In Vitro Biomechanical Analyses of The PCL and Medial Ligaments of The Human Knee

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Abstract

Previous studies have shown that surgical treatments of PCL injuries are not successful in all cases and there is room for improvement. The effectiveness of an isolated PCL reconstruction, in the setting of what actually is a multi-ligament injury, may be inadequate, and therefore the biomechanical contribution of other ligaments in a PCL-deficient knee need to be better understood.

A new apparatus was used to analyze the effect of medial ligaments transection on the kinematics of the PCL-deficient knee during simulated clinical tests and activities of daily living. We observed that the anterior translation of the medial side of the joint increased after transection of the POL; however, this increase was small. Transection of neither the POL nor dMCL affected the posterior translation of the medial aspect of the joint; however, both contributed to resisting loads crossing the joint, which increase after the PCL transection.

Keywords

Knee, Ligaments, PCL, Medial Ligament, Kinematics, Force Contribution
Summary for Lay Audience

The posterior cruciate ligament (PCL) is one of the main ligaments in the knee joint which helps stabilize against the shearing motion. Injury to the PCL is common in knee injuries and it usually happens in sports or car accidents. There are several surgical techniques to treat PCL injury and regain pre-injury joint motion; however; studies show that these techniques have a high failure rate and need to be improved.

Since PCL injuries are combined with injuries to other structures of the knee; understanding the role of those structures in stabilizing of the PCL-deficient knee can be the key to PCL reconstruction improvement. Over the past few years, investigations on the effect that structures on the lateral side of the joint (the outward-facing side of the knee) have on the kinematics of the PCL-deficient knee have resulted in the reconstruction of lateral structures as an additional step in the PCL reconstruction. On the contrary, there is not enough data on the role of structures on the medial side of the knee (inward-facing side of the knee) when the PCL is injured.

In this study, a new joint motion simulator was used to analyze effects that ligaments on the medial side of the knee have on the kinematics of the knee. It was found that the PCL transection resulted in abnormal joint motion. During activities of daily living such as stair ascent and descent, PCL transection resulted in abnormal joint motion.

Medial structures of the knee had an effect on the kinematics of the joint when a torque was applied on the shinbone. Even though transection of medial ligaments did not affect the kinematics during other loading scenarios such as backward load or internal moment, our study discovered that they had a relatively large force contribution to resisting those motions. Thus, damage to these structures can lead to abnormal loading patterns on a reconstructed PCL and cause failure.
Co-Authorship Statement

Chapters 1, 2 and 5 were written solely by Alireza Moslemian.

Chapter 3 of this study (Influence of the posterior cruciate ligament on the stability of the knee during experimentally simulated clinical tests and activities of daily living) was co-authored by Dr. Alan Getgood and Dr. Ryan Willing. This paper was drafted by Alireza Moslemian and modified by Drs. Getgood and Willing.

Chapter 4 of this study (The Biomechanical Contribution of Medial Ligaments in The Stability of The PCL-Deficient Knee) was drafted by Alireza Moslemian and modified by Dr. Willing.
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Chapter 1

1 Introduction

1.1 Motivation

The posterior cruciate ligament (PCL) is considered to be the largest and strongest ligament of the human knee joint [1], [2]; however, studies show that PCL injury accounts for up to 44% of acute knee injuries that occur during sports and vehicle accidents [3]–[7]. This percentage includes both isolated PCL injury and multi-ligament injuries. PCL injuries commonly occur during these accidents due to a large posterior-directed load applied to the tibia when the knee is flexed [8], although hyperextension of the joint is another mechanism of PCL injury which commonly occurs in sports [9]. As a treatment, different ligament reconstructions have been developed to restore the normal kinematics of the knee following a PCL injury; although, studies show that these reconstruction techniques have failure rates up to 30% [10]. Patients with a failed reconstruction can experience pain and an increased risk of osteoarthritis due to abnormal kinematics, and they must undergo more operations which can be costly. Thus, current reconstruction methods have the potential to be improved.

PCL injuries are rarely just a tear in the PCL, and 95% of PCL injuries happen in combination with injuries to other ligamentous structures of the knee joint [2], [3], [8], [11]. Therefore, having a better understanding of the role that secondary joint stabilizers play in a human knee can be beneficial to improve existing reconstruction techniques. Since the injury to the posterolateral structure is the most frequent secondary injury associated with PCL injury [8], there have been various studies investigating the effect of posterolateral structures of the knee joint on the kinematics of the knee and specifically on the kinematics of PCL-deficient knees [11]–[14]. As a result, existing PCL reconstructions are followed by reconstruction of posterolateral structures, if needed [10]. On the contrary, it is still unclear how posteromedial structures of the knee, which are the second most common associated injury, contribute to knee stabilization and if they need to be repaired.
in case of an injury. The deficiency of medial structures might increase the load exerted on the reconstructed PCL and result in PCL reconstruction failure. Additionally, if abnormal kinematics persist after the PCL reconstruction because of the deficiency of medial structures, it can have detrimental effects on cartilage health [15]. The current study aims to characterize the contribution of the deep medial collateral ligament (dMCL), superficial medial collateral ligament (sMCL) and posterior oblique ligaments (POL) during simulated clinical tests and simulated activities of daily living including gait, stair ascending and descending.

1.2 Objective

This study aims to use a new platform for biomechanical testing of the knee joint for this experiment and investigates the contribution of the posterior cruciate ligament and medial ligaments on the biomechanics of the knee joint. Therefore, the objectives of this study are to:

1. Investigate the effect of PCL injury on the kinematics of the knee during simulated clinical tests using an AMTI VIVO joint motion simulator.

2. Investigate the effect of different joint reduction methods on the kinematics of the joint.

3. Investigate the effect of PCL injury on the kinematics of the knee during activities of daily living.

4. Characterize the contributions of the posterior oblique ligament and deep medial collateral ligament in the stability of the PCL-deficient knee during simulated clinical tests and activities of daily living.

5. Characterize the load response of the posterior oblique ligament, deep and superficial medial collateral ligament during simulated clinical tests and activities of daily living.
1.3 Organization of Thesis

Chapter 2 discusses the anatomy of the human knee joint followed by the anatomy and mechanics of the posterior cruciate ligament and medial ligaments. A brief review of previous experimental studies on these aforementioned ligaments using different testing modalities is included in Chapter 2.

Chapter 3, comprised of the first manuscript of this study, investigates the effect of the PCL on the kinematics of the knee using a new servohydraulic joint motion simulator. This chapter starts with discussing PCL injuries and the lack of in vitro studies investigating the kinematics of the knee joint during activities of daily living and continues with an outline of the experimental setup used in the first part of the study. Results gathered in this chapter using the new apparatus are then compared to results published in prior studies which used different joint motion simulators. This chapter also investigates the effect of simulated muscle forces on the kinematics of the joint in comparison with a simple axial compressive force acting on the tibia.

Chapter 4, comprised of the second manuscript of this research, focuses on the effects of the POL, dMCL and sMCL on the biomechanics of PCL-deficient knees during simulated clinical tests and activities of daily living. This chapter starts with a review of previous experimental studies on the POL and dMCL, and their contribution to the kinematics of a PCL-deficient knee joint. Afterward, a brief overview of methods including the experimental setup, calculation process for medial tibial kinematics and force contribution measurements of each ligament is presented.

A final discussion of results and conclusions are presented in Chapter 5 of this thesis. Limitations and strengths of this study are presented in this chapter. This chapter also summarizes the clinical impact that this thesis can have on treatment decision making.
Chapter 2

2 Background

This chapter starts with a brief review of human knee joint anatomy to familiarize the reader with different terminologies used in the text. This is followed by anatomy and biomechanics of different ligamentous structures of the knee joint. Moreover, the reader is provided with a review on previous in vivo and in vitro studies that have examined these ligamentous structures. In the last part of this chapter, the reader will be provided with information regarding different joint motion simulators that have been used for previous in vitro studies.

2.1 Knee Joint Anatomy

Before discussing the anatomy of the knee joint, a review of some common anatomical terminologies used for the knee joint is required in order to understand knee joint motions. As demonstrated in Figure 2-1 Common planes used to define anatomical motions, three anatomical planes are defined to describe the motions of the human body. Universal directional terms used in this study can be classified as shown in Figure 2-2. Anterior-posterior: anterior being towards the front of the body and posterior being toward the back. Medial-lateral: medial being towards the mid-sagittal plane and lateral being away from the mid-sagittal plane. Superior-inferior: superior being above and inferior being below. Proximal-distal: proximal being closer to the torso and distal being farther from the torso.
Figure 2-1 Common planes used to define anatomical motions [16]

Figure 2-2 Anatomical terminologies used to describe directions. (Image courtesy of Complete Anatomy, Dublin, Ireland)
Human joints can have up to six degrees of freedom including three translations (anterior-posterior, medial-lateral, and superior-inferior) and three rotations (flexion-extension, internal-external and abduction-adduction). As shown in Figure 2-3, flexion of the knee is defined as a bending motion in the sagittal plane that decreases the angle between the distal and proximal segment of the knee joint, and extension is defined as the motion increasing the angle. Abduction of the knee is defined as the motion in the frontal plane that moves the distal part of the joint away from the midline of the body. The opposite motion that brings the distal segment towards the midline is called adduction. Inward rotation of the distal segment of the joint is called internal rotation and the opposing rotation is called external rotation.

Figure 2-3 Anatomical definitions of different rotation
The knee is described as a synovial joint which acts as a hinge that has 2 degrees of freedom: flexion-extension and internal-external rotation; however, this is a simplified description of the knee motions since the knee joint has small motions in other directions as well. As shown in Figure 2-4, the knee joint consists of 4 osseous portions: the tibia (shinbone), femur (thighbone), patella (kneecap) and fibula, with the fibula being an extra-articular bone [17]. Three of these bones comprise two joints, the tibiofemoral and patellofemoral joint. The tibiofemoral joint refers to the articulation between the tibia and femur which is involved in flexion-extension and internal-external rotation of the joint. The articulation between the patella and femur forms the patellofemoral joint, which is involved in flexion-extension of the joint. There are two separate fibrocartilage menisci in between the femur and tibia, the medial and lateral meniscus, which help to distribute the load to the tibia by increasing the contact surface and stabilizing the joint. Menisci also protect the bone by absorbing shocks.
The human knee is surrounded by strong capsule, ligaments, and muscles which stabilize the knee joint during different tasks. The muscles stabilize the joint both actively (applying loads by contracting) and passively (loaded when they are under tension). As shown in Figure 2-5, the knee muscles are divided into two main groups, the extensor (quadriceps femoris) and flexor mechanism (hamstrings). Knee ligaments are also passive stabilizers of the knee joint. There are four main ligaments in the human knee that stabilize the tibiofemoral joint, comprising two cruciate ligaments and two collateral ligaments. As shown in Figure 2-6, the anterior cruciate ligament (ACL) and posterior cruciate ligament (PCL) are located in the intercondylar space of the tibiofemoral joint and they help control the joint in the anteroposterior and internal/external directions. The medial collateral ligament (MCL) and the lateral collateral ligament (LCL) are located on the sides of the tibiofemoral joint and they constrain the abduction and adduction of the joint, respectively. There are many more additional capsuloligamentous stabilizers involved in stabilization of

**Figure 2-5 Muscle groups surrounding the knee joint.** (a) Anterior view (b) posterior view

(Image courtesy of Complete Anatomy, Dublin, Ireland)
the knee joint. This study focuses on the PCL and ligaments on the medial side of the knee joint.

**Figure 2-6 Cruciate ligaments.** Anterior and posterior cruciate ligaments in the human knee joint (a) lateral view (b) transverse view of the left knee (Image courtesy of Complete Anatomy, Dublin, Ireland)
2.2 Posterior Cruciate Ligament (PCL)

The posterior cruciate ligament is the primary ligamentous structure that inhibits the posterior translation of the tibia. The PCL attaches to the posterior side of the tibia, hence the name “posterior”, and the medial wall of the femoral notch [9], as shown in Figure 2-6. It has an average length of 38 mm and width of 13 mm [18], and it is the strongest ligament in the human knee joint [1], [2]. The PCL consists of two inseparable bundles, the anterolateral (AL) and posteromedial (PM) bundles [19]–[21]. The AL band becomes slack when the knee is in full extension and taut when the knee is flexed; however, the PM band acts in reverse as it gets taut in full extension and slack as the knee flexes [9], [22], [23].

Although studies show that the PCL is the strongest ligament in the knee, isolated and combined PCL injuries, together, account for 44% of acute knee injuries happening in sports and vehicle accidents [3]–[7]. PCL injury will result in abnormal kinematics of the joint and can increase the risk of osteoarthritis in patients [24]–[28]. There are three well-known injury mechanisms for the PCL: 1) excessive posterior displacement of the tibia when the knee is flexed at 90 degrees 2) hyperextension of the joint, and 3) hyperflexion of the joint [9]. PCL injuries mostly happen in sports activities and vehicle collisions when a posterior-directed load is applied to the flexed tibia [24], [29]. In order to diagnose PCL injuries, different physical examinations have been developed, with the most valuable of them being the posterior drawer test [20]. During the posterior drawer test, the examiner applies an approximately 100 N posterior-directed force on the tibia with the knee flexed to 90 degrees. Based on the resulting displacement of the tibia with respect to the femur, PCL injury can be identified [30]. The grade of the PCL injury can be categorized based on the magnitude of the posterior translation. A Grade I injury, or partial tear of the PCL, is defined as 0-5 mm posterior translation of the tibia. A complete tear of the PCL is classified as a grade II injury in which the tibia translates posterior 5-10 mm. A posterior translation more than 10 mm is an indication of a multi-ligament injury, a grade III PCL injury [31].

Various studies have attempted to quantify the effect that a PCL injury can have on knee joint biomechanics. An *in vivo* study on eight PCL-deficient patients was performed by
Li et al. in which they examined the kinematics of the tibia during weightbearing flexion via imaging the knee with MRI [32]. It was observed that near full extension, PCL injury did not have a significant effect on the position of the tibia. On the contrary, when the knee was flexed further, PCL injury caused a posterior translation of the tibia. *In vivo* kinematics of the knee during the drawer test have been studied by Logan et al. [33]. A significant increase in the laxity of the medial compartment of the tibia, from $2.3 \pm 1.9$ mm to $8.2 \pm 3.3$ mm, was observed in PCL-deficient knees compared to intact knees.

Although *in vivo* studies can result in more realistic kinematics with all the muscles being active, the measurement and comparison of these motions are challenging and error-prone. Kinematics measurements in *in vivo* studies are usually performed by imaging the knee, or by using motion tracking. In most cases, imaging equipment is stationary, which limits their usage to analyzing motions where the patient is stationary, such as lunging [32], and they can be harmful to test subjects if patients are exposed to ionizing radiation for long periods of time. Recently, a few studies have overcome these limitations by using mobile fluoroscopy imaging systems [34]. Using motion tracking, the kinematics measured are generally prone to some error since motion tracking markers are usually located on the skin, and the skin moves relative to the bone [35]. Moreover, the comparison between the intact knee and injured knee in *in vivo* studies is not reliable since it is not ethically accepted to cause injury in patients; therefore, there is rarely corresponding “healthy” data for knees with injured ligaments. Thus, using cadaveric knees can be useful for better understanding how a certain ligament injury can affect the kinematics of the joint. One limitation of *in vitro* studies, however, is the lack of joint motion simulators capable of performing complex motion such as activities of daily living (ADL). In recent years, joint motion simulators have evolved from custom-designed machines to robotic arms capable of having more complex motions [36]–[38]; however, robotic arms capable of applying high magnitude forces are bulky and potentially dangerous to work with. For instance, a KUKA industrial arm capable of applying load up to 5000 N requires a working space of around 80 m$^3$. More recently, compact servohydraulic joint motion simulators, which are capable of applying high magnitude loads, have been used to analyze the behavior of joints [39]–[42].
A VIVO six degrees of freedom (6-DoF) joint motion simulator (Advanced Mechanical Technology, Inc., Watertown, MA, USA) is one of the servohydraulic joint motion simulators that have been used to analyze knee implants [41], [42]. The VIVO can manipulate joints via relative motions of two actuators. The lower actuator translates in the medial-lateral, anterior-posterior, and proximal-distal directions, and also provides internal-external rotation. The upper actuator provides flexion-extension and adduction-abduction rotations. All forces and moments through the joint are measured using a 6-DoF load cell located within the lower actuator and closed-loop controls allow the VIVO to operate each DoF independently in either force or displacement control mode. The VIVO is the first joint motion simulator that fully expresses motions in the Grood and Suntay [43] coordinate conventions. Figure 2-7 shows a mounted specimen on the VIVO.

![Figure 2-7: The experiment set up on The AMTI VIVO joint motion simulator](image)

Various in vitro studies have investigated the kinematics of the PCL-deficient knee by mounting cadaveric knees on joint motion simulators and observing the effect that the PCL had on the kinematics under different loads and motions. An analysis of ten cadaveric knees which were subjected to a 134 N posterior-directed load was performed by Petersen et al. [44]. A significant increase in posterior tibial translation was detected at all
flexion angles, with the maximum increase occurring at 90° of flexion. Additional in vitro studies have analyzed the kinematics of the PCL-deficient knee under simulated clinical tests and they all have observed an increased posterior tibial translation due to the PCL injury [11, 38, 44–49]. The force contribution of the PCL has been investigated in a small number of studies by observing the changes in the resultant joint forces due to PCL transection under repeated identical kinematics [11, 13, 44, 49]. These studies have reported that 85% to 95% of the posterior stability of the knee at 90° is provided by the PCL. None of these in vitro studies, however, have been able to investigate the effects that PCL transection can have on knee kinematics during complex activities of daily living.

2.3 Medial Ligament Complex

Ligamentous structures on the medial side of the joint provide resistance to abduction and internal-external rotation of the joint [50]. The medial ligament complex of the knee consists of three ligaments: the superficial medial collateral ligament (sMCL), deep medial collateral ligament (dMCL) and posterior oblique ligament (POL).

The sMCL is the largest ligament on the medial side of the knee and it has one femoral and two tibial (distal and proximal) attachments, as shown in Figure 2-8 [51]. The proximal tibial attachment of the sMCL is connected to soft tissues at the medial joint line and the distal attachment is connected to the tibia directly [51].
Figure 2-8 Medial ligaments. (a) Superficial medial collateral and posterior oblique ligaments (b) deep medial collateral ligament of the medial ligamentous complex (adopted from Wijdicks et al. [52])

As shown in Figure 2-8, the POL has a bony femoral attachment and three fascial attachments to the distal semimembranosus tendon which are named the superficial, central and capsular arms [53], [54]. The femoral attachment of the POL is slightly posterior and proximal to the sMCL attachment [51]. The superficial arm is parallel to the posterior side of the sMCL and it merges with the semimembranosus tendon distal insertion [51]. The central arm, which is the thickest part of the POL, courses from the distal semimembranosus and has an attachment to the medial meniscus [51]. The capsular arm of the POL fans out posterior to the central arm and connects to the anterior portion of the distal semimembranosus tendon. The capsular arm does not have any osseous attachment to the tibia [51]. A study performed by Hughston et al. showed that the POL gets slack as the knee is flexed [53].

The deep medial collateral ligament refers to the vertical thickening of the knee capsule under the sMCL [51]. The dMCL consists of two portions, the meniscofemoral and
The meniscotibial portion of the dMCL is shorter and attaches to the tibia just distal to the edge of the tibial plateau [51].

In a study by Robinson et al., the tensile strength of the sMCL, dMCL, and POL was reported to be $534 \pm 85$ N, $194 \pm 82$ N and $425 \pm 151$ N, respectively [56]. The effect of the medial ligaments on the biomechanics of the joint has been reported in various studies. Robinson and colleagues investigated the effect of these medial ligaments on the kinematics of twenty four cadaveric knees [36]. It was observed that in an intact knee, an injury to either of dMCL or POL does not have a significant effect on the internal and external rotation; conversely, injury to the sMCL increased the internal-external rotation of the joint significantly. Moreover, no significant changes in the posterior translation of the joint were reported after transection of the medial ligaments. These results agree with other studies which have looked at the effect of medial ligaments [57]–[59].

Studies show that the medial ligaments of the knee joint have more significant roles in the absence of the primary anterior stabilizer of the joint, the ACL [57], [60]–[62]. Haines et al. reported an increase in the anterior translation of the ACL-deficient knee caused by an injury to medial ligaments [57]. To the knowledge of the author, there has been just one study looking at the contribution of medial ligaments in the absence of the PCL [37]. This study investigated the kinematics of the PCL-deficient knee under a posterior drawer test when the knee was at $90^\circ$, and it was observed transection of both the dMCL and POL increase the posterior translation of the tibia when a posterior-directed load was applied on the tibia. The role of medial ligaments on the rotational stability of the PCL-deficient knee, however, has not been completely elucidated.

Different methods have been used to measure the force contribution of medial ligaments. Griffith et al. measured the load response of the POL and sMCL using buckle transducers [63]. Buckle transducers consist of a crossbar that sits underneath the ligament and a rectangular stainless steel frame equipped with strain gauges which measure the strain applied on the frame through the crossbar. One of the limitations of this method is that the tension applied on a ligament when a buckle transducer is mounted can affect the function of the ligament. Another approach to measuring a ligament’s load is using superposition.
This approach assumes that any change in the load response of a system subjected to a consistent, repeated motion is associated with a change made to the system. Thus, the measured change in the load response of the joint caused by a ligament transection is defined as the load that was previously applied by that ligament. This method has been used in several PCL studies [11], [13], [36], [44], [49], [58], [62]. The same method was used by Robinson et al., and they observed a 60% force contribution attributed to medial ligaments in intact knees when a posterior load and internal moment were applied on the tibia simultaneously [36]. Sakane and colleagues reported up to 38.1 ± 25 N of force applied by the medial ligaments using the same method [62].
Chapter 3

3 Influence of the Posterior Cruciate Ligament on the Stability of the Knee During Experimentally Simulated Clinical Tests and Activities of Daily Living

This chapter begins with a brief review of the available literature on the PCL injury and its effect on the kinematics of the tibia. A new servohydraulic joint motion simulator is used to investigate the role that the PCL plays on the kinematics of the joint under simulated clinical examinations and activities of daily living. Additionally, the study presented in this chapter will analyze the effect that simulated muscle loads can have on the kinematics.

3.1 Introduction

Despite decades of studies, sub-optimal clinical outcomes persist for surgical treatments for knee ligament injuries. For instance, previous studies have shown that 20% to 60% of patients receiving operative treatment for PCL injuries will prematurely develop arthritis [24]–[28]. While there is compelling evidence that knee osteoarthritis originates from cartilage injury sustained during these events [15], past clinical studies have also identified pathomechanical motion patterns in PCL deficient knees [45]. This suggests that either reconstructed PCL’s are not providing the same biomechanical contributions as they did prior to the injury, concomitant injuries that are not addressed during surgery have a significant contribution, or perhaps both. This change in joint mechanics can further exacerbate cartilage degradation [15]. Thus, there remains a need for studies focused on better understanding the contributions of knee ligaments to knee joint biomechanics and motion.

Much of our understanding of knee ligament biomechanics has been developed through in vitro studies using cadaveric knees or clinical studies comparing injured and healthy patients [11], [46]. Clinical studies give the best indication of the overall net effect of an injury/repair on knee joint motion, especially during activities of daily living (ADL) such
as gait or stair climbing [64], [65]; however, it is difficult to control for the effects of inter-patient variability, exact injury pattern, and motion adaptation. Cadaveric studies offer greater control but are limited by the (lack of) sophistication of the loading apparatus, such that primary outcomes are usually restricted to basic kinematics during simple flexion/extension motions and joint laxity/stability [47].

Furthermore, few in vitro cadaver studies have employed loads and motions representative of activities of daily living [66], [67]. This has likely been a result of the lack of joint motion simulator apparatuses capable of accommodating such complex six degrees of freedom (6-DoF) loading scenarios, as well as consensus as to what loads should be used to represent those activities. More recently, however, compact and versatile servohydraulic joint motion simulators have become commercially available and have been shown to be effective tools for studying the biomechanics of cadaver joints [39] and implant components subjected to loads and motions representative of activities of daily living [40], [41]. For the knee, instrumented TKR loading data have been compiled into a standardized load set and made publicly available (https://orthoload.com) [68]. Despite having this standardized load set not accurately depicting intact knee loading, it can provide a reasonable loading protocol to test knees under more physiologically loading than simple passive flexion/extension.

The primary objective of this cadaveric biomechanics study is to examine changes in knee motion as a result of PCL transection during simulated ADL. Prior to that, however, kinematics and stability data collected for intact and PCL-deficient knees undergoing simple passive flexion-extension motions will be compared with previously published findings, primarily to demonstrate consistency between results collected on the 6-DoF servohydraulic joint motion simulator and previous analysis techniques. Since previously-established testing protocols were using different combinations of simple compressive loadings or muscle-based loading, we will explore the sensitivity of our results to the loading mechanism as well. We hypothesize that PCL transection will increase the posterior translation and internal-external rotation of the joint [11], [38], [44]–[49]. Furthermore, we hypothesize that the loading mechanism (compression versus simulated muscle forces) will have a significant effect on joint kinematics. Finally, we anticipate that
the PCL dissection will increase the posterior displacement of the joint during simulated ADL.

3.2 Methods

*Specimen preparation*

Twelve fresh-frozen non-arthritic cadaveric knees (from donors aged 53-98 years, 4 male, 5 female, 2 pairs) were used in this study. Specimens were CT scanned for further analyses of the posterior slope of the tibia. The posterior slope of the medial plateau for all specimens was within the range reported for normal knees in a previous study (5 degrees to 15.5 degrees [69]). Each specimen was thawed 24 hours before testing at room temperature. Biomechanical testing of each joint was performed using a VIVO joint motion simulator. Mounting the specimens on the VIVO first required that the femur and tibia/fibula each be transected approximately 200 mm proximal to or distal from the approximate transepicondylar axis, respectively. The proximal and distal segments of the joint were skeletonized to expose approximately 75 mm of clean bone for secure potting into dental model stone (Modern Materials Golden Denstone Labstone, Modern Materials, Kulzer GmbH, Hanau, Germany). The femoral pot (approximately 130 mm length of 2” diameter polyvinyl chloride pipe) was mounted to the upper actuator of the VIVO using a custom-designed aluminum fixture. This fixture allowed adjustment of the adduction-abduction and internal-external rotations and proximal-distal position of the femur, such that the flexion axis of the specimen aligned with the mechanical flexion axis of the VIVO. This alignment was confirmed using a FARO GAGE digital coordinate measuring machine (FARO Technologies, FL, USA) whose coordinate system was co-registered to that of the VIVO. First, the flexion axis of the specimen was adjusted to be parallel to the mechanical flexion axis of the VIVO. This alignment was achieved by adjusting the abduction-adduction and internal-external rotation of the femur in the fixture so that the medial-lateral translation of the tibia (with respect to the VIVO) was minimal throughout flexion of the joint. The tibia/fibula (shank) was manually guided through its entire flexion-extension range of motion by an investigator while applying a small reducing load. During this motion, the tip of the FARO GAGE was held in a small hole on the distal tibia made by
the operator, such that the arc of motion of that reference point was recorded. The flexion axis of the specimen was estimated based on the centre of a circle fit to the path of the reference point. The flexion axis of the specimen was then matched with the flexion axis of the VIVO by translating the femur until the centre of the circle fit was within 2 mm of the VIVO/digitizer’s origin. This mounting method was developed to minimize tibial motions, which would occur as the tibia “chases” an improperly positioned femur in force control. A 2 mm criteria was deemed an acceptable distance to keep the motion of the tibia minimal. When focusing on relative kinematics of the joint, small misalignment that can result from this alignment method will have only small effects on measured outcomes. The tibia and fibula were potted in situ into the tibial pot (approximately 100 mm length of 5” diameter acrylonitrile butadiene styrene pipe) which was installed onto the lower actuator using a cylindrical aluminum clamp. This potting technique resulted in the neutral alignment of the joint residing near the middle of the operating ranges of the VIVO’s actuators.

*Intact joint testing*

Once installed, the neutral path of motion of the knee was recorded. This baseline loading scenario consisted of continuous cyclical flexion of the knee from full extension (0°) to flexion (90°) with a 100 N compressive force acting parallel to the long axis of the tibia and passing through the approximate joint centre. During this motion, remaining degrees of freedom were set to maintain zero loads; meaning no external loads were applied in other directions and the specimen was able to move freely in those directions. This loading scenario was then repeated with each of five additional forces or moments applied to the tibia to measure the stability of the joint, including a posterior-directed force of 134 N, a 5 Nm internal or external moment, and a 10 Nm adduction or abduction moment. These loading scenarios were achieved through communication between the load cell (beneath the tibia) and the proportional-integral-derivative (PID) controller of the VIVO. The load cell was set to measure loads and moments at the joint level. Therefore, the PID controller displaced the femur and tibia (via motions of the lower and upper actuator) to reach the desired load and moment at the joint level (measured by the load cell). Each
flexion/extension motion was repeated four times at a rate of 0.04 Hz, and all kinematics were recorded during the 2nd and 3rd cycles at a sampling rate of 100 Hz.

To investigate the effect of joint reduction method, the aforementioned loading scenarios were repeated, but with simulated muscle forces compressing the joint instead of the 100 N compressive force. Simulated muscle forces were comprised of a 50 N quadriceps force and two hamstring forces each applying 25 N. The constant 50 N quadriceps force was created using a 24 mm Airpel anti-stiction pneumatic actuator (Airpot Corp, Norwalk, CT, USA) via a 250 lbs. test microfilament braided line (Scotty, Sidney, BC, Canada) sutured to the quadriceps tendon and routed through pulleys to provide an appropriate line of action. Two virtual springs were introduced at the approximate medial and lateral hamstrings insertions so that the total 50 N flexor force acted parallel with the long axis of the femur. During simulated muscle forces, the lower actuator was set to apply 0 N axial load on the tibia (in other words, no external load applied, but joint contact forces would result from muscle actions).

After all the cyclical flexion/extension loading scenarios were completed, the specimen was subjected to simulated activities of daily living (ADL), including gait, stair ascending, and stair descending. For this experiment, standardized “AVER75” loading data were used, but all loads were reduced by 75% to prevent specimen damage. All degrees of freedom were manipulated in force-control mode, except for the flexion angle, which was prescribed. These loads were cycled, and kinematic data were recorded at the same rates as the cyclical flexion/extension loading scenarios. Figure 3-1, 3-2 and 3-3 show loads and moments applied to specimens to simulate activities of daily living.
Figure 3-1 Simulated gait. (a) Loads, (b) moments and (c) flexion angle used to simulate gait.
Figure 3-2 Simulated stair ascent. (a) Loads, (b) moments and (c) flexion angle used to simulate stair ascent.
Figure 3-3 Simulated stair descent. (a) Loads, (b) moments and (c) flexion angle used to simulate stair descent.
**PCL-deficient joint modelling**

Following the biomechanical testing of the intact joints, the PCL was transected via a small trans-patella tendon portal under arthroscopic guidance to simulate a grade II injury of the PCL, without damaging other joint stabilizing structures. Subsequently, the PCL-deficient knee was subjected to the same loading scenarios as the intact knee, and the resulting kinematics were used to measure changes in knee stability.

**Data analysis**

In three specimens, the range of motion limits of the VIVO was met in at least one direction during the cyclical flexion/extension testing after transection of the PCL, and therefore their complete dataset could not be collected and they were excluded from the data analysis. In addition, when simulated stair ascending and descending were applied on specimens, three of the remaining specimens passed limits of range of motion resulting in six specimens remaining in the final dataset for stair ascending and descending. The kinematic data were filtered using a second-order low-pass Butterworth filter (dual-passed) with a cutoff frequency of 1 Hz. The kinematics of the knee during cyclical flexion/extension tests were sampled at 0°, 15°, 30°, 45°, 60°, 75° and 90°, and the average data at each angle (of the flexion and extension portions of the cycle) was calculated. Output kinematics variables included the AP positioning of the tibia during the neutral and posterior-force loading, the IE positioning of the tibia during neutral, external and internal moment loading, and the AA positioning of the tibia during the neutral, adduction and abduction moment loading. Three-way repeated-measures ANOVAs were performed for each output variable, considering joint condition (intact versus PCL-deficient), flexion angle and joint loading (simple compression versus simulated muscles) as within-subject variables. These comparisons were performed with a threshold of \( \alpha = 0.05 \), after correcting with Bonferroni correction, for statistically significant differences. The influence of PCL transection on the AP kinematics of the tibia during simulated ADL was of particular interest and was measured at 10% intervals of each motion cycle. The AP kinematics were averaged over the whole cycle during each activity, and this average value was used to compare AP kinematics during ADLs before and after sectioning the PCL. Two-tailed
t-tests were conducted to compare the average AP kinematics of each ADL loading scenario. A threshold of $\alpha = 0.05$ for statistically significant differences, after applying a Bonferroni correction, was again used for this comparison.

### 3.3 Results

**Effect of PCL transection**

Transection of the PCL had a significant effect on the overall kinematics of the joint during neutral motion and posterior-directed loading ($p = 0.005$ and $p = 0.001$, respectively). Transection of the PCL resulted in a slightly more anterior positioning of the tibia under simple compression at $15^\circ$ and $30^\circ$ (Figure 3-4 a); however, with the addition of a posterior-directed tibial force, the tibia was significantly more posterior at all flexion angles after the PCL was cut. When simulated muscle forces were used (Figure 3-4 b), transection of the PCL resulted in a more posterior position of the tibia at all angles, by up to $6.2 \pm 2.9$ mm at $90^\circ$ ($p = 0.001$). Likewise, with the addition of a posterior-directed tibial force, the tibia was significantly more posterior at all flexion angles after the PCL was cut, by up to $9.7 \pm 3.6$ mm at $75^\circ$ ($p < 0.001$).
Figure 3-4 Anterior-Posterior kinematics of the tibia. Mean AP kinematics of the tibia during neutral flexion and 134 N posterior-directed loading with: (a) the compressive force applied, (b) the simulated muscle force applied. Asterisks (*) show a significant difference in AP kinematics after PCL transection during neutral flexion, and downward triangles (▽) show a significant increase in AP kinematics after PCL transection with the addition of posterior-directed force.
The mean neutral IE kinematics of the knee did not change significantly after the PCL was transected with either simulated muscle or compressive forces applied (Figure 3-5) ($p > 0.05$). PCL transection had significant effects on internal and external rotation of the joint ($p < 0.001$). With an internal moment applied, PCL sectioning resulted in increased internal rotation of the tibia at all angles, by up to $3.6 \pm 1.2$ degrees at $90^\circ$ ($p < 0.001$) when a compressive load was applied or $3.2 \pm 1.1$ degrees at $90^\circ$ ($p < 0.001$) when simulated muscle forces were used. During external loading of the joint, with an external moment applied, PCL transection resulted in increased external rotation of the tibia at all angles, by up to $1.5 \pm 0.6$ degrees at $90^\circ$ ($p < 0.001$) when a compressive load was applied or by up to $1.1 \pm 0.5$ degrees at $0^\circ$ ($p = 0.001$) when simulated muscle forces were used.
Figure 3-5 Internal-External rotation of the tibia. Mean IE kinematics of the tibia during neutral flexion, 5 Nm internal and external moment with: (a) the compressive force (b) the simulated muscle load applied. Upward (Δ) and downward triangles (▽) show a significant increase in IE kinematics after PCL transection with the addition of internal and external moment, respectively.
Transection of the PCL resulted in more abducted positioning of the neutral AA kinematics at 90° with a compressive force applied (p=0.042). With an adduction moment applied on the tibia, the PCL-deficient knee was more adducted at 75° by 0.5 ± 0.4 degrees (p = 0.031) and 90° by 0.7 ±0.5 degrees (p=0.014) when a compressive force was applied (Figure 3-6). Moreover, the adduction of the joint did not change significantly when simulated muscle forces were applied (p>0.05). Abduction of the joint was significantly affected by PCL transection (p <0.001). With an abduction moment applied, PCL transection increased the abduction of the knee at all flexion angles by up to 1.0 ± 0.3 degrees at 90° (p<0.001) while a compressive force was applied; however, with simulated muscle forces (Figure 3-6 b), the abduction of the joint increased from full extension to mid flexion (45°) by up to 0.7 ± 0.6 degrees at 0° (p=0.023).
Figure 3-6 Adduction-Abduction of the tibia. Mean AA kinematics of the tibia during neutral flexion, 10 Nm adduction and abduction moment with: (a) the compressive force applied, (b) the simulated muscle load applied. Upward (Δ) and downward triangles (V) show a significant increase in AA kinematics after PCL transection with the addition of adduction and abduction moment, respectively. Asterisks (*) show a significant difference in AP kinematics after PCL transection during neutral flexion.
Activities of Daily Living

AP kinematics of the intact and PCL-deficient knees during simulated activities of daily living are shown in Figure 3-7 and Figure 3-8. During gait, PCL transection did not change these kinematics significantly (Figure 3-7). During stair ascending and descending, however, PCL-deficient knees exhibited a significant posterior offset of the tibia in comparison with the intact knee, on average by $2.4 \pm 1.4 \text{ mm (p = 0.02)}$ and $1.9 \pm 1.4 \text{ mm (p = 0.04)}$, respectively.

**Figure 3-7 Anterior-Posterior kinematics of the tibia during simulated gait.** Average of the anterior/posterior position of the tibia during gait. Solid lines represent AP kinematics of intact knees and dashed lines represent AP kinematics of the PCL-deficient knees. Dotted lines indicate the flexion angle of the joint during each cycle.
Figure 3-8 Anterior-Posterior kinematics of the tibia during simulated stair ascending and descending. Average of the anterior/posterior position of the tibia during: (a) stair ascending and (b) stair descending. Solid lines represent AP kinematics of intact knees and dashed lines represent AP kinematics of the PCL-deficient knees. Dotted lines indicate the flexion angle of the joint during each cycle.
**Effect of joint-reducing load**

The mean neutral AP positioning of the tibia when only a simple compressive force was applied are shown in Figure 3-4 (a), as well as the altered AP positioning of the tibia when a posterior-directed load was also applied. Figure 3-4 (b) shows the corresponding data recorded with simulated muscle loads used instead of the simple compression. Our statistical analyses showed that using simulated muscle forces had a significant effect on AP kinematics during both neutral motion and posterior-directed loading of the joint (p < 0.001). Considering the intact knees, using simulated muscle loads resulted in a more posterior positioning of the tibia when subjected to both neutral (p<0.001) and posterior-directed forces (p<0.001), in comparison with the corresponding motions when a simple compressive force was applied. A similar trend was observed in the PCL-deficient knees (p < 0.001 and p = 0.004, respectively).

The mean neutral IE kinematics, with either a compressive force or simulated muscle loads, are shown in Figure 3-5. These graphs also include the rotational positioning of the joint when internal and external moments are applied to the tibia. Joint reduction loads had a statistically significant effect on the neutral IE rotation of the joint (p = 0.016). As flexion increased, simulated muscle loads caused the tibia to rotate more externally, in both intact (p = 0.015) and PCL-deficient knees (p = 0.020), than when a simple compressive load was used. Joint reduction load had significant effects on internal and external rotation of the joint (p = 0.018 and p = 0.011, respectively). With an internal moment applied to the tibia, using simulated muscle loads resulted in the tibia being less internally rotated, in both intact (p = 0.020) and PCL-deficient knees (p=0.029), than when the simple compressive force was used. Likewise, when an external moment was applied to the tibia, using simulated muscle loading resulted in the tibia being less externally rotated, in the PCL-deficient knee, than when the simple compressive force was used (p=0.006). Figure 3-6 shows the mean neutral AA kinematics of the knee, as well as the AA kinematics with the abduction and adduction moments applied on the tibia. In PCL deficient knees, knees were more adducted (varus) when simulated muscle loads were used in comparison with the compressive force (p = 0.020). When an adduction moment was applied, the resulting position was not sensitive to how the joint was reduced (simulated muscle loads or a simple compressive
Conversely, joint reduction load affected the abduction rotation of the joint (p = 0.001). When an abduction moment was applied, however, the knee was more abducted under simple compressive force loading than simulated muscle loads, in both intact (p = 0.027) and PCL-deficient knees (p < 0.001).

3.4 Discussion

The first objective of this study was to compare kinematics and stability of intact and PCL-deficient knees using a new servohydraulic joint motion simulator. Judging by the neutral path of motion of the knee during flexion under simple compressive loading, our experiment seems to promote knee motions in agreement with well-established patterns. For instance, our results showed that as the intact knee is flexed, the femur translates anteriorly. This behavior, known as “femoral rollback”, was also reported in an in vitro study by Kia et al. [70] and Pinskerova et al. [71], in which the neutral path of AP motion of the tibia of an intact knee is observed to translate anteriorly by flexing the knee, under simple compressive loading. This femoral rollback occurs as a result of tightening of the PCL at full flexion which pulls the tibia anterior. Also during the neutral motion of the joint, the tibia rotated externally as the knee reached full extension. This external rotation of the tibia is known as the “screw-home mechanism”, which will tighten cruciate ligaments and increase the stability of the joint at full extension [72]. This behavior was also previously reported by Markolf et al. [73].

Comparing the neutral paths of motion of intact and PCL-deficient knees, we found statistically significant differences in the AP motions. PCL transection had a significant effect on AP neutral paths of motion at every angle tested when simulated muscle loads were used, but only at 15° and 30° when a simple compressive force was applied. Transection of the PCL did not appear to have a statistically significant effect on IE neutral path kinematics. These findings are supported by a study by Kumagai et al., which found the same AP kinematic patterns as ours for both intact and PCL-deficient knees (the tibial motion during flexion of the knee); however, they reported the kinematics of the knee joint between 20° and 90° only [45]. They also observed a similar anterior tibial translation (4 mm) after transecting the PCL under neutral motion. This trend was not observed under
simulated muscle loading in our study because there was a posterior component of force acting on the tibia increasing with flexion. These findings highlight the importance of carefully considering how the joint is reduced using in vitro models, and how additional scenarios should be considered, when possible.

Comparing the AP kinematics of the knee in response to posterior force, we found significantly more posterior positioning of the tibia after PCL transection in both simulated muscle and compressive loading. Other in vitro studies in which a similar magnitude of the posterior force was applied on the tibia found similar increases in the posterior translation of the tibia after PCL transection [11], [48], [49], [74]. Our results were also supported by the findings of Gollehon et al., in which they observed significant increases in posterior translation under 125 N of posterior force after PCL transection [47]. Our results also showed that when the specimen was extended from 15° to 0°, the intact joint had less translation under posterior-directed loading. This increase in the stability of the joint was caused by the screw-home mechanism of the joint [72]. This agreement between findings obtained using the new joint motion simulator and previous analysis techniques described in previously published studies helps establish confidence in this new testing platform.

The effect of PCL transection on IE kinematics of the knee has scarcely been discussed in the literature. Our findings indicate that when either an internal or external moment was applied to the tibia, the PCL-deficient knees had significantly greater rotations in comparison with the intact knees; regardless of whether the joint was reduced using simple compression or simulated muscle loads. The rotational laxity of the intact knees increased from 0° to 30°. This interesting trend is caused by the screw-home mechanism of the joint as explained before; making the joint stiffer at full extension. Previous in vitro studies quantified this trend and showed an average of 12° internal-external rotational laxity at full extension [47], [72]. An in vitro study by Markolf et al. demonstrated that the rotational laxity of the intact knee increases from 0° to 20° [73]. Thus, when the PCL is transected, the joint could have greater rotational laxity. These results indicate that the IE stability should also be considered when designing new PCL reconstruction techniques, with PCL sectioning having a greater effect on internal rotation (3.6° ± 1.2°) than external rotation (1.5° ± 0.6°).
Comparing the AA kinematics of intact and PCL-deficient knees under abduction and adduction moments, it was observed that abduction increased at all flexion angles in the PCL-deficient knee when the joint was reduced with a compressive force. Conversely, adduction only increased at 75° and 90° due to the PCL transection. Under simulated muscle loads, only the abduction increased significantly from 0° to 45° after PCL transection. These results imply that AA laxity may be less sensitive to PCL-injury than is IE laxity.

To the best of our knowledge, there have not been any studies directly comparing the two loading conditions used in this study (axial compression and simulated muscle loads). Therefore, the second objective of this study was to evaluate the sensitivity of the kinematics in relation to how the joint is reduced. By applying simulated muscle loads, the neutral AP kinematics of the knee was more posterior in both intact and PCL-deficient states. Moreover, the posterior translation of both the intact and PCL-deficient knee due to a posterior force increased significantly in comparison with the compressive force. These effects were likely due to a posterior-directed component of force which increases during flexion in our simulated muscle loads due to the contributions of the virtual hamstrings. The neutral IE kinematics of the knee during simulated muscle loads were more externally rotated in comparison with the compressive force. Surprisingly, during internal loading of the tibia, the knee rotated less internally in both intact and PCL-deficient knees with simulated muscle loads. On the other hand, only the PCL-deficient knees rotated more externally under an external moment when simulated muscle loads were added. The posterior translation of the tibia caused by the simulated muscle loads likely resulted in altered contact mechanics that could be a cause of these significant changes in IE kinematics. Comparing the neutral AA kinematics of the PCL-deficient knee, the knee was more adducted during simulated muscle loading in comparison with the compressive load; however, with the abduction moment added, the simulated muscle loads had less abduction than the compressive loading.

The last objective of this study was to look at the effect of PCL transection on knee kinematics during simulated activities of daily living for comparison with clinical in vivo studies. AP kinematics were focused on because the PCL is a main anteroposterior
stabilizer. The kinematics recorded for simulated gait in this study agree with anterior-posterior kinematics recorded by Benoit et al. for stance phase [35]. They observed that during gait, the tibia moves anteriorly in the beginning of the stance phase and posterior at the end of the stance phase; similar patterns are observed in our study. Other studies have also recorded the same anterior-posterior kinematics of the joint during normal gait for intact knees [75], [76]. In contrast to our hypothesis, no significant difference in AP kinematics during gait was observed after PCL transection; however, during stair ascending and descending, the tibia was generally shifted posteriorly. A Study by Orita et al. showed that PCL dissection translated the tibia anterior during gait which is in contrast with our study [65]. They concluded that this anterior translation could occur by changes in loading pattern of muscles after PCL injury as observed in other studies [77], [78]. Thus, not simulating muscle loads during ADL can be the cause of the difference between Orita’s study and ours. Effect of PCL dissection on kinematics of the joint during stair ascent was investigated by Goyal et al. [79]. Goyal and colleagues observed that the PCL dissection translated the tibia posterior during stair ascent; however, this effect was observed only during the swing phase of the stair ascent cycle. Our findings are also supported by an in vivo study by Iwata et al., who found that during stair descending, the PCL-deficient knee had more posterior translation in its early swing phase in comparison with the intact knee [64]. One interesting finding of this study was that, in contrast to Iwata et al. results, we observed a significant difference in the average position of the tibia throughout the entire motion, rather than at a specific point of the cycle. This difference between our in vitro results and Iwata et al.’s in vivo study, is likely due to our limited ability to accurately simulate muscle loads. Thus, in future studies, use of in vivo muscle loads may increase the accuracy of ADL motion simulations.

A limitation of this study was that testing order could not be randomized, therefore some of the changes in joint behavior may have been a result of joint laxity increasing due to repeated manipulations. Future studies may be able to employ virtual ligaments to parametrically add/remove ligaments of interest in a randomized sequence. Another limitation of this study was the limited range of motion of the VIVO which was reached in three of our specimens. This limitation of the VIVO was more problematic when working with more lax specimens with compromised ligaments. Furthermore, since the specimen
was removed from the VIVO for transection of the PCL, there was the possibility for positioning error when the specimen was mounted back on the VIVO. A correction algorithm based on the position of the tibia at full extension in the baseline scenario was applied to the PCL-deficient results to account for this possible positioning error. Finally, the application of virtual hamstring loadings instead of applying forces through hamstring muscles was another limitation of this study. Although this simplification can reduce the accuracy of our simulation, it was consistent across specimens, and we were still able to achieve a similar AP pattern as other studies [46]. Moreover, in this study, the quadriceps load was simulated by a single load parallel to the femur, whereas a previous study by Ahmed et al. showed that simulating muscle forces with multiple loads acting on each muscle of quadriceps affected the kinematics of the joint [80].

We performed simple tests of knee joint biomechanics before and after PCL transection and measured trends in close agreement with previous literature. Furthermore, we measured the effect of PCL transection for more complicated ADL loading, with results in agreement with reported outcomes from previous in vivo studies of PCL injury patients. Thus, apart from an improved understanding of the influence of PCL injury on knee joint biomechanics, this study demonstrates that new servohydraulic joint motion simulators can be used to analyze knee stabilizers under complex loading conditions. The results of this study also suggest that knee kinematics are sensitive to experimental decisions about how the joint will be reduced and that ADL simulation should be considered for more physiologically relevant measures of joint motion.
Chapter 4

4 The Biomechanical Contribution of Medial Ligaments in the Stability of the PCL-Deficient Knee

This chapter focuses on the biomechanical role of medial ligaments on the PCL-deficient knee. Changes in the translation of the medial side of the joint due to transection of medial ligaments have been analyzed. Further, the load developed in those ligaments to resist the force applied to the joint was measured using superposition. At the end of this chapter, results of this study were compared with published literature on the biomechanics of medial ligaments.

4.1 Introduction

The human knee joint is comprised of various muscles and ligaments that stabilize the joint throughout different motions. Amongst those ligaments, the posterior cruciate ligament (PCL), which prevents the posterior translation of the tibia, is the strongest ligament of the knee [1], [2]. Nevertheless, up to 44% of acute knee injuries happening in sports or vehicle accidents are PCL injuries which could either be isolated or combined with injuries to other ligaments [3]–[7]. Over the past few decades, different reconstruction techniques have been developed in order to regain normal kinematics following a PCL injury; however, studies show that these techniques have high failure rates [10]. Thus, these reconstruction techniques need to be improved, and in order to do that, we need to understand the mechanism of the PCL injury better.

One explanation for the failure of reconstruction techniques may be that 95% of PCL injuries are combined with injuries to other ligamentous structures of the knee joint [2], [3], [8], [11]. Thus, there is a need for better understanding the contributions of other ligamentous structures to joint stability. There have been numerous in vitro studies looking at the contribution of posterolateral ligamentous structures to the knee biomechanics [11]–[14]. Consequently, PCL reconstruction is routinely supplemented with posterolateral reconstruction, if injuries are present. On the contrary, it is still unclear which medial
structures need to be addressed during PCL reconstruction. Few in vitro studies have looked at the contribution of medial structures of the joint to the stability of the intact and ACL-deficient knee [36], [57], [58], [63]. On the contrary, to the best of our knowledge, there have been just three studies which investigated the role of the medial ligamentous complex in the PCL-deficient knee [37], [81], [82]; however, neither of those studies have properly investigated the role of medial ligaments on the stability of the PCL-deficient knee. Two of those studies did not report the magnitude of displacement caused by medial ligament transections. The study conducted by Ritchie and colleagues just focuses on the effect of medial ligaments’ transection on the kinematics of the PCL-deficient knee when the knee is flexed at 90°. Moreover, there has not been any study investigating the biomechanical contribution of the medial ligaments during more complex motions such as activities of daily living. Thus, there is a need for a comprehensive analysis of the PCL-deficient kinematics and influence of medial ligaments’ transection on it.

The primary objective of this in vitro cadaveric study is the examine changes in knee joint kinematics, focused on the medial side AP kinematics, as a result of POL or dMCL transection in a PCL deficient knee. Furthermore, the relative force contributions of each medial ligament in stabilizing both the intact and PCL deficient knee is examined. We hypothesize that transection of either medial ligament will cause increased AP translations on the medial side of the joint. We further hypothesize that, in comparison with the dMCL, the POL force (tension) will be greater in extension but lesser in flexion. Finally, we hypothesize that these medial ligaments will have a greater overall contribution towards maintaining joint stability when an internal moment is applied to the tibia.

4.2 Methods

Specimen Preparation

Ten frozen intact knees (from donors aged 40-63, 3 pairs, 1 male, 5 female) were used in this study. Specimen were selected based on their body mass index (BMI <30) and age (less than 65 years). Specimens were thawed 20 hours prior to testing at room temperature. In order to exclude outlier specimens, the posterior slope of the medial plateau was
measured through CT scanning specimens. The average tibial slope of the medial plateau for all specimens was $10.6 \pm 1.9$ degrees which lies within the reported range measured for normal knees [69]. Biomechanical experiments were performed for each specimen using a VIVO six degrees of freedom (6-DoF) joint motion simulator (Advanced Mechanical Technology, Inc., Watertown, MA, USA). In order to mount the specimens on the VIVO, the femur and tibia/fibula were transected approximately 20 centimeters from the approximate transepicondylar axis (TEA) [83] of the knee. The proximal and distal segment of the specimen were skeletonized from approximately 12 centimeters of approximate TEA to expose bones for potting into pipes using dental model stone (Modern Materials Golden Denstone Labstone, Modern Materials, Kulzer GmbH, Hanau, Germany). The femur was potted (12 cm length of 2” diameter PVC pipe) and mounted to the upper actuator of the VIVO using a custom-made aluminum fixture which allowed adjustment of specimens in all degrees of freedom except flexion. Fixture adjustments were used to align the flexion axis of the specimen with the mechanical flexion axis of the VIVO. The alignment was confirmed using a FARO GAGE digital coordinate measuring machine (FARO Technologies, FL, USA) co-registered to the VIVO’s coordinate system. During alignment, the flexion axis of the specimen was set parallel to the VIVO’s flexion axis by adjusting the internal-external and abduction-adduction of the fixture. To measure this, the tibia/fibula was manually guided from full extension to full flexion and back to full extension by an investigator while applying a small axial load on the tibia. Throughout this motion, the tip of the FARO GAGE was held in a pre-defined hole on the distal tibia approximately 18 centimeters from the flexion axis in order to record the arc of motion of the reference point. The centre of a circle fit to this arc of motion was selected as the estimated flexion axis of the knee. The alignment of the custom-designed fixture in the anterior-posterior, medial-lateral and vertical direction was repeated until the centre of the fitted circle was within 2 mm of VIVO/digitizer’s origin. The fixture was then adjusted in the medial-lateral direction so that midpoint of the medial and lateral epicondyles was approximately at the origin of VIVO coordinate system. Afterward, the tibia and fibula were attached together distally using a wood screw and potted into the tibial pot (8 centimeters length of 5” diameter ABS pipe). The tibial pot was mounted on the lower actuator via an aluminum clamp. Using this mounting technique, the neutral alignment of
the joint resided approximately near the middle of the working range of the lower actuator, minimizing the likelihood of exceeding the simulators allowable ROM during motion testing. Once potting was completed, three arbitrary points on the femoral pot and three arbitrary points on the tibial pot were drilled and digitized to be used for co-registration with reconstructed three-dimensional models in further stages.

*Input Loads and Motions*

Once mounted on the VIVO, a baseline loading scenario was applied to the joint to define the neutral path of the motion for the knee. This loading scenario comprised of continuous cyclical flexion and extension of the knee from 0° to 90° (and back to 0°) over a period of 25 s, reduced using a 10 N axial compressive load (parallel to the long axis of the tibia) applied through the joint centre. All the remaining degrees of freedom were unconstrained and free to move as a result of the applied loads, motions and joint mechanics. Subsequently, this loading scenario was repeated with four additional loading scenarios at two loading levels superimposed on the tibia which represented loads applied on the tibia during clinical tests of the knee. The first loading level which was used for analyzing the kinematics of the joint included: a constant posterior directed force (−67 N), an internal or external torque (± 2.5 Nm), and a posterior force of 50 N combined with an internal torque of 2.5 Nm. The flexion/extension motion was repeated four times over a period of 100 s for described loading scenarios and all the kinematics were sampled at 100Hz. The second loading level was applied on specimens in order to measure the force contribution of each ligament which included the same cyclical loading scenarios with their magnitude doubled. The second loading level was applied on the intact and the PCL-deficient knee, and the recorded kinematics were applied back on the specimen at each stage of the experiment while the joint’s resistance (joint torque and posterior load) to the motion was measured. Based on the superposition principle, the change in the joint’s reaction forces following a ligament transection was associated with the load applied by the ligament. For the simulated clinical tests, the load applied by each ligament was reported as its percent contribution to the resistance load of the intact and PCL-deficient knee.
Following cyclical flexion/extension loading scenarios, activities of daily living (ADL) including gait, stair ascending and descending were applied to the specimen using “AVER75” loading data by Bergmann et al. (https://orthoload.com) [68]. All degrees of freedom were operated in force-control mode, except for flexion of the joint and all the loads were reduced by 75% to prevent damage to soft tissues. At each stage of the study, ADLs were simulated for four cycles over a period of 100 s and resulting kinematics were recorded with a sampling rate of 100 Hz. The kinematics recorded from the intact and PCL-deficient stage were then applied back on the specimen to measure the resistance load of the joint following ligament transections. Similar to the simulated clinical tests, the load applied by each ligament was measured by measuring the change in the joint’s resistance load.

Recorded kinematics and forces were smoothed using a second-order Butterworth filter and down-sampled to 1024 sample points. For cyclical flexion/extension loading scenarios, the average of the flexion and extension phases of cycles was calculated at 30° increments of flexion. For ADL, the kinematics of the joint were calculated at every 10% of the cycle.

**Simulated Ligamentous Injury**

After biomechanical testing of the intact knee was computed, the PCL was transected arthroscopically by a surgical fellow (PR) trained by an orthopaedic surgeon specializing in soft tissue reconstruction (AG) to simulate a grade II injury. The PCL-deficient knee was subjected to the same kinematics and loads as the intact knee and the resulting biomechanics were recorded. Following biomechanical testing of the PCL-deficient knee, the surgical fellow separated medial ligaments by a scalpel based on projection and thickness of their fibers. These medial ligaments were then tagged by sutures for further transection of these ligaments by the operator in order to reproduce a grade III injury. Subsequently, either the POL (4 specimens, randomized) or the meniscofemoral section of the dMCL (remaining 4 specimens) was transected via a surgical window on the medial side of the knee, which was closed using surgical staples afterward. Following simulated injury to medial ligaments, the knee was subjected to the aforementioned loads and motions to measure the kinematics of the joint. This process was repeated after transection of the
other ligament (POL or dMCL). Finally, the sMCL was transected and the specimen was subjected to the intact and PCL-deficient knee kinematics to measure the joint's resistance. Dissection of all medial ligaments was carefully done by the trained operator using a scalpel. Figure 4-1 shows a flow chart of the protocol used in this study to measure the kinematics of the knee and the load response of ligaments.

![Flow chart of the protocol](image)

**Figure 4-1** The protocol used in this study to measure the kinematics of each stage as well as the load response of each ligament

*Measuring Medial Tibial Translation with Respect to the Flexion Axis*

After biomechanical testing of knees was complete, models of bony anatomy were created from CT scans using a threshold-based segmentation technique and a marching-cubes reconstruction algorithm. Reconstructed femurs and tibias were then co-registered on the VIVO's coordinate system by using the drilled points on the femoral and tibial pots, which were identifiable in the CT reconstructions, as registration landmarks. Once co-registered, two spheres were fit to the posterior aspects of the femoral condyles using in-house code developed in MATLAB (MathWorks, Natick, MA, USA) and a line passing through their centres was selected as the geometrical centre axis (GCA) [83]. To enable consistent identification of the most medial point across the tibiae of all specimens, one specimen was selected as the base model and other tibias were scaled to the same medial/lateral width as that specimen. Scaled models were co-registered to the position/alignment of the base tibia
using iterative closest point (ICP) in MeshLab (National Research Council, Rome, Italy). A line passing through the most medial and lateral points of the base model was used to identify each specimen’s medial point. From this data, we could compare the anatomic GCA of each mounted specimen to the mechanical flexion axis of the VIVO. When the joint was at full extension, the average absolute misalignment error of our method was $5.3^\circ \pm 2.4^\circ$ in the transverse plane and $3.6^\circ \pm 2.4^\circ$ in the frontal plane. On each specimen, the closest medial point to the line was selected as the specimen’s medial point. Once medial points were selected, the recorded kinematics was converted into the Cartesian coordinate system using a custom-developed MATLAB code (Appendix A). The kinematics were then applied on the reconstructed models with the femur flexing and extending along its flexion axis, and the tibia moving in all remaining directions. During these simulations for each specimen, the AP position of the medial point relative to the flexion axis was recorded (Figure 4-2).

![Figure 4-2 The kinematics of the medial point with respect to the femoral flexion axis](image)

*Data analysis*

Two specimens were excluded from our dataset due to damage to pots during experiments (one specimen) or reaching the range of motion limits of the VIVO in the internal rotation direction (one specimen). After transection of each ligament, the translation of the medial point under superimposed loads (relative to its position during baseline) was compared using a three-way repeated-measures ANOVA with a significance value of $\alpha = 0.05$. This comparison was done considering specimen condition (intact, PCL transected, first medial
ligament transection and second medial ligament transection), the order of medial ligament transection and flexion angle. All ADLs were subdivided into their stance and swing phases based on the magnitude of the ground reaction force. The portion of the motion cycle where the ground reaction force exceeded 100 N was considered as the stance phase of the cycle and the portion where the ground reaction force less than 100 N was considered as the swing phase. During ADLs, medial joint translation relative to the baseline at 0° flexion angle were compared using a mixed three-way repeated-measures ANOVA. In this comparison, the condition of the joint and the flexion angle were considered as within-subject variables and the medial ligament cutting sequence was considered as a between-subject variable. Furthermore, to analyze the effect of flexion angle on the contribution of each ligament, a one-way repeated-measures ANOVA with a significance level of α = 0.05 was used. A Bonferroni correction was used in all comparisons.

4.3 Results

**Medial kinematics**

*Simulated Clinical Tests*

Translations of the medial section of the tibia under 67 N posterior-directed load are shown in Figure 4-3. In the POL cohort, the transection of the PCL increased the posterior translation of the medial point at 30°, 60° and 90° by up to 10.0 ± 7.4 mm at 90° (p=0.01). Likewise, for the dMCL cohort, medial joint translations when a posterior-directed load was applied increased after PCL transection, at 60° by 8.1 ± 6.4 mm (p=0.01) and 90° by 11.2 ± 7.4 mm (p<0.01) with respect to intact PCL kinematics. Subsequent transection of either medial ligament did not change the kinematics of the medial point significantly. When the posterior-directed load was combined with an internal torque, PCL transection increased the posteromedial translation of the tibia in both cohorts at 60° and 90°, up to 3.3 ± 1.7 mm at 90° (p<0.01) in the POL cohort and 4.3 ± 1.7 mm at 90° (p<0.01) in the dMCL cohort (Figure 4-4). Isolated transection of the POL and further transection of the dMCL did not have a measurable effect on the posteromedial translation of the tibia under the paired loading. Transection of the dMCL (with remaining medial ligaments intact) had
no statistically significant effect on kinematics; conversely, subsequent transection of the POL increased the posteromedial translation of the joint at full extension by 2.6 ± 2.5 mm (p=0.048).

**Figure 4-3** Anterior-Posterior kinematics of the medial joint during posterior loading. Mean AP translation of the tibial medial point during 67 N posterior loading for (a) the POL and (b) dMCL cohort. Error bars represent the standard deviation for each stage. Asterisks (*) show significant increases in the posterior translation after transection of the PCL.
Figure 4-4 Anterior-Posterior kinematics of the medial joint during combined loading of the joint. Mean AP translation of the tibial medial point during simultaneous 50 N posterior-directed load and 2.5 Nm Internal torque for (a) the POL and (b) dMCL cohort. Error bars represent the standard deviation for each stage. Asterisks (*) show significant increases in the posterior translation after transection of the PCL. Triangles (Δ) show significant increases in the posterior translation after transection of the POL.
Figure 4-5 and Figure 4-6 show the medial joint translations as a result of internal or external torques applied to the tibia, across varying stages of ligament transection. With an applied internal torque, the internal rotation of the tibia resulted in the posterior translation of the medial joint. We measured a significant increase in the posteromedial translation of the intact knee after the PCL transection. This increase occurred at 90° flexion in both the POL and dMCL cohort by 2.0 ± 1.5 mm (p=0.01) and 2.5 ± 1.5 mm (p<0.01), respectively. Isolated transection of the POL or dMCL had no measurable effect on the medial joint translation with an applied internal torque at any angle of flexion (p>0.05) (Figure 4-5), nor did a simultaneous transection of both ligaments (p>0.05). With an applied external torque, the ANOVA did not identify a significant change in the medial joint translation due to the PCL transection in either cohort (p>0.05). Isolated transection of the POL increased the anterior translation at all flexion angles by up to 2.9 ± 2.7 mm at 0° (p=0.03); however, further transection of the dMCL did not affect the kinematics in the POL cohort. No statistically significant change in the kinematics was measured after transection of the dMCL (p=0.10) and POL (p=0.10) in the dMCL cohort.
Figure 4-5 Anterior-Posterior kinematics of the medial joint during internal loading of the joint. Mean AP translation of the tibial medial point during 2.5 Nm Internal torque for (a) the POL cohort (b) the dMCL cohort. Error bars represent the standard deviation for each stage. Asterisks (*) show significant increases in the posterior translation after dissecting the PCL.
Figure 4-6 Anterior-Posterior kinematics of the medial joint during external loading of the joint. Mean AP translation of the tibial medial point during 2.5 Nm external torque for (a) the POL cohort (b) the dMCL cohort. Error bars represent the standard deviation for each stage. Asterisks (*) show significant increases in the posterior translation after transection of the POL.
**Activities of Daily Living**

The medial joint translation during simulated gait, stair ascending and descending are shown in Figure 4-7. During simulated gait, transection of the PCL and medial ligaments did not have a significant effect on translations of the medial point. There were also no statistically significant differences when comparing the medial kinematics of the tibia after transecting either the PCL, dMCL or POL during simulated stair ascending (Figure 4-8). During simulated stair ascending, the ANOVA did not identify any significant changes after transections of the PCL and medial ligaments (p>0.05).

![Simulated Gait](image1)

**Simulated Gait**

- **Figure 4-7 Anterior-Posterior kinematics of the medial joint during simulated activities of daily living.** Mean AP translation of the tibial medial in the intact knee point during simulated (a) gait (b) stair ascending (c) stair descending. Error bars represent the standard deviation of the AP translation.
Figure 4-8 The change in the anterior-posterior kinematics of the medial joint. Change in the translation of the medial joint relative to the intact knee due to transection of each ligament during simulated activities of daily living for (a) the POL and (b) dMCL cohort. Error bars represent the standard deviation of the change.

**Force and moment contribution**

**Simulated Clinical Tests**

Figure 4-9 (a) and (b) show the average contribution of the PCL to the stability of the intact knee with an isolated posterior-directed load and posterior-directed load combined with an internal torque, respectively. With a posterior-directed load applied on the tibia, the applied load was resisted by forces developed in soft tissues and articular components; up to 88% ± 14% of this load was resisted by the PCL tension. The flexion angle had a significant effect on the PCL’s contribution (p<0.05). When a posterior-directed load was combined with the internal torque, the PCL’s contribution to the resistance force of the joint increase...
significantly with increasing the flexion angle (p<0.05); however, the maximum contribution of the PCL was only 67% ± 20%, unlike in the isolated posterior loading scenario (Figure 4-9).

![Figure 4-9 Force contribution of the PCL in intact knees. Force contribution of the PCL to the posterior resistance force developed in the joint in response to (a) a 134 N posterior-directed load and (b) a 100 N posterior-directed load and 5 Nm internal torque.](image)

In the PCL-deficient knee, the dMCL, POL, and sMCL had small contributions to the load response of the joint during posterior loading of the tibia compared to other structure of the joint; however, their contributions increased as the posterior loading was paired with the internal torque (Figure 4-10). During the isolated posterior loading, the dMCL’s and POL’s contributions to the load response of the joint were not affected by the flexion angle (p>0.05); however, the sMCL’s contribution significantly increased from 7% ± 6% at 0° to 14% ± 9% at 30° (p=0.048). When a combined posterior-directed force and internal torque was applied to the tibia, the changes in the contribution of the dMCL and sMCL were not statistically significant. Flexion increased the sMCL’s contribution to the load response of the joint from 11% ± 10% at 0° to 42% ± 24% at 30° (p=0.027).
Figure 4-10 Force contribution of medial ligaments in PCL-deficient knees. Force contribution of medial ligaments to the posterior resistance force developed in the PCL-deficient knee in response to (a) 134 N posterior-directed load (b) 100 N posterior-directed load and 5 Nm internal torque.

The contributions of medial ligaments to the rotational stability of the knee during various loading scenarios are shown in Figure 4-11 and Figure 4-12. In the PCL-deficient knee with an applied internal torque, the contribution of the dMCL was greatest at 30°, contributing up to 23% ± 23% of the total restraint torque and the flexion angle of the joint did not affect its contribution significantly (p>0.05). By flexion of the joint from 0° to 60°, the contribution of the POL decreased to 9% ± 6% (p= 0.033). Conversely, by flexion of the knee from 0° to 30°, the contribution of the sMCL to rotational stability increased from 14% ± 19% to 30% ± 26% (p=0.014), as shown in Figure 4-12.

The contribution of medial ligaments to the rotational stability of the PCL-deficient knee when the tibia was subjected to a 5 Nm external torque is shown in Figure 4-11. For the applied external torque, the ANOVA indicated a significant decrease in the dMCL’s contribution from 12% ± 5% at 30° to 5% ± 3% at 60° (p=0.003). The POL’s contribution was also affected by the flexion angle, decreasing from 12% ± 4% at 0° to 3% ± 4% at 30° (p=0.031). The sMCL provided the largest contribution amongst medial ligaments, and its contribution increased from 21% ± 11% at 0° to 73% ± 7% at 60° (p=0.025).
When the 100 N posterior-directed load and 5 Nm internal torque were applied to the tibia simultaneously, the dMCL’s contribution to the rotational stability was greatest at 60° by contributing 24% ± 20%; however, its contribution was not sensitive to the flexion angle (p>0.05). The POL’s contribution was affected by the flexion angle and decreased from 35% ± 18% at 0° to 8% ± 4% at 60° of flexion (p=0.023). As shown in Figure 4-12 (b), during this combined loading of the tibia, the sMCL contributed 18% ± 17% to rotational stability of the joint, and by flexion of the knee to 30° this contribution increased to 40% ± 25% (p<0.01).

![5 Nm External Torque](image)

**Figure 4-11 Moment contribution of medial ligaments during external loading.** Contribution of medial ligaments in the IE direction in the PCL-deficient knee when the tibia was subjected to a 5 Nm external torque.
Figure 4-12 Moment contribution of medial ligaments during different loading scenarios. Contribution of medial ligaments in the IE direction in the PCL-deficient knee when the tibia was subjected to a (a) 5 Nm internal torque (b) 100 N posterior load and 5 Nm internal torque.
Activities of Daily Living

The AP load response of the PCL and medial ligaments during simulated activities of daily living are shown in Appendix A. During simulated gait of the intact knee, the maximum anterior load applied by the PCL was $3.1 \pm 14.6$ N. The maximum posterior loads applied by the dMCL, POL and sMCL was $3.4 \pm 8.4$ N, $2.9 \pm 4.6$ N and $3.1 \pm 10.2$ N, respectively. The average contribution of dMCL and POL increased significantly by the PCL transection by $2.3 \pm 3.9$ N (p<0.01) and $0.7 \pm 2.1$ N (p=0.002) in the anterior direction, respectively. During the simulated stair ascending and descending, there were no statistically significant differences found comparing the load response of medial ligaments in the intact and PCL-deficient knee (p>0.05). The maximum anterior load applied by the PCL in the intact knee during simulated stair ascending and descending was $10.4 \pm 26.4$ N and $10.9 \pm 22.7$ N, respectively.

4.4 Discussion

This in vitro experiment provides new insights into the biomechanical contribution of medial ligaments in translational and rotational stability of the tibia. The first objective of this study was to measure and compare the kinematics of the medial side of the knee following transection of the PCL and two of the medial ligaments, the POL and dMCL. Comparing the relative anteromedial and posteromedial translation of the tibia during simulated clinical tests, significant differences were identified after transection of the PCL and medial ligaments; however, no measurable differences were found in the kinematics of the medial joint during simulated activities of daily living.

Studies show that the dMCL and POL get injured prior to the sMCL [84], [85]. Our medial ligaments sectioning order followed the clinically observed order of injuries. Various studies focused on the stabilizing role of medial ligaments in the intact knee joint [36], [58], [86]. Robinson and colleagues recorded the kinematics of the tibia with 150 N posterior drawer test and applied 5 Nm internal-external torque [36]. They did not observe any significant change in internal-external laxity of the intact joint after dissection of either dMCL or POL. Coobs et al. compared the rotational laxity of the knee after transection of
POL and sMCL, and noted a significant increase in the rotational laxity after both ligaments were transected [58]. A small number of studies also investigated the role of medial ligaments in ACL-deficient knee. Haimes et al. reported an increase in the rotational laxity of the joint when ACL transection was followed by the medial ligament transection [57].

To the knowledge of the author, the study performed by Ritchie and his colleagues is the only study that quantified the translation of the tibia in PCL-deficient knees [37]. They reported an increase in the posterior translation of the flexed (90°) PCL-deficient knee after transection of the POL and dMCL when they applied posterior-directed load and simultaneous posterior-directed load and internal torque. Conversely, our results showed that transecting neither the dMCL nor POL had measurable effect on the posteromedial translation of the tibia with an applied posterior load or simultaneous posterior load and internal torque. This contrast in our results can be due to intra-specimen variability since the increase observed in Ritchie’s study were less than 0.6 mm and a small rotation of the tibia can cancel that translation on the medial side. This contrast might also be caused by a difference in loading methods. During the combined loading of the tibia, a significant change in the posteromedial translation was noted when the second medial ligament (POL) was transected in the dMCL cohort. Thus, in the PCL-deficient knee, the POL had a more substantial role in stabilizing the joint than the dMCL when subjected to simultaneous posterior load and internal torque.

A previous study by Coobs et al. was conducted to analyze the role of POL and sMCL [58]. They observed a significant increase in the rotational laxity of the tibia when both POL and dMCL were transected with the maximum change occurring at 90°. The role of these ligaments in rotational stability of the intact knee was further analyzed by Robinson et al.[36]. The result from the subsequent transection of medial ligaments in their study concluded that the POL and dMCL do not affect the kinematics of the intact knee significantly and the sMCL acts as the primary restraint to the rotation. In contrast to the intact knee, we observed an increase in the anteromedial translation of the PCL-deficient knee when subjected to external torque. This contrast in our results is likely due to having more external rotation in the PCL-deficient knee and therefore more engagement of the
POL. Similar to the study conducted on the intact knee, we did not find any significant effect after transection of the POL and dMCL when an internal torque was applied.

One of the advantages of this study compared to others was the capability of the joint motion simulator used in this study to simulate complex motions such as activities of daily living. Our data indicated that during simulated gait, stair ascending and descending, no statistically significant change occurs in the posteromedial and anteromedial translation of the tibia due to the transection of medial ligaments. This finding is not surprising since, during those activities, the compressive force applied to the joint and the contact forces caused by it have a greater role in stabilizing the knee.

An interesting finding of this study was that in the PCL-deficient knee, medial ligaments contributed up to 88% + 21% collectively when the posterior load was combined with the internal loading. This can imply that under the posterior loading paired with an internal torque, medial ligaments are under higher tensions and can have a higher chance of rupture. As the main rotational stabilizers of the knee, medial ligaments had the highest contribution to IE rotation and amongst those medial ligaments, the sMCL had the greatest contribution to the rotational stability. The POL and dMCL had a greater contribution to the rotational stability when the internal torque was applied, to the point that at some flexion angles, their contribution was greater than the sMCL. This study is one of the first in vitro studies that have investigated the load response of ligaments during simulated activities of daily living. The load applied by the PCL and medial ligaments in response to the simulated activities of daily living were minimal and their directions varied from anterior to posterior depending on the anterior translation of the tibia. These low magnitude load responses of ligaments can also be produced by the VIVO’s error in reproducing the kinematics of the intact and PCL-deficient knee. Thus, we cannot be certain that the measured changes in the load response of the knee are representative of ligament forces since ADLs do not engage the PCL and medial ligaments substantially.

A prior study conducted by Griffith et al. noted a decrease in the POL’s response to the 5 Nm internal torque as the knee was being flexed from 0° to 90°, and under the same loading scenario, the sMCL’s load increased from 0° to 60° and decreased from 60° to 90°.
The exact same pattern was observed in the PCL-deficient knee in our study. On the other hand, in another study by Coobs et al. opposite patterns were observed using the superposition method, as the POL had a larger load response to the external torque than the internal torque [58].

One of the limitations of this study was the assumption regarding some of the loading scenarios used. Loads used to simulate activities of daily living of the intact knee were gathered using instrumented TKRs (https://orthoload.com) [68]; therefore, extrapolations to the intact knee may not be reliable. Moreover, these loads were reduced to 25%, a method used by other studies to prevent damage to the specimen [87]. In addition, another limitation of this study was the accuracy of the joint motion simulator in applying the desired loads to the specimen. The maximum observed error during force-control testing of the knee was 11.5 N for the posterior-directed load and 0.7 Nm for internal-external torque. The last limitation of our study was the small sample size (n=4) for each cohort.

In summary, this study compared the kinematics of the PCL-deficient knee following sequential transection of the dMCL and POL and our results indicate that the dMCL restrained the simultaneous posterior and internal motion of the tibia, and the POL restrained the external motion. Although the transection of these ligaments does not change the kinematics significantly, they have a notable force and moment contribution to the stability of the knee. Medial ligaments of the knee did not have a significant role during simulated activities of daily living, mostly because of the nature of these activities (mostly anterior-directed loads). Joint kinematics appears to be relatively insensitive to the status of the medial ligaments, but they are subjected to greater loads after PCL transection when the tibia was subjected to a posterior load. Comparing the POL and dMCL, reconstructing the POL might result in more successful PCL reconstruction since the POL seems to have a higher contribution to the stability of the medial joint than the dMCL, both in kinematics and load contribution. Clinical studies would be required to further test this conclusion since throughout most ADL these ligaments had very small contribution and their clinical importance may be negligible.
Chapter 5

5 General Summary and Future Works

This chapter reviews the objectives and hypotheses of the current study and the experiments conducted to achieve those objectives. This chapter also discusses the limitations and strengths of the methods used in this study. Additionally, the future directions that this study can take and the significance of this study in possible modifications for available PCL reconstruction techniques are considered.

5.1 Summary

An injury to the posterior cruciate ligament of the knee is one of the common knee injuries in sports and vehicle accidents. This injury will result in abnormal kinematics and therefore cause early osteoarthritis by increasing the contact pressure. As a solution for abnormal kinematics, different surgical techniques have been developed to reconstruct the PCL by placing soft tissues from different part of the body in the PCL's place. Prior studies show that these techniques are not successful in all the cases, and they need to be modified. Injury to the PCL is, in most cases, coincident with injury to other ligaments of the knee. Thus, reconstruction of other injured ligaments may result in more successful PCL reconstructions. Because there is a lack of studies that have investigated the role of medial structures in the stability of the PCL-deficient knee, it is still unclear which medial ligaments need to be reconstructed additionally in the PCL-deficient knee. The majority of our understanding of the role various ligaments have in the stability of the human knee has been achieved through cadaveric studies. Cadaveric studies are limited by the lack of information on loads that represent complex motions and the capability of apparatuses used to simulate those motions. As a result, most cadaveric studies have characterized ligaments under simple motions such as anterior-posterior loading of the tibia. Thus, there is inadequate data on how each ligament comes into play during complex motions such as walking. Recently, servohydraulic joint motion simulators have been used to investigate implants subjected to activities of daily living [42].
In Chapter 3, the capability of the VIVO joint motion simulator in simulating knee joint biomechanics was assessed by comparing its results with results from prior studies that investigated how the PCL transection affect the kinematics (Objective 1, chapter 3). The agreement between the findings of Chapter 3 and previously published literature on the effect of the PCL on the kinematics indicated that the new servohydraulic joint motion simulator is suitable for analyzing knee stabilizers. The second objective of this study was investigating the effect that the PCL can have on the kinematics of the tibia during activities of daily living (Objective 3, Chapter 3). During simulated activities of daily living, PCL transection resulted in posterior offset of the tibia; however, this change in the position of the tibia was only significant during stair ascending and descending. This study also suggests that the kinematics of the knee is sensitive to how the joint is reduced (simulated muscle forces vs. axial compressive load) (Objective 2, Chapter 3).

With increased confidence in the VIVO, this platform was then used to investigate other stabilizers of the knee under more complicated motions. The role of medial structures in the stability of the intact and ACL-deficient knee was analyzed in prior studies; however, there was scarcely any info on the effect of the medial structures on the kinematics of the PCL-deficient knee. The kinematics of the medial tibia in the PCL-deficient knee were analyzed in Chapter 4 to determine whether or not the transection of the POL and dMCL has any effect on PCL-deficient knee kinematics (Objective 4, Chapter 4). The result of this study showed that the POL only had a significant effect on kinematics when an external moment was applied and the dMCL affected the kinematics when a simultaneous posterior-directed load and internal moment were applied on the tibia. However, the change in the kinematics was small, and perhaps not clinically significant. While these ligaments had a significant effect on the kinematics of the medial tibia during simulated clinical tests, neither the POL nor dMCL affected the kinematics significantly when activities of daily living were simulated.

Another way to analyze the biomechanical effect of these medial ligaments is to investigate how they contribute to the stability of the joint through the load they exert on the joint during motions (Objective 5, Chapter 4). Results of this study showed that during most of the tested loading scenarios, the sMCL had a large force contribution. Moreover, the dMCL
and POL had up to 50% contribution collectively to the stability of the PCL-deficient knee when an internal moment was applied. This collective contribution decreased as the knee was flexed; however, the flexion angle had a completely opposite effect on the contribution of each individual ligament. By flexing the joint from 0° to 90°, the POL’s contribution decreased and the dMCL’s contribution increased gradually. These changing patterns in the contribution of the dMCL and POL are logically in agreement with the anatomy of these ligaments, indicating the POL and dMCL are taut at extension and flexion, respectively.

This work has shown that the posterior oblique and deep medial collateral ligament have significant effects, although small, on the rotational stability of the PCL-deficient knee. Even though those ligaments had a small contribution to the kinematics of the joint, they had an important contribution to resisting forces and moments that act to posteriorly displace the medial side of the tibia. This contribution was at its maximum when the knee was at extension. Therefore, damage to the medial structures can affect the load balance of the joint and result in larger loads being exerted on the reconstructed PCL and its failure. It is important that further work is done to investigate the role of medial ligaments on the kinematics of the PCL-deficient knee further by analyzing various injury scenarios of those ligaments. Complete understanding of medial ligaments’ roles could suggest additional reconstruction of medial ligaments in the process of the PCL reconstruction to achieve more natural kinematics.

5.2 Limitations and Strengths

In the first part of this study (Chapter 3), surgical transection of the PCL required by dismounting the specimen along with a part of the custom-designed fixture from the VIVO which resulted in some positioning errors. Those positioning errors altered the anterior-posterior alignment of the specimen on the VIVO at 0° flexion angle by an average of $2.6 \pm 2.4$ mm. A correction algorithm was used to account for this positioning error. This positioning error was minimized in the second part of this study by removing the whole fixture during transections. Another limitation of this study was simulating muscle forces with constant loads while, in reality, the magnitude of the load exerted by each muscle
depends on the flexion angle. Even though this simplification can reduce the accuracy of this simulation, we achieved the same anterior-posterior motion as other studies.

The loads used to represent activities of daily living was another limitation of the study. Firstly, ADL loads used in this study were gathered through an instrumented total knee replacement; therefore, they may not be representative of intact knees. Secondly, ADL loads were reduced to 25% to prevent damaging the ligaments, a method used by other studies. This reduction has a small effect on our study since all specimens are subjected to the same loads. Another limitation of this study was the force application uncertainty of the VIVO which was ± 11.5 N for the anterior-posterior load and ± 0.7 Nm for the internal-external moment. The last limitation of this study was the small sample size we had for each cohort. A statistical analysis using G*Power software (Heinrich Heine University, Düsseldorf, Germany) showed that in order to have a large effect size with the power of 0.8, a minimum sample size of 15 is required.

Despite our limitations, our study had a number of strengths. Other studies have analyzed the effect of different simulated muscle loads on joint kinematics. For instance, how applying just quadricep loads can result in different kinematics compared to applying both quadriceps and hamstring loads. This study was the first to directly compare the effect joint reduction method can have on the joint kinematics of both the intact and PCL-deficient knee. This study also is the first to investigate the effect of these joint reduction methods (simulated muscle loads vs. axial load) on the response of the joint to superimposed loads.

The new joint motion simulator used in this study allowed us to investigate the role different ligaments play during more complex motion such as gait. During each activity of our daily lives, different sets of loads and moments are being applied on our knees. Thus, understanding how knee ligaments contribute to the stability of the knee during those motions can help surgeons decide to treat a ligament injury operatively or non-operatively. For example, if an injury to a ligament does not affect the kinematics significantly, the patient can resume activities of daily living and compensate for the injured ligament by training the surrounding muscles over time. Previous *in vivo* studies have investigated the effect of the PCL injury during a few activities of daily living. One of the main limitations
of these *in vivo* studies is the intra-patient variability which makes their comparison unreliable. This study was the first *in vitro* study to measure the contribution of medial ligaments in the PCL-deficient knee.

Another strength of this study was its focus on the kinematics of the medial joint. Previous studies on the role of medial kinematics focused on either AP translation or IE rotation of the whole tibia [36], [57], [58], [63]; however, in the knee joint, these motions happen in combination with each other causing different kinematics at different parts of the joint. Therefore, if we focus on the motions of the whole tibia rather than focusing on the area that these ligaments are supposed to stabilize, we might not fully understand the contributions of these ligaments.

### 5.3 Future Work

One interesting future study would be to develop a system capable of simulating more accurate muscle loads. Quadriceps and hamstrings apply different loads during flexion to extension than during extension to flexion. Hamstrings flex the joint by applying tension to the posterior portion of the tibia, whereas the quadriceps extend the knee by applying loads to the anterior portion of the tibia. Therefore, loading scenarios applied to the tibia can have different effects during flexion motion than during the extension motion. For instance, a posterior-directed load applied to the tibia during flexion motion may cause injury to the PCL. Thus, the dynamic simulation of muscle loads can lead to a better grasp of the stabilizing effect of ligaments.

Since our results indicate that the sMCL had a large load contribution to the stability of the PCL-deficient knee, it would be interesting to analyze how the POL and dMCL act in the absence of the sMCL. The result of this study showed that the sMCL had a slightly larger contribution in load response of the joint. Thus, in the absence of the sMCL, other medial ligaments may contribute more to the load response of the joint. As a result of increased load applied by those ligaments, their transection may significantly affect the kinematics of the PCL/sMCL-deficient knee.
The activities of daily living investigated in this study relied on the contact pressure for stabilization of the joint; therefore, in order to take this analysis a step further, investigation of ADLs with more extreme AP or IE loadings such as jumping forward is required. This apparatus can also be used to investigate the effect of the ACL on kinematics since it was observed in Chapter 3 that the large axial load present in ADLs in combination with the posterior slope of the tibia translated the tibia anteriorly.

One of the interesting features of this newly developed joint motion simulator is the capability of the device to introduce nonlinear virtual springs. In this feature, two arbitrary points in space can be identified as the insertions of the virtual spring and the VIVO's control system will apply the appropriate loads on the specimen based on the properties of the nonlinear spring including stiffness, length and slack length. Therefore, the results of this study, in combination with this feature, can be very useful in tuning existing ligament models. So far, the existing ligament models were developed and tuned based on how closely the model can follow the kinematics recorded during experiments. Thus, tuning of ligaments can be affected by how fine other structures of the joint are modeled. This new feature opens the stage for a hybrid computational-experimental modeling and tuning a ligament based on how it changes the kinematics of the real joint. For instance, an additional study would be to optimize the insertion and stiffness of the virtual ligaments based on the data that was gathered on ligament loads and then test the optimized ligaments under same loading scenarios to investigate how natural the kinematics can get. This additional study requires the development of a protocol that allows data processing and optimizations to happen along with the experimentation.

5.4 Significance

This study is the first to use the VIVO joint motion simulator for analyzing the biomechanical role of knee ligaments. This new servohydraulic joint motion simulator not only allows the analysis of the knee under more complicated motions but also opens the floor for a new type of studies, hybrid computational-experimental studies. This study was also the first to properly investigate the role of medial ligaments on the kinematics of the PCL-deficient knee under more complicated motions such as activities of daily living. The
knowledge from this study could aid surgeons in deciding what medial structures to address during PCL reconstruction. Since our result showed that the PCL and medial ligaments share some of the load together, not repairing those ligaments may place higher demands on the reconstructed PCL and cause reconstruction failure.
References


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Appendices

Appendix A. The Load Response of Each Ligament to ADLs

The anterior-posterior load applied by each ligament in response to simulated activities of daily living. Solid lines represent the load that a ligament applied in the intact knee. Dashed lines represent load applied by a ligament in the PCL-deficient knee.

Appendix A. 1 Load response of ligaments during simulated gait. Load response of (a) the PCL, (b) dMCL, (c) POL and (d) sMCL to simulated gait in the AP direction at 10% increments of the cycle. Solid lines represent the response of ligaments in the intact knee, and dashed lines represent the responses in the PCL-deficient knee.
Appendix A. 2 Load response of ligaments during stair ascent. Load response of (a) the PCL, (b) dMCL, (c) POL and (d) sMCL to simulated stair ascending in the AP direction at 10% increments of the cycle. Solid lines represent the response of ligaments in the intact knee, and dashed lines represent the responses in the PCL-deficient knee.
Appendix A. 3 Load response of ligaments during stair descent. Load response of (a) the PCL, (b) dMCL, (c) POL and (d) sMCL to simulated stair descending in the AP direction at 10% increments of the cycle. Solid lines represent the response of ligaments in the intact knee, and dashed lines represent the responses in the PCL-deficient knee.
Appendix B. In-House Developed Codes for the Medial Joint Kinematics

The “Run_all_medial_study_specimens” contains the specimen ID for each specimen and the point coordinate of the most medial and lateral points on both tibia and femur. “Process_medial_study_specimen_V2” receives the data for each specimens and starts reading all the files for the specimen. In the next step, “Extract_data_general_V4” filters and calculates the kinematics of the specimen at 30 degrees increments. “VIVO_Positions_updated_V4” then aligns the femoral and tibial model based on calculated kinematics and reports the position of the most medial point relative to the femoral flexion axis.

Appendix B. 1 Flow chart of functions used in data processing of medial tibial kinematics
Appendix B.(i) Run_all_medial_study_specimens

make_models=1;
anchoring=4;
specimen='GL1707145L';
fem_pts=[38861 64874]; % Node ID of the medial and lateral points on the femur
tib_pts=[85481 73434]; % Node ID of the medial and lateral points on the tibia
rot=-5
% Calls the "Process_medial_study_specimen_v2"
Process_medial_study_specimen_V2(specimen,R_offset,fem_pts,tib_pts,make_models,anchoring,rot)

Appendix B.(ii) Process_medial_study_specimen_V2

function[]=Process_medial_study_specimen_V2(specimen,R_offset,fem_pts,tib_pts,make_models,anchoring,rot)
[]=Process_medial_study_specimen_V2(specimen,R_offset,fem_pts,tib_pts,make_models,anchoring,rot)
T_offset=[0 0 0];
% Selects the proper model
fname_upper_spec=[specimen '/vtks/' specimen '_Femur_VIVO.vtk'];
fname_lower_spec=[specimen '/vtks/' specimen '_Tibia_VIVO.vtk'];
if anchoring==4
% % % Process result for each stage of the experiment by calling "Extract_data_general_V4"
[pose_data,force_data]=Extract_data_general_V4(specimen,'intact','cont_flex_f',→
→fname_upper_spec,fname_lower_spec,[],[],fem_pts,tib_pts,T_offset,R_offset,make_models,anchoring,rot);

[pose_data,force_data]=Extract_data_general_V4(specimen,'PCL_cut','cont_flex_f',→
→fname_upper_spec,fname_lower_spec,[],[],fem_pts,tib_pts,T_offset,R_offset,make_models,anchoring,rot);

[pose_data,force_data]=Extract_data_general_V4(specimen,'POL_cut','cont_flex_f',→
→fname_upper_spec,fname_lower_spec,[],[],fem_pts,tib_pts,T_offset,R_offset,make_models,anchoring,rot);

[pose_data,force_data]=Extract_data_general_V4(specimen,'deepMCL_cut','cont_flex_f',→
→fname_upper_spec,fname_lower_spec,[],[],fem_pts,tib_pts,T_offset,R_offset,make_models,anchoring,rot);
end
Appendix B.(iii) Extract_data_general_V4

function[pose_data,force_data]=Extract_data_general_V4(specimen,condition,str_pattern,→
→fname_upper_spec,fname_lower_spec,fname_fem,fname_tib,fem_pts,tib_pts,T_offset,R_offset, →
→make_model,model_orientation,rot)

true_chirality=specimen(end);

% Creates the proper excel file
excel_filename=[specimen '\ specimen '_results_Posterior slop.xlsx']

sheetname=condition;

% Reads all the file available for the condition
inventory=dir([specimen '\ condition]);
% Separates the files based on their names
count=0;
for i=1:1:size(inventory,1)
  if strncmpi(inventory(i).name,str_pattern,length(str_pattern))
    fname_temp=[specimen '\ condition '\ inventory(i).name];
    if exist(fname_temp)==2
      count=count+1;
      motion{count}=inventory(i).name;
    end
  end
end

% Reads the name of each motion and its kinematics
for i=1:1:length(motion)
  fname=[specimen '\ condition '\ motion{i}];
  fid=fopen(fname,'r');
  tline=fgetl(fid);
  tline=fgetl(fid);
  tline=fgetl(fid);
  colons=strfind(tline,':');
  periods=strfind(tline,'.');
motion_name{i} = tline((colons(end)+2):(periods(end)-1));
if ~isempty(strfind(motion{i},'_M_'))
    motion_name{i} = [motion_name{i} ' - With Muscles'];
end

if ~isempty(strfind(motion{i},'_NoForce_'))
    motion_name{i} = [motion_name{i} ' - No Force'];
end
fclose(fid);

if exist(fname)==0
    pose_temp=nan*ones(7,6);
else
    % Filters the result
    [data smooth_data]=cyclicsmooth_forces(fname);

    % Calculates the kinematics at 0, 30, 60 and 90 degrees
    [pose_temp,force_temp]=return_discrete_poses_and_forces(smooth_data(:,1:6),
                                                             smooth_data(:,7:12),
                                                             nowrite);
    end
    pose_data{i}=pose_temp;
    force_data{i}=force_temp;
end

% This is where we insert code to calculate medial/lateral epicondyle motions
[npoints_F points_F trias_F quads_F] = Read_VTK_Surface(fname_upper_spec);
save upper_spec_info.mat npoints_F points_F trias_F quads_F

[npoints_T points_T trias_T quads_T] = Read_VTK_Surface(fname_lower_spec);
save lower_spec_info.mat npoints_T points_T trias_T quads_T

if make_model==1
    if exist([specimen '\animations'])==0
        mkdir([specimen '\animations']);
    end
if exist([specimen 'animations' condition])==0
    mkdir([specimen 'animations' condition]);
end
end

for i=1:1:length(motion)
    med_temp_array=[];
    lat_temp_array=[];

    if make_model==1
        if exist([specimen 'animations' condition ' motion_name{i}'])==0
            mkdir([specimen 'animations' condition ' motion_name{i}']);
        end

        if exist([specimen 'animations' condition ' motion_name{i} ' pics'])==0
            mkdir([specimen 'animations' condition ' motion_name{i} ' pics']);
        end
    end

    for j=1:1:size(pose_data{i},1)
        pose=pose_data{i}(j,:);

        if make_model==1
            fname_tib=[specimen 'animations' condition ' motion_name{i} ' tib_plateau num2str(j) '.vtk'];
            fname_fem=[specimen 'animations' condition ' motion_name{i} ' fem_plateau num2str(j) '.vtk'];
            fname_pic=[specimen 'animations' condition ' motion_name{i} ' pics num2str(j)];
        end

    % Moves the tibial and femoral model based on the given kinematics and gives back the AP position of the medial and lateral point of the tibia with respect to the femoral flexion angle.
    [med_temp, lat_temp]=VIVO_Positions_updated_V4(fname_upper_spec, fname_lower_spec, pose(4), pose(5), pose(6), pose(2), pose(1), pose(3), R_offset, T_offset, 'r', fname_tib, fname_fem, model_orientation, tib_pts, fem_pts, make_model, true_chirality, fname_pic, rot);

        med_temp_array=[med_temp_array; med_temp];
        lat_temp_array=[lat_temp_array; lat_temp];
    end
end
end

med_data{i}=med_temp_array;
lat_data{i}=lat_temp_array;
end
delete upper_spec_info.mat
delete lower_spec_info.mat

% Write summarized data to excel
% Kinematics
data=cell2mat(pose_data);
formatted_data=[];
line1=[];
line2=[];
for i=1:1:size(pose_data,2)
    line1_temp={motion_name{i},'','','','','',''};
    line1=[line1, line1_temp];
    line2_temp={'ML (mm)', 'AP (mm)', 'Vert (mm)', 'FE (deg)', 'AA (deg)', 'IE (deg)', 'Medial (mm)', '→ Lateral (mm)' ,'};
    line2=[line2, line2_temp];
    if i==size(pose_data,2)
        formatted_data=[formatted_data [data(:,((1:6)+6*(i-1))) med_data{i} lat_data{i}]];
    else
        formatted_data=[formatted_data [data(:,((1:6)+6*(i-1))) med_data{i} lat_data{i}] nan*ones(7,1)];
    end
end
xlsxwrite(excel_filename,[line1;line2],sheetname);
xlsxwrite(excel_filename,formatted_data,sheetname,'A3')

% Forces
data=cell2mat(force_data);
formatted_data=[];
line2=[];
for i=1:1:size(pose_data,2)
    line2_temp={'ML (N)', 'AP (N)', 'Vert (N)', 'FE (Nm)', 'AA (Nm)', 'IE (Nm)', ''};
    line2=[line2, line2_temp];
if i==size(pose_data,2)
    formatted_data=[formatted_data data(:,((1:6)+6*(i-1)))];
else
    formatted_data=[formatted_data data(:,((1:6)+6*(i-1))) nan*ones(7,1) nan*ones(7,1) nan*ones(7,1)];
end
end
xlswrite(excel_filename,[line2],sheetname,'A14');
xlswrite(excel_filename,formatted_data,sheetname,'A15')

Appendix B.(iv)  VIVO_Positions_updated_V4

function[med_ap,lat_ap]=VIVO_Positions_updated_V4(fname_upper_spec,fname_lower_spec,FE,AD,IE,AP,ML,VL,Roffset,T,chirality,fname_T,fname_F,lock_component,tib_pts,fem_pts,make_models,true_chirality,fname_pic,rot)

  % Opens the segmented model
  if isempty(make_models)
      [npoints_F points_F trias_F quads_F] = Read_VTK_Surface fname_upper_spec;
      [npoints_T points_T trias_T quads_T] = Read_VTK_Surface fname_lower_spec;
  else
      if exist('upper_spec_info.mat')==0
          [npoints_F points_F trias_F quads_F] = Read_VTK_Surface fname_upper_spec;
      else
          load upper_spec_info.mat
      end
      if exist('lower_spec_info.mat')==0
          [npoints_T points_T trias_T quads_T] = Read_VTK_Surface fname_lower_spec;
      else
          load lower_spec_info.mat
      end
  end

  % Calculate the alpha beta gammas for the Grood and Suntay coordinate system.
  if chirality=='r'
      gamma=((IE+Roffset(3)))*pi()/180;
else
   gamma=(-(I+Roffset(3)))*pi()/180;
end

beta=(AD-Roffset(2))*pi()/180+(pi()/2);
% alpha=(FE+Roffset(1))*pi()/180;
alpha=(FE)*pi()/180;

% Calculate translations in terms of S (grood-suntay directions) and then H (tibia wrt femur)
q1=ML;
q2=AP;
q3=VL;

S3=(q1*cos(beta)+q3)/((cos(beta)*cos(beta))-1);
S1=((1*S3)-q3)/(cos(beta));
S2=q2;

S=[S1;S2;S3];

U=[1 0 cos(beta);
   0 cos(alpha) sin(alpha)*sin(beta);
   0 -1*sin(alpha) cos(alpha)*sin(beta)];

H=U*S;

% Calculate the transposed rotation, R^T, givin in appendix of grood-suntay
R_trans_1_1=cos(gamma)*sin(beta);
R_trans_1_2=sin(gamma)*sin(beta);
R_trans_1_3=cos(beta);

R_trans_2_1=-cos(alpha)*sin(gamma)-cos(gamma)*sin(alpha)*cos(beta);
R_trans_2_2=cos(alpha)*cos(gamma)-sin(gamma)*sin(alpha)*cos(beta);
R_trans_2_3=sin(beta)*sin(alpha);

R_trans_3_1=sin(alpha)*sin(gamma)-cos(gamma)*cos(alpha)*cos(beta);
\[ R_{trans\_3\_2} = -\cos(\gamma)\sin(\alpha) - \cos(\alpha)\cos(\beta)\sin(\gamma); \]
\[ R_{trans\_3\_3} = \cos(\alpha)\sin(\beta); \]

\[
R_{trans} = \begin{bmatrix}
R_{trans\_1\_1} & R_{trans\_2\_1} & R_{trans\_3\_1} \\
R_{trans\_1\_2} & R_{trans\_2\_2} & R_{trans\_3\_2} \\
R_{trans\_1\_3} & R_{trans\_2\_3} & R_{trans\_3\_3}
\end{bmatrix};
\]

% Calculate the rotation / translation of the tibia wrt to femur. The equation is given as \( R = [R]r + H \) where \( r \) is coordinates in tibial frame and \( R \) is coordinates in femoral frame, \( R \) is the matrix we just calculated the transpose of above, and \( H \) is the transformation vector from femur to tibia

\[
R = \text{transpose}(R_{trans});
\]
\[
\text{points\_T2} = R \times \text{points\_T};
\]
\[
\text{points\_T2} = \text{points\_T2} + H;
\]

if lock_component==4

% First move origin to femur flexion axis:

\[
\text{points\_F2} = \text{points\_F};
\]
% Now rotate everything about femurs described flexion axis by alpha

\[
R = \begin{bmatrix}
1 & 0 & 0 \\
0 & \cos(\alpha) & -1\sin(\alpha) \\
0 & \sin(\alpha) & \cos(\alpha)
\end{bmatrix};
\]
\[
\text{points\_F3} = R \times \text{points\_F2};
\]
\[
\text{points\_T3} = R \times \text{points\_T2};
\]
\[
\text{origin} = (\text{points\_F3(:,fem\_pts(1))} + \text{points\_F3(:,fem\_pts(2))})/2;
\]
\[
\text{points\_F4} = \text{points\_F3-origin};
\]

if true_chirality=='L'

\[
\text{i\_dir} = (\text{points\_F4(:,fem\_pts(1))} - \text{points\_F4(:,fem\_pts(2)))}/\rightarrow \rightarrow\text{norm((points\_F4(:,fem\_pts(1)) - points\_F4(:,fem\_pts(2))))});
\]
else

\[
\text{i\_dir} = (\text{points\_F4(:,fem\_pts(2))} - \text{points\_F4(:,fem\_pts(1)))}/\rightarrow \rightarrow\text{norm((points\_F4(:,fem\_pts(2)) - points\_F4(:,fem\_pts(1))))});
\]
end

% Rotates the coordinate system so that the femoral flexion axis is aligned with the x axis, then reports the y Value of the medial point.
k\_temp = \[0 ; 0 ; 1\];
j\_dir=cross(k\_temp,i\_dir)/norm(cross(k\_temp,i\_dir));
k\_dir=cross(i\_dir,j\_dir)/norm(cross(i\_dir,j\_dir));

points\_T4=points\_T3-origin;
R=[i\_dir';j\_dir';k\_dir'];
points\_F5=R\*points\_F4;
points\_T5=R\*points\_T4;

\% Rotates the whole model so that the posterior surface of the tibia is vertical
deg=(rot)*pi()/180;
R=[1 0 0;
 0 cos(deg) -1*sin(deg);
 0 sin(deg) cos(deg)];
points\_F6=R\*points\_F5;
points\_T6=R\*points\_T5;

\% medial/lateral condyle AP position:
med\_ap=points\_T6(2,tib\_pts(1));
lat\_ap=points\_T6(2,tib\_pts(2));
end
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