FACTORS ASSOCIATED WITH PRE-CLINICAL DISABILITY AND RECOVERY OF MUSCLE AND PHYSICAL FUNCTION FOLLOWING TOTAL KNEE ARTHROPLASTY

by

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(Under the Direction of M. Elaine Cress)

ABSTRACT

Physical functional performance reflects the cumulative abilities of multiple physiological systems including joint and muscle function. Aging, inactivity, and disease can slowly degrade peak capacities and abilities of older adults necessary for living independently. In the first study, high functioning individuals (HIGH) were compared to individuals living independently but exhibiting lower levels of physical function (LOW). Group membership was determined by a threshold score of 57 on the Continuous scale Physical Functional Performance Test. Groups were compared on ability to perform tasks with or without difficulty or modification and mobility factors including gait speed, stride length, and steps/day. Our results indicate that older adults with high function had 27% greater gait speed, 18% greater stride length, 47% greater steps/day, and reported one less task requiring modification compared to those with low function.

In the second study, individuals with osteoarthritis (OA) scheduled for total knee arthroplasty (TKA) were examined. The purpose of this study was to examine 1) decrements in joint, muscle, and physical function associated with OA, and 2) the time course of three months recovery in joint, muscle, and physical function after TKA. Prior to surgery, individuals with OA had significantly more use-related pain and reduced muscle and physical function compared to controls. TKA reduced use-related pain 83% by 1 month post surgery. From 1 month to 3 months, the surgical limb of TKA patients increased knee range of motion by 13%, quadriceps strength by 16%, muscle quality by 13%, extensor power by 55%, and steps/day by 46% while physical function increased 19% to a level predictive of independent living. Range of motion was a significant predictor of physical function.

These results indicate that older adults living independently but with lower levels of function modify more mobility-related tasks and take fewer steps/day. Individuals recovering from TKA surgery can expect significant pain relief in one month followed by improved joint, muscle, and physical function by three months.

INDEX WORDS: osteoarthritis, total knee arthroplasty, pre-clinical disability, physical reserve

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DEDICATION

Frank Joseph Petrella
(Joshua 1:9)

"Nothing in the world can take the place of persistence. Talent will not; nothing is more common than unsuccessful men with talent. Genius will not; unrewarded genius is almost a proverb. Education will not; the world is full of educated derelicts. Persistence and determination alone are omnipotent. The slogan 'Press On' has solved and always will solve the problems of the human race."
- Calvin Coolidge, 1932

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CHAPTER I

INTRODUCTION

Osteoarthritis (OA) is the most common non-inflammatory joint disease in the United States affecting more than 20 million Americans or 12.1% of adults (1). The overall prevalence of diagnosed OA increases with age and most commonly occurs in older Caucasian females (2). Reporting of symptoms associated with chronic joint diseases can occur as early as the second decade of life (3). Radiographic evidence of changes in the joint that are associated with OA occur as early as in the third decade, however most of the diagnoses occur in the fifth or sixth decade (4). The span between the onset of symptoms and the full manifestation of OA suggests that the disease process is active long before clinical, overt OA is diagnosed.

The pathology of OA develops over two to four decades, reducing an individual's physical ability and increasing the risk of disability (Figure 1.1) (5, 6). The etiology of OA is not fully understood but initiating factors include joint trauma, instability, and congenital deformities (7). Once the pathology of OA is initiated, disablement is characterized by a loss of joint integrity due to articular cartilage degeneration (8). As degeneration continues, articular cartilage becomes thin even to the point of exposing subchondral bone. Cartilage-coated osteophytes begin to grow from the bone and may break off causing inflammation of the synovial membrane, synovitis, and joint effusion (7). These pathological changes lead to impairments in the knee joint limiting the range of motion and causing use-related pain (9).

The primary impairment of OA is an altered joint structure or malalignment (9) due to the thinning of articular cartilage and development of osteophytic processes altering the surface contours of the bone (7). According to the Nagi Model, impairments are structural abnormalities within a specific body system (Figure 1.1) (6, 10). An altered joint structure can result in bone on bone contact and limited range of motion during mobility. Radiographic imaging is the most common method of assessing structural damage in OA (11). A positive association exists between radiographic evidence of OA and self-reported disability (12, 13). The structural damage due to OA can result in an impaired knee exhibiting stiffness, loss of motion, and joint effusion (14). These impairments result in functional limitations that reduce an individual's ability to perform tasks important for living independently (15).

Limitations in mobility and mobility-related tasks are among the earliest indicators of disability and mortality in an OA population (16-18). The structural changes to the joint can result in decreased mobility and increased risk of limitations in mobility associated tasks such as walking or climbing stairs (15, 18, 19). The impairments associated with OA result in several mobility limitations including slower walking velocity, shorter stride length, wider stance, and decreased range of motion (19). Functional limitations such as these put an individual at risk for disability or dependency in the roles necessary for maintaining one's place within society.

Disability is a mismatch between an individual's abilities and the physical demand of the environment or living status (Figure 1.1) (10). This imbalance can be alleviated by either increasing an individual's ability or reducing the demand of the environment. For example, the older adult who uses analgesics to relieve pain is

attempting to increase the ability to tolerate pain. The older adult who reduces the need to use stairs by only using the ground floor is alleviating disability by reducing the demand of the environment that stimulates pain. For older adults with OA, disability is most often reported when the ability to climb stairs or get up and down from a chair result in hardship (13). In these instances, disability occurs when the individual's ability is insufficient to meet the demands of the current environment.

Pain, the most prevalent symptom of OA, results from altered joint structure, however confounding factors such as muscle weakness and asymmetry are also disabling factors. Accounting for pain, weakness, and psychosocial factors reduces the relationship between radiographic evidence of OA and disability (11, 20, 21). Muscle weakness, joint laxity, or asymmetric muscular activity may also contribute to an unstable joint (5, 11). Joint laxity results in malalignment of the knee and increases the risk of OA progression as well as a decline in physical function (22). Stress on an unstable joint can lead to strain on innervated tissue resulting in pain that leads to decreased muscle use (11). Disuse leads to muscle weakness that can then be classified as a secondary impairment of OA (Figure 1.2).

Physical inactivity or disuse can be just as responsible for the physical decline leading to impairments, limitations, and disability as diseases (23). Muscle strength and power are physiological factors important for the performance of daily activities such as standing from a chair, climbing steps, and basic mobility (24, 25). A major impairment that may be implicated in pain-related disuse is a loss of quadriceps strength in the affected limb(s) (26-33). In OA, quadriceps weakness is assumed to be due to disuse atrophy presumably because the individual minimizes use of the painful limb. In

contrast, quadriceps weakness also exists in persons with knee OA who have no history of joint pain (5). This may be due to the fact that individuals function at a low level to prevent the stimulation of pain, a pattern of behavior previously shown in cardiovascular disease patients (34). The role of quadriceps weakness in OA is not well understood. Weakness may be a risk factor for not only the development of OA but also the progression of the disease as well (29, 30, 35). As the impairments progress, the individual develops functional limitations especially in the area of mobility (36). Quadriceps strength, knee pain, and age determine the level of functional limitations more so than severity of radiographic evidence of OA (37).

Interventions can be introduced at different stages of the Nagi Model (Figure 1.2) in order to slow or delay disablement associated with OA. An intervention reduces the limitations or difficulties for a given environment by either reducing the demand of the environment or increasing the physical capacity. Interventions include analgesics, exercise, or surgery. The primary goal of each of these modalities for OA treatment is to manage pain and/or maintain or improve function. Minimizing pain has a positive impact on the individual's physical function and quality of life (38). Concomitant to these changes, the individual can also modify activities of daily living in order to reduce the perceived difficulty with changing the environment. Once pain is alleviated, an individual has the capacity to increase physical activity with positive influences on muscle and strength.

Muscle strength is important for living independently. Studies show that OA can ultimately affect quadriceps strength due to joint pain leading to inactivity. The insidious pain associated with OA limits the ability to be chronically active and therefore is

believed to deteriorate strength and reduce the physical reserve of individuals that allows them to remain independent. For muscle strength, the physical reserve is defined as the difference between an individual's peak strength and the level of strength required to perform activities independently (39). This reserve provides a "margin of safety" from the risk of becoming dependent (40). As the muscle strength reserve is eroded in association with OA, individual's progress toward a threshold below which a person is at greater risk of being unable to perform basic activities particularly in mobility (39).

The relationship between strength and physical function is curvilinear (24, 39). Each unit change in strength below the threshold is associated with a 17-fold change in function while above the threshold this relationship is a one unit change (39). A person with high peak strength will not change function with a change in strength, while those below the threshold will see a significant change in function. For those with high peak strength, function is unaffected due to the physical reserves in strength providing a margin of safety and absorbing age or disease-related changes without a resulting change in function. As the physical reserve deteriorates, individuals progress toward a threshold where one is at greater risk of being unable to perform basic activities particularly in mobility (39). Individuals below this threshold exhibit lower levels of strength. Once below the threshold, each unit change in strength results in a significant change in function (39). The ability to predict those at risk of functional decline associated with a loss of independence can provide an opportunity for early intervention in individuals who are still ambulatory yet at high risk for disability.

Individuals who maintain their independence but have peak strength levels at or near the threshold represent a sub-population that can be classified as pre-clinical

disability. Pre-clinical disability is an intermediary stage of disablement that precedes and predicts disability (36). Lower levels of maximal strength yet continued independence characterizes an individual in the pre-clinical disability stage. These individuals have reduced the demand of their current environment to accommodate their level of physical ability without sacrificing their independence (36). Methods for reducing the demand of the environment include environment modifications, external aids, or task modification (10). With task modification, individuals modify the procedures, time spent, or frequency of performing activities in order to reduce the physical or mental perceived (10, 36).

Physical function is an integration of physical ability such as muscle strength and range of motion with physical performance such as climbing stairs (41). Both these domains are mediated by psychosocial factors such as motivation or perceived ability (41). An individual with decreased abilities resulting in lower performance may not fully realize their lower physical ability. An individual who modifies tasks may also rely on greater assistance from family or some other social support in order to remain independent. This population is at high risk of becoming disabled given any further loss of physical ability.

Two potential indicators of pre-clinical disability are how individuals modify the way they perform tasks and decrements in mobility (42, 43). Modification of a task reduces the perceived demand so that the task is not considered difficult (43). These self-initiated strategies are used to forestall any change in environment necessitated by loss of physical ability. Having difficulty in mobility predicts the onset of limitations in activities essential for living independently and caring for oneself (16, 36) including

walking speed and stair climbing (36). Further research is needed on the measures of task modification and mobility as indicators for early detection of impending disability. Once the strategies for modification have been exhausted, an individual must seek other forms of treatment to minimize the impact of OA on ability and independence.

Individuals attempt to manage pain by self-initiated strategies such as task modification and physician-initiated interventions such as injections or fluid drainage. Alleviation of pain does not alter the pathology of OA (44). Only after pain management has failed to provide sufficient relief and the ability to perform daily tasks interferes with quality of life do physicians recommend surgery to replace the knee with a prosthesis (45). Thus, one of the more aggressive surgical treatments of OA is total knee arthroplasty (TKA) also referred to as total knee replacement. TKA is a widely performed and well-accepted procedure with approximately 362,000 surgeries performed in 2000 (46). The primary reason given for TKA surgery is relief of pain (47). TKA surgery provides the benefit of immediate pain relief from the pathological changes associated with OA. TKA relieves pain by intervening upon the pathology of OA and the associated structural changes. With reduced swelling and structural malalignment alleviated, greater knee flexion, one of the markers of recovery, is then possible (48). By correcting the alignment and joint integrity, TKA will alleviate pain and unmask the secondary impairment of lower levels of strength. TKA should slow or reverse the disablement process and allow the individual to maintain or even improve strength levels by reducing pain and allowing for increased activity (Figure 1.2). The expectation is that without pain or limited mobility, the OA patient can rebuild their physical reserves after surgery.

Greater strength provides a greater physical reserve and a "margin of safety" against the dangers of acute and chronic disease on physical function.

Patients of a TKA often exhibit quadriceps muscle weakness for as long as two years following surgery (26). Continued quadriceps weakness may be due to continued pain-related disuse, loss of type II fibers, surgical trauma, psychological factors, a selective inhibition, or the failure to activate available muscle fibers (40). Absence of recovery in muscle function may be related to persistent pain (48) leading to disuse (5), task modification (43), and/or low self-perception (49). Recovery of muscle function is associated with increased muscle size (50), increased ability to activate the muscle (51), and decreased joint pain. Reduced muscle strength and power translates into greater self-perceived functional limitation as well as lower functional performance (24, 52).

The effects of interventions on indicators of pre-clinical disability are not well studied. TKA reduces the symptoms, primarily pain, associated with OA and can theoretically lead to increases of the individual's physical capacities. The individual can now focus on an enabling process by either restoring function or increasing their access to the environment (35). Task modification and low mobility characterize individuals who maintain a high environmental demand despite a low physical capacity (36, 43). Once the symptoms of pain are alleviated, the individual should be able to increase physical capacity without being limited by use-related pain and no longer having to rely on strategies designed to reduce the demand of the independent environment.

Physical function recovery would be expected to be due to reduced pain (53) leading to increased activity. Higher levels of activity should lead to increased muscle strength (54) and power (55), improved range of motion (56), and improved self-

perception (57). Absence of recovery of physical function may be due to continued pain (58, 59), inactivity, disuse, task modification, and low self-perception of functional ability (59).

Purpose

The Nagi Model describes the causal pathway to disability that can be applied to chronic disease-related loss of physical capacities (6). The objective of this research is to identify factors that are indicative of pre-clinical disability in a population of community-dwelling older adults, as well as in the clinical population of older adults diagnosed with osteoarthritis. Early warning signs of disability include strategies of modified task performance and decrements in performance of measures of mobility. A second objective of the study is to examine decrements in muscle and physical function associated with OA, as well as the time course of recovery at one and three months after the intervention of total knee arthroplasty.

Hypothesis

1. Older adults below the threshold of independence have slower gait speed, shorter stride length, fewer steps/day, and modify more mobility tasks than older adults above the threshold of independence.
2. Prior to surgery, individuals with diagnosed osteoarthritis have reduced joint, muscle, and physical function compared to the non-surgical limb and age, sex, and height-matched controls.
3. During a three month recovery period, individuals with TKA surgery have a greater change in joint, muscle, and physical function compared to age, sex, and height-matched controls.

4. During a three month recovery period, change in physical function is due to decreased use-related pain and improved strength in the surgical limb.

Significance of the Study

The significance of this study stems from the need to determine characteristics of individuals considered to be at risk for disability. By identifying risk factors of disability and prior to the manifestation of functional limitation, strategies for primary prevention of disability can be initiated before functional decline begins. To effectively address the impending onset of OA cases expected in the next five to thirty years, priorities should include methods for preserving the independence of older adults with OA. Primary prevention will be crucial in fighting the epidemic of arthritis-related disability as the population ages. Apart from the public health standpoint, many older adults perceive independence as more important to their quality of life than disease state (60).

Performance measures of mobility such as walking speed and number of steps per day and task modification are precise and accurate predictors of disability (61). Early detection of those at or below the threshold of independence may allow for the timely intervention and motivation needed to delay or reverse the disablement process. Additionally, the effects of the surgical intervention, total knee arthroplasty, on improvements in muscle strength of individuals diagnosed with osteoarthritis are unclear. Explanation of the absence of recovery in thigh muscle characteristics and the resulting effect on physical function would provide valuable information for rehabilitation interventions aimed at improving physical function independence. While evidence suggests TKA reduces pain, this study will quantify performance changes as a direct result of the surgical intervention. In addition, it will help to delineate the ability to

recover muscular function that is critical for functional performance and independent living.

Figure Legends:

Figure 1.1: The Nagi Model (1976) modified to include the concept of environmental demand.

Figure 1.2: The Nagi Model (1976) modified for the pathology of osteoarthritis and the intervention of total knee arthroplasty.

Figure 1.1

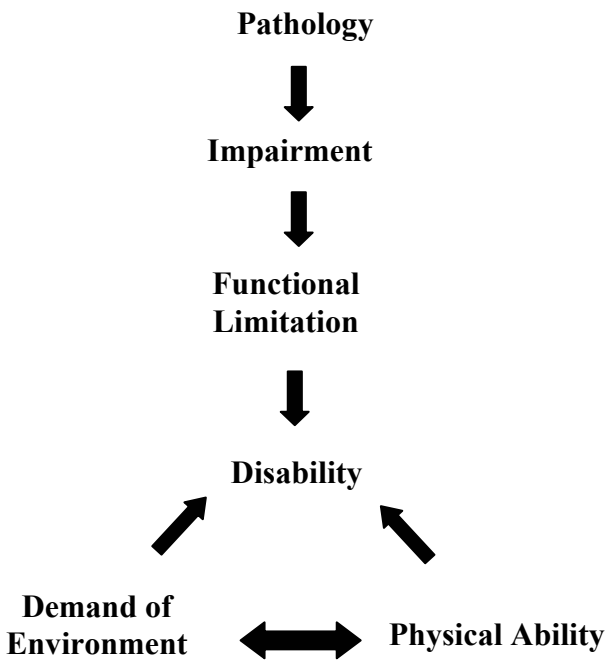
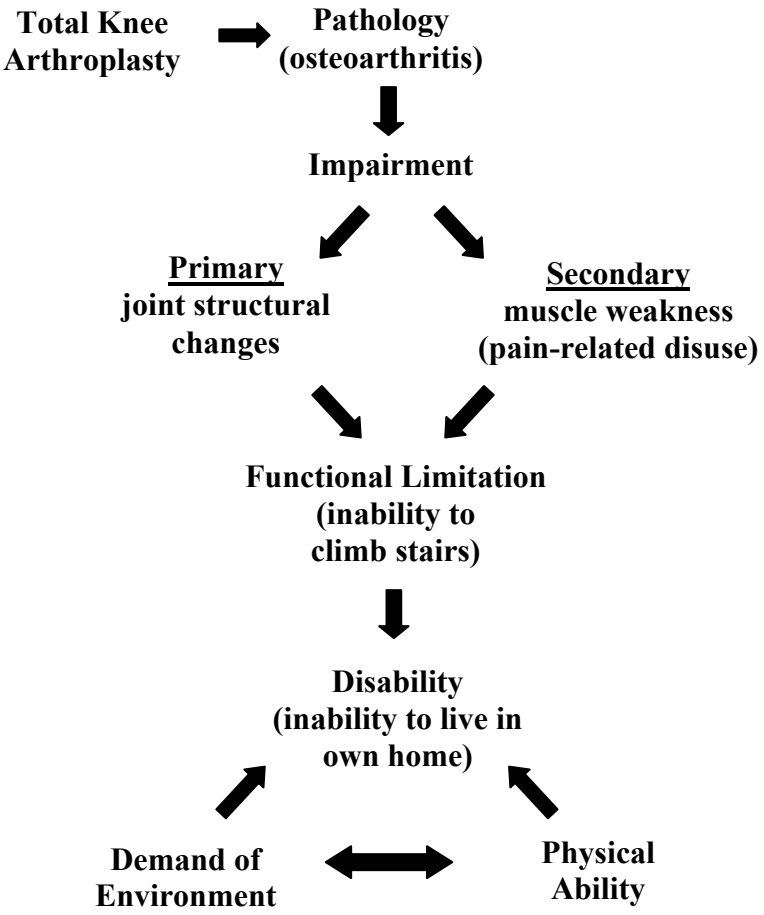

Pathology
Impairment
Functional Limitation
Disability
Demand of Environment
Physical Ability

Figure 1.2


Total Knee Arthroplasty
Pathology (osteoarthritis)
Impairment
Primary joint structural changes
Secondary muscle weakness (pain-related disuse)
Functional Limitation (inability to climb stairs)
Disability (inability to live in own home)
Demand of Environment
Physical Ability

CHAPTER II

REVIEW OF LITERATURE

Osteoarthritis (OA) is the most common non-inflammatory joint disease in the United States with more than 30 million diagnoses of Americans, or 12.1% of adults (4). More than half the population exhibits radiological evidence of OA in at least one joint after the age of 65 (4). As of 1998, it was a leading cause of work-site disability second only to chronic heart disease (62). Onset of the disease can occur as early as the 3rd decade affecting approximately 3% of Americans. Incidence of OA increases to 25% of Americans between ages 45-64 and 50% for those over the age of 65 (63, 64). The disease affects both men and women with symptoms usually presenting in the 4th or 5th decade with men more affected than women until after the age of 55 (7).

This review consists of three sections. In the first section, the normal anatomy and physiology of the knee is explained along with the pathology, symptoms, and age-related changes associated with OA. In the second section, total knee arthroplasty (TKA) is explained along with adaptations of skeletal muscle during recovery from surgery. In the third section, the relationship of physical function and individual capacity is explained.

Osteoarthritis

Normal Anatomy and Physiology

The knee is a synovial joint, the most moveable classification of joint, consisting of the following parts: a fibrous joint capsule, synovial membrane, and synovial cavity (7). The synovial cavity is lined with a synovial membrane and articular cartilage and

filled with synovial fluid. The synovial membrane lines the non-articulating aspects of the joint while the articular cartilage covers and pads the articulating bony surfaces of the femur and tibia (7). The articular cartilage is a layer of connective tissue comprised of an extracellular matrix composed primarily of collagen, chondrocytes, proteoglycans, and water. Another important distinction between the synovial membrane and articular cartilage is that the synovium is highly vascularized and can regenerate quickly while the articular cartilage has no blood vessels, lymph vessels, or nerves (7). This avascularization makes the articular cartilage insensitive to pain and slow to heal after injury or trauma.

The purpose of articular cartilage is to reduce friction within the joint as well as distribute the forces of weight bearing (7). This function is largely accomplished by the high water content of the articular cartilage which acts as a shock absorber and lubricant (14). Chondrocytes are single cartilage cells which secrete the extracellular matrix and proteoglycans while residing within the lacunae of the matrix. These cells are highly isolated within the matrix and receive nutrition through diffusion of synovial fluid (14). Chondroyctes respond to trauma, pH changes, and pressure to produce rapid turnover of proteoglycans in order to maintain homeostasis (14). Proteoglycans are complexes of protein and polysaccarides that act as water pumps for determining the viscoelastic properties of the cartilage (65). Proteoglycan pumps respond to weight bearing pressure and provide a lubricating film on the articulating surface of the cartilage as well as draw synovial fluid back into the cartilage once pressure has been released (7). Articular cartilage is supported by underlying subchondral bone (14).

Pathology

OA is a disease that is directly correlated with age (8) and affects the anatomy and structure of any joint particularly weight-bearing joints such as the knee. Idiopathic and secondary OA are the two main classifications of the disease sharing similar pathological features but different etiologies. Regardless of classification, OA is characterized by the fundamental pathological feature of degeneration and loss of articular cartilage in synovial joints. The main sites effected by OA are the femoral condyles, tibial plateau, and the posterior aspect of the patella (14).

Idiopathic, or primary, OA is the most common type of classification but is not associated with any known risk factors or etiology (7). Secondary OA can be caused by any condition that directly damages cartilage due to chronic, excessive, or abnormal force to the joint resulting in instability (8). Risk factors associated with secondary OA include joint trauma, long-term mechanical stress, inflammation, joint instability, congenital or skeletal deformities, hyper-parathyroidism, neurologic disorders leading to diminished pain or proprioception, and hematologic or endocrine disorders causing chronic bleeding and effusion in the joints (7).

The development of OA is characterized by loss of articular cartilage. The etiology of articular cartilage loss is unknown but most likely due to enzymatic breakdown of the cartilage matrix. Trauma or inflammation to the joint results in the up-regulation of proteolytic enzymes referred to as aggrecanases (66). These enzymes degrade the proteoglycan complexes within the cartilage (7). Defective proteoglycans effect articular cartilage by interfering with the pumping action important for regulating movement of water and synovial fluid in and out of the articular cartilage. Cartilage that

assimilates too much fluid becomes less able to withstand weight-bearing stress and is at greater risk of degradation (7, 14).

As proteoglycans degrade, fragments and catabolic products are released into the synovial fluid further inflaming the joint (67). As the joint continues to be inflamed, further cytokines and free radicals infiltrate the cartilage and up-regulated the activity of acid metalloproteinases which degrade the collagen components of the extracellular matrix (66, 67). As the structural components of the matrix degrade, the chondrocytes are exposed to mechanical stresses and enzymatic degradation (7). Chondrocytes exposed to these stressors produce altered proteoglycans and release more catabolic factors (67) resulting in a cycle of further degradation to the components of the articular cartilage (7). As continued disruption occurs to the cartilage, more fluid is retained resulting in reduced stiffness and weight bearing capabilities (67).

Early in the OA disease process, articular cartilage begins to lose it color and erode. The cartilage gradually forms longitudinal fissures, a process known as fibrillation (7). As fibrillation develops, cartilage becomes thin and may expose subchondral bone. The unprotected bone becomes sclerotic and develops cysts which communicate within the longitudinal fissures (67). As pressure increases within the cysts, their contents are forced through the longitudinal fissures and into the synovial cavity causing a rupturing of the articular cartilage. Cartilage-coated osteophytes begin to grow outward from the bone altering the surface contours and joint anatomy (7). These osteophytes may break off and inflame the synovial membrane causing synovitis and joint effusion (67). These changes result in clinical manifestations of OA that appear during the 5^{th} or 6^{th} decade, however, asymptomatic changes in articular surface are common by age 40 (8).

Clinical Manifestations

Abnormalities or disease to joints may be difficult to diagnose due to the small number of symptoms produced by the joint (7). The primary symptoms of OA are pain and stiffness in one or more joints most commonly noticed in weight-bearing joints first (7, 68). Joint stiffness is typically the first symptom manifested (69). Stiffness may be due to changes in the bone surface, proliferation of osteophytes within the synovial cavity, or swelling within the joint. Swelling in the joint cavity may be due to an inflammatory response of the synovial fluid or blood increasing the joint capsule volume (67). While joint pain is the most prevalent symptom reported in relation to OA (47), the actual cause of joint pain is unknown. Possible explanations include distention of articular cartilage as well as stretching and inflammation of the joint capsule (7). Protruding subchondral bone into the joint cavity may also result in pain due to weight-bearing forces through bone on bone articulation (14). Other symptoms associated with OA include point tenderness, reduced range of motion, muscle atrophy, or joint deformity (7).

Lower limb muscles are important joint stabilizers and weakness in those muscles may contribute to the development of OA (29, 30). The quadriceps muscles play a prominent role not only in movement but also in shock absorption, proprioception, and stabilization of the knee joint (70). A reduction in muscle performance, particularly in response to mechanical stresses, could reduce ability to protect the joint (29). A common idea is that articular damage causes pain and joint effusion resulting in disuse and inactivity-related atrophy of the muscle (30). However, some literature suggests that muscle weakness may precede and contribute to the development of OA (29, 30).

Several studies show that quadriceps weakness is present in individuals with radiographic evidence of OA but not with muscle atrophy or joint pain (30). This finding would support the idea of muscle weakness as a risk factor and not as a result of pain-related disuse. Additionally, the muscle weakness was detected in the quadriceps muscles and not the hamstrings which would contradict the idea of pain-related disuse (30). These findings were confirmed by a longitudinal study which found reduced quadriceps strength relative to body weight to be a risk factor for the development of OA at 31.3 months follow-up (29). The quadriceps muscles brake the leg during the descent phase of walking as well as provide the anteroposterier stability of the knee (29, 30). Quadriceps weakness could affect the mechanical loading of the joint and increase the impact forces at heel strike (29).

Theories of Etiological Causes of OA

Aging appears to increase the risk of OA by compromising the ability of articular cartilage chondrocytes to maintain or restore the cartilage tissue (5, 8). Damage to cartilage tissue through trauma or some other factor stimulates chondrocytic synthesis and proliferation of proteoglycans and collagen for the maintenance and restoration of cartilage integrity (8). This synthetic response can last for years, but over time chondrocytes become less responsive to anabolic factors. This age-related change results in an imbalance between synthesis and degradation eventually leading to the thinning of cartilage and resulting fibrillation (7). Chondrocytes of aging cartilage also synthesize smaller and fewer proteoglycans (8). Changes to the proteoglycans reduce the ability to maintain homeostasis of the water content of cartilage leading to reduced stiffness and strength of the articular cartilage.

OA-related in Lower Limb Strength

A major impairment associated with OA is a loss of muscle strength in the affected limb(s) (26, 31, 33, 71-73). Weakness and atrophy of muscles acting across a damaged joint is common (40) and primarily seen during knee extension of the effected limbs (26, 33, 40). Wigren et al (1983) was one of the earliest studies to show significantly less strength for flexion and extension in patients with OA when compared to a previously published data set of controls that were healthy. Individuals with OA can expect to have 65% of the flexion strength of the non-diseased limb (26), and 55-78% of the flexion strength of healthy controls (31). For extensor strength, individuals diagnosed with OA have 59-81% the strength of the non-diseased limb (26, 72, 73), and 36-75% the strength of healthy controls (31, 71, 73). There was no difference between the non-diseased limb of the individuals with OA and the group that was considered healthy (33). In summary, individuals with OA experience a decline in strength and muscle size, however, the declines in strength may be greater than those expected from losses in muscle size (73, 74).

OA-related in Lower Limb Muscle Size

A well-accepted relationship exists between muscle strength and size as assessed by cross sectional area (CSA) (75-78). Young (1986) was one of the first to publish comments on selective muscle wasting associated with joint disorders. Early studies examined thigh muscle wasting by examining circumference only (48). Using circumference measurements to assess muscle size limits the ability to determine differential atrophy among the muscles of the thigh. Sargeant et al (1977) found atrophy was almost all localized to the quadriceps muscle in patients with unilateral orthopedic

problems. Biopsy data revealed a 40% difference in mean fiber cross-sectional area (CSA) of the quadriceps despite only a 12% difference in fat-free thigh volume (79). Thus, a decrease in muscle size is primarily confined to the quadriceps muscles. Other findings suggest a 5% difference in mid-thigh circumference corresponds to a 22-33% difference in quadriceps CSA area for individuals with unilateral knee injuries (48). Advances in technology allow for more accurate assessment of individual muscle changes particularly MRI analysis of anatomical CSA (76). Proton MRI has been used previously to assess skeletal muscle, subcutaneous fat, and interstitial fat (78, 80-82). No studies have been found which use this technique to assess changes in the quadriceps muscle in populations with OA.

Preferential loss of the quadriceps mechanism may be due to immobilization of the knee joint, joint effusion, pain, or perhaps some type of reflex inhibition (48). All these factors are apparent in a population with OA (71). Immobilization of the knee almost completely immobilizes the quadriceps while hip movement would need to be restricted to immobilize the hamstrings (48). Joint effusion can also result in quadriceps atrophy. Small volume increases in the knee joint which do not result in swelling can still produce a 60% inhibition of quadriceps (48). Pain in the knee joint may result in an arthrogenic inhibition of the quadriceps muscle (83, 84). Arthrogenic muscle inhibition (AMI) is attributed to an altered afferent signal resulting in an inhibited efferent stimulation of the motor neurons of the quadriceps (30). Inhibiting the quadriceps muscle would result in less activation of the muscle leading to atrophy.

OA-related Changes in Lower Limb Muscle Activation

Quadriceps weakness in knee OA may be due in a large part to arthrogenous muscle inhibition (AMI) (74). AMI is characterized by a failure to fully activate the available muscle during voluntary effort (30, 74, 83, 85). Joint pathology may reduce the excitability of the alpha and gamma motorneurons and decrease the ability to activate the available muscle (85). This inhibition is associated with altered afferent neural impulses from articular mechanoreceptors (83) and appears to be highly selective of the quadriceps muscles (74). Unconscious down-regulation of quadriceps activation by AMI may be a protective reflex to prevent further muscle or joint damage as well as to maintain a balance in motor output between the limbs (73). AMI is also associated with muscle atrophy and weakness (72) and appears to be independent of pain, occurring both in the presence and lack of presence of pain (83). Arthrogenic inhibition could help explain the disparity between muscle strength and muscle size in patients with OA (73).

The inability to fully activate the quadriceps muscle would reduce force output by either failing to recruit all available motor units or failing to attain maximal discharge rate from the motor units recruited (86). Merton (1954) was the first to externally activate muscle using electrical stimulation. By activating the muscle proximal to the neuromuscular junction, any change in force with stimulation would suggest failure to maximally activate due to the central nervous system. Newham (1991) was the first to utilize the burst method by delivering a train of electrical pulses which was more sensitive to failure than single or double pulses (51, 87). Central activation failure is determined by comparing the ratio of MVC force to burst augmented force (51, 71, 87). Any ratio less than 1.0 suggests failure. Activation testing of OA patients shows a

consistent failure to maximally activate the muscle. OA patients can activate approximately 66% - 76.2% of the muscle theoretically available (72, 73).

OA-related Changes in Lower Limb Power

Muscle strength is a function of cross sectional area and activation of the muscle. Muscle power is defined as muscle force divided by time to reach activation. Muscle force and power while different are related to physical function in older adults (75, 88). Changes in CSA and activation can results in negative effects on power generation by the extensor muscles. Recent research has studied the influence of peak power on independent living in older adults (89). Leg extensor power (LEP) is needed for many activities of daily living (55) such as walking, climbing stairs, and rising from a seated position (89). Muscular power is a functionally relevant measure of physiological capacity since power rather than strength is often used in mobility (25). Maintaining leg power throughout life may reduce fall risk and associated fractures (55). LEP has been safely measured in an OA population, prior to surgery. The affected limb produced approximately 70% of the power output of the unaffected limb. Few if any studies have been published that report on recovery of lower limb extensor power after TKA surgery.

OA-related Changes in Lower Limb Pain

OA may lead to impairment and disability through pain-related disuse due to structural changes to the knee joint (9). Disuse or inactivity induces or accelerates the loss of physical capacity (90) including extensor strength and power. Population estimates suggest that one third of all individuals over the age of 65 have radiographic evidence of OA and 25-50% of those individuals experience knee pain and disability (9, 11). Individuals with diagnosed OA and individuals without radiological evidence of OA

but symptomatic knee pain had lower peak torques in isometric and isokinetic flexion (19-39%) and extension (18-37%) compared to sex-matched healthy controls (31). Alleviation of joint pain will produce an increased maximal voluntary contraction (91). Strategies designed to manage pain should have a positive impact on lower limb strength.

OA-related Changes in Physical Function

Knee osteoarthritis is a major contributor to physical disability in non-institutionalized older adults (13, 15, 92) specifically in activities requiring ambulation (11). Poor strength, power, and pain-related disuse can have a significant impact on physical function and level of independence for individuals with OA (11). A clinical diagnosis of OA does not necessarily indicate increased disability in the performance of functional activities. Studies show that OA progresses beyond a threshold where the radiographic changes of OA are characterized as moderate before symptomology increases. Once individuals exhibit moderate evidence of OA with increased symptomology, then does an individual begin to exhibit performance decrements (13, 93). Decrements are typically evident in stair climbing, walking a mile, and housekeeping (13, 15).

Total Knee Arthroplasty

The primary goal for treatment of OA is either to manage pain or to maintain or improve function (94). Treatments for OA can include analgesics, exercise, weight control, or surgery (11, 94). Alleviating use-related pain allows an individual the ability to increase physical activity with the resulting positive influences on muscle and strength. Several studies show that increasing muscle strength and activity can reduce the symptomology and disability associated with OA (94). In seeking relief from

symptomology, individuals attempt to manage pain by self-initiated strategies such as task modification and physician-initiated interventions such as injections or fluid drainage. Unfortunately, the alleviation of pain does not alter the pathology of OA (44). Current research has focused on managing the symptoms associated with OA but do not effect the underlying processes that cause OA.

Treatment of symptomology associated with OA typically begins with pharmocologic interventions to mange pain (94). Only after non-surgical treatments for pain management have failed do physicians recommend surgery to replace the knee with a prosthesis (45). Thus, one of the more aggressive surgical treatments of OA is total knee arthroplasty (TKA) also referred to as total knee replacement. The most common medical indication for TKA surgery is degenerative structural damage to the knee joint accompanied by pain and functional impairment that does not improve with non-operative treatment (95). The primary reason given for TKA surgery is relief of pain (47). TKA surgery is a cost effective intervention that provides the benefit of immediate pain relief from the pathological changes associated with OA (45).

TKA is one of the most prevalent surgeries within the United States and has become a reliable surgical procedure for the treatment of painful degenerative arthritis by relieving pain, increasing knee function, and improving quality of life (95). According to the American Academy of Orthopedic Surgeon's (AAOS), approximately 362,000 surgeries were performed in 2000, with each surgery costing $23,000 (excluding physician's fees and rehabilitation costs). Approximately 72.7% of the surgeries performed are on individual's aged 65 and older with 66.2% of those being women. The average female undergoing TKA is age 68 and the average length of hospital stay is 5.6

days. With the current trends of aging in the population, the AAOS estimates as many at 475,000 surgeries will be performed annually by 2030. Current research suggests that alleviation of pain is immediate however the restoration of function is unclear (94).

TKA involves total replacement of the osteoarthritic joint's articulating surfaces with metal alloy prostheses. By replacing the joint surfaces, the symptomology associated with degraded cartilage is immediately relieved. The primary reason given by individuals for electing to have TKA is the relief of pain (47). Many patients also elect to have surgery to relieve functional limitations such as the inability to go shopping, socialize, or drive a car as well as the desire to eliminate use of a cane, and to live independently (95). Several issues of TKA surgery have been examined including persistent muscle weakness and incomplete recovery of physical function after surgery. Only three longitudinal studies examine the changes in individuals after total knee arthroplasty. One of the longitudinal studies failed to collect data before surgery thus all changes were assessed relative to measurements taken 3-months after surgery. Several studies use a cross sectional design to compare changes in the surgical limb to either the non-surgical limb or a control group.

Recovery of Lower Limb Muscle Strength

Wigren (1983) conducted one of the first longitudinal studies of changes in quadriceps muscle strength after TKA surgery. Strength data prior to surgery was not collected but found significant increases in strength using three months post-surgery as baseline for knee extension. The authors commented that the three month strength levels were unchanged from pre-surgery but no data was reported. Compared to three month

values, flexor strength significantly increased 26% at 12 months and increased an additional 10% at 24 months post-surgery.

The authors also included cross-sectional data examining strength in the surgical and non-surgical limbs compared to a previously published data set of strength in healthy volunteers. Results show that 36 months post-surgery there was no difference in strength between healthy controls and the non-surgical limb but a significant difference of strength in the surgical limb. However, the article reports contradicting results with a separate analysis that states there was no significant difference between the surgical limb and non-surgical limb of TKA patients at 36-months post-surgery. The contradiction in reporting may be due to the study having a low number of subjects at the 36-month time-point thus lacking the power to show a difference between limbs.

Persistent loss of muscle strength during knee extension has been shown in isometric (28, 33, 71-73, 96, 97) and isokinetic (26, 28, 32, 96) testing of maximal voluntary contractions in the surgical limb. Studies vary in post-surgical follow-up assessment ranging from one month to ten years after surgery. At one month post surgery, TKA patients can expect a 67% decrement in strength in the surgical limb when compared to the non-surgical limb or healthy controls (71). At three months post-surgery, the surgical limb has shown a varied response in strength ranging from a 17-22% decrease (96, 97) to a 10% increase (26) from pre-surgery values. The surgical limb is still typically 24-34% weaker than the non-surgical limb at three months (26, 96).

One year after TKA surgery, the surgical limb has been shown to gain 42% extensor strength compared to pre-surgical measurements (26). Again, these changes in strength are still typically 13-29% less than the strength of the non-surgical limb

measured at the same time point. At two to three years after surgery, the surgical limb has improved 28-60% in strength but remains 16-24% below the values of the non-surgical limb when compared relative to pre-surgical values (26, 73).

The majority of the studies examining the recovery from TKA surgery are cross-sectional in design. Only one longitudinal study of pre-surgery and multiple post-surgical assessments was found during review of the literature. Berman (1990) examined 68 patients pre-surgery with follow-up assessment at 7-12, 13-23, and 24+ months. Postoperatively, hamstring peak torque values were able to attain knee flexion strength levels of the non-surgical limb within a period of 7 to 12 months after surgery, whereas the quadriceps strength showed a deficit of 16% at two years of follow-up examination (26).

Few studies were found that compared recovery of quadriceps strength to healthy controls (28, 32, 33, 73). A design that relies on comparisons to apparently healthy subjects would control for any undiagnosed disease of the non-surgical limb as well as any changes due to pain-related disuse and inactivity. Marked decrements in isometric muscle extension strength were measured as early as one month post-surgery with a 67% decrement compared to a recreationally active control group (32). There was no significant difference in strength between the non-surgical limb and the control group. Two separate studies found that at one to two years post-surgery, TKA patients had a 19-38% decrement in strength comparing the surgical limb to the control group and a 17-26% decrement in the non-surgical limb when compared to controls (32, 73). These reduced levels of quadriceps strength have been shown to persist as far as 13 years post-surgery with the surgical limb still presenting with a 27-28% decrement in strength

compared to healthy controls (28). These data suggest that perhaps the non-surgical limb also exhibits persistent muscle weakness if not directly related to OA pathology then possibly related to pain in the osteoarthritic limb resulting in disuse. Three studies found that strength in the surgical limb recovered to pre-operative values (72, 96, 97) and two studies found a recovery to the non-surgical limb (33, 73). Three studies found significantly less strength in the surgical limb when compared to the non-surgical limb (26, 32, 97) and four studies found significantly less strength when compared to a control group (32, 33, 71, 73). No studies to date have been found examining the recovery of leg power after total knee arthroplasty.

Recovery of Lower Limb Muscle Size

Only two studies were found that examined changes in muscle size before and after TKA surgery. Madsen et al. (1997) used dual energy X-ray absorptiometry (DEXA) to assess lower limb size. While a 3% difference in lean mass of the two legs was found, a 20% decrement in strength existed for the surgical limb (84). These findings suggest a failure to activate the available muscle in the surgical limb. A cross-sectional study of one-year post-surgery found significantly higher lean mass of the surgical limb compared to sex-matched controls (32). CSA assessment was done by anthropometric measures and could not detect changes in intramuscular fat which may make the findings questionable. Muscle CSA reported by Walsh et al. (1998) for CSA were very low compared to published norms (80) calling their validity into question. Proton MRI is an accurate and valid method of assessing skeletal muscle (8, 78, 80-82). No studies have been found which use this technique to assess changes in the quadriceps muscle in populations with OA.

Recovery of Lower Limb Muscle Activation

Several studies have reported on the recovery of voluntary activation deficits after knee replacement surgery. Muscle activation has been assessed by electromagnetic stimulation (EMS) utilizing two techniques, twitch interpolation and burst superimposition. Two studies used the twitch interpolation technique, a comparison of EMS twitch height at rest to twitch height during an attempted maximal contraction (72, 73). Prior to surgery, this technique shows voluntary activation of 66.4-76.2% in the surgical limb and 79.3% in the non-surgical limb (72, 73). After surgery, individuals could voluntarily activate 74.2% of the available muscle in the surgical limb one month after surgery (71). The non-surgical limb had voluntary activation of 92.7% at one-month post surgery (71). At 18 months after surgery, the surgical limb is 76.9% activated (72) and increases to 84.9% at 33 months post surgery (73). Control subjects matched on sex and age exhibit voluntary activation ranging from 90.9-94.3% (71, 73). An increase in activation may be due to removal of inhibitory factors associated with pain resulting in a reduction in AMI (71).

Recovery from Pain in the Lower Limb

Several studies have found significant improvements in self-rated pain six months to one year post-surgery using the SF36 questionnaire (57-59). Despite an improvement in pain ratings, individuals with OA continue to report greater pain levels than norms of apparently healthy individuals. These findings suggest a significant relief from pain after surgery but at an elevated level compared to the general population. Pain has also been assessed by self-rated intensity and use of medications before and after surgery (58). Six months after surgery, 33.8% of the sample reported severe pain and one-year after

surgery 14.7% rated pain as severe. Pain prevalence as also assessed by analgesic intake which dropped 17.6% in the first 6 months and an additional 5.4% at the one-year follow up (58). The number of individuals no longer requiring the use of analgesics doubled 12 months after surgery. The post-surgical reduction in pain was associated with increased walk distance and reduced difficulty with stair climbing (58). The alleviation of pain itself resulted in improved function in some mobility tasks. Approximately one-third of the sample continued to report dissatisfaction with the surgery at one year but this was due to increased pain in the non-surgical limb at one-year post-surgery. These data suggest that OA was operating in more than one limb and that the replacement intervention was successful in alleviating pain in the surgical limb.

Recovery of Physical Function

Several studies report on the physical functional changes after total knee arthroplasty. Walsh et al (1998) examined the recovery of objectively measured functional limitations after TKA surgery. One year after surgery, TKA patients were 13-18% and 43-51% slower in both walking and stair climbing speed respectively when compared to healthy age and sex-matched controls. Ouellet and Moffett (2002) conducted a longitudinal study examining gait analysis as a primary measure but did include functional measures including an 8-foot up and go task as well as a 6-minute walk. Prior to surgery, TKA patients took 21% longer in the up and go task and walked 72% of the distance covered by controls during the 6-minute walk (18). At two months after surgery, function declined with TKA patients taking 58% longer to complete the up and go task and only walking 58% of the distance completed by controls during the 6-

minute walk. These values exhibit significant decrements compared to baseline measures.

Physical Function

Disablement

Physical function is the integration of physiological capacities and physical performance capability mediated by psychosocial factors (41). Disablement is the process whereby individuals lose physical function and the ability to remain independent (39). The Nagi Model (1976) describes how chronic and acute conditions effect specific physical capacities and the performance of activities of daily life (ADL) (10). The pathway of disablement is initiated by a pathology which results in an impairment(s) leading to functional limitations and ultimately disability. The pathological condition of osteoarthritis can lead to impairments such as structural changes to the joint. These impairments lead to functional limitations such as the inability to use stairs. If functional limitations accumulate or become severe, the individual is no longer able to fulfill social roles. Functional limitations refer to the individual's capability without reference to environmental demands while disability is a social process and must take into account the demands of the societal environment (10). Disability signifies a relationship between the individual and the environment and the patterns of behavior that develop from the reduced (or lost) ability to perform expected social roles of extended duration because of a chronic disease or impairment (10).

Physical Thresholds and Function

Age-related losses in physical capabilities typically begin in the 4th decade; however, incidences of disability do not occur until after the age of 75 (39). During this

time span, age and/or chronic diseases are eroding the physiological capacities of individuals without a concomitant loss in physical function. For all activities, a minimum amount of physical capacity, including muscle strength and power, is required to perform that activity in a normal manner (24, 25). Physical reserve is the excess physiological capacity not tapped during daily activities (24) or the difference between an individual's peak physical capacity and the level of ability required to perform activities independently. Physical reserve provides a margin of safety that absorbs age or disease-related changes without a resulting loss in function (40).

As physical reserve deteriorates, individual's approach a threshold of independence below which any further loss of peak capacity results in a 8-17 fold decrease in physical function (39). Thresholds characterize the curvilinear relationship between capacity and function as physical capacities change on a continuous scale whereas functional changes are typically quantal (40). If an individual is beneath the threshold, small improvements in a physical capacity such as strength may be accompanied by considerable improvement in function. However, an individual above the threshold may experience a gradual loss of strength that is not apparent until the individual is suddenly unable to perform a crucial function (40).

If physical capacity falls below the level of ability required for performance of daily tasks then resultant functional limitation can bring about loss of independence (24). Disability occurs when physical demands of the current living status are greater than individual physical capacity (10). As the population ages, a large increase in the number of adults will be living at or near the threshold of ability, needing only a minor or acute illness to render them dependent (40).

Pre-Clinical Disability

In spite of declining fitness and function, many older adults remain in a current living status that requires higher levels of function. Individuals who maintain independence with physical capacities near or below the threshold have been identified as pre-clinical disability (36, 43). Disability is an imbalance between the individual's capabilities and the environmental demand (10) and can be alleviated by increasing capability or by reducing demand. Individuals classified as pre-clinical disability may develop strategies to modify the method of task performance in order to accommodate physical changes and remain independent (98). Task modification strategies reduce physical demand on the individual by altering the environment or modifying the procedures, time spent, or frequency of performance of daily tasks (10, 42). These self-initiated strategies are used to forestall any change in living status that may be necessitated by loss of physical ability. Individuals who remain independent yet modify task performance have been found in those with pre-clinical disability and therefore are at a greater risk of progressing to dependency with subsequent losses in their physical capacities (43).

Disability usually occurs first within the domain of mobility. Mobility difficulty predicts the onset of limitations in activities essential to living independently in the community and caring for oneself (16, 36, 99). The use of mobility measures may provide another simple and effective prediction for those at risk of becoming disabled. For individuals in the transitional stage of pre-clinical disability, the subtleties of changes in mobility during early stages of decline are less well defined. Changes in mobility are most likely to be evident during higher effort assessments, such as the 6-minute walk, or

over longer time frames such as a 7-day average of steps/day. The ability to predict those at the threshold of limitation or at risk for disability would be indispensable for the prevention of age or diseases-related physical disability (43). Prediction and prevention of medical problems is the foremost method of optimizing health and reducing health care costs of older adults (90). The continued loss of physiologic reserves below the threshold of independence can eventually progress into disability despite modification strategies aimed at delaying that progression (39).

CHAPTER III

FACTORS ASSOCIATED WITH PRE-CLINICAL DISABILITY IN OLDER ADULTS 63-71 YEARS OF AGE[1]

[1] Petrella, J.K. and M.E. Cress. To be submitted to *Journal of the American Geriatrics Society*.

Abstract

Introduction. Individuals modifying performance of everyday tasks may comprise a subset of community-dwelling adults in early stages of limitation and at high risk of disability. The purpose of this study was to examine differences in mobility and task modification between independent older adults above and below an empirically derived physical threshold that has been linked to independence.

Methods. Twenty community-dwelling older adults (72.8 ± 6 years) were categorized into groups based on functional performance using the Continuous scale Physical Functional Performance Test total score (Cs-PFP TOT). Individuals with Cs-PFP TOT ≥ 57 were assigned to the higher functioning group (HIGH; n=10) with all others assigned to the lower functioning group (LOW; Cs-PFP TOT < 57; n=10). Dependent variables included gait speed, stride length, steps/day, and number of tasks reported with modification.

Results. HIGH had significantly faster gait speed (HIGH: 1.57 ± .3; LOW: 1.14 ± .3 m/s), longer stride length (HIGH: 0.76 ± 0.1; LOW: 0.62 ± 0.1 m), and more steps/day (HIGH: 9,503 ± 4,623; LOW: 5,048 ± 2,917) compared to LOW ($p < .05$). Groups reported having difficulty with a similar number of tasks (HIGH: 0.4 ± 1; LOW: 1.0 ± 1) but LOW reported modifying a significantly larger number of tasks (HIGH: 0.3 ± 1; LOW 1.4 ± 1). Gait speed, stride length, steps/day, and number of tasks modified were significant predictors of physical functional reserve.

Conclusions. Individuals compensate for reduced mobility by modifying the method of performing a task. Older adults with lower physical function also exhibit reduced

mobility compared to older adults with high function despite a similar independent living status.

Introduction

Aging, inactivity, and disease can slowly degrade the abilities of older adults necessary to living independently (1, 2). Losses in ability result in functional limitations that can accumulate and develop into disability (3, 4). Prevention of disability is a priority of aging research as nearly 40% of individuals 65 years and older exhibit limitations in the ability to carry out daily activities (5). This age category is the fastest growing segment of the population and may lead to increased prevalence of disability with associated increases of health care costs (6).

Physical reserve is the physiological capacity in excess of that needed during daily activities (7) or the difference between an individual's peak physical capacity and the level of ability required to perform activities independently. Typically, age-related losses in peak physical capacity begin in the 4th decade, but incident age-associated disability does not increase until approximately age 75 (8). During this time-span, age, inactivity, and/or diseases erode physical reserve by decreasing maximal or peak ability without a concomitant loss in physical function. Physical reserve provides a margin of safety that absorbs age or disease-related changes without a resulting loss in function (9). As physical reserve deteriorates, individual's approach a threshold of independence below which any further loss of peak capacity is associated with an 8 to 17-fold decrease in physical function (8). If physical capacity falls below the level of ability required for performance of daily tasks, then resultant functional limitation can bring about loss of independence (7). Disability occurs when physical demands of the current living status are greater than individual physical capacity (4). In spite of declining function, many individuals remain in homes that require high levels of physical function.

Individuals who maintain independence with physical capacities near or below the threshold have been identified as having pre-clinical disability (2, 10). These individuals develop strategies to modify the method of task performance in order to accommodate physical changes and remain independent (11). Task modification strategies reduce physical demand on the individual by altering the environment or modifying procedures, time spent, or frequency of performance of daily tasks (4, 12). These self-initiated strategies can be used to forestall changes in living status that may be necessitated by loss of physical ability. Individuals with pre-clinical disability remain independent yet modify task performance and are therefore at a greater risk of progressing to dependency with subsequent losses in their physical capacities (2).

Disability is typically first detected within the domain of mobility (10, 13). Frequently measured parameters of mobility include gait speed, stride length, and number of steps taken per day (10, 14-16). Declines in mobility, including gait speed and stride length, have been shown to predict disability in older adults (10, 17) but the relationship of daily mobility (steps/day) to independence has not been well examined. For the transitional stage of pre-clinical disability, the subtleties of changes in mobility during early stages of decline are less well defined. Changes in mobility are most likely to be evident during higher effort assessments such as the 6-minute walk, or over longer time frames such as a 7-day average of steps/day. This study provides unique insight into differences in mobility for a group nearing dependency and accommodating physical declines when compared to higher functioning individuals.

The purpose of this study was to characterize mobility and task modification of individuals living independently yet below the threshold of independence in physical

function. The threshold criteria used was 57 on the Continuous scale Physical Functional Performance Test. We believe these individuals represent a sub-population of independent community dwellers with pre-clinical disability. We hypothesized that individuals living independently but with performance below the threshold of independence would have slower gait speed, shorter stride length, fewer steps/day, and modify more tasks than those above the threshold. The ability to detect individuals with little symptomology but at high risk for disability would allow for early intervention to those who desire to remain independent (2).

Methods

Participants

We recruited 20 older adults (aged 65-95) for participation in this cross-sectional study. Eligibility criteria included men and women 65 years and older living independently within the community. Thirty-four individuals responded to various recruitment strategies including fliers and radio announcements. Respondents were assessed by SF36 Physical Function domain to include individuals scoring greater and less than 85 to ensure a broad range of physical abilities. Scores of greater than 85 on the SF36PF are more likely to occur in older adults without chronic conditions and associated disability (18, 19). A score of less than 85 is associated with a transition to disability (20, 21). Exclusion criteria included: inability to walk, unstable cardiovascular disease or diabetes, unhealed bone fracture, severe hypertension, or leg amputation. Twenty-two respondents were cleared by their personal physician with two individuals declining to participate. Participants reviewed and signed a consent form approved by the Institutional Review Board Human Subjects Committee.

Eligible participants were classified into two groups based on physical functional performance as assessed by the Continuous scale Physical Functional Performance Test (Cs-PFP) (22, 23). Participants scoring ≥ 57 on the Cs-PFP were classified as higher functioning (HIGH) with all others classified as lower functioning (LOW). A Cs-PFP total score of 57 is associated with thresholds in oxygen consumption and strength that accurately predict functional limitations and dependency in living status (8). Participants completed a self-reported instrument of task performance (difficulty and modification) and performance-based mobility measures including stride length, walk speed, and steps/day.

Physical Functional Performance

Physical functional performance was determined using the Cs-PFP, a valid, performance-based measure specifically designed to discriminate function across a broad range of physical ability (22, 24). The Cs-PFP quantifies physical performance in 16 tasks important for living independently by measuring distance moved, time for task completion, and/or weight carried (22). Performance yields five domain scores including upper and lower body strength (UBS, LBS), upper body flexibility (UBF), balance and coordination (BALC), endurance (END), and a summary score (Cs-PFP TOT) (24). Scores range 0 to 100 with higher scores reflecting higher function (23). Description of test set-up, administration, and scoring has been published elsewhere (22, 23) and is available online (http://www.coe.uga.edu/cs-pfp/).

Self-reported Physical Function

The SF36 is a valid and reliable measure of health status (25). For this study, the physical functional domain (SF36PF) was administered for screening purposes. The

SF36PF consists of ten questions assessing health-related limitations on an array of strenuous to basic physical activities (26). Scores range from 0 to 100 with higher scores reflecting higher self-perceived function. Self-reported function was also determined by the SF12 questionnaire, a shorter, valid version of the SF36 (27, 28). Results include a physical (SF12 PCS) and mental component summary (SF12 MCS).

Task difficulty (TD) and modification (TM) were determined by a modified version of the Supplement on Aging Questionnaire, National Health Interview Survey (29). This questionnaire determines functional decline in individuals based on self-report (2). The questionnaire covers 27 different tasks related to mobility, upper extremity function, instrumental activities of daily living, and self-care. As the focus of this study is mobility, a subset of six tasks related to mobility were selected for analysis and reported here. Tasks include walking for ½ mile, 150 feet, and around the home, getting out of bed, walking up 10 steps, and walking down 10 steps. Results are reported as the number of tasks with or without difficulty and the number requiring modification.

Mobility Performance

Performance-based mobility was assessed by gait speed, stride length, and average steps/day. One Cs-PFP tasks is the 6-minute walk, a commonly used measure of endurance appropriate for older populations (30, 31). Participants were instructed to cover as much distance as possible on a closed walking course while wearing a DigiWalker Stepcounter. The stepcounter is a valid, reliable pedometer that displays total number of steps taken since the counter was last reset (32, 33). Stride length (m) was determined by dividing total distance covered during the 6-minute walk by number

of steps taken. Gait speed (m/s) was determined by dividing total distance during the 6-minute walk by time spent walking.

Average daily walking habits (steps/day) were assessed by stepcounter over a seven-day period. Participants were instructed on placement of the pedometer before use. A walking log was given along with instructions on how to record number of steps per day. Participants wore the pedometer for seven consecutive days. At the end of each day, number of steps taken for that day was recorded on the walking log. Participants mailed the completed walking log to the investigator.

Statistical Analysis

Statistical analyses were performed using SPSS version 10.0 (SPSS Inc., Chicago, IL). Independent t-tests were used to detect group differences in physical function, task difficulty, task modification, gait speed, stride length, and number of steps taken per day. A discriminant-loading approach to predictive discriminant analysis was used to determine significant predictors of physical functional reserve as determined by the threshold of independence. Significance was set at alpha = .05

Results

Selected physical characteristics and functional outcomes are listed in Table 3.1. All participants lived in their own home (95 %) or apartment (5%). The majority of the sample lived with a spouse (80 %) with all others living alone. Physical functional performance was significantly higher in the HIGH group (TOT $p < .001$; CI: HIGH 62.5-79.6, LOW 42.8-52.0). The HIGH group had significantly greater performance for all domain scores of the Cs-PFP as shown in Table 3.1 (UBS $p = .018$; CI: HIGH 65.4-87.9, LOW 57.4-66.4. LBS $p = .001$; CI: HIGH 53.6-77.6, LOW 35.0-47.2. UBF $p = .018$; CI:

HIGH 72.2-84.9, LOW 57.2-74.8. BALC $p < .001$; CI: HIGH 57.4-74.3, LOW 34.1-44.5. END $p < .001$; CI: HIGH 65.9-80.9, LOW 41.6-52.2). The HIGH group had significantly higher self-rated physical component scores (SF12 PCS $p = .004$; CI: HIGH 49.8-55.9, LOW 38.0-48.7) but similar mental component scores (SF12 MCS $p = .795$; CI: HIGH 51.1-59.5, LOW 50.5-58.8) and physical function scores (SF36PF $p = .051$; CI: HIGH 81.5-95.5, LOW 70.2-86.8).

The HIGH group had significantly higher performance on all measures of mobility including gait speed (m/s) ($p = .004$; CI: HIGH 1.35-1.80, LOW 0.94-1.34), stride length (m) ($p = .015$; CI: HIGH 0.72-0.80, LOW 0.51-0.72), and steps/day ($p = .019$; CI: HIGH 6,195.4-12,810.1, LOW 2,961.7-7,134.8) (Figure 3.1, 3.2). One potential outlier maintained approximately 20,000 steps per day. This participant was still involved with full-time work requiring high amounts of walking. Because the participant was passed the age of retirement, met all criteria for the study, and intended to remain employed, this data point was included in the analysis. Groups did not differ in number of tasks reported as difficult (TD $p = .331$; CI: HIGH –.5-1.3, LOW –.01-2.0) but there was a significant difference in number of tasks being modified (TM $p = .047$; CI: HIGH -.18-.78, LOW .38-2.4) (Figure 3.3).

Predictive discriminant analysis was conducted to determine whether age, task modification, stride length, gait speed, and steps/day could predict individuals above the threshold of independence. The overall Wilk's lambda was significant, $\Lambda = .287$, X^2 (5, $N = 20$), $p = .008$. Based upon discriminant loadings, gait speed, stride length, steps/day, and task modification are predictors of physical functional reserve (Table 3.2). The group means on the discriminant function were 1.4 for HIGH and –1.6 for LOW.

Prediction of group membership correctly classified 94.1% of individuals with a kappa value of .881, indicating moderately to highly-accurate prediction.

Discussion

This study demonstrates that individuals below the threshold of independence have shorter stride length, slower gait speed, fewer steps/day, and more task modification than individuals above the threshold. These changes are evident despite all subjects living independently within the community and having similar physical characteristics. Groups did not differ in mental capabilities suggesting any differences in functional performance can be attributed to physical differences.

In the HIGH group, the average functional performance was 14.0 units above the threshold. This difference is a measure of physical reserve. The LOW group performance was 9.6 units below the threshold, an indicator of pre-clinical disability. Group means on self-reported physical function (SF36PF) ($p = .051$) approached significance and provide some insight as a score of less than 85 on the SF36PF is associated with a transition to disability (20, 21). In our sample, the LOW group perceived themselves to be at a level of function associated with transition to disability and performed below the threshold of independence despite a living status similar to the HIGH group. These characteristics are indicative of pre-clinical disability.

The HIGH group had greater mobility performance on stride length, gait speed, and steps/day. These measures were taken during a fast walk and should not be considered as parameters of usual gait. Langlois and colleagues (1997) suggest 1.22 m/s is the minimal gait speed needed to cross the street safely. In the HIGH group, average

walk speed was 1.57 m/s, another indication of physical reserve. In the LOW group, an average walk speed of 1.14 m/s was below this suggested minimum.

Performance measures of daily activity (steps/day) were significantly different between groups. Reduced mobility may place individuals at greater risk of becoming dependent (34). We believe this work to be the first to show global mobility as assessed by steps/day in relation to physical function. We found a significant difference in number of steps per day with the HIGH group having 1.88-fold greater steps/day despite similar living status and environmental demand. Studies suggest 10,000 steps/day are associated with significant health benefits (15, 35, 36). Our results indicate individuals who average approximately 10,000 steps/day and have a peak that exceeds 10,000 steps/day should have significantly higher physical function, which is also above the functional threshold indicative of greater independence. Individuals classified LOW averaged approximately 5,000 steps/day and could be at increased risk of disability requiring perhaps a single event to thrust them below the ability to remain independent.

LOW group modified significantly more mobility tasks than the HIGH group. Modification of tasks is a primary characteristic of individuals in the transitional stage of pre-clinical disability (10). Individuals who perform below the threshold of independence yet remain of independent living status are compensating for declines in physical capacity by altering methods used to accomplish the task. Gait speed, stride length, steps/day, and number of tasks modified were predictors of physical functional reserve. Guralnik (1995) also found that difficulty with mobility predicted future disability. We did not directly assess disability risk, but individuals classified as LOW exhibited difficulty with mobility and mobility-related tasks and could thus be at high

risk for disability. This study supports mobility as a factor essential for independence and adequate functional capacity. Characteristics of individuals classified as pre-clinical disability (LOW) include reduced stride length, gait speed, steps/day, and modification of more tasks than the HIGH group.

A limiting factor of this cross-sectional study design is inability to assess temporal sequence of the relationship between mobility and physical function. Also, participants met criteria for inclusion, but a variety of acute and chronic diseases that effect the outcomes could be undetected yet existent. The authors were unaware of the amount of social support each individual received within their environment, particularly those with spouses or immediate family. Individuals receiving high levels of support from others could maintain independence despite having reduced physical capacities and reserves. In future studies, these individuals need to be studied over time to understand if lower mobility leads to institutionalization and dependency.

In conclusion, gait speed, stride length, steps/day, and task modification are associated with physical reserve in older adults. Individuals with greater capacity in mobility and task performance tend to retain adequate physical reserves in later life. Individuals performing below the threshold of independence exhibit lower mobility characteristics and may be at a stage of pre-clinical disability that has a greater risk of dependency. Interventions that target mobility are recommended in order to maintain independence in older adults.

Acknowledgments

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Table 3.1 Selected demographic, physical, and functional characteristics of individuals above and below the threshold of independence

Measure	HIGH (n = 10)	LOW (n = 10)	p value
Age (years)	71 ± 6	75 ± 5	0.093
Height (cm)	167.7 ± 10	170.0 ± 11	0.619
Weight (kg)	74.1 ± 16	75.1 ± 15	0.897
Female (%)	70	70	
Cs-PFP TOT	71.0 ± 12	47.4 ± 6	<0.001
Upper Body Strength	76.6 ± 16	61.9 ± 6	0.018
Lower Body Strength	65.6 ± 17	41.1 ± 9	0.001
Upper Body Flexibility	78.5 ± 9	66.0 ± 12	0.018
Balance/Coordination	65.9 ± 12	39.3 ± 7	<0.001
Endurance	73.4 ± 10	46.9 ± 7	<0.001
SF12 PCS	52.8 ± 4	43.4 ± 8	0.004
SF12 MCS	55.3 ± 6	54.6 ± 6	0.795
SF36PF	88.5 ± 10	78.5 ± 12	0.051

Notes: Values are Means ± SD. HIGH = individuals scoring ≥ 57 on the Cs-PFP total; LOW = individuals scoring < 57 on Cs-PFP total; Cs-PFP TOT = Continuous scale Physical Functional Performance Test Total Score, SF12 PCS = SF12 physical component score, SF12 MCS = SF12 mental component score, SF36PF = SF36 physical function score

Table 3.2 Predictive discriminant analysis: Correlations of predictor variables for independence level in older adults

Variable	Correlational Coefficient
Age (years)	- 0.19
Task Modification	- 0.34
Stride Length (meters)	0.50
Gait Speed (meters/second)	0.56
Number of Steps/day	0.38

Notes: Correlation coefficients are the within-group correlations between the predictors and the discriminant function. A negative sign indicates inverse relationship.

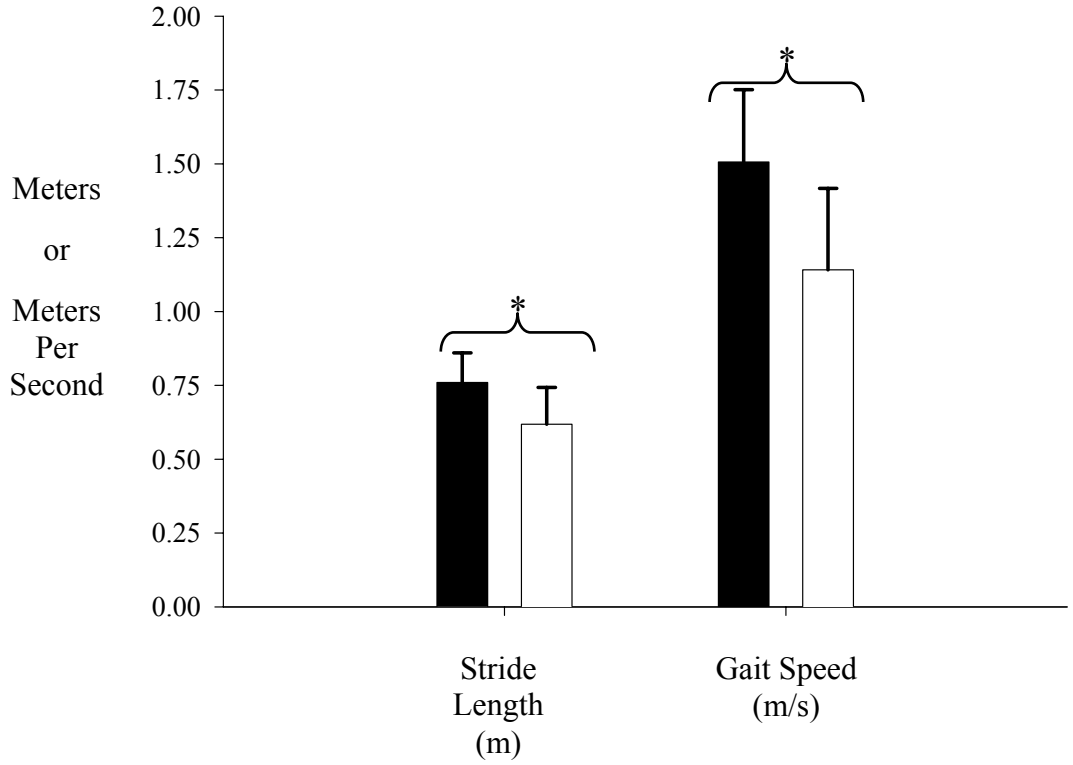
Figure Legends:

Figure 3.1: Differences in Stride Length and Gait Speed between High and Low Functioning Older Adults. Values are Means ± SD. $p < .05$, * = significant difference between groups. Closed bars = HIGH, Open bars = LOW, HIGH = individuals scoring ≥ 57 on the Cs-PFP total; LOW = individuals scoring < 57 on Cs-PFP total; Stride expressed in meters (m); Walk Speed expressed in meters per second (m/s).

Figure 3.2: Differences in Average and Peak number of Steps/Day between High and Low Functioning Older Adults. Values are means ± SD. $p < .05$, * = significant difference between groups. Closed bars = HIGH, Open bars = LOW, HIGH = individuals scoring ≥ 57 on the Cs-PFP total; LOW = individuals scoring < 57 on Cs-PFP total; Average = 7-day average steps/day; Peak = One day peak during seven day pedometer testing.

Figure 3.3: Differences in Number of Tasks reported Difficult or Requiring Modification between High and Low Functioning Older Adults. Values are Means ± SD. $p < .05$, * = significant difference between groups. Closed bars = HIGH, Open bars = LOW, HIGH = individuals scoring ≥ 57 on the Cs-PFP total; LOW = individuals scoring < 57 on Cs-PFP total

Figure 3.1


2.00
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Length
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Figure 3.2

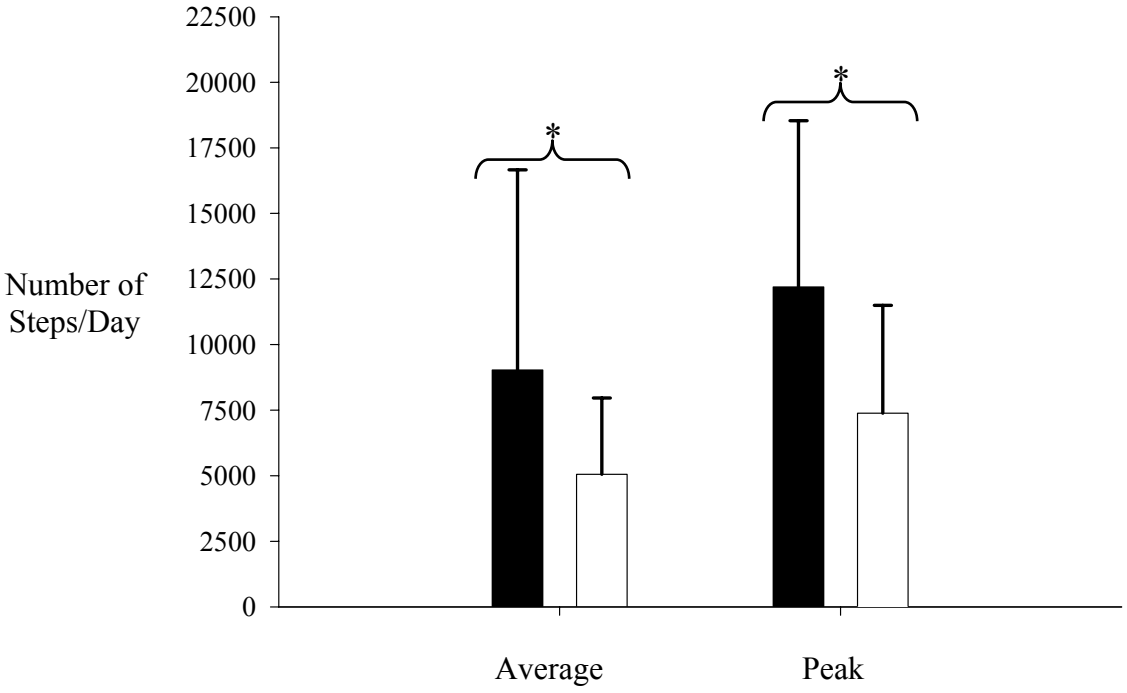
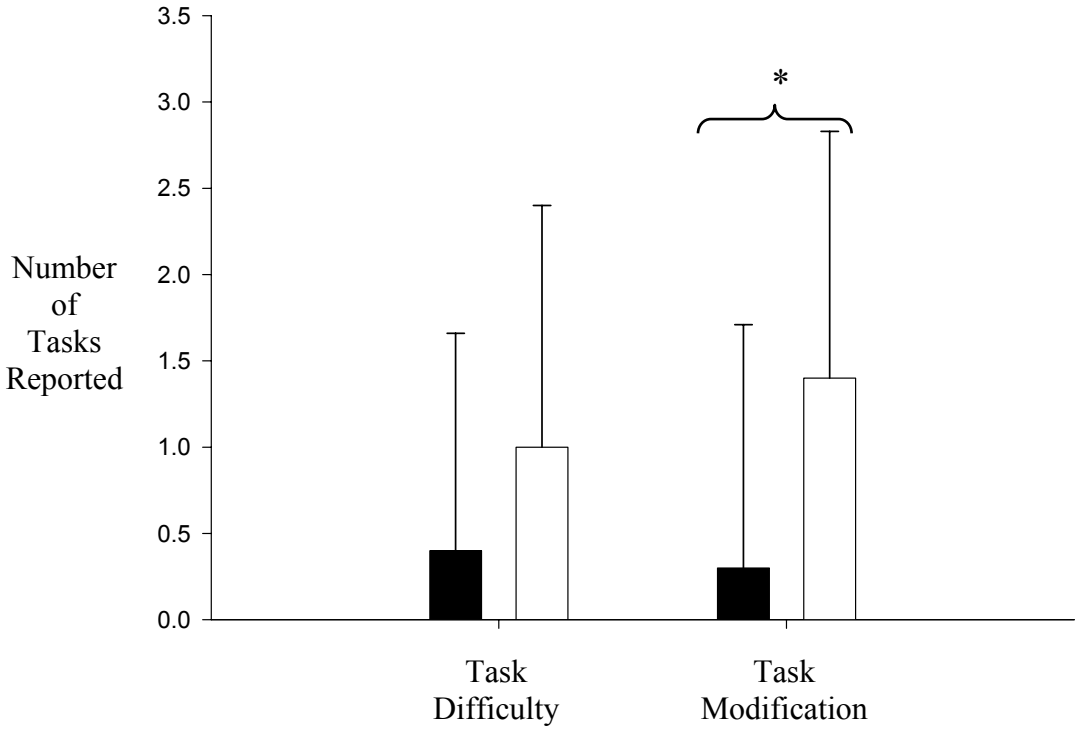

Number of Steps/Day
22500
20000
17500
15000
12500
10000
7500
5000
2500
0
*
*
Average
Peak

Figure 3.3


3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
Number of Tasks Reported
*
Task Difficulty
Task Modification

CHAPTER IV

RECOVERY OF MUSCLE AND PHYSICAL FUNCTION FOLLOWING TOTAL KNEE ARTHROPLASTY[1]

[1] Petrella, J.K., M.E. Cress, M.S. Ferrara, G.A. Dudley, O.M. Mahoney. To be submitted to *Journal of Gerontology: Biological Sciences*.

Abstract

Introduction. Osteoarthritis (OA) develops over two to four decades reducing physical abilities. Total knee arthroplasty (TKA) is a surgical intervention recommended for reinstalling function and mobility. The purpose of this study is two-fold: 1) To examine decrements in joint, muscle, and physical function associated with OA, and 2) To examine time course of recovery in joint, muscle, and physical function after TKA.

Methods. Thirteen candidates for TKA were compared to controls prior to surgery (baseline) with follow-up testing one month and three months post-surgery. Outcome measures included use-related pain, range of motion, isometric peak force, muscle activation, force per cross sectional area, extensor power, perceived function, and performance-based physical function.

Results. At baseline, TKA group reported greater use-related pain compared to controls which decreased 83% ($p < .05$) by one-month. From one month to three months post-surgery, the TKA surgical limb increased range of motion by 13%, muscle force by 16%, extensor power by 55%, force/CSA by 13%, and steps/day by 46% ($p < .05$). Concurrent to these changes, physical function of the TKA group improved 19% ($p < .05$). Change in functional range of motion in the surgical limb of the TKA group explained 54% of the variance in physical function recovery.

Conclusions. Use-related pain is alleviated within one month of surgery followed by improvements in range of motion, strength, force per cross sectional area, extensor power, and activity. Individuals recovering from TKA surgery improved physical function to a level predictive of independent living by three months post-surgery.

Introduction

Osteoarthritis (OA) is the most common non-inflammatory joint disease in the United States affecting more than 20 million Americans or 12.1% of adults (1). The pathology of OA develops over two to four decades, reducing an individual's physical ability and increasing the risk of disability (2, 3). OA effects multiple physiological systems including joint structure and muscle function. Pain, particularly use-related pain, is the most prevalent symptom of OA (4, 5). Use-related pain typically results from altered joint structure and is considered a primary impairment of OA (6-8). OA-related damage to the cartilage results in pain and joint effusion that can lead to disuse-related atrophy of the muscle (9). Reduced activity can cause decreased strength and reduce the physical capacity of individuals necessary for remaining independent. (10-12). Thus, a secondary impairment of OA that can be masked by the prevalent symptom of pain is disuse-related muscle weakness.

Progression of OA can lead to disuse and the depletion of physical reserves. Strategies can be employed in order to maintain function and forestall disability including altered or reduced activity, use of medication for pain relief, and modification of tasks. If these strategies have failed to provide sufficient pain relief and function, total knee arthroplasty is a surgical intervention that is often recommended. By alleviating the symptom of use-related pain, TKA should provide the opportunity for reinstalling function and mobility. Increased mobility would be expected to increase muscle function and allow for the rebuilding of physical reserve. Increasing muscle strength can be expected to provide a greater physical reserve and a "margin of safety" against the dangers of acute and chronic disease on physical function. However, patients who have

had TKA often exhibit quadriceps muscle weakness for as long as two years following surgery (13). Continued quadriceps weakness may be due to several factors including pain-related disuse, psychological factors, selective inhibition, or failure to activate available muscle fibers (14).

Individuals who maintain their independence but have reduced levels of peak strength represent a stage in the disablement process called pre-clinical disability. In pre-clinical disability, an intermediary stage of disablement that precedes and predicts disability (15), individuals can reduce the demand of their current environment by modifying task performance without sacrificing their independence (15). Individuals who report task modification are at increased risk of disability, particularly mobility-related disability (15, 16).

The effects of total knee arthroplasty on improvements in muscle and physical function are unclear. Understanding recovery of joint function and quadriceps muscle and the resulting effect on physical function can provide valuable information for rehabilitation interventions aimed at improving physical function independence. The purposes of this study are to examine: 1) decrements in joint, muscle, and physical function associated with OA, and 2) the time course of recovery in joint, muscle, and physical function after total knee arthroplasty. We hypothesized that prior to total knee arthroplasty, individuals with end-stage OA would have reduced joint, muscle, and physical function compared to the non-surgical limb or matched control. We also hypothesize during three months recovery from TKA surgery, patients will have a greater change in joint, muscle, and physical function compared to matched controls. In addition, changes in physical function would be due to improvements in muscle strength

and pain-related use. This study will quantify functional changes as a direct result of TKA surgery. We will further examine the data for factors contributing to the recovery of physical function.

Methods

Participants

We recruited 15 older adults with degenerative OA of the knee from the Athens Orthopedic Clinic, Athens, Georgia. Eligibility criteria included men and women 20 years of age and older who were scheduled to have total knee arthroplasty within in the next six weeks. Patients were excluded from the study due to previous lower limb joint replacement, unstable diabetes, hypertension, cardiovascular or thyroid disease, any neuromuscular disease that would effect muscle strength, or any contraindications to magnetic resonance testing including pacemakers, aneurysm clips, or any other metal implants. Potential participants were identified by medical history and contacted by telephone for an explanation of the study design including any risks or benefits they would receive. All participants were recruited by the staff of the same orthopedic practice. The practice performed approximately 45 to 50 arthroplasties per month. We averaged two participants recruited per month. The orthopedic surgeon (OMM) cleared all participants who agreed to be in the study. Fifteen patients (TKA) volunteered for the study with nine scheduled for unilateral knee surgery and six for bilateral surgery.

Fifteen apparently healthy adults of the same sex and similar age and height to individuals in the TKA group were enrolled as a cohort comparison (CON). Eligibility criteria included adults of the same gender with similar age and height to TKA patients. Exclusion criteria were the same as TKA patients as well as no known chronic joint

disease of the lower limb. Once a respondent met the criteria and was an appropriate match to a TKA participant, a clearance form was sent to the individual's personal physician. Participants signed an informed consent from approved by the Institutional Review Board at the University of Georgia, Athens, Georgia.

Study Design

Thirteen candidates for TKA surgery were compared to controls of the same sex and similar age and height prior to surgery (baseline) with follow-up testing at one month and three months post-surgery. Once the TKA patient was assessed at the one month post-surgical time point, the control was scheduled for testing. After surgery, TKA patients participated in standard home-based physical rehabilitation followed by center-based rehabilitation. Outcome measures included use-related pain, joint range of motion, muscle characteristics and function, perceived physical function, and physical performance of daily tasks.

Joint Function

Use-related pain intensity and location on the body were assessed using an 11-point Numerical Graphical Rating Scale (17, 18). Validity and reliability has been tested in older populations (19). The 11-point scale was used to determine the anatomical location of pain while intensity was rated on a 0 to 10-point scale with higher scores reflecting greater intensity of pain. Pain scores were assessed after each trial of isometric muscle strength and after each task performed during the Continuous scale Physical Functional Performance Test (Cs-PFP). Participants were asked if they perceived any pain in the lower limb and the data was recorded.

Range of motion was assessed to the nearest degree using a goniometer (20). Participants were tested for active (AROM) range of motion on knee flexion and passive (PROM) range of motion on knee extension. For both flexion and extension of the knee, the fulcrum of the goniometer was aligned with the lateral midline of the femur using the greater trochanter for reference. The distal arm of the goniometer was aligned with the lateral midline of the fibula using the lateral malleolas for reference. A more detailed description may be found elsewhere (20).

Participants were supine with one knee bent and the foot flat on the table for knee flexion measurement. For AROM flexion, participants were asked to pull the foot as far back as possible without using the upper limbs for assistance. For PROM flexion, the lower leg was pushed back as far as possible within pain tolerance of the participant. For PROM extension, the leg was extended with the heel on a small box and the measurement was taken. The peak values of two trials were recorded for both limbs. Functional range of motion was calculated by peak flexion – peak extension. Assessments were made at the Exercise Vascular Biology Lab, University of Georgia.

Muscle Function and Characteristics

Isometric peak force (IVC_{peak}) of the quadriceps muscle of both limbs was assessed using a custom-made chair with a fixed 6" x 22" platform extending 70^{o} below the horizontal surface of the chair. The platform was fixed to a loadcell attached to a Macintosh microcomputer. Force was collected in foot-pounds at a sampling rate of 1,000 Hz. A rolled towel was placed beneath the knee and the leg to be tested was secured to the platform with a velcro strap. All measurements were corrected for the weight of the limb. Participants were verbally encouraged to straighten the leg "as hard

as possible" for three seconds. Three trials were performed with three minutes rest between trials. All strength values recorded in foot-pounds were converted to Newton-meters (Nm) with the correction factor of 1.0 foot-pound x 1.355818. Force per cross sectional area (force/CSA) was calculated by IVC_{peak} divided by quadriceps cross sectional area of contractile tissue (Nm/cm^2). Strength testing was assessed in the Muscle Biology Laboratory, University of Georgia.

The central activation ratio (CAR) is an assessment of voluntary muscle activation. Ability to activate the muscle was assessed concurrently with IVC_{peak} measurements using a burst superimposition technique. Two 6.98 x 10.16 cm electrodes (DynaMed Corp., Marietta, GA) were placed over motor points of the quadriceps muscles. Participants performed a three second peak isometric contraction. At the two-second mark of the IVC_{peak}, a 100 Hz 12-pulse burst was delivered to the quadriceps muscles by a stimulator (Digitimer Stimulator Model DS7A) using customized software (TestPoint Software 4.0, ©2000). Each participant was encouraged to be tested at a stimulation intensity of at least 100 mAmps (21). Ultimately, the intensity of stimulation was determined by the participant's tolerance level of the electrical stimulation. Any increase in force due to the external electrical stimulus suggests a failure to fully activate the muscle. The percent of muscle activated is calculated by the following formula (22-24):

CAR = (IVCpeak force / peak force during electrical stimulation) x 100

If CAR was less than 95%, subjects were encouraged to try harder after a three minute rest (22). Subjects were given three attempts to attain a CAR of 0.95 or greater. CAR measurements were taken at the Muscle Biology Laboratory, University of Georgia.

Average cross sectional area (CSA) of contractile and non-contractile tissue of the quadriceps muscles was determined using T1 proton-weighted magnetic resonance (MR) imaging. A total of 30 trans-axial MR images 1-cm thick spaced 0.5-cm apart were collected with a 1.5-Tesla magnet (TR/TE 500, 2.0 NEX, 256 x 192 matrix; General Electric, Milwaukee, Wis., USA) using a whole body coil. The echo and repetition times were selected to optimize the signal-intensity contrast between muscle and fat (25). Participant's feet were strapped together and knees and hips fully extended as determined by participant's pain tolerance. The total scan time was twelve minutes. All MR images were taken at St. Mary's Hospital Imaging Facility, Athens, Georgia.

MR images were downloaded to compact disk and analyzed on a LINUX Redhat operating system. A software program (X-vessel, East Lansing, MI) was used to outline the quadriceps muscles by tracing the perimeter of the region of interest (ROI) (25). The signal intensity threshold for contractile tissue was determined by a histogram plot of the ROI (26, 27). The number of pixels within the ROI was converted to CSA (cm^2) by spatial calibration of pixels to centimeters. The average CSA of the quadriceps was calculated by averaging the CSA of eight slices beginning with the first proximal slice containing no gluteal muscle and the next seven distal slices (28). This region of the thigh represents the maximal CSA of individual muscles of the quadriceps group (28, 29).

The Nottingham Leg Power Rig (Nottingham, England) was used to assess single leg extensor power (LEP) for each limb separately. The equipment consists of a backless chair that can be adjusted to accommodate the leg length and range of motion of the participant. Power is generated when the participant, using one foot, pushed the pedal

forward and down "as hard and as fast as possible" transmitting energy from the pedal by a connecting chain to the flywheel. The average power was derived from the final velocity of the flywheel (30). The task required approximately 250-300 ms to complete. Peak power was calculated and recorded in watts by the microcomputer (30). Each subject repeated this procedure until a plateau in LEP was achieved with a minimum of five trials, 30 seconds rest given between trials. Extensor power was assessed at the Aging and Physical Performance Laboratory, University of Georgia.

Physical Function

The Continuous scale Physical Functional Performance Test (Cs-PFP) is a performance-based measure of function specifically designed to discriminate across a broad range of physical function for older adults (31, 32). The Cs-PFP is used to quantify physical performance in 16 common tasks important for living independently. Functional performance is quantified by using the weight carried, time to task completion, and distance traveled. A modified version of the Cs-PFP that uses ten tasks was used (33). The shorter version contains tasks that are quantified by time including laundry transfer, putting on and removing a jacket, floor sweeping, climbing stairs, and getting up and down from the floor. Tasks that are quantified by time and weight include carrying a pan of weight and carrying groceries. Tasks that are quantified by distance include the 6-minute walk and highest reach (32). Testing requires approximately thirty minutes to complete and scores are scaled from 0 – 100 by utilizing the following formula based upon the lower and upper extremes of performance by previously tested older adults (32):

CS-PFP corrected task score = (observed score – lower limit) / (upper limit – lower limit) x 100.

Participants are asked to perform tasks as quickly as possible and with maximal effort but within their own perception of safety. Task performance yields five domain scores including upper and lower body strength (UBS, LBS respectively), upper body flexibility (UBF), balance and coordination (BALC), endurance (END), and a summary score (Cs-PFP Total) (32). Functional testing was assessed at the Aging and Physical Performance Laboratory, University of Georgia.

The SF36 is a valid measure of health status that contains 36 questions and yields eight domains (34). The eight domains assessed include self-perception of ability for physical function, role-physical (role limitations due to physical problems), bodily pain, general health, vitality/energy, social function, role-emotional (role limitations due to emotional problems), and mental health (34). Scores range from 0 to 100 with higher scores reflecting higher self-perceived function and health (35). The SF36 was administered prior to Cs-PFP testing so the participant did not gain insight into their level of function from the performance measure.

Task difficulty (TD) and modification (TM) were determined by a modified version of the Supplement on Aging Questionnaire, National Health Interview Survey. (68) This questionnaire assesses functional decline in individuals who may not be identified by other methods (36, 37). Ten tasks related to mobility were selected for analysis including walking for ½ mile, walking for 150 feet, walking around the home, getting out of bed, doing heavy housework, getting out of a car, walking up 10 steps, walking down 10 steps, stooping/crouching/kneeling, and cutting one's own toenails. Participants indicated if a task was performed with or without difficulty or modification. The number of tasks modified (TM) and identified as being difficult (TD) were recorded.

The Instrumental Activities of Daily Living (IADL) Questionnaire was used to assess difficulty with eight complex tasks important for remaining independent (38). Participants assessed the level of difficulty with performance of tasks including housework, car/bus travel, shopping, meal preparation, laundry, financial management, self-medication, and use of the telephone. Scores range from 0 – 24 with higher scores reflecting higher function.

Activity

A pedometer was used to assess average number of steps/day. This valid and reliable instrument displays the total number of steps taken since the pedometer was last reset (39, 40). The pedometer contains a horizontal, spring-suspended lever arm that, when worn on the waist over the midline of the leg, measures the vertical accelerations of the hip. With each vertical displacement, an electrical contact is made and a step is recorded. Participant recorded the daily number of steps taken for seven consecutive days on the walking log.

Self-reported physical activity was assessed by using the Older Adult Exercise Status Inventory (OA-ESI), a self-reported 7-day recall specifically validated on older adults (41). Activity is organized into 38 exercise/leisure categories and five indoor/outdoor work categories. The day of the week and number of minutes spent in an activity was recorded. Expenditure in kilocalories (kcals) was estimated by the following formula (41):

[(# minutes spent in activity / 60) x MET x body weight in kg] / 7 days

Results are reported as daily work energy expenditure, daily leisure energy expenditure, and total daily energy expenditure in kilocalories (kcals).

Statistical Analysis

The independent variable of the study was TKA surgery (CON group verses TKA group, n = 13). The control group was matched to the TKA group on sex, age, and height to reduce 3rd variables that may be influencing the outcome measures. Outcome measures for joint function included use-related pain ratings and functional range of motion. Outcome measures for muscle function included IVC_{peak}, pain during IVC_{peak}, force/CSA, CAR, LEP, pain during LEP, and quadriceps CSA of contractile and non-contractile tissue. Outcome measures for physical function included Cs-PFP total , SF36 domains, IADL's, task difficulty, and task modification. Outcome measures for activity included number of steps/day and kcals/day.

A series of one-way repeated measures analysis of variance were used to examine the effects of TKA surgery on muscle and physical function at the pre-surgical time-point with follow-up at one month and three months post-surgery. For the testing of muscle characteristics and function, we compared the surgical limb of the TKA group (TKA-S) to the matched surgical limb of the control group (CON-S). Since the sample included patients that had bilateral surgery, the weakest limb at baseline was selected for analysis as the surgical limb.

In order to include testing of limb to limb comparisons within the same participant, a secondary data analysis of unilateral TKA patients (TKA_{UNI} verses CON_{UNI}, n = 9) was conducted using a 2 x 3x 3 mixed model repeated measures analysis of variance. The primary statistic examined was the interaction term of the repeated measures ANOVA. Contrasts were planned to compare the non-surgical limb of TKA_{UNI} to the control matched non-surgical limb (TKA_{UNI}NS to CON_{UNI}NS) and the within-

subjects factor of surgical limb to non-surgical limb ($TKA_{UNI}S$ to $TKA_{UNI}NS$). The family-wise error rate was corrected using a modified Bonferonni adjustment (42):

$$\alpha_{family\text{-}wise} = [\ (\alpha) * (\text{Degrees of Freedom}_{\text{Between Subject}})\ ] / \#\text{ of planned contrasts.}$$

Outcome measures that contain the additional limb to limb analysis of the unilateral TKA patients include functional range of motion, IVCpeak, force/CSA, CAR, LEP, and quadriceps CSA.

Pearson's correlation of change scores from pre-surgery to one and three months post-surgery were used to detect significant relationships among performance variables and physical function. Forward stepwise linear regression was used to explain the variance in recovery of physical function after TKA surgery. Significance was set at $\alpha = .05$ except for contrasts which were adjusted to $\alpha = .0167$.

Results

Participants

Of the thirty participants, twenty-six completed all time points of the study. Four participants (TKA, n = 2; CON, n = 2) missed one follow-up time point for reasons unrelated to the study. Two participants (TKA, n = 1; CON, n = 1) missed the MR imaging appointment of the one-month post-surgical time point due to scheduling conflicts. To account for the missing MR data, values for the second time point were calculated by interpolation of the first and third time point values recorded for the participants (43).

At baseline, there were no significant differences between TKA and CON for age ($p = .569$) and height ($p = .525$) (Table 4.1) but TKA weighed significantly more than CON at all time points ($p < .05$). Neither TKA or CON significantly changed in weight

over the course of the study. TKA consisted of seven males and six females with one African American and one Hispanic individual. The CON group consisted of eight males and five females with two African Americans.

Joint Function

At baseline, TKA reported significantly more use-related pain compared to the CON (Figure 4.1). Additionally, the surgical limb of the TKA group had 23% less ($p < .05$) functional range of motion than the control-matched limb (Table 4.2). Upon further analysis, the surgical limb of TKA_{UNI} had 15% less functional range of motion than the non-surgical limb ($TKA_{UNI}S = 102.2 \pm 14^{o}$, $TKA_{UNI}NS = 120.2 \pm 6^{o}$, $p < .05$). The TKA_{UNI} non-surgical limb had 15% less functional range of motion compared to CON_{UNI} non-surgical matched limb ($TKA_{UNI}NS = 120.2 \pm 6^{o}$, $CON_{UNI}NS = 142 \pm 5^{o}$, $p < .05$).

The changes in use-related pain are reported in Figure 4.1. After surgery, use-related pain decreased 83% ($p < .05$) in TKA group by one month post-surgery with no further change at three months post-surgery. At three months follow-up, TKA reported significantly greater use-related pain ($p < .05$) than CON. TKA surgical limb significantly increased functional range of motion by 13% from one month to three months post-surgery (Table 4.2). Upon further analysis, there was a significant change from baseline in the TKA_{UNI} non-surgical limb (Pre-surgery: 120.2 ± 6^{o}, 3 mo: 129 ± 7^{o}, $p < .05$).

Muscle Function and Characteristics

At baseline, TKA surgical limb had less strength, muscle activation, and extensor power compared to control matched surgical limb (Table 4.3). No differences were detected between TKA-S and CON-S on quadriceps CSA contractile and non-contractile

tissue. Force/CSA in the TKA-S was 41% less than CON-S at baseline ($p < .05$) (Figure 4.2). Upon further analysis, TKA_{UNI} surgical limb had less strength compared to the non-surgical limb (91.6 ± 45 verses 176.2 ± 99 Nm, $p < .05$) at baseline but there was no difference between the TKA_{UNI} non-surgical limb and control matched non-surgical limb in muscle strength. TKA_{UNI} surgical limb had less muscle activation during strength testing compared to the non-surgical limb (86.4 ± 7 verses 95.3 ± 3%, $p < .05$) with no difference between TKA_{UNI} non-surgical limb and CON_{UNI} matched non-surgical limb ($TKA_{UNI}NS$ = 95.3 ± 3%, $CON_{UNI}NS$ = 93.7 ± 11%, $p = .614$). In the TKA_{UNI} group, leg extensor power was less in the surgical limb compared to the non-surgical limb (99.0 ± 65 verses 188.9 ± 115 watts, $p < .05$) and no difference between $TKA_{UNI}NS$ and $CON_{UNI}NS$ (188.9 ± 115 verses 242.4 ± 131 watts, $p = .527$) at baseline. Force/CSA in $TKA_{UNI}S$ was 38% less than $TKA_{UNI}NS$ ($p < .05$). There was no difference in force/CSA or quadriceps CSA contractile and non-contractile tissue between $TKA_{UNI}NS$ and $CON_{UNI}NS$ at baseline.

During follow-up, TKA surgical limb improved 16% ($p < .05$) in IVC_{peak} from one to three months post-surgery. Muscle activation significantly increased in the surgical limb by one month with no further change at three months (Table 4.3). Leg extensor power increased 55% ($p < .05$) in TKA surgical limb by three months follow-up (Table 4.3) and force/CSA increased 13% ($p < .05$) from one month to three months post-surgery (Figure 4.2). Upon further analysis, no significant recovery was detected in the non-surgical limb of TKA_{UNI} for strength, muscle activation, extensor power, force/CSA, or quadriceps CSA by three months follow-up.

Physical Function

Figure 4.3 and Table 4.4 shows the progression of physical function (Cs-PFP total and domain scores) for both TKA and CON during the three-month follow-up from surgery. Prior to surgery, TKA was significantly lower than CON in physical function by 37% ($p < .05$). No differences were detected in IADL performance at baseline or during follow-up (Table 4.5). Compared to CON, TKA reported on average 1.0 more tasks ($p < .05$) that required modification and 6.9 more tasks ($p < .05$) that were considered difficult to perform when assessed pre-surgery (Table 4.5).

CON improved 7% ($p < .05$) in physical function from pre-surgery to one-month post-surgery while TKA improved 19% ($p < .05$) on physical function assessment from one month to three months post-surgery (Figure 4.3). Despite the significant improvement, TKA exhibited physical function 43% lower ($p < .05$) than CON group at three months post-surgery. Based on self-report, TKA increased physical function and the roles associated with that function from one to three months follow-up from surgery (Table 4.5). Self-reported pain also improved significantly ($p < .05$) from one to three months follow-up. All other domains are summarized in Table 4.5. The number of tasks reported difficult by TKA decreased to 4.15 tasks ($p < .05$) on average at one month follow-up and 2.08 tasks ($p < .05$) on average at three months. TKA also reported, on average, one positive task modification by three months surgery though this was not statistically significant.

Activity

Activity increased 46% ($p < .05$) in TKA group from one to three months as assessed by daily number of steps/day (Table 4.6). No changes were detected in self-reported activity.

Predictors of Recovery

To determine factors that contribute to the change in physical function from one to three months post-surgery, Pearson's correlation of change scores for all performance-based variables exhibiting significant increases one to three months were correlated to the change in function score (Cs-PFP total). Variables that increased in the surgical limb during that period of follow-up included force/CSA, IVC_{peak}, LEP, steps/day, and functional ROM (Figure 4.2, Tables 4.2, 4.3, 4.6). The only variables to significantly correlate with change in Cs-PFP total was change in functional range of motion in the surgical limb (TKA-S: $r = .643$, $p = .000$). The change in functional range of motion in TKA-S explained 54.1% ($p = .000$) of the variance in Cs-PFP total recovery.

Discussion

The major finding of this study was that TKA surgery resulted in a significant increase of physical function by three months post-surgery. The increase in physical function occurred from one month to three months post-surgery and was accompanied by significant improvements in joint and muscle function. Pain was alleviated by one month post-surgery while muscle and physical function showed recovery from one month to three months post-surgery. Thus, pain was relieved early in the recovery process, however muscle and physical function took longer to recover.

Prior to TKA surgery, individuals with diagnosed OA had reduced joint, muscle, and physical function when compared to the non-surgical limb and to matched controls. Joint function at baseline was characterized by significant use-related pain and reduced range of motion. Muscle function was impaired in the surgical limb of those scheduled for TKA surgery but not in the non-surgical limb. Impairments in muscle function included reduced strength, force/CSA, and extensor power compared to the non-surgical limb and to matched controls. Physical functional performance was also reduced at baseline suggesting an increased risk of loss of independence and disability.

The improvements in joint function following TKA surgery were characterized by significant relief from use-related pain and increased functional range of motion. By replacing the worn cartilage and eliminating bone on bone articulation, TKA surgery corrects the primary cause of use-related pain associated with OA. We found significant pain relief at one month follow-up with no further change at three months. At the end of three months follow-up, the TKA group was still experiencing some use-related pain yet this was 83% less than the levels of pain reported prior to surgery. The time course of pain relief at one month is earlier than previously reported findings of pain relief noted at three months post-surgery (44). Despite this significant reduction in pain, we found no changes in strength or physical function concurrent with pain relief. Once symptomatic pain is alleviated, it may be that individuals can begin an enabling process that increases range of motion, strength, force/CSA, extensor power, and activity resulting in improvements of physical function.

Muscle function in the surgical limb of TKA patients improved significantly from one to three months post-surgery. The TKA group showed increased strength,

force/CSA, and extensor power in the surgical limb. LEP increased from surgery to three months while IVC_{peak} increased from one to three months post-surgery. The changes in strength are most likely due to neural factors. Neural deficits of up to 20% have been detected in the voluntary activation of quadriceps muscles of individuals prior to TKA surgery (23). Measures of central activation showed significant improvements from surgery to one month post-surgery with no further changes detected. The increase in muscle activation was not accompanied by any significant changes in muscle strength or cross sectional area. Not all participants could tolerate the target stimulation of 100 mAmps calling into question the ability to maximally activate the muscle by electrical stimulation and artificially increase estimated muscle activation.

Peak force per unit of muscle mass (force/CSA) estimates specific tension of muscle (45, 46). Strength improved significantly from one to three months but without concomitant increases in muscle CSA. On average, the surgical limb was 21% smaller in quadriceps muscle contractile tissue and contained 30% more non-contractile tissue compared to controls. These CSA values were stable throughout recovery, but there was a significant change in force/CSA suggesting improved muscle quality either through increased synchronicity, activation, recruitment, or decreased inhibitory factors.

Several measures of physical function increased during recovery. We detected significant recovery of performance-based physical function within three months post-surgery. The recovery in physical function during this short-term follow-up was significant enough to elevate the TKA group above the threshold of independence. A score of 57 on the Cs-PFP is associated with thresholds in oxygen consumption and strength that accurately predict functional limitations and dependency in living status

(47). At baseline, the TKA surgery group was eight units below the threshold (Cs-PFP threshold of 57 – TKA group average of 49) but improved to one unit above the threshold by three months. One of the primary goals given by individuals with OA is the improvement of function (4) and this sample was successful in significantly improving function by three months of follow-up. There was also a small but significant change in physical function for the control group from pre-surgery to one-month follow-up with an effect size = .52.

By in large, despite decrements in physical reserve, this TKA group was not disabled at baseline and did not change during follow-up. IADL levels were near the ceiling of the measure. Results from self-reported quality of life assessments were aligned with performance-based measures during recovery. The TKA group reported improvements in self-rated pain, physical function, and the social roles associated with physical ability (SF-role physical). This finding was supported by the TKA group reporting fewer tasks being difficult by the end of follow-up. There was a small increase in the number of tasks requiring modification by one-month surgery but that number returned to baseline levels by three months. TKA group reported several positive modifications in task performance such as alternating feet up stairs or no longer using arms to stand up from a chair. These positive modifications are important factors to the long-term maintenance of muscle and physical function and may be overlooked in current assessment of recovery of individuals with TKA.

Obesity is a major risk factor associated with OA (48-50). As obesity is common in OA and can exacerbate the associated symptoms, increased activity and number of steps/day are important factors for weight management during the recovery process.

Although changes in self-reported activity were not significant, the TKA group reported an additional 160 kcals/day of activity-related energy expenditure. Additionally, performance-based mobility (steps/day) improved by 46% from one to three months post-surgery. It appears that once use-related pain was diminished at one month, the TKA group increased activity levels from one month to three months post-surgery.

The short-term recovery from TKA surgery was widely variable as suggested by the large standard deviations in physical function. Functional ROM was strongest predictor of physical function recovery suggesting that individuals with sufficient range of motion can better perform tasks important for living independently. This finding is not surprising in light of the fact that rehabilitation is primarily aimed at increasing range of motion during the first few months of recovery. In addition to improved range of motion, recovery of physical function occurred concurrently with improvements in strength, force/CSA, extensor power, and activity levels.

This study was limited by several factors. Our sample included both unilateral and bilateral surgeries. Individuals who have both knees replaced may recover differently than individuals with only one knee replacement. Also, due to participant drop out, the final sample contained an unequal distribution of males and females in the two groups. Any sex differences in recovery could confound the results of this study. Finally, a longer follow-up of recovery is warranted. Several physiological systems showed significant recovery within three months, however, the TKA group still exhibited significantly less joint, muscle, and physical function compared to the control group.

The results of this study suggest that pain is alleviated within one month of surgery. The reduction in use-related pain is followed by improvements in range of

motion, strength, muscle quality, extensor power, and activity. Physical function is a composite performance reflecting these cumulative changes in joint and muscle function. Individuals recovering from TKA surgery improved physical function to a level predictive of independent living by three months post-surgery. Range of motion was a significant predictor of physical function with other important factors of joint and muscle function contributing to increased physical function.

Future studies should investigate the influence of current rehabilitation protocols on TKA recovery. Pain relief is an important factor in the recovery of physical function. Functional recovery occurs by 3 months with rehabilitation and normal daily activity and is reinforced by the positive modifications to their performance of daily activity. This may imply that TKA patients could sustain a more intensive rehabilitation. Rehabilitation that focuses on other aspects of muscle function such as strength training may provide functional and morphological changes after surgery. Also, many patients expressed apprehension of the ability of the knee prosthesis to sustain activity. Future studies could examine the patient's apprehension of increasing activity levels after TKA surgery as well as follow recovery for a longer period of time.

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Table 4.1 Selected physical characteristics for controls and individuals with total knee arthroplasty

			Weight (kg)		
Group	Age (y)	Height (cm)	Pre-surgery	1 month	3 months
CON	63.5 (11)	175.1 (9)	79.3 (9)	80.3 (10)	80.3 (10)
TKA	61.3 (10)	177.2 (9)	100.3 (25)[a]	97.5 (23)[a]	96.6 (21)[a]

Notes: All data are means ± SD. Body weight is shown for all three follow-up time points. CON = control group, TKA = total knee arthroplasty group. $p < .05$, [a] = significant difference from CON group.

Table 4.2 Comparisons of range of motion of the surgical limb between controls and individuals with total knee arthroplasty during three months recovery

	Pre-Surgery	1 month	3 months
Range of Motion			
Extension (°)			
CON	-1.7 (1)	-0.8 (1)	-1.2 (1)
TKA	4.7 (5)[a]	7.2 (5)[a]	4.5 (4)[a]
Flexion (°)			
CON	138.2 (6)	140.2 (5)	140.8 (6)
TKA	112.3 (13)[a]	107.2 (11)[a]	117.8 (11)[a,c]
Functional (°)			
CON	139.8 (6)	141.0 (6)	142.0 (6)
TKA	107.6 (16)[a]	100.1 (13)[a]	113.3 (14)[a,c]

Notes: All data are mean ± SD. CON = control group, TKA = total knee arthroplasty group. S = TKA-surgical limb or CON-surgical matched limb. NS = TKA-non-surgical limb or CON-non-surgical matched limb. Function range of motion = (Flexion – extension). Negative extension values = hyperextension. [a] = $p < .05$ compared to CON-S, [b] = compared to pre-surgery, [c] = $p < .05$ compared to one month.

Table 4.3 Comparisons of muscle function and characteristics of the surgical limb between controls and individuals with total knee arthroplasty during three months recovery

	Pre-Surgery	1 month	3 months
Muscle Function			
IVCpeak (Nm)			
CON	182.2 (65)	176.6 (59)	179.7 (64)
TKA	83.6 (41)[a]	80.3 (31)[a]	93.3 (34)[a,c]
Pain with IVCpeak			
CON	0.0 (0)	0.0 (0)	0.0 (0)
TKA	3.3 (3)[a]	2.0 (2)[a]	0.8 (1)[b]
Central Activation Ratio			
CON	0.968 (0.03)	0.974 (0.01)	0.965 (0.03)
TKA	0.865 (0.09)[a]	0.965 (0.03)[b]	0.945 (0.09)
Leg Extensor Power (watts)			
CON	239.5 (108)	236.3 (103)	253.6 (127)
TKA	92.6 (58)[a]	121.8 (73)[a,b]	143.4 (95)[b,c]
Pain with Leg Extensor Power			
CON	0.0 (0)	0.0 (0)	0.0 (0)
TKA	1.3 (2)[a]	0.9 (1)	0.5 (1)
Muscle Characteristics			
Quadriceps-Contractile (cm^2)			
CON	59.7 (16)	59.2 (16)	59.2 (16)
TKA	47.9 (18)	47.3 (22)	47.7 (18)
Quadriceps-Non-contractile (cm^2)			
CON	4.5 (1)	4.7 (2)	4.4 (1)
TKA	6.8 (2)	7.0 (3)	6.3 (2)

Notes: All data are mean ± SD. CON = control group, TKA = total knee arthroplasty group. IVC_{peak} = peak isometric voluntary contraction (70^o). Central Activation Ratio = (IVCpeak / peak force during burst of stimulation). Quadriceps- Contractile = cross sectional area of contractile tissue, Quadriceps-Non-contractile = cross sectional area of non-contractile tissue, [a] = $p < .05$ compared to CON, [b] = $p < .05$ compared to pre-surgery, [c] = $p < .05$ compared to one month.

Table 4.4 Comparisons of Cs-PFP total and domain scores between controls and individuals with total knee arthroplasty during three months recovery

	Pre-Surgery	1 Month	3 Months
Total Score			
CON	78.7 (15)	83.9 (14)[b]	83.3 (13)
TKA	49.2 (15)[a]	48.9 (18)[a]	58.2 (17)[a,b,c]
Upper Body Strength			
CON	78.2 (20)	83.0 (20)	80.4 (18)
TKA	56.3 (20)	52.2 (23)[a]	60.8 (25)[c]
Lower Body Strength			
CON	77.9 (18)	84.3 (18)[b]	83.3 (17)
TKA	45.8 (17)[a]	46.4 (18)[a]	56.6 (19)[a,b,c]
Upper Body Flexibility			
CON	74.3 (6)	78.0 (5)	76.8 (5)
TKA	66.6 (13)	67.7 (11)[a]	66.0 (17)
Balance/Coordination			
CON	76.4 (16)	82.5 (15)	83.8 (14)
TKA	45.2 (17)[a]	45.9 (20)[a]	57.2 (16)[a,b,c]
Endurance			
CON	80.7 (15)	85.7 (13)[b]	85.2 (13)
TKA	49.0 (16)[a]	48.7 (18)[a]	58.1 (16)[a,b,c]

Notes: All data are means ± SD. CON = control group, TKA = total knee arthroplasty group, Cs-PFP = Continuous Scale Physical Functional Performance Test. [a] = $p < .05$ compared to CON, [b] = $p < .05$ compared to pre-surgery, [c] = $p < .05$ compared to one month.

Table 4.5 Comparisons of physical function between controls and individuals with total knee arthroplasty during three months recovery

	Pre-Surgery	1 Month	3 Months
SF36 Domain Scores			
General health			
CON	79.0 (17)	79.8 (17)	81.3 (13)
TKA	66.8 (22)	70.2 (17)	67.9 (22)
Bodily pain			
CON	90.2 (12)	86.6 (12)	86.2 (14)
TKA	36.7 (11)[a]	43.2 (22)[a]	64.4 (16)[a,b,c]
Mental health			
CON	88.0 (11)	83.1 (17)	85.8 (16)
TKA	79.4 (11)	70.5 (15)	84.6 (11)[c]
Physical function			
CON	96.5 (4)	97.7 (4)	97.3 (4)
TKA	42.7 (15)[a]	53.1 (20)[a]	71.2 (16)[a,b,c]
Role function-mental			
CON	97.4 (9)	87.2 (32)	92.3 (28)
TKA	82.1 (32)	76.9 (39)	97.4 (9)
Role function-physical			
CON	94.2 (11)	88.5 (28)	96.2 (14)
TKA	15.4 (26)[a]	5.8 (11)[a]	53.8 (39)[a,b,c]
Social function			
CON	96.2 (9)	92.3 (13)	97.1 (10)
TKA	62.5 (27)[a]	47.1 (28)[a]	81.7 (20)[c]
Vitality/energy			
CON	73.8 (15)	71.5 (16)	74.6 (20)
TKA	51.9 (13)[a]	50.0 (17)[a]	65.0 (8)[b,c]
Instrumental ADL			
CON	24.0 (0)	24.0 (0)	24.0 (0)
TKA	23.4 (1)	22.8 (2)	23.9 (0.3)
Task Difficulty			
CON	0.31 (0.9)	0.08 (0.3)	0.08 (0.3)
TKA	7.23 (1.7)[a]	4.15 (2.1)[a,b]	2.08 (1.3)[a,b,c]
Task Modification			
CON	0.15 (0.4)	0.31 (0.6)	0.00 (0.0)
TKA	1.31 (0.9)[a]	2.46 (1.7)[a,b]	1.84 (1.1)[a]
Task Modification: positive			
CON	0.0 (0)	0.0 (0)	0.0 (0)
TKA	0.0 (0)	0.5 (1)	1.0 (2)

Notes: All data are means ± SD. CON = control group, TKA = total knee arthroplasty group, ADL = activities of daily living, [a] = $p < .05$ compared to CON, [b] = $p < .05$ compared to pre-surgery, [c] = $p < .05$ compared to one month.

Table 4.6 Comparisons of activity between controls and individuals with total knee arthroplasty during three months recovery

	Pre-Surgery	1 Month	3 Months
Number of Steps/day			
CON	8,282.4 (3,037)	9,062.6 (3,400)	9,879.6 (2,879)
TKA	3,954.3 (2,616)[a]	3,554.9 (2,333)[a]	5,776.5 (2,572)[a,c]
OA-ESI (kcals/day)			
Total			
CON	650.5 (397)	596.3 (482)	868.5 (693)
TKA	754.5 (462)	670.2 (538)	990.4 (680)
Work-related			
CON	300.0 (275)	330.0 (375)	537.7 (502)
TKA	503.6 (443)	447.8 (514)	558.2 (531)
Activity/Rehabilitation			
CON	350.5 (241)	304.1 (156)	330.8 (245)
TKA	250.9 (190)	269.9 (167)	432.2 (259)

Notes: All data are means ± SD. CON = control group, TKA = total knee arthroplasty group, OA-ESI = Older adult exercise status inventory, kcals = kilocalories, [a] = $p < .05$ compared to CON, [b] = $p < .05$ compared to pre-surgery, [c] = $p < .05$ compared to one month.

Figure Legends:

Figure 4.1 Recovery of Use-related Pain associated with Cs-PFP during three months follow-up. Data are means ± SE. Closed circles = Control Group, Open circles = TKA group. * = significant difference from pre-surgery, † = significant difference from one month, ‡ = significant difference from Control group.

Figure 4.2 Recovery of Force per CSA of quadriceps contractile tissue during three months follow-up. Data are means ± SE. Closed circles = Control surgical matched limb, Open circles = TKA surgical limb, Closed triangles = Control non-surgical limb, Open triangles =TKA non-surgical limb. $p < .05$. * = significant difference from pre-surgery, † = significant difference from one month, ‡ = significant difference from Control surgical matched limb, § = significant difference from TKA non-surgical limb.

Figure 4.3 Recovery of Physical Function (Cs-PFP total) during three months follow-up. Data are means ± SE. Closed circles = Control Group, Open circles = TKA group. $p < .05$. * = significant difference from pre-surgery, † = significant difference from one month, ‡ = significant difference from Control group.

Figure 4.1

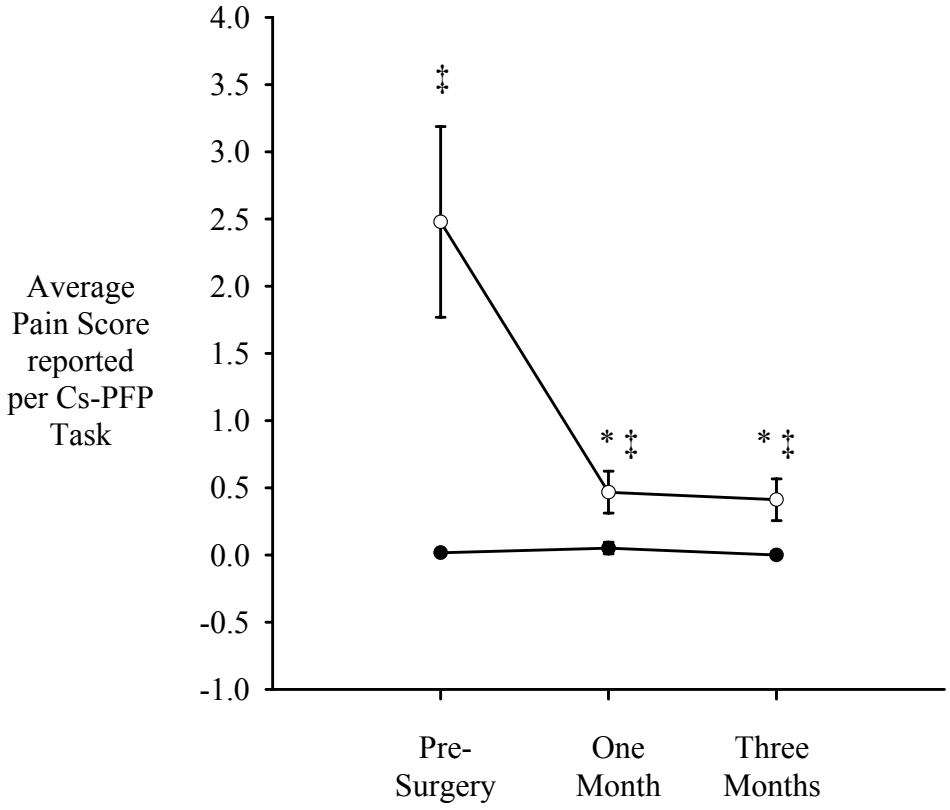

Average Pain Score reported per Cs-PFP Task
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
-0.5
-1.0
‡
* ‡
* ‡
Pre-Surgery
One Month
Three Months

Figure 4.2

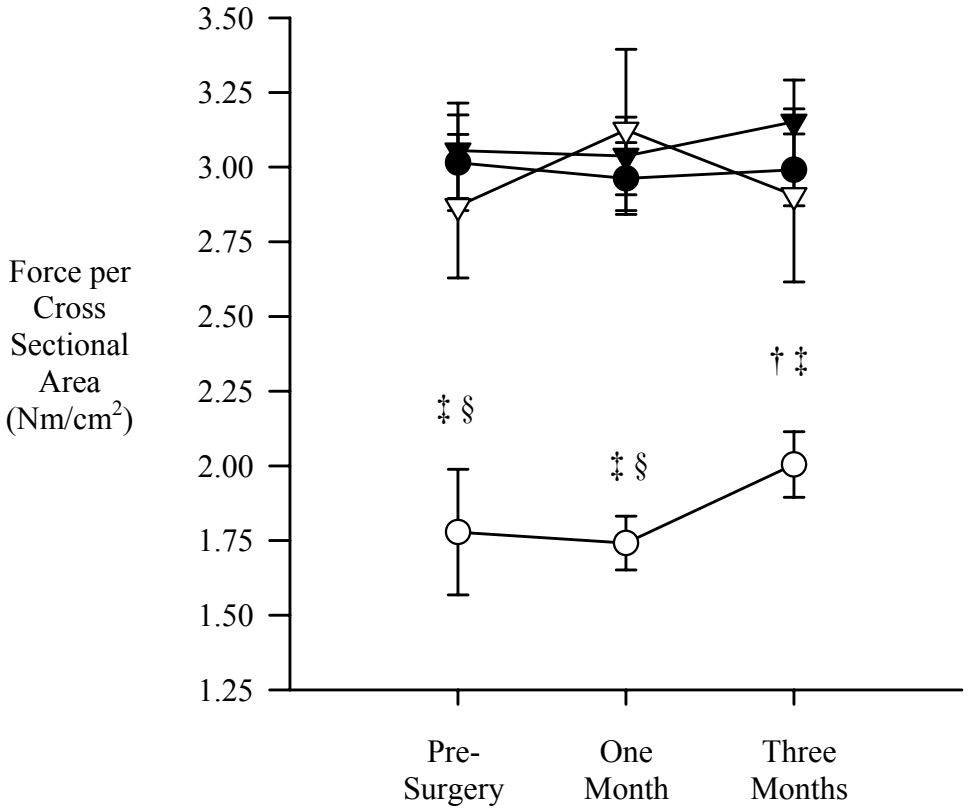
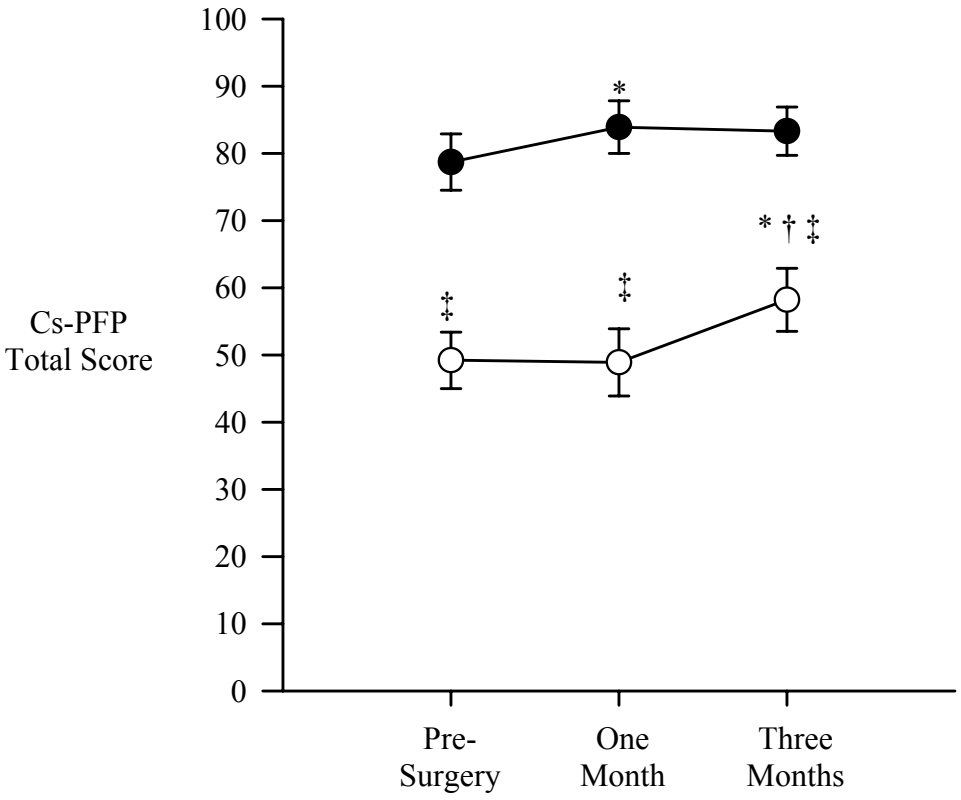

Force per Cross Sectional Area (Nm/cm²)
3.50
3.25
3.00
2.75
2.50
2.25
2.00
1.75
1.50
1.25
‡ §
‡ §
† ‡
Pre-Surgery
One Month
Three Months

Figure 4.3


Cs-PFP
Total Score
100
90
80
70
60
50
40
30
20
10
0
*
* † ‡
‡
‡
Pre-
Surgery
One
Month
Three
Months

CHAPTER V

SUMMARY

Physical function is a composite performance that reflects physical abilities in multiple physiological systems. Physical reserve is the excess physical capacity above that required to perform daily activities. Age, inactivity, or disease can reduce physical reserves resulting in functional limitation and risk of disability. Greater peak physical abilities result in greater physical reserves that provide a margin of safety against the risk of disability. These studies focused on two areas impacting physical reserves of older adults, pre-clinical disability and recovery from total knee arthroplasty (TKA).

Individuals who report modification of task performance can be classified as pre-clinical disability. In the first study, we compared high functioning individuals (HIGH) to individuals living independently but with lower levels of physical function (LOW). These groups were assessed on task modification and factors of mobility including gait speed, stride length, and number of steps/day. Group membership was determined by using a threshold score 57 on the Cs-PFP total. A score of 57 is indicative of a functional threshold associated with levels of physical abilities needed to maintain independence.

Our results show gait speed for LOW was 27% slower and stride length was 18% shorter when compared to HIGH. The LOW group also showed 47% fewer steps taken per day and reported 4.6 times as many tasks requiring modification as the HIGH group. These task modification and mobility characteristics provide several indicators for increased risk of disability. These characteristics could identify an individual in the stage

of pre-clinical disability and allow for timely and effective interventions to increase physical reserves and forestall the possible loss of independence.

Osteoarthritis (OA) is one of the leading causes of disability effecting multiple physical capacities including joint, muscle, and physical function. In the second study, we examined the decrements in joint, muscle, and physical function in a sample with diagnosed OA and how those functions changed during three months recovery from total knee arthroplasty (TKA). Prior to surgery, individuals with OA had significantly more use-related pain and reduced muscle function and physical function compared to controls at baseline. TKA surgery reduced use-related pain 83% by one month post surgery. From one to three months post surgery, TKA patients increased knee range of motion by 13%, quadriceps strength by 16%, muscle quality by 13%, extensor power by 55%, and number of steps/day by 46%. Concurrent to these changes, physical function increased 19% by three months follow-up. At baseline, the TKA surgery group was eight units below the functional threshold (Cs-PFP threshold of 57 – TKA group average of 49) but improved to one unit above the threshold by three months. One of the primary goals given by individuals with OA is the improvement of function and this sample was successful in significantly improving physical function during the short time course of three months recovery.

These results indicate that physical reserves are reduced in pre-clinical disability but can be increased after TKA surgery. Interventions aimed at increasing mobility can improve the physical reserve and decrease the risk of disability. In a clinical population, individuals recovering from TKA surgery can expect significant pain relief followed by improved joint, muscle, and physical function. Short-term improvements in physical

function occurred with increases in peak capacities of range of motion, quadriceps strength, force/CSA, extensor power, and number of steps taken per day. By increasing peak capacities of TKA patients, the physical reserves can be improved and provide a margin of safety against risk of disability.

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APPENDIX A

RAW DATA

ID	GROUP	Sex	AGE YR	HT CM	WT0 KG	WT1 KG	WT3 KG	IADL0 (#)	IADL1 (#)	IADL3 (#)	SF36 BP0	SF36 BP1	SF36 BP3	SF36 GH0	SF36 GH1	SF36 GH3
1	TKA	M	59	190.0	113.2	115.0	116.1	23	24	24	34.4	13.3	57.8	87.0	77.0	57.0
2	TKA	M	68	169.0	99.3	97.7	101.8	24	24	24	**	68.9	100.0	50.0	77.0	45.0
4	TKA	F	62	163.5	69.8	67.5	69.1	24	24	24	24.4	35.6	56.7	82.0	67.0	87.0
5	TKA	F	70	169.5	68.9	64.3	64.1	23	23	24	35.6	34.4	56.7	37.0	57.0	62.0
6	TKA	F	60	177.4	100.0	97.3	97.7	24	24	24	24.4	34.4	57.8	32.0	42.0	52.0
7	TKA	M	54	180.5	158.0	146.6	132.3	24	24	24	45.6	11.1	93.3	47.0	57.0	25.0
8	TKA	M	77	181.0	85.7	87.3	87.3	24	24	24	45.6	45.6	56.7	57.0	62.0	57.0
9	TKA	M	64	166.0	95.7	94.1	97.7	24	24	24	34.4	82.2	80.0	67.0	100.0	97.0
10	TKA	M	35	178.0	131.1	128.1	125.7	23	24	24	46.7	24.4	45.6	92.0	72.0	57.0
11	TKA	M	62	182.0	112.5	105.9	106.4	24	24	24	34.4	68.9	93.3	67.0	52.0	85.0
12	TKA	M	55	186.0	92.5	**	90.7	24	**	24	45.6	62.8	80.0	90.0	**	67.0
13	TKA	F	63	174.8	74.3	74.3	70.5	22	17	24	24.4	34.4	45.6	57.0	72.0	82.0
14	TKA	F	64	176.0	103.2	103.6	100.9	21	19	24	24.4	45.6	56.7	97.0	100.0	100.0
15	TKA	M	55	195.0	164.1	158.0	**	24	24	**	24.4	22.2	**	45.0	57.0	**
16	TKA	F	72	169.5	91.8	85.7	86.8	24	21	23	56.7	68.9	56.7	97.0	77.0	77.0
1	CON	M	68	189.0	101.8	101.4	101.4	24	24	24	80.0	80.0	80.0	67.0	62.0	72.0
2	CON	M	71	169.0	84.3	81.1	82.5	24	24	24	93.3	100.0	93.3	100.0	100.0	90.0
4	CON	F	62	163.5	73.2	74.3	74.3	24	24	24	93.3	93.3	93.3	62.0	62.0	57.0
5	CON	F	71	174.5	70.7	70.5	69.1	24	24	24	82.2	93.3	100.0	92.0	92.0	97.0
6	CON	F	61	171.5	67.3	67.0	67.0	24	24	24	100.0	93.3	93.3	100.0	95.0	85.0
7	CON	M	55	176.5	78.4	79.8	77.3	24	24	24	67.8	67.8	56.7	60.0	70.0	70.0
8	CON	M	76	186.0	78.9	78.6	77.1	24	24	24	100.0	93.3	68.9	92.0	87.0	87.0
9	CON	M	71	173.0	79.3	79.3	78.9	24	24	24	100.0	93.3	93.3	90.0	90.0	95.0
10	CON	M	31	186.0	85.0	85.7	85.5	24	24	24	100.0	100.0	100.0	90.0	100.0	95.0
11	CON	M	64	175.5	81.2	86.8	85.7	24	24	24	93.3	93.3	93.3	75.0	75.0	85.0
12	CON	M	61	181.0	76.8	78.4	**	24	24	**	93.3	100.0	**	75.0	95.0	**
13	CON	F	62	173.3	68.4	68.6	70.2	24	24	24	100.0	80.0	100.0	85.0	90.0	90.0
14	CON	F	65	164.3	75.7	77.0	78.0	24	24	24	93.3	68.9	80.0	62.0	62.0	67.0
15	CON	M	63	184.5	97.1	95.2	97.5	24	24	24	68.9	68.9	67.8	52.0	52.0	67.0
16	CON	F	71	159.0	72.0	**	**	24	**	**	100.0	**	**	62.0	**	**

** = missing data

ID	GROUP	SF36 MH0	SF36 MH1	SF36 MH3	SF36 PF0	SF36 PF1	SF36 PF3	SF36 RE0	SF36 RE1	SF36 RE3	SF36 RP0	SF36 RP1	SF36 RP3	SF36 SF0	SF36 SF1	SF36 SF3
1	TKA	76.0	76.0	80.0	45.0	40.0	60.0	100.0	100.0	100.0	75.0	0.0	0.0	25.0	0.0	50.0
2	TKA	80.0	72.0	76.0	35.0	75.0	95.0	100.0	100.0	100.0	25.0	25.0	100.0	100.0	75.0	75.0
4	TKA	88.0	64.0	92.0	60.0	45.0	65.0	100.0	100.0	100.0	50.0	0.0	100.0	100.0	62.5	100.0
5	TKA	48.0	60.0	64.0	50.0	25.0	90.0	0.0	0.0	100.0	0.0	0.0	100.0	62.5	37.5	87.5
6	TKA	80.0	68.0	88.0	20.0	40.0	50.0	100.0	66.7	100.0	0.0	0.0	25.0	37.5	75.0	62.5
7	TKA	80.0	80.0	80.0	55.0	75.0	80.0	66.7	100.0	100.0	0.0	25.0	75.0	62.5	12.5	100.0
8	TKA	80.0	84.0	72.0	60.0	80.0	70.0	100.0	100.0	100.0	0.0	25.0	50.0	37.5	62.5	87.5
9	TKA	72.0	88.0	100.0	55.0	75.0	80.0	33.3	100.0	100.0	0.0	0.0	75.0	62.5	87.5	100.0
10	TKA	92.0	56.0	92.0	20.0	40.0	75.0	100.0	100.0	100.0	0.0	0.0	25.0	37.5	25.0	100.0
11	TKA	88.0	92.0	100.0	45.0	80.0	85.0	100.0	100.0	100.0	0.0	0.0	75.0	50.0	75.0	100.0
12	TKA	72.0	**	60.0	75.0	**	95.0	66.7	**	100.0	0.0	**	100.0	75.0	**	100.0
13	TKA	92.0	48.0	80.0	45.0	45.0	75.0	100.0	0.0	100.0	0.0	0.0	0.0	50.0	37.5	62.5
14	TKA	76.0	80.0	92.0	20.0	35.0	60.0	100.0	100.0	100.0	0.0	0.0	0.0	100.0	37.5	87.5
15	TKA	72.0	76.0	**	55.0	90.0	**	100.0	66.7	**	0.0	25.0	**	37.5	12.5	**
16	TKA	80.0	48.0	84.0	45.0	35.0	40.0	66.7	33.3	66.7	50.0	0.0	75.0	87.5	25.0	50.0
1	CON	80.0	80.0	92.0	90.0	100.0	90.0	100.0	100.0	100.0	75.0	100.0	100.0	100.0	100.0	100.0
2	CON	96.0	52.0	92.0	95.0	100.0	100.0	100.0	0.0	100.0	100.0	100.0	100.0	100.0	87.5	100.0
4	CON	92.0	92.0	92.0	95.0	100.0	100.0	100.0	100.0	100.0	100.0	0.0	100.0	100.0	62.5	100.0
5	CON	92.0	76.0	92.0	100.0	90.0	95.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
6	CON	80.0	88.0	88.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
7	CON	64.0	48.0	44.0	100.0	100.0	100.0	100.0	33.3	0.0	100.0	100.0	50.0	100.0	75.0	100.0
8	CON	100.0	100.0	100.0	90.0	90.0	90.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
9	CON	96.0	96.0	96.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
10	CON	96.0	92.0	92.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
11	CON	84.0	84.0	64.0	90.0	90.0	90.0	100.0	100.0	100.0	75.0	75.0	100.0	100.0	100.0	62.5
12	CON	84.0	84.0	**	100.0	100.0	**	100.0	100.0	**	100.0	100.0	**	100.0	100.0	**
13	CON	96.0	100.0	96.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
14	CON	72.0	76.0	76.0	95.0	100.0	100.0	66.7	100.0	100.0	100.0	100.0	100.0	75.0	100.0	100.0
15	CON	96.0	96.0	92.0	100.0	100.0	100.0	100.0	100.0	100.0	75.0	75.0	100.0	75.0	75.0	100.0
16	CON	88.0	**	**	95.0	**	**	100.0	**	**	100.0	**	**	100.0	**	**

** = missing data

ID	GROUP	SF36 VT0	SF36 VT1	SF36 VT3	TM0 (#)	TM1 (#)	TM3 (#)	TD0 (#)	TD1 (#)	TD3 (#)	TM+0 (#)	TM+1 (#)	TM+3 (#)
1	TKA	60.0	30.0	70.0	3	4	2	6	5	5	0	0	0
2	TKA	55.0	50.0	60.0	1	0	2	6	3	0	0	3	3
4	TKA	55.0	45.0	60.0	2	4	2	4	4	1	0	0	1
5	TKA	40.0	30.0	55.0	2	2	3	8	7	1	0	0	1
6	TKA	30.0	40.0	60.0	1	1	0	6	4	3	0	0	6
7	TKA	45.0	50.0	70.0	1	4	1	9	0	2	0	2	1
8	TKA	45.0	60.0	60.0	1	2	2	6	4	2	0	0	0
9	TKA	40.0	60.0	65.0	2	3	1	6	3	2	0	0	1
10	TKA	55.0	95.0	70.0	0	0	2	10	2	1	0	0	0
11	TKA	75.0	60.0	85.0	0	1	3	9	3	1	0	0	0
12	TKA	50.0	**	75.0	3	**	0	1	**	0	0	**	0
13	TKA	40.0	30.0	65.0	1	2	0	9	8	3	0	0	0
14	TKA	65.0	50.0	65.0	2	5	4	8	5	3	0	0	0
15	TKA	45.0	55.0	**	3	2	**	6	2	**	0	2	**
16	TKA	70.0	50.0	60.0	1	4	2	7	6	3	0	0	1
1	CON	70.0	75.0	75.0	1	1	0	0	0	0	0	0	0
2	CON	85.0	75.0	100.0	1	0	0	0	0	0	0	0	0
4	CON	85.0	60.0	90.0	0	0	0	1	0	0	0	0	0
5	CON	85.0	75.0	75.0	0	0	0	0	0	0	0	0	0
6	CON	70.0	75.0	80.0	0	0	0	0	0	0	0	0	0
7	CON	35.0	25.0	20.0	0	0	0	0	0	0	0	0	0
8	CON	90.0	90.0	70.0	0	0	0	0	0	0	0	0	0
9	CON	85.0	85.0	85.0	0	0	0	0	0	0	0	0	0
10	CON	70.0	80.0	90.0	0	0	0	0	0	0	0	0	0
11	CON	65.0	65.0	65.0	0	1	0	3	1	1	0	0	0
12	CON	75.0	80.0	**	0	0	**	0	0	**	0	0	**
13	CON	80.0	75.0	85.0	0	0	0	0	0	0	0	0	0
14	CON	60.0	70.0	70.0	0	0	0	0	0	0	0	0	0
15	CON	80.0	80.0	65.0	0	2	0	0	0	0	0	0	0
16	CON	65.0	**	**	2	**	**	0	**	**	0	**	**

** = missing data

ID	GROUP	WRK0 KCAL	WRK1 KCAL	WRK3 KCAL	ACT0 KCAL	ACT1 KCAL	ACT3 KCAL	TOT0 KCAL	TOT1 KCAL	TOT3 KCAL
1	TKA	252.7	776.3	424.0	442.7	264.0	509.3	695.4	374.8	933.3
2	TKA	553.4	705.1	240.0	0.0	193.4	186.7	553.4	898.4	426.7
4	TKA	142.0	82.0	266.5	238.9	295.1	459.8	380.9	377.0	726.3
5	TKA	612.4	156.2	192.3	258.2	222.8	200.3	870.6	379.0	392.6
6	TKA	514.3	1688.3	669.9	321.4	321.0	544.3	835.7	2009.3	1214.3
7	TKA	812.3	582.9	552.8	158.0	185.9	128.5	970.3	768.7	681.4
8	TKA	330.5	486.2	729.3	171.4	151.7	353.4	501.8	637.9	1082.8
9	TKA	41.0	208.4	586.2	239.2	286.2	830.5	280.2	494.6	1416.7
10	TKA	988.2	1267.3	2154.5	573.7	341.6	843.9	1562.0	1608.9	2998.4
11	TKA	8.0	128.6	136.8	229.7	268.6	764.8	237.7	397.2	901.5
12	TKA	406.3	**	323.9	535.2	**	495.5	941.5	**	819.4
13	TKA	1720.0	47.8	1086.9	23.9	307.0	263.7	1743.9	354.8	1350.6
14	TKA	44.2	0.0	147.8	33.2	40.7	21.6	77.4	40.7	169.4
15	TKA	492.3	0.0	**	492.3	770.9	**	984.5	770.9	**
16	TKA	636.0	140.8	303.8	45.9	129.4	448.5	681.9	270.2	752.3
1	CON	229.1	1463.1	1270.6	581.8	474.4	597.3	810.8	1937.5	1867.9
2	CON	253.0	504.2	689.5	528.0	226.0	334.9	781.0	730.3	1024.4
4	CON	533.3	26.5	222.9	377.3	0.0	152.1	910.6	26.5	375.0
5	CON	444.3	102.7	207.3	235.6	201.3	130.8	679.9	304.0	338.1
6	CON	57.7	502.9	57.4	226.7	395.1	177.9	284.4	898.0	235.3
7	CON	894.5	210.0	1846.1	192.2	588.1	866.1	1086.6	798.1	2712.2
8	CON	0.0	294.8	297.2	371.8	306.0	407.3	371.8	600.7	704.5
9	CON	136.0	264.4	312.3	264.4	451.4	325.5	400.4	187.0	637.8
10	CON	97.1	0.0	146.6	110.8	202.0	668.7	208.0	202.0	815.3
11	CON	382.8	502.2	330.5	286.1	477.4	97.9	668.9	979.6	428.4
12	CON	57.6	39.2	**	584.4	247.8	**	642.0	287.0	**
13	CON	45.6	101.7	556.8	19.5	218.2	250.5	65.1	319.9	807.3
14	CON	583.8	445.5	467.7	138.2	183.7	44.3	722.0	629.2	512.0
15	CON	693.2	163.3	585.0	979.2	285.7	247.2	1672.4	448.9	832.2
16	CON	92.6	**	**	360.9	**	**	453.4	**	**

** = missing data

ID	GROUP	TOT0 PFP	TOT1 PFP	TOT3 PFP	LBS0 PFP	LBS1 PFP	LBS3 PFP	UBS0 PFP	UBS1 PFP	UBS3 PFP	UBF0 PFP	UBF1 PFP	UBF3 PFP
1	TKA	52.27	55.95	62.04	50.48	47.04	60.56	83.30	70.56	81.27	82.43	82.43	82.43
2	TKA	61.62	73.53	60.90	61.39	68.77	59.16	72.71	82.32	70.42	50.33	71.16	53.17
4	TKA	48.09	48.58	56.56	37.31	41.85	45.28	45.19	42.56	45.26	59.99	58.04	63.80
5	TKA	50.04	41.13	62.10	43.29	39.46	55.63	57.20	50.73	73.29	76.38	65.92	74.33
6	TKA	41.96	44.04	50.29	31.84	32.22	42.06	33.96	27.92	31.81	73.37	70.07	56.31
7	TKA	38.48	44.63	62.14	33.56	42.86	65.12	55.84	53.55	79.46	67.04	60.73	75.96
8	TKA	35.50	40.66	45.21	34.21	39.87	45.01	32.75	40.30	40.82	45.41	43.38	38.47
9	TKA	72.07	55.44	77.00	71.17	51.43	83.32	85.00	66.89	86.69	77.83	82.07	82.24
10	TKA	70.13	72.86	82.32	71.53	74.35	83.96	74.13	68.52	79.34	72.77	74.26	86.29
11	TKA	62.48	79.35	83.76	61.45	82.98	87.46	69.98	95.25	100.00	74.30	78.79	80.99
12	TKA	87.59	**	84.35	90.39	**	85.37	99.00	**	91.61	77.53	**	82.37
13	TKA	54.71	29.47	53.76	51.59	27.65	49.98	57.10	27.53	43.64	80.94	72.82	75.68
14	TKA	19.70	23.58	34.45	15.78	24.45	31.04	22.83	27.64	30.12	53.24	63.27	54.95
15	TKA	53.58	67.66	**	50.38	55.10	**	63.71	63.78	**	62.82	78.18	**
16	TKA	32.47	26.72	25.75	32.41	29.64	27.86	41.58	25.14	28.31	51.76	57.47	33.50
1	CON	88.07	91.88	87.02	89.39	92.83	83.10	96.45	100.00	86.23	71.13	74.11	70.81
2	CON	88.38	91.55	93.75	91.76	96.77	100.00	96.13	100.00	100.00	85.09	85.09	86.27
4	CON	52.44	59.04	62.23	49.13	57.49	60.15	52.75	65.07	62.32	69.89	69.51	75.59
5	CON	54.61	61.65	57.50	39.04	46.51	43.91	44.86	38.00	41.12	68.38	79.35	76.73
6	CON	79.42	83.14	85.94	82.92	89.22	89.73	68.61	72.33	77.59	71.79	76.56	74.23
7	CON	85.07	93.17	96.28	86.45	97.21	99.45	88.00	95.88	99.17	83.46	84.02	84.02
8	CON	90.98	92.29	87.16	90.06	94.39	88.46	86.60	88.76	85.58	79.14	77.85	77.85
9	CON	80.99	79.42	70.89	80.92	81.17	72.64	91.42	89.01	80.72	75.90	78.51	67.17
10	CON	96.01	97.66	93.09	94.62	100.00	92.20	91.93	95.69	75.36	76.45	75.91	75.91
11	CON	94.93	96.18	96.93	97.71	100.00	100.00	100.00	100.00	100.00	66.72	72.99	70.71
12	CON	76.35	79.04	**	79.55	79.16	**	86.04	87.85	**	77.83	78.56	**
13	CON	72.34	80.30	82.76	67.49	78.58	87.57	57.73	77.19	78.87	79.67	81.77	82.55
14	CON	58.43	66.22	73.48	60.19	62.76	69.70	51.67	57.78	60.07	69.40	74.40	76.85
15	CON	80.87	98.49	95.44	82.87	99.24	96.95	90.53	98.86	97.73	69.30	83.41	80.16
16	CON	65.65	**	**	61.09	**	**	65.86	**	**	63.79	**	**

** = missing data

ID	GROUP	BALC0 PFP	BALC1 PFP	BALC3 PFP	ENDR0 PFP	ENDR1 PFP	ENDR3 PFP	PFP0 PAIN	PFP1 PAIN	PFP3 PAIN	STEPS0 (#/DAY)	STEPS1 (#/DAY)	STEPS3 (#/DAY)
1	TKA	31.86	51.72	50.41	44.90	54.41	58.20	10.0	7.0	12.0	**	1088.9	**
2	TKA	70.13	74.23	61.98	61.42	73.05	57.67	**	3.0	0.0	**	**	**
4	TKA	52.41	54.36	67.62	52.11	51.44	61.24	7.0	0.0	0.0	5702.2	3028.3	7391.0
5	TKA	43.92	29.08	55.68	50.92	40.01	63.27	20.0	7.0	5.0	**	2267.7	5600.0
6	TKA	41.60	48.39	61.69	45.03	49.95	55.80	20.0	5.0	9.0	4652.6	7550.0	5067.0
7	TKA	31.35	37.71	50.34	36.27	43.60	57.88	9.0	0.0	0.0	2149.5	3611.7	4581.0
8	TKA	39.43	46.64	48.67	35.87	39.75	46.09	33.0	17.0	12.0	1166.0	2477.0	4532.0
9	TKA	63.94	50.32	70.86	71.52	50.96	73.15	8.0	3.0	3.0	4466.0	4124.0	6560.0
10	TKA	67.98	74.75	84.69	72.23	75.97	83.57	0.0	2.0	3.0	2033.0	3700.0	**
11	TKA	61.82	71.40	75.42	60.90	76.61	80.97	35.0	2.0	3.0	3750.0	6877.0	10898.0
12	TKA	83.92	**	82.22	86.57	**	82.83	6.0	**	0.0	**	**	**
13	TKA	44.82	21.89	50.10	56.39	29.04	57.22	48.0	0.0	0.0	9647.7	785.1	6517.3
14	TKA	14.17	12.58	39.91	18.74	22.38	35.90	83.0	4.0	1.0	1879.9	2381.6	5009.4
15	TKA	51.83	72.15	**	52.79	72.52	**	31.5	0.0	**	**	**	**
16	TKA	23.77	23.06	25.73	30.65	26.41	23.87	11.0	4.0	0.0	2175.0	1159.9	1432.4
1	CON	86.48	92.05	97.03	87.40	91.06	87.19	0.0	0.0	0.0	**	8633.4	**
2	CON	79.55	82.11	85.92	87.70	90.63	92.62	2.0	1.0	0.0	8420.3	10084.7	9013.0
4	CON	45.70	50.93	57.64	52.34	58.12	61.53	0.0	0.0	0.0	4739.3	6948.9	6056.0
5	CON	57.93	69.34	62.34	60.39	70.58	65.21	0.0	0.0	0.0	9017.4	8740.4	8498.1
6	CON	83.90	84.12	88.59	82.37	86.56	89.62	0.0	0.0	0.0	9122.0	16084.7	12332.0
7	CON	78.15	89.06	93.77	85.82	92.88	96.55	0.0	5.0	0.0	3785.0	5349.7	9848.9
8	CON	94.64	95.64	87.80	94.76	94.50	89.15	0.0	0.0	0.0	4463.4	4705.3	4893.0
9	CON	76.13	72.37	66.00	80.87	78.81	68.69	0.0	0.0	0.0	8251.4	8541.3	8772.6
10	CON	100.00	100.00	100.00	100.00	100.00	100.00	0.0	0.0	0.0	9174.7	6702.9	10312.3
11	CON	94.99	94.70	100.00	96.41	97.35	98.63	0.0	0.0	0.0	6086.9	10240.7	10077.7
12	CON	63.76	72.50	**	75.22	77.22	**	0.0	0.0	**	4807.9	**	**
13	CON	71.41	76.03	78.06	78.37	82.86	83.20	0.0	0.0	0.0	8703.1	7964.1	14584.1
14	CON	54.38	65.88	77.89	61.24	70.13	78.60	0.0	0.0	0.0	11425.6	11991.3	11651.4
15	CON	70.59	100.00	93.94	81.36	100.00	96.97	0.0	0.0	0.0	14004.6	12575.0	12714.1
16	CON	64.17	**	**	67.94	**	**	0.0	**	**	8690.6	**	**

** = missing data

ID	GROUP	SURG LIMB	IVCPK0 NM	IVCPK1 NM	IVCPK3 NM	IVCPK0 PAIN	IVCPK1 PAIN	IVCPK3 PAIN	CAR0	CAR1	CAR3	CAR0 PAIN	CAR1 PAIN	CAR3 PAIN
1	TKA-S	Left	94.69	100.43	120.18	3.0	3.0	4.0	0.88	1.00	0.92	2.5	3.0	4.0
2	TKA-S	Right	95.63	86.65	84.82	4.0	2.0	0.0	0.80	0.89	0.95	4.0	3.0	3.0
4	TKA-S	Bilat-L	68.59	44.70	61.39	4.0	0.0	0.0	0.96	0.95	0.95	6.0	0.0	0.0
5	TKA-S	Left	14.11	43.55	56.16	6.0	3.0	2.0	0.76	0.96	0.98	6.0	3.0	2.0
6	TKA-S	Left	70.46	76.96	85.48	0.0	0.0	0.0	0.92	1.00	1.00	8.0	7.0	5.0
7	TKA-S	Left	65.64	107.91	141.37	3.0	3.0	0.0	0.78	0.99	1.00	3.0	3.0	2.0
8	TKA-S	Right	106.97	100.49	124.93	8.0	5.0	0.0	0.85	1.00	1.00	8.0	5.0	0.0
9	TKA-S	Left	133.47	101.48	103.15	0.0	1.0	0.0	0.98	0.97	0.99	0.0	0.0	0.0
10	TKA-S	Left	173.63	135.87	120.51	10.0	0.0	2.0	0.87	0.95	0.69	10.0	0.0	0.0
11	TKA-S	Bilat-R	52.77	99.84	140.48	0.0	0.0	0.0	0.77	0.98	0.99	2.0	0.0	0.0
12	TKA-S	Bilat-L	171.86	**	151.97	2.0	**	0.0	0.99	**	0.96	2.0	**	0.0
13	TKA-S	Bilat-R	44.58	52.51	64.67	0.0	0.0	1.0	0.75	0.92	0.84	0.0	0.0	0.0
14	TKA-S	Bilat-L	97.06	57.17	64.29	5.0	4.0	1.0	0.99	0.98	1.00	5.0	4.0	0.0
15	TKA-S	Bilat-L	164.95	173.83	**	1.0	0.0	**	0.88	0.97	**	0.0	0.0	**
16	TKA-S	Right	69.63	36.95	45.54	0.0	5.0	0.0	0.94	0.96	0.98	0.0	5.0	0.0
1	TKA-NS	Right	299.24	271.26	274.70	3.0	1.0	0.0	1.00	0.98	0.99	0.0	1.0	0.0
2	TKA-NS	Left	121.73	119.52	136.07	4.0	0.0	0.0	0.91	0.79	0.92	4.0	3.0	3.0
4	TKA-NS	Bilat-R	83.26	56.16	70.34	5.0	0.0	0.0	0.95	0.93	0.97	5.0	0.0	0.0
5	TKA-NS	Right	106.82	120.44	75.99	6.0	3.0	0.0	0.99	0.96	0.97	6.0	3.0	4.0
6	TKA-NS	Right	80.73	105.02	108.29	0.0	0.0	0.0	0.97	0.99	0.99	9.0	6.0	5.0
7	TKA-NS	Right	222.14	249.92	237.53	3.0	3.0	0.0	0.97	0.99	0.99	3.0	3.0	3.0
8	TKA-NS	Left	143.70	130.13	143.83	0.0	0.0	4.0	0.93	0.94	0.97	5.0	3.0	4.0
9	TKA-NS	Right	199.17	207.03	230.75	0.0	0.0	0.0	0.97	1.00	1.00	0.0	0.0	0.0
10	TKA-NS	Right	348.02	377.22	350.99	0.0	0.0	0.0	0.91	0.95	0.95	0.0	0.0	4.0
11	TKA-NS	Bilat-L	150.78	133.30	146.02	2.0	0.0	1.0	0.91	0.98	0.96	2.0	1.0	0.0
12	TKA-NS	Bilat-R	213.23	**	167.32	0.0	**	0.0	0.94	**	1.00	2.0	**	0.0
13	TKA-NS	Bilat-L	47.21	39.59	69.36	3.0	0.0	0.0	0.70	0.76	1.00	5.0	0.0	0.0
14	TKA-NS	Bilat-R	110.24	55.05	72.55	0.0	1.0	0.0	1.00	0.93	0.98	3.0	4.0	0.0
15	TKA-NS	Bilat-R	213.18	185.18	**	4.0	0.0	**	0.92	0.96	**	1.0	0.0	**
16	TKA-NS	Left	64.48	71.37	43.64	0.0	0.0	0.0	0.93	1.00	0.98	0.0	0.0	0.0

** = missing data

ID	GROUP	SURG LIMB	LEP0 WATTS	LEP1 WATTS	LEP3 WATTS	LEP0 PAIN	LEP1 PAIN	LEP3 PAIN	MLEG0 CSA(CM^2)	MLEG1 CSA(CM^2)	MLEG3 CSA(CM^2)	SUBCUT0 CSA(CM^2)	SUBCUT1 CSA(CM^2)	SUBCUT3 CSA(CM^2)
1	TKA-S	Left	85.0	114.2	103.3	3.5	2.0	4.0	141.50	142.88	144.18	109.77	116.78	121.91
2	TKA-S	Right	137.8	185.8	168.7	2.0	0.0	0.0	147.10	136.80	148.42	84.17	85.88	85.69
4	TKA-S	Bilat-L	59.1	83.5	88.7	0.0	0.0	0.0	76.72	67.58	76.43	131.83	115.14	121.04
5	TKA-S	Left	37.8	45.1	52.7	0.0	2.0	1.0	66.02	62.45	58.87	124.12	122.82	121.52
6	TKA-S	Left	92.1	139.0	157.9	3.0	0.0	0.0	98.16	101.82	95.80	132.19	122.33	144.39
7	TKA-S	Left	43.3	175.2	245.7	4.0	0.0	0.0	152.63	142.33	138.39	175.10	157.01	130.24
8	TKA-S	Right	104.4	160.9	136.6	3.0	0.0	2.0	126.20	124.68	126.29	41.27	40.44	38.39
9	TKA-S	Left	144.1	131.9	171.9	0.0	1.0	0.0	115.30	103.21	116.25	98.06	95.61	106.93
10	TKA-S	Left	227.9	265.4	347.4	0.0	0.0	0.0	229.40	262.57	199.15	84.55	112.61	80.50
11	TKA-S	Bilat-R	142.8	178.7	256.7	0.0	0.0	0.0	152.18	145.83	151.97	63.19	62.12	60.57
12	TKA-S	Bilat-L	218.5	**	297.5	2.0	**	0.0	147.49	**	138.32	56.89	**	56.73
13	TKA-S	Bilat-R	52.4	56.5	65.3	2.0	0.0	0.0	89.37	73.00	78.80	120.04	109.49	105.49
14	TKA-S	Bilat-L	58.4	35.3	44.8	0.0	2.0	0.0	86.94	80.30	79.56	243.45	251.64	208.37
15	TKA-S	Bilat-L	159.4	180.4	**	4.0	0.0	**	200.10	176.57	**	109.60	100.75	**
16	TKA-S	Right	19.0	11.7	24.5	0.0	5.0	0.0	77.70	67.11	72.81	162.31	144.04	152.83
1	TKA-NS	Right	284.0	268.4	297.7	0.0	0.0	0.0	177.61	169.90	175.25	117.14	123.09	103.51
2	TKA-NS	Left	209.7	268.4	253.9	0.0	0.0	0.0	154.60	147.12	152.51	76.83	77.97	76.95
4	TKA-NS	Bilat-R	61.5	92.8	94.2	3.0	0.0	0.0	82.92	71.04	74.89	120.37	116.04	107.75
5	TKA-NS	Right	92.8	82.9	69.4	0.0	0.0	0.0	61.31	65.38	69.45	118.92	117.11	115.29
6	TKA-NS	Right	121.1	155.0	209.7	0.0	0.0	0.0	97.59	99.93	96.66	121.49	118.54	133.22
7	TKA-NS	Right	160.9	235.3	237.9	0.0	0.0	0.0	189.89	169.58	151.70	157.89	135.50	112.09
8	TKA-NS	Left	165.5	178.7	88.7	3.0	0.0	3.0	129.56	127.46	125.06	38.64	37.06	39.89
9	TKA-NS	Right	195.3	211.8	277.6	0.0	0.0	0.0	130.43	121.90	129.40	103.12	101.01	103.94
10	TKA-NS	Right	431.4	671.3	839.8	0.0	0.0	0.0	263.96	245.17	250.87	84.03	81.45	75.08
11	TKA-NS	Bilat-L	262.5	216.3	268.4	0.0	0.0	0.0	168.44	156.73	159.18	64.28	58.16	55.80
12	TKA-NS	Bilat-R	230.3	**	304.5	0.0	**	0.0	156.32	**	141.68	60.56	**	56.02
13	TKA-NS	Bilat-L	74.4	64.4	66.6	3.0	2.0	0.0	91.99	72.84	79.94	113.46	110.32	101.14
14	TKA-NS	Bilat-R	42.1	30.4	47.0	4.0	1.0	0.0	86.03	82.30	80.41	240.15	228.43	220.70
15	TKA-NS	Bilat-R	253.9	248.4	**	2.0	0.0	**	218.60	179.09	**	105.29	98.01	**
16	TKA-NS	Left	39.1	25.2	32.3	0.0	0.0	0.0	77.24	70.63	74.80	143.69	140.94	136.35

** = missing data

ID	GROUP	SURG LIMB	QUAD0 CSA(CM^2)	QUAD1 CSA(CM^2)	QUAD3 CSA(CM^2)	Qcon0 CSA(CM^2)	Qcon1 CSA(CM^2)	Qcon3 CSA(CM^2)	Qnoncon0 CSA(CM^2)	Qnoncon1 CSA(CM^2)	Qnoncon3 CSA(CM^2)
1	TKA-S	Left	66.85	68.12	71.56	59.54	58.78	64.71	7.31	9.34	6.85
2	TKA-S	Right	68.29	65.60	71.91	63.26	59.61	66.38	5.02	5.99	6.63
4	TKA-S	Bilat-L	32.37	31.46	33.93	25.31	23.21	27.33	7.07	8.25	6.60
5	TKA-S	Left	31.30	29.43	27.55	26.97	25.49	24.01	4.33	3.94	3.54
6	TKA-S	Left	47.51	49.19	46.65	40.97	40.22	39.42	6.54	8.97	7.22
7	TKA-S	Left	68.81	66.86	63.42	58.25	53.96	53.67	10.56	12.89	9.75
8	TKA-S	Right	56.14	58.40	60.30	51.27	53.26	54.80	4.87	5.14	5.50
9	TKA-S	Left	50.88	49.90	54.64	41.23	40.25	47.69	9.64	9.66	6.96
10	TKA-S	Left	100.09	112.52	89.29	93.20	104.43	82.60	6.90	8.09	6.69
11	TKA-S	Bilat-R	61.31	61.78	64.79	52.73	57.71	61.30	8.58	4.07	3.49
12	TKA-S	Bilat-L	63.87	**	60.02	56.12	**	55.32	7.75	**	4.70
13	TKA-S	Bilat-R	39.97	32.59	37.06	36.75	29.82	32.81	3.22	2.77	4.25
14	TKA-S	Bilat-L	51.07	46.12	44.45	41.53	38.46	35.72	9.54	7.66	8.74
15	TKA-S	Bilat-L	93.57	89.27	**	84.53	78.38	**	9.04	10.89	**
16	TKA-S	Right	36.46	34.50	34.15	31.72	29.83	29.01	4.74	4.67	5.13
1	TKA-NS	Right	90.39	88.27	88.82	81.44	77.85	79.50	8.96	10.42	9.33
2	TKA-NS	Left	74.12	72.43	75.69	69.57	67.62	71.24	4.55	4.81	4.45
4	TKA-NS	Bilat-R	35.68	31.69	32.49	29.26	26.83	27.39	6.42	4.87	5.10
5	TKA-NS	Right	33.31	33.15	32.99	30.05	30.23	29.96	3.26	3.15	3.03
6	TKA-NS	Right	43.23	42.97	41.47	36.93	36.84	34.97	6.30	6.13	6.50
7	TKA-NS	Right	92.32	82.63	74.39	75.73	65.47	64.71	16.59	17.16	9.67
8	TKA-NS	Left	55.30	57.87	57.24	50.79	53.34	51.67	4.50	4.53	5.57
9	TKA-NS	Right	64.34	62.18	65.35	51.62	51.58	54.75	12.72	10.61	10.59
10	TKA-NS	Right	126.56	119.10	118.93	121.71	112.54	112.45	4.85	6.56	6.47
11	TKA-NS	Bilat-L	80.38	75.80	77.06	73.19	72.26	74.06	7.19	3.54	3.00
12	TKA-NS	Bilat-R	66.27	**	60.91	60.47	**	54.91	5.80	**	6.00
13	TKA-NS	Bilat-L	40.75	33.27	35.13	36.13	27.77	31.10	4.62	5.50	4.03
14	TKA-NS	Bilat-R	42.05	43.51	40.79	33.41	31.55	29.91	8.64	11.96	10.87
15	TKA-NS	Bilat-R	107.92	98.95	**	100.51	87.57	**	7.41	11.38	**
16	TKA-NS	Left	36.79	33.89	35.59	29.59	29.58	32.00	7.20	4.30	3.59

** = missing data

ID	GROUP	SURG LIMB	F/CSA0 Nm/cm^2	F/CSA1 Nm/cm^2	F/CSA3 Nm/cm^2	EXT0 DEG	EXT1 DEG	EXT3 DEG	AROM0 DEG	AROM1 DEG	AROM3 DEG	PROM0 DEG	PROM1 DEG	PROM3 DEG	FROM0 DEG	FROM1 DEG	FROM3 DEG
1	TKA-S	Left	1.47	1.58	1.72	10	6	2	104	109	116	104	111	118	94	103	114
2	TKA-S	Right	1.40	1.35	1.18	9	5	8	117	111	121	117	117	121	108	106	113
4	TKA-S	Bilat-L	2.51	1.79	2.08	3	3	2	105	109	116	111	110	112	102	106	114
5	TKA-S	Left	0.49	1.58	2.17	8	16	3	117	100	120	117	100	123	109	84	117
6	TKA-S	Left	1.59	1.77	2.01	5	5	4	115	108	115	123	111	117	110	103	111
7	TKA-S	Left	1.04	1.85	2.44	0	2	2	88	103	115	88	109	118	88	101	113
8	TKA-S	Right	1.93	1.75	2.11	6	4	2	117	119	139	121	122	145	111	115	137
9	TKA-S	Left	3.00	2.34	2.01	4	3	0	123	110	123	128	115	128	119	107	123
10	TKA-S	Left	1.73	1.21	1.35	2	15	15	109	120	114	117	127	120	107	105	99
11	TKA-S	Bilat-R	0.93	1.60	2.12	-1	5	2	112	120	124	117	123	128	113	115	122
12	TKA-S	Bilat-L	2.84	**	2.55	1	**	0	131	**	132	131	**	134	130	**	132
13	TKA-S	Bilat-R	1.12	1.63	1.83	-2	4	1	136	96	122	140	105	125	138	92	121
14	TKA-S	Bilat-L	2.17	1.38	1.67	0	9	7	126	107	119	128	11	125	126	98	112
15	TKA-S	Bilat-L	1.81	2.06	**	5	2	**	113	113	**	118	120	**	108	111	**
16	TKA-S	Right	2.04	1.15	1.46	17	16	10	91	82	87	96	87	90	74	66	77
1	TKA-NS	Right	3.41	3.23	3.20	3	2	-1	119	131	134	119	135	134	116	129	135
2	TKA-NS	Left	1.62	1.64	1.77	-1	-2	0	126	121	130	126	129	134	127	123	130
4	TKA-NS	Bilat-R	2.64	1.94	2.38	2	4	3	110	100	106	116	105	102	108	96	103
5	TKA-NS	Right	3.30	3.69	2.35	0	5	0	132	142	143	132	142	147	132	137	143
6	TKA-NS	Right	2.03	2.64	2.87	4	4	-1	121	123	125	123	125	126	117	119	126
7	TKA-NS	Right	2.72	3.54	3.40	1	-1	-1	121	127	130	128	129	133	120	128	131
8	TKA-NS	Left	2.62	2.26	2.58	3	2	3	122	121	130	125	125	134	119	119	127
9	TKA-NS	Right	3.58	3.72	3.91	5	2	-3	126	128	127	132	133	133	121	126	130
10	TKA-NS	Right	2.65	3.11	2.89	-3	-2	-1	112	119	119	115	126	128	115	121	120
11	TKA-NS	Bilat-L	1.91	1.71	1.83	-1	1	0	122	116	118	126	121	122	123	115	118
12	TKA-NS	Bilat-R	3.27	**	2.83	2	**	0	119	**	131	121	**	133	117	**	131
13	TKA-NS	Bilat-L	1.21	1.32	2.07	-2	0	1	136	85	115	138	93	118	138	85	114
14	TKA-NS	Bilat-R	3.06	1.62	2.25	-1	6	3	122	111	120	125	115	122	123	105	117
15	TKA-NS	Bilat-R	1.97	1.96	**	6	3	**	114	105	**	117	112	**	108	102	**
16	TKA-NS	Left	2.02	2.24	1.26	2	0	0	117	123	123	121	129	125	115	123	123

** = missing data

ID	GROUP	MATCH LIMB	IVCPK0 NM	IVCPK1 NM	IVCPK3 NM	IVCPK0 PAIN	IVCPK1 PAIN	IVCPK3 PAIN	CAR0	CAR1	CAR3	CAR0 PAIN	CAR1 PAIN	CAR3 PAIN
1	CON-S	Left	207.32	196.28	224.43	0.0	0.0	0.0	0.99	0.99	0.99	0.0	0.0	0.0
2	CON-S	Right	219.20	178.02	211.40	0.0	0.0	0.0	1.00	0.99	1.00	1.0	0.0	0.0
4	CON-S	Left	117.71	98.38	107.39	0.0	0.0	0.0	0.98	0.98	0.98	0.0	0.0	0.0
5	CON-S	Left	74.94	128.25	117.12	0.0	0.0	0.0	0.91	0.96	0.96	0.0	0.0	0.0
6	CON-S	Left	126.36	118.17	118.36	0.0	0.0	0.0	0.95	0.95	0.93	0.0	0.0	0.0
7	CON-S	Left	171.54	175.70	161.56	0.0	0.0	0.0	0.95	0.97	0.96	0.0	0.0	0.0
8	CON-S	Right	206.56	200.80	174.89	0.0	0.0	0.0	1.00	0.99	0.99	0.0	0.0	0.0
9	CON-S	Left	191.32	194.10	196.82	0.0	0.0	0.0	0.94	0.96	0.94	0.0	0.0	0.0
10	CON-S	Left	319.86	316.31	311.66	0.0	0.0	0.0	0.98	0.98	0.99	0.0	0.0	0.0
11	CON-S	Right	213.66	179.23	179.92	0.0	0.0	0.0	0.94	0.95	0.88	0.0	0.0	0.0
12	CON-S	Left	189.95	183.32	**	0.0	0.0	**	0.96	0.97	**	0.0	0.0	**
13	CON-S	Right	160.91	159.27	152.62	0.0	0.0	0.0	0.97	0.98	0.96	0.0	0.0	0.0
14	CON-S	Left	111.81	107.71	107.45	0.0	0.0	0.0	1.00	0.98	0.97	0.0	0.0	0.0
15	CON-S	Left	247.22	243.23	272.68	0.0	0.0	0.0	0.98	0.98	0.99	0.0	0.0	0.0
16	CON-S	Right	95.99	**	**	0.0	**	**	0.93	**	**	0.0	**	**
1	CON-NS	Right	235.99	235.59	287.19	0.0	0.0	0.0	0.99	0.99	0.99	0.0	0.0	0.0
2	CON-NS	Left	195.96	185.34	192.74	0.0	0.0	0.0	1.00	1.00	1.00	1.0	0.0	0.0
4	CON-NS	Right	116.87	100.82	105.50	0.0	0.0	0.0	0.98	0.98	0.96	0.0	0.0	0.0
5	CON-NS	Right	92.72	111.75	134.13	0.0	0.0	0.0	0.89	0.95	0.97	2.0	0.0	0.0
6	CON-NS	Right	128.78	138.48	124.68	0.0	0.0	0.0	0.95	0.96	0.96	0.0	0.0	0.0
7	CON-NS	Right	222.48	220.32	208.54	0.0	0.0	0.0	0.97	0.97	0.97	0.0	0.0	0.0
8	CON-NS	Left	206.07	177.99	170.89	0.0	0.0	0.0	1.00	1.00	0.98	0.0	0.0	0.0
9	CON-NS	Right	160.08	192.25	219.43	0.0	0.0	0.0	0.67	0.93	0.97	0.0	0.0	0.0
10	CON-NS	Right	332.11	323.76	347.14	0.0	0.0	0.0	0.98	0.98	0.99	0.0	0.0	0.0
11	CON-NS	Left	155.65	153.78	163.63	0.0	0.0	0.0	0.95	0.93	0.94	0.0	0.0	0.0
12	CON-NS	Right	243.75	239.60	**	0.0	0.0	**	0.99	0.99	**	0.0	0.0	**
13	CON-NS	Left	138.92	142.36	123.95	0.0	0.0	0.0	0.99	0.99	0.97	0.0	0.0	0.0
14	CON-NS	Right	114.46	106.82	112.52	0.0	0.0	0.0	1.00	0.99	0.97	0.0	0.0	0.0
15	CON-NS	Right	267.69	272.13	266.32	0.0	0.0	0.0	0.99	0.98	1.00	0.0	0.0	0.0
16	CON-NS	Left	77.43	**	**	0.0	**	**	0.96	**	**	0.0	**	**

** = missing data

ID	GROUP	MATCH LIMB	LEP0 WATTS	LEP1 WATTS	LEP3 WATTS	LEP0 PAIN	LEP1 PAIN	LEP3 PAIN	MLEG0 CSA(CM^2)	MLEG1 CSA(CM^2)	MLEG3 CSA(CM^2)	SUBCUT0 CSA(CM^2)	SUBCUT1 CSA(CM^2)	SUBCUT3 CSA(CM^2)
1	CON-S	Left	315.5	308.1	351.7	0.0	0.0	0.0	160.57	160.07	159.57	42.42	41.56	40.72
2	CON-S	Right	277.6	248.4	274.5	0.0	0.0	0.0	128.84	129.51	126.66	52.15	50.83	48.97
4	CON-S	Left	176.9	157.9	160.9	0.0	0.0	0.0	88.60	89.17	89.21	101.33	103.43	104.16
5	CON-S	Left	102.7	71.8	111.4	0.0	0.0	0.0	89.82	91.25	94.04	91.37	92.43	91.58
6	CON-S	Left	68.5	100.3	90.7	0.0	0.0	0.0	96.52	97.18	100.12	82.28	76.17	73.30
7	CON-S	Left	284.1	256.7	300.9	0.0	0.0	0.0	134.93	135.19	131.47	46.80	48.40	43.56
8	CON-S	Right	227.9	251.1	262.5	0.0	0.0	0.0	137.94	138.60	133.97	40.08	39.66	37.01
9	CON-S	Left	243.0	223.2	170.3	0.0	0.0	0.0	144.66	141.91	147.92	36.10	36.48	36.07
10	CON-S	Left	437.2	360.5	507.9	0.0	0.0	0.0	155.47	154.51	156.66	51.52	46.75	47.47
11	CON-S	Right	251.1	297.5	326.9	0.0	0.0	0.0	134.34	129.87	130.93	51.54	49.22	46.03
12	CON-S	Left	205.4	284.1	**	0.0	0.0	**	129.26	130.37	**	24.69	25.78	**
13	CON-S	Right	223.2	201.3	165.5	0.0	0.0	0.0	101.17	104.47	105.22	93.48	96.92	87.25
14	CON-S	Left	111.4	152.2	148.1	0.0	0.0	0.0	75.19	77.69	80.63	139.59	141.74	144.35
15	CON-S	Left	393.8	443.1	425.8	0.0	0.0	0.0	172.22	167.22	176.71	48.26	44.78	49.79
16	CON-S	Right	47.3	**	**	0.0	**	**	102.65	**	**	112.00	**	**
1	CON-NS	Right	300.9	323.1	369.7	0.0	0.0	0.0	147.55	158.39	169.23	40.40	42.22	44.04
2	CON-NS	Left	189.5	199.3	185.8	0.0	0.0	0.0	119.63	115.76	117.04	54.63	54.11	53.71
4	CON-NS	Right	167.1	171.9	173.6	0.0	0.0	0.0	86.98	87.07	85.93	86.92	88.33	88.97
5	CON-NS	Right	110.5	88.7	125.2	0.0	0.0	0.0	100.65	96.23	98.91	94.16	91.13	90.32
6	CON-NS	Right	72.4	108.7	149.4	0.0	0.0	0.0	97.65	99.71	102.33	83.37	75.16	73.14
7	CON-NS	Right	315.5	274.5	277.6	0.0	0.0	0.0	148.66	145.86	145.48	48.32	50.12	45.25
8	CON-NS	Left	205.4	209.7	287.3	0.0	0.0	0.0	133.80	132.65	130.38	36.27	36.20	35.41
9	CON-NS	Right	256.7	230.3	223.2	0.0	0.0	0.0	145.55	145.33	145.97	37.53	37.01	35.11
10	CON-NS	Right	360.5	339.0	431.4	0.0	0.0	0.0	158.13	158.13	159.58	49.63	49.63	47.95
11	CON-NS	Left	225.5	311.8	300.9	0.0	0.0	0.0	136.65	136.75	135.17	55.77	56.85	49.91
12	CON-NS	Right	211.8	294.0	**	0.0	0.0	**	127.76	135.04	**	26.03	27.88	**
13	CON-NS	Left	160.9	167.1	133.0	0.0	0.0	0.0	88.62	90.56	93.66	82.56	78.13	80.40
14	CON-NS	Right	153.8	156.4	168.7	0.0	0.0	0.0	82.73	85.74	89.82	130.42	132.95	137.20
15	CON-NS	Right	494.0	545.2	414.8	0.0	0.0	0.0	173.04	167.17	173.46	46.71	44.76	47.36
16	CON-NS	Left	53.7	**	**	0.0	**	**	95.61	**	**	105.91	**	**

** = missing data

ID	GROUP	MATCH LIMB	QUAD0 CSA(CM^2)	QUAD1 CSA(CM^2)	QUAD3 CSA(CM^2)	Qcon0 CSA(CM^2)	Qcon1 CSA(CM^2)	Qcon3 CSA(CM^2)	Qnoncon0 CSA(CM^2)	Qnoncon1 CSA(CM^2)	Qnoncon3 CSA(CM^2)
1	CON-S	Left	83.31	82.36	81.41	76.83	75.57	74.31	6.48	6.79	7.10
2	CON-S	Right	67.25	65.69	64.04	61.85	60.68	60.06	5.39	5.00	3.99
4	CON-S	Left	47.07	46.80	46.19	43.89	43.20	43.43	3.17	3.60	2.76
5	CON-S	Left	50.44	51.20	51.60	43.43	41.90	44.58	7.01	9.30	7.03
6	CON-S	Left	52.17	51.79	52.33	47.24	47.81	48.52	4.93	3.98	3.81
7	CON-S	Left	69.03	67.90	65.97	64.59	64.10	62.71	4.44	3.80	3.26
8	CON-S	Right	65.19	65.29	62.17	63.05	62.89	59.60	2.14	2.39	2.57
9	CON-S	Left	73.66	73.96	76.24	68.53	68.25	71.77	5.13	5.71	4.47
10	CON-S	Left	84.79	85.57	85.42	79.81	79.93	79.99	4.99	5.64	5.33
11	CON-S	Right	63.86	62.97	61.47	59.49	59.57	56.61	4.37	3.40	4.86
12	CON-S	Left	57.66	57.82	**	54.94	55.59	**	2.71	2.23	**
13	CON-S	Right	49.96	50.90	49.80	47.53	47.74	45.88	2.43	3.16	3.93
14	CON-S	Left	37.73	37.97	40.66	34.17	33.77	35.57	3.56	4.20	5.08
15	CON-S	Left	90.11	87.51	90.06	85.64	83.93	87.06	4.47	3.58	3.00
16	CON-S	Right	46.62	**	**	43.60	**	**	3.02	**	**
1	CON-NS	Right	82.30	81.45	80.59	75.00	74.55	74.09	7.30	6.90	6.50
2	CON-NS	Left	61.69	59.71	58.49	56.44	55.02	54.92	5.24	4.69	3.57
4	CON-NS	Right	45.02	44.77	43.35	41.46	40.60	39.68	3.57	4.17	3.67
5	CON-NS	Right	52.55	50.22	52.16	47.03	44.49	45.00	5.52	5.73	7.15
6	CON-NS	Right	50.59	52.23	52.82	46.41	48.23	49.24	4.18	4.00	3.58
7	CON-NS	Right	76.82	74.84	73.09	72.69	71.43	69.58	4.13	3.41	3.51
8	CON-NS	Left	63.63	63.31	61.63	59.79	59.22	57.96	3.84	4.09	3.67
9	CON-NS	Right	71.15	71.96	73.10	66.27	67.28	67.81	4.88	4.68	5.29
10	CON-NS	Right	85.64	85.64	85.54	80.63	80.63	79.79	5.01	5.01	5.75
11	CON-NS	Left	67.31	68.65	66.05	62.90	64.29	61.40	4.40	4.36	4.65
12	CON-NS	Right	59.98	60.32	**	57.45	57.41	**	2.53	2.86	**
13	CON-NS	Left	44.84	45.18	44.70	40.75	41.97	42.53	4.09	3.21	2.17
14	CON-NS	Right	40.18	40.61	43.40	35.08	36.93	37.99	5.10	3.68	5.41
15	CON-NS	Right	83.31	82.27	82.56	80.01	78.81	79.69	3.30	3.46	2.87
16	CON-NS	Left	44.52	**	**	42.04	**	**	2.48	**	**

** = missing data

ID	GRP	Match Limb	F/CSA0 Nm/cm^2	F/CSA1 Nm/cm^2	F/CSA3 Nm/cm^2	EXT0 DEG	EXT1 DEG	EXT3 DEG	AROM0 DEG	AROM1 DEG	AROM3 DEG	PROM0 DEG	PROM1 DEG	PROM3 DEG	FROM0 DEG	FROM1 DEG	FROM3 DEG
1	CON-S	Left	2.50	2.41	2.80	-3	-2	-2	129	132	129	133	138	134	132	134	131
2	CON-S	Right	3.29	2.72	3.26	-2	-1	-1	137	140	147	141	147	151	139	141	148
4	CON-S	Left	2.49	2.11	2.29	-1	-1	-1	137	144	144	143	147	148	138	145	145
5	CON-S	Left	1.60	2.84	2.44	-3	-2	-2	141	138	142	145	145	149	144	140	144
6	CON-S	Left	2.48	2.29	2.26	-2	-1	-1	146	143	146	150	148	148	148	144	147
7	CON-S	Left	2.46	2.54	2.39	-2	-1	-1	139	142	142	142	148	148	141	143	143
8	CON-S	Right	3.04	2.96	2.72	-1	0	-1	148	145	149	151	151	155	149	145	150
9	CON-S	Left	2.59	2.64	2.54	-2	0	-1	145	149	149	146	152	150	147	149	150
10	CON-S	Left	3.72	3.67	3.61	-1	-1	-1	138	140	140	146	146	146	139	141	141
11	CON-S	Right	3.33	2.79	2.95	1	1	0	136	140	137	140	143	140	136	139	137
12	CON-S	Left	3.21	3.06	**	-1	-1	**	146	138	**	149	149	**	147	139	**
13	CON-S	Right	3.14	3.09	3.08	-1	0	0	136	140	138	146	143	142	137	140	138
14	CON-S	Left	3.03	2.96	2.80	-2	0	-1	134	138	135	140	146	140	136	138	136
15	CON-S	Left	2.68	2.69	2.90	-3	-3	-3	130	131	132	135	142	147	133	134	135
16	CON-S	Right	2.04	**	**	-1	**	**	132	**	**	137	**	**	133	**	**
1	CON-NS	Right	2.92	2.93	3.59	-3	-1	-2	129	125	126	133	130	132	132	126	128
2	CON-NS	Left	3.22	3.12	3.25	-2	0	0	135	140	140	141	146	155	137	140	140
4	CON-NS	Right	2.61	2.30	2.46	-1	-1	-1	140	139	142	145	142	145	141	140	143
5	CON-NS	Right	1.83	2.33	2.76	-2	-2	-2	140	137	137	142	144	142	142	139	139
6	CON-NS	Right	2.57	2.66	2.35	-2	-1	-2	147	144	144	151	150	151	149	145	146
7	CON-NS	Right	2.84	2.86	2.78	-1	0	-2	139	138	143	144	143	148	140	138	145
8	CON-NS	Left	3.20	2.79	2.73	-1	0	-1	150	146	148	153	155	154	151	146	149
9	CON-NS	Right	2.24	2.65	3.00	-2	0	0	142	149	147	142	151	150	144	149	147
10	CON-NS	Right	3.82	3.72	4.03	-1	-1	-1	136	138	138	146	145	146	137	139	139
11	CON-NS	Left	2.29	2.22	2.47	0	0	0	135	140	135	141	142	140	135	140	135
12	CON-NS	Right	3.93	3.87	**	-1	-1	**	146	136	**	148	146	**	147	137	**
13	CON-NS	Left	3.16	3.14	2.70	-1	-1	-1	142	148	145	143	147	147	143	149	146
14	CON-NS	Right	3.02	2.68	2.75	-2	-1	-2	134	139	137	140	145	143	136	140	139
15	CON-NS	Right	3.10	3.20	3.10	-3	-3	-4	126	131	133	133	142	148	129	134	137
16	CON-NS	Left	1.71	**	**	-1	**	**	132	**	**	138	**	**	133	**	**

** = missing data