greater MG and LG activity in these subjects may be a strategy that is used to brace the knee during pre-heel strike in response to the perturbation.

During loading response in young subjects, 86.8% of the variance in peak knee flexion angle in the initial response to the perturbation is explained by the initial response of LG and VM, and by TTDPM at 45 degrees flexion. Greater errors in proprioception may be compensated by reducing the knee flexion angle during loading phase in response to the perturbation, which may be accomplished by an increase in VM activity and a reduction in LG activity. A similar strategy is not seen in older subjects.

During midstance in young subjects, 51.8% of the variance in peak knee flexion in the initial response to the perturbation is explained by the initial response of LH. In these subjects, an increase in LH activity may be a strategy to prevent full knee extension during midstance in response to the perturbation. Such a strategy is also not seen in older subjects.

These data suggest that normal aging is associated with changes in knee control strategies. In future work, we hope to include more subjects in our data analysis to further elucidate the mechanisms behind the change in such strategies. Additionally, we also hope to address whether the loss of knee control strategies are related to other conditions related to age, such as knee osteoarthritis. If we are able to establish such a connection, we hope to develop rehabilitation programs for adults to

help mitigate the changes in knee control strategies that could otherwise predispose this population to OA.

## REFERENCES


Akazawa, K., T. E. Milner, et al. (1983). "Modulation of reflex EMG and stiffness in response to stretch of human finger muscle." J Neurophysiol **49** (1): 16-27.

Bergman E., B.T. Fundin, et al. (1999). "Effects of Aging and Axotomy on the Expression of Neurotrphin Receptors in Primary Sensory Neurons." J Comp Neurol **410** (3): 368-386.

Dietz, V. (1992). "Human neuronal control of automatic functional movements: interaction between central programs and afferent input." Physiol Rev **72** (1): 33-69.

Garsden, L. R. and J. E. Bullock-Saxton (1999). "Joint reposition sense in subjects with unilateral osteoarthritis of the knee." Clin Rehabil **13** (2): 148-155.

Horak, F.B. and L.M. Nashner (1986). "Central Programming of Postural Movements: Adaptation to Altered Support-Surface Configurations." J Neurophysiol **55** (6): 1369-1381.

Horak, F.B., S.M. Henry, et al. (1997). "Postural Perturbations: New Insights for Treatment of Balance Disorders." Phys Ther **77** (5): 517-533.

Johansson, H. (1991). "Role of knee ligaments in proprioception and regulation of muscle stiffness." J. Electromyography Kines **1**: 158-179.

Johansson, H., P. Sjolander, et al. (1991). "Receptors in the knee joint ligaments and their role in the biomechanics of the joint." Crit Rev Biomed Eng **18** (5): 341-368.

Kawamura Y., H. Okazaki, et al. (1977). "Lumbar motorneurons of man: I) Numbers and diameter histograms of alpha and gamma axons and ventral roots." J Neuropathol Exp Neurol **36** (5): 853-860.

Lacour, M., L. Bernard-Demanze, et al. (2008). "Posture control, aging, and attention resources: Models and posture-analysis methods." Clinical Neurophysiol **38** (6): 411-421**.**

Li, K.Z., and U. Lindenberger (2002). "Relations between aging sensory/sensorimotor and cognitive functions." Neurosci Biobeh Rev **26** (7): 777-783.

Marigold, D.S. and A. E. Patla (2002). "Strategies for dynamic stability during locomotion on a slippery surface: effects of prior experience and knowledge." J Neurophysiol **88** (1): 339-353.

McGibbon, C. and D. Krebs (2004). "Discriminating age and disability effects in locomotion: neuromuscular adaptations in musculoskeletal pathology." J Appl Physiol **96** (1): 149-160.

McHugh, M. P. and D. E. Hogan (2004). "Effect of knee flexion angle on active joint stiffness." Acta Physiol Scand **180** (3): 249-254.

McIlroy, W.E. and B.E. Maki (1995). "Adaptive changes to compensatory stepping responses." Gait and Posture **3** (1): 43-50.

Mirbagheri, M., H. Barbeau et al. (2000). "Intrinsic and reflex contributions to human ankle stiffness: variation with activation level and position." Exp Brain Res **135** (4): 423-436.

Moorehouse K. and K. Granata (2007). "Role of Reflex Dynamics in Spinal Stability: Intrinsic Muscle Stiffness Alone is Insufficient for Stability." J Biomechanics **40** (5) 1058-1065.

Nichols, T.R. and J.C. Houk (1977). "Improvement of linearity and regulation of stiffness that results from the actions of the stretch reflex." J Neurophysiol **39** (1): 119-142.

Nielsen, J.B. and T. Sinkjaer (2002). "Afferent feedback in the control of human gait." J Electromyogr Kinesiol **12** (3): 213-217.

Odedsson, L.I. and C. Wall III et al. (2004). "Recovery from perturbations during paced walking." Gait and Posture **19** (1): 24-34.

O'Connor, K.W., P.J. Louglin, et al. (2007). "Postural adaptations to repeated optic flow stimulation in older adults." Gait and Posture **28** (3): 385-391.

Pai, Y.C., J.D Wening, et al. (2003). "Role of feedforward control of movement stability in reducing slip related balance loss and falls among older adults." J Neurophysiol **90** (2): 755-762.

Pai, Y.C. and T.S. Bhatt (2007). "Repeated-slip training: an emerging paradigm for prevention of slip related falls among older adults." Phys Ther **87** (11): 1478-1491.

Scheidt, R. J. Dingwell, et al. (2001). "Learning to Move Amid Uncertainty." J Neurol Phys **86** (2): 971-985.

Sjolander, P., H. Johansson, et al. (2002). "Spinal and supraspinal effects of activity in ligament afferents." J Electromyogr Kinesiol **12** (3): 167-76.

Tomlinson B.E., and D. Irving (1977). "The number of limb motor neurons in the human lumbosacral cord throughout life." J Neurol Sci **34** (2): 213-219.

Woollacott M.H., Shumway-Cook A, et al (1986). "Aging and Posture Control: Changes in Sensory Organization and Muscular Coordination." Int J Aging Human Development **23** (2): 97-114.

Woollacott, M., B. Inglin, et al. (1988). "Response preparation and posture control. Neuromuscular changes in the older adult." Ann NY Acad Sci **515**: 42-53.

Zhang, L. Q., G. Nuber, et al. (1998). "In vivo human knee joint dynamic properties as functions of muscle contraction and joint position." J Biomech **31** (1): 71-76.

Zhang, L. Q. and G. Wang (2001). "Dynamic and static control of the human knee joint in abduction-adduction." J Biomech **34** (9): 1107-1115.