An investigation of the biomechanical factors influencing knee joint function following total knee replacement.

by
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ABSTRACT:

Knee replacement surgery is often performed in cases where the pain due to knee osteoarthritis can no longer be effectively controlled by conservative measures. The surgery—which replaces the articular surfaces of the tibia, femur and sometimes the patella with a mix of metal and polyethylene—is one of the most commonly performed lower limb surgeries. Despite patient reports of reduced pain and increased participation in activities of daily living, knee function following total knee arthroplasty (TKA) does not return to normal. Reductions in both passive and active knee range of motion, alterations in magnitude and timing of muscle activity, and changes in knee joint kinetics have all been reported. Comparatively little is known about why knee function is altered following TKA. In an attempt to address this gap in the literature, this thesis was designed to examine the biomechanical factors thought to contribute to reduced knee extensor moments following TKA. In particular, the contribution of alterations in passive knee moments, muscle moment arm lengths, and muscle activation magnitude and timing were examined in detail to determine which factors contributed to reduced knee extensor moment following TKA. To accomplish this goal, two groups--6 healthy controls and 6 individuals who were at least one year post-TKA—were examined in four different studies.

The initial study of this thesis, a comprehensive examination of patients and controls during gait and stepping onto a raised surface, had two purposes. This moment data was needed to clearly define the knee moment deficits that existed in the patient – the remainder of the thesis focused on determining why these specific deficits existed. A secondary aim of this first study was to allow for the assessment of muscle activation patterns in this population during weight bearing tasks. Results of study one confirmed the existence of knee extensor moment deficits in the TKA group and also demonstrated that patients exhibited increased gastrocnemius EMG, prolonged stance phase activation of biceps femoris and reduced knee flexion during the loading phase of stance. Analysis of results across the two tasks suggested that reduced knee moments in members of the TKA group may be related to changes in gastrocnemi activation, however, differences in knee joint kinematics between patients and controls made it difficult to draw this conclusion.

Study number two was designed to further probe the muscle activation strategies adopted by members of the TKA group. In order to avoid complications involved when comparing muscle activation patterns between groups using different kinematic strategies, seated knee extension was examined. This task was chosen as it challenged the knee musculature while at the same time restricting both groups of participants to use the same knee joint kinematics. The results of this analysis revealed that, while EMG magnitude for quadriceps and hamstrings were similar for both groups, patients exhibited increased amplitude of EMG in both the medial and lateral gastrocnemi.

While the first two studies of this thesis focused on the active knee joint moment, in the third study the force required to passively move the knee from a flexed to an extended position was measured and used to estimate the passive moment of the knee joint. This study was based on the rational that if the passive knee moment was altered following TKA it could potentially affect the net knee joint moment. The comparison of patient and control results showed that, while small changes in passive moment were evident in patients, these differences were not large enough to account for changes in the knee extensor moment.

The goal of the final thesis study was to examine moment arms of the knee musculature in an attempt to determine if TKA resulted in changes to this variable. However, various methodological issues arose in the course of completing this study. As a result, limited data were produced that sufficiently addressed the question posed. Despite the problems that arose, important issues regarding *in vivo* moment arm determination were realized and are included for discussion in this thesis.

Together, the four studies provided a unique opportunity to observe knee function over a range of activities. The following conclusions were reached.

- Changes in passive knee moment did not seem to contribute to reductions in knee extensor moment observed following TKA.
- Quadriceps and hamstring muscle function, as evidenced by EMG recording during the seated knee extension task, appeared intact following TKA, suggesting

that alteration in the function of these muscles were not directly responsible for reductions in knee extensor moments.

- Changes in medial and lateral gastrocnemi activation were observed during knee extension and weight-bearing tasks. These changes may account for reduced knee extensor moment, particularly during the step-up task.
- Knee extensor moment reductions during gait appear to be related to the reduced knee flexion exhibited by patients during the stance phase of gait. It was hypothesized that these changes in knee kinematics were directly linked to the increased activation of the gastrocnemi during gait.

Although low subject numbers limited the generalizability of the results these conclusions will serve to guide future research in this area and ultimately help improve function and quality of life in this patient population.

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Dedication

To my students at Memorial University of Newfoundland – your enthusiasm, desire to learn and hard work provided the inspiration I needed to complete this project. I will be forever grateful to you all.

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Chapter 1

Background information and literature review

Introduction:

The first knee joint replacements were performed in the early 1970s, however, most initial designs were plagued by failure (Andriacchi and Hurwitz 1997). This was due to gross simplifications of knee joint anatomy (i.e. some prostheses were designed to function as a simple hinge), to the non-physiological articular surface loading that resulted due to excessive articular surface conformity (Andriacchi 1986), and a lack of understanding of the asymmetrical nature of knee loading patterns during weight bearing (Andriacchi and Hurwitz 1997). Since that time, total knee arthroplasty (TKA), reserved primarily for the treatment of severe knee osteoarthritis (Dieppe et al 1999), has grown to become one of the most commonly performed orthopaedic surgeries in the lower limb.

Over the past 40 years, improved understanding of knee function and advances in both technology and surgical techniques, have resulted in enhanced prosthesis function and durability. Despite these improvements, patients continue to exhibit long-term functional deficits (Andriacchi and Hurwitz 1997) and to report greater difficulty with lower limb function during activities of daily living then do those with healthy knee joints (Walsh et al 1998; Finch et al 1998). While researchers have worked diligently to quantify post-operative changes in knee function, and while they have proposed numerous hypotheses to explain why deficits in knee function persist, relatively little effort has been expended in examining these hypotheses. As a result, current understanding of the factors contributing to post-operative functional deficits remains limited.

For clinicians working with individuals who have undergone TKA, understanding the factors underlying knee deficits is imperative, because a problem whose cause is not understood cannot be effectively and efficiently treated. If this next step in research is not taken, we run the risk of applying misdirected treatments and possibly limiting functional recovery. The goal of this research was, therefore, to perform a comprehensive and detailed analysis aimed at identifying factors responsible for functional deficits following TKA. If one stops to consider the myriad of possible factors that influence function during any given task, it is apparent that the list is potentially endless (i.e. mechanical,

neurological, physiological, and psychosocial). The challenge of identifying and adequately describing the contribution of each factor would be a daunting one. To make this task more manageable, the specific goal of this research was limited to exploring the biomechanical factors leading to altered knee extensor moments in individuals following TKA. Reduced knee extensor moments are a common problem in this clinical population. Given the integral role the knee joint plays in supporting the body during weight bearing(Winter 1980) and absorbing energy that results with foot contact on the floor (Jefferson et al. 1990), deficits in the knee extensor moment can potentially have a large impact on lower limb performance. Attempts to optimize function in this population, would therefore seem to have to include interventions aimed at maximizing the knee extensor moment. As discussed above, in order for these interventions to be as effective and efficient as possible, factors underlying the extensor moment deficits must be better understood. The choice was made to specifically study biomechanical factors as changes in joint geometry and alterations in knee joint kinematics and sensory function known to occur following the surgery all have the potential to affect the biomechanics of the joint. It should be noted, however, that this choice does not in anyway suggest that other factors are not important – in all likelihood numerous factors contribute.

1.1 Background information

1.1.1 The healthy knee joint

The knee joint, one of the largest and most complex joints in the human body (McGinty et al. 2000), is comprised of 3 bones and two main articulations – the tibiofemoral (TF) and patellofemoral (PF) joints.

Tibiofemoral joint:

The TF joint is formed by the articulation of the medial and lateral condyles of the femur with the two tibial plateaus and is described as containing a medial and lateral compartment (Blackburn and Craig 1980). Two menisci (medial and lateral) are located on the medial and lateral tibial plateaus respectively. The menisci, fibrocartilage, crescent shaped wedges (McDermott and Amis 2006), act to absorb shock (Voloshin and Wosk

1983), decrease joint surface stress by distributing loads over a greater surface area (Baratz et al. 1986), and function as secondary stabilizers due to the increased joint conformity they help create (Allen et al. 2000).

The TF joint has 6 degrees of freedom (DoF) with the greatest motion occurring about the medial/lateral axis (approximately 0 – 120º of active flexion). Initial investigations of knee joint kinematics suggested that flexion/extension was accompanied by femoral rollback – a rolling of the medial and lateral femoral condyles on the tibia that was thought to permit greater range of knee flexion (Williams and Logan 2004). Recent research, suggests that femoral rollback, as defined above, does not occur (Pinskerova et al. 2000). Although controversy still exists, current research suggests that the medial femoral condyle remains relatively stationary while the lateral condyle rolls posteriorly (Pinskerova et al. 2000, Freeman and Pinskerova 2003, Johal et al. 2005). As a result of this asymmetrical rollback, tibial internal (femoral external) rotation is observed during knee flexion (Johal et al. 2005). This axial rotation appears to be intimately linked to knee motion from 0 – 20ºof flexion; beyond 20º, the amount of axial rotation depends on loading and muscular forces (Hill et al. 2000; Benoit et al. 2007)

Due to the incongruity between the femoral condyles and tibial plateaus, the TF joint has little inherent bony stability (Williams et al. 2001). While the menisci help improve congruity, most stability at the knee comes from the combined 'actions' of muscles, ligaments, and joint capsule (Williams et al. 2001). The four primary ligaments that contribute to knee stability are the anterior and posterior cruciate ligaments (ACL and PCL) and the medial and lateral collateral (MCL and LCL) ligaments. A detailed review of the functions of these ligaments is beyond the scope of this work (see Woo et al. 1998; Sanchez et al. 2006; Bray et al. 2005; Woo et al. 2005). Briefly, the ACL and PCL are primarily responsible for providing sagittal plane and axial rotation stability while the MCL and LCL provide frontal plane passive support (Guiliani et al. 2009). The ACL and PCL are also thought to help guide tibiofemoral motion during knee flexion and extension (see review by Mcginty et al. 2000). As a result, ligament laxity or absence may result in altered knee joint kinematics. For example, Velde et al. (2009) have

recently shown that ACL deficient knees exhibit abnormal anterior translation and increased internal tibial rotation. Logan et al. (2004) have also observed altered tibiofemoral motion during weight bearing when the PCL is absent.

As reviewed by Williams et al. (2001), the tensile strength of these ligaments is insufficient to withstand loads applied during demanding activities. In these situations, additional stability is provided by muscles (through the compression forces they produce and their ability to resist bone motion) and the joint compression forces that result when weight-bearing (Schipplein and Andriacchi 1991). Active muscle contribution to knee stability during functional activities requires some means by which knee muscle forces can be precisely controlled. Fundamental to this control are the various sensory receptors located in the joint and surrounding tissues. Working together, sensory receptors in knee ligaments, the menisci, joint capsule and tendons form a complex network of sensory information that are integral to knee joint stability and function (Solomonow 2006; William et al. 2001; Solomonow and Krosgaard 2001). Knee ligament injury or laxity, therefore, not only impairs passive joint restraints, but also has been shown to affect muscle function at the knee joint (see Yasuda et al. 1999; Shultz et al. 2004).

Patellofemoral joint:

The PF joint, the second of the two joints comprising the knee, exists as the articulation between the trochlear groove of the femur and the articular surface of the patella. Normal function of the PF joint is integral to optimal knee performance. Stability of the joint is provided by a combination of passive (ligaments and retinaculi) and dynamic (muscles – primarily the quadriceps) structures (McGinty et al. 2000; Feller et al. 2007). Patellar gliding during knee motion is essential to normal knee kinematics (McGinty et al. 2000; Feller et al. 2007), while a lack of patellar stability has been implicated in the development of patellofemoral pain syndrome (Sheehan et al. 2009). The patella also acts to increase the moment arm of the knee extensors, thereby improving their mechanical efficiency (Draganich et al. 1987; Ward et al. 2005). In so doing the patella functions as a mechanical fulcrum that acts to help reduce loads on the knee joint during quadriceps activation.

1.2 Total knee arthroplasty

As discussed briefly above, TKA is a surgical procedure that is most commonly performed as treatment for severe knee osteoarthritis (OA). During knee replacement surgery, the articular surfaces of the femoral condyles are replaced by metal, while the tibial plateaus are removed and a metal tray is implanted. Polyethylene spacers, placed on the tibial tray, ensure that no metal on metal articulation occurs. The spacers also provide surgeons with flexibility in determining joint spacing, which is essential to ensuring proper tensioning of ligaments and other soft tissues (Barrett et al. 1990). In some cases, the articular surface of the patella are also replaced, however, controversy still appears to exist related to the merits of this procedure (Helmy et al. 2008). Due to the limited life span of the prosthetic materials, TKA used to be done almost exclusively in individuals over the age of 65. Recent advances in material engineering and implant design have resulted in longer lasting prostheses that can be implanted in younger individuals (Kurtz et al. 2009; Jain et al. 2005).

Knee prostheses can be done on one compartment of the knee (typically the medial) or on the total joint (Lutzner et al. 2009). As the investigation outlined in this document will focus exclusively on total knee replacements, unicompartmental designs will not be discussed (the reader is referred to Lutzner et al. 2009 and Brouwer et al. 2005 for more information regarding unicompartmental replacements). Knee replacement design is constantly changing, with numerous models and designs of prosthesis available to surgeons. Each design varies slightly from the other and makes different claims regarding performance and function (Victor 2006). A review of these many variations is not possible in this document - however, there are two design characteristics that warrant specific attention. The first is related to the mobility of the polyethylene tibial insert, and the second relates to whether the PCL is retained or removed during surgery.

Fixed-bearing vs. mobile-bearing:

As the names suggest, the primary difference between these two design attributes is the mobility of the polyethylene tibial tray. In fixed-bearing designs, the tray is fixed to the tibial plate; in a mobile-bearing model, a certain degree of axial rotation is permitted by

the tibial tray. Original prostheses were designed to be fixed-bearing. However, with improved understanding of the forces experienced by the implanted materials and also a better appreciation for normal knee kinematics, Goodfellow and O'Connor introduced the idea of mobile-bearing replacements (Barrett et al. 1990). When first introduced, it was argued that a main benefit of this design feature was that mobile-bearing knees would result in more normal knee kinematics (i.e. axial rotation of tibia during knee flexion/extension) and, as a result wear rates would be lower (Barrett et al. 1990). Despite these early claims, researchers comparing mobile and fixed bearing designs have found little, if any, difference in short term or long term performance and longevity (see Barrett et al. 1990; Harrington et al. 2009; Vogt and Saarbach 2009; Ladermann et al. 2008).

PCL retaining vs. PCL sacrificing:

Original prostheses required that both the ACL and PCL be sacrificed. Problems arising with these designs (i.e. limited knee flexion and posterior tibial subluxation) were felt to be due to the lack of PCL (Conditt et al. 2004). Design changes that resulted in the PCL being retained or having its function substituted for (Delp et al. 1995) were subsequently devised. In PCL substituting prosthesis, the design was altered so that PCL function would be mimicked by the design. This was usually done by the addition of a tibial spine that interacted with a femoral cam (Delp et al. 1995). In theory, this design modification provided increased anterior-posterior stability and more normal femoral roll-back (Victor 2006, Delp et al. 1995). The choice of whether to implant a PCL retaining or PCL substituting design is generally based on surgeon preference, but, in situations where OA has left the PCL lax and non-functioning, preference may be given a PCL substituting prosthesis (Victor 2005). Despite considerable research examining the potential benefits of PCL retaining vs. substituting prostheses, a recent Cochrane Review (Jacobs et al. 2005) on the subject found no evidence to suggest that the performance of one was better than the other.

The ACL in knee joint replacement:

Despite the importance of the ACL to knee stability and normal joint kinematics (Guiliani et al. 2009), TKA with ACL retention is uncommon (Moro-oka et al. 2007).

The relatively low number of bi-cruciate retaining knee replacements is due primarily to the technical difficulty of the surgical procedure (Stiehl et al. 2000; Pritchett 1996). Also, the ACL is sometimes too degenerated to be worth saving (Cloutier et al. 1999), so it is removed. Despite these difficulties, and the relatively low incidence rates for bi-cruciate retaining TKA, favourable results have been shown in the literature. Pritchett (1996) examined individuals who had undergone bilateral TKA (a bi-cruciate retaining prosthesis in one knee and a PCL retaining prosthesis in the other). This author reported that patients 'preferred' the feel of the bi-cruciate retaining prosthesis over the PCL retaining one. Knee joint kinematics also appeared more 'normal' when the ACL was retained (Moro-oka et al. 2007), and Clouteir et al. (1999) have highlighted the benefits of knee motion being guided by ligaments as opposed to constraints imposed by the prosthesis. These apparent benefits notwithstanding, ACL retention during TKA remains the exception as opposed to the rule.

1.3 Literature review: Knee joint function following TKA

1.3.1 Knee joint sensory function in individuals following TKA:

In addition to being a primary weight bearing joint of the lower limb, sensory information from the knee joint also makes an important contribution to overall lower limb and whole body function. As with most other joints in the body, the knee joint contains numerous mechanoreceptors. These mechanoreceptors, contained primarily in ligaments, the joint capsule and menisci, serve numerous roles. Their part in modulation of muscle activity was examined in section 1.2.1. In addition to their influence on muscle activation, knee mechanoreceptors have been shown to be involved in the detection of knee joint motion (Pap et al. 1999), knee joint position sense (Bonfim et al. 2003), dynamic balance (Courtney et al. 2006) and recovery from standing postural perturbations (Bloem et al. 2000; Gage et al. 2007 and 2008). While this is not meant to be an exhaustive list, it does provide evidence of the importance of knee joint sensory information to functional activity. As TKA typically results in removal of the ACL (and sometimes the PCL) as well as the menisci, it is not surprising that questions have arisen related to how the sensory function of the knee may be altered following TKA.

Most of the research examining knee sensory function post-TKA has focused on knee joint proprioception (see for example Gage et al. 2007; Swanik et al. 2004; Cash et al. 1996; Issac et al. 2007). Research to date has resulted in conflicting findings regarding proprioception following TKA. Knee position sense (Ishii et al. 1997; McChesney and Wollacott 2000; Wada et al. 1997) and balance control (McChesnet and Wollacott 2000) in individuals who have undergone TKA have both been shown to be similar to healthy controls. In contrast, Gage et al. (2008) have reported altered balance control and Pap et al. (2000) have reported decreased knee joint position sense in individuals who have undergone TKA. While some of these contradictions are due to differences in experimental paradigms, it seems fair to conclude that at present the true effects of TKA on knee joint proprioception are unknown. That having been said, it appears reasonable, given the rich sensory function of structures removed during the surgery, that knee joint sensory function is not normal in individuals who have undergone TKA.

1.3.2 Knee joint kinematics and kinetics during gait in individuals following TKA:

By far the most common kinematic deficit identified during gait in this population is decreased stance phase knee flexion – patients typically weight bear on a straighter knee (Jevsevar et al 1993; Andriacchi et al 1982; Draganich et al 1999, Weidenhelm et al 1993; Wilson et al 1996, Myles et al 2002; Yoshida et al. 2008). Myles et al (2002) and Wilson et al (1996) have also shown that maximum swing phase knee flexion is reduced following TKA.

In contrast to kinematic analyses, far fewer studies have examined knee kinetics in this population. Most of the existing kinetic research has demonstrated alterations in both the magnitude and pattern of stance phase knee moments following TKA. In one of the first studies examining kinetics during gait post-TKA, Andriacchi et al. (1982) observed three typical moment patterns. While control subjects exhibited the characteristic biphasic flexion/extension knee moment during stance, patients produced either a primarily flexor

pattern (16/35), a primarily extensor pattern (11/35), or a normal pattern (8/35). In all cases, the moments produced by patients were smaller than those of controls. Wilson et al (1996) reported the same three stance phase moment patterns in their study of patients who were an average of 46 months post TKA (5/16 knee extension moment, 7/16 knee moment dominated by a flexion pattern, and 4/16 normal biphasic pattern). Simon et al (1983) observed similar knee moment patterns. In all of these cases, even if a normal sagittal plane moment pattern was observed, magnitudes were less than control values. As part of a larger study, Jevsevar et al (1993) reported that peak surgical knee extensor moments during stance were reduced in a group of patients at least one year post-arthroplasty. Although this decrease was not significant, Jevsevar's results demonstrated a strong trend toward decreased peak knee moments in the post-op group.

1.3.3 Knee kinematics and kinetics during stepping-up and stair climbing in individuals following TKA:

Andriacchi et al (1982) were the first to examine stair climbing in a TKA population. They reported that patients used decreased knee range during both stair ascent and descent. Recent work by Myles et al (2002) has supported these findings, reporting decreased active knee flexion during both stair ascent and descent in patients 24 months post-TKA. In contrast, Wilson et al (1996) reported decreased knee flexion range during descent only and Catani et al (2003) observed knee flexion deficits only during ascent.

As was the case with level gait, knee moments are also altered when individuals ascend stairs following TKA. Andriacchi et al (1982) identified two main moment patterns during stair climbing – one dominated by a knee extensor moment (all controls used this pattern) and a pattern consisting of extension and then flexion moments (most patients exhibited this pattern). Net joint moments during stair climbing have subsequently been examined by Andriacchi et al (1993),Wilson et al (1996), and Catani et al (2003), all of whom reported decreased knee extensor moments in the stance limb. However, the variations in moment patterns initially identified by Andriacchi et al (1982) have not been consistently observed in these more recent studies.

Fewer studies have examined stair descent following knee arthroplasty. As a result, knee performance during this task is less well defined. A recent study by Catani et al. (2003) examined patients who had undergone unilateral TKA between 11 and 20 months post-op. They reported that patients exhibited normal peak knee extension moments when descending stairs. The 57˚ range of knee flexion exhibited by patients in this study did not differ significantly from control subjects. This was in contrast to the significantly reduced knee excursion (49˚ vs. 57˚) and peak knee moment exhibited by these same patients during stair ascent. Wilson et al (1996) reported slightly different findings during stair descent, with patients an average of 46 months post-op demonstrating significantly decreased knee excursion and reduced knee extension moment.

Analogous to these evaluations of stair climbing is the work of Draganich et al. (1999), Gage (1999) and Byrne et al. (2002). These authors examined the knee performance of patients required to step on to a raised surface. Over 50% of the patients examined in these studies could not step on to the highest height used (30cm step for Draganich; 20cm step for Gage and Byrne). Participants who could complete the step-up did so with less surgical knee flexion (Gage 1999; Draganich et al. 1999), decreased stance phase peak external knee extension moments when the surgical limb led the step-up (Draganich et al. 1999), and less surgical knee joint work during lead limb stance (Byrne et al. 2002).

Gage (1999) also examined CoM displacement, velocity and acceleration in both the medial/lateral (M/L) and anterior/posterior (A/P) directions during both step-up and step-down. He concluded that members of the TKA group exhibited decreased postural control due to observed increases in CoM velocity (A/P and M/L) during surgical single limb support, and ‘inappropriately small’ decelerative forces at the end of the stepping movement. Patients in the Gage study also demonstrated decreased surgical knee joint angular velocity during a step-up and step-down task.

1.3.4 Knee kinematics and kinetics during other activities in individuals following TKA:

Farquhar et al. (2008) examined knee joint kinetics while participants performed a sit-to-stand task. Compared to healthy controls, Farquhar found that individuals who had undergone TKA at least one year earlier exhibited a decreased knee extensor moment and increased hip extensor moment when standing from a seated position. Su et al. (1998) performed a similar analysis, however they examined individuals prior to and following TKA rising from 4 different chair heights. While all patients were able to successfully rise from a seated position, at lower chair heights patients exhibited decreased peak knee extensor moments. This decrease in extensor moment may have been related to decreased mechanical demand resulting from the significantly greater anterior CoM displacement exhibited by patients. As part of their examination of function post-TKA Jevsevar et al. (1993) had patients rise from a chair adjusted to knee height. Their moment data agreed with that of Su et al, although the decreases in peak knee extensor moment were non-significant. Patients in the Jevsevar study, all of whom were more than one year post-TKA surgery, also used approximately 10˚ less knee flexion then controls when rising from a chair.

Obstacle avoidance has also been examined in the post-arthroplasty population. Byrne and Prentice (2003) reported that patients performed less knee flexor work during the elevation phase of swing over both 6cm and 18cm obstacles. In addition, the maximum amount of knee flexion produced by patients during swing was significantly less than controls. Despite these differences, patients were able to maintain toe clearance at control levels for both obstacle heights by increasing hip work and hip hiking and via increased lateral toe displacement during swing.

Myles et al (2002) examined sagittal plane range of motion during slope ascent/descent and while getting into and out of a bathtub. In keeping with the findings presented above, they found decreased flexion/extension excursion for both tasks at 24 months post-op. In recent work, Gage (2007 and 2008) examined postural responses to small magnitude (i.e. 7.5º) sagittal and frontal plane perturbations in individuals who had undergone TKA.

While all patients were able to maintain balance when perturbed, they demonstrated decreased knee flexion in response to both A/P and M/L perturbations.

1.3.5 Kinetics at the ipsilateral hip and ankle following TKA:

As reviewed above, knee moment deficits following TKA are common. Relatively little is known however, about how the performance of the ipsilateral ankle and hip are affected by the surgery. The work of Byrne et al. (2002) provided a detailed examination of ipsilateral hip and ankle function in this population. In this work, Byrne reported that individuals who had undergone TKA produced increased net hip extensor work when performing a step-up. This increase in contributions from the hip appeared to be a compensation for the decreased knee extensor work observed in the same participants. The only other report of ipslateral joint function is from Winter (1980), who reported data from one individual who had undergone TKA. During stance phase of gait, this individual exhibited a decreased knee extensor moment and increased hip extensor moment.

Winter (1980) used the idea of the support moment (the algebraic sum of the moment of the hip, knee and ankle) to explain this shift from knee to hip that has been observed following knee replacement. Winter's argument was that while the ankle, knee and hip all contribute to lower limb support, it is the net affect (i.e. the support moment) that determines whether limb collapse occurs. If the net extensor moment produced by the ankle, knee and hip is sufficient to counteract the flexor moments acting on the body (i.e. due to gravity or external loads) then limb collapse with not occur. Alternatively of the net extensor moment is not adequate, then limb collapse is likely. Winter (1980) also showed that while the pattern of the support moment remains relatively constant during gait, the contribution of the hip and knee to this moment was quite variable. This led Winter to conclude that contributions of the hip and knee to overall lower limb support may vary depending on the task and or the characteristics of the individual. Extending this argument, it appears that following TKA, the contribution of the hip to the total support moment is increased to ensure lower limb support is maintained in the face of

decreased knee extensor moments. As Byrne et al. (2002) highlighted, this strategy may result in increased loads being placed on the hip joint, potentially resulting in increased risk of injury to the hip.

1.3.6 Activation of the knee musculature following TKA:

It seems reasonable to hypothesize that the knee musculature would not function normally following TKA. It nothing else, the absence of the ACL and often the PCL would likely impact muscle activation. Despite this rational, relatively few published reports of muscle activity post-TKA exist. Three groups of investigators (Wilson et al. 1996, Benedetti et al. 2003, Mizner and Snyder-Mackler 2005) have combined kinematic and kinetic data with electromyography (EMG) to analyze gait following TKA. The results did not provide any conclusive evidence of the effect of TKA on lower limb EMG - Wilson et al. (1996) reported increased quadriceps activity, Mizner and Snyder-Mackler (2005) observed normal quadriceps EMG amplitude, and Benedetti et al. (2003) reported prolonged activation of rectus femoris and hamstrings.

Mizner and Synder-Mackler (2005) and Farquhar et al. (2008) reported lower limb EMG data during a sit-to stand task in individuals who had undergone TKA. Results from these authors suggested the EMG magnitude may change as patients recover from the surgery. Mizner's group tested individuals at 3 months post-surgery and found decreased quadriceps activation, while Farquhar's group tested at one year after surgery and reported no EMG magnitude changes. Gage et al. (2007, 2008) also examined lower limb EMG in a TKA population. These investigations, which examined muscular and kinematic responses to support surface translations following TKA, found that patients exhibited some minor changes in temporal pattern of EMG. One difficulty arising in all of the above studies is that often, in addition to EMG changes, the patients group being studied performs the task using kinematics that differ from those of healthy individuals. These differences in kinematics, make interpretation of EMG findings difficult. The researcher must try to determine whether the observed EMG differences are indicative of

some underlying change in muscle activation or whether they are merely reflective of the fact that the lower limb kinematics differ between the groups.

The only studies that have avoided the problems discussed above are those investigating muscle inhibition post-TKA. This work, performed primarily by Stevens et al. (2003) and Mizner et al. (2003) has demonstrated that following TKA, patients exhibit increased amounts of quadriceps muscle inhibition. One of the drawbacks of this work however, is that most of the investigations have been done within 6 months of knee replacement surgery. As a result it is likely that quadriceps inhibition, resulting from the effects of surgery on the knee joint, may still be influencing muscle function. These muscle inhibition studies need to be carried out on individuals at least 1 year post-TKA in order to provide better insight into the extent of muscle inhibition in this population.

1.4 Summary of research questions:

As previously discussed, the primary aim of the proposed research is to examine the biomechanical factors that contribute to deficits in the knee extensor moment following knee arthroplasty. The specific questions, along with a brief rationale for each, are outlined below:

1. *Do patients exhibit altered muscle activation magnitude and timing following TKA?* **Rationale:** Moments produced by muscles are a primary factor in determining the magnitude of the net joint moment. Changes in either the magnitude of muscle activation or the pattern of activation therefore have the potential to impact the net joint moment. Specifically, activation magnitude, muscle onset and offset timing, and the degree of co-activation will be examined. Muscle activation will be examined during three different movements: seated knee extension, gait, and stepping-up. As discussed above, individuals who have undergone knee joint replacement often exhibit changes in knee joint kinematics during weight bearing tasks. As such changes complicate the interpretation of EMG data, it is preferable to examine muscle activation during a task where knee

kinematics are constrained to be similar for everyone. Seated knee extension is just such a task, hence its inclusion in this thesis. The seated knee extension work will be carried out in Study 2, while the gait and step-up data will be examined together in Study 1.

Hypotheses:

- Patients will exhibit prolonged activation of quadriceps and hamstrings during seated knee extension, gait, and stepping-up.
- Patients will exhibit increased co-contraction of quadriceps and hamstrings during seated knee extension, gait, and stepping-up.
- Patients will exhibit increased activation magnitudes for the quadriceps during seated knee extension, gait, and stepping-up.

1. *Does passive resistance to knee motion change following TKA?*

Rationale: The net knee joint moment is comprised of both active and passive components. To date no efforts have been made to quantify passive resistance to knee motion in the TKA population. Following knee replacement, changes in articular surface friction (Zilhmann et al 2005), altered joint geometry (Victor and Bellemans 2006) and potential alterations in soft-tissue tension suggest that passive resistance to joint motion may be changed. Study 3 of this thesis will specifically address this issue in order to determine if altered passive resistance to knee motion is a factor contributing to the reduction in knee moments following TKA.

Hypothesis:

- Individuals who have undergone TKA will exhibit increased sagittal plane passive moments compared to age matched individuals with no history of knee pathology.

3. *Are moment arms of muscle crossing the knee joint altered following TKA?*

Rationale: In addition to force magnitude, moment arm is directly related to the moment a force can produce. It therefore seems imperative that any examination of factors influencing the net knee moment following TKA must include an investigation aimed at quantifying the moment arms of knee musculature in this population. Study 4 of this thesis will examine the in vivo moment arms of muscle crossing the knee joint as individuals perform a knee extension task.

Hypothesis:

- Moment arms of the knee musculature in members of the TKA group will differ from moment arms found in the healthy control group. The direction of this difference (i.e. greater or less) is unable to be predicted based on available literature.

4. *Do any of the changes observed in passive resistance, muscle activity and moment arm lengths have the potential to reduce the net knee extension moment during gait and stepping-up?*

Rationale: The overall goal of this thesis was to examine the biomechanical factors that influence the net knee extensor moment in an attempt to understand why sagittal plane knee moments are reduced following TKA. Chapter 7 of this thesis will be used to consolidate the findings of Studies 1, 2, 3 and 4 in an effort to answer this bigger question.

Hypotheses:

- Changes in passive knee moments will not be of a large enough magnitude to have a substantial affect on the net knee extensor moment.
- Changes in muscle activation, particularly increases in co-contraction, will be observed to occur simultaneously with reduced knee extensor moments during gait and stepping-up. As a result, it is hypothesized that these muscle activation changes will be partially responsible for the reduced knee extensor moment observed following TKA.

- Reduced quadriceps moment arms, will mean these muscles have less of an ability to produce a knee extensor moment following TKA, although it is not anticipated that the change will be large enough to account for the reduction in knee extensor moment observed in this population.

Chapter 2
General methodology

2.1 Overview of experimental protocol:

A series of four studies were performed to address the specific research questions posed by this thesis. One of the unique aspects of these studies was that they were carried out using the same participant group, with all testing being done on the same day. Although this meant individual participants were at the lab for an extended period of time (3-4 hours) it was felt to be essential to the overall results of the thesis. The alternatives – using different participants for each study or having the same participants come in on consecutive days – were not considered; as it was felt they would introduce unnecessary variability.

The four studies that were completed were:

- ***Study 1***: Muscle activation patterns and lower limb kinematics and kinetics during functional tasks.
- ***Study 2***: Muscle activation patterns during seated knee extension
- ***Study3***: Passive knee joint moments
- ***Study 4***: In vivo knee muscle moment arms

While each of the studies conducted had a unique methodology, all shared common participants and all required the collection of 3D kinematic data and/or EMG data. These common factors will be described below to provide the reader with a broad overview of the methods used. Details specific to individual studies are provided in the chapters that follow.

2.2 Participant characteristics:

Six individuals, an average of 32 months post TKA (range 19 – 72 months), were examined and their results compared to six age matched controls with no history of lower

limb arthritis or serious lower extremity injury (see subject demographics Table 1). Strict inclusion criterions were used to select members of the TKA group. A primary consideration was time post-surgery. As research (Farquhar et al. 2008;) has shown that changes in both knee joint kinematics and kinematics are evident up to at least 1-year following the surgery, only individuals who had undergone knee replacement at least 1-year prior to testing were included. Additionally, as both surgical technique and prosthesis design have the potential to affect knee kinematics all members of the TKA group had had knee replacement surgery performed by the same orthopaedic surgeon using the same type of prosthetic implant (a mobile bearing design, the DePuy low contact stress (LCS) prosthesis (DePuy Orthopaedics, Warsaw, IN)). In all cases members of the TKA group had replacement surgery secondary to severe knee osteoarthritis. All patients were deemed appropriate for the study (i.e. non-complicated, unilateral TKA) by their treating surgeon and if they did not have arthritis in other lower limb joints. The decision regarding the presence or absence of arthritis in other joints was made based on patients reports of pain in either ipsilateral or contralateral joints. No attempts were made to objectively quantify the integrity of other joints (i.e. through x-ray). Given the strict inclusion criteria for members of the TKA, it was difficult to recruit large numbers of individuals who met these criteria. This resulted in the relatively small number of participants examined. Details of individual participant characteristics are presented in Appendix A1. Prior to participation all subjects signed a consent form approved by the University of Waterloo's ethics review board and completed a Par-Q questionnaire designed to assess their fitness to participate. They also completed a WOMAC questionnaire, deigned to assess lower limb function.

Table 2.1: Subject demographics; mean values and (standard deviations) are reported for both patients and controls.

Group	***Gender***	***Age (years)***	***Mass (kg)***	***Height (m)***	***Time post-op (months)***
Patient	4 females 2 males	70.67 (10.5)	90 (17.8)	1.67 (0.15)	32 (20.12)
Control	3 females 3 males	69.7 (4.4)	75.9 (16.75)	1.72 (0.08)	NA

2.3 Collection of 3D kinematic data:

Three-dimensional ankle, knee and hip joint kinematics were recorded from all participants. For members of the TKA group, kinematic data were collected from the limb on which joint replacement surgery had been performed. The limb tested in control participants was chosen such that equal numbers of right and left limbs were contained in both groups.

Kinematic data were collected using an Optotrak (NDI, Waterloo, ON) active marker system. A 4-segment (foot, shank, thigh, and pelvis) lower limb model was used. A cluster of four non-collinear infrared light emitting diodes (IREDs) was affixed to the shank, thigh and pelvis using a rigid plate that was securely fastened at mid-segment level. For the foot, only three markers were used. These were placed directly on the footwear of participants in a non-collinear fashion (approximately on posterior heel, dorsum of foot and on lateral aspect of foot). An additional marker was placed on the heel of the non-tested limb to aid in identification of limb contact times during gait and stepping-up. Kinematic data were collected at a rate of 60Hz and were low pass filtered at a rate of 6Hz, using a dual pass, Butterworth filter prior to any further data analysis.

In order to perform 3D kinematic and kinetic analysis, the position and orientation (pose) of all segments in the model was needed. To accomplish this using skin based markers, a multi-step process, based on the calibrated anatomical system technique (CAST) described by Cappozzo et al. (1995) was performed. Before describing the process, definitions of the various Cartesian coordinate reference frames required are reviewed below. These are also illustrated in Figure 2.1.

Global reference frame: This frame represented the Cartesian coordinate system of the laboratory space. It was determined during the camera registration process and was purposefully defined so that + X was in the direction of travel for all movements.

Marker (technical) reference frame: This frame was defined using the global coordinates of the segment markers. It was initially established for each segment using

data collected during a standing calibration trial. Unit vectors defining the X, Y and Z axes of the marker frame were determined and used to create an orientation matrix that represented the relationship between the marker frame and the global frame.

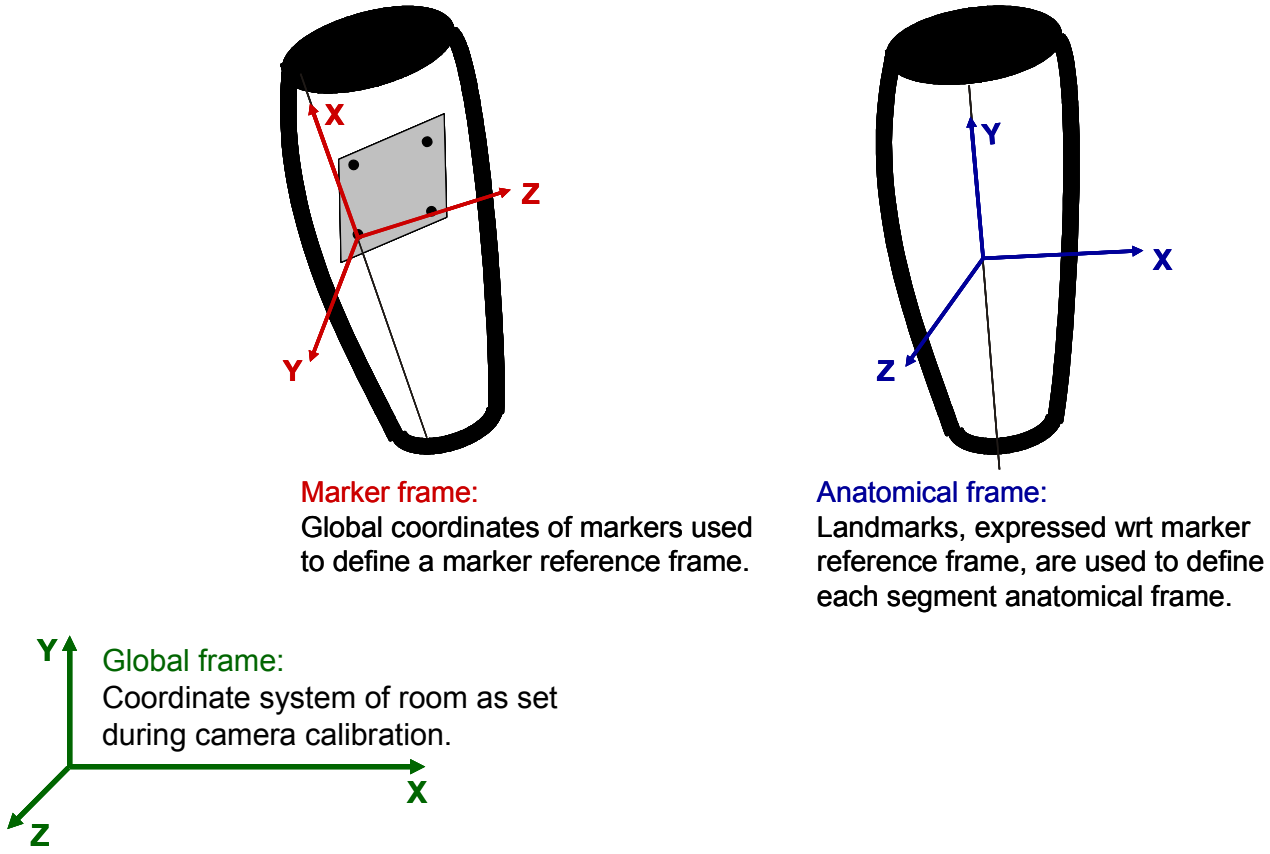


Figure 2.1: Illustration of marker, anatomical and global frames used in 3D kinematic analysis. The marker and anatomical frames were first defined using data from standing calibration trial. The global frame was defined as part of the process of camera registration. The orientation matrix relating marker and anatomical frame, determined as part of the calibration process was used in conjunction with a least squares minimization approach to determined the global coordinates of bony landmarks on a frame by frame basis during individuals trials. This relationship was then used to provide pose information for the anatomical frame, which in turn was used estimate 3D segment and joint kinematics.

Anatomical (bone-embedded) reference frame: Defining this frame was a two step process. First, the locations of anatomical landmarks were determined using a digitizing probe (NDI, Waterloo, ON) that consisted of a pointed tip and a body with 6 IREDs. When placed on the landmark of interest, 3D coordinates of the landmark with respect to

(wrt) the marker frame were determined. The specific landmarks digitized were based on Eng and Winter (1995) and are described in Table 2. Once landmark coordinates were determined, they were used to define the anatomical frame for each of the segments as defined by Eng and Winter (1995). The specifics of the anatomical frame definitions used for each segment are provided in Table 2. In general, the Z axis was defined as running from lateral to medial, the Y axis as vertical (usually running between adjacent joint centers) and the X axis as the cross product of the two. The resulting unit vectors expressed the relationship between the marker and the anatomical frame. This relationship was assumed to be constant.

2.3.1 Determining segment position and orientation:

In order to estimate the position and orientation (pose) of a bony segment at discrete time intervals, the global coordinates of the anatomical landmarks used to define the segments anatomical frame need to be determined. Using an approach similar to Cappozzo et al. (1995), this was done using a two-step process.

Define marker and anatomical reference frames:

An initial standing 'calibration' trial was collected with participants in anatomical position. This data was used to define the orientation matrix of the marker reference frame wrt global. A digitizing probe (Optorak, Northern Digital, Waterloo, ON) consisting of 6 non-collinear markers and a pointed tip was then used to determine the location of the various anatomical landmarks required to define the foot, shank, thigh and pelvis anatomic reference frames (see Table 2). Anatomic landmarks were defined wrt the marker reference frame of the appropriate segment. The transformation matrix (2.1) that resulted ($\mathbf{R}_{m2a}$) was recorded and kept for later use.

$$\mathbf{R}_{m2a} = \begin{bmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{bmatrix} \quad \textbf{(2.1)}$$

where $\mathbf{R}_{m2a}$ defined the orientation of the marker frame relative to the global frame ($a_{i,1}$, $a_{i,2}$, $a_{i,3}$ were the unit vectors of the anatomical frame expressed wrt to the marker frame; $a_{1,i}$, $a_{2,i}$, $a_{3,i}$ were the unit vectors of the marker frame expressed wrt the anatomical frame).

Determine global coordinates of anatomical landmarks during trial:
During movement trial individual marker coordinate data was recorded using the Optotrak system. This global marker data was then used to construct an orientation matrix, $\mathbf{R}_{m2g}$, that represented the orientation of the marker reference frame expressed with respect to global. This matrix was needed for future calculations.

As the marker data collected using skin-based markers is affected by noise (see Appendix A2 for review of noise sources), the global marker coordinates collected in this manner did not accurately represent global marker position. In order to reduce the effects of this noise on joint kinematics and kinetics an optimization approach based on Challis (1995) was employed. This approach, which employed the principals of singular value decomposition (SVD), is described in more detail in Appendix A3. Briefly, instead of using the global maker coordinates to $\mathbf{R}_{m2g}$ from equation was determined using a least squares method that was equivalent to minimizing:

$$\frac{1}{n}\sum_{i=1}^{n}\left(\left[R_{m2g}\right]p_l + o_g - p_g\right)^T\left(\left[R_{m2g}\right]p_l + o_g - p_g\right) \qquad \textbf{(2.2)}$$

where n was the number of non-collinear markers per segment; p_l were the 3D coordinates of individual markers expressed with respect to the local marker reference frame and determined during the standing calibration trial; p_g were the marker coordinates, expressed with respect to the global frame (as captured by Optorak); o_g was the position vector locating the origin of the marker frame with respect to global frame (unknown), to be determined in conjunction with $\mathbf{R}_{m2g}$.

$$\mathbf{R}_{m2g} = \begin{bmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{bmatrix} \quad \textbf{(2.3)}$$

where $\mathbf{R}_{m2g}$ defined the orientation of the marker frame relative to the global frame ($a_{i,1}$, $a_{i,2}$, $a_{i,3}$ were the unit vectors of the global frame expressed wrt to the marker frame; $a_{1,i}$, $a_{2,i}$, $a_{3,i}$ were the unit vectors of the marker frame expressed wrt the global frame).

By combining matrix 2.1 and 2.3, using standard matrix operations, the matrix $\mathbf{R}_{a2g}$ (2.3) was determined.

$$\mathbf{R}_{a2g} = \begin{bmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{bmatrix} \quad \textbf{(2.3)}$$

where $\mathbf{R}_{a2g}$ defined the orientation of the anatomical frame relative to the global frame ($a_{i,1}$, $a_{i,2}$, $a_{i,3}$ were the unit vectors of the anatomical frame expressed wrt to the global frame; $a_{1,i}$, $a_{2,i}$, $a_{3,i}$ were the unit vectors of the global frame expressed wrt the anatomical frame). The unit vectors of the anatomical landmarks with respect to the global reference frame could then be used determined orientation angles of individual segment and also the relative angles representing hip, knee and ankle joint angles (as per Winter 2005).The X,Y,Z Cardan angle system was used and all relative angles expressed proximal segment orientation in relation to distal (i.e. the knee joint angle represented thigh orientation relative to shank.

2.4 Collection of EMG data:

Muscle activity was recorded from all major muscles crossing knee of the tested limb: vastus medialis (VM), vastus lateralis (VL), rectus femoris (RF), medial hamstrings

Table 2.2: Summary of digitized landmarks and X, Y and Z axes that defined the anatomical reference frame of the foot, shank, thigh and pelvis.

	Landmark #1	Landmark #2	Landmark #3	X Axis	Y Axis	Z Axis
Pelvis	Right ASIS*	Left ASIS	Right iliac Left iliac	Vector normal to plane of the iliac crests and pelvis CoM	(0,1,0)	**X** x **Y****
Thigh	Greater trochanter	Medial epicondyle	Lateral epicondyle	**Y** x **Z**	Knee joint→ Hip joint***	Medial → Lateral epicondyle
Shank	Tibial tubercle	Medial malleolus	Lateral malleolus	**Y** x **Z**	Ankle joint → knee joint	Medial → Lateral epicondyle
Foot	Tubercle of navicular	Middle toe	Posterior calcaneous	Middle toe → posterior heel	(0,1,0)	**X** x **Y**

* ASIS: anterior surperior iliac spine
** x indicates vector cross product. **Bold** capitalized letters indicate vectors
*** → indicates a vector traveling from A → B

(MH), biceps femoris (BF) and medial and lateral gastrocnemius (MG and LG respectively). The electromyography (EMG) signal was collected using two silver – silver chloride electrodes (MediTrace 130, Mansfield, MA) placed as per Winter (1991).

A reference electrode was also placed over a non-active site. The site of electrode placement was prepped by shaving and debridement with alcohol to further enhance signal quality. The resultant signal was collected using a Bortec (Bortec Medical, Calgary, AB) system that differentially amplified the signal (input impedance 10 GOhms; MRR 115bB (at 60Hz)) before collecting at a rate of 1200Hz using the Optorak system (NDI, Waterloo, ON).

Prior to data collection, all participants performed maximum voluntary isometric contractions of quadriceps (resisted seated knee extension), hamstrings (resisted seated knee flexion) and gastrocnemius (resisted toe raises in standing). Participants were given an opportunity to practice this contraction prior to collection of these trials and trials were participants did not appear to be giving maximal efforts were repeated following an adequate rest period. Results from the maximum contraction trials were used to normalize all EMG signals based on the work of Burden et al. (2003). Briefly, for each raw maximum voluntary contraction (MVC) trial, root mean square amplitude (RMS) was calculated over consecutive periods of 50ms for each muscle over the duration of each MVC trial. Individual muscle RMS results were then examined and the maximum 50ms RMS was chosen as the normalization constant for that particular muscle. This value was kept and during data analysis was used to normalize all EMG signals collected during gait and stepping-up trials.

2.4.1 Analysis of EMG data:

The analysis of EMG consisted of calculating RMS magnitude (using raw, amplitude normalized EMG), muscle onset and offset and the magnitude of muscle co-contraction. As calculations for the former two variables were dependent on the specific task being examined, details of these calculations will be provided in later chapters.

Co-contraction, or the amount of simultaneous activation of agonist/antagonist muscle pairs, was determined in a similar manner irrespective of the study. We were specifically interested in the amount of co-contraction occurring in 4 muscle groups (BF and VL, MH and VM, LG and VL, MG and VM). This was quantified using the equation (2.4) below, adapted from Rudolph et al.(2000).

$$CCI = \frac{\sum_{i=1}^{n} \frac{lowerEMG_i}{higherEMG_i}\left(lowerEMG_i + higherEMG_i\right)}{n} \qquad (2.4)$$

$LowerEMG_i$ is the i^{th} frame of LE EMG of the less active of the 2 muscles and $higherEMG_i$, the LE EMG of the more active muscle. The resulting CCI measures identified as BFVL, MHVM, LGVL, MGVM, were calculated for specific time periods depending on the activity being examined. Variable *n* represented the total number of frames in the interval of interest. Division by the total number of frames in each period resulted in a single value that characterized the average co-contraction throughout the time frame examined. As outlined by Rudolph et al. (2000) this method of determining co-contraction will result in high values when both muscles in the pair were activated at high levels over the time period of interest. Low amounts of co-contraction were observed when either both muscles have relatively low activation or one muscle was highly activated while the other produces lower levels of activity.

Chapter 3

A kinematic, kinetic and EMG analysis of gait and stepping-up in individuals who have undergone knee joint replacement.

3.1 Introduction

As outlined in Chapter 1, the primary research question being proposed for this thesis is "Do biomechanical factors such as muscle activation, moment arm length and passive joint moment contribute to the deficits in knee extensor moment observed following total knee arthroplasty (TKA)?" The rational behind this research is based on the reasoning that without improved understanding of the factors contributing to knee extensor moment deficits, interventions aimed at improving knee function will potentially be done ineffectively and inefficiently. The importance of this investigation is further highlighted by the fact that the frequency of TKA is on the rise (Kurtz et al. 2007) and that the surgery is being performed in younger and younger individuals (Kurtz et al. 2007; Jain et al. 2005; Crowninshield et al. 2006) who place increased mechanical demands on the prosthesis (Andriacchi 1993; Crowninshield et al. 2006). If these growing numbers of more active individuals are to remain as active as possible following TKA then research aimed at improved understanding of the numerous factors that influence knee function following TKA is needed.

The first study undertaken this thesis was a full kinematic, kinetic and electromyographical (EMG) analysis of level walking and a step-up task (starting from a standing position and stepping on to a step). For both these tasks the knee joint must contribute to the overall support moment (Winter 1980) of the lower limb. In other words, the knee contributes to the net lower limb extensor moment needed to prevent lower limb collapse (1980). The tasks differ, however, with respect to their specific muscular demands. During gait, the knee extensor musculature must act both eccentrically (to control knee flexion during weight and late stance) and concentrically (to assist with knee extension during midstance) (Robertson and Winter 1980). While unassisted gait is certainly possible in the face of significant knee impairment (Siegel et al. 2006), the resulting pattern of walking is less then optimal. When stepping-up there is little, if any, eccentric work performed at the knee (Byrne et al. 2002). Knee function is key to propulsion of the body upward, with knee moments being greater then those of

normal walking (Mandeville et al. 2007; McFadyen and Winter 1988). The challenging nature of stepping-up, particularly to individuals who have undergone TKA, was demonstrated by Draganich et al. (1996) and Byrne et al. (2002). Both studies reported that 50% of the post-arthroplasty patients tested could not step onto a raised surface when the limb leading onto the step was the one with the replaced joint. Byrne et al's (2002) work not only illustrated the challenge posed by stepping-up, it also highlighted a strategy used by individuals to compensate for deficits in knee extensor moments – those in the surgical group increased hip extensor work when stepping up. This was an apparent attempt by patients to ensure the overall lower limb extensor moment was maintained in the face of decreased knee extensor moments. Winter (1980) has reported similar results in his case study assessment of an individual following TKA.

The specific questions asked in this study were:

- *When walking and stepping-up, how do the knee extensor moments of members of the TKA group differ from those of control participants? (NOTE: these results were not needed to answer any of the specific questions posed in Chapter 1. However before examining the factors that contribute to knee extensor moment deficits in this clinical population these deficits must first be quantified)*

- *How do knee muscle activation amplitude, timing and co-contraction differ between members of the TKA group and control participants?*

Answers to the questions posed above served two important purposes in the overall thesis. Firstly, the results provided a detailed description of the knee extensor moment deficits present in the study participants. Although knee extensor moment deficits have been previously quantified during both these tasks (Byrne et al. 2002; Andriacchi et al. 1982; Simon et al. 1983), this thesis examined moment deficits in a select group of individuals. Previously published knee moment data, therefore could not be used - moments specific to the individuals tested were needed. Secondly, the muscle activation data collected while members of the TKA group walk and step to a height provided initial insight into muscle activation patterns in the individuals being studied. This EMG data

enabled us to begin to examine the relationship between alterations in muscle activation and knee extensor moment deficits.

3.2 Methods

Six individuals, an average of 32 months post TKA (range 19 – 72 months), were examined and their results compared to six age matched controls with no history of lower limb arthritis or serious lower extremity injury (see subject demographics Table 2.1). Further details on subject characteristics and selection criterion can be found in Chapter 2.

Ten gait and 20 step-up trials were performed by each participant. For the gait trials, participants walked along an 8m walkway in which an AMTI (Advanced Mechanical Technology Inc, Watertown MA) force plate was embedded. Starting position and limb were manipulated to ensure the desired limb hit the force plate. Gait trials were performed consecutively and continued until 10 trials with appropriate force plate strikes were collected. Following completion of gait trials, an additional AMTI force plate (Advanced Mechanical Technology Inc, Watertown MA) was added. This plate, which was affixed to a marble base, was placed on the floor in front of the embedded plate, so that a 17.2cm step was created. Participants were instructed to stand with both feet on the embedded plate and to step onto the upper plate. Step-up trials ended when the trailing limb hit the upper plate so no analysis of the step-down motion was performed. Twenty step-up trials were completed: 10 in which participants led with their test limb (for patients the limb containing the replaced knee; for controls either the right or left limb so that the number of right and left limbs in both groups was equal) and 10 where the non-test limb (i.e. the other lower limb) led. Trials in which the test and non-test limb lead during the step-up were randomized. As the focus of the current research was on lead limb knee function, trials where the non-test limb lead were not analyzed for the current work.

3.2.1 Data collection:

During all gait and step-up trials kinematic, kinetic and EMG data were collected. Details of the kinematic and EMG collections were outlined in Chapter 2. Briefly, EMG was recorded from medial and lateral gastrocnemi, hamstring and vasti muscles as well as from rectus femoris. All EMG data were collected at a rate of 1200Hz and stored in raw format for post-collection processing. Three dimensional kinematics of the lower limb were captured during both gait and step-up trials using an active marker system (Optotrak, Northern Digital, Waterloo, ON). A total of 3 Optotrak cameras recorded the 3D position data of a series of markers placed on the foot, shank, thigh and pelvis. Four markers each were mounted on a rigid plate and securely fastened to the shank, thigh and pelvis. Three markers, mounted in a non-colinear fashion, on the subjects footwear, were used to track foot position. A multistep process, based on the calibrated anatomical system technique (CAST) described by Capozzo et al. (1995) was used to determine 3D segment and joint position and orientation based on marker location. Following completion of this process 3D kinematics of the foot, shank, thigh and pelvis were known as were the ankle, knee and hip joint angles.

Results of the kinematic analysis were used in conjunction with force plate data to determined 3D joint moments. Custom designed software (Mishac Inc., Waterloo, ON) was used for this purpose. The net internal ankle, knee and hip moments were determined using a standard 3D inverse dynamics analysis (Winter 2005). Anthropometric data were obtained from Winter (2005). All moments were expressed with respect to the reference frame of the distal segment (i.e. knee joint moments were reported with respect to the shank reference system) and represented the net moment exerted by muscles and passive tissues in resisting the moment created by any external force, gravity and segment inertia. Note that some labs report net external joint moments, as opposed to the internal reaction to this moment (for example Smith et al. 2004; Catani et al. 2003; Milner et al. 2008). This can create confusion when comparing results. **In order to avoid any potential misunderstanding all moments reported in this paper (those being presented as results and those being discussed from other authors) will be expressed as internal moments unless otherwise indicated.** Moments were normalized to subject mass.

In order to compare 3D ankle, knee and hip moment data between groups, joint angular impulse – the integral of the moment vs. time curve was determined. Integrals of the moment profiles were determined for the various phases (see sec 3.2.2 for definition of the phases used) of gait and stepping-up using the trapezoid rule.

3.2.2 Temporal windowing of data:

All data were windowed so that only data from specific time periods of interest were analyzed. For gait the windowing period was from tested limb heel contact ($LdHC_g$) to toe off ($LdTO_g$). Force plate data were used to identify both events. Heel contact was deemed to have occurred when vertical force exceed 15N, while toe off was identified as the instant when vertical force fell below 5N. For step-up trials, data were windowed from tested (i.e. lead) limb heel contact on the upper plate ($LdHC_{su}$) to non-tested (i.e. trail) limb heel contact ($TrHC_{su}$). $LdHC_{su}$ was determined using vertical force data (i.e. when force exceed 15N). $TrHC_{su}$ was identified by examining the vertical trajectory of the heel marker to determine when minimum displacement had occurred.

The stance phase of gait and stepping-up were further divided into smaller intervals to facilitate data analysis. The stance phase of gait was divided into a preparation phase ($Prep_g$:120 frames prior to $LdHC_g$ to $LdHC_g$), weight acceptance phase (WA_g: from $LdHC_g$ to the point of maximum knee flexion during stance), midstance phase (MS_g: from maximum knee flexion to maximum knee extension during stance) and late stance phase (LS_g: from frame where maximum knee extension to $LdTO_g$). Sagittal plane knee angles were examined on a trial by trial basis to determine when maximum knee flexion and extension occurred. Stepping-up was divided into double support (DS_{su}) and single support (SS_{su}) phases. Double support consisted of the period from $LdHC_{su}$ to $TrTO_{su}$. The period from $TrTO_{su}$ to $TrHC_{su}$ was classified as SS_{su} (Fig 3.1). $TrTO_{su}$ was deemed to have occurred when vertical force data from the floor embedded plate fell below 5N.

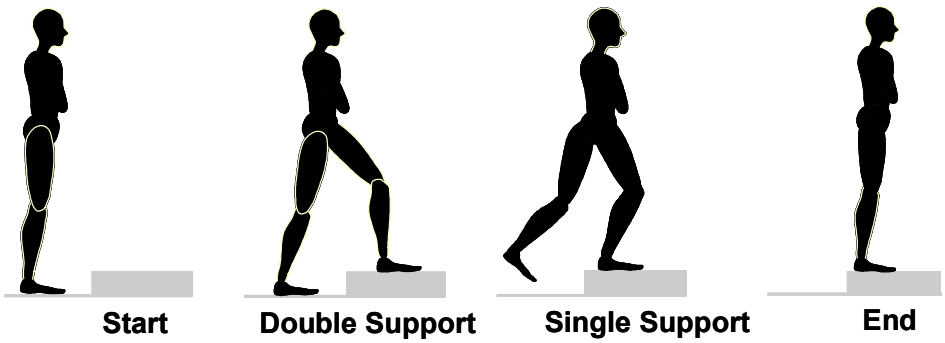


Figure 3.1: Double support and single support phases when stepping-up. Double support (DS) was from $LdHC_{su}$ to $TrTO_{su}$, single support (SS) from $TrTO_{su}$ to $TrHC_{su}$.

3.2.3 EMG analysis:

The magnitude of EMG activity and the amount of co-contraction between the quadriceps and either the hamstrings or gasctrocnemi were determined using methods described in Chapter 2. Briefly EMG RMS of amplitude normalized EMG was determined during the prep, DS and SS phases of stepping-up and for prep, WA, MS and LS phases of gait. Co-contraction indices (CCI) were also determined during these same phases for BF and VL, MH and VM, LG and VL and MG and VM.

Identification of muscle onset/offset frames was done visually. Specifically, the full-wave rectified EMG data were dual-pass filtered using a critically damped low pass filter at a cut-off frequency of 50Hz (as per Hodges and Bui 1996), This processed signal was the examined to determine muscle onset (the frame at which a prolonged burst of EMG activity was evident) and offset (the frame at which the observed burst of activation ceased). Data used for this visual analysis were not windowed as described above, rather EMG from the complete trial was examined. Trials were analyzed in a random manner and the examiner was unaware of the exact frames of interest (i.e. heel contact and toe off times) for the trial. Random trials were picked for re-analysis on 2 separate days to ensure the on / off times selected were reliable. For gait trials, muscle onset and offset times were determined using the above method, as all muscles had clear and well defined on and off times. For the step-up trials, muscle offset did not occur until after the trial had been completed. As a result only muscle onset times were quantified for step-up trials.

To allow for visual display of EMG the raw, full-wave rectified signal was low pass filtered with a dual pass, critically damped filter at a frequency of 6Hz creating a linear enveloped (LE) signal. Linear enveloped data for each trial were then time normalized to 100 frames. The traditional 60/40 time normalization was used for gait trials (60% stance, 40% swing). For step-up trials, an examination of DS and SS times showed that on average participants spent 40% of their time in DS and 60% in single support. As a result, normalization of step-up trial EMG was done using a 40/60 split. Once time normalized, EMG data were averaged within patients for like trial types. This ensemble averaged data were used for qualitative and visual analysis only. Three dimensional moment profiles for ankle, knee and hip were also time-normalized and ensemble averaged in a similar manner. Once again, these ensemble averaged moment were used only for display purposes.

3.2.4 Statistical analysis

A two-way repeated measures ANOVA was employed to statistically examine data from this study. ANOVA factors used were group (patient and control) and trial, where trial was the repeated factor. The trial effect was not of interest, however, it was included to reduce the model error sums of squares, thereby improving the accuracy of predictions for between group differences. Co-contraction index, RMS, muscle onset / offset times, knee angular impulse and knee position were analyzed in this manner. An alpha level of $p=0.05$ was used for all analyses. A similar analysis was also used to assess differences between medial and lateral CCI measures (ie: LGVL vs MGVM and BFVL vs MHVM) within patient and controls participants. For this analysis group and side (medial and lateral) were the factors, with side being the repeated measure.

3.3 Results

Patients and controls differed with respect to the time required to complete both the gait and step-up tasks. During gait, patients spent 0.82s (±0.05s) in stance compared to 0.73s (±0.07) spent by controls. This difference reached statistical significance ($F_{1,9.03} = 5.28$, $p=0.04$). Swing time did not differ significantly between the groups (patients 0.44s

(±0.04); controls 0.44s (±0.07)). When stepping to a height, patients spent 0.42s (±0.01) DS and 0.59s (±0.01) in test limb SS. This differed from the 0.31s (±0.01) and 0.54s (±0.01) spent by controls in these same periods. Only the DS time intervals differed significantly (main effect of group: $F_{1,9.84}$ =15.78, p =0.003)

3.3.1 Kinematics and kinetics:

Kinematic and kinetic analysis focused on sagittal plane measures only (see Appendix A4 for frontal plane results).

During both gait and stepping-up, members of the TKA group used knee, hip and ankle joint kinematics that were similar to control subjects (Table 3.1 and 3.2). The only exception to this was observed during the WA phase of gait, where patients ended this phase with significantly less knee flexion than controls. Patients exhibited only 16º of knee flexion in comparison to the 22º of flexion observed in controls ($F_{1,9.01}$ =5.49,p=0.04).

Sagittal plane knee moment profiles during gait and stepping-up are presented in Figure 3.2. Note that the gait moments are from 5 patients – there were technical problems with vertical force readings for the sixth subject meaning moment data could not be used. Therefore, all kinetic gait data presented below were based on only these 5 subjects. Kinematic and EMG data were based on averages of all 6 patients.

Table 3.1: Summary of sagittal plane knee angles at various times during stance phase of gait. See text for description of HC,WA, MS and TO.

	Patient	Control
Lead HC	7.9º (±5.3)	7.3º (±6.4)
End of WA	16.2º (±4.5º)*	22.8º (±3.3º)
End of MS	14.1º (±4.7º)	10.6º (±3.8º)
Trail HC	23.7º (±4.0º)	23.2º (±7.0º)
Lead TO	54.6º (±5.9º)	53.3º (±10.0º)

*indicates significant difference between patients and controls (See text for details)

Table 3.2: Summary of sagittal plane test limb knee angles at various times during the step-up task.

	Patient	Control
Lead TO	79.6º (±9.5)	53.0º (±9.5)
Lead HC	53.6º (±9.3)	60.5º (±17.3)
Trail TO	49.7º (±10.9)	55.1º (±4.2)
Trail HC	14.4º (±7.0)	11.9º (±8.9)

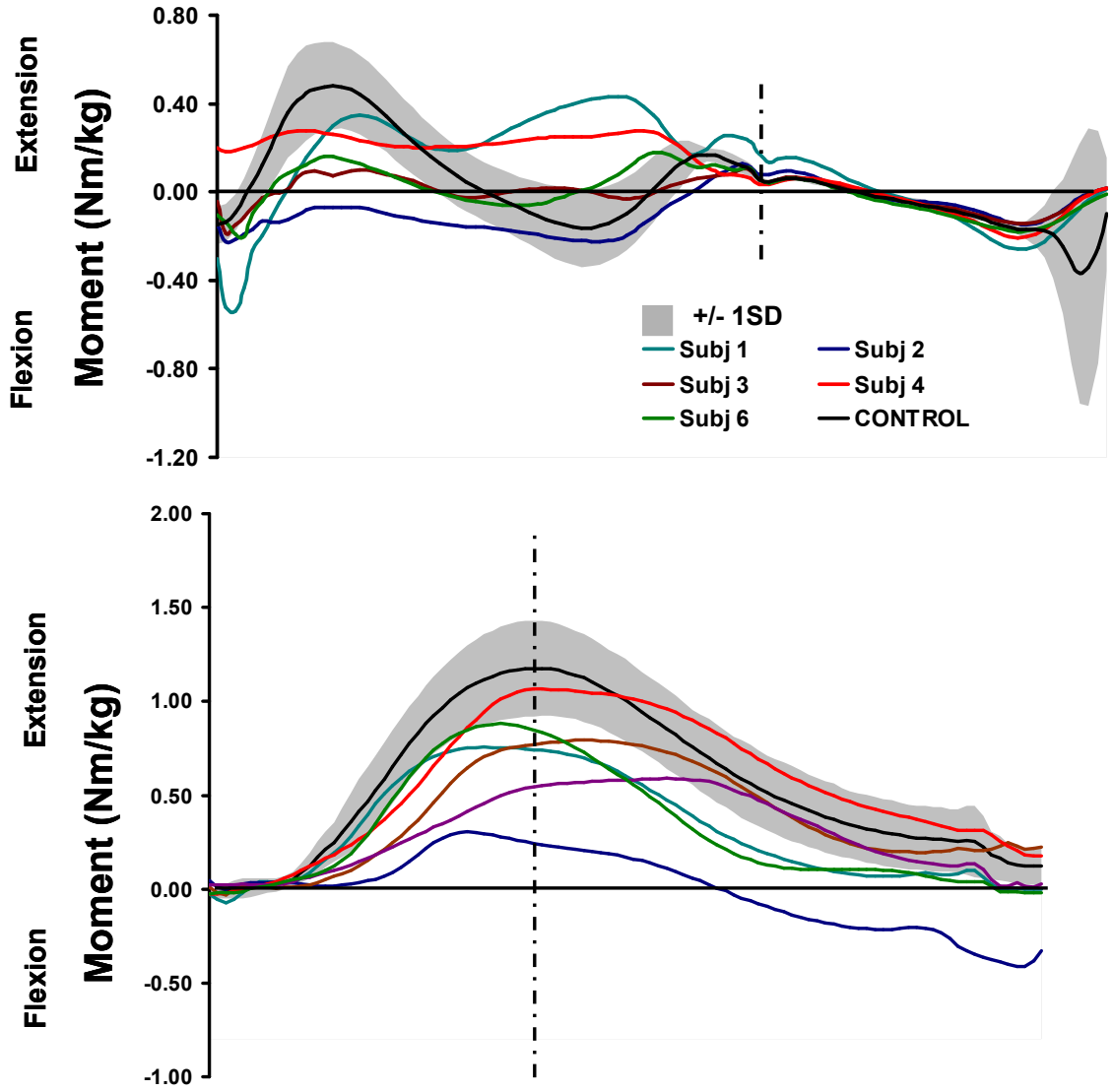


Figure 3.2: Sagittal plane knee moments during gait (upper panel) and step-up (lower panel) for members of the TKA group plotted against mean and ±1 SD of control average moment. Vertical lines: upper panel – toe off; lower panel: separates double and single support.

Gait and step-up angular impulse results (see Fig 3.3) showed that sagittal plane impulse measures were significantly greater in controls than patients during the MS phase of gait. During this phase, patients produced 0.01Nm/kg/s (±0.02Nm/kg/s) of extensor impulse at the knee compared to 0.04 Nm/kg/s (±0.03Nm/kg/s) produced by controls ($F_{1,9.2}$ =5.06, p=0.04). Angular impulse results at the ankle and hip did not differ significantly between the groups (see Fig. 3.4).

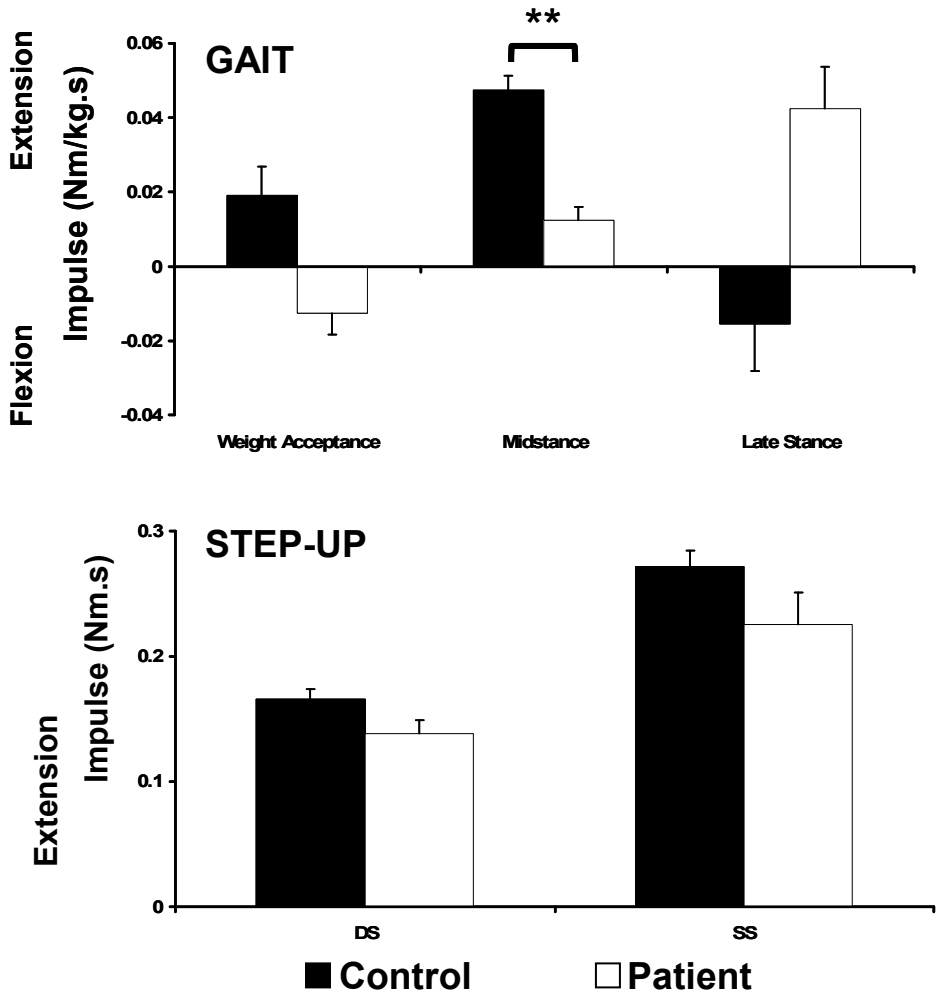


Figure 3.3: Sagittal plane angular impulse at the knee during gait (upper panel) and step-up (lower panel). Error bars represent standard error. ** indicate significant difference between patients and controls. DS = double support, Ss = single support

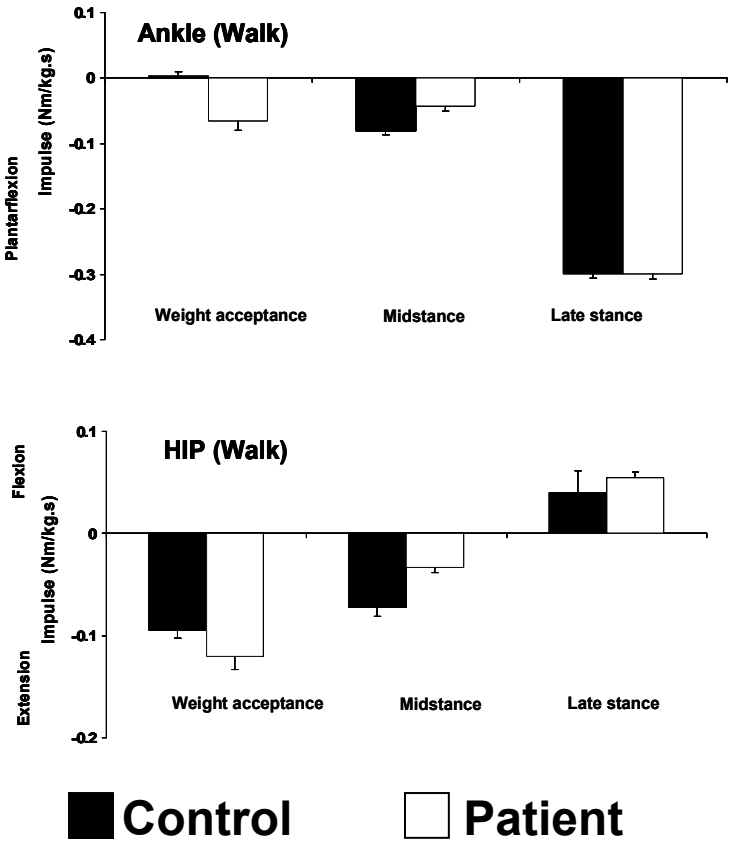
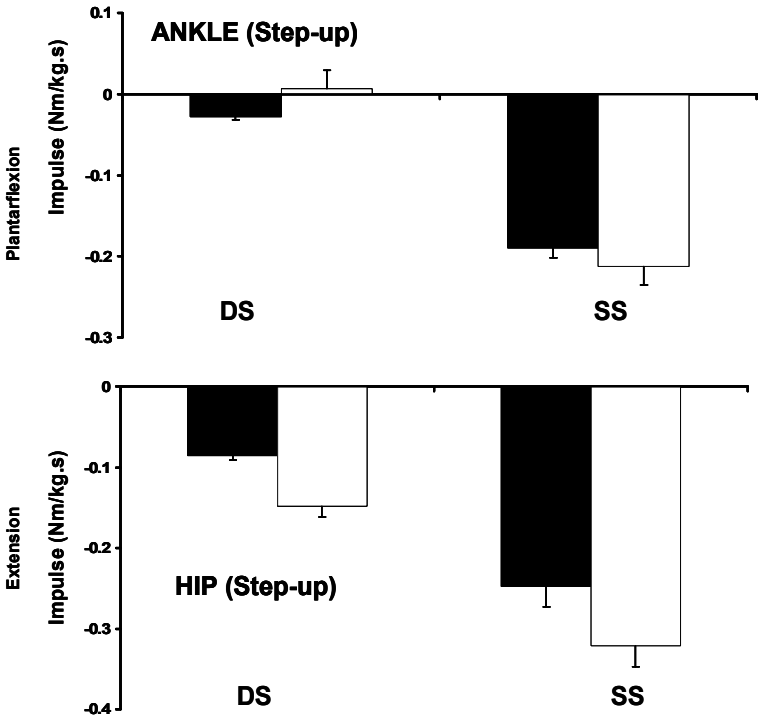


Figure 3.4: Ankle and hip sagittal plane angular impulse during walking (upper plots) and stepping-up (lower plots). Error bars represent standard error of the mean.



3.3.2 Quadriceps EMG:

The greater demands experienced by the quadriceps when individuals were stepping-up were demonstrated in the ensemble averaged VL, VM and RF EMG. (Fig. 3.5 a,b and c). While some patients maintained control like EMG profiles during both tasks, magnitude and timing differences were evident. For the step-up tasks, subjects 1, 4 and 6 consistently produced greater VL and VM activation than controls. Due to the variability of these magnitude values, no significant differences were found in quadriceps EMG RMS for the quadriceps during either gait or stepping up (Fig. 3.6 and 3.7).

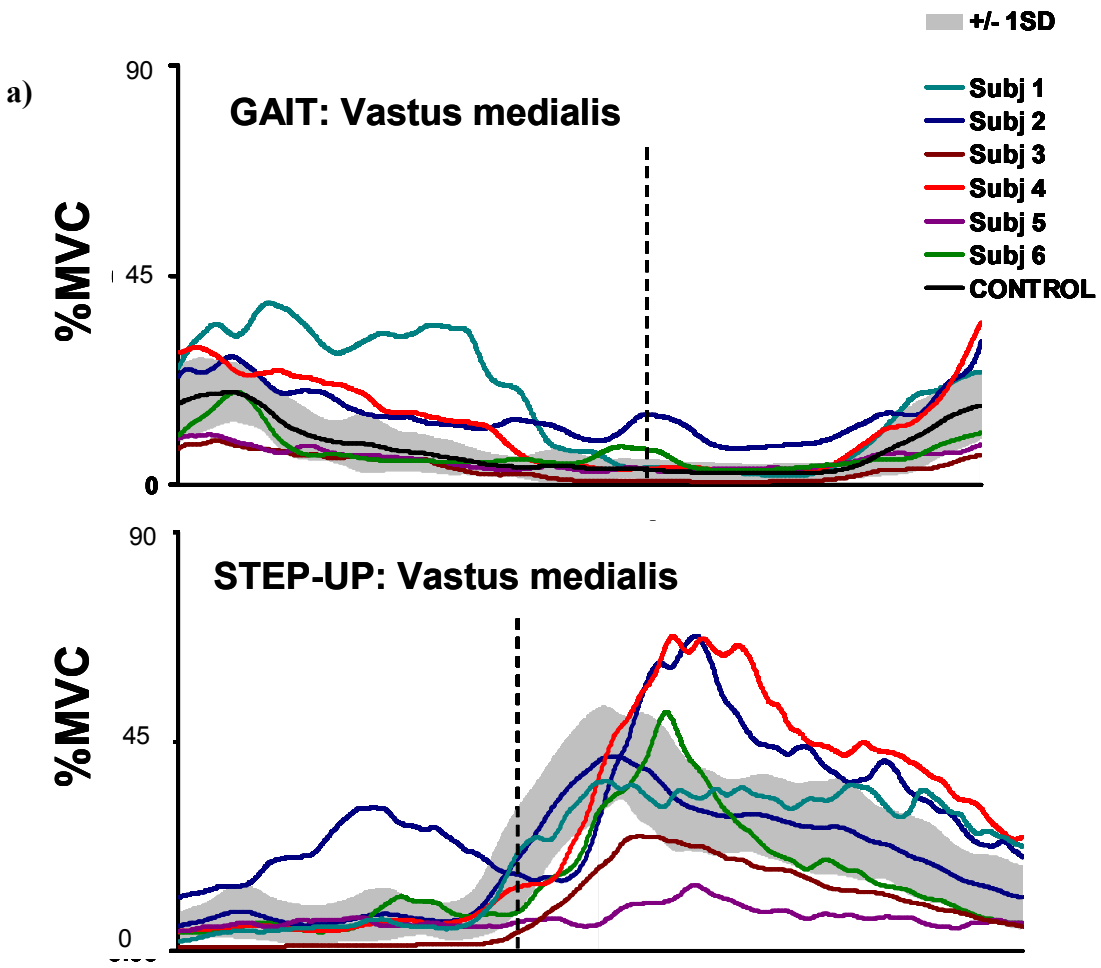
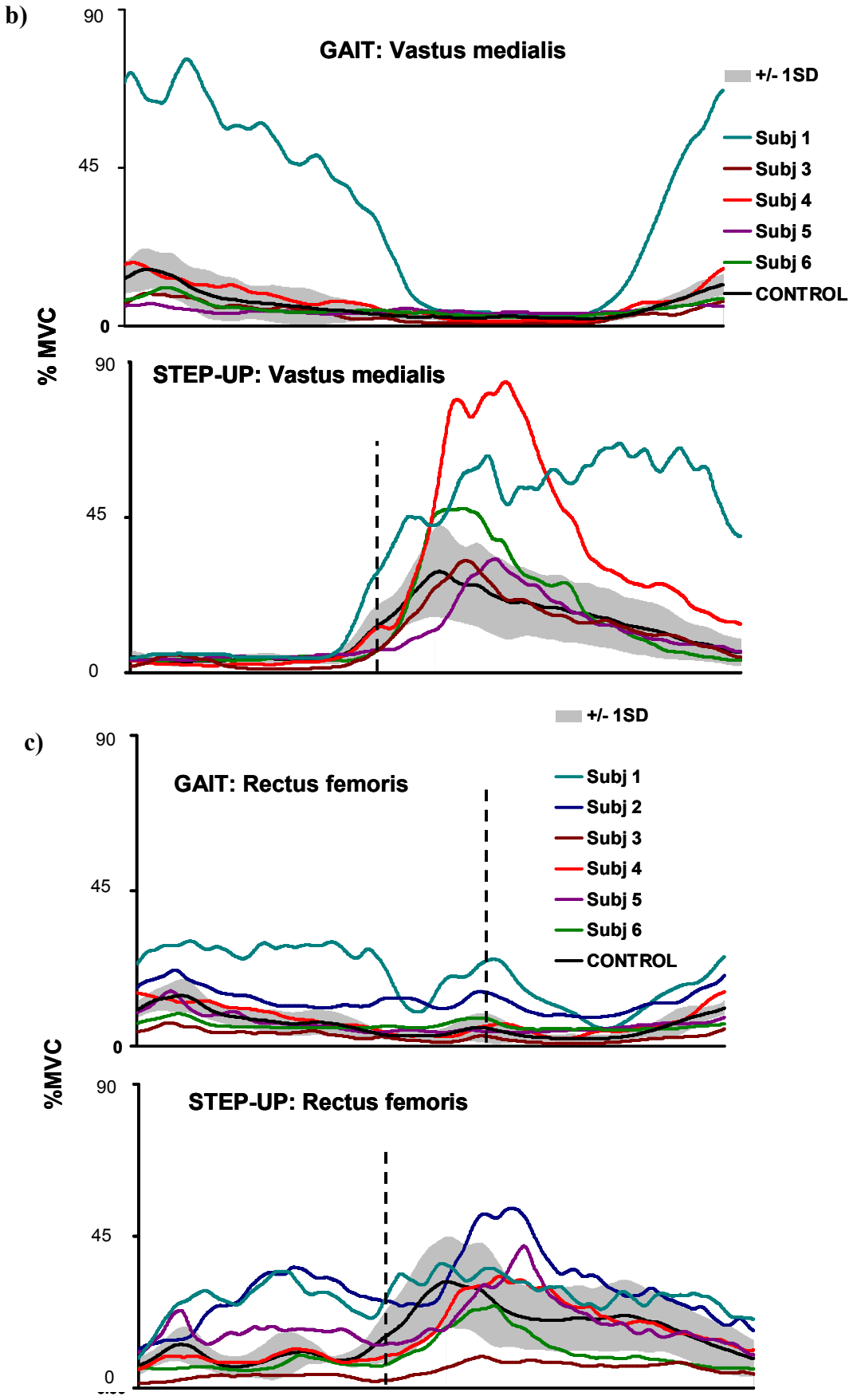


Figure 3.5: Ensemble averaged a)VL, b)VM and c) RF EMG during gait and step-up (See next page for b and c). Shaded areas: ±1SD of control EMG. Individual patient profiles averaged across subject gait and step-up trials. Subject 2 data for VM not included due EMG noise. Vertical axis is amplitude normalized EMG, horizontal axis is %stance. Vertical line: gait trials = toe off, step-up trials = start of single support.


b)
90
45
0
% MVC
GAIT: Vastus medialis
+/- 1SD
Subj 1
Subj 3
Subj 4
Subj 5
Subj 6
CONTROL
STEP-UP: Vastus medialis
c)
+/- 1SD
GAIT: Rectus femoris
Subj 1
Subj 2
Subj 3
Subj 4
Subj 5
Subj 6
CONTROL
%MVC
STEP-UP: Rectus femoris

3.3.3 Hamstring EMG

Hamstring activation during the stance phase of both gait and stepping-up was considerably lower than in the quadriceps (Fig 3.8). Although there were 2 patients who activated BF using a pattern that differed from controls, in general patient and controls patterns were similar. The primary magnitude differences were observed in BF during gait and step-up, where patients produced more EMG than controls near the end of the stance. EMG magnitude differences were only statistically significant for BF during the LS phase of gait (Fig 3.6 and 3.7) (main effect of group: $F_{1,9.16}$ =8.09,p=0.02).

3.3.4 Gastrocnemius EMG:

During both gait and stepping-up, patients' gastrocnemi RMS magnitudes were generally greater than controls (Fig 3.6 and 3.7). These differences were significant during the WA phase of gait, where lateral gastrocnemius RMS was significantly greater in patient' (main effect of group: $F_{1,9.3}$ =6.84, p = 0.03). Medial and lateral gastrocnemius RMS values were approximately twice as large in patients as in controls during the double support phase when stepping up [MG: $F_{1,10}$ =6.5, p = 0.03; LG: $F_{1,10}$=6.87, p=0.03].

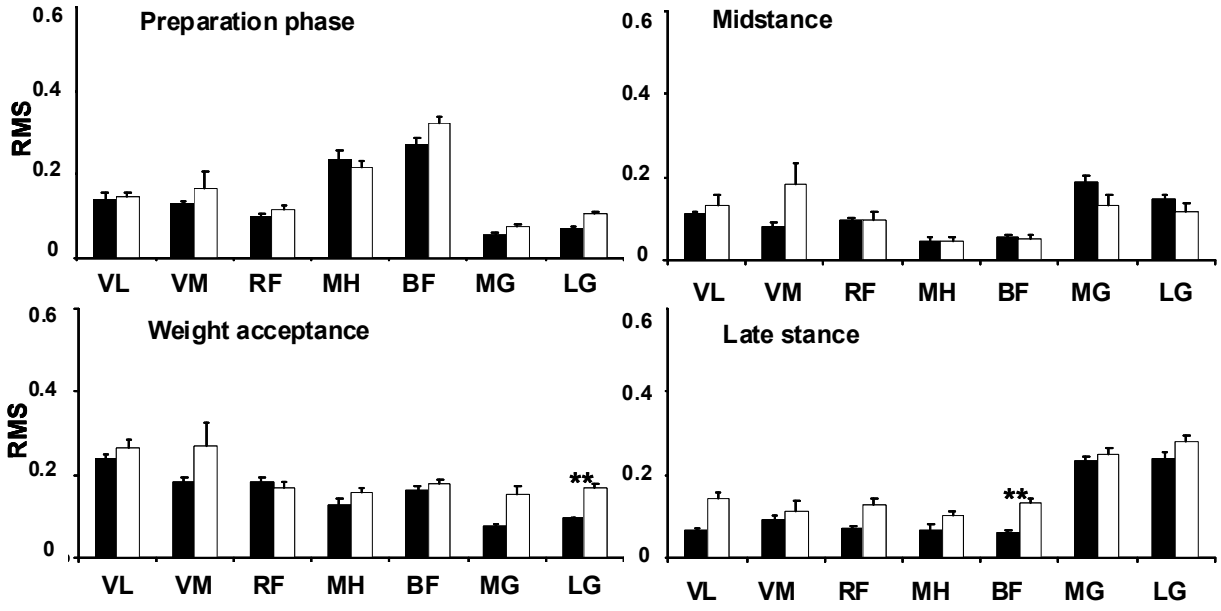


Figure 3.6: RMS values for lower limb EMG during the gait phases indicated (see text for stance phase definitions). Error bars represent standard error. ** indicates significant differences between patients and controls.

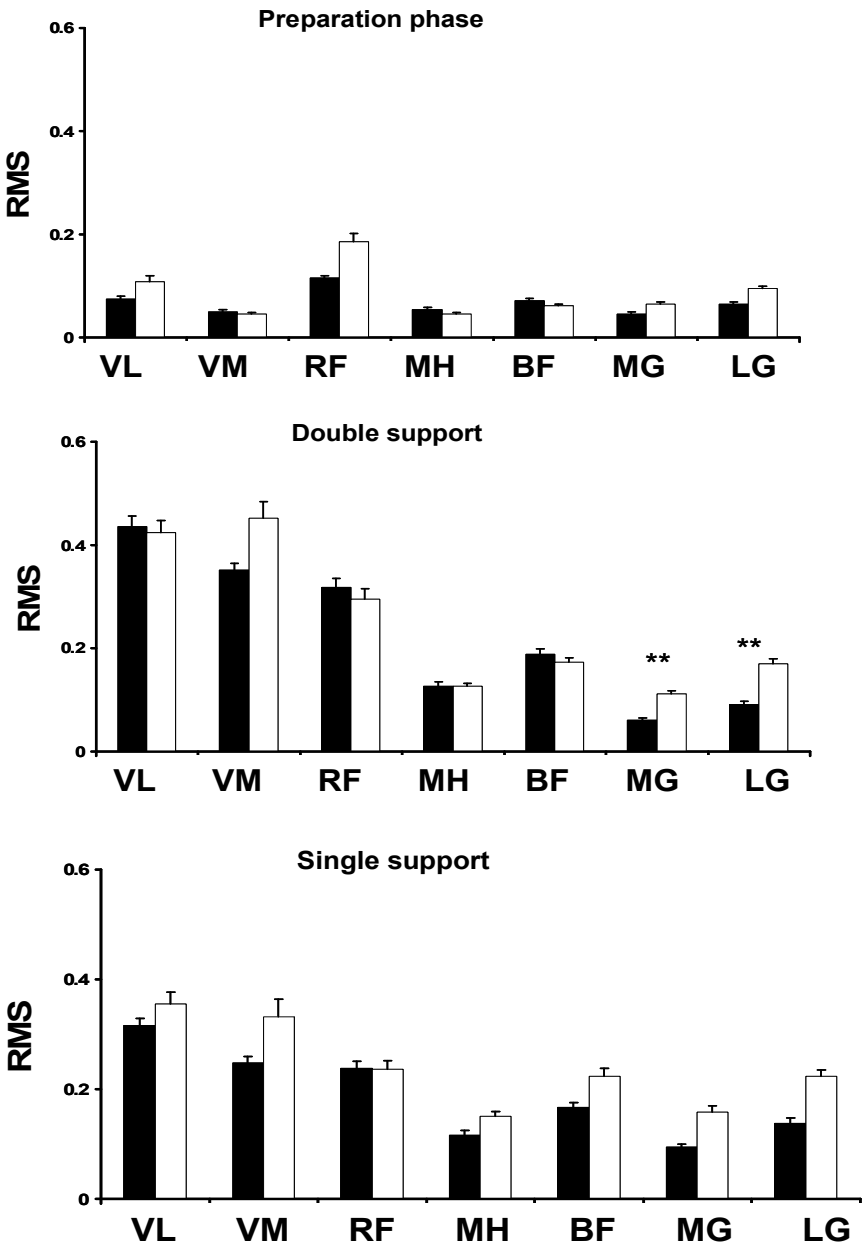


Figure 3.7: RMS values for lower limb EMG during 3 phase of step-up task (see text for stance phase definitions). Error bars represent standard error. ** indicates significant differences between patients and controls.

3.3.5 Temporal patterns of muscle activation:

The primary differences in muscle timing occurred while participants completed the gait trials. During these trials, controls activated MH and BF for 19% (±1.5%) and 26% (±2%) of total stance time respectively. In contrast, MH and BF were activated for 50% (±4%) and 52% (±4%) of stance respectively by patients (see Fig. 3.10). Both differences were statistically significant (main effect of group: MF $F_{1,9.13}$ =6.55, p = 0.03; BF $F_{1,9.16}$=5.89, p = 0.04). Patients also activated rectus femoris significantly earlier than controls, with muscle onset taking place 0.19ms prior to HC in patients and 0.04ms later (0.15ms before heel contact) in controls [main effect of group $F_{1,10.4}$ =7.72, p = 0.02]. No difference between patients and controls existed for either percent of stance activated or on-time with respect to heel contact for any other muscle groups. Muscle timing characteristics did not differ significantly between patients and controls during the step-up task. Summary of timing results was depicted in Table 4.

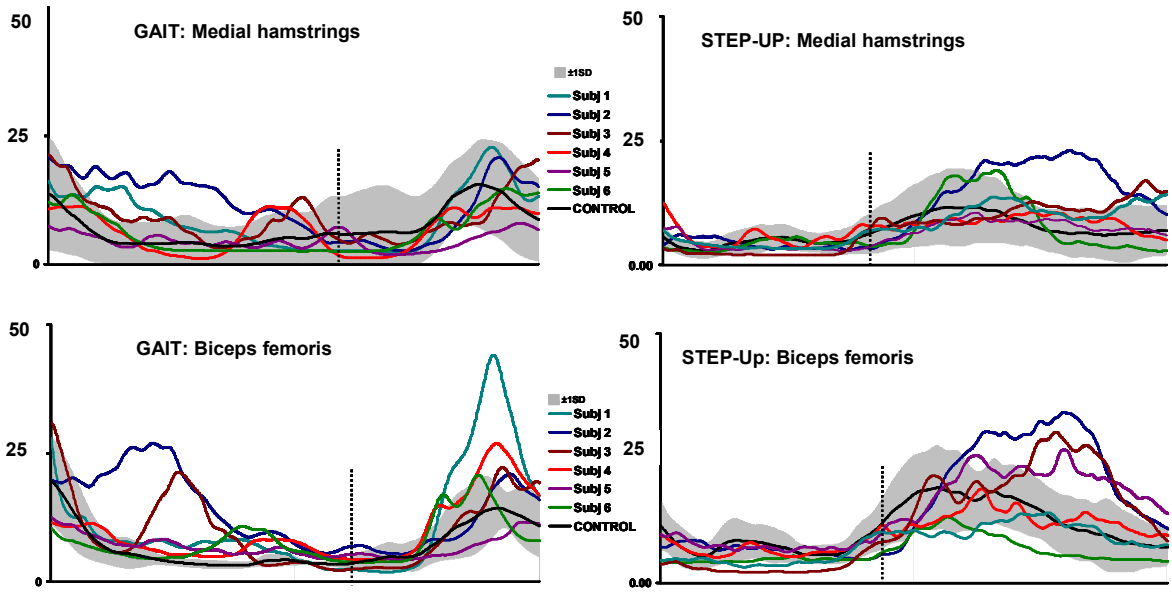


Figure 3.8: Ensemble averaged MH and BF EMG during gait (left column) and step-up (right column). Shaded areas represent ±1SD of control EMG profiles. Individual patient profiles are averaged across subject gait and step-up trials. Vertical axis is amplitude normalized EMG, horizontal axis 100% of gait stride or stance phase of step-up. Vertical line: gait trials = toe off, step-up trials = start of single support.

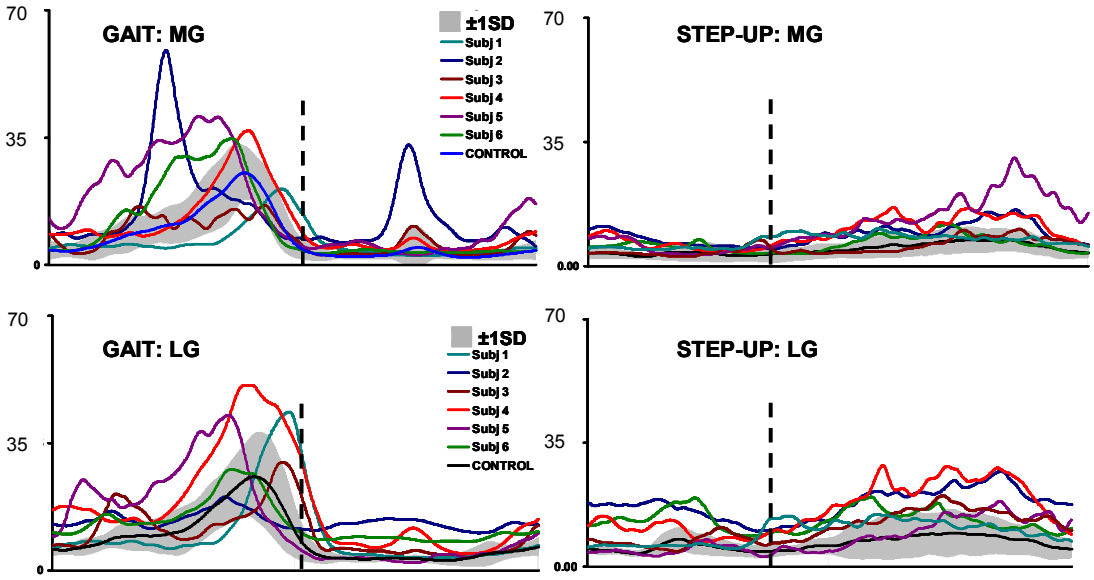


Figure 3.9: Ensemble averaged MG and LG EMG during gait and step-up. Shaded areas represent ±1SD of control EMG profiles. Individual patient profiles are averaged across subject gait and step-up trials. Vertical axis is amplitude normalized EMG, horizontal axis represents either 100% of step-up stance or 100% of gait stride. Vertical line: gait trials = toe off, step-up trials = start of single support.

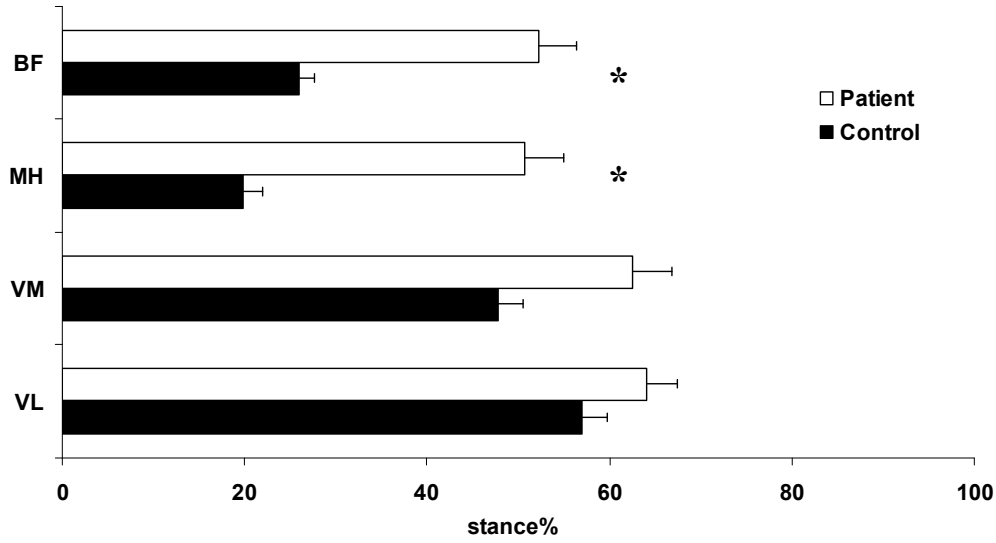


Figure 3.10: Horizontal bars: percentage of time each muscle was active during the stance phase of gait. Horizontal axis represents percentage of stance, with 0% indicating heel contact and 100% indicating toe off. Note muscle onset occurred prior to heel contact for all muscles. The figure only displays the portion of muscle activation time that occurred during stance. Errors bars indicate standard error. * indicates a significant difference between patients and controls.

3.3.6 Muscle co-contraction:

Co-contraction levels were generally greater during stepping-up then during walking (Fig. 3.11 and 3.12). All individuals studied displayed greater co-contraction of lateral musculature then medial, with a significant main effect of side ($P<0.0001$ for all analyses) when comparing BFVL to MHVM and LGVL to MGVM, irrespective of the activity. Results of the 2-way repeated measures ANOVA comparing patient and control CCI values found no differences between patients and control CCIs during gait. When stepping up, patients co-activated LG and VL more than controls during DS (significant main effect of group $F_{1,10.4}=14.4, p=0.03$). The larger MGMH CCI for patients during DS was not statistically significant, although a strong trend existed ($p=0.05$).

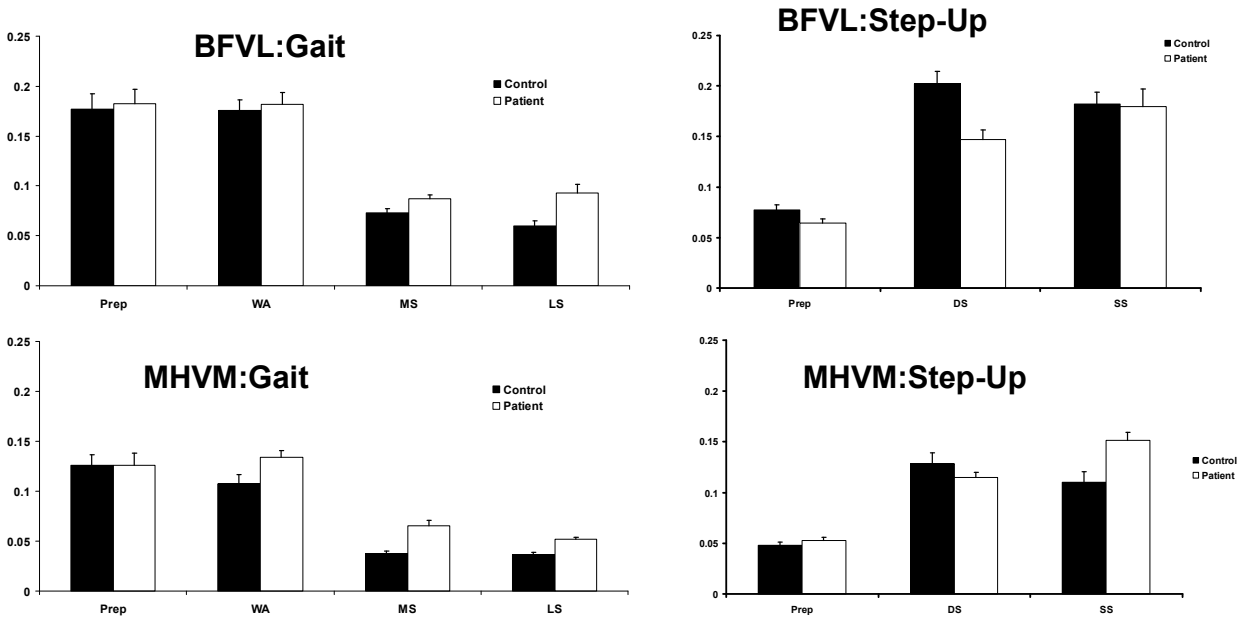


Figure 3.11: Co-contraction indices for medial and lateral hamstrings during gait and stepping-up. Error bars represent standard error. For gait trials: Prep = preparation phase (see text for definition); WA = weight acceptance; MS=midstance; LS = late stance. For step-up trials: Prep = preparation phase (see text for definition), DS = double support, SS = single support.

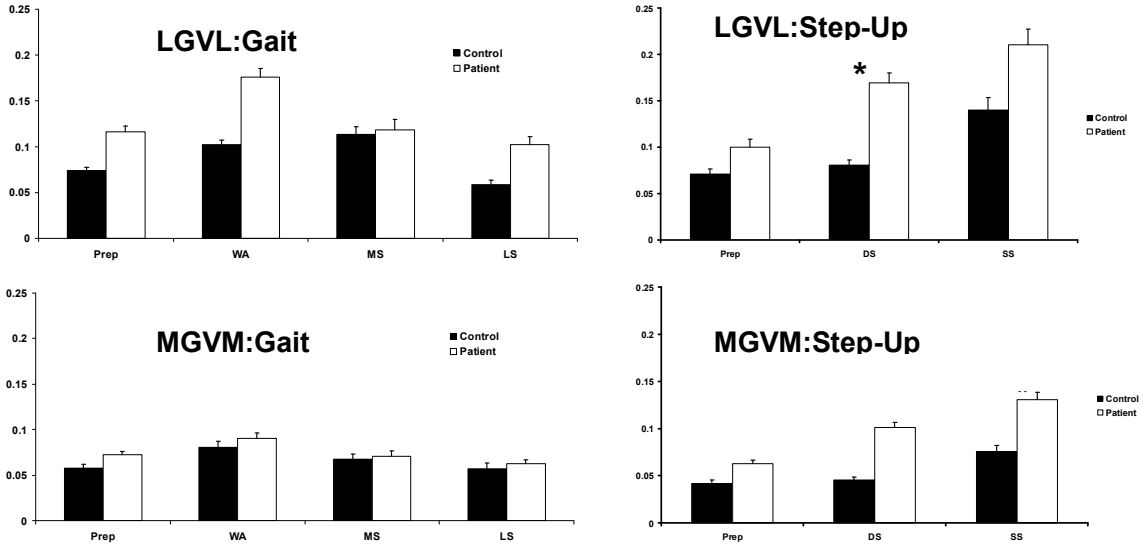


Figure 3.12: Co-contraction indices for medial and lateral gastrocnemi during gait and stepping-up. * indicate patient CCI values significantly greater than controls as indicated. Error bars represent standard error. See figure 11 caption of description of phases abbreviated on the horizontal axes of graphs.

3.4 Discussion

The two tasks investigated in the current study were chosen because both greatly challenge the knee joint and are critical to activities of daily living. When walking, the knee must simultaneously assist with energy absorption while contributing to lower limb support (Winter 1980). While performing a step-up motion, knee joint contributions to whole body support and propulsion upward are vital to ensuring successful and safe completion of the task. The purpose of this kinematic, kinetic and EMG analyses was two-fold: firstly it was intended to provide a detailed description of sagittal plane knee moments; secondly results from the EMG analysis would provide initial insight into the muscle activation patterns employed by study participants.

3.4.1 Sagittal plane kinematics and kinetics:

Results of the sagittal plane moment analysis agree with those of previous authors. Namely, reductions in stance phase knee extension moment were observed during gait and stepping-up. These qualitatively observed differences were quantified by determining

Table 4: Summary of muscle onset / offset data for gait and stepping-up. Values represent mean (±1SD) for both TKA and control groups. Negative values for on times indicate that muscle was activated prior to heel contact, positive values indicate activation after heel contact.

		% On time during stance	On time wrt HC (gait)**	On time wrt HC (SU)**
VL	**Patient**	**64 (±22.1)**	**-0.18(±0.04)**	**0.027 (±0.03)**
	Control	**57 _±17.5)**	**-0.16(±0.03)**	**-0.012 (±0.01)**
VM	**Patient**	**63 (±26.2)**	**-0.17(±0.03)**	**-0.03 (±0.01)**
	Control	**48 (±17.2)**	**-0.15(±0.05)**	**-0.022 (±0.01)**
RF	**Patient**	**64 (±21.8)**	**-0.19*(±0.04)**	**0.44 (±0.04)**
	Control	**60 (±14.0)**	**-0.15(±0.03)**	**0.26 (±0.03)**
MH	**Patient**	**50* (±27.0)**	**-0.28(±0.1)**	**-0.006 (±0.07)**
	Control	**19 (±9.8)**	**-0.31(±0.14)**	**0.08 (±0.04)**
BF	**Patient**	**52* (±26.0)**	**-0.25(±0.07)**	**-0.04 (±0.01)**
	Control	**26 (±13)**	**-0.26(±0.11)**	**0.077 (±0.04)**
MG	**Patient**	**87 (±8.2)**	**0.09(±0.18)**	**-0.20 (±0.04)**
	Control	**86 (±4.5)**	**0.12(±0.11)**	**-0.03 ±(0.08)**
LG	**Patient**	**91 (±6.1)**	**0.02(±0.17)**	**-0.12 (±-0.04)**
	Control	**88 (±5.1)**	**0.03(±0.12)**	**-0.11 (±0.05)**

****negative values mean muscle onset occurred prior to heel contact (HC)**
***** indicates significant main effect of group ($p<0.05$). See text for details.

knee angular impulse. Statistical comparison of these results revealed that members of the TKA group produced significantly less knee extensor impulse during the MS phase of gait. Although no statistically significant, differences in sagittal plane angular impulse were observed during stepping-up, members of the TKA did exhibit trends in this regard.

Direct interpretation of the kinetic differences has to be done in conjunction with an examination of the kinematics used by the groups. Typically patients who have undergone TKA have been reported to walk with less knee flexion during the stance phase of gait, irrespective of how long they are post-op. Results from the current study agree with these previous findings - patients exhibited both decreased knee flexion at the

end of the WA phase of gait and reduced knee extension excursion during MS. This finding is not unique to individuals who have undergone TKA, as a similar kinematic pattern has been found in individuals with knee osteoarthritis (OA) (Hubley-Kozey et al. 2006; Astephen et al. 2008a and 2008b; Childs et al 2004; Fisher et al 1997), and those who have suffered a torn anterior cruciate ligament (ACL) (Rudolph et al. 2000 and 2001).

This reduced knee flexion during WA has often been referred to as 'quadriceps avoidance gait' (Andriacchi 1993). The theory behind this idea of quadriceps avoidance is that individuals walk with less knee flexion in an effort to reduce the demand on the quadriceps muscle, therefore enabling them to walk with a reduced quadriceps activation (and thus reduced knee joint load). While theoretically this idea is sound, the near normal average quadriceps activation by members of the TKA group (see Fig 3.7) suggested that individuals were activating their quadriceps in a manner similar to controls. In other words they did not appear to be avoiding quadriceps activation by walking with a less flexed knee.

Examination of individual participant results, however, suggested a somewhat different story. Not all members of the TKA group demonstrated the same degree of angular impulse reduction during MS. Angular impulse data is presented in Figure 3.13 for four members of the TKA group – those individuals at the extremes. Two of these individuals (subjects 3 and 4) had markedly reduced knee extensor angular impulse, while the other two exhibited more mild reductions in angular impulse. Examination of the EMG data for these same four participants (see Fig 3.14) revealed that the two with dramatically reduced impulse measures (i.e. subject 3 and 4) also exhibited minimal quadriceps activation during MS. It was concluded that for these two patients, quadriceps avoidance may help explain the reduced knee moments they exhibited. The remaining subjects, however, exhibited reduced knee extensor moments but elevated or normal quadriceps EMG – clearly they were not adopting a quadriceps avoidance strategy.

Given the small number of participants examined in the current study it is not possible to establish whether the patterns identified above (i.e. a portion of individuals using quadriceps avoidance and a portion not) would apply to a more general population. Further research, with expanded subject numbers, is needed to confirm this finding. The results do, however, indicate that the reduced knee extensor moment exhibited during gait by members of the TKA group cannot be blamed entirely on an avoidance of quadriceps activation.

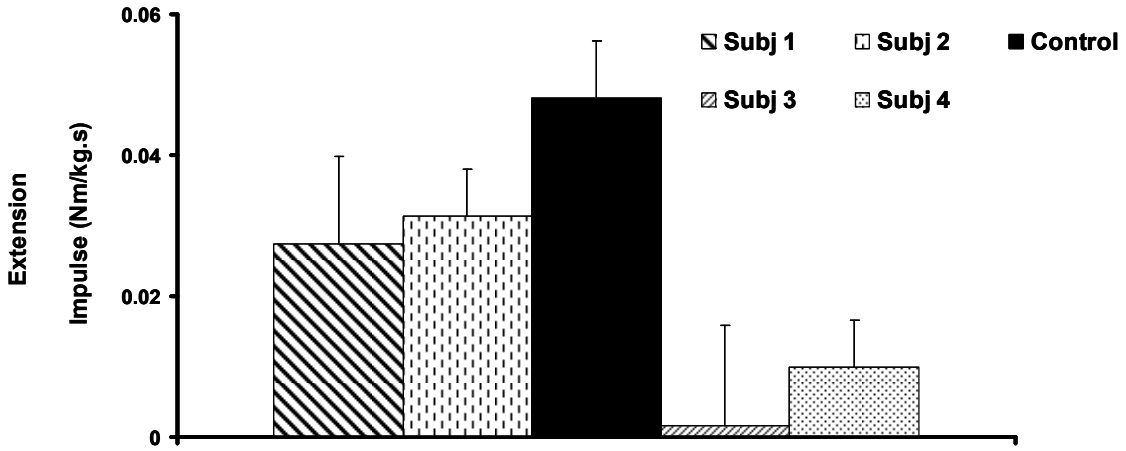


Figure 3.13: Individual patient angular impulse data during the MS phase of gait. Error bars represent standard error of mean. Time spent in MS was comparable for controls and subject 1 and 2. Subjects 3 and 4 spent less time than controls in MS. See text for additional details.

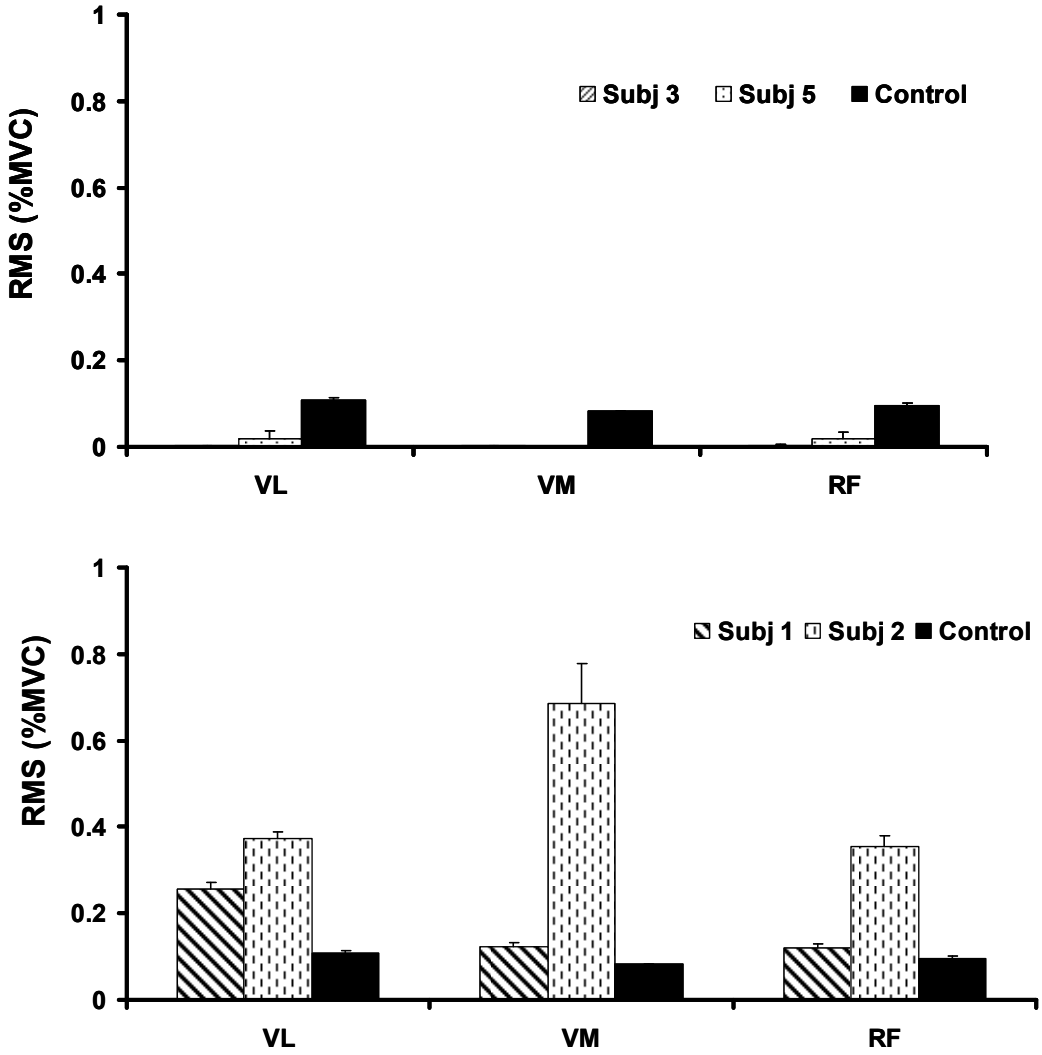


Figure 3.14: RMS EMG for individual members of the TKA group during the MS phase of gait. Upper panel contains results for subject 3 and 4, two patients who exhibited markedly reduced knee extensor impulse during MA. The lower panel reports results from two patients whose knee angular impulse results differed less dramatically from those of controls. Subject numbers reported in this image correspond to those in figure 3.13. Error bars represent standard error of the mean.

3.4.2 Muscle activation: amplitude

Although quadriceps avoidance may have contributed to the reduced knee extensor moment in some members of the TKA group, it was clearly not to blame for reduced knee extensor moments in all members of the TKA group. Were there other alterations in muscle activation that could help explain the knee moment reductions observed in the

patients group? An examination of the EMG profiles during the two weight-bearing tasks (see Figs 3.5, 3.8 and 3.9) illustrated that overall patterns of EMG used by patients and controls were similar. Despite these similarities, changes in EMG magnitude were observed in medial and lateral gastrocnemi and magnitude and timing differences were seen in the hamstrings. Specifically lateral gastrocnemi magnitude was increased during the WA phase of gait; medial and lateral gastrocnemi activation was elevated during the DS phase when stepping-up and BF activation was higher in members of the TKA group during the LS phase of gait and patients exhibited prolonged activation of both medial and lateral hamstrings during stance. As both the gastrocnemi and BF are knee flexors it was tempting to conclude that increased activation of these muscle groups lead directly to reduced knee extensor moments. More in depth examination of these results, however, showed that this was generally not the case.

3.4.3 Reduced knee extensor moments during the MS phase of gait:

The only significant reduction in knee extensor angular impulse observed occurred during the MS phase of gait. During this phase members of the TKA group exhibited knee extensor angular impulse values that were approximately 40% less than those of controls. As discussed above, for two members of the TKA group this reduction was likely due to reduced activation of the quadriceps. For the others quadriceps activation was either normal or elevated. This would seem to suggest that the reduced knee extensor moment could not have been due to alterations in quadriceps muscle activation. This assessment would appear correct if similar EMG levels are equated with similar force (and therefore moment) magnitudes. However, members of the TKA employed knee joint kinematics that differed from those of controls. As muscle length has an effect on a muscles force production, alterations in knee joint position would presumably alter the length of both the quadriceps and hamstrings, therefore, having a potential differential effect in the two groups. Members of the TKA group were less flexed at the beginning of WA then controls. Weight bearing on a straighter knee would have the effect of shortening the quadriceps and potentially lengthening the hamstrings. This would mean that in members of the TKA group, the quadriceps would produce less force for a given

amount of EMG while hamstrings force production would conceivably be greater. The net affect of this length related change in force production would be decreased knee extensor force and increased knee flexor force. This combination would result in exactly the scenario observed in members of the TKA group – a decreased knee extensor moment. It is not possible, with the data available, to confirm the validity of this hypothesis at this time. Further research, employing musculoskeletal models of the knee joint to predict individual muscle forces, needs to be done in order verify these ideas.

3.4.4 Gastrocnemi activation and knee extensor moments during gait:

At HC, members of both groups exhibited similar amounts of knee flexion. During WA , however, patients flexed their knee less, resulting in the significantly reduced knee flexion angle at the beginning of MS. This reduced knee extensor moment observed in members of the TKA group during MS was hypothesized to be related to changes in muscle mechanics resulting due to the altered knee position. Given that gastrocnemi activation was increased during WA, we were interested in examining if this increased gastrocnmei activation could have contributed to the altered knee kinematics seen in patients.

Although the gastrocnemi are capable of producing frontal plane moments (Lloyd and Buchanan 2001, Shelburne et al. 2006) the primary action of these muscles is in the sagittal plane (Lloyd and Buchanan 2001; Shelburne et al. 2006). Gastrocnemi function during gait has received considerable research attention of late (Neptune et al. 2001, McGwan et al. 2007; Stewart et al. 2008; Schmitz et al. 2008; Neptune et al. 2008; Liu et al. 2008). Results of forward simulation and induced acceleration analyses done by Neptune et al. (2001) during normal gait have reported that the gastrocnemi, normally considered knee flexors, were capable of inducing knee extension acceleration during WA (see Fig. 8 Neptune et al. 2001). As, by definition, the knee flexes throughout WA, this extension acceleration would act to limit the amount of flexion that occurred. Although there are obvious limitations related to forward simulation analyses (Zajac et al 2002 and 2003), application of Neptune's results to findings from the current study would

indicate that the decreased knee flexion observed in members of the TKA group (see Table 3) may have resulted due to increased LG activation. Given the link we have hypothesized between straight leg gait and the reduction in knee extensor moments exhibited by members of the TKA group, an additional hypothesis we propose is that alterations in activation of the gastrocnemi have the effect of reducing knee flexion during the WA phase of gait which subsequently leads to reduced knee extensor moments during MS. This is the first study we are aware of to suggest a link between gastrocnemi activation during WA, reduced knee flexion during stance and decreased knee extensor moments. Replication of these findings with a larger sample size could have considerable implications for rehabilitation of the TKA population, as traditionally little attention has been paid to the gastrocnemi muscles in rehabilitation following TKA (Meiner et al. 2008).

3.4.5 Sagittal plane contributions of the gastrocnemi: stepping-up

Could reasoning similar to that discussed above be applied to explain the effects of increased gastrocnemi activation during DS when stepping up? During this portion of stepping-up, members of the TKA group exhibited increased activation of both medial and lateral gastrocnemi. As the knee was extending during this portion of the step-up, the extensor moment generated by the gastrocnemi in patients could have assisted with knee extension. Robertson et al. (2008) used just this argument to explain increased gastrocnemi activation during the ascent phase of a full squat. This argument was contradicted, however, by simulation studies (Neptune et al. 2001) and experimental results using functional electrical stimulation (Stewart et al. 2007). Both Neptune et al. (2001) and Stewart et al. (2007) have shown knee joint acceleration arising due to gastrocnemi activation switched from extensor to flexor when the knee was extending (i.e. during the MS portion of gait). Collectively this suggests that gastrocnmi function when stepping-up, where the knee was continually extending, would act to produce a knee flexion moment. Thus increased gastrocnemi activation during this phase may have accounted for the decreased knee extensor moment observed in members of the TKA group. Although these change in knee moment did not reach statistical significance, as

Fig 3.5 illustrates, the two groups exhibited markedly different moment and impulse magnitudes during DS. The lack of statistical significance is more likely due to the low power of the study, as opposed to a reflection of a lack of difference.

3.4.6 Reasons for increased gastrocnemi activation in members of TKA group:

It appears, based on the above analyses, that increased gastrocnemi activation may have had a direct effect on knee extensor moment when stepping-up and through effects on knee kinematics, may have indirectly contributed to reduced knee extensor moments during the MS phase of gait. If this was indeed the case, the as yet unanswered question is why do patients have elevated levels of gastrocnemi activation. Previous research examining muscle activation in those with knee OA provided some interesting insight into this very question. Hubley-Kozey et al. (2008), observed increased gastrocnemi activation during gait in individuals with severe knee OA. These authors attributed the gastrocnemi EMG changes to an attempt to ensure a more generalized knee joint stability. Although direct comparison between the Hubley-Kozey work and the current study was not possible (they only examined gait, not stepping-up), the results of Hubley-Kozey suggest two possible explanations for the increased gastrocnmei activity in members of the TKA group. If Hubley-Kozey's hypothesis that gastrocnemi activation is increased to compensate for decreased knee stability, then perhaps members of the TKA group were experiencing knee instability. In an effort to ensure that the resulting knee instability did not lead to lower limb collapse (i.e. the support moment was maintained) members of the TKA group may have increased gastrocnemi activation. As minimal efforts have been made to quantify functional knee instability post-TKA and no attempts have been made to link instability to muscle performance, the validity of this conclusion cannot be judged at this time. Research investigating complaints of instability and the link between such complaints and muscle activation are needed in this population. Researchers in the knee OA field have recently begun to take the approach of assessing EMG changes based on knowledge of reports of knee instability in the individuals being studied (see Schmitt and Rudolph 2008 and 2007; Lewek et al. 2005). It is suggested that similar efforts be

adopted to better explain the interaction between changes in gastrconemi activation and joint instability following TKA.

A second possibility raised by the Hubley-Kozey findings is that the increased gastrocnemi activation observed in members of the TKA group in the current study is simply a carryover of a motor pattern learned prior to knee replacement. In other words, in the years prior to their surgery patients may have increased activation of their gastrocnemi in an effort to minimize pain and / or improve joint stability, two common sequela of knee OA. Several years of activating the gastrocnemi in this manner may have subsequently lead to this activation pattern become a learned pattern for them. Post-surgically, despite minimal pain and instability the previously learned motor pattern continued. In order to test which of these two is most likely the case, it is necessary to get patients to activate their knee musculature during a non-weight bearing task where knee stability is not challenged. This was done during the seated knee extension study (see Chapter 4). Further discussion of the role of habit vs. joint instability will therefore be carried our after the results of this study are outlined.

3.4.7 Differences in hamstring EMG:

In addition to these EMG differences in the gastrocnemi, members of the TKA group also exhibited increased hamstring EMG during the late stance phase of gait (See Fig. 3.6 – only BF difference was statistically significant). Additionally during stance, patients activated the hamstrings for approximately 50% of stance, while hamstring activation lasted for only 26% of stance in controls. As the period of activation observed in controls was comparable to previously published data (Ounpuu and Winter 1989; Winter 1991), these results suggest that members of the TKA prolonged activation of the hamstrings. It is likely that this prolonged activation was responsible for the increased LS hamstring activation observed in the members of the TKA group. Despite these differences in magnitude and contraction duration, no differences were noted in the knee extensor moment during this phase. As such, it was concluded that the observed difference in hamstring activation did not substantially impact the knee extensor moment.

Before proceeding, it should be noted that prolonged hamstring activation post-TKA has been observed previously in this population. Beneditti et al. (2003), reported prolonged hamstring activation in individuals 6, 12 and 24 months post-TKA. While they did not quantify this, data provided by the authors clearly indicated it occurred. These authors suggested a link between this and the stiff knee gait participants in their study exhibited. Rudolph et al. (2001) have also reported prolonged hamstring activation in a group of individuals who complained of periods of knee instability (referred to as non-copers) following anterior cruciate ligament (ACL) rupture. This same group of individuals also walked with less knee flexion during stance and decreased knee extension moments – just as patients in the current study and in Benedetti's work. What was interesting about the work of Rudolph et al. (2001) was that a comparable group of ACL deficient individuals, who were not experiencing symptoms of knee instability (copers), exhibited normal kinematic, kinetic and EMG profiles. Rudolph et al. (2001) suggested that the differential hamstring activation observed in the copers and non-copers was indicative of attempts by the non-copers to stabilize the knee joint – a claim that was similar to that of Bendetti et al. (2003). As well several authors have reported similar findings in the OA literature (Schmitt and Rudolph 2007; Childs et al. 2004; Astephen et al. 2008; Al-Zahrani and Bakheit 2002), making similar conclusions about the nature of the prolonged hamstring activation.

As patients in the present study adopted a similar strategy (prolonged hamstring activation, decreased knee flexion during WA and reduced knee extensor moments), it was hypothesized that the prolonged hamstring activation was an attempt to ensure knee stability was maintained throughout stance. As was the case during our discussion of increased gastrocnemi activation, collectively these results suggest that either instability is still an issue for this group or patients are simply continuing to use muscle activation patterns learned prior to surgery. Additional research, some of which will be done in study 2 of this thesis, will help determine which, if either of these two hypotheses is correct.

3.4.8 Rectus femoris EMG:

In addition to prolonged hamstring activation, patients also demonstrated earlier onset of RF during gait. A similar finding was evident during step-up, although it was not statistically significant. In healthy individuals, rectus femoris is typically observed to have two periods of activation during gait: a major burst that begins just prior to heel contact (thought to assist with knee extension in preparation for heel contact and to help support the knee during weight acceptance) and a second, smaller burst, just prior to toe off (Winter 1991; Shiavi 1985; Arsenault et al. 1986; Annasswamy et al. 1999). On the basis of this description, it could have been argued that early activation of RF by patients in the current study was an indication that patients were starting preparations for HC earlier then controls in order to ensure the knee was ready to accept weight at HC (Schmitt and Rudolph 2007). While this argument was plausible, recent work by Nene et al. (2001) and Byrne et al. (2005) called into question the validity of surface recorded RF EMG. Both authors compared surface and fine needle EMG recorded from RF to surface EMG from VL, Nene during gait and Byrne during isometric contractions. The collective results from both research groups suggested that the RF burst typically observed during late swing / early stance appears to result due to cross-talk from the vasti, with Nene et al. (2001) providing strong evidence to suggest that the only true RF activity during gait is the burst that occurs near TO. These conclusions call into question the validity of the findings of the current study in relation to early pre-stance activation of RF in patients. The early activation of RF observed in members of the TKA groups occurred during the late swing, early stance phase described by Nene et al. (2001) as resulting from cross-talk. Cross-correlation analysis (using Igor Pro) on a random selection of trials confirmed the possibility of cross-talk. As such conclusions regarding the behaviour of RF following TKA were not possible using the present data. Further research, possibly using fine wire EMG will need to be done before any definitive statement can be made about possible RF onset differences post knee arthroplasty.

3.4.9 Limitations:

Ideally, much larger subject numbers were desired for this research, however, difficulty finding participants who had undergone TKA with the same surgeon and prosthethic implant and were free of co-morbidities resulted in only a small number of eligible patients being recruited. Because of these small numbers, results cannot be applied to a more general population of individuals who have undergone TKA. Despite this lack of generalizability, it is felt that the issues raised and questions asked will act as the basis for future, larger studies that will serve to further elucidate factors underlying alteration in locomotor mechanics following TKA.

3.4.10 Conclusions:

The first aim of the current study was to quantify the differences in knee extensor moments that existed between members of the TKA group and controls. Secondly, it aimed to examine muscle activation strategies used by this population during weight bearing tasks, in an attempt to make links between moment deficits and EMG. While the results confirmed findings from previous studies (i.e. decreased stance phase knee extensor moments, reduced knee flexion during stance phase of gait and prolonged activation of the hamstrings) new information related to increased gastrocnemi EMG amplitude has been reported. This finding of altered gastrocnemius EMG represented a first for research in this population. It was hypothesized that this increased gastrocnemi EMG were either directly (step-up) or indirectly (gait) related to deficits in knee extensor moments. Additionally, examination of both hamstring and gastrocnemi EMG raised the question of whether differences in activation of these two muscle groups were related to knee instability or whether they represented habitual movement patterns. Additional research, some of which will be reported in Chapter 4, is needed to definitively answer this question.

In closing, this work has provided a detailed examination of kinematic, kinetic and EMG data collected from two groups – one with healthy knee joints and the other who had undergone TKA. Although small subject numbers limit the generalizability of the results, a major strength of this study was the fact that it examined this particular patient

population during 2 functional and demanding weight bearing tasks. The simultaneous examination of the two tasks, meant results could be directly compared without the complications that often occur when trying to compare between studies (i.e. methodological differences; variability between participants groups; data processing and analysis differences). The results obtained, as well as the numerous questions raised, provide a sound basis on which future research, aimed at enhancing function in individuals who have undergone TKA, will be based.

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Chapter 4

Muscle activation strategies used during seated knee extension in individuals who have undergone TKA

4.1 Introduction

In the previous Chapter gastrocnemi activation in members of the TKA was observed to be increased during both gait (the LS phase) and stepping-up (during DS). Patients also activated their hamstrings for a significantly greater portion of the stance phase of gait then did healthy controls. Irrespective of whether the EMG differences observed during gait and stepping-up are related to deficits in the knee extensor moment, a question posed in the previous chapter was why did members of the TKA group exhibit these changes in EMG. Several hypotheses must be considered: 1) The EMG differences identified in members of the TKA were remnants of pre-surgical motor patterns. In the years prior to undergoing TKA, individuals altered muscle activation patterns in an attempt to minimize pain and enhance joint stability. Post-surgically, with pain and joint stability markedly improved, individuals continue pre-surgical patterns as they have become habit 2) EMG changes represent a fundamental change in how the neuromotor control system activates muscles in this clinical population perhaps arising due to altered sensory function of the joint 3) Following surgery individuals continue to experience knee joint instability and the altered muscle activation strategies represent an attempt to ensure knee joint stability was maintained.

The question of the source behind the altered EMG patterns observed in members of the TKA group is directly related to one of the primary questions posed in this thesis – does total knee arthroplasty result in some fundamental change in the way patients activate muscle surrounding the knee joint. In order to answer this question, and in so doing provide further insight into why knee moments are reduced following TKA the current study was undertaken. In designing this study, consideration had to be given to the task used. Previous analyses of EMG data in this clinical population (including our own reported in Chapter 3) have generally been done during functional, weight bearing tasks. Conclusions based on EMG results from these studies are complicated by the fact that there are an indeterminate number of motor control strategies (i.e. kinematic combinations at ankle, knee and hip) available for a person to use during tasks such as gait, climbing stairs and stepping onto raised surfaces. The muscle force (and hence

EMG) used to accomplish these tasks directly influences kinematics. If two individuals exhibit different kinematics, then in all likelihood this was accomplished by using different patterns and/or magnitudes of muscle activity. This was exactly the case in the previous study of this thesis (see Chapter 3). While difference in gastrocnemi and biceps femoris EMG were identified in members of the TKA group, interpretation of these differences were clouded by that fact that patients and controls used different knee kinematics during gait. It was possible that the changes in EMG exhibited by patients were simply reflective of these altered kinematics as opposed to being indicative of some underlying change in muscle control.

The current study represents an attempt to remove the effect of variations in kinematics from the examination of muscle activation post-TKA. This was done by requiring participants to complete a task that had predominantly one degree of freedom. The task chosen was a dynamic seated knee extension movement, in which participants extended their knee against a variety of loads, while lower limb muscle activity was recorded. In addition to being a relatively kinematically constrained task, seated knee extension targets key muscles of interest in this population. The primary agonist muscles involved are the quadriceps (Aagard et al 2000; Alkner at al 2000), while hamstring activation is required to slow shank motion near the end of knee extension. As such this task was ideally suited to provide insight into EMG magnitude and timing differences following TKA.

The magnitude of muscle activation is key to any investigation aimed at examining human movement - it plays a central role in determining the amount of force a muscle can produce. An equally important feature of neuromuscular control is the ability to recruit individual muscles in a manner that allows for efficient movement production (Basmajian 1977). While assessment of EMG magnitude provides insight into changes in muscle activation, two other important variables that have received attention in the knee literature are the amount of muscle co-contraction and the relative distribution of muscle activation between medial and lateral knee musculature (i.e. how medial vs. lateral hamstring activation amplitudes compare). These specific measures were chosen based

on examination of literature that has examined function in individuals with knee osteoarthritis (OA). Specifically individuals with knee OA have been found to exhibit: increased lateral hamstring activation during gait (Hubley-Kozey et al. 2006); increased medial co-contraction when subjected to lateral balance perturbations (Lewek et al. 2005, Schmitt and Rudolph 2008); increased hamstring/quadriceps co-activation during gait in those with sever OA (Hubley-Kozey et al. 2008).

While Benedetti et al. (2003) and Wilson et al. (1996) qualitatively reported increased co-contraction following TKA, co-contraction has not been quantified in this population and researchers have yet to investigate if medial/lateral activation distribution is altered in this population. Changes in either muscle co-contraction magnitude and/or medial/lateral activation distribution are of particular interest in relation to TKA because both have the potential to negatively impact joint loading. Co-contraction can have this effect through the increased compression forces it has been shown to create (Cholewicki et al. 1995; Lutz et al. 1993); and altered medial/lateral distribution of muscle activation due to the large role played by muscle forces in determining the distribution of knee joint loading (Shelburne et al. 2006).

The current study was designed to answer the question of whether individuals who have undergone TKA exhibit differences in muscle activation magnitude, co-activation or medial/lateral activation distribution during a task selected because it forces patients and controls to use the same gross kinematic patterns. The following specific hypotheses were proposed:

- *Patients would employ greater magnitudes of EMG during completion of the knee extension task at all weights tested.*
- *Patients would activate all quadriceps muscles earlier than controls at all weights tested.*
- *Co-contraction would differ between patients and controls, with patients exhibiting greater co-contraction of lateral muscles than controls.*
- *Both patients and controls would activate lateral hamstrings to a greater extent than medial hamstrings.*

4.2 Methods

Subject demographics have been previously described in detail in Chapter 2. Briefly six individuals, an average of 32 months post TKA (range 19 – 72 months) were examined in this study. Their results were compared to six healthy controls of similar age. Control subjects had no history of lower limb arthritis or serious lower extremity injury. As detailed in Chapter 2, all members of the TKA group had knee replacement surgery performed by the same surgeon, using the same mobile bearing design, a DePuy low contact stress (LCS) prosthesis (DePuy Orthopaedics, Warsaw, IN). In all cases TKA was performed secondary to osteoarthritis. Prior to data collection knee flexion and extension strength and active range of motion were assessed for all participants using manual muscle testing and goniometry respectively.

For the duration of data collection, all participants sat on a bench, legs flexed at the knees and feet not touching the floor. While in this position, participants completed a total of 18 knee extension trials with each limb. These 18 trials were comprised of 6 blocks of 3 trials completed with a load attached at the ankle. The 6 masses used were: 0kg (W0), 1.1kg (W1), 2kg (W2), 2.9kg (W3), 3.8kg (W4) and 4.7kg (W5). These specific masses were chosen as they represented the weight increments available in the commercially purchased ankle weight used for this study. In all cases, trials started with the 0kg condition and, progressed, in order, to the heaviest mass. Blocks of trials at each mass were alternated between the right and left limb, so participants had a rest period of approximately 4 minutes between loads on any given limb. For the current study, only data from one limb per participant were analyzed. For members of the TKA group, the tested limb was the one that had undergone knee arthroplasty. For control participants, the test limb was determined *a priori* to ensure equal numbers of right and left limbs were examined in each experimental group.

For the current study kinematic data collection procedures described in Chapter 2 were employed to capture the three dimensional (3D) kinematics of the shank, thigh and pelvis. Briefly, an Optotrak (NDI, Waterloo, ON) active marker system was used to record the position of four markers securely affixed on rigid plates to the shank, thigh and

pelvis. Using methods described in Chapter 2, segment and joint kinematics (hip and knee) were determined using 3D marker positions. As the addition of weights at the ankle altered the location of the shank center of mass (CoM), adjustments had to be made to account for this change. This was done by using the parallel axis theorem (Winter 2005) to adjust shank center of mass position in trials where mass was added at the ankle.

Muscle activity was recorded from all major muscles crossing the surgical (control) knee joint: vastus medialis (VM), vastus lateralis (VL), rectus femoris (RF), medial hamstrings (MH), biceps femoris (BF) and medial and lateral gastrocnemius (MG and LG respectively). All EMG data were normalized to EMG collected during a maximum voluntary effort. Details of the data collection and analysis procedures used are given in Chapter 2.

4.2.1 Data analysis

To facilitate further analysis of the normalized EMG signal, the start and end of the knee extension motion was determined. Several random trials from different participants were examined to determine the most reliable way of doing this. The method selected used sagittal plane knee angular acceleration data (determined by double differentiating the sagittal plane knee angles). Custom designed software (designed using Visual Basic 6.0) automated the approach, which identified the start of knee motion as occurring at the frame where knee angular acceleration was different from zero for at least 0.17s. Termination of knee extension was identified as the frame at which the knee extension angle reached its peak – this process was also automated. All automatically detected start and end frames were examined for outliers following data processing. While end frame detection was never in error, there were several instances were start frames were obviously incorrect (i.e. end frame occurred before start frame or duration of knee extension was unusually long or short). Trials with errors were visually examined and start times were corrected – this had to be done for approximately 20% of the trails.

All further analyses were done on data truncated to end at the final frame of knee extension. Note that no truncation was done to the beginning of the trial as EMG activation prior to the start of motion was of interest. Two primary analyses were then performed on the normalized and truncated EMG data - one focusing on EMG amplitude, the other on activation timing. During the process of EMG analysis, the presence of 60HZ noise was found in 3 subjects (2 patients and 1 control participant). To remove this noise, all EMG signals from the affected participants were subjected to a notch filter as described by Mello et al. (2007). The filter coefficients for the dual-pass Butterworth notch filter (59-61Hz) were determined using Matlab (filter function in Matlab 2006a), while filter design and implementation was done using custom written software.

Variables of interest for the current study were EMG amplitude (as determined using the RMS values) and muscle co-contraction which have both been described in detail in Chapter 2.

Analysis of muscle onset timing was done using combined automated computer detection and visual inspection. Initial attempts at using approaches outlined by Hodges and Bui (1996) proved unsuccessful, since for most trials the methods suggested by these authors resulted in quadriceps onset occurring following movement initiation. Examination of the raw EMG suggested that this was due to high levels of resting EMG prior to the start of knee motion. Instead of using full wave rectified EMG, as advocated by Hodges and Bui (1996), the full wave rectified EMG was low pass filtered using a dual pass, critically damped filter at a cut-off of 6Hz to create a linear envelope (LE). LE EMG during the resting portion of the trial (generally the first 200 frames of data, however this was sometimes manually adjusted if motion started at or near the 200^{th} frame) was then averaged and subtracted from the LE EMG signal, thereby removing the bias that existed. The mean and standard deviation (SD) of the first 200 frames of this zeroed LE EMG signal were then determined for each individual muscle. Muscle onset was said to occur when the zeroed LE EMG signal exceeded the mean ±3SD for at least 1000 (0.8s) frames of data (see Fig 4.1 for illustration of this method). Following automatic detection of EMG onset times, all onset results were visually examined to ensure their accuracy. As a

result of this visual examination, onset times were sometimes changed by a researcher who had no knowledge of the frame number at which motion started for the trial being examined. The most common reason for changing onset time was because the automatic detection algorithm missed the initial burst of EMG data that, upon visual inspection, clearly indicated the onset of muscle activity. This visual examination resulted in 105/200 trials being changed for VL, 102/200 for VM and 97/200 for RF. No changes were needed for BF, MH, MG or LG. Since all muscles remained on until after full knee extension was reached, no analysis of muscle off times was carried out for the current study. All onset times were reported with respect to the onset of knee motion. In addition, the onset timing of VL with respect to VM, VM with respect to RF, and RF with respect to VL were also determined.

4.2.2 Statistical analysis

Two-way repeated measures ANOVAs were employed to statistically examine data from this study. ANOVA factors used were group (patient and control) and weight (W0, W1, W2, W3, W4, W5). Co-contraction, RMS, muscle onset time, total time to completion of knee extension, and total knee range of motion covered during knee extension were analyzed in this manner. Significant main and interaction affects were examined using Tukey's test using a Bonferroni correction of the alpha level. For analyses where no significant main effect of weight was found, weight data were pooled and a 2-way repeated measures ANOVA (factors group and trial) was used to assess differences between patients and controls. An alpha level of $p=0.05$ was used for all analyses.

The analyses described above probed the differences between patient and controls for each of the individual muscles examined in the current study. In addition to these analyses, comparisons were made between medial and lateral muscle groups. Three-way repeated measures analysis of variance (Factors: group, weight and side [lateral or medial]) were used to assess whether there were within group differences in the way that medial and lateral muscles were activated or co-activated. To this end, a within group analysis was done comparing the following variables: MH and BF; MG and LG; MHVM and BFVL; and MGVM and LGVL. A similar analysis was done comparing VM, VL and

RF; in this case the third factor in the analysis was muscle not side. An alpha level of p = 0.05 was used, with post-hoc Tukey tests being used to evaluate significant interaction effects.

4.3 Results:

All participants were able to successfully lift each of the weights used in the study with no reports of fatigue or discomfort. Both groups exhibited the same amount of knee motion during knee extension. Knee extension in members of the TKA group ranged from 69 º of flexion to 6 º of extension. Controls extended from 73 º to 7 º. The amount of time required to complete the knee extension portion of the motion was 2.43sec (±0.54) for patients, while controls completed it in 2.25sec (±0.57). Neither the knee range of motion nor the time to complete it differed significantly between the two groups.

Muscle activation patterns during seated knee extensions showed all participants activated both quadriceps and hamstrings during the task. Figure 4.1 provides sample LE EMG data during a 0kg trial. All participants exhibited these patterns of activation in the quadriceps, BF, MG and LG. The pattern displayed for medial hamstrings was found in 8/12 participants. Visual examination of LE EMG data showed that the remaining four subjects (two patients and two controls) did not show a burst of activity in MH.

Figures 4.2 provides a summary of RMS levels in hamstring and quadriceps muscles at each of the loads tested. Both patients and controls exhibited similar RMS for hamstrings and quadriceps. For all muscles except VL the group*weight interaction effect was non-significant ($p>0.05$). While the group*weight interaction effect for VL was significant ($p=0.0069$), post-hoc analysis revealed no differences of interest for the current study. A significant main effect of weight existed for the quadriceps (VL: $p =0.0008$; VM: $p = 0.0016$; RF: $p<0.0001$). Despite this significant main effect,

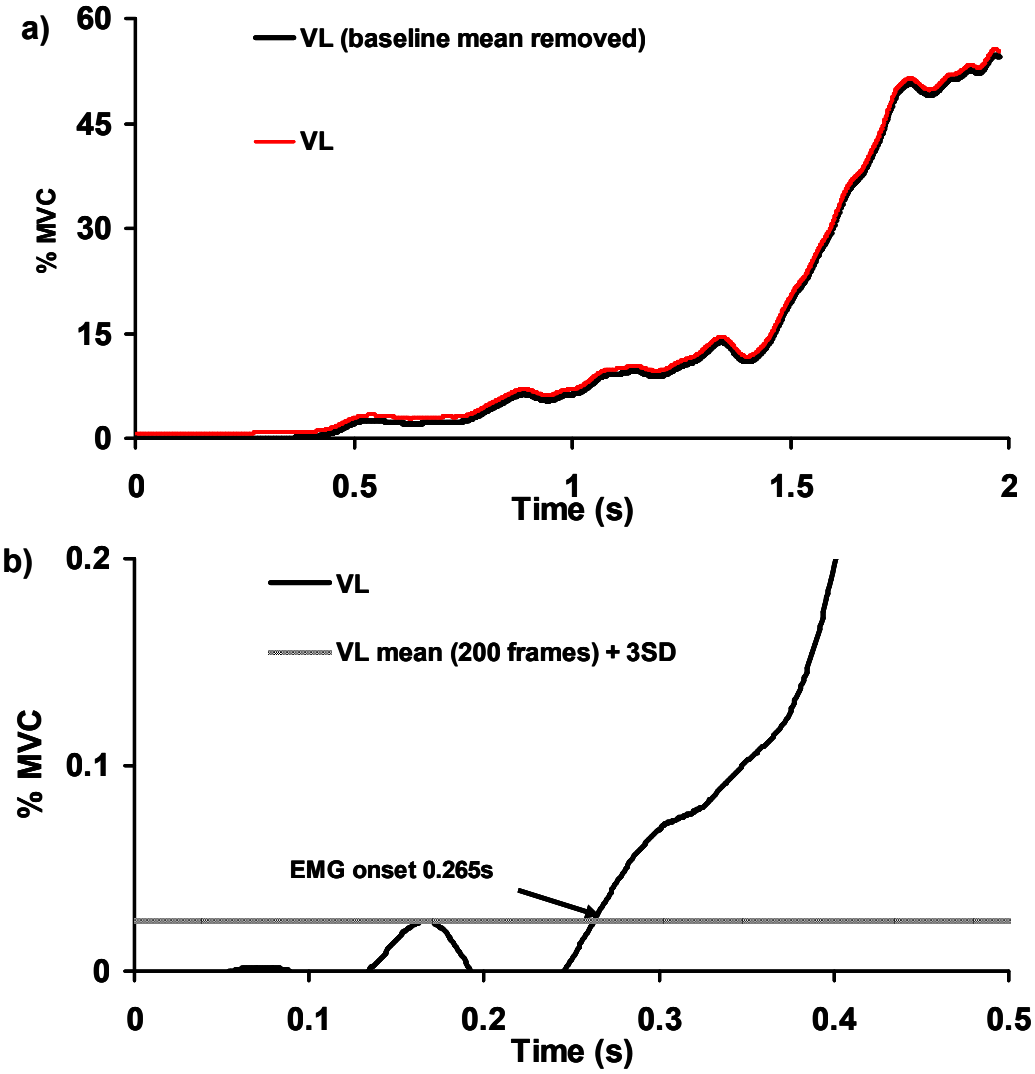


Figure 4.1: a) Upper panel displays linear envelope EMG for vastus lateralis (VL) during a weighted knee extension trial. The shaded line is the 'raw' linear enveloped data, the solid dark line has had the mean of the first 200 frames of the LE signal subtracted from it. b) An enlargement of the first 0.5s of figure a) showing the linear enveloped VL signal with baseline removed (dark line). The shaded grey line represents the mean+3 SD of the zeroed VL signal (first 200 frames were used to find mean and SD, see text for details). The point indicated, where the VL signal surpasses the mean + 3SD level, represents the time selected as the onset of VL activity during this trial.

examination of post-hoc tests showed no evidence of EMG scaling with weight. BF and MH had no significant main effect of weight, so data from all weight trials were pooled and a one-way repeated measures ANOVA was performed. Results of these analyses revealed no significant effect of group on either BF or MH activation levels.

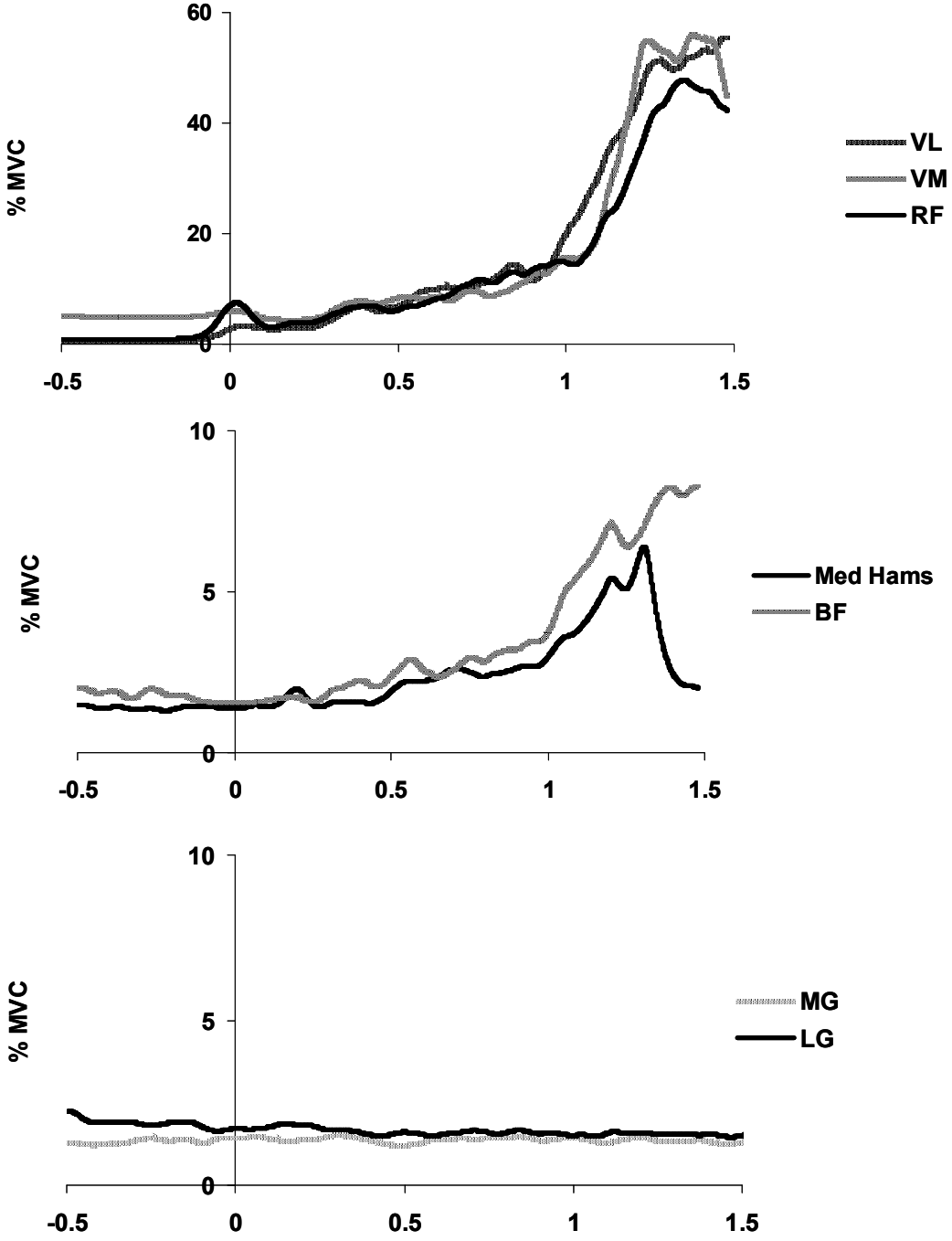


Figure 4.2: Linear enveloped EMG from three muscle groups during knee extension. Time 0 represents the start of knee motion. End of data signified the completion of knee extension motion. Patterns depicted are typical of all participants, with the exception of medial hamstrings. As outlined in the text, 8/12 participants exhibited the pattern above, while 4/12 showed no burst of MH activity during the task.

Patients showed consistently greater activation of MG and LG than controls (Fig. 4.4). Irrespective of which portion of the knee extension motion was examined this observation was supported by a significant main effect of group for both muscles (MG: $F_{1,5}$=6.96, p=0.025; LG: $F_{1,5}$=6.82,p=0.026). Neither the main effect of weight nor the interaction effect was significant for MG or LG.

Both groups exhibited greater BF activation compared to MH during weighted knee extension. BF levels averaged approximately 11%MVC for both groups, while MH levels reached only 5%MVC (Fig. 4.5a). While the group*muscle interaction did not reach statistical significance, a main effect of muscle ($p<0.0001$) was found.

Quadriceps activation was less consistent between the two groups (Fig. 4.5b). Controls activated VL to near 25% MVC, while VL and RF were activated at slightly lower levels (VL=23%, RF=23%). Post-hoc analyses of a significant group*muscle interaction effect (p=0.0004) revealed that VL activation was significantly greater than VM (p=0.0003). For patients, there were no significant differences in the level of activation of the 3 superficial quads.

Co-contraction levels appeared greater for patients when gastrocnemi CCIs were examined and greater for controls when hamstring CCIs were considered (Fig. 4.6). Only the LGVL CCI during the 2nd half of knee motion was statistically different between the groups, with patients values exceeding controls (main effect of group p=0.03). When medial and lateral (side) co-contraction levels were compared using a 3-way repeated measures ANOVA (group, side and weight) there was no main effect of weight. Data were collapsed across weight groups and the analysis, now a 2-way ANOVA, was redone (see results in Fig. 4.7). When comparing BFVL to MHVM, a significant main effect of side during both portions of knee extension ($p<0.0001$) confirmed that lateral co-contraction was greater then medial. During the 2nd half of knee motion, this significant main effect of side was further confirmed by a significant group*side interaction effect ($p<0.0001$). Post hoc analysis of this interaction revealed that the lateral hamstring CCI exceeded medial for both patients and controls. A similar main effect of side was found

for lateral gastrocnemi CCI during the second half of knee motion ($p<0.00001$: LGVL>MGVM). This main effect of side was driven primarily by greater co-contraction laterally in patients, as evidenced by a significant group*side interaction effect ($p<0.0001$). Post-hoc analysis of this interaction revealed the only difference of interest to be in patients where LGVL was significantly greater then MGVM ($p<0.0001$).

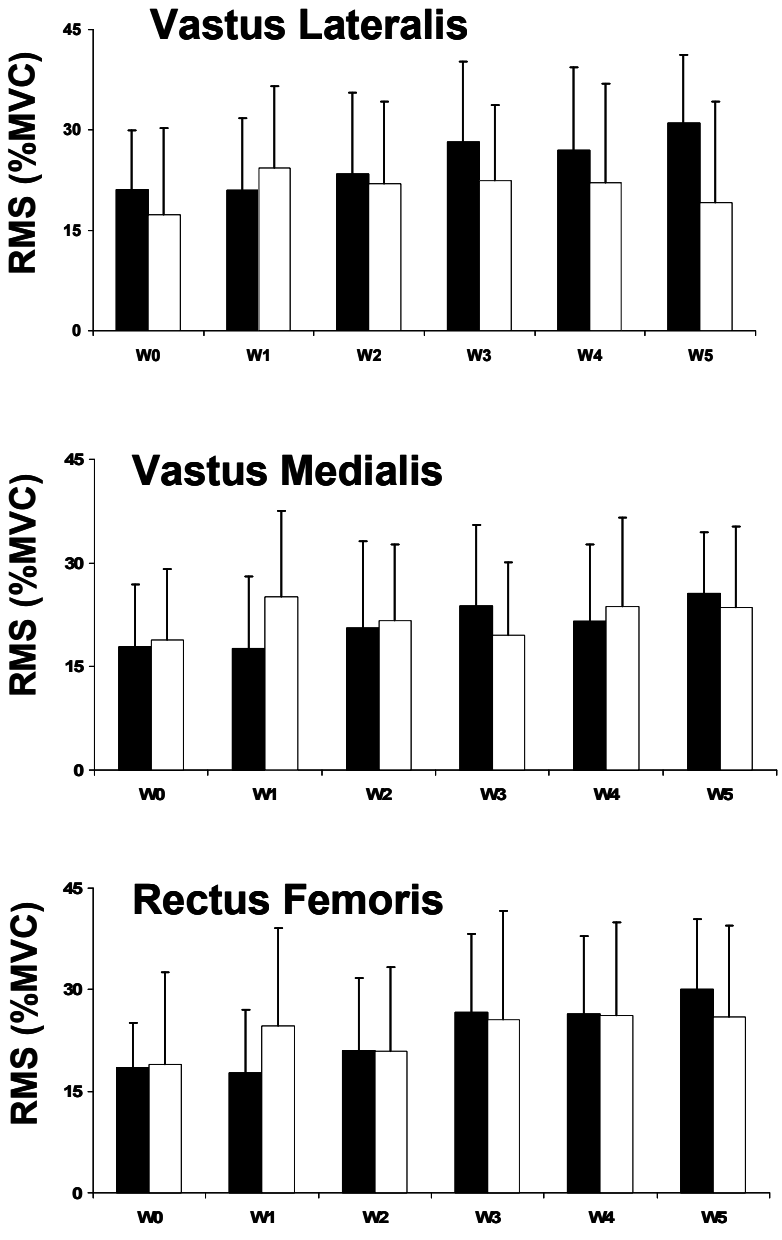


Figure 4.3: RMS of VL, VM and RF were calculated from start to end of knee extension motion. No significant main or interaction effects. Errors bars represent standard error. (W0-W5 represent the six loads lifted – see text for details)

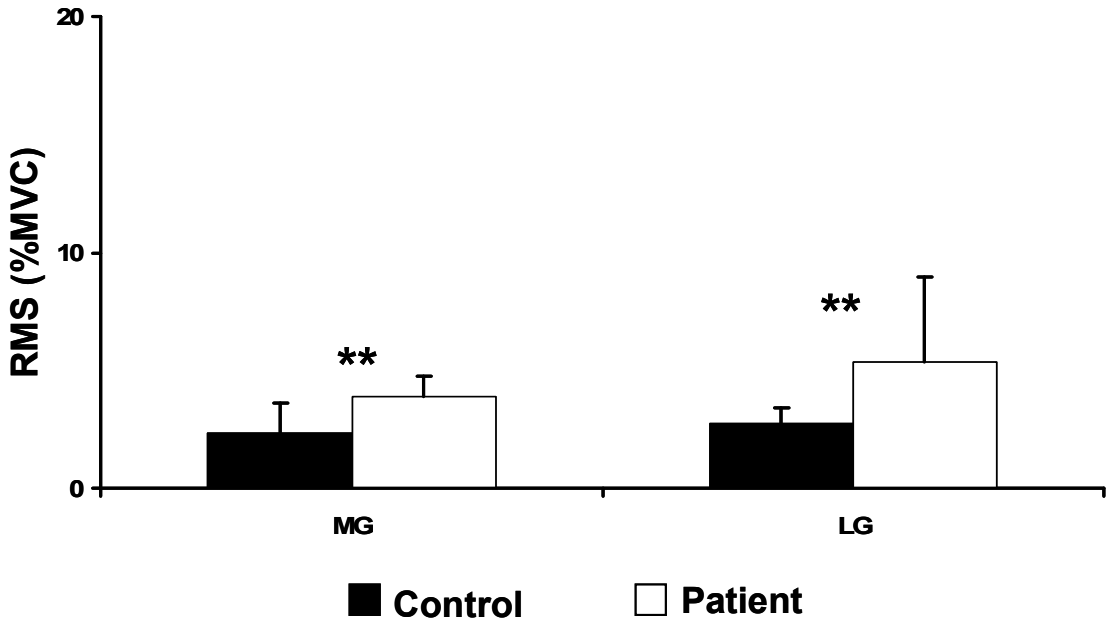


Figure 4.4: Medial and lateral gastrocnemius RMS values for patients and controls (first half of knee motion – similar results found for second half of motion). ** indicates significant group effect. Error bars represent standard error.

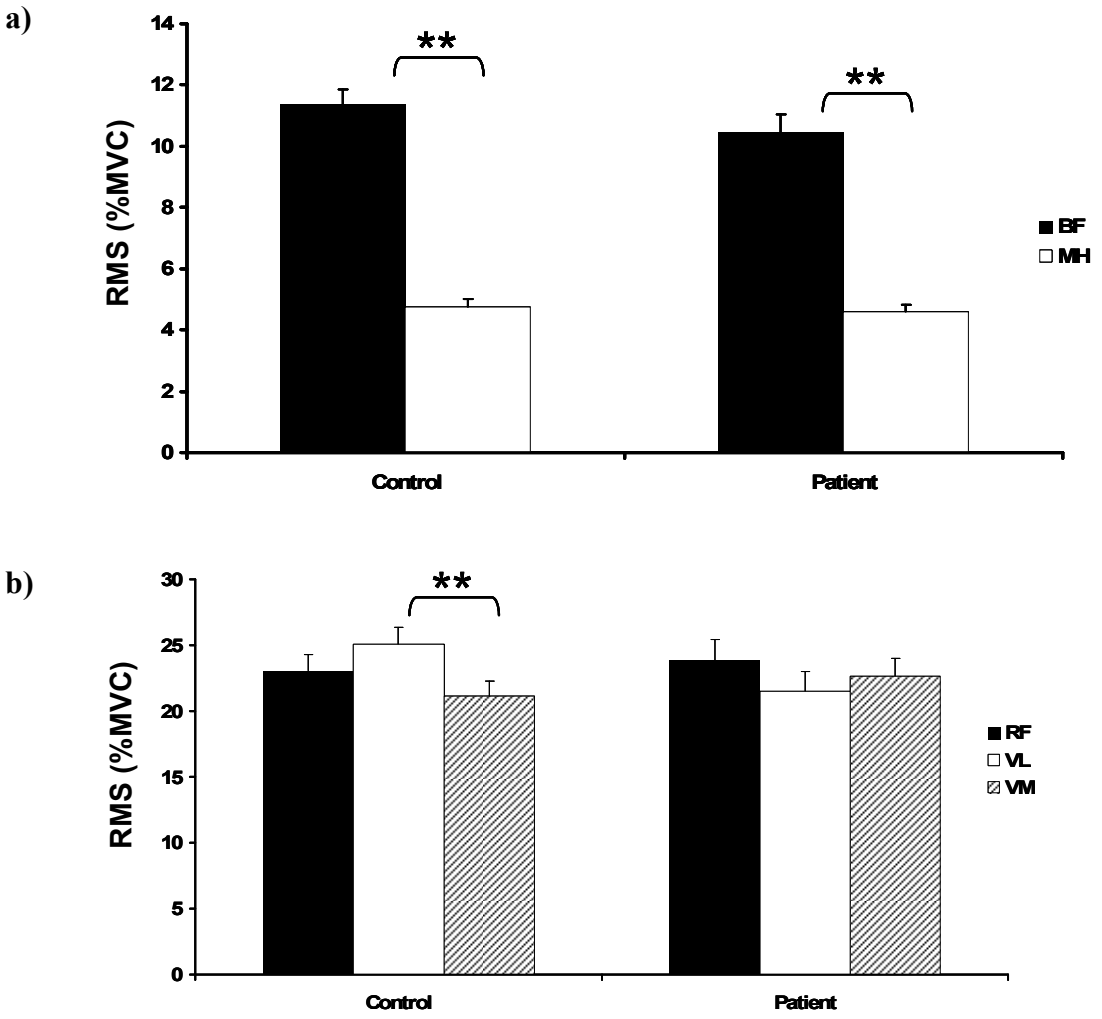


Figure 4.5: a) Biceps femoris (BF) and medial hamstring (MH) RMS for controls and patients during seated knee extension. b) Rectus femoris (RF), vastus medialis (VM) and vastus lateralis (VL) RMS EMG during seated knee extension. (** $p<0.0001$). Error bars represent standard error of the mean.

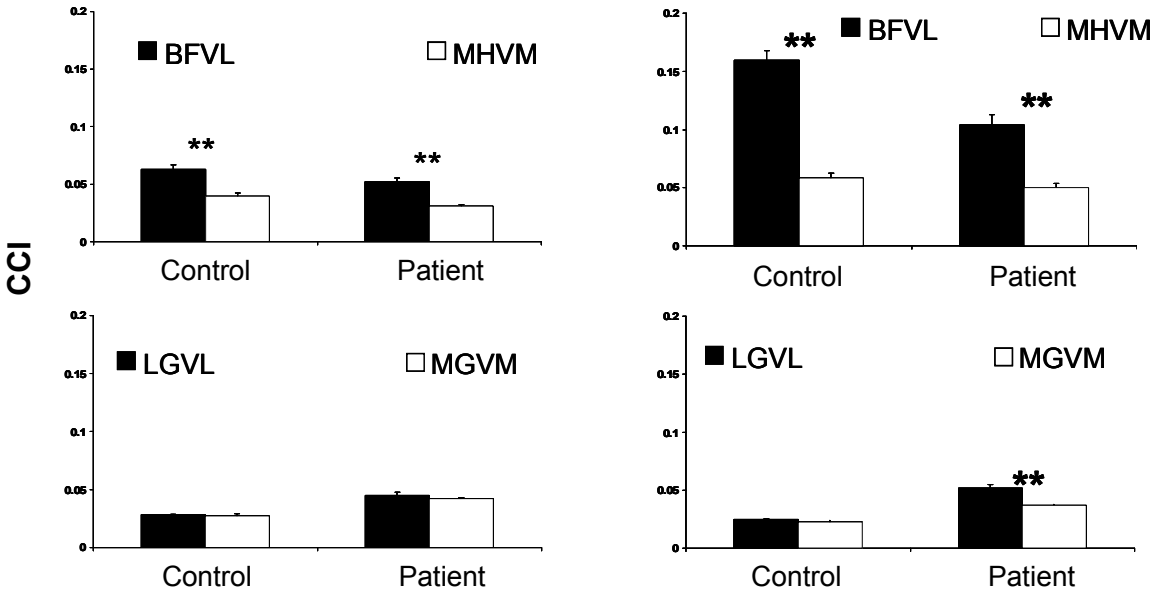


Figure 4.6: Within group comparison of medial vs. lateral co-contraction of the hamstrings/quadriceps (BFVl vs. MHVM) and gastrocnemi/quadriceps (LGVL vs. MGVL) during the first (left column) and second (right column) half of the knee extension motion. Error bars represent standard error.. ** denotes significant difference as determined using post-hoc analysis of the significant group*side interaction effect.

In general, the results of the EMG timing analysis agree with what would be expected for this type of motion. Hamstring activation occurred after the start of motion, with MH becoming active an average of 0.53s (± 0.78) after the start of motion and BF being activated 1.17s (± 0.64) following initiation of knee extension. No significant main or interaction effects existed for either of the hamstrings. Pooling of data with respect to load magnitude did not alter the results of the statistical analysis. When quadriceps muscle onset was examined both RF and VM appeared to be activated earlier in patients then in controls. Statistical analysis of this data failed to find any main or interaction effects. Pooling of data across load magnitudes did not affect this result.

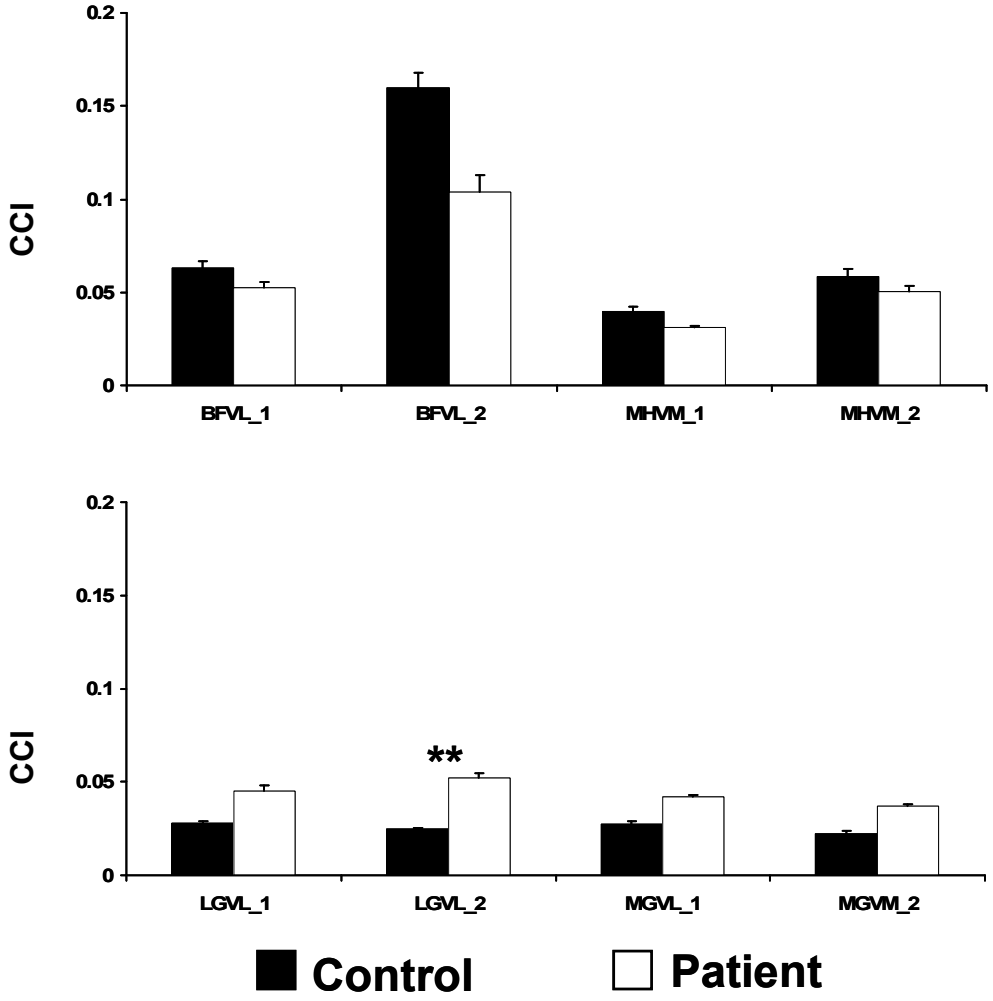


Figure 4.7: Patient and control CCI measures during the first (_1) and second (_2) portion of knee motion for the muscle groups indicated. * denotes patient values significantly greater then controls.

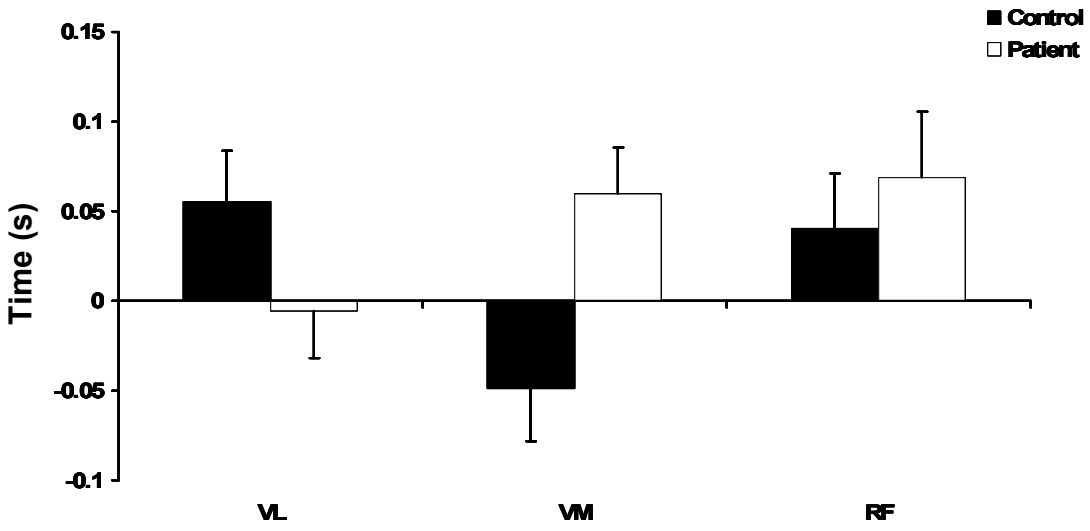


Figure 4.8: Muscle onset time expressed with respect to the start of knee motion for VL, VM and RF. Negative times indicate muscle activation occurred after the start of knee motion. Error bars indicates standard error.

4.4 Discussion:

This paper is the first to report results based on an EMG analysis of dynamic seated knee extension following TKA. It could be argued that seated knee extension was not a functional task, and as such would provide little insight into functional performance in this population. While this argument would have some merit, dynamic seated knee extension was specifically chosen for the current investigation because it was both demanding for the quadriceps (Anderson et al 2006), while at the same time has sufficient kinematic constraints (i.e. motion is isolated to the knee joint) to ensure that alterations in kinematic patterns do not cloud interpretation of EMG data.

4.4.1 Gastrocnemi EMG: amplitude and co-contraction

When walking and stepping onto a raised surface, members of the TKA group exhibited increased activation of their medial and lateral gastrocnmi muscles (see Chapter 3). In the

current examination these same individuals also exhibited elevated LG and MG EMG, irrespective of the weight being lifted. The existence of increased gastrocnemi activation in this controlled knee extension task AND during gait and stepping-up suggests that members of the TKA group may be exhibiting some fundamental change in their ability to activate the gastrocnemi muscles. While the small subject numbers used in the current study mean this finding cannot be generalized to a larger population, the results do represent the first report of altered gastrocnmei function in this clinical group.

Increased gastrocnemi activation have been previously reported in individuals with severe (Hubley-Kozey et al. 2008) knee osteoarthritis (OA), leading to the hypothesis that the changes in gastrocnmei activation observed in members of the TKA group may in fact represent a muscle activation pattern learned prior to surgery. Indeed several authors (Andriacchi et al. 1982; Hilding et al. 1995; Wilson et al. 1996) have suggested that post-surgical knee function in those who have undergone TKA may, at least partially, be due to habitual movement patterns learned prior to surgery. An alternative explanation, suggested in the previous chapter, is that both groups exhibit increased gastrocnemi activation in response to knee instability. As the demands on the knee were relatively low during seated knee extension (i.e. it is a non-weight bearing activity), the need for enhanced stability would be minimal. This line of reasoning seems to provide added support to the habitual movement pattern hypothesis.

In order to gain more insight into the exact nature of the changes in gastrocnemi activation, replication of the current study with a large, homogeneous patients group is needed. Additionally, pre-post studies of individuals undergoing TKA need to be carried out so that the link between pre and post surgical gastrocnemi activation levels can be established. While this has been done by Smith et al (2004) looking at sagittal plane knee moments, no efforts have been made to longitudinally examine change in muscle activation pre and post surgically. This work is integral if the hypothesis regarding the role of habitual movement patterns is to be definitively elucidated. Such work will have important clinical application, as the gastrocnemi are very often not included in rehabilitation programs aimed at improving function in this population. Clearly, results

from this and the previous study, indicate that this could potentially limit the recovery of this clinical group. Discovering the underlying cause behind the increased gastrocnemi activation is of great importance given the link between altered gatrocnemi activation and decreased knee extensor moments proposed in the previous chapter. It will also have effects on the interventions used with this population. If altered calf muscle activation is merely habit gait retraining can be implemented to help patients return to a more normal pattern. If on the other hand it is due to a need for enhanced knee joint stability, then removal of this strategy may put the knee under undue strain and potentially shorten the life of the prosthesis.

4.4.2 Quadriceps activation:

As was the case in our examination of gait and stepping-up, quadriceps activation during weighted knee extension was similar between members of the TKA group and controls. This finding further confirms the conclusions drawn in the previous chapter, namely that members of the TKA do no appear to have an inherent inability to activate their quadriceps. This finding, however, did not agree with our hypothesis that EMG magnitudes would be increased in members of the TKA group. As this was the first investigation of EMG during knee extension in a post-arthroplasty population, this hypothesis was based on the relatively large body of work that has examined EMG magnitudes in individuals with OA. Hubley-Kozey et al (2008) reported increased activation of VL and VM during the stance phase of gait in individuals one week prior to undergoing TKA. Individuals with less severe OA have also been examined by Hubley-Kozey et al. (2006) and by Zeni et al (2009) – both groups reported increased levels of VL activation and normal levels of VM recruitment during stance phase of gait. In light of increased VL activation in individuals with OA, the results of the current study could be used to refute the argument that knee function following TKA is determined largely by habitual patterns learned prior to surgery. If this were indeed the case, increased levels of VL activation would have been expected in members of the TKA group. This seems to conflict with our previous conclusions related to increased gastrocnemi activation.

Alternatively, it could be argued that since increased VL activation in individuals with knee OA is hypothesized to occur in order to enhance knee joint stability (Hubley-Kozey et al. 2006), the demands of the knee extension task were perhaps not great enough to create feelings of instability - hence increased VL activation was not needed. However, the gait discussed in the previous chapter also reported no change in EMG magnitudes. This finding, coupled with the results of the current study, suggests that following TKA quadriceps EMG magnitudes were not altered. Confirmation of this finding by expanding subject numbers using the current experimental protocol will add strength to this argument.

In addition to muscle activation amplitude, the timing of muscle onset can also potentially alter the net joint moment. During our examination of gait in the previous chapter, patients activated their hamstrings for a greater percentage of stance then controls. This finding was similar to that of Bendetti et al. (2003) who examined individuals following TKA and has also been observed in those with moderate (Hubley-Kozey et al. 2006) and severe (Astephen et al. 2008; Hubley-Kozey et al. 2008) knee OA. Because of the presence of prolonged activation in those with knee OA it was hypothesized that patients were merely continuing to activate their muscles in the way they had prior to surgery. If this were the case, then why wasn't prolonged hamstring activation observed when participants performed seated knee extension? This is felt to be related directly to the nature of the knee extension task. Unlike gait where periods of minimal hamstring activity occur, once the hamstrings are activated when performing seated knee extension they remain on until after the knee reaches full extension. Members of the TKA, therefore have no opportunity to prolong activation of this muscle group as it was already activated for the entire task.

Although no significant difference in quadriceps onset times were observed, patients did exhibit strong trends suggesting they activated both VM and RF earlier then controls. Although our RF findings must be interpreted with care due to possible crosstalk (see discussion in Chapter 3) we are confident that the earlier onset of VM is of functional significance. Beneditti et al. (2003), who examined EMG during gait, individuals who

were 6, 12 and 24 months post-TKA, have also reported prolonged activation of the quadriceps muscles. Benedetti et al (2003) attributed the prolonged quadriceps activation observed during gait directly to the reduced knee flexion exhibited by their participants. Similar patterns of decreased knee flexion were reported in our gait study (see Chapter 3) and have been observed by Wilson et al (1996). While the suggestion that prolonged quadriceps EMG during gait was needed to support a less flexed knee might be plausible, in the current study there were no weight bearing demands being placed on the joint. As such, early activation of cannot be attributed to prevention of lower limb collapse.

An alternative explanation for the early VM onset may be related to the specific role VM plays at the knee joint. Vastus medialis is generally regarded as providing medial/lateral stabilization to the patella (Toumi et al. 2007). Delayed onset time of VM was hypothesized to be one of several factors contributing to the development of patellar femoral pain syndrome (Boucher et al 1992). Unlike individuals with patellar femoral pain, patients in the current study exhibited earlier activation of VM then healthy controls. It was possible that this early activation was an attempt to ensure patellar stability during knee extension.

A different explanation can be found in the work of Pincivero et al (2004). These authors examined the force production efficiency of VL, VM and RF at numerous knee joint angles. They found that the greatest efficiency in all three muscles occurred at greater knee flexion angles. Additionally, Pincivero et al (2004) suggested that, based on their results, VM appeared to have enhanced ability to contribute to knee extensor force at flexion angles between 70 and 90º. This was precisely the starting point for the knee extension performed by participants in the present study. This suggested that members of the TKA group may have been taking advantage of the increased efficiency of VM in an effort to successfully complete the knee extension task. Both hypotheses - enhanced patellar stability and improved VM efficiency - provided possible explanations for why patients activated VM earlier during seated knee extension. With data from the current study, it was not possible to determine which, if either, of these explanations was correct. Additional insight into the reasons behind prolonged VM activation was found in the

results a gait analysis performed on these same subjects (see Chapter 3). In this study, VM onset/offset was normal for members of the TKA group. As patellar stability was important for gait and knee extension, one could conclude that the early activation of VM observed during knee extension was therefore not likely related to patellar stability. Certainly at this point, a definitive conclusion cannot be drawn – this will require addition research designed to replicate the knee extension findings with a larger sample and examine more deeply the functional implications of early VM onset.

4.4.3 Hamstring activation:

There has been considerable research examining muscle activation during seated knee extension in healthy individuals (Aargaard et al. 2000; Andersen et al. 2006; Escamilla et al. 1998; Wilk et al. 1996). Based on an examination of the EMG data provided by these authors, lateral hamstring activation amplitude was greater than the activation that occurs in the medial hamstrings. Results of the current examination of knee extension agreed with this finding – lateral hamstring EMG was greater than medial for both patients and controls (see Figure 4.6). Additionally, the amplitude of EMG produced by patients and controls did not differ for either medial or lateral hamstrings. Collectively these two findings directly address two of the hypotheses of this study. The lack of difference between the groups suggests that, at least for the group of post-arthroplasty patients examined in the current study, hamstring activation amplitude and the medial/lateral distribution of activation were not altered. While the small subject numbers examined prevent generalizing this finding, the results did provide initial insight into an area of muscular control not previously examined in individuals who have undergone TKA.

Given the lack of magnitude differences for the hamstrings and quadriceps it was not surprising that the hamstring/quadriceps CCIs (i.e. BFVL and MHVM) did not differ significantly between patients and controls. Comparison of these CCI results to other TKA literature was not possible because this measure has not been previously quantified in this patient population. Benedetti et al. (2003) and Wilson et al. (1996) both suggested co-activation was increased post-TKA based on evidence of muscle onset/offset timing differences, but neither researcher objectively quantified the amount of co-contraction.

Aargard et al. (2000) did report that lateral hamstrings exhibited 3-times the co-activation as medial hamstrings during seated knee extension in healthy individuals. Once again, comparison with this finding was not appropriate as Aargard does not formally quantify co-contraction, but rather based this conclusion on non-normalized integrated EMG in medial and lateral hamstrings during knee extension.

Irrespective of the inability to compare results with previous research, the lack of between group differences in CCIs suggests that, for members of the TKA group, no evidence of increased co-activation was evident. Typically, increased co-contraction occurs when joint stability was of paramount importance (Solomonow 1987). Not surprisingly then, co-contraction has often been observed to increase in situations where instability exists. For example it has been observed in individuals with ACL deficient knees (Rudolph et al. 2000) and in individuals with knee OA and known knee laxity (Schmitt and Rudolph 2008a and 2008b; Lewek et al. 2005). As such, the lack of increase in co-activation may be merely a reflection of the relatively undemanding nature of seated knee extension. This having been said however, it is important to recognize that these co-activation results do show that members of the TKA group do have the ability to exhibit normal co-activation levels. Whether this can be generalized to a larger population of individuals who have undergone TKA will require additional study.

4.5 Summary

This study represents one of the first attempts at examining muscle function following TKA during a task designed to specifically target the knee joint, while limiting the possibility of alternative kinematic patterns being used by patient and control groups. The results suggest that the general pattern and timing of muscle activation were similar for patients and controls. Members of the TKA group also exhibited normal magnitudes of quadriceps and hamstring EMG and were able to differentially activate medial and lateral muscle groups in a manner similar to healthy control subjects. They also produced normal amounts of co-contraction between the quadriceps and hamstrings. The only differences observed between the groups were increased gastrocnemi EMG and earlier

onset of VM. Collectively these results point to relatively intact control of knee joint muscles during seated knee extension. The differences that were identified could potentially represent changes in how medial and lateral gastrocnemi are controlled following TKA. This results, combined with results from our previous examination of the same individuals performing weight bearing tasks, suggests that this alteration in gastrocnemi activation may be indicative of some inherent inability of individuals to activate the these muscles normally. Additionally, evidence of similar disturbances in gastrocnemi activation in individuals with severe knee OA (Hubley-Kozey et al. 2008), suggests the gastrocnemi activation changes in the TKA group may indicate a carryover of habitual movement patterns learned prior to surgery. Clearly further research, aimed at replicating the above research with considerably greater subject numbers, is needed before any generalizable conclusions can be drawn. These limitations not withstanding, this research does demonstrate the potential benefits of using seated knee extension to gain insight into muscle performance following TKA, and it shows the benefits of using a relatively kinematically constrained task to enable more focused assessment of muscle function in clinical populations.

Chapter 5

Passive Knee Moments Following Total Knee Arthroplasty

5.1 Introduction

Chapter 3 and 4 of this thesis have focused on examining how magnitude and pattern of muscle activation differs between individuals with healthy knee joints and those who have undergone total knee arthroplasty (TKA). These investigations have been carried out in an effort to determine if changes muscle activation strategies explain the deficits in knee extensor moment commonly observed in this population. By focusing on muscle activation, these previous chapters have been concerned with examining the active portion of the net joint moment. In addition to this active component, the net joint moment is also influenced by the moments arising from passive tissues of the knee joint. Given the contribution of these passive moments to the net joint moment it is plausible that net knee extensor moments may be reduced following TKA due to changes in the passive moments of the knee joint. As no previous research has examined passive knee moment following TKA the goal of this study was to quantify the magnitude of the sagittal plane passive knee moment in individuals who had previously undergone total knee arthroplasty. In so doing, study results will enable us to answer the question "Do changes in the passive knee joint moment exist following TKA?" If changes do exist do they have the potential to contribute to reduced net extensor knee moments?

Due to difficulties inherent in measuring individual tissue forces in vivo, passive joint moments have been most commonly quantified by determining the net passive joint moment (Esteki and Mansour 1996). This has been done by passively moving a joint through range and measuring the force (moment) required to produce the motion. Two methods of passively moving the joint have been used: motion has been produced by an investigator moving the joint (Reiner and Edrich 1999; Silder et al 2007; McFaull and Lamontagne 1998; McGill et al 1994) or by an isokinetic dynamometer (Magnusson et al 1998; Blackburn et al 2004; Nordez et al 2006). If muscle activity is kept to a minimum, motion is done at a slow speed, and the effects of gravity are accounted for, the force data can then be used to calculate passive joint moment (see Reiner and Edrich 1999). This approach has been used by Yoon and Mansour (1982) and Mansour and Audu (1986) to assess sagittal plane passive hip and knee moments respectively; by McGill (1994) to

assess passive tissue properties in lumbar spine; by Reiner and Edrich (1999) in knee, hip and ankle joints; by Silder et al (2007), Edrich et al (2000) and McFaull and Lamontagne (1998) in the knee joint; and by Dionysian et al (2005) in the finger joints.

As a primary objective of this research is to assess whether or not changes in the passive knee moment can account for deficits in the knee extensor moment during functional activities, a key variable that will be examined is the magnitude of the passive knee moment. An additional variable of interest, however, is how any changes in passive knee moment may alter knee joint stiffness. From a mechanical perspective joint stiffness is defined as the amount of force (moment) required to produce a given amount of linear (angular) deformation at the joint. It is easily determined by finding the slope of the passive moment / angle curve. Joint stiffness has been quantified in lower limb joints by Nordez et al (2006), Blackburn et al (2004) and Magnusson et al (1998), and in the lumbar spine by McGill et al (1994). While Latash and Zatsiorski (1993) raise some issues with the application of the term 'stiffness' to complex physical structures such as joints, it is a concept that adds to our understanding of passive joint resistance. Knee joint stiffness has been examined in healthy young individuals (Nordez et al 2006) and in older runners (Magnusson and Larsson 1997). Silder et al (2007) have examined stiffness at the ankle, knee and hip. Although individuals often complain of increased knee joint stiffness following TKA (Moffett et al. 2004), it is unclear whether there is a true increase in mechanical joint stiffness (i.e. patients may be feeling muscle tightness or increased resistance to joint motion. The proposed study will provide additional insight into this question.

5.2 Methodology

Experimental data from six individuals, an average of 32 months post TKA (range 19 – 72 months), were examined and results compared to six age matched controls with no history of lower limb arthritis or serious lower extremity injury. The reader is referred to Chapter 2 for details of subject demographics.

Passive knee moment data were collected using a protocol modified from that of Reiner and Edrich (1999). The setup used was designed to allow for simultaneous collection of kinematic and kinetic data. With subjects seated in a specially designed chair, the experimenter slowly moved the knee in the sagittal plane from a seated flexed position to full extension and back again. The force required to move the knee through range was recorded using a mini low profile (MLP) load cell (Transducer Techniques, Temecula, CA) that was attached just above the ankle malleoli using a custom designed ankle brace. The load cell was mounted such that it measured forces exerted perpendicular to the long axis of the shank. Load cell data were collected at a rate of 1200Hz using Optotrak (NDI, Waterloo, ON) software. A Daytronic signal conditioning device (Daytronic Corporation, Dayton, Ohio) was used to amplify the force signal prior to sampling.

The three-dimensional (3D) kinematics of the knee joint were recorded during knee motion with an Optotrak (NDI, Waterloo, ON) active marker system. Cluster of four infrared light emitting diodes (IREDs), attached to a rigid plate, were affixed to both the shank and thigh at mid-segment level and used to track 3 dimensional segment motion. The procedures used to determine knee joint kinematics were based on calibrated anatomical system technique (CAST) (Cappozzo et al. 1995) and were described in detail previously (Chapter 2). Note that for the present study, only a 2 segment model (shank and thigh) was employed, as knee joint angular motion was the focus. All other details of the analysis are similar to those described in Chapter 2.

In order to ensure that the knee motion was purely passive, muscle activity in the quadriceps (vasutus medialis and lateralis) and hamstrings (medial hamstrings and biceps femoris) was monitored using electromyography (EMG). Two silver – silver chloride electrodes (MediTrace 130, Mansfield, MA) were placed as per Winter (1991) on the muscles of interest and a reference electrode was placed on anterior shank. A Bortec EMG system (Bortec Medical, Calgary, AB) was used to differentially amplify the signal (input impedance 100GOhms; MRR 115dB (at 60Hz)) before data was collected at a rate of 1200Hz. EMG was visually inspected during data collection – if muscle activity increased during the trial then the it was repeated. During data analysis the raw EMG

signals were also full-wave rectified and dual-pass filtered using a low pass Butterworth filter (cut-off 6Hz) to create a linear envelope (LE). The LE signal was normalized to maximum voluntary contraction, using EMG data collected while subjects exerted maximum effort in quadriceps, hamstrings and gastrocnemi (see Chapter 2 for details of this process). The normalized, LE EMG was examined to ensure muscle activity was minimal during all passive motion trials. 10 out of 12 subjects exhibited EMG that was less than 5% MVC in all muscles for all passive motion trials. The two other participants (1 patient and 1 control) exhibited EMG that reached as high as 10% in some muscles. All data analysis was redone with this two subject excluded and the results did not change. The data presented below, therefore, represents all 12 participants.

Force and kinematic data at the knee were collected with the hip at 2 different angles – sitting and lying supine. Because of differences in subject anthropometry the exact hip angle (as measured using a manual goniometer) differed slighted between individuals. On average control hip angles were 98±2.6º in sitting and 178±2.6º in supine lying. Patients exhibited an angle of 101.2±6º in sitting and 174±2º in supine. The angles were not statistically different between the groups.

5.2.1 Passive Moment Calculation:

Force and kinematic data were combined as per Reiner and Edrich (1999) and used to calculate the passive knee moment. Briefly, the force required to move the shank was applied at a known distance l from the knee joint center – these data were used to calculate the total moment at the knee joint. This moment was comprised of active (muscle forces) and passive components, as well as a contribution from the effects of inertia and the gravitational moment. The active moment, which was assumed zero due to the lack of EMG activity, and the inertial moment, which was assumed to have no effect due to the slow nature of the motion, were both disregarded. The gravitational moment was calculated using the mass m and the center of gravity location (l_{CoG}) of the shank. Anthropometric values, needed for this calculation, were taken from Winter (2005). The passive moment at the knee was then estimated using the following expression:

$$M_{pas} = Fl - mgl_{CoG}\cos(\theta) \quad \textbf{(5.1)}$$

where g was acceleration due to gravity, and θ the angle as defined in Figure 5.1. Passive knee moment was then plotted against knee angle to allow for qualitative assessment of the data (see sample data in Fig 5.2).

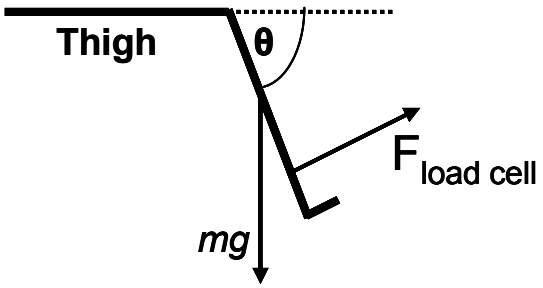


Figure 5.1: Schematic of thigh, shank and foot segments illustrating the variables used in the passive moment calculation (equation 1). mg: force of gravity acting on the shank, $F_{load\ cell}$: the force exerted by the examiner to move the knee joint, θ represents the knee angle used in the calculation.

Visual examination of knee angle data, revealed that maximum knee flexion and extension angle attained by each subject differed. To facilitate further data analysis, angle profiles were examined to determine the range of knee motion that all subjects attained during all passive motion trials (see Fig. 5.3 for illustration). The range of knee motion that met this criterion was from 15º - 55º. Subsequently, passive knee moments that occurred outside this range of knee motion were not examined as part of the current work.

The passive moment data calculated using equation (1) were a function of time – each discrete data point occurred at an equally spaced time interval. In order to average passive moment data across trials and compare between subjects, the data needed to be expressed as a function of knee joint angle. This was accomplished by fitting the passive moment data with a cubic spline (Forsyth et al. 1977). One degree angle increments were used when fitting data. Trials were then averaged within the 2 different hip angle conditions, so that each participant had 2 moment-angle curves, each representing the passive moment-angle relationship for a given hip angle.

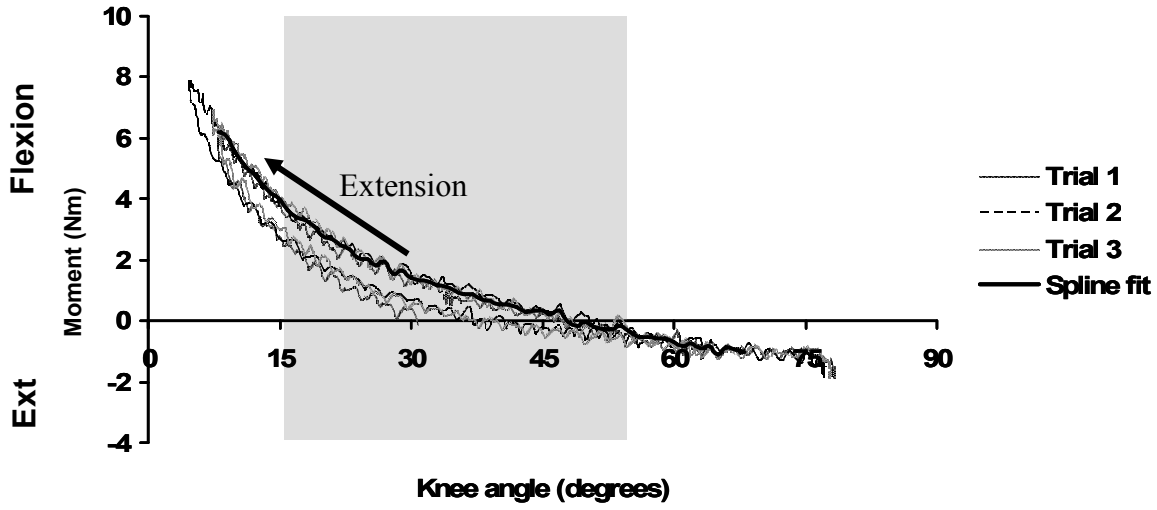


Figure 5.2: Three trials of raw experimental data (seated position) from a typical control subject. Shaded region demonstrates knee angle range used for all further analysis. ─ determined by cubic spine fit of the extension portion of the three trials illustrated. The upper portion of the curve indicates the knee extension phase of the motion. Note presence of slight hysteresis between the flexion and extension portions. The flexion portion of the curve was not used for further analysis because a majority of patients had great difficulty relaxing during this portion of the motion, rendering the movement no longer fully passive. Ext = extension.

A fourth order polynomial (see equation 5.2) was then fit to the average moment-angle curve for each hip angle as per Nordez et al (2006). The curve fitting was done using the curve fit function of Igor Pro (Wavemetrics, Lake Osewego, Oregon). As the slope of this curve represented stiffness, the polynomial was differentiated (see equation 5.3) and knee stiffness subsequently calculated for 5º increments of knee motion.

$(M(\theta)= a\,\theta^{4}+b\,\theta^{3}+c\,\theta^{2}+d\,\theta+e$, (a-e coefficients) **(5.2)**

$dM/d\theta = 4a\,\theta^{3} + 3b\,\theta^{2} + 2c\,\theta + d$ **(5.3)**

where a – e are coefficients determined during the polynomial curve fitting process and θ represented the sagittal plane knee angle. The goodness of fit produced by equation (5.2) was determined by calculating the R^2 value.

5.2.2 Statistical Analysis:

Statistical analysis was performed using SAS software. T-tests were used to assess the whether WOMAC results differed between the two groups. A 2-way repeated measures ANOVA (factors: group and hip position) was used to assess difference in both passive moment and stiffness data. To facilitate these comparisons passive moment and stiffness values were assessed at 5º intervals starting at 15º. In total stiffness and moment results were compared at 9 different points through–out the total knee range tested. This was done at both hip angles. An alpha level of p=0.05 was used.

5.3 Results

WOMAC results, as summarized in Table 5.1, indicated that members of the TKA group reported significantly more pain than controls (p=0.03) and experienced significantly more functional difficulties then controls (p=0.03). Reports of stiffness were not significantly different between the groups (p= 0.06), although patients' stiffness scores were substantially greater then that of controls (2.5 vs. 0.2, out of a possible maximum score of 8).

Patients were only able to actively flex their knees to approximately 110º (±10), in contrast to controls that had normal active knee flexion. When knee motion was assessed during the passive moment trials, average passive knee motion for controls ranged from 6.8º to 64º, while patients exhibited a slightly lower range (7.6º to 56º). These differences were not statistically significant for the start angle, however, controls did exhibit significantly greater knee flexion then patients (p = 0.04). As discussed in the methods section, only data corresponding to knee angles between 15º - 55º were used.

Some members of the TKA had difficultly relaxing when the knee was being passively moved from an extended to a flexed position. Examination of EMG levels for these participants revealed high levels of muscle activity during this portion of the movement. As a result, this portion of the data was discarded for all participants. The data presented below, therefore, represented the passive knee extension portion of the motion only.

Table 5.1: WOMAC results.

	Patient	*Control*
Pain (/20)*	3.2	**0.2**
Stiffness (/8)*	2.5	**0.2**
Physical function (/68)*	**12**	**0**

* A higher score indicates more pain, more stiffness and more deficits in physical function.

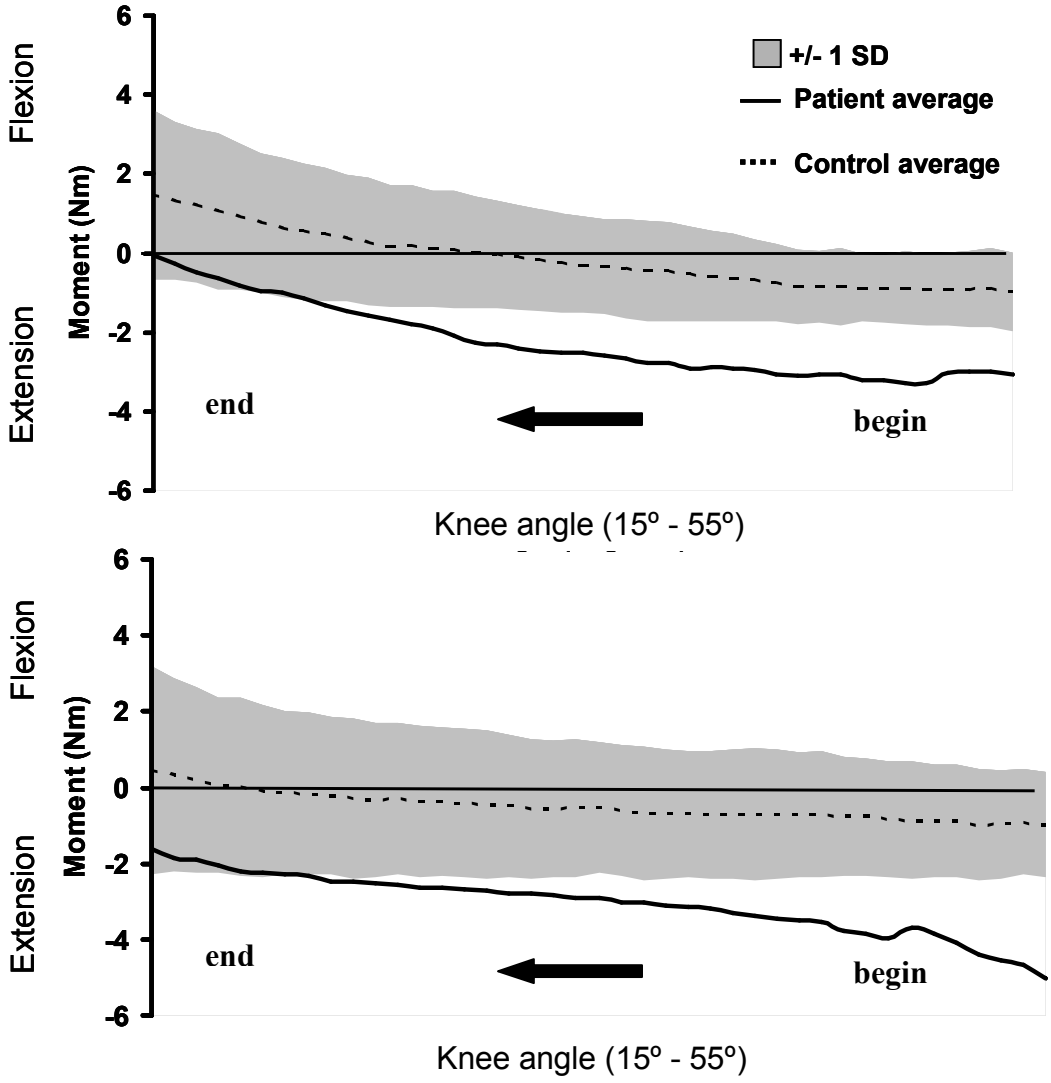


Figure 5.3: Passive knee moment in seated position (upper graph) and in supine lying (lower graph). The moments plotted represent the average passive moment acting on the knee as the knee was moved from a flexed to extended position (as determined by equation 1 in the text). Shaded region represents ± 1SD of all control results.

The average patient and control passive moment-angle relationship is depicted in Figure 5.3. Similar results are presented in Figure 5.4, however, in this case average patient and control data at each hip angle are presented on the same figure. Collectively, Figure 5.3 and 5.4 illustrate that patients and controls exhibited similar trends in passive knee moment.

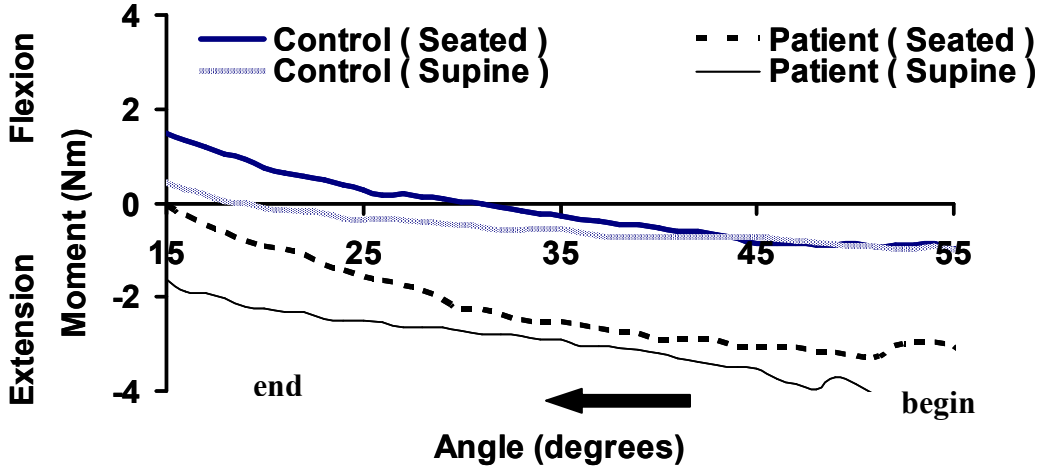


Figure 5.4: Average passive moment for patients and controls in sitting and supine. Averages based on data from all participants in each experimental group. The passive moment value indicates the amount of resistance to knee motion that is produced by passive joint structures.

Generally, however, passive moment values for patients were biased toward the extensor moment portion of the curve. The fourth order polynomials fitted to patient and control passive moment-angle data had generally high correlation values. Patient R^2 values were 0.98 ±0.03 (range 0.91 – 0.999) and 0.97±0.3 (range 0.92 – 0.996) when seated and supine respectively. Control correlations were slightly more variable and lower, with a value of 0.95±0.08 (range 0.77 – 0.996) in sitting and 0.87±0.2 (range 0.45 – 0.99) in supine. Stiffness values calculated using the differentiated polynomial equation showed that stiffness in patients and controls at the knee angles tested did not differ significantly in either sitting or supine (Fig 5.5).

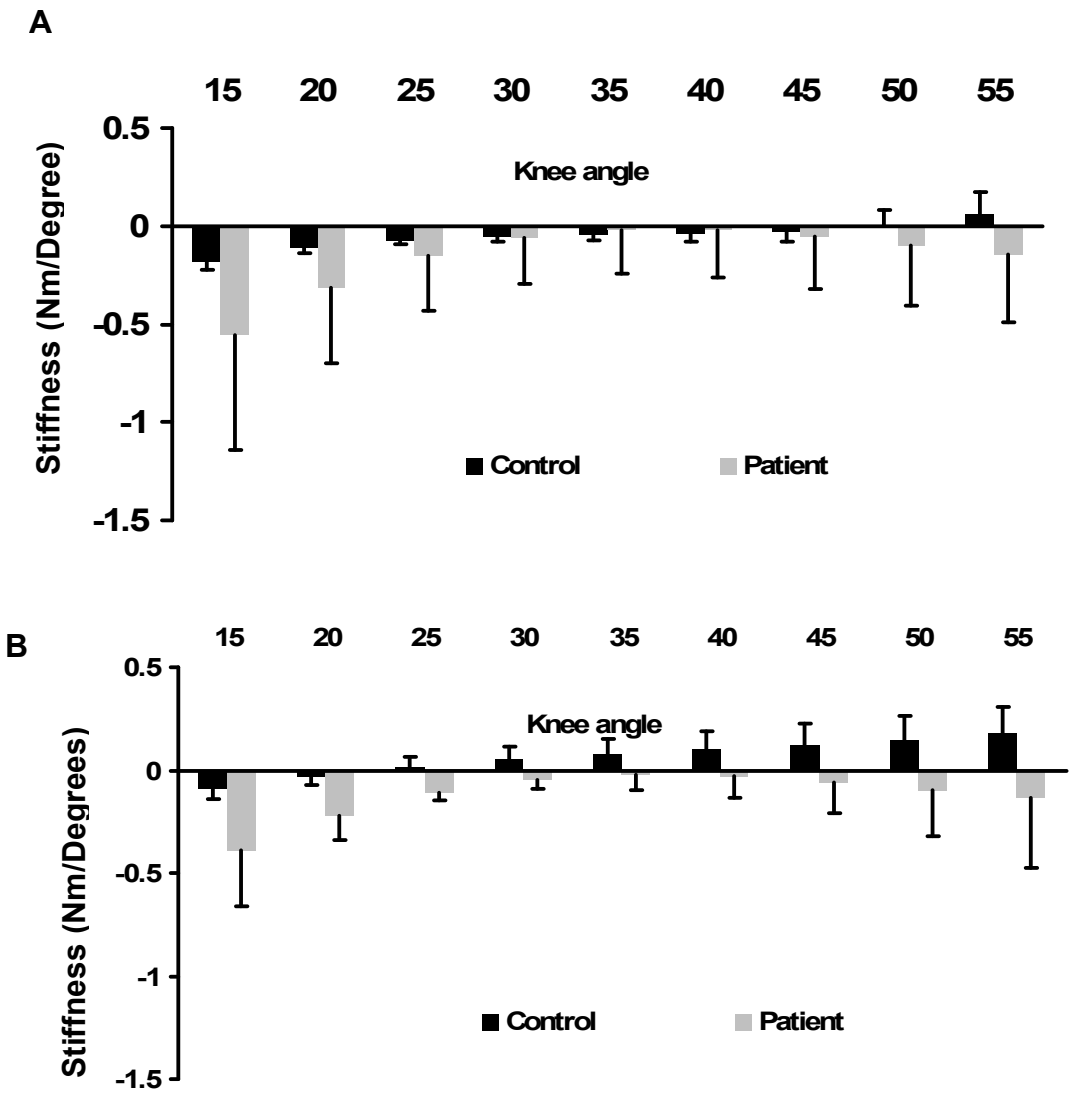


Figure 5.5: Passive knee stiffness (A: seated, B: supine) at knee angles ranging from 15 – 55 degrees. Negative values represent stiffness that is acting in the extensor direction; positive values indicate stiffness is flexor in nature. Error bars denote ±1 standard error.

5.4 Discussion

The aim of the current study was to quantify the magnitude of the sagittal plane passive knee moment in an effort to determine whether changes in this measure could account for the deficits observed in the net knee extensor moment following TKA. A secondary aim was to quantify passive knee stiffness in this population in order to determine if patient complaints on increased knee stiffness (Moffett et al. 2004) could be attributed to changes in mechanical knee joint stiffness. These goals were examined by experimentally

quantifying the passive knee moment in a small group of individuals who had undergone TKA at least 1 year ago. This passive moment data was also used to estimate passive knee stiffness in these same individuals. This research represents the first attempt to quantify in vivo passive knee moment and stiffness in this population. Results showed that the passive moment-angle relationship for both patients and controls follow expected patterns, thus suggesting no dramatic changes in the passive characteristics of replaced joints. For the small group tested, however, there appeared to be some slight differences in the magnitude of passive knee moment following knee joint replacement. Although these differences did not reach statistical significance, they do provide some initial insight into the effects of TKA passive knee resistance.

5.4.1 Passive knee moment

For the range of knee motion examined, the pattern of passive resistance exhibited by all participants was similar to patterns reported by previous authors (McFaull and Lamontagne 1998, Silder et al 2007, Magnusson 1998, Reiner and Edrich 1999). Further research, employing a greater knee range of motion, is obviously needed to determine the passive properties of the knee joint from 0 – 15º of flexion and beyond 55º of flexion. Previous research has also confirmed that the passive knee moment is substantially affected by hip position (Reiner and Edrich 1999; McFaull and Lamontagne 1998) and less so by ankle position (Reiner and Edrich 1999). As illustrated in Figure 5.4, the change from a seated to a supine position affected passive knee moments in both groups involved in the present study. As individuals went in to more hip extension, passive resistance to knee extension decreased. As others have commented (Reiner and Edrich 1999), this was thought to be due to decreased stretch on the hamstrings as the hip is extended.

While passive knee moments in both groups followed patterns similar to previous studies, there is one main trend of interest that warrants further examination. At both hip angles tested, patients exhibited greater passive knee moment than controls, particularly at knee angles between 25º and 55 º (see Fig. 5.4). While the moment produced during this

portion of the knee extension motion was extensor (i.e. the moment due to gravity exceeded the moment delivered by the load cell) it is important to recognize that moment magnitudes in this portion of the curve were very small. As such, any errors that occurred in the shank mass, CoM position, moment arm of the CoM or moment arm of the load cell could have resulted in a polarity reversal in the passive moment (i.e. a flexor moment could be estimated as an extensor moment or vice versa). For this reason, the discussion that follows does not focus specifically on the direction of the moment, but rather on possible factors that may contribute to the increase observed in members of the TKA group.

Two alternative sources of the increased resistance exist: either it was produced by muscle activation or it came from passive tissues. While EMG activity was monitored to ensure that minimal activation occurred, muscle activation was not totally absent. As a result, minimal active muscle force contributions to the passive moment likely occurred. However, since muscle activation levels did not differ between patients and controls, active muscle force could not be used to explain the observed passive moment differences between the two groups.

There are several possible sources of passive tissue moments that may have contributed to this increased midrange resistance. All patients in the study demonstrated deficits in active knee flexion (107º +/- 10º). While the amount of active flexion exhibited by patients was within the limits required to perform the tasks examined, it was substantially less then the typical maximum range for knee flexion (~140º). This restriction in active knee motion is not uncommon following TKA (Victor and Bellemans 2006; Dennis et al. 1998a). Dennis et al (2007) and Victor and Bellemans (2006) have both reviewed possible reasons for limited active knee motion in this population. While patient characteristics (i.e. preoperative knee range and adherence to post-operative physiotherapy programs) could affect post-operative knee motion (reviewed in Dennis et al. 2007), the factor which has received the most attention is the post-operative tibiofemoral kinematics that occur to produce knee flexion and extension (Victor and Bellemans 2006; Dennis et al. 2007). Many investigations have determined that tibio-

femoral kinematics differ post knee replacement (Long et al. 1996; Rhoads et al 1993; Uvehammer et al. 2000; Dennis et al. 1998b). While the specifics of these differences vary depending on the type of prosthesis used, Victor and Bellemans (2006) suggest that abnormalities in femoral rollback, femoral rotation and posterior condylar offset can all result in altered knee motion post-TKA. Although the focus of the Dennis and Victor papers was on the effect of altered tibiofemoral kinematics on knee flexion range, it could be argued that alterations in tibiofemoral kinematics would invariably affect resistance to knee motion. If the tibia and femur are moving in a non-normal manner with respect to one another, then increased resistance to motion may result. As such, the increased moment exhibited by members of the TKA group in the 25º to 55º range of knee flexion could be due to alteration in tibiofemoral joint kinematics. Alternatively the increased passive moment could be due to tension in passive tissues. This was unlikely, however, because in this range of knee motion, there would be minimal stress placed on passive tissues (McFaull and Lamontagne 1998)

It was also plausible that friction could have contributed to the slight changes in passive resistance observed in the current study (Zhilmann et al 2005). In healthy joints the effects of friction are thought to be minimal due to the very low coefficient of friction afforded by hyaline cartilage and synovial fluid (Sathasivam and Walker 1997). The higher coefficient of friction in replaced joints (0.03 – 0.1 compared to 0.002 – 0.03 in healthy joints) has led several authors to suggest that friction should not be ignored when examining prosthesis function (Wimmer and Andriacchi 1997; Sathasivam and Walker 1997; Zilhmann et al 2005). Can friction help explain the increased passive moment observed in patients in the current study? In order for friction forces to occur, the joint surfaces must be subjected to a compression (or normal) force. In synovial joints, compression forces can be created by body weight (as is the case in weight bearing activity), muscle contraction or, to a lesser extent, tension in ligaments. The passive motion performed in the current study would therefore result in very little compressive forces since the activity was non-weightbearing and muscle contraction was minimal, making it unlikely that frictional forces would be a factor.

Irrespective of the mechanism by which the slight increase in passive knee moments were generated in members of the TKA group a primary aim of the current work was assess the potential impact of these changes on the net knee extensor moment in these individuals. Numerous investigations have demonstrated that knee extensor moment is reduced during gait (Andriacchi et al 1982, Wilson et al 1996, Simon et al 1983), stair climbing (Andriacchi et al 2003,Wilson et al 1996 and Catani et al 2003), and stepping on to a raised surface (Draganich et al 1999, Byrne et al 2002) following TKA. As the net moment is a function of both passive and active forces, a change in passive resistance could potentially affect the resultant moment. To determine if the observed changes would have an affect the net moments of individual patients were compared with their passive knee moment profiles (see Table 5.2 below). The results of this analysis showed that while passive moment magnitudes for patients ranged from -4Nm to 9Nm, in 4 of the 5 patients, the moment deficit ranged between 16 – 39Nm. This suggests that the passive moment changes observed in patients would not have been large enough to account for the relatively large knee moment deficits. There was one exception however. Subject one had only a 2Nm moment deficit. This small magnitude deficit could have been due to an increase in passive moment.

Table 5.2: Passive and active moment data for all members of the TKA group. Peak moment: peak active moment during MS of gait; Control-patient: average control peak active MS moment – patient peak active MS moment; Passive moment; range of passive moment values for individual members of TKA group.

	Peak moment *	**Control – patient** **	**Passive moment****
Subj 1	35Nm	2Nm	-3Nm to 7Nm
Subj 2	-2Nm	39Nm	-4Nm to 1Nm
Subj 3	11Nm	26Nm	-4Nm to 9Nm
Subj 4	21Nm	16Nm	-3Nm to -1.5Nm
Subj 5	NA	NA	-2Nm to 1Nm
Subj 6	16Nm	21Nm	-4Nm t0 3Nm

* during MS phase of gait

** Average control peak MS moment (37Nm) – patient peak MS moment

*** passive moment range observed between 15º and 55º

In considering the analysis presented in Table 5.2, it must be recognized that this only applies when the knee is flexed more the 15º. Near full knee extension, where others have reported passive moments of between 10 – 20Nm (Reiner and Edrich 1999; Silder et al 2007), the relatively high magnitude passive moment would certainly have the potential to substantially reduce the net knee extensor moment. Given this fact, further investigations of passive knee moment in the terminal portion of knee extension (i.e. 15º - full extension) needs to be undertaken with the TKA population.

5.4.2 Passive knee stiffness

In addition to exhibiting greater passive moments (from 25º - 55º), patients' knees also appear to be stiffer than those of controls (Figure 5.5). While passive moment quantifies the magnitude of the moment either contributing to or opposing passive motion, stiffness provides insight into how this moment changes as a function of displacement (in this case joint angle). It is represented by the slope of the force (moment) vs. displacement (angle) curve. This finding agreed with the results of the WOMAC analysis. Although WOMAC results from the present study indicated that members of the TKA group did not report more stiffness than controls, the trend certainly existed (control stiffness 0.2/8; patients 2.5/8).

The approach used to quantify knee stiffness in the current study was similar to the one used by Nordez et al (2006). These authors reported stiffness magnitudes of approximately 0.25 Nm/º at 65º knee flexion and 0.45Nm/º at 45º of knee flexion. This compares to stiffness values (at 45º of knee flexion) of 0.05Nm/º (patient group) and 0.03Nm/º (controls) in the current study. Comparison of stiffness values between the two studies is complicated by that fact that Nordez's group quantified knee stiffness with the hip in 60º of flexion, compared to the current study where the subject were seated in a relaxed sitting position (i.e. hip ~ 90º flexion). Compared to relaxed sitting, a hip angle of 60º would place the hamstrings on greater stretch, therefore resulting in increased passive resistance to knee extension. This may account for the higher stiffness values observed in the Nordez study when compared to the current work.

5.4.3 Limitations:

It is important to recognize that there are several limitations for this current work, with the primary limitation being the small sample size examined. Because of these small numbers, results cannot be generalized to a larger population. Despite this fact, the study does provide initial evidence regarding the passive motion properties of replaced knee joints, and it provides a strong foundation upon which future research can be based. Additionally, the 15º - 55º knee range of motion examined provides no insight into passive resistance in extreme extension and flexion. To have a complete picture of the passive behaviour of replaced knee joints, these ranges must also be examined and will be the basis of future work.

Testing participants in a gravity dependent position created two primary issues for data collection and analysis. This testing position appeared to make it difficult for participants to relax during the passive knee motion. Through EMG monitoring, trials that were not truly passive were omitted. As the majority of participants had great difficulty relaxing during the extension to flexion portion of the motion, only data from the flexion to extension phase could be analyzed. As the flexion and extension portions of the motion are normally averaged (see Reiner and Edrich 1999) to determine the moment-angle curve, the approach taken in the current study would likely result in a slight over estimation of passive resistance. As the same approach was taken for both patients and controls, this over estimation would occur with both groups and is unlikely to have adversely affected the results. Testing in a side lying (non-gravity dependent position) would help eliminate this problem.

Testing in a gravity dependent position also meant that the effects of gravity had to be accounted for when determining passive moment. Any errors in estimating this force, or its moment arm would ultimately impact the passive moment. The use of skin based markers to determine segment kinematics and the moment arm of the force of gravity meant these measures were potentially affected by errors due to skin motion artifact (see Chapter 2). We are confident that these errors are low however, due to the use of singular value decomposition, which has been shown to reduce error in kinematic data (Challis

1995). Additionally, because the limb motion was so slow, it is anticipated that the amount of error introduced by skin motion would be minimal.

5.4.4 Conclusion

In conclusion, the current study represents one of the first attempts to quantify passive knee moment and stiffness following total knee arthroplasty. While the general shape of the passive moment-angle curve is similar for patients and controls, members of the patient group exhibit slight increases in both passive knee moment and knee stiffness. The magnitude of passive moment changes are unlikely to be large enough to account for deficits in the net knee extensor moment often found in this clinical population. That having been said the changes may account for increased complaints of knee stiffness in this population. Clearly, considerable more work needs to be done in this area to better elucidate the effects of TKA on passive knee moment and stiffness and to also gain a better understanding of the potential effects of these differences on knee function.

Chapter 6

In vivo moment arms following total knee arthroplasty: methodological issues.

"In the task of helping an injury repair itself … we must understand the mechanical principles that make the joint work in the first place… The **muscle moment arm** is one of the basic principles essential to this understanding". Burford et al. 1997

6.1 Introduction

As discussed preciously, the net joint moment is comprised of both active and passive components. Given the relatively large magnitude of the active vs. the passive moment (see Chapter 5), the dominant factor in the production of the net knee moment is active moment produced by muscles. These active moments are a function of both the muscle force and that forces moment arm. This is illustrated clearly by the following equation:

$$Moment = \underline{r} \times \underline{F} \quad \textbf{(6.1)}$$

where $\underline{r}$ is a vector from the axis of rotation to some point on the line of force $\underline{F}$. Using the definition of vector cross product, equation 1 can be further simplified to:

$$Moment = |r||F|\sin\theta \quad \textbf{(6.2)}$$

where ϴ is the angle between r and F and $|r| and |F|$ represent the magnitude of $\underline{r}$ and $\underline{F}$ respectively. Application of basic trigonometry shows that $|r|\sin\theta$ is the perpendicular distance from the axis (or center) of rotation to the line of force – it is the moment arm of force F.

Given the influence of moment arm length on a muscle's moment production ability, it seems obvious that any efforts aimed at improving understanding of knee moment deficits following TKA must examine the effects of the surgery on moment arms of the knee musculature. Following TKA, both joint geometry (D'Lima et al 2001) and kinematics (D'Lima et al 2001; Karrholm et al. 1994) are altered. These changes can alter the muscle line of pull and/or the relationship between the muscle and the axis of rotation, thereby effecting moment arm lengths.

Despite this potential for moment arm changes and the possible impact of these changes on knee function post-TKA surprisingly few researchers, have attempted to quantify how moment arm lengths are affected by knee replacement surgery. Instead, researchers have

attempted to explain observed knee moment behaviour based on what the moment arm is 'expected' to be doing given knowledge of joint geometry and contact point motion (i.e. see Draganich et al 2002; Andriacchi et al 1982). The investigations of moment arm following TKA that have been carried out have generally been done *in vitro* (D'Lima et al. 2001; Ostermeier et al. 2006; Brown et al. 2005) or using finite element models of the knee (Pal et al. 2007), thereby limiting the applicability of the results to *in vivo* situations. The intent of the current research was, therefore, to quantify *in vivo* moment arms of the knee, in an attempt to determine if moment arm changes could account for (or at least contribute to) alterations in knee extensor moments in this population.

Researchers have devoted considerable effort towards quantifying moment arms in lower limb muscles. As outlined by Herzog and Read (1993), most of this moment arm research has been closely linked with efforts to estimate individual muscle forces. Although other approaches are possible, knowledge of moment arm length is needed in order to determine individual muscle forces using an inverse dynamics based approach. The ability to estimate individual muscle forces is integral to performing forward simulation analyses and quantifying loads experienced by joints. While the importance of these areas of research cannot be disputed, moment arms calculated for these purposes are often influenced by the need to ensure moment equilibrium equations can be solved (see Tasaopolous et al. 2006). As such the calculated moment arms likely did not represent the effectiveness of a given force to produce a moment about some functional joint axis. An example of this is the practice of determining quadriceps moments arms with respect to the tibio-femoral contact point (TFCP) – a point used to estimate the point of contact

between the tibia and femur (see Tsaopoulos et al. 2009; Kellis and Baltzopoulos 1999; Wretenberg et al. 1996). As reviewed by Tsapolous et al. (2006) the TFCP is often used to simplify the moment equilibrium equations. If the joint shear and compression forces are assumed to act through the TFCP, they will not contribute to the net moment therefore removing them from the equations. While this approach may be perfectly valid for the chosen application, knee joint motion does not occur about the TFCP. Moment arms calculated with respect to this point will therefore not be accurate.

Key then, to the work being undertaken for this thesis, was to choose a method of moment arm calculation that produced results that represented, as closely as possible, what was actually happening in the knee joint. In other words, to find a method that produced moment arms indicative of effectiveness of a given force to produce a moment about some functional joint axis. This task proved to be challenging and numerous methods were tried with mixed results. What follows below is a description of the process and some of the results. As will be described in detail below, in the end, no satisfactory approach for moment arm estimation was found. Despite this lack of conclusive results, the decision was made to include this work in the current thesis because it was felt that the work done and the issues raised form the basis for future work in this area. Also, to not include a discussion of moment arms in this work would be to ignore a key component known to contribute to the overall net knee moment.

6.2 Background: Methods for determining moment arm

A seminal paper reviewing MA calculation methods was published by An et al in 1984. In this paper, An provided a detailed overview of the 3 primary methods that can be used to calculate a muscles MA – the geometric method (GM), tendon excursion method (TEM) and direct load measurement method (DLMM). In a more recent review, Tsaopoulos et al. (2006) focused specifically on the issues and challenges surrounding MA determination at the knee joint.

6.2.1 Tendon excursion method (TEM):

The TEM was derived using the principal of virtual work (An et al. 1998) and simply put states that a muscles moment arm is equal to the change in muscle length over the change in joint angle. While detailed derivations are available elsewhere (Yamaguci 2001) briefly the TEM method can be understood as follows:

- If we assume that the no work is done moving the knee (i.e. the work of shortening equals the work of rotation, no energy storage in joint structures) then:

  $$(\text{Force})(\Delta l) = (\text{Moment})(\Delta\theta) \qquad \textbf{(6.3)}$$

  Where Δl is change in muscle length, $\Delta\theta$ change in joint angle and force is the force producing the change in length

- If the moment is assumed to depend only on the muscle force (i.e. inertial and gravitational forces can be ignored) then:

  $$\text{Moment} = (\text{Force})(\text{MA}) \qquad \textbf{(6.4)}$$

  (MA = moment arm)

- Rearranging equation 6.2 and 6.3 leads to the basic equation used in the TEM method:

$$MA = dl/d\theta \quad \textbf{(6.5)}$$

- The method is also based on the assumption that the motion being examined is planar.

The three assumptions above necessitate that this method be applied ideally to situations where:

- No muscle activation is present (Tsaopoulus et al 2006): muscle contraction will result in energy storage in elastic tendons and other tissues and violate a main assumption of this method.
- Any motion that does occur must be quasistatic to ensure that inertial forces are minimal (Yamaguchi 2001).
- The motion that occurs must be planar.

Despite these assumptions, the simplicity of this approach has resulted in it being used by numerous researches to quantify moment arms of muscles crossing the knee joint (for example: Arnold et al 2000; Visser et al. 1990; Spoor et al 1990; Burford et al. 1997 and 2001).

In the early 1990's, with the introduction of SIMM, an interactive graphics based musculoskeletal modeling package (Delp et al. 1990), an application of the TEM referred to as the 'partial velocity' method (PVM) began to be used quite extensively. The PVM

appears to have the advantage that it can be used to determine MAs in situations where motion is not purely planar. Since the extension motion of the knee is accompanied by rotation about the longitudinal axis of the tibia during the terminal 20º of extension (Johal et al. 2005), knee motion cannot be considered purely planar. This makes the PVM appear to be particularly well-suited for moment arm investigation at the knee.

6.2.2 Direct load measurement (DLMM):

As the name suggest the DLMM determines moment arm length by directly measuring the forces and moments acting on segments. It uses these values in force and moment equilibrium equations to determine the moment arm of individual muscles (An et al. 1984). It can be used in both 2D and 3D motion, however, it is not suited to *in vivo* measurement as muscle forces must be determined directly – a task that though possible it not practical for most human motion. Because of this limitation it was not considered appropriate for the current study and will not be discussed further.

6.2.3 Geometric method (GM):

When using the GM, MAs are determined by calculating the perpendicular distance from some origin of rotation to the line of action of the muscle. For 2D motion, the origin of interest is some point about which rotation is assumed to occur, while in 3D analyses, rotation is assumed to occur about an axis.

Researchers have used numerous origins (or centers) of rotation when employing the 2D GM approach. At the ankle, Magnaris (2004) has used the instant center of rotation. Knee moments arms have been determined using centers of rotation that have included the

tibiofemoral contact point (Herzog and Read 1993; Yamaguchi and Zajac 1989; Kellis and Baltopoulos 1999; Tsapoulos et al. 2007; Wrentenberg et al. 1996), the anterior and posterior cruciate ligament intersection point (Gill and O'Connor 1996; Imran et al 2000) and the geometrical center of the posterior femoral condyles (Tsapoulos et al. 2009). Each of the above origins of rotation has its advantages and disadvantages (see Tsaopoulus et al 2006 for a review), however, all will result in errors, when the motion being studied is not purely planar (Maganaris, 2004). As reviewed above, such is the case during knee extension.

For 3D applications of the GM, motion occurs about one (or several) axes of rotation. As the current work was primarily concerned with sagittal plane knee motion, the axis of interest was the flexion/extension axis. The location of this axis has been the focus of much research and discussion over the past 160 years (see Leyvraz and Raktomanana 2000). Up until the early 1990's knee flexion/extension was said to occur about a variable flexion/extension axes that was located in the posterior femoral condyles and was perpendicular to the sagittal plane (See review by Smith et al. 2003). Work by Hollister et al (1993) challenged this concept, and suggested instead that sagittal plane knee motion occurred about a fixed axis. Using cadaver knees and a devise referred to as an axis finder, Hollister described the axis as passing through the origins of the medial and lateral collateral ligaments (i.e. does not lie purely in the sagittal plane). Alternatively, it has been suggested that the flexion/extension axis is not purely transepicondylar, but rather that it projects distal and posterior to the medial epicondyle (Freeman and Pinskerova 2003). Determining the axis of knee motion is further complicated by the apparent

longitudinal rotation that accompanies sagittal plane knee motion, particularly during terminal extension (Johal et al. 2005).

Perhaps partly due to this lack of agreement on knee joint axes location, very little research has attempted to quantify 3D moment arms of knee musculature. That which does exist has computed the patellar tendon moment arm as the common perpendicular between the helical axis and the muscle line of action (See Boyd and Ronsky 1998; Krevolin et al. 2004; Sheehan 2007b; Wilson and Sheehan 2009). Helical motion, which occurs about the helical axis, is the simplest motion path describing rigid body motion (Shiavi et al., 1987), and represents rotation about and translation along the helical (screw) axis. Numerous authors have used the helical axis to quantify knee motion in a variety of populations (Hart et al. 1991; Ramakrishnan and Kadaba 1991; Jonsson and Karrholm 1994; Sheehan et al. 2008).

Irrespective of whether 2D or 3D motion is being examined, the GM requires that the origin / axis of rotation and the line of action of the muscle be identified as accurately as possible – this requires the collection of kinematic data. Researchers have used a variety of methods ranging from magnetic resonance imaging (Sheehan 2007b), video fluoroscopy (Kärrholm et al 1994; Kellis and Baltzopooulos, 1999; Tsaopoulus et al. 2009), or embedded and skin based marker systems (Boyd and Ronsky 1998; Krevolin et al. 2004, Dhaher and Francis 2006). While most initial work in this area was done using cadavers (Krevolin et al. 2004; Herzog and Read 1993; Boyd and Ronsky 1998 (using cats)) more recent work has focused on determining in vivo measures in humans

(Sheehan 2007a; Kärrholm et al 1994; Kellis and Baltzopooulos, 1999; Magnaris 2004; Tsaopoulus et al. 2009).

6.2.4 Summary:

Two factors were considered in deciding which method to use for moment arm calculation in the current study.

- The method had to determine moment arm length with respect to a center or axis of rotation that corresponded to knee joint kinematics (i.e. knee motion does not occur around the TFCP, so it would not be considered appropriate).
- Ideally, the method should account for the 3D motion that is known to occur at the knee joint.
- The method had to have been previously used to determine moment arms at the knee joint (this would ensure that results of the current could be partially validated by comparing to previously published work).

Based on the review of literature and the criteria established above it was decided to use the partial velocity method (PVM) (Delp and Loan 1995). This method, an application of the tendon excursion approach, has been used extensively in the literature to determine moment arms at numerous joints (Lloyd and Besier 2003; McClean et al. 2004; Buchanan et al. 2005; Besier et al. 2009; Arnold and Delp 2001). Based on descriptions provided by Delp and Loan (1995) it also appeared to take in to account the 3 dimensional nature of knee motion. One limitation of using this approach was that the TEM assumes no elastic storage of energy (i.e. no active muscle contraction). This would obviously not be the

case during active motion. Despite the inability to meet this assumption, the decision was made to use this approach.

6.3 Methods

Characteristics of the subjects examined this study have been previously described in Chapter 2.

In order to determine moment arm lengths, 3D kinematic data for the shank and thigh were required. This was done using methods identical to those described in Chapter 4 and so will not be repeated in detail. Briefly, (X,Y,Z) coordinate data from clusters of 4 markers attached to both the shank and thigh were collected using an Optotrak (NDI, Waterloo, ON) active marker system. Data collected during a standing calibration trial were used to determine the relationship between markers and the underlying rigid bone. This relationship was subsequently used to determine shank and thigh position and orientation (pose) during movement trials. Details of how this information was used in moment arm calculation are provided below.

The motion chosen for analysis was seated knee extension. This task was identical to the one used in Chapter 4, however, for the current work, only non-weighted knee extension trials would be examined.

6.3.1 Partial velocity method

The details below were based on Delp and Loan (1995). Using the 'partial velocity' method the moment arm of a muscle was defined as:

$$MA = {}^{S}V^{Pm} \bullet \hat{f} \tag{6.6}$$

where $\hat{f}$ was the unit vector that represented the line of action of the muscle of interest (in this case the patella tendon) and ${}^{S}V^{Pm}$ was the partial velocity of point P_m (some point on the muscle line of action) and S was the locally defined anatomical reference frame of the shank (see Figure 1). The partial velocity, ${}^{S}V^{Pm}$ of any point P_m was given by:

$${}^{s}V^{Pm} = \underline{u} + (\underline{\omega} \times \underline{r}) \tag{6.7}$$

where $\underline{r}$ position vector of point P_m with respect to the thigh reference frame and $\underline{u}$ and $\underline{\omega}$ are the the linear and angular components of the partial velocity respectively.

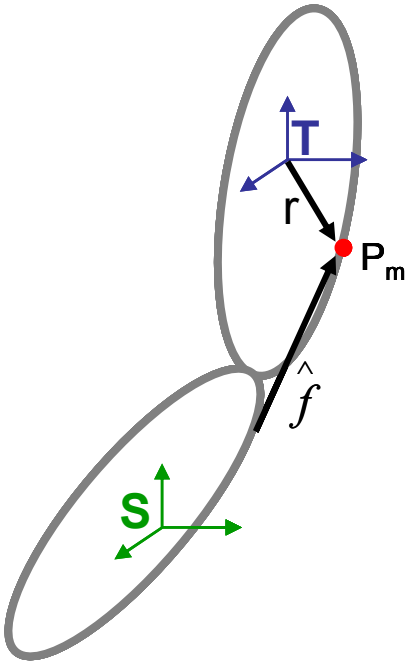


Figure 6.1: Reference frames T and S were the anatomical frames of the thigh and shank respectively. Vector *r* represented the position of muscle insertion point Pm with respect to the reference frame T. Vector $\hat{f}$ was the unit vector that represented the line of action of a representative muscle. The orientation and position of segment T with respect to segment S were represented by 3 rotational ($q_{1\text{-}3}$) and 3 translational ($q_{4\text{-}6}$) generalized coordinates. (Figure adapted from Delp and Loan 1995).

The translational component of the partial velocity, $\underline{u}$, was defined as:

$$\underline{\mu} = (\partial tx/\partial q_i)\,x + (\partial ty/\partial q_i)\,y + (\partial tz/\partial q_i)\,z \qquad \textbf{(6.8)}$$

where tx, ty and tz represented the translations along axis x, y and z respectively, between the two body segments of interest (in this case the thigh and shank) and q_i represented the generalized coordinates (3 translations and 3 rotations) of the joint in question. The rotational component of the partial velocity, **ω,** was defined as:

$$\underline{\omega} = (\partial r_1/\partial q_i)\,x + (\partial r_2/\partial q_i)\,y + (\partial r_3/\partial q_i)\,z \qquad \textbf{(6.9)}$$

where r_1, r_2 and r_3 represented the rotations about axes x, y and z and q_i has been previously defined.

For the current study, the translational component of the partial velocity ($\underline{u}$) was (0,0,0) for each of the rotational generalized coordinates (i.e. translational motion was independent of rotational motion – see Appendix A5). Given the zero value of $\underline{u}$ it did not contribute to the partial velocity of point P_m, resulting in equation (6.7) being reduced to:

$${}^{s}V^{Pm} = \left(\underline{\omega} \times \underline{r}\right) \qquad \textbf{(6.10)}$$

Substituting equation (6.10) into equation (6.6) produced the following formula for calculating moment arm length:

$$MA = \left(\underline{\omega} \times \underline{r}\right) \bullet \underline{\hat{f}} \qquad \textbf{(6.11)}$$

The rotational partial velocity component, $\underline{\omega}$, was computed to equal (1,0,0), (0,1,0) and (0,0,1) for the 3-rotational degrees of freedom of the knee joint (see Appendix A5 for sample calculations**)**. Using the properties of the scalar product [a·(b x c)=b·(c x a)] equation (6.11) was simplified to:

$$MA = \underline{\omega} \bullet \left(\underline{r} \times \hat{\underline{f}} \right) \qquad \textbf{(6.12)}$$

Given the values for $\underline{\omega}$ determined above, equation 6.12 can be simplified to:

$$\underline{MA} = \underline{r} \times \hat{\underline{f}} \qquad \textbf{(6.13)}$$

The magnitude of the MA vector therefore represent the moment of the force as determined using the partial velocity method.

As Figure 6.2 illustrates, application of the PVM method to the knee model used in the current study would be identical to using a 2D geometric approach.[1] This was an unexpected finding. The PVM had been chosen because, initial examination of the methods appeared to account for the 3D motion of the joint – clearly application of the method to the current knee model did not do this. This led to a re-evaluation of the approach used for moment arm calculation.

[1] In Delp and Loan's (1995) use of the PVM, they defined the anterior/posterior translation of femur with respect to tibia as a function of the sagittal plane knee angle. Because of this link, the translation and rotational components of knee motion were no longer independent. As such, the translational component of the partial velocity would have a magnitude. As a result equation 6.6 couldn't be simplified as it was above and the moment arm calculated using the PVM would differ from r x f.

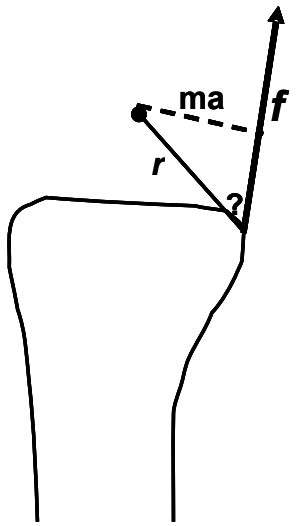


Figure 6.2: Schematic of moment arm calculation using $\underline{r} \times \hat{\underline{f}}$ where $\underline{r}$ is the position vector from some point on the femur to a point on a given muscle; $\hat{f}$ is the unit vector representing the line of action of a given muscle; θ is the angle between *r* and $\hat{f}$**.** The magnitude of the resulting cross product will equal: $|\underline{r}|\left|\hat{\underline{f}}\right|\sin\theta$. With $\left|\hat{\underline{f}}\right| = 1$ ($\hat{f}$ is a unit vector), this magnitude will therefore equal: $|\underline{r}|\sin\theta$. As is shown in section 6.5, this is precisely the formula that would be used to determined MA length using the 2D GM.

6.3.2 Moment arm calculation: Attempt #2

The decision was then made to determine muscle moment arm length with respect to the finite helical axis of the knee joint – an application of the 3D GM. Sheehan (2007a and 2007b) recently used this method to determine the moment arm of the patellar tendon during knee flexion /extension. To facilitate comparison of results with those of Sheehan, the decision was also made to only calculate the moment arm of the patellar tendon, as opposed to moment arms for all knee musculature. Given that the forces exerted by the quadriceps are transmitted to the tibia via the patellar tendon, making the moment arm of

this structure key to active extensor moment production, this simplification was felt to be justified.

Motion of any rigid body can be thought of as some combination of rotations about and translations along a given axis in space (de Lange et al. 1990). This axis is referred to as the instantaneous (for continuous motion) or finite (when described using finite position data recorded experimentally) helical axis. Finite helical axis (FHA) orientation and position, two quantities needed for moment arm calculation using this approach, are not easy to accurately determine during *in vivo* studies. The primary problem encountered is that determining helical axis orientation and position requires the differentiation of kinematic data, therefore, making it very susceptible to the noise inherent in this type of data (Woltring et al. 1985; de Lange et al. 1990; Cheze et al. 1998). Despite this known limitation, the decision was made to attempt this calculation. In light of the efforts which had been made to reduce noise in the kinematic data (see Chapter 2), it was hoped that noise would not negatively impact MA results.

Detailed background and methods for determining the finite helical axis in human motion can be found in Woltring et al. (1985). For the purposes of the current work, methodological details presented below were taken from Sheehan et al. (2007a and 2007b) and Boyd and Ronsky (1998). The first step in determining patellar tendon moment arm with respect to the finite helical axis (FHA), was to find the orientation and location of the FHA at each time interval of interest. This was done by calculating the relative angular velocity ($\underline{\omega}$) of the tibia with respect to the femur**.** For the current

investigation of patellar tendon moment arms, $\underline{\omega}$ represented the relative angular velocity of the tibia with respect to the femur. It was determined by finding the skew symmetric matrix (S) as indicated below:

$$S = \begin{bmatrix} 0 & -\omega_z & \omega_y \\ \omega_z & 0 & -\omega_x \\ -\omega_y & \omega_x & 0 \end{bmatrix} = \left(\dot{R}_{T2F}\right)\left(R_{T2F}\right)^T \quad \textbf{(6.14)}$$

where R_{T2F} was the orientation matrix that indicated the relative orientation of the tibia with respect to the femur. It was determined by computing the X,Y,Z Euler angles that described the relative orientation of the tibial anatomical frame with respect to the femoral reference frame (see Chapter 2 for details). $\dot{R}_{T2F}$ represented the first derivative of R_{T2F}. It was calculated by using the finite central difference method to determine the first derivative of each element of R_{T2F}. The relative angular velocity, **ω,** was subsequently obtained from the components of the skew symmetric matrix (S) found in equation (13) such that $\underline{\omega} = \lfloor \omega_x \quad \omega_y \quad \omega_z \rfloor$. Because the FHA is ill-defined as **ω** approaches zero, data points were ω was <0.3rad/s were eliminated from further analysis (Sheehan 2007a).Once **ω** was known the position vector, vec_1, locating the FHA with respect to the origin of the tibial reference frame (see Fig 6.3) was calculated using procedures outlined in Boyd and Ronsky (1998) and Sheehan (2007a):

$$\underline{vec_1} = \frac{\hat{\underline{\omega}} \times \underline{\mu}}{\left\| \underline{\omega} \right\|^2} \quad \textbf{(6.15)}$$

where $\hat{\underline{\omega}}$ is the angular velocity unit vector (ω/||ω||) with ||ω|| being the magnitude of the angular velocity vector ω. The variable μ represented the relative linear velocity of origin of tibial system (i.e. CoM of tibia) expressed with respect to femur. This value was determined using the linear velocity of shank and thigh CoM, which were easily determined during 3D kinematic calculations described in Chapter 2.

Once the position vector for the FHA, vec_1, was known and using vec_2, the position of the tibial tubercle in relation to the origin of the tibial reference frame, $\underline{r}$, the location of the FHA in relation to a point on the patellar tendon (ie. the tibial tubercle) was determined using:

$$\underline{r} = \underline{vec_2} - \underline{vec_1} \tag{6.16}$$

The common perpendicular between the FHA and the patellar tendon (ie. the moment arm) was then found using:

$$MA = \frac{\hat{\underline{F}} \times \hat{\underline{\omega}}}{\left\| \hat{\underline{F}} \times \hat{\underline{\omega}} \right\|} \bullet \underline{r} \tag{6.17}$$

where $\hat{\underline{F}}$ was the unit vector defining the line of action of the patellar tendon. (See Appendix A6 for details on how patellar tendon orientation was determined). As detailed by Sheehan (2007b), the MA calculated using this equation represented the component of $\underline{r}$ that was along the common perpendicular (calculated using the cross product portion of equation 16) of the patellar tendon line of force ($\hat{\underline{F}}$) and FHA ($\hat{\underline{\omega}}$).

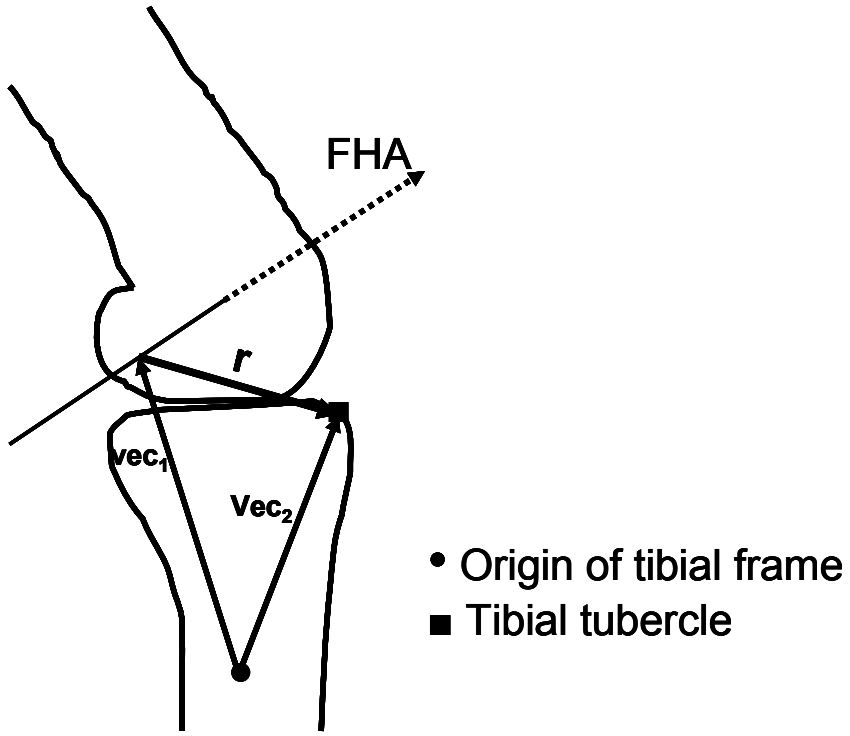


Figure 6.3: vec_1: the position vector of the FHA with respect to origin of tibial reference frame; vec_2: the position vector indicating location of tibial tubercle with respect to origin of tibial refernce frame; $\underline{r}$ a position vector defining FHA location in relation to some point on the patellar tendon (ie. tibial tubercle).

6.4 Results and discussion: Finite helical axis method

Before presenting moment results for this section, recall that FHA orientation (i.e. as represented by **ω**) becomes ill-defined as **ω** approaches zero. To avoid this problem any time interval when **ω** <0.3rad/s were not included in the analysis. This restriction had minimal affect on calculation for control subjects – once knee motion had begun they maintained angular velocity measures of >0.3rad/s until near the end of knee extension. Several patients, however, exhibited intermittent periods during the knee extension motion when **ω** dropped below 0.3rad/s. As a result, FHA orientation, and therefore MA length, were unable to be determined at these instants in time. Examination of the

moment arm results obtained for patients and controls are provided in figure 6.4. The data in this figure represents moment arm data which has been spine fit to allow averaging across like knee angles. Each curve in figure 6.4 showed the moment arm calculated at the knee angles indicated, averaged across three knee extension trials for each participant. Note the limited knee range over which some of the patient moment arm data was calculated. In all cases, this limited range was due to angular velocity measures dropping below the level needed to calculate FHA orientation.

Qualitatively, patellar tendon moment arms for patients and control did not appear to differ markedly. Also the general trend matched published reports in that greater patellar tendon moment arm lengths occurred as the knee became more extended. In all cases, however, moment arm magnitudes were higher then results presented by Sheehan (2997b), on which the current work was based. In addition, the shape of the curves appeared more undulating then was expected – again a difference from the work of Sheehan (2997b).

There were several limitation and potential sources of error that must be recognized when interpreting these results:

1. FHA orientation and position was known to be particularly susceptible to noise in kinematic data (Woltring et al. 1985; de Lange et al. 1990; Cheze et al. 1998), primarily due to the need to use differentiated data during calculations. Efforts were made in the current study to reduce the amount of noise in the kinematic data. Specifically, rigid mounting of marker clusters and a least-squares

optimization approach were employed to help reduce noise in the raw data, while digital filtered was used to remove high frequency noise during data processing (see Chapter 2 for details). Collectively, these efforts would have reduced the amount of noise arising due to instrumentation error and inter-marker motion. However, noise arising due to marker cluster motion with respect to the underlying bone would not have been reduced using these methods. As a result FHA orientation and position were not accurate – this would have lead to errors in MA estimation. Sheehan (2997a) avoided this noise by using Cine phase MRI to directly record bone motion. This would possibly account for some of the errors in moment arms found in the current study.

2. In order to determine the moment arm of the patellar tendon, the unit vector of this tendon had to be determined for use in equation 6.17. Because skin based marker data were not an appropriate means of tracking patellar motion, several assumptions had to be made in order to determine the orientation of the patellar tendon. For example the tendon was assumed to be inelastic and of constant length and the patellar tendon angle with respect vertical was assumed to be accurately represented by equation 6B.4 (see Appendix A6). Any or all of these assumptions likely contained some degree of error. that could have affected the accuracy of patellar tendon orientation and therefore moment arm results. Once again the Sheehan (2997b) study did not have this problem as patellar tendon position could be directly imaged.

3. The anatomical reference frames used to define segment orientation differed between the current study and the Sheehan (2007b) work. Such difference in reference frame definition may have resulted in different FHA orientation (i.e. relative angular velocity) which may have contributed to the different results obtained for the two studies.

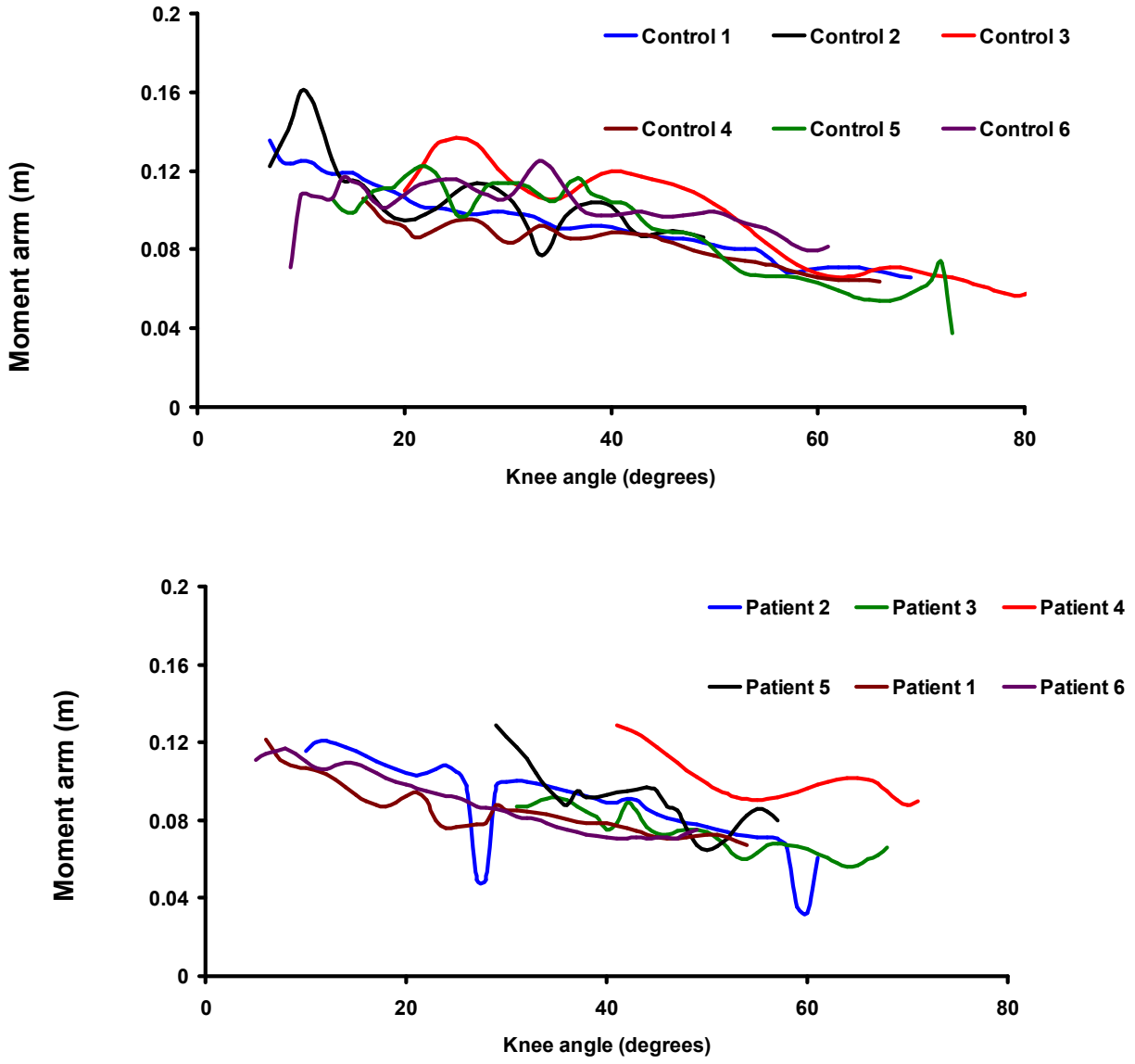


Figure 6.4: Control (upper) and patient (lower) patellar tendon moment arms determined with respect to the finite helical axis of the knee joint. See text for discussion of these results.

6.5 Moment arm: attempt #3

Given the lack of success of the PVM and 3D GM, the decision was made to use a 2D GM approach. Although not ideal, this approach was adopted in an effort to produce moment arm data that might begin to provide some insight into the effect if TKA on muscle moment arms. In this approach, the patellar tendon moment arm was determined during 3 trials of seated knee extension for both patients and controls using the following equation:

$$MA = \left\| \underline{r} \times \underline{\hat{f}} \right\| \qquad \textbf{(6.18)}$$

where $\underline{\hat{f}}$ represented the direction of pull of the patellar tendon, as defined by a unit vector, $\| \ \|$ represented the magnitude of , and $\underline{r}$ was the position vector from the knee joint center (JC) to any point on the patellar tendon line of action (note this was the same equation arrived at in simplifying the partial velocity equations in section 6.3.1 above) (see Figure 6.2). The patellar tendon point chosen was the tibial tubercle - the attachment point of the patellar tendon to the tibia. For the purpose of the current investigation, the JC for the knee was defined as a point that, in transverse and sagittal planes was midway between the medial and lateral epicondyles. In the frontal plane, the center was displaced 2.5cm below the line joining the two epicondyles. Global position of the knee joint center was previously determined as part of the kinematic analysis performed in Chapter 5. Consequently, no re-calculation of these data was done for the current study; rather already existing data were used. The methodology used to determine $\hat{f}$ was described in Appendix A6.

Application of equation 6.18 resulted in moment arms expressed as a function of time. As individuals subject kinematics varied at any given instant in time, comparisons between subjects were not possible with this time based moment arm data. To facilitate this comparison, moment arm data were fit using a cubic spline (see Chapter 5 for details), using angle increments of 1º. The resulting moment arm curves, expressed as a function of knee joint angle, could now be averaged within subjects and easily compared between groups.

6.6 Results and discussion: 2D Geometrical approach

Due to the exploratory nature of this research no statistical analysis was carried out.

Patellar tendon moment arm lengths attained using $\left\| \underline{r} \times \underline{\hat{f}} \right\|$ are illustrated in Figure 6.5.

The patient results are averaged across 3 seated knee extension trials while control data were averaged across 18 trials (3 each per participant). As there was variability within subjects with respect to the start and end angle for the knee extension motion, only angles attained by at least 11 of the 12 participants were included for analysis. As such, the angle range for the data below ran from 15º - 55 º. While data from two patients (4 and 5) appeared higher than control averages, the remaining four patients exhibited 'moment arms' that were within the range of controls.

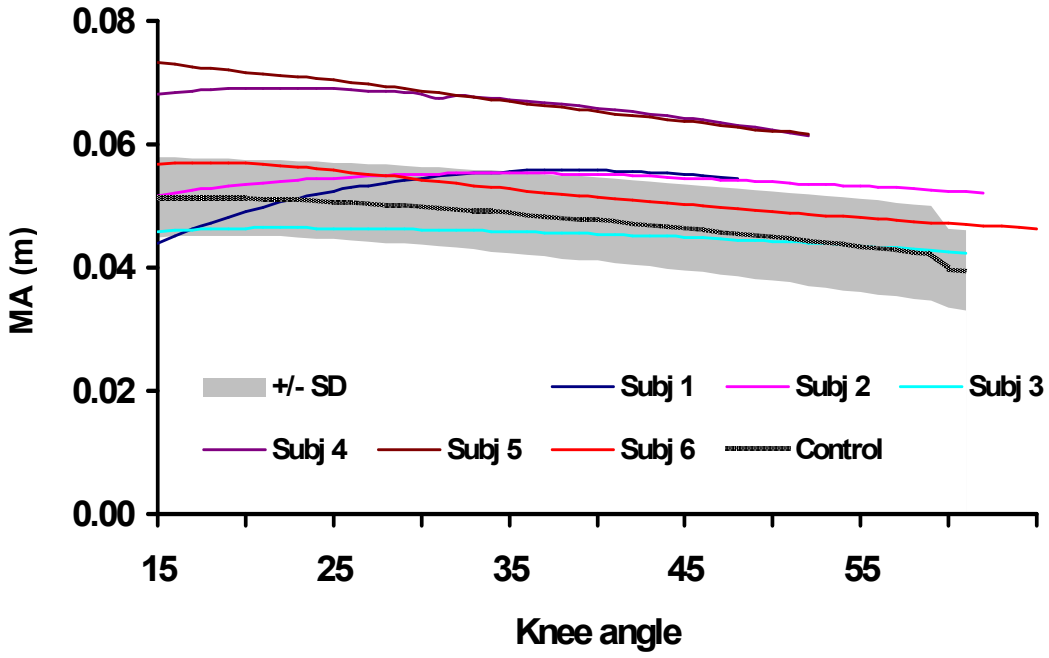


Figure 6.5: Patellar tendon moment arm as a function of knee joint angle. Subj 1 – 6 were members of the TKA group. Control data and ±SD determined by averaging moment arm results for all 6 control subjects. Averaging was done by first fitting a cubic spline to the moment arm estimates. Spline fitting enabled moment arms to be averaged across like angles for all subjects.

Direct comparison of these results with other literature was not possible given that none of the previous researchers have used the joint center employed in the current study. As differences in joint center location can directly impact moment arm length, comparisons between studies using different methods was problematic. These potential differences notwithstanding, the result presented in Figure 6.5 do generally agree with previous results. The largest patellar tendon moment arm occurred when the knee was more extended and moment arm values for healthy controls ranged from approximately 4cm – 6cm. These results agree with the published results of Imran et al. (2000), Kellis and Baltzopopoulos (1999), and Wretenberger et al. (1996). They did differ slightly from the lengths reported by Sheehan (2007b) where MAs were observed to decrease to a greater

extent with knee flexion then was observed in the current study. This could be attributed to the fact that Sheehan determined the moment arm with respect to the finite helical axis of the knee. Sheehan demonstrated (see Figure 2 in Sheehan 2007b) this axis moved noticeably anterior as knee flexion increases. This anterior movement of the axis would result in a decreased distance between the axis and the patella tendon – hence the reduced moment arm with flexion. In the current study, knee joint center position would not exhibit such a dramatic forward motion, therefore, the smaller observed change in moment length.

The intent of this work was to examine whether changes in patellar tendon MA were evident following TKA. Comparison of patient and control results (see Figure 6.5) showed that while four members of the TKA group had MAs similar to controls, the remaining two patients exhibited increased MA lengths. However, caution must be exercised when interpreting these results. Specifically the following issues likely affected the validity of the MA data determined using this 2D approach:

1. The method used assumed that the motion being examined was purely planar. In reality, knee extension, particularly during the last 20º of motion, is accompanied by tibial external rotation. Because this rotation is not accounted for by the simply calculation implied by equation 8, moment arms calculated will be in error. As the effect of TKA on tibial rotation is not yet completely understood (Banks et al. 2003), the error in the results likely affected patients and control differently, further complicating interpretation.

2. The validity of the moment arm results was also affected by the choice of center of rotation used. In the case of 2D, planar motion occurring between two bodies (i.e. rotation + translation), the point about which motion occurs is the instantaneous center of rotation (ICR). The point used in the current study was a knee joint center, defined based on the location of the medial and lateral epicondyles. This inaccurate definition of joint center would preclude further interpretation of the data.

3. Patella tendon length was assumed to be constant – in other words the tendon was assumed to act like a non-elastic structure. This has been shown to be an invalid assumption (Sheehan and Drace 2000), with changes in tendon length of up to 6.6% being reported during volitional activity. Sensitivity analyses, performed by Imran et al. (2000), however suggested that the assumption of a non-elastic patella tendon would not substantially alter moment arm lengths.

6.7 General summary of moment arm work:

The goal of the work described had been to determine if patellar tendon moment arms were altered following TKA. Unfortunately, due to the methodological issues outlined above, firm conclusions cannot be drawn with regard to this issue. That having been said however, the work undertaken to take this point has highlighted several key issues related to *in vivo* moment arm determination.

- ***2D or 3D?*** Knee joint motion is clearly 3 dimensional, particularly as the knee nears full extension. In using 2D approaches to determine knee muscle moment

arms, this non-sagittal plane motion will result in errors in moment arm lengths. While 2D moment arms are necessary in the case of 2D modeling work aimed at estimating muscle forces, their value for interpreting the affects of injury, disease or surgical interventions on knee function must be questioned. Recent work by Sheehan (2007a and 2007b), using cine-MRI to capture joint kinematics, looks promising in this regard. Because cine-phase MRI directly determines bone velocity there is no need to differentiate data thus avoiding the noise associated with calculation (Sheehan et al. 1999). Sheehan used MRI data to determine the finite helical axis of the knee joint – a process that is relatively free of noise. Moment arm lengths can then be determined with respect to the helical axis. To date Sheehan has examined moment arms of the patellar tendon (Sheehan 2007b) and individual quadriceps muscles (Wilson and Sheehan 2009) in this manner. This approach seems to be the most promising.

As the 2D calculations are much simpler, it would be advantageous to know what information is potentially lost by not doing 3D calculations. While 2D and 3D studies of patellar tendon moment arms exist, comparison of results of these studies is complicated due to differences in methodologies and participant characteristics. To overcome this problem, and determine the effect of using 2D methods to assess a 3D motion, a study calculating 2D and 3D moment arms on the same sample of subjects needs to be carried out.

- ***What is the axis of rotation of the knee joint?*** If *in vivo* moment arms are to truly represent the effectiveness of a muscle force to produce limb rotation, then the axis about which this rotation occurs has to be defined correctly. As reviewed in this document, there is still much controversy regarding the axis of rotation of the knee joint. Continued research, aimed at defining knee joint motion *in vivo* is needed in order to assist with knee MA calculations.

- ***Should the same knee axis be used for healthy individuals and those with knee pathology?*** Knee kinematics, and in the case of TKA, knee geometry, are often altered in clinical populations. Using an axis defined by joint anatomy may therefore not be appropriate for clinical populations as they will likely have different axes of rotation then those with healthy knees. One approach that avoids this problem is to use the finite helical axis for moment arm calculation. As described above, this axis is determined based on the kinematics of the joint. As such it would vary between individuals who exhibit different joint kinematics. It would therefore, likely, result in MA differences in clinical populations. As illustrated above, one problem with this approach is that kinematic data collected using skin based markers is often noisy. This noise is known to affect helical axis calculations (Woltring et al. 1985), resulting in inaccurate MA estimates. Recent use of this approach by Sheehan (2007b), using kinematic data collected using cine-MRI, appears to offer a promising solution to this noise problem.

6.8 Conclusions

Enhanced understanding of the effects of injury, disease and surgical procedures on knee joint function is integral to continued improvements in individual's ability to function optimally after these events. Central to this improved understanding is better insight into how such events affect muscle moment arms. Changes in muscle moment arm lengths have the potential to alter the moment generating capacity of muscle and consequently the ability of the knee joint to function normally. The current study attempted to quantify the *in vivo* patellar tendon moment arm, in an effort to better understand why TKA results in decreased knee extensor moments. Although methodological issues interfered with our ability to accomplish this goal, the work that was done in the attempt highlighted some of the issues that need to be addressed in order to enable accurate determination of *in vivo* knee muscle moment arms. If function following knee arthroplasty is to be optimized it is imperative that these issues be addressed so the moment arms of all the knee musculature can be determined in individuals who have undergone this surgical procedure.

Chapter 7
Answering the bigger question.

7.1 Introduction

In a recent systematic review of literature reporting gait analysis results following TKA, McClelland et al. (2007) stated that, while the "absence of a normal sagittal moment pattern in patients was the most common kinetic finding in 'the TKA literature', little is known about the cause of this phenomenon." This thesis attempted to fill that knowledge gap, by performing a detailed examination of knee moments deficits following TKA. To realize this goal, two groups - one comprised of patients who had undergone TKA and the other comprised of healthy controls, - were assessed while performing a variety of tasks. As there were a myriad of factors that could potentially influence knee joint moments post-arthroplasty a decision was made to limit the current investigation to three biomechanical factors – muscle activation, passive knee moments and muscle moment arms. As reviewed in Chapter 1, each has the potential to affect knee joint moments, yet comparatively little had been quantified about how each is affected by knee joint replacement and no efforts have been made to link changes in these factors to the reduced sagittal plane knee moment. The specific questions examined in this thesis were:

1. *Do patients exhibit altered muscle activation following TKA?*

   Muscle force is a key contributor to the net joint moment. Therefore, any investigation of the biomechanical factors affecting the net joint moment following TKA must include an examination of the muscle contribution to the net moment. One means by which such insight can be gained in through examination of muscle activation using surface electromyography (EMG). All previous investigations of EMG in individuals who have undergone TKA have been done

using tasks where multiple degrees of freedom have enabled patients and controls to use different kinematics to attain their goal. This made it difficult to determine whether EMG differences were due to some fundamental disruption in muscle function OR whether they were needed to complete the task using different kinematics. To remove altered kinematics as a variable in EMG examination, the seated knee extension study (see Chapter 4) required participants to complete a seated knee extension task. This task effectively forced patients and controls to use the same knee kinematics. As a result, any changes in the pattern or magnitude of EMG between those with and without knee replacement could be more easily interpreted.

The EMG analysis was also preformed during two weight bearing tasks – gait and stepping-up. By combining kinetic data with the EMG results, this analysis enabled the direct relationship between muscle activity and knee joint moments to be examined. These results were presented in Chapter 3.

2. *Does the passive knee moment differ between members of the TKA group and controls?*

The net internal joint moment has both an active and passive component. The passive component, while relatively small during mid-range joint motion, is created by tension in ligaments, joint capsule and elastic tissue contained within muscles. No previous attempts have been made to quantify the passive knee moment in those who have undergone TKA. Because of this, the potential impact

of alterations in passive knee moments on the net knee moment was not known. As detailed in Chapter 5, the current investigation quantified the passive knee moment for a portion of the total knee range of motion (15º - 55º of knee flexion).

3. *Are moment arms of muscle crossing the knee joint altered following TKA?*

A muscle's moment arm (MA) is a key determinant of the magnitude of the moment it can generate. MAs of knee muscles have been quantified *in vitro* in the knee arthroplasty population, however, no published reports of *in vivo* MAs exist. The initial proposal for this thesis was to quantify *in vivo* MAs of the knee musculature in individuals who had undergone TKA. As described in detail in Chapter 6, various methodological issues were encountered that resulted in insufficient MA data being determined. Despite this lack of results, the considerable time and effort invested in this portion of the thesis did result in several key issues related to *in vivo* MA determination being highlighted. The discussion presented in Chapter 6 outlines the work done in an attempt to determine MA length in the post-arthroplasty population. It also highlights several key areas that need to be addressed before further progress can be made in this important research area. Because of the attention paid to this topic in Chapter 6, no further discussion related to it will be occur in this concluding chapter.

4. *Do any of the changes observed in passive moment, muscle activity and moment arm lengths have the potential to reduce the net knee extension moment during gait and stepping-up?*

The strength of this thesis lies in the fact that all studies were carried out on the same individuals during a single testing session. This decision was made in order reduce variability that would be introduced by using either different participants for each study and/or collecting data for each of the studies on different days. Adopting this approach enabled comparison between of results from all four studies to be done. This resulted in far greater insight into why knee moments were reduced then looking at the results of any one of the studies in isolation would have.

In addition to the three biomechanical variables that were the specific focus of the thesis, there were several other 'bigger picture' issues needed to be considered when attempting to draw conclusions from the results. These issues include:

- **Habit:** Several authors (Andriacchi 1982; Smith et al. 2004;ADD REFs) have suggested that alterations in knee function following TKA could explained by individuals continuing to use movement patterns they learned prior to surgery. These movement patterns are often adopted in an attempt to reduce pain and/or enhance knee stability. While pre-surgically these patterns are perhaps necessary; following surgery, dramatic reductions in pain and improved knee stability are typically reported. This suggests that pre-surgical movement patterns are no longer needed. It is hypothesized that patients persist with these patterns because they have become accustomed to moving in that manner. As will be discussed later, it is possible that the alterations in muscle function and knee kinematics observed in members of the TKA group may be representative of patterns learned prior to surgery. While this issue cannot be definitively resolved with the data

from the current studies, results that provide interesting insight into this matter will be discussed.

- **Muscle weakness:** Numerous reports of muscle weakness following TKA have been published (see Meier et al. 2008 for review). It seems reasonable to therefore suggest that alterations in knee function following TKA may be due to this weakness. Although work by Mizner and Snyder-Mackler (2005) has suggested a link between muscle weakness and altered knee loading during gait and sit-to-stand, the impact that muscle weakness has on knee function following TKA is still largely undefined. As no attempts were made to objectively quantify muscle strength in this thesis we cannot comment directly on this issue. As was the case with habitual patterns of motion the results did however shed some light on this issue. These will be discussed near the end of this document.
- **Knee instability:** Given the important contribution of the knee to the lower limb support moment (Winter 1980), stability at this joint is integral for overall lower limb function. As Byrne et al. (2002) and Winter (1980) have both reported, the ipsilateral hip has the ability to compensate for decreased knee contributions to the support moment. Such a strategy, however, places greater load on the hip potentially making it more suspectible to injury and also potentially reducing the ability of the hip musculature to contribute to trunk and pelvic control.

  While static knee stability is a desired outcomes of TKA (Yercan et al. 2005) and is tested intraoperatively, recent work examining patients with knee OA has suggested that it is patients' qualitative perception of joint instability that actually

has the largest impact on knee function (Schmitt and Rudolph 2007 and 2008). Given that instability has been linked with certain muscle activation and joint kinematics patterns in knee OA (see Scmitt and Rudolph 2007 and 2008, Lewek et al. 2005) it is plausible to suggest that a similar link exists in those who have undergone knee arthroplasty. As this research has yet to be undertaken, the potential role that knee joint instability plays in knee function following TKA is as yet unknown. The issue of the potential role of knee instability in knee function post-TKA will be discussed at several points throughout the remainder of this document.

7.2 Passive knee moments following TKA:

Patients who have undergone TKA often complain of knee stiffness (Moffet et al. 2004). No previous research has reported passive knee moments in this population, making this thesis the first study to provide experimental results in this area. Using methods previously employed to examine passive resistance in healthy knee joints (Reiner and Edrich 1999; McFaull and Lamontagne 1998; Silder et al. 2007), subjects were seated on a raised bench so that their feet hung freely. A load cell, attached to a custom designed ankle brace, measured the force required to passively move the knee from a flexed (~60º) to a fully extended position and back again. This force data, in combination with 3D knee joint kinematics, were used to determine the passive moment exerted by the knee during this motion. Due to the effect of bi-articular muscles on passive resistance to knee motion (Reiner and Edrich 1999; Silder et al. 2007), testing was done in both sitting and supine, to capture the effect of changes in hip position on resistance provided by the hamstrings. The resulting passive moment data were fit with a cubic spline and then averaged, within

subjects and across knee joint angles, for each of the two hip angles tested. The resulting passive moment-angle data were then fit with a fourth order polynomial and integrated to determine joint stiffness as per Nordez et al. (2006). The range of knee motion analyzed was 15º - 55º of knee flexion. Despite the fact that no significant differences existed for either knee stiffness or passive moment curves, patients exhibited a strong trend towards increased passive moment, particularly at knee angles between 25º and 55º. Based on a review of the relevant literature, it was hypothesized that the most likely cause of this increased resistance was altered tibio-femoral kinematics, although changes in passive tissue stiffness and increased prosthetic joint friction (Zihlmann et al. 2005) may also have contributed.

Could the observed changes in passive knee moment have contributed to the deficits in knee extensor moment that patients in the current study demonstrated? Control knee moments during gait peaked at approximately 37Nm, while peaks for patients ranged from 16-39Nm. This large difference between patient and control moment magnitude could not be accounted for by the relatively small differences in passive moment observed between the two groups (see Table 5.2). It is therefore unlikely that the changes observed in passive moment would have had a substantial impact on sagittal plane knee moments in members of the TKA group. The possible exception was near full knee extension where others have reported passive moments between 10 – 20Nm (Reiner and Edrich 1999; Silder et al 2007). This magnitude of passive moment certainly had the potential to affect the net knee moment. As such, an investigation of passive knee

moment in the terminal portion of knee extension (i.e. 15º - full extension) needs to be undertaken with the TKA population.

As reviewed in Chapter 5, there were numerous limitations in this work. Despite these limitations, the results showed that for the small number of patients tested, changes in passive knee moments not appear to be sufficient enough to account for the observed changes in sagittal plane knee moments. Future research, with a larger sample size and expansion of the range of knee motion examined, is needed before more generalized conclusions can be drawn.

Conclusion #1:

Although minor changes were observed in the passive resistance to knee motion seen in members of the TKA group, these changes were relatively small in comparison to the magnitude of sagittal plane knee moments. This suggests that alteration in the passive knee moment would be unlikely to contribute to deficits in knee extensor moments observed during gait and stepping-up.

7.3 Muscle activation strategies:

Normal joint function is partially dependent on the ability of the joint's musculature to be activated in a manner that can produce the desired performance (i.e. support, propulsion, stability, power etc.) (An 2002). It was therefore imperative that the current series of investigations, aimed at better understanding knee moment deficits following TKA,

include a detailed examination of knee muscle function. Based on a review of the literature, the aspects of muscle activation felt to be most relevant to the population being studied were: muscle activation amplitude; the temporal pattern of activation; magnitude of co-activation; and the activation distribution between medial and lateral knee musculature.

In designing the studies for the current work, it was recognized that comparisons of EMG data between healthy and clinical populations is often complicated by the fact that patients often employ different kinematics from controls when performing the same task. Since the muscle force (and hence EMG) directly influences joint kinematics, if two individuals exhibit different kinematics, it is possibly because they used different patterns and/or magnitudes of muscle activity. The challenge was, therefore, to find a task that would force patients and controls to use the same gross joint kinematics. The task chosen was seated knee extension. This task was ideal in that it required only motion at the knee, and compensatory motion at either the ankle or the hip would not be possible. It was assumed that, because of joint kinematic similarities between patients and controls, the EMG collected during this task would provide direct insight into patients' ability to 'normally' activate lower limb musculature.

Results of the seated knee extension analysis (see Chapter 4) showed that members of the TKA group retained the ability to activate both the hamstrings and quadriceps at magnitudes similar to controls. Examination of individual patient EMG data confirmed this finding with 5/6 individuals exhibiting normal or slightly elevated quadriceps activation. Activation amplitudes for both medial and lateral gastrocnemi (MG and LG

respectively) were, however, increased in all members of the TKA group. The only temporal pattern changes observed were in vastus medialis (VM) and rectus femoris (RF) – patients activated these muscles earlier than controls, although differences did not reach statistical significance. As a result of the increased gastrocnemi EMG, co-contraction of these muscles with the quadriceps was also increased during seated knee extension. While changes in onset timing of quadriceps muscles has been previously reported following TKA (Benedetti et al. 2003), the increased amplitude observed in both medial and lateral gastrocnemi during knee extension has not been previously reported in this population.

The purpose of including the knee extension examination in the current body of work was to gain insight into muscle activation in a manner that was not clouded by difference in joint kinematics. As discussed in Chapter 4, changes in the temporal pattern of the qudriceps were hypothesized to be due to the specific demands of the knee extension task, as opposed to being representative of a fundamental change in muscle function. When the elevated levels of EMG in both medial and lateral gastrocnemi during knee extension were considered, however, it was noted that members of the TKA also exhibited increased gastrocnemi activation during portions of both the gait and step-up tasks. These universal changes in gastrocnemi activation lead to the hypothesis that changes in gastrocnemi activation in patients may in fact indicate a fundamental change in how this muscle group was activated by members of the TKA group. This appears to be the first documented account of a disturbance in gastrocnemi activation in this

population. It indicates the need for additional research in this area, in an effort to replicate these findings using a larger sample size.

Conclusion #2:

Members of the TKA group appear to have the ability to produce normal amplitudes of EMG in the quadriceps and hamstrings. Activation of the medial and lateral gastrocnemi muscles appears altered, with evidence of increased EMG amplitude being found during seated knee extension, gait and stepping-up.

7.4 Knee extensor moments following TKA – why are they reduced?

The initial study performed for this thesis was a 3 dimensional (3D) kinematic and kinetic analysis of participants walking and also stepping-onto a 17cm step. Results from this study confirmed that members of the TKA produced less sagittal plane knee angular impulse during both tasks (only the difference during midstance of gait reached statistical significance). Additionally, members of the TKA group all walked with a straighter knee, exhibiting less knee flexion during weight acceptance (WA) and little or no knee extension during midstance (MS). As discussed in Chapter 3, there appeared to be two relatively distinct behaviors that could explain why knee moments were reduced – both were directly linked to the reduced amount of knee flexion patients exhibited during gait.

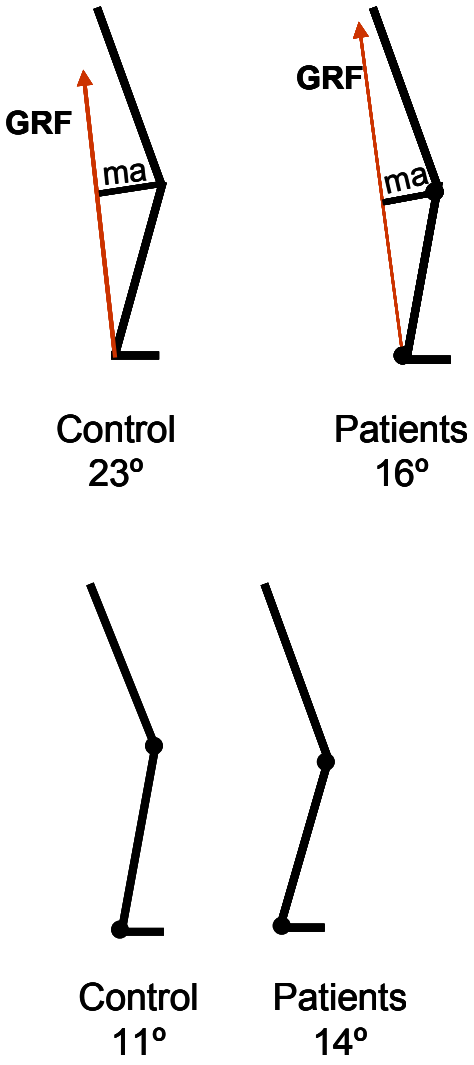
Walking with a straighter knee is a common kinematic finding in individuals with knee pathology (Berchuck et al. 1990, Jevsevar et al. 1993, Draganich et al. 1999, Weidenhelm et al. 1993, Yoshida et al. 2008, Rudolph et al. 2001). As Berchuck et al. (1990)

discussed, this decreased knee flexion is thought to reduce the demands placed on the knee extensors, thereby decreasing quadriceps activation and subsequently lowering knee joint forces. As a result of this hypothesized sparing of the quadriceps this gait pattern has typically been referred to as quadriceps avoidance gait.

As stated above members of the TKA did have reduced knee flexion during weight acceptance – this resulted in a less flexed knee at the start of MS (patients 16º of flexion and controls 23º) (Fig. 7.1a). As a result of this straighter knee, the sagittal plane projection of the ground reaction force (GRF) vector passed closer to the knee joint in members of the control group. The vector was 5cm away from the joint center in controls and only 3cm in patients (see Fig. 7.1a). Although just a crude estimate of the demands placed on the knee musculature (Wells 1981), the fact that the GRF vector is located closer to the knee joint center would create less of an external flexor moment, thereby placing less demand on the knee extensors. Following Andriacchi's line of reasoning this therefore suggested that members of the TKA group would be able to counter these external forces using less quadriceps activation.

Examination of individual patients EMG, however, indicated that only two members of the TKA group exhibited decreased quadriceps activation compared to controls. The remaining individuals had normal or elevated levels of quadriceps EMG. For the two patients with reduced quadriceps activation it seems that they may have adopted a quadriceps avoidance strategy. The remaining members of the TKA group however, appeared to be doing just the opposite – walking with a straighter knee yet using elevated

levels of quadriceps. This was clearly not a strategy to reduce knee joint loading and it certainly did not agree with the idea that knee moments were reduced due to lowered muscle activation.



a. Knee flexion angle of patients and controls at the start of midstance. GRF represents sagittal plane projection of the ground reaction force vector. **ma** indicates the perpendicular distance from knee joint center to GRF.

b. Knee flexion angle of patients and controls at the end of midstance.

Figure 7.1: Schematic representation of the three characteristics of knee joint kinematics thought to result in decreased internal knee extensor moment in members of the TKA group during the midstance phase of gait. See text for further discussion of each.

The above argument is, however, based on the assumption that similar EMG levels are equated to similar force (and therefore moment) magnitudes. However, members of the TKA employed knee joint kinematics that differed from those of controls. As muscle

length has an effect on a muscles force production, alterations in knee joint position would presumably alter the length of both the quadriceps and hamstrings, therefore, having a potential differential effect in the two groups. Members of the TKA group were less flexed at the beginning of WA then controls. Weight bearing on a straighter knee would have the effect of shortening the quadriceps and potentially lengthening the hamstrings. This would mean that in members of the TKA group, the quadriceps would produce less force for a given amount of EMG while hamstrings force production would conceivably be greater. The net affect of this length related change in force production would be decreased knee extensor force and increased knee flexor force. This combination would result in exactly the scenario observed in members of the TKA group – a decreased knee extensor moment. It is not possible, with the data available, to confirm the validity of this hypothesis at this time. Further research, employing musculoskeletal models of the knee joint to predict individual muscle forces, needs to be done in order verify these ideas.

Conclusion #3

Reduced MS knee extensor moments appeared to have two possible causes:

- Quadriceps avoidance was evident in two members of the TKA group.
- The other members of the TKA group had normal or increased levels of quadriceps activity. In these individuals decreased knee flexion at the end of WA and reduced knee extension during the MS phase likely resulted in the quadriceps being capable of producing less force due to its shortened length. This lowered force production was hypothesized to have lead to the reduced knee moments observed in these particular individuals.

7.4.1 Gastrocnemi activation and knee moments during gait:

Given the apparent influence that altered knee joint kinematics during gait has on the knee joint moment we felt it necessary to examine this characteristic of patient gait more deeply. Specifically we were interested in determining if muscle activation strategies adopted by patients could help account for this altered pattern of movement. The obvious starting point of this analysis was with the gastrocnemi. This was the only muscle group where members of the TKA group exhibited consistent EMG differences throughout all three activity tasks. In addition, it was the one muscle group where activation levels were significantly increased during the WA phase of gait.

Although the gastrocnemi are capable of producing frontal plane moments (Lloyd and Buchanan 2001, Shelburne et al. 2006) the primary action of these muscles is in the sagittal plane (Lloyd and Buchanan 2001; Shelburne et al. 2006). Gastrocnemi function during gait has received considerable research attention of late (Neptune et al. 2001, McGwan et al. 2007; Stewart et al. 2008; Schmitz et al. 2008; Neptune et al. 2008; Liu et al. 2008). Results of forward simulation and induced acceleration analyses done by Neptune et al. (2001) during normal gait have reported that the gastrocnemi, normally considered knee flexors, were capable of inducing knee extension acceleration during WA (see Fig. 8 Neptune et al. 2001). As, by definition, the knee flexes throughout WA, this extension acceleration would act to limit the amount of flexion that occurred. Although there are obvious limitations related to forward simulation analyses (Zajac et al 2002 and 2003), application of Neptune's results to findings from the current study would indicate that the decreased knee flexion observed in members of the TKA group may have resulted due to increased LG activation. Given the link we have hypothesized

between straight leg gait and the reduction in knee extensor moments exhibited by members of the TKA group, an additional hypothesis we propose is that alterations in activation of the gastrocnemi have the effect of reducing knee flexion during the WA phase of gait which subsequently leads to reduced knee extensor moments during MS. This is the first study we are aware of to suggest a link between gastrocnemi activation during WA, reduced knee flexion during stance and decreased knee extensor moments. Replication of these findings with a larger sample size could have considerable implications for rehabilitation of the TKA population, as traditionally little attention has been paid to the gastrocnemi muscles in rehabilitation following TKA (Meiner et al. 2008).

Conclusion #4

Increased LG activation observed during the WA phase of gait is hypothesized to be related to decreased WA knee flexion in members of the TKA group. This fact, combined with the link previously established between straight knee gait and reduced knee moments, lead to the conclusion that elevated activation of LG during WA is indirectly linked to reduced knee moments during the MS phase of gait.

7.4.2 Knee extensor moments when stepping-up:

Despite the fact that extensor impulse was not significantly reduced when stepping-up, patients produced less extensor impulse than controls during both double and single support phases (see Fig 3.3). These changes in extensor impulse were accompanied by

increased MG and LG activity. As was the case for the examination of increased gastrocnemi activation during gait, an extensive review of the literature pertaining to gastrocnemi function was performed to determine the potential impact of increased gastrocnmei activation on the sagittal plane knee moment. Two key papers uncovered in this review were the work of Neptune et al. (2001) and Stewart et al. (2007). Using different approaches – Neptune used forward simulations and induced accelerations, while Stewart's work was a functional electrical stimulation study- these authors both investigated the role of the gastrocnemi during various phases of gait. They found that during the midstance phase of gait (i.e. when the knee is extending) the gastrocnemi appear to produce a knee flexion moment (i.e. they resist knee extension). When stepping onto a step, the primary knee action is extension, suggesting that step-up gastrconemi activity may result in a flexor moment being produced. Since a flexor moment would act to reduce the net knee extensor moment, it is conceivable that increased LG and MG activation may explain the slight deficits observed in knee extensor impulse when members of the patient group stepped onto the raised surface.

It must be recognized that this hypothesis was based on research that examined gastrocnemi function when walking. As such, extending these findings to draw conclusions regarding gastrocnemi during stepping-up may not be appropriate. One substantial difference suggesting a need for caution was the considerable difference in knee geometry between walking (the focus of the work of both Neptune et al. 2001 and Stewart et al. 2007) and stepping-up (the task of interest for the current discussion). While the knee was extending in both cases, extension ranged from ~22º to 10º during

MS of gait, whereas the knee extended from ~65º to 53º during the double support phase of stepping up. Because segment configuration is known to affect muscle function it is possible that the gastrconemi would not function as knee flexors during stepping-up. Further research, including forward simulations and function electrical stimulation studies, performed during a step-up task, would be needed to confirm this. At this stage, however, available data and the literature seem to suggest that increased gastrocnemi EMG, in combination with a lack of EMG changes in other knee muscles, may result in decreased knee extensor moments. Further research, employing substantially larger numbers of patients and specifically targeting the role of gastrocnemi function when stepping-up, is needed to confirm this hypothesis.

Conclusion #5:
Increased MG and LG EMG during the step-up double support phase *may* contribute to the reductions in knee extensor angular impulse observed in members of the TKA group.

7.5 Why was gastrocnemi activation elevated in members of the TKA group?

The only consistent EMG finding across gait, stepping-up, and seated knee extension was evidence of elevated gastrocnemi activation in members of the TKA group. As discussed above, the increased gastrocnemi activation during gait and stepping-up was linked to reduced knee moments in the post-arthroplasty group. Viewed in isolation this finding could be interpreted to mean that altered gastrocnemi was directly linked to the particular demands of these weight bearing tasks. However, the fact that these same individuals also

exhibited increased gastrocnemi EMG during weighted knee extension suggested a more fundamental change in the way the gastrocnemi are controlled post-TKA.

The as yet unanswered question is why did members of the TKA group have elevated levels of gastrocnemi activation. Previous research examining muscle activation in those with knee OA provided some interesting insight into this very question. Hubley-Kozey et al. (2008), observed increased gastrocnemi activation during gait in individuals with severe knee OA. A common 'reason' given for increased gastrocnemi activation in those with knee OA is that it occurs in an effort to maintain knee joint stability. As such, one explanation for the increased gastrocnemi activation exhibited by members of the TKA group could be that it is related to attempts to maintain knee stability. As the demands on the knee were relatively low during seated knee extension (i.e. it is a non-weight bearing activity), the need for enhanced stability would be minimal. It is impossible to confirm this idea, as research has yet to be done to quantify knee instability and its link to muscle activation patterns in this population. Research, modeled after the OA work of Schmitt and Rudolph (2008 and 2007) and Lewek et al. (2005), is currently being planned to address this very question. Until such research is completed the hypothesis that patients increased activity in their gastrocnemi in order to enhance knee joint stability cannot be definitively rejected.

An alternative explanation for increased gastrocnemi activation is that members of the TKA group are simply continuing with a motor pattern learned prior to knee replacement. In other words, patients learned to activate their gastrocnemi in this manner when their

knee OA was severe and joint instability and pain had substantial impact on joint function. Post-surgically, despite minimal pain and instability the previously learned motor pattern continues.

Several authors have suggested the possibility that learned patterns of motion impact function post-knee replacement (Andriacchi et al. 1982; Hilding et al. 1995; Wilson et al. 1996). Recent work by Smith et al. (2004) has provided some of the first concrete evidence that habit may influence knee function post-arthroplasty. These authors examined knee moments pre and post TKA finding that pre-surgical knee moment patterns persisted post-surgically. Based on Smith's results, the evidence of elevated gastrocnemi activation in those with knee OA and the evidence, in the current studies, of this activation pattern occurring during both weight bearing and non-weight bearing tasks, a strong case can certainly be made that members of the TKA group were exhibiting habitual increase in gastrocnemi activation.

In order to gain more definitive insight into the exact nature of the changes in gastrocnemi activation, replication of the current study with a large, homogeneous patient group is needed. Additionally, pre-post studies of individuals undergoing TKA need to be carried out so that the link between pre and post surgical gastrocnemi activation levels can be established. While this has been done by Smith et al (2004) looking at sagittal plane knee moments, no efforts have been made to longitudinally examine change in muscle activation pre and post knee replacement. This work is integral if the hypothesis regarding the role of habitual movement patterns is to be definitively elucidated. Such work will have important clinical application, as the gastrocnemi are very often not

included in rehabilitation programs aimed at improving function in this population. If future research confirms that altered calf muscle activation is habitual in nature than gait retraining can be implemented to help patients return to a more normal pattern of muscle activation. If on the other hand research finds that gastrocnemi activation is elevated in order to provide enhanced knee joint stability, then removal of this strategy may put the knee under undue strain and potentially shorten the life of the prosthesis.

7.6 Alternative hypotheses:

Members of the TKA group exhibited significantly reduced sagittal plane knee angular impulse during the MS portion of gait and non-significant reductions in angular impulse during the LS phase and also when stepping-up. Based on the results from the four studies completed, the following explanations have been hypothesized for these deficits:

- During gait, quadriceps avoidance was implicated in two subjects while decreased external demands arising due to a less flexed knee were suggested to be factors for the remaining subjects. Increased gastrocnemi activation was felt to contribute to the reduced knee flexion observed, and thus was felt to be indirectly related to the reduced knee extensor moment.
- When stepping-up, increased gastrocnemi activation during the DS phase was felt to directly reduce the knee extensor moment due to the flexor moment created by this muscle group during the step-up motion.

These findings are based on a very limited number of patients and therefore need to be replicated on a larger scale before they can be generalized. Despite this fact, these results

do represent a first attempt to tease apart the factors underlying reduced knee moments in this population. It is important to recognize, however, that the hypotheses reached are not the only possibilities. This thesis would not be complete without acknowledging that others do exist. Since effective interventions to improve knee joint moments will depend on the underlying cause of altered patient kinematics each of these hypotheses was examined in light of available research and results of the present investigations.

7.6.1 Habit:

Results of the studies performed for this thesis have provided evidence to both support and refute the hypothesis that habit influence knee function post-TKA. These findings are review below:

Findings supporting the hypothesis that habitual patterns are a factor in post-surgical function:

In addition to the increased gastrocnemi activation observed in both members of the TKA group and in those with knee OA, the strongest support for the role of habitual movement patterns comes from the knee kinematics used by members of the TKA group. As reviewed above, patients walked with less knee flexion during WA, a pattern that has also been observed in individuals with knee OA (Astephen et al. 2008a, 2008b; Hubley-Kozey et al. 2006; Childs et al. 2004; Schmitt and Rudolph 2007; Fisher et al. 1997).

Findings refuting the role habitual patterns in post-surgical function:

As reviewed on numerous occasions in this document, individuals with knee OA tend to co-contract muscle around the knee joint and/or preferentially activate lateral muscle over

medial. If knee function following TKA was dependent solely on patterns learned prior to surgery, then it could be argued that members of the TKA group should have exhibited these same patterns of muscle activation during seated knee extension. This, however, was not the case, suggesting that patients had the ability to activate muscles in a manner appropriate for the task, as opposed to relying on habitual patterns of muscle activation.

An additional argument against habit influencing function following TKA comes from an examination of the co-contraction indices (CCI) found in members of the TKA group during gait. Specifically, co-contraction results from the current study (gait task) were compared to those of Hubley-Kozey et al. (2009). These authors reported co-contraction results for the period from 100ms prior to HC until the end of weight acceptance (i.e. the point of maximum knee flexion). Data were provided for three groups – healthy controls, patients with moderate knee OA, and patients identified as having severe OA. If habitual movement patterns influence knee function post-TKA, it would be expected that patients in the current investigation would exhibit co-contraction values more similar to individuals with severe OA. However, this was not the case, as members of our patient group had co-contraction levels that were very similar to the healthy controls from the Hubley-Kozey study. (There was one exception – lateral gastrocnemius co-contraction with vastus lateralis. For this muscle pair, patients' results matched most closely with Hubley-Kozey's severe OA group). It should be noted that the comparison of CCI results from the current work with those of the Hubley-Kozey work was not ideal - the group tested by Hubley-Kozey was much larger, experimental protocols likely differed somewhat, and the co-contraction time interval differed slightly (we looked at the interval

from heel contact to maximum knee flexion; Hubley-Kozey also included 100ms prior to heel contact). These limitations not withstanding, it does suggest that habitual movement patterns were not the only factor influencing knee function following TKA.

On the basis of the arguments for and against habitual movement patterns presented above, at this point no firm conclusions regarding there role in increased gastrocnemi activation can be drawn.

7.6.2 Muscle weakness:

The reduced knee flexion exhibited by members of the TKA group during gait was attributed to increased gastrocnemi activation and was felt to be indicated of attempts by patients to reduce the load on the knee joint. An alternative explanation, however, could be that patients walked with less knee flexion due to the fact that they did not have enough strength in their quadriceps to support their body weight on a more flexed knee joint. Indeed, quadriceps weakness following TKA has been reported by several authors (see Meier et al. 2008 for review) and muscle weakness has been hypothesized to contribute to post-surgical deficits in knee function (Green and Schurman 2008;Yoshida et al. 2008; Mizner and Snyder-Mackler 2005;). While muscle strength was not quantitatively measured in the current series of studies, in all likelihood members of the TKA group were weaker then controls. It is unlikely that this weakness contributed to reductions in knee moments during gait however. This argument is based on a comparison of knee moments produced by members of the TKA group when walking and stepping-up. Sagittal plane moments during gait did not exceed 0.5Nm/kg while moments of >1Nm/kg existed when stepping-up. All patients, except one, produced knee extensor

moments exceeding 0.5Nm/kg during step-up trials suggesting that patients' knee musculature was capable of producing net moments similar to control values during gait. If quadriceps weakness was truly a limiting factor during gait, moment magnitudes for step-up trials should have been equally limited.

7.6.3 Knee instability:

An additional hypothesis that has been raised in this thesis is that patients walk they way they do in an effort to combat knee instability. This hypothesis is based primarily on work from researchers examining knee function following anterior cruciate ligament rupture. Following rupture of the anterior cruciate ligament (ACL), researchers at the University of Delaware (Rudolph et al. 2000; Rudolph et al. 2001) have identified two groups of individuals – those who cope well with the injury (copers) and those who report knee instability (non-copers). The work of the Delaware group suggested yet another reason why patients may walk with a straighter knee. Rudolph et al. (2001) reported that non-copers exhibited decreased knee flexion during weight acceptance and prolonged biceps femoris (BF) activation during stance – exactly the behaviors observed in members of the TKA group. This was in contrast to copers (patients with ACL deficiency and no complaints of instability) who had normal knee kinematics and hamstring activation when walking. Rudolph et al. concluded that non-copers were using changes in knee kinematics and hamstring activation to try and counter knee instability. Given the similarities between members of the TKA and those with ACL deficiency – both lack the stability and sensory feedback provided by the ACL – it is reasonable to suggest that patients in the current study may have been attempting to compensate for

altered knee stability. As limited experimental evidence exists to describe *in vivo* knee stability post-TKA, this is an area where further research is needed.

7.7 Knee extensor moments – conclusion:

In summary, the following conclusions were drawn from this body of work:

1. Changes in passive knee moment did not appear to be a factor contributing to knee extensor moment deficits in the TKA population.
2. Quadriceps and hamstring function, as evidenced by EMG recordings, appeared intact in approximately half of the TKA group. The remaining individuals exhibited evidence of decreased activation of the quadriceps during gait. This was felt to be linked directly to attempts by patients to reduce knee joint loading as opposed to being related to a fundamental change in the way these muscles are activated. Changes in the neuromuscular control systems ability to activate these muscles does therefore not appear to be responsible for knee extensor moment deficits observed in members of the TKA group.
3. Activation of medial and lateral gastrocnemius was altered in all members of the TKA group. Magnitude changes in EMG may have accounted for the difference in knee extensor moments when stepping-up. During level walking a direct relationship between patient sagittal plane knee moments and increased gastrocnemi activation seems unlikely for the individuals tested in the current study.
4. It was hypothesized that the midstance phase reductions in knee moments observed in members of the TKA group were related to the particular kinematic

strategy adopted by patients during stance. In particular, patients weight bore on a less flexed knee that appeared to reduce the demand on the quadriceps, resulting in a decreased knee extensor moment.

7.8 Limitations:

It seems fitting to conclude this document with a discussion of the various limitations of this series of studies. Some of the limitations discussed below were known prior to research beginning, some became evident during the data collection process, and still others did not become apparent until data analysis began. Irrespective of when they arose, all influenced the applicability of these results and, more importantly perhaps, provided valuable opportunities for learning.

- An obvious limitation was the small number of subjects examined – this fact limits the applicability of these results to a more generalized population. Because of this, whenever possible it was emphasized that the results apply only to the subjects studied and not to the broader population of individuals who have had knee replacement surgery. Although a limitation, small numbers were was also seen as an asset, since they enabled a much more detailed examination of individual subject results. Without this detailed examination a likely conclusion from the thesis might have been that increased BF EMG was related to the increase in knee abduction moment during the step-up task. By examining individual patient data, however, this was found to not be the case. Such attention to individual data would have not been possible with a larger subject pool.

- Given the small numbers of subjects examined, it would have been perhaps more appropriate to used non-parametric statistics. This type of analysis is more forgiving on unequal variances and non-normal distributions than the parametric statistics used. However, the two-way and three-way ANOVAs used for data analysis do not have non-parametric counterparts. Based on discussions with a statistician, the decision was made to use parametric statistics.
- As was reviewed extensively in Chapter 2, the use of skin based marker systems to capture kinematic data is subject to errors. Although efforts were made to reduce the impact of these errors, they were not eliminated. As a result, kinematic results, particularly for non-sagittal plane motions, likely do not represent the true motion of the bony skeleton. Since most of the analysis focused on sagittal plane, however, the impact of frontal and transverse plane errors in kinematics would be minimal.
- The use of EMG to assess muscle function is limited by that fact the results from this type of analysis only allow for the analysis of muscle activation – a variable that is one step removed from muscle force. As activation is only one of many variables that influences the force produced by a muscle, it is possible that difference members of the TKA may have exhibited differences in force production, even though EMG activity did not differ.

7.9 Future research:

As is often the case with a project of this scope, considerably more questions were raised then were answered. The following five areas for future research have been identified:

1. *Gastrocnemi function post-TKA:* As the only finding common to the three tasks examined in this thesis, increased MG and LG activation post-TKA warrants further investigation. The amplitude increases were hypothesized to contribute to changes in knee moments during stepping-up, and may have been partially responsible for reduced knee flexion during gait. As such, the alterations in gastrocnemi function found were not trivial and need to be replicated and examined further.

2. *Quadriceps avoidance following TKA:* two members of the TKA group appeared to adopt this strategy while the remaining patients exhibited normal or elevated levels of quadriceps activation. Further research is needed to determine whether the different strategies employed by subjects in the current study are evident in a larger sample of patients. Confirmation of the existence of two apparently distinct strategies may have implications for treatment interventions aimed at this population. Those using quadriceps avoidance will need gait retraining so they begin to produce more normal activation of their quadriceps. Those using reduced knee flexion as a means of reducing knee loads will need to undergo gait retraining so they can being to walk with more normal knee flexion.

3. *Knee stability post-TKA:* Knee instability was identified as a possible contributor to knee function post-arthroplasty. Considerably little attention has been paid to *in vivo* knee stability in this population. Studies designed to place potentially destabilizing loads on the knee joint are needed to assess muscle behaviour in

these situations. Recent work by Lewek et al. (2005) and Schmitt and Rudolph (2008), examining individuals with knee OA, utilized just such a research paradigm. Both applied lateral perturbations either in standing (Lewek et al.) or during stance phase of gait (Schmitt and Rudolph) to individuals with knee OA while recorded joint kinematics and muscle activation. Both groups found a strong link between muscle co-contraction and participant reports of knee joint instability (interestingly, those with objective evidence of static knee instability did NOT show this same link). Although the approaches used by Lewek and Schmitt differed slightly, in general they make an effort to link reports of knee instability with observed muscle activation patterns in an effort to better understand the role knee instability plays in knee function. A similar paradigm would likely provide similar insight into the relationship between knee instability and muscle activation in those who have undergone knee arthroplasty.

4. *What role does muscle weakness play?* Recent work by LaStayo et al. (2009) has reported that strength and quadriceps muscle volume gains in individuals greater than one year post-TKA were greater when an eccentric muscle strengthening program was used compared to traditional strength training. The authors, however, did not perform any type of movement analysis on their participants to assess whether these strength gains were associated with more 'normal' knee kinematics and kinetics. This research is clearly needed and will be one of the first studies stemming from this thesis.

5. *Do movement strategies adopted following TKA place increased loads on the knee joint?*

   During the weight acceptance phase of gait patients walked with a less flexed knee joint. The knee flexion that occurs during weight acceptance is thought to absorb energy created by contact with the floor (Perry 1981). Therefore a reduction in knee flexion in members of the TKA group could increase compression and shear forces on the joint (Rudolph et al. 2001). Similarly, increased internal knee abduction moments have been associated with increased loading of the medial joint compartment (Schipplen and Andriachhi 1991). Although the effects of reduced stance phase knee flexion and increased frontal plane moments on joint loads has been proposed by numerous authors (see, for example, Zeni et al. (2009); Briem and Snyder-Mackler 2009; Childs et al. 2004) it has not, to our knowledge, been quantified experimentally. Clearly, this is an area where further research is needed if improved understanding of the impact of movement patterns on joint loading following TKA is to be achieved.

6. *What is the effect of TKA on moment arm lengths?*

   The moment arm work reported on in the current research needs to continue in an effort to better understand the effects of knee joint replacement on moment arm lengths of the knee musculature.

7. *Are reduced knee moments a bad thing?*

This question arose from reading the work of Hilding et al. (1995). These authors suggested that deficits in knee moments are not necessarily a bad thing, since they may provide a loading environment that potentially does less harm to the prosthesis. Hilding et al. based these conclusions on examination of loosening rates in individuals who exhibited two different moment patterns at the knee – one with a peak close to normal (but with an overall pattern that was very atypical), the second with a pattern that was more normal, but with a peak that was very low. They found greater risk of loosening in the group with the more normal peak value. One way of interpreting this would be to say that normal magnitude moments are not healthy for a replaced knee joint – this was the conclusion reached by Hilding's group. However, a point not made by Hilding was that the temporal pattern of the two moment profiles examined differed noticeably. Perhaps it is the temporal pattern of loading that is important rather then the magnitude. If indeed the temporal characteristics prove important, this suggests that researchers must begin to consider not just peak loads but also temporal aspects of prosthesis loading in their efforts to enhance prosthesis function and longevity. Future research, aimed at examining the link between the effect of temporal patterns of loading and prosthesis function, needs to be done.

APPENDIX A1: Participant demographics

Table A.1: Individual participant characteristics for both TKA and control groups. Group means and standard deviations (SD) are also provided.

SUBJECT	GROUP	GENDER	TEST LIMB	AGE	MONTHS POST-OP	MASS	HEIGHT
1	TKA	Female	Left	51	31.00	101	1.76
2	TKA	Female	Right	80	25.00	78	1.68
3	TKA	Female	Left	75	26.00	68	1.64
4	TKA	Male	Left	78	19.00	112.5	1.77
5	TKA	Male	Right	72	19.00	103	1.77
6	TKA	Female	Right	68	72.00	77	1.39
MEAN				**70.67**	**32.00**	**89.92**	**1.67**
SD				**10.54**	**20.12**	**17.85**	**0.15**
7	Control	Male	Right	74	NA	93	1.83
8	Control	Female	Right	74	NA	49.89	1.61
9	Control	Male	Left	70	NA	73	1.76
10	Control	Male	Left	66	NA	95.7	1.77
11	Control	Female	Right	71	NA	71.2	1.69
12	Control	Female	Left	63	NA	72.6	1.64
MEAN				**69.67**	**NA**	**75.90**	**1.72**
SD				**4.41**	**NA**	**16.75**	**0.08**

APPENDIX A2: ISSUES SURROUNDING KINEMATIC DATA COLLECTION

Prior to beginning data collection several issues related to the collection and analyses of each of these types of data had to be resolved. While most of these issues can be effectively described in the chapters that follow, kinematic data collection using surface based markers was deemed to require additional discussion. Specifically, in designing the protocols for the proposed studies considerable attention was devoted to ensuring skin motion artefact was minimized in all kinematic data. What follows is an overview of the types of errors associated with kinematic data as well as an examination of the methods that can be used to minimize the effect of these errors.

Determining segment kinematics:

The description of body segment and joint kinematics and calculation of joint kinetics require an accurate estimate of bone position and orientation (pose) during movement. In order to estimate segment pose, the motion of $n \geq 3$ non-collinear points on the segment of interest must be known. The most common means by which this data are gathered is through optoelectronic stereophotogrammetry, during which the trajectory of surface based markers is recorded and used to infer position of underlying bony landmarks (Cappello et al. 1996a). Other means of pose estimation exist, including:

- *Intracortical bone pins:* In this approach segment kinematics are tracked using $n \geq 3$ non-collinear markers, mounted on pins that have been embedded directly into bone. The bone pin marker data are recorded and analysed in a manner similar to surface based markers. This approach has been used by Lafortune et al (1992); Fuller et al. (1997) and Reinschmidt et al. (1997). The primary advantage of this method is that marker location is firmly fixed with respect to the segment, thereby avoiding errors related to skin motion that are often present when skin based markers are used. An excellent review of the pros and cons of this method is provided by Ramsey and Wretenberg (1999).

- *External fixation:* This approach is similar to the intracortical bone pin method in that markers are firmly affixed to bony segments. In this case, however, rather then mounting markers on bone pins, markers are mounted on external fixation devices that are in place for treatment of fractures or other bone/joint injuries. As such it can only be used in individuals with pre-existing conditions that require external fixation. Andriacchi et al. (1998) and Cappello et al. (1996b) have both used this method to examine thigh kinematics.

- *Roentogen stereophotogrammetric analysis (RSA):* Instead of placing markers externally, this approach uses metallic balls implanted in bone to track segment kinematics. Radiography is used to record segment position and the resulting x-ray images are used to determine segment kinematics using theory similar to the theory applied when external markers are used. This approach has been used extensively since its introduction in the mid-1970s (Karrholm 1989).

- *Video fluoroscopy:* In this approach video fluoroscopy images captured during dynamic motion. The images recorded are then used in conjunction with known geometry of prosthetic components to recreate 3D joint pose information (see Banks and Hodges 1996; Steihl et al. 1995 and1997).

- *Cine phase MRI:* Cine MRI is capable of directly estimating velocity of tissues or structures it images. Past application had been limited to assessment of blood flow and heart motion (Sheehan et al. 1999). Recent work by researchers in Maryland has resulted in this technology being applied to the assessment of human motion, in particular dynamic knee motion (see Sheehan 1997; Redmann and Sheehan 2003; and Sheehan 2007). Although presently limited to a flexion/extension motion, continuing technological advances will likely enable a greater variety of motions to be studied in this manner.

While the methods described above can provide the data required for movement analysis they are either too invasive (1 and 2) or present too much of a radiation exposure risk (3

and 4) to be used an a wide-spread basis. Practically, intracortical bone pins and external fixation devices are most often employed to test the effects of soft tissue artefacts (Leardini et al. 2005; Cappozzo et al. 1996; LaFortune et al. 1992). Cine phase MRI, the newest and least invasive of the techniques, is currently limited by availability and cost associated with MRI technology.

Skin based markers systems:

As a result of the issues associated with other available methods and until a more satisfactory approach becomes available, surface based marker systems will likely remain the most widely used means of estimating segment kinematics. Before examining some of the problems associated with the skin based marker approach, a brief review of the steps required to get from marker data to rigid body pose is necessary[2]. These steps are summarized in Figure 2B.1. Following fixation of $n \geq 3$ non-collinear markers on each segment a standing calibration trials is generally collected. Data from this trial are then used to determine the location of skin based markers with respect to certain bony landmarks (the landmarks used will depend on the specific model being implemented). Once the relationship between bony landmarks and surface markers is determined, it is assumed constant. Marker data are then collected as the individual performs the task of interest. The relationship between markers and boney landmarks determined during the calibration trial is then used to determine the global coordinates of bony landmarks. Once the global coordinates of these landmarks are known, they can be used to determine the pose of each segment at discrete time intervals. Pose information can then be used to estimate joint kinematics and for input into kinetic analyses.

Skin based marker systems: errors and methods for error reduction.

Despite being used quite extensively, skin based marker data are prone to errors that subsequently result in inaccuracy in segment and joint kinematics (Cappozzo et al. 1996).

[2] This description of methods used to determine global position and orientation of segments from surface based markers is very general, Details of the process, as used in the current work, are provided in Chapter 3. The description provided above is meant only to provide some context to the discussion of errors in kinematic data determined using skin based marker systems.

Specifically, the following errors are known to affect kinematic data collected in this manner (Chiari et al. 2005):

- Instrumentation error
- Skin movement artefact (SMA)
- Error in bony landmark identification.

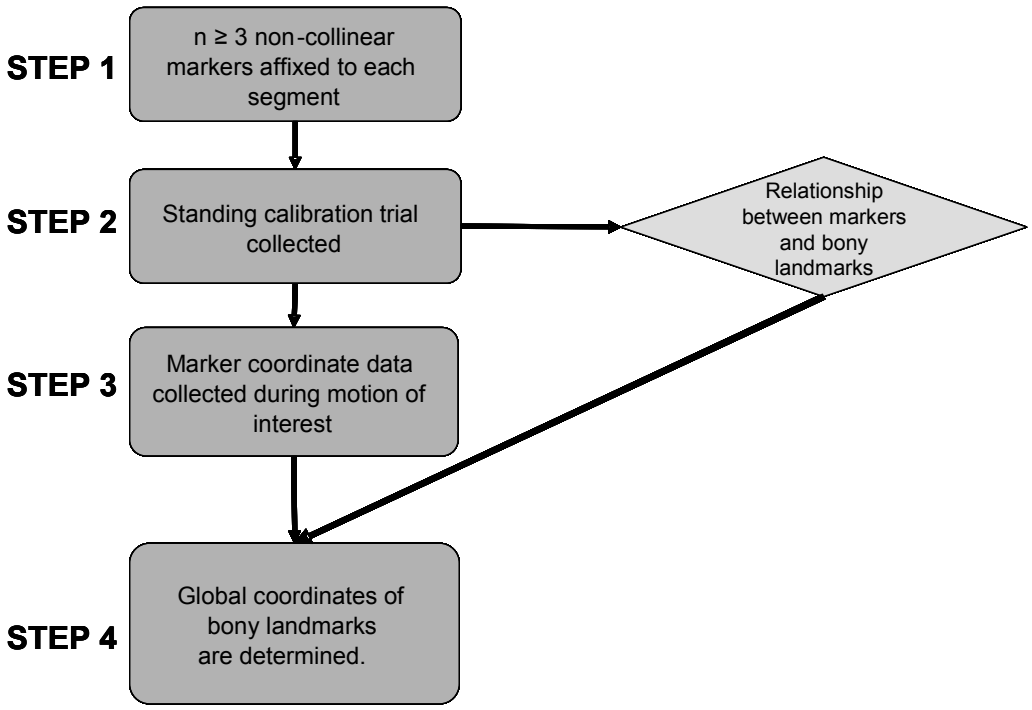


Figure 2B.1: Schematic outlining the process used to determine position and orientation of segments using a skin based marker system. Note that this is a generalized description of this process. For specific details of the steps used in the current investigations the reader is directed to Chapter 3.

Instrumentation error:

Chiari et al. (2005) provide a detailed review of the instrumentation error that can occur when stereophotogrammetric methods are used to collect skin based marker data. Briefly, such error can be either systematic (i.e. due to problems with system calibration) or random (i.e. arising from electronic noise, quantization error, interpolation of missing marker data, etc.). Irrespective of the source, instrumentation error will primarily effect step 4 of the process outlined in Figure 2B.1. During this step, marker coordinates are assumed to accurately represent marker position in global space – the presence of

instrumentation error will mean this is not the case. An obvious step to reduce this type of noise is to ensure accurate camera calibration is performed. Additionally, high frequency noise can be relatively easily removed using digital filtering (Winter 1991) in the data processing stage. Various least squares minimization approaches have also been developed to more accurately determine marker position globally (see review in Chiari et al. 2005). These mathematical means of reducing error, which are also utilized to help compensate for SMA, will be discussed in more detail in the next section.

Skin motion artefact errors:

Motion between skin based markers and the underlying skeleton are common when skin based markers are used to collect kinematic data during dynamic tasks. This motion results in errors in pose estimation that are much greater than those due to instrumentation error (Leardini et al. 2005). The magnitude of the error introduced will vary depending on the marker position on the segment (Cappello et al. 1997), the marker set-up used (Cappozzo et al. 1997), the means of marker attachment to skin (Manal et al. 2000), and the task being examined (Fuller et al. 1997). It is beyond the scope of this paper to provide a detailed review of error magnitudes when using skin based markers, however the interested reader is directed to the work of Reinschmidt et al. (1997), Holden et al. (1997), Lucchetti et al. (1998), Manal et al. (2003); Schwartz et al. (2004), Chiari et al. (2005), Fuller et al. (1997) and Leardini et al. (2005) for a sampling of research in this area. It should be noted however, that the effects of errors due to SMA are often more noticeable in planes where motion magnitudes are small. A 2º error will have relatively little effect in the sagittal plane where the total motion is > 100º. The same errors, occurring in the frontal plane where total motion may be <10º could have potential greater impact of outcomes. This logic has been confirmed by Reinschmidt et al. (1997) who examined knee kinematics during running using both surface mounted markers and intracortical bone pins. Reinschmidt et al. (1997) concluded that errors induced by skin movement artefact were too large to enable frontal and transverse plane angles to be interpreted with any degree of confidence.

The errors arising due to SMA can be divided into two distinct types (Leardini et al. 2005). The first occurs when the location of individual markers change with respect to one another. This is problematic, because the relationship between marker and underlying bony landmarks (see Step 2, Figure 2B.1) assumes that marker location with respect to other markers on the segment remains constant. Efforts to reduce this error can be implemented during experimental set-up (i.e. marker placement decisions) and in the data analysis stage (i.e. mathematical methods used to determine marker position).

As Capello et al (1997) have shown using comparisons to intracortical bone pin data, markers located directly on bony sites result in the highest noise magnitudes. The first step in avoiding SMA between markers is therefore to avoid placing markers over bony prominences. Additionally, placing segment markers on a common rigid plate, so that marker locations are relatively fixed with respect to each other, is another technique shown to reduce intermarker motion (Manal et al. 2000; Holden et al. 1997). During data analysis, least-mean squares minimization techniques have been developed that can help reduce the noise resulting in non-constant intermarker distances – this noise can be due to instrumentation error or SMA. These approaches, most of which are designed to take advantage of the redundancy created by having $n \geq 3$ markers per segment, use various methods of optimizing orientation matrix definition so as to reduce errors in pose estimation. A complete description of these various methods is beyond the scope of this work, however, the reader is directed to Chiari et al. (2005) for an excellent review of this topic. One approach commonly applied uses singular value decomposition to solve the lesast squares minimization problem (see Challis 1995). This will be the method used in the current study and as such it will be described in detail in Chapter 3.

The second type of SMA that occurs is due to motion of whole marker cluster with respect to the underlying bone. Such motion will result in a non-constant relationship between marker reference frame and the anatomical frame. This will introduce errors into Step 4 (Fig 2B.1) of the process, resulting in inaccurate segment pose estimates. Dealing with SMA arising due to marker cluster motion with respect to bone is more complicated then the problem of intermarker motion and until recently has been a relatively neglected

issue. Leardini et al. (2005) provide a detailed review of currently available methods. Despite these attempts at reducing error Leardini et al. (2005) concluded that "…the objective of a reliable estimation of skeletal motion in *in vivo* experiments of human movement has not yet been achieved satisfactorily".

Error due to bony landmark misidentification:

A crucial element in determining the relationship between markers and bony landmarks (Step 2, Figure 2B.1) is the accurate identification of bony landmarks. As Della Croce et al. (2005) reviewed extensively; errors in bony landmark identification can have a substantial impact on segment and joint kinematics determined with skin based markers. These errors affect two types of landmarks – those that are palpable and those that are internal (such as joint centers) (Della Croce et al. 2005). Inaccuracies in landmark identification can be particularly problematic in light of their importance for accurate determination of the anatomical coordinate systems and joint axes. As alteration in coordinate system definition and joint axes location have been shown to influence both the kinematics (Most et al. 2004; Piazza and Cavanagh 2000) and kinetics (Manal et al. 2002) of the knee joint, attention to the details of landmark determination is important.

Summary: errors in kinematic data

When determining segment and joint kinematics using surface based markers, errors inherent in the data collection process can impact negatively on experimental results. In order to reduce the potential impact of error on kinematic data in the series of studies proposed for this thesis the following mechanisms will be implemented:

Marker placement:

Markers will be affixed to rigid mounting plates prior to being attached to shank, thigh and pelvic segments. This will eliminate having to place markers over bony landmarks and will also reduce intermarker motion artefact.

Instrumentation error:

An Optotrak (Northern Digital, Waterloo Ontario) motion measurement system will be used to collect kinematic data. This system has been shown to have an accuracy of 0.3mm. Additionally, as the camera calibration procedure used has been designed and tested by Opotrak, instrumentation errors arising from this portion of the process should be minimal.

Skin motion artefact:
In addition to attention to marker attachment detail, a least squares approach utilizing singular value decomposition (as per Challis 1995) will be used to help reduce errors arising from inconsistent intermarker distances.

Error due to landmark misidentification:
Bony landmark palpation will be performed by the same investigator for all subjects. The investigator has a clinical background and experience with locating anatomical landmarks through the skin. This approach will help ensure consistency in landmark definition across all subjects.

While these efforts are expected to reduce errors due to instrumentation, intermarker motion and landmark identification, they will not address the issue of marker cluster motion with respect to bony landmarks. As a result, care must be taken when interpreting knee data from the frontal and transverse planes, as error rates will likely remain relatively high in these planes.

APPENDIX A3: Dealing with skin motion artefact

Skin movement artifact is a common problem known to affect the accuracy of kinematic data collected using skin based marker systems (see Capozzo et al. 2005 and Chapter 2). In an effort to reduce the effect of skin movement artifact on kinematic results for this series of investigation a least squares minimization procedure that utilized singular value decomposition was used. The specific approach was taken from Challis (1995) and readers are directed to this paper for additional details on the approach. What follows are the specifics of how this approach was applied to the current work.

Let point $\boldsymbol{p_l}$ the x,y and z coordinates of a given marker, expressed with respect to the marker reference frame. For an given marker, the locally defined coordinates were determined using data from the standing calibration trial and as such remained constant throughout the process described below. In order to express $\boldsymbol{p_l}$ with respect to a different reference frame, say *g* (the global frame), the following equation can be used:

$$\boldsymbol{p}_g = \mathbf{R}_{m2g}\,\boldsymbol{p}_l + \boldsymbol{o}_g \qquad \textbf{(A3.1)}$$

where $\mathbf{R}_{m2g}$ is the orientation matrix expressing the orientation of the marker reference frame with respect to the global frame and $\boldsymbol{o}_g$ is the position vector of origin of marker frame with respect to global frame. In a non-optimized approach to finding $\mathbf{R}_{m2g}$ and $\boldsymbol{o}_g$, global marker data would be used to directly establish the relationship between marker and global frames. However, the marker data are known to be affected by errors (see Chapter 2) the resulting kinematics would also contain error. To minimize this error, an optimization procedure can be used to determine these two quantities – in the current work a least squares optimization approach was employed. Using least squares optimization, determining $\mathbf{R}_{m2g}$ and $\boldsymbol{o}_g$ becomes a matter of minimizing the following equation:

$$\frac{1}{n}\sum_{i=1}^{n}\left(\left[R_{m2g}\right]p_l + o_g - p_g\right)^T\left(\left[R_{m2g}\right]p_l + o_g - p_g\right) \qquad \textbf{(A3.2)}$$

where n are the number of non-collinear markers per segment (n=4 for the current study). By applying simplification as outlined in Challis (1995) minimizing equation A3.2 is the same as maximizing:

$$\frac{1}{n}\sum_{i=1}^{n}\left(p_{gi}^{'\,T}\left[R_{m2g}\right]p_{li}^{'}\right) \qquad \textbf{(A3.3)}$$

where $p_g^{'}$ and $p_l^{'}$ are defined as follows:

$$p_{gi}^{'} = p_{gi} - \overline{p_g} \qquad (\overline{p_g} = \frac{1}{n}\sum_{i=1}^{n} p_{gi}\ ) \qquad \textbf{(A3.4)}$$

$$p_{li}^{'} = p_{li} - \overline{p_l} \qquad (\overline{p_l} = \frac{1}{n}\sum_{i=1}^{n} p_{li}\ ) \qquad \textbf{(A3.5)}$$

Equation A3.3 can be rearranged as illustrated by Challis to give:

$$\frac{1}{n}\sum_{i=1}^{n}\left(p_{gi}^{'\,T}\left[R_{m2g}\right]p_{li}^{i}\right) = tr\left(\left[R_{m2g}\right]^{T}\left[C\right]\right) \qquad \textbf{(A3.6)}$$

where tr( ) refers to the trace of a matrix and $[C]$ is the cross-dispersion matrix that is computed as follows:

$$[C] = \frac{1}{n}\sum_{i=1}^{n} p_{gi}^{'}\,p_{li}^{'} \qquad \textbf{(A3.7)}$$

As p_{gi} represents the global marker coordinates, p_{gi} 's for each marker are known at each instant in time of interest (i.e. from Optorak data). In addition the coordinates of individual markers, expressed with respect to the marker frame (i.e. p_{li}) are also known from the standing calibration trial. Using these known values $[C]$ was easily determined using equation A3.4 and A3.5. Once $[C]$ was known, the SVD of $[C]$ was defined by:

$$[C] = [U][W][V]^{T} \qquad \textbf{(A3.8)}$$

For the purpose of the current work, the SVD of $[C]$ was determined using methods described by Wilkonsin et al. (1986) using custom software (M. Ishac).

The results of the SVD calculation can then be used to determine $\lfloor R_{m2g} \rfloor$ based on the fact that equation A3.3 will be maximized (see Challis for details) when:

$$[R_{m2g}] = [U][V]^T \qquad \textbf{(A3.9)}$$

In order to prevent the situation where the matrix determined using A3.9 is a reflection matrix rather then a rotation matrix, Challis (1995) made the following modification to equation A3.9:

$$[R_{m2g}] = [U]\begin{bmatrix} 1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & \det([U][V]^T) \end{bmatrix}[V]^T \qquad \textbf{(A3.10)}$$

Once $\lfloor R_{m2g} \rfloor$ was determined it represented the orientation of the marker reference frame with respect to the global frame and could be used to determine the global position of markers. Because of the optimization approach used, errors resulting in non-constant inter-marker distance would be reduced, therefore, improving the accuracy of segment kinematics determined in this manner. It should be noted that error arising due to motion of the whole marker cluster with respect to underlying bone was not addressed with this algorithm and, therefore, likely affected segment pose.

APPENDIX A4: Frontal plane knee moments following TKA

Although this thesis focused on sagittal plane knee moments, frontal plane moments were also calculated and analysed. Frontal plane knee moments are of importance to this population as high internal knee abduction moment has been linked to OA development (Amin et al.2004) and have been identified as an indicator of disease progression and severity (Landry et al.2007; Mundermann et al. 2005; Astephen et al. 2008). These links between frontal moments and OA are thought to be related to the fact that the frontal plane knee moment influences medial / lateral distribution of knee forces (Schipplen and Andriachhi 1991). As increased joint loading is associated with an elevated risk of prosthesis failure (Andriacchi et al. 1997; Mundermann et al. 2008) it is of interest to know whether the frontal plane knee kinetics, and therefore, frontal plane knee load distribution, return to normal levels following surgery. A literature search found only two studies that reported frontal plane knee moments post-arthroplasty. These analyses, both carried out during level walking, showed peak internal knee abduction moments returned to control levels post-arthroplasty (Milner and O'Bryan 2008; Mandeville et al. 2008). Given the association between altered joint loading and prosthesis failure (Andriacchi et al. 1997), better insight into frontal plane knee moments post-arthroplasty would appear key to prolonging the life of the prosthesis.

Methods:

Participants and methods used for this analysis were identical to those employed for the sagittal plane analysis reported on in the main body of this chapter.

Results:

Figure A4.1 contains the ensemble averaged frontal plane knee moments for all members of the TKA group during both gait and stepping-up. Angular impulse was used to quantify the changes observed in these frontal plane moments. While differences in frontal plane impulse were evident in both gait and stepping-up (Fig. A4.2), only the changes during stepping-up were statistically significant ($p<0.0001$ for both double support and single support phases).

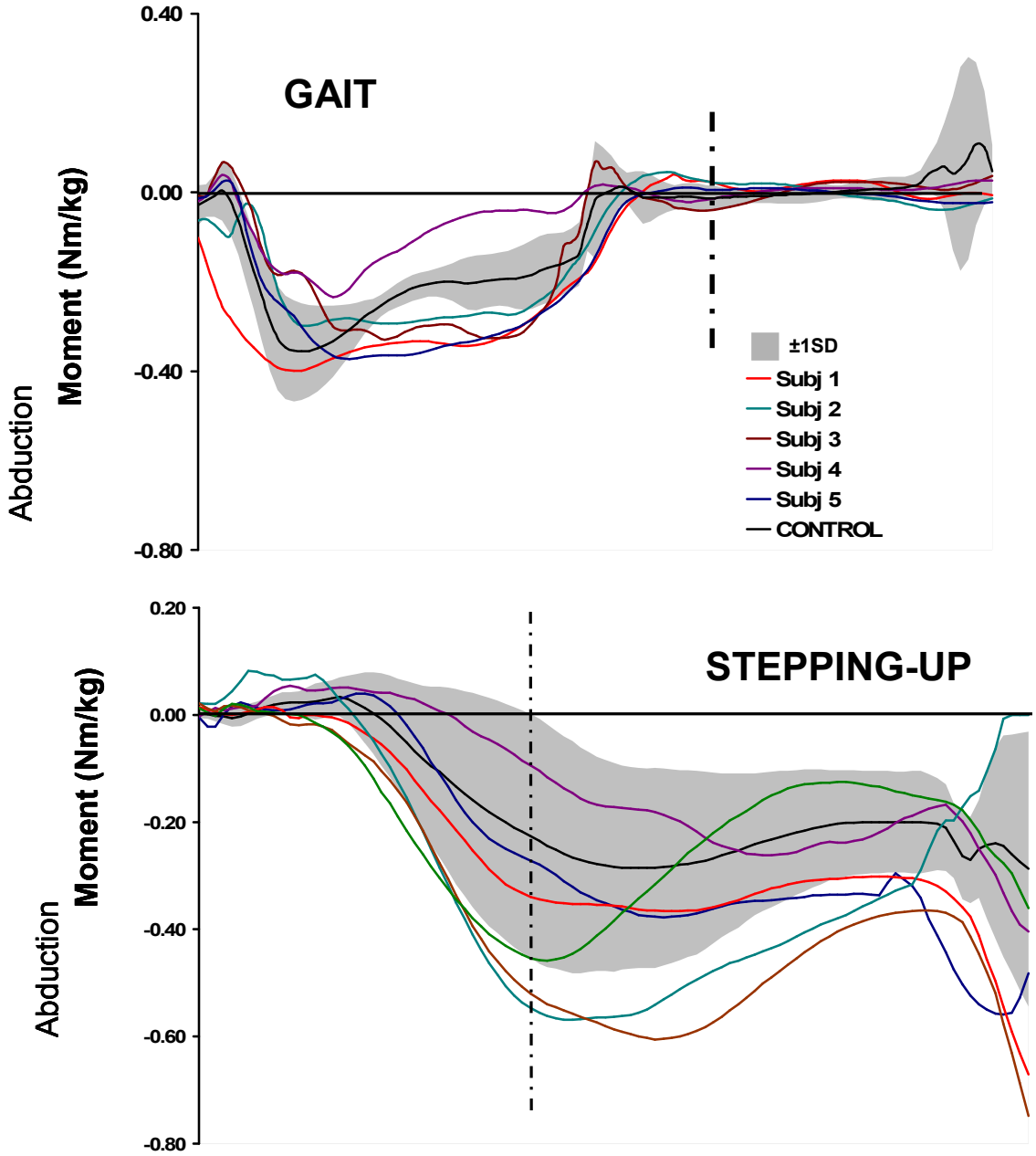


Figure A4.1: Frontal plane knee moments during gait (upper panel) and stepping-up (lower panel) for members of the TKA group. Only data for 5 patients are shown for the gait trials due to problems with force plate data for the sixth individual.

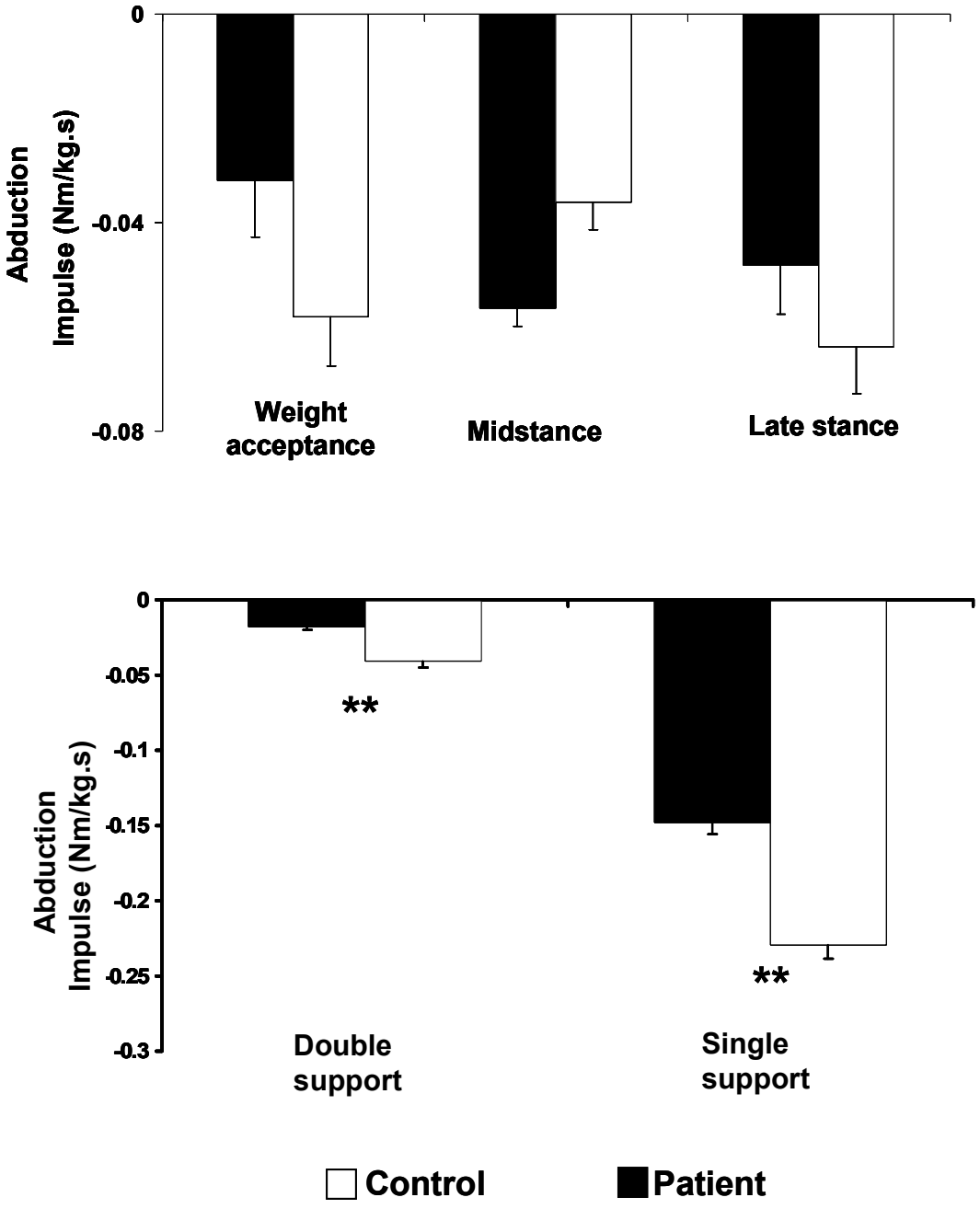


Figure A4.2: Frontal plane angular impulse data during gait (upper panel) and stepping-up (lower panel). Error bars represent standard error of the mean. ** indicates significant difference between controls and patients.

Discussion:

Members of the patient group exhibited increased internal knee abduction moments compared to controls. More specifically, frontal plane knee angular impulse was increased during the DS_{su} and SS_{su} portions of step-up trials (see Fig. A4.2). Increases were also observed during WA_g and LS_g phases of gait, however, these were not significant. These findings were contrary to those reported by Milner and O'Bryan (2008) and Mandeville et al. (2008) who both found that following TKA peak knee abduction moments were similar to those with healthy knee joints.

The contradictory conclusions drawn between the present study and the literature were likely due to the different means used to quantify the knee moment. While Milner and O'Bryan (2008) and Mandeville et al. (2008) examined peak moments, angular impulse was used in the present research. The peak moment only provided information about demand placed on the joint at one instant in time – details related to the time course of moment application were lost. As Thorp et al. (2006) have shown, angular impulse provides a means by which both moment magnitude and timing characteristics can be quantified. For example, figure A4.3 shows a sample frontal plane knee moment profile for gait and stepping-up. If just peak values were considered the frontal plane moments would look similar; however, angular impulse clearly differentiated between them (angular impulse for gait:-0.16Nm/kg.s; step-up:-0.27Nm/kg.s). A longitudinal study performed by Hilding et al. (1999) provided additional insight into the importance of examining moment profiles when examining moments in individuals who have undergone TKA. In their work, which examined the relationship between frontal plane moment patterns and tibial component loosening over a 2 year period, Hilding et al. (1999) found that the peak internal knee abduction moment was not a good predictor of tibial component loosening following TKA. Rather Hilding et al. (1999) reported that prosthesis loosening was more likely to occur in individuals where the moment profile over time differed markedly from controls. Re-examination of the frontal plane moment profiles for individuals provided by Milner and O'Bryan (2008) and Mandeville et al. (2008) suggested that if these authors had determined frontal plane impulse instead of

simply examining peak values, their conclusions may have agreed with those of the present study.

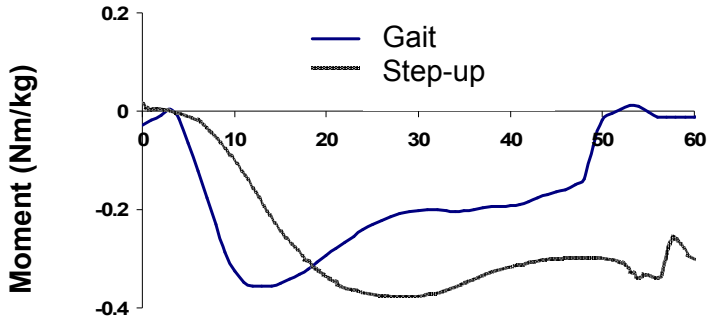


Figure A4.3: Ensemble averaged internal frontal plane knee moments for control subjects during the stance phase gait and stepping up. Negative values denote an internal knee abduction moment. Notice the similar peak moments for both tasks and the prolonged abduction moment when stepping-up. Angular impulse measures were -0.16Nm/kg.s for gait and -0.28Nm/kg.s for step-up.

Frontal plane moments and knee joint loading:

The internal knee abduction moment influences medial / lateral distribution of forces in the knee (Schipplen and Andriachhi 1991) – with high internal abduction moments being linked to high medial compartment loading. In a healthy knee joint, considerable asymmetry exists between force distribution in the medial and lateral joint compartment - with approximately 60% of the load being concentrated on the medial side during weight bearing activities (Schipplein and Andriacchi 1991). Most of this asymmetry has been linked to a limb alignment which results in ground reaction force projection passing medial to the knee joint during weight bearing (Shelburne et al. 2006). This association between frontal plane knee moments and knee joint loading raises concerns when considered in light of the findings of the current investigation. Higher frontal plane angular impulse in members of the TKA during the step-up task, may have indicated that the medial compartment of the prosthetic joint was being subjected to higher than normal loads. This could have implications for prosthesis longevity, as abnormal prosthesis loading has been shown to increase wear rates and component loosening (Kuster et al. 1997). Continued investigation of frontal plane moments in this population is therefore warranted, to better elucidate the effects of TKA on frontal plane loading.

APPENDIX A5:

Partial linear velocity: $\mu = (\partial tx/\partial q_i)\,x + (\partial ty/\partial q_i)\,y + (\partial tz/\partial q_i)z$

- For each **rotational** generalized coordinate:

  $\underline{\mu_i} = (\partial tx/\partial q_i)\,x + (\partial ty/\partial q_i)\,y + (\partial tz/\partial q_i)z = (0,0,0)$

  This is because translational motion was independent of rotation.

- For each **translational** generalized coordinate:

  $\underline{\mu_4} = (\partial tx/\partial q_4)\,x + (\partial ty/\partial q_4)\,y + (\partial tz/\partial q_4)z = (1,0,0)$

  $\underline{\mu_5} = (\partial tx/\partial q_5)\,x + (\partial ty/\partial q_5)\,y + (\partial tz/\partial q_5)z = (0,1,0)$

  $\underline{\mu_6} = (\partial tx/\partial q_6)\,x + (\partial ty/\partial q_6)\,y + (\partial tz/\partial q_6)z = (0,0,1)$

  Because the partial derivative of any generalized coordinate with respect to itself will be 1. Its partial derivative with respect to any other translational coordinate will be 0, because translational motions, along different axes are independent of one another.

Partial angular velocity: $\underline{\omega} = (\partial r_1/\partial q_i)\,x + (\partial r_2/\partial q_i)\,y + (\partial r_3/\partial q_i)z$

- For each **rotational** generalized coordinate:

  $\underline{\omega_1} = (\partial r_1/\partial q_1)\,x + (\partial r_2/\partial q_1)\,y + (\partial r_3/\partial q_1)z = (1,0,0)$

  $\underline{\omega_2} = (\partial r_1/\partial q_2)\,x + (\partial r_2/\partial q_2)\,y + (\partial r_3/\partial q_2)z = (0,1,0)$

  $\underline{\omega_3} = (\partial r_1/\partial q_3)\,x + (\partial r_2/\partial q_3)\,y + (\partial r_3/\partial q_3)z = (0,0,1)$

- For each translational generalized coordinate:

$\underline{\omega} = (\partial r_1/\partial q_i)\,x + (\partial r_2/\partial q_i)\,y + (\partial r_3/\partial q_i)z = (0,0,0)$

This is because the rotational motion was independent of translation.

APPENDIX A6: Methods used to determine patellar tendon orientation.

In order to define patellar tendon orientation, two points on the line of action of the patellar were needed. One point chosen was the tibial tubercle, the point of attachments of the patellar tendon to the tibia (see TT_{PT} in Figure A6.1b). Because this point was one of the anatomical landmarks digitized during the standing calibration trial, its position with respect to the marker frame was therefore known. Finding coordinates for the tibial tubercle at each instant in time, was accomplished using the following generic equation:

$$\boldsymbol{p}_g = \mathbf{R}_{m2g}\,\boldsymbol{p}_l + \boldsymbol{o}_g \qquad \textbf{(A6.1)}$$

$$\mathbf{R}_{m2g} = \begin{bmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{bmatrix} \qquad \textbf{(A6.2)}$$

where $\boldsymbol{p}_g$ and $\boldsymbol{p}_l$ represented the coordinates of the tibial tubercle with respect to the global and marker frames respectively. $\mathbf{R}_{m2g}$ was the orientation matrix that defined the orientation of the global frame with respect to the marker frame at any given point in time. See Chapter 2 for details of how $\mathbf{R}_{m2g}$ was determined. $\boldsymbol{o}_g$ was the position vector of the origin of the marker frame with respect to the global frame.

As surface markers were not an effective means for tracking patellar location due to the considerable amount of skin motion artefact and small size of the patella, a second point on the line of action of the patellar tendon was not readily available. To solve this problem a mathematical patellar model was added. This model, from Delp (2009) consisted of a locally defined axis system and coordinates for the insertion points of the

four quadriceps muscles and the patellar tendon. The unit vector of the patellar tendon, vec_{PT} (see Figure A6.1), was directed from the tibial tubercle to the patellar tendon insertion on the patella (P_{PT}). It was assumed that the patellar tendon lay in the xy (sagittal) plane only, therefore meaning that:

$$P_{PT} = (T_x, T_y, 0) \quad \textbf{(A6.3)}$$

where T_x and T_y are illustrated in Figure A6.1. It was assumed that the length of vec_{PT} was constant at 0.06m (White 1986). Using this assumption and the angle θ from Figure A6.1b, T_x and T_y were easily determined using simple trigonometry. The angle θ was determined using equation (A6.4) taken from the work of White (1986). .

$$\theta = 15 + 0.317\varphi - 0.0084\,\varphi^2 + 0.00013\varphi^3 \quad \textbf{(A6.4)}$$

where φ, illustrated in Figure A6.1a, was defined as the angle between the long axis of the shank and thigh. Once θ, T_x and T_y is determined then the global position of P_{PT} can be calculated using basic trigonometry.

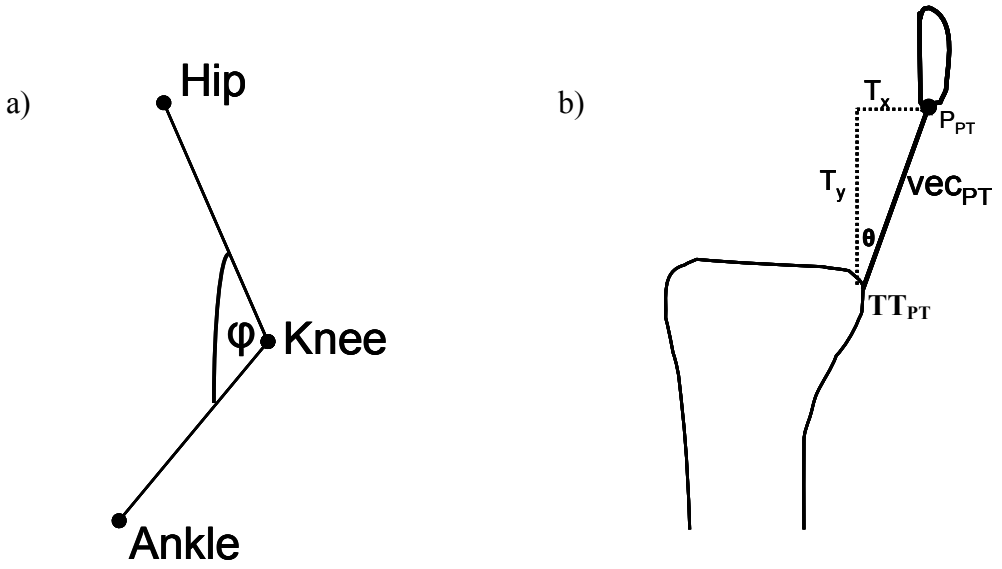


Figure A6.1: a) Angle φ, as used in equation A6.4, is illustrated in this figure. b) Schematic of approach used to determine vec_{PT}. See text for details.

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