8-7-2024 4:00 PM

The Role of Transverse Plane Malalignment in Posterolateral Corner Complex Injury

Larissa M. Madia, *Western University*

Supervisor: Willing, Ryan, *The University of Western Ontario*
Co-Supervisor: Getgood, Alan, *The University of Western Ontario*
A thesis submitted in partial fulfillment of the requirements for the Master of Engineering Science degree in Biomedical Engineering

Abstract

The posterolateral corner (PLC) is a complex of ligaments and soft tissues in the knee that primarily constrain external tibial rotation. Neglecting PLC injury is a contributing factor in graft failure following cruciate ligament reconstruction. This emphasizes the importance of understanding PLC injury mechanisms, considering their disposition to multi-ligament tears. The PLC can be damaged by hyperextension. Tibial torsion, a twisting of the tibia longitudinally relative to the femur, offsets the transverse knee alignment, consequently altering the loading of soft tissues. Therefore, the objective of this investigation was to determine if tibial torsion affects loading of PLC components during hyperextension, as an indication of an underlying injury pattern. A joint motion simulator was used to record flexion torque magnitudes following the sectioning of individual PLC components in cadaveric specimens. Tibial torsion affected the load distribution of PLC components.

Keywords

Posterolateral Corner, External Tibial Torsion, Tibial Deformity, Force Contribution

Summary for Lay Audience

The posterolateral corner (PLC) complex is a collection of soft tissues and ligaments located at the back, outside corner of the knee joint. The primary components are the lateral collateral ligament (LCL), the popliteofibular ligament (PFL), and the popliteus tendon, which together constrain external tibial rotation. Isolated injury to this complex is rare. It is frequently damaged alongside anterior and posterior cruciate ligaments. Clinicians may prioritize treatment of these major ligaments, neglecting the injury to the PLC. Consequently, untreated PLC injuries lead to cruciate ligament graft failure [4].

The most common mechanism of PLC injury is a combination of forces involving varus stress and hyperextension of the knee joint, occurring in situations like motor vehicle accidents or athletics. Researchers from the Kliniek ViaSana (Mill, Netherlands) recognized that several patients with PLC injuries also exhibited underlying abnormal tibial torsion, a twisting of the tibia along its longitudinal axis, prompting consideration that tibial transverse malalignment may be a contributing factor to PLC injury. It is hypothesized that excessive external tibial torsion alters the loading pattern within the knee joint, thereby increasing the stress on specific ligaments. These increased loads may heighten PLC susceptibility to injury when subjected to hyperextension forces.

There is limited research exploring loading of the posterolateral corner complex during hyperextension stress in the presence of a torsional deformity. We aimed to pioneer this area of study by establishing the loading pattern of PLC components during hyperextension in patients with abnormal tibial alignment. We used a joint motion simulator to evaluate force contributions of PLC components. We measured joint reaction forces before and after cutting a soft tissue structure, attributing the change in forces to be representative of the loads passing through the cut tissue. Results indicate large contributions from the LCL and posterolateral capsule + PFL in restraining hyperextension. Simulated rotational deformities significantly influenced the loading pattern of PLC structures. Considering our findings, when a patient presents with a knee injury, tibial torsion, and describes a mechanism involving hyperextension, clinicians should be particularly vigilant in assessing for potential PLC damage.

Co-Authorship Statement

This thesis was drafted by Larissa Madia and revised by Dr. Ryan Willing and Dr. Alan Getgood.

Acknowledgments

I would like to express my gratitude to my supervisors, Dr. Willing and Dr. Getgood, for their guidance and support throughout my thesis. Through their expertise and insightful feedback, I have learned so much about research. I would also like to thank Dr. Takaaki Hiranaka for his involvement in my experiments.

I would like to thank the members of the Biomechanical Engineering Research Lab, past and present, for enriching my experience with knowledge and camaraderie. I would also like to extend a special thanks to Emma Donnelly, for her constant support and mentorship.

I would like to extend my sincere appreciation to the donors of the cadaveric specimens, whose generous contributions made this research possible.

Lastly, but certainly not least, I want to thank my family for supporting me during this journey. Dad, thank you for all the times you filled up my gas tank to get to London, and Mom, thank you for coming along for the ride, both literally and figuratively.

Thank you all for being a part of the past two years of my life.

Table of Contents

List of Figures

Chapter 1

1 Introduction

1.1 Motivation

The posterolateral corner (PLC) complex was previously referred to as the “dark side of the knee” because of its intricate anatomy, diagnostic challenges, and limited treatment options [22]. Ongoing research of this complex and advancements in clinical practice has since shone a light on this region, leading to an improved understanding from an anatomical and biomechanical perspective. Despite these improvements, continued research on this complex is necessary to improve diagnostic techniques and identify risk factors associated with PLC injuries. The aim of these research efforts is to ultimately reduce both the incidence and severity of PLC injuries.

The primary components of the PLC complex are the lateral collateral ligament (LCL), the popliteofibular ligament (PFL), and the popliteus tendon, which together constrain external tibial rotation and stabilize the knee during varus stress [5] (Figure 1-1).

Injuries to the posterolateral corner occur most often as the result of a high-energy impact, such as a motor vehicle accident or a sports-related collision (Figure 1-2) [16]. The mechanisms of PLC injury include posterolateral forces to the anteromedial thigh, contact and non-contact hyperextension, a valgus force to a flexed knee, and severe tibial external rotation with a flexed knee [6]. The most common mechanism of injury is a combination of forces involving varus stress and hyperextension of the knee joint [6].

It has been established that injuries to the PLC contribute to knee instability, but it was previously believed that injuries to the PLC were rare. This is because isolated injury to the PLC complex scarcely occurs, accounting for only ~2% of acute knee injuries [6]. However, only 28% of PLC injuries occur in isolation [22]. Therefore, 72% of PLC injuries involve concurrent damage to another structure, most frequently the anterior cruciate ligament or the posterior cruciate ligament [27]. This explains why PLC injuries were perceived as rare and are often overlooked in reporting; clinicians may prioritize

assessment of the cruciate ligaments, which may result in the PLC injury being overlooked [27, 32, 47]. When left untreated, the altered loading that results from PLC injuries leads to increased stress on other ligaments, particularly the ACL [32]. This increased stress can result in complications like graft failure of the ACL, chronic pain, instability, and functional limitations [4, 32].

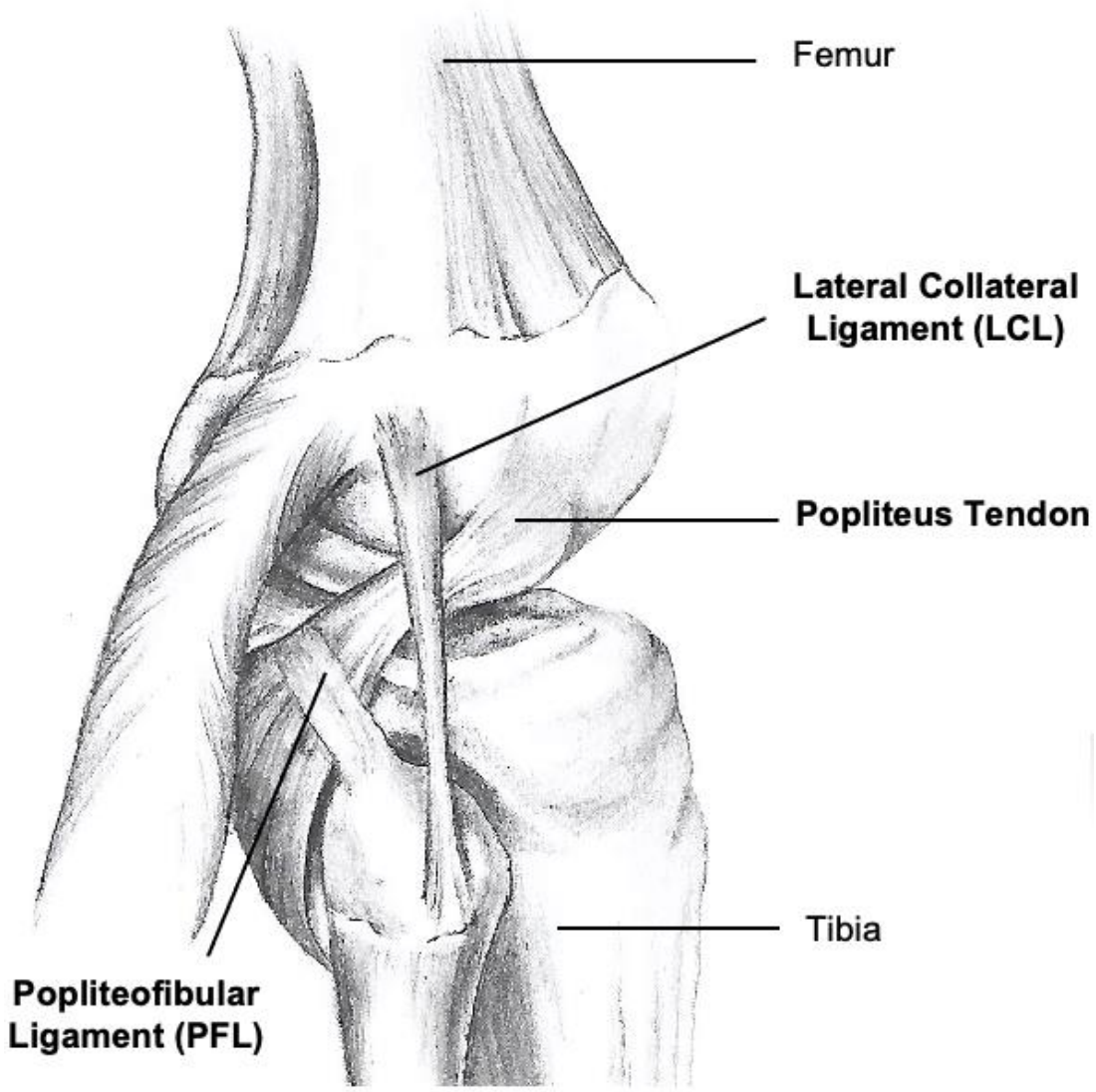


Figure 1-1: Major stabilizing structures of the posterolateral corner (PLC) complex. Figure inspired by LaPrade et al. "The Posterolateral Attachments of the Knee" [7].

The change in load distribution that results from PLC injury can increase risk of early onset osteoarthritis, a degenerative joint disease, because of the altered biomechanics of the knee joint. Specifically, the loading distribution resulting from PLC tears stresses certain areas of the joint, which accelerates cartilage degeneration in that area [63]. Osteoarthritis can therefore develop more rapidly and cause symptoms like pain, joint stiffness, swelling, and muscle weakness [63]. Over time these symptoms may impair the patient from completing daily tasks due to the functional limitations these symptoms impose on the joint [63]. By studying the loading patterns of the PLC, we aim to provide healthcare professionals with the tools to identify PLC injuries early and accurately to prevent further complications and provide a foundation for further investigation into multi-ligament injuries.

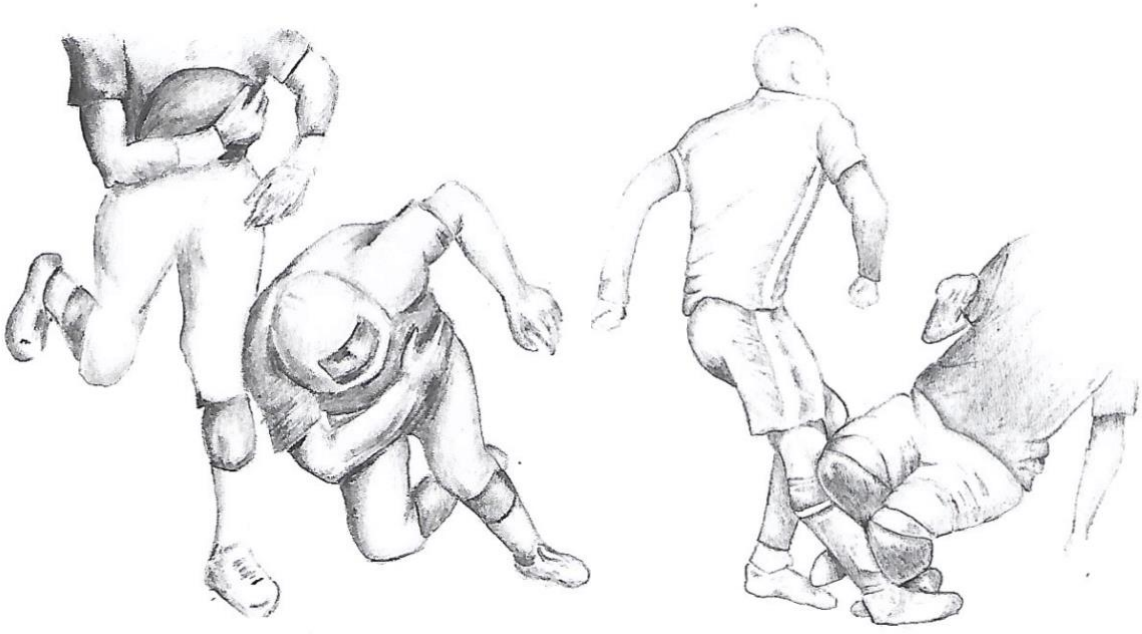


Figure 1-2: Contact hyperextension is a cause of posterolateral corner injury often occurring in sporting events. The image on the right displays a hyperextension and varus stress, as the defender is approaching from the medial side (inside) of the knee, directing the blow in the posterolateral direction.

Previous literature has explored two key topics we investigate in our study: cadaveric studies have quantified the resisting moments of soft tissue structures of the PLC in resisting knee hyperextension, while separate research has established that internal and external tibial rotations alter transverse plane torques at the knee [3, 8]. In reference to the latter, Bates et al. utilized a robotic manipulator arm to determine if tibial rotation offsets affect knee loading profiles and found that transverse plane rotational offsets altered the forces and torques of the ACL during simulated athletic tasks [8]. Our study aims to address a gap in the literature by exploring the effect of transverse plane rotational offsets on the loading of the PLC during knee hyperextension. A rotational offset within the transverse plane is also known as a transverse plane malalignment (Figure 1-3). This tibial deformity refers to an abnormal twisting of the distal tibia bone relative to the proximal tibia bone along the tibia's longitudinal axis. This rotation can be measured using the tibial external rotation angle, which is the angle between the line connecting the posterior condyles of the tibia, and a line intersecting the malleoli (the bony projections on either side of the ankle) (Figure 1-3).

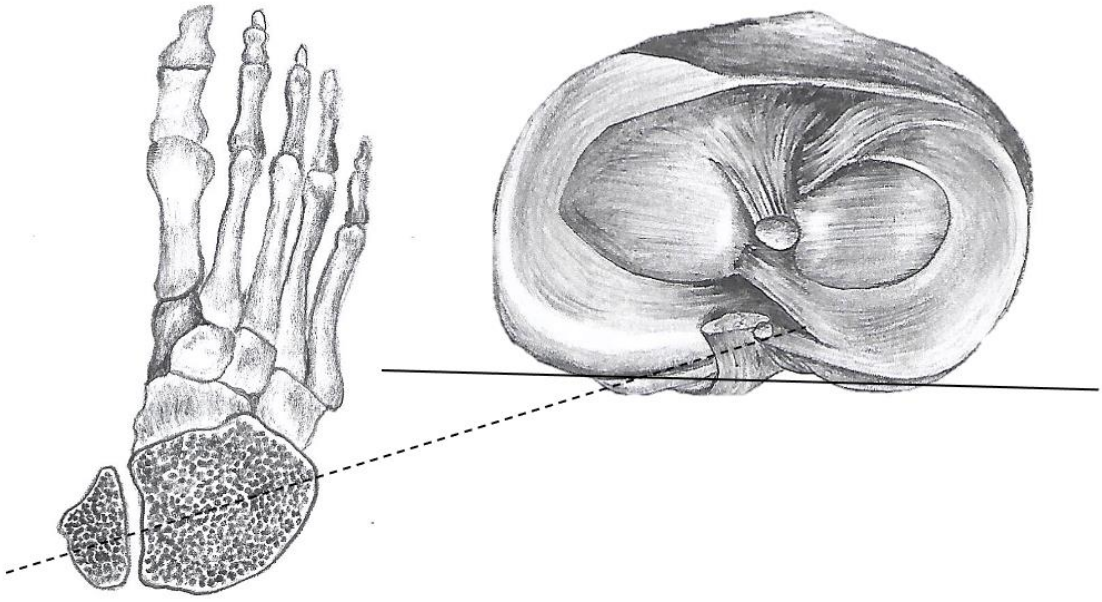


Figure 1-3: Transverse plane malalignment. Axial depiction of the right tibia. The degree of tibial torsion can be measured using the angle between the line connecting the posterior condyles of the tibia (solid line), and a line intersecting the malleoli (transmalleolar axis) (dotted line).

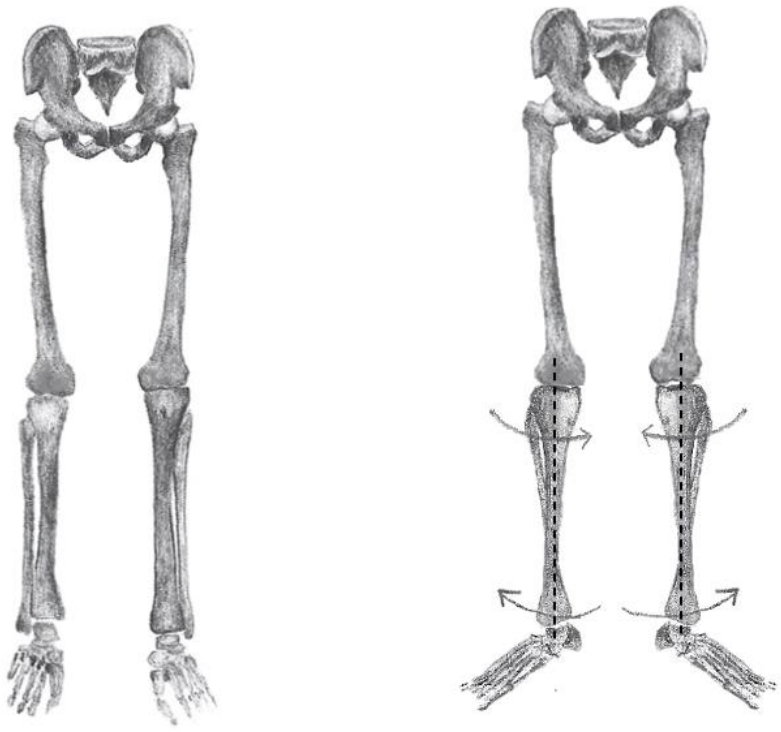


Figure 1-4: External tibial torsion. Tibial torsion is an abnormal twisting of the tibia relative to the femur along its longitudinal axis (dashed line). At the proximal tibia, the fibula is visible due to an inward rotation, whereas more distally the tibia rotates such that the fibula becomes hidden, resulting in an externally rotated presentation of the feet.

Researchers from Kliniek ViaSana (Mill, Netherlands) recognized patients presenting to the clinic with PLC injuries also exhibited underlying abnormal tibial torsion, specifically external tibial torsion. This prompted the consideration that tibial malalignment may be a contributing factor to PLC injury risk. We hypothesize that the altered loading patterns within the knee joint caused by external tibial torsion might increase the susceptibility of the PLC to injury when exposed to hyperextension forces. This hypothesis, in addition to the observations of the clinicians, is based on two key considerations. The first consideration is based on a compensatory pattern that is characteristic of a patient with excessive external tibial torsion. This compensatory pattern, which consists of a relative internal rotation of the proximal tibia with respect to the femur serving as an attempt to maintain a “normal” foot angle, may decrease the leverage of the PLC. Leverage refers to

the mechanical advantage that a structure possesses to counteract forces acting upon it. With reduced leverage, the PLC may require greater tension to counteract the forces of hyperextension. This increased tension can potentially strain the ligaments and soft tissues of the PLC complex, predisposing them to fatigue, overloading, or injury. Therefore, individuals with excessive external tibial torsion may be more susceptible to PLC injuries due to the altered biomechanics and decreased leverage of the posterolateral corner.

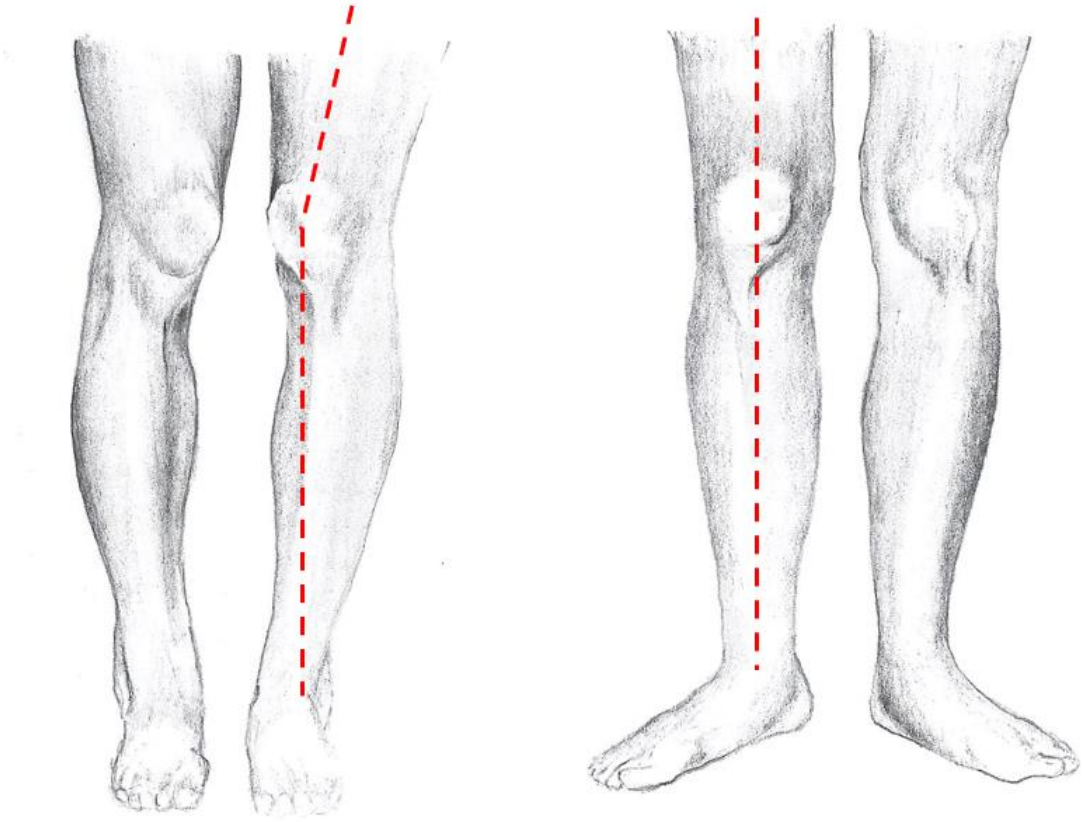


Figure 1-5: Two potential injury situations during a hyperextension injury to a patient presenting with excessive external tibial torsion. Left: Feet neutral, proximal tibia rotated inwardly. Compensatory Alignment Injury. Right: Feet pointed outward, proximal tibia in a “normal” alignment. Non-compensatory Alignment Injury.

Alternatively, if those with external tibial torsion undergo a hyperextension force, there is the potential that the knee is in a “normal” rotational position, not in the internally rotated compensatory pattern. Yet, because of the deformity, the foot is angled outward. This outward foot angle causes some of the force (upon landing, for example) to be directed in

a way that induces an external rotation torque on the knee. These forces cause the knee to experience a relative external rotation, which, when combined with hyperextension forces, places additional stress on the PLC. Shon et al. explain that the PLC is responsible for resisting external rotation and hyperextension forces [17]. Under normal circumstances, the PLC can manage these forces, however, when the knee is subjected to abnormal external rotation due to the outward foot angle, the PLC is forced to endure greater strain. This strain can predispose the PLC to injury through increased tension required to counteract the abnormal forces caused by the external rotation torque which can exceed the capacity of the PLC, leading to damage. Bates et al. showed that transverse plane rotational offsets, such as external tibial torsion, can significantly impact the kinetics of the knee joint, and make certain structures more vulnerable to injury, like the ACL [8].

Research has yet to explore the loading distribution of the posterolateral corner complex during hyperextension stress in the presence of a torsional deformity. Understanding the role that these joint stabilizers play in the human knee can assist in the diagnosis of PLC injuries by identifying the key structures vulnerable to hyperextension injuries, especially in cases of rotational malalignments. This knowledge underscores the importance of the PLC in knee stability and can be used to enhance reconstruction techniques in the future.

1.2 Objectives

This thesis aims to conduct biomechanical analysis of the PLC complex during a loading scenario consisting of both pure hyperextension and hyperextension with varus torque. By replicating a hyperextension injury, we aim to determine if simulated torsional deformities of the tibia impact the loading profiles of posterolateral corner structures. Utilizing the robotic six-degrees-of-freedom joint motion simulator designed to test biological joint specimens, we set to achieve the following:

1. To determine the distribution of loads among the components of the PLC complex during hyperextension at neutral rotation.

2. To compare the effects of the Compensatory and Non-Compensatory Alignment Injury mechanisms, each simulating excessive external tibial torsion, a deformity in which there is malalignment within the transverse plane.

1.3 Organization of Thesis

Chapter 2 describes the anatomy of the intact human knee joint. Specific focus is placed on the anatomy and physiology of the primary ligament complex of interest in this work, the posterolateral corner (PLC) complex and the clinical importance of injuries to the PLC. This is followed by a description of PLC injuries and transverse plane malalignment, developed from a literature review of previously published studies investigating the PLC, its injury patterns, and knee malalignment.

Chapter 3 describes the testing that was conducted to investigate the biomechanical role of the PLC in knee stability. This chapter describes the way in which loads passing through PLC components are assessed following sectioning using a robotic joint motion simulator. The results of the study are presented in this chapter, beginning with discussing the relative role of each PLC component in knee stability during hyperextension in a neutral transverse alignment. This is followed by an exploration of cases in which the knee is rotated internally, and in cases in which the knee is rotated externally. This chapter concludes by offering insight into the differences between this study's findings and the results of previous studies, explaining any contradictions.

Chapter 4 discusses the conclusions of Chapter 3 and addresses the strengths and limitations of this work, followed by a discussion of future work directions and the significance of findings.

Chapter 2

2 Background

This section provides a brief overview of the anatomy of the knee joint, introducing the terminology readers will encounter within this thesis. Subsequently, this chapter will examine the biomechanics of different ligamentous structures of the knee joint and their roles in knee stability, specifically that of the PLC. PLC injuries and the way they are diagnosed, classified, and treated are also discussed. This chapter concludes with a discussion of malalignment and superposition.

2.1 Knee Joint Anatomy

Anatomical Planes

Before exploring the intricacies of the dynamic knee joint, establishing a foundation in anatomical terminology is imperative. The three anatomical planes of the body provide a reference for describing body movements and positions. These planes are termed sagittal, frontal (coronal), and transverse (horizontal) (Figure 2-1).

The sagittal plane is a vertical plane dividing the body into left and right halves. Bending within this plane is known as *Flexion-Extension (FE)*. Flexion is the reduction of the angle between two body segments, whereas extension is the motion that increases this angle. These movements are important to completing exercises like walking and squatting.

The frontal plane divides the body into front and back sections. In this plane, we observe *Abduction/Adduction (AA)* movement. Abduction involves movement away from the body's mid-line, whereas adduction involves movement toward the mid-line. These motions are evident when performing maneuvers like getting out of bed, getting out of the car, or other side-stepping movements. In discussions of the knee joint, varus and valgus (VV) may be used in place of AA. Varus corresponds to adduction, whereas valgus refers to abduction, each of the tibia with respect to the femur.

The third plane, the transverse plane, is a horizontal plane that divides the body into upper and lower portions. This plane facilitates the movement of *Internal-External rotation (IE).* Internal rotation involves rotation of the tibia towards the mid-line of the body, whereas external rotation involves the rotation of the tibia away from the mid-line, each with respect to the femur.

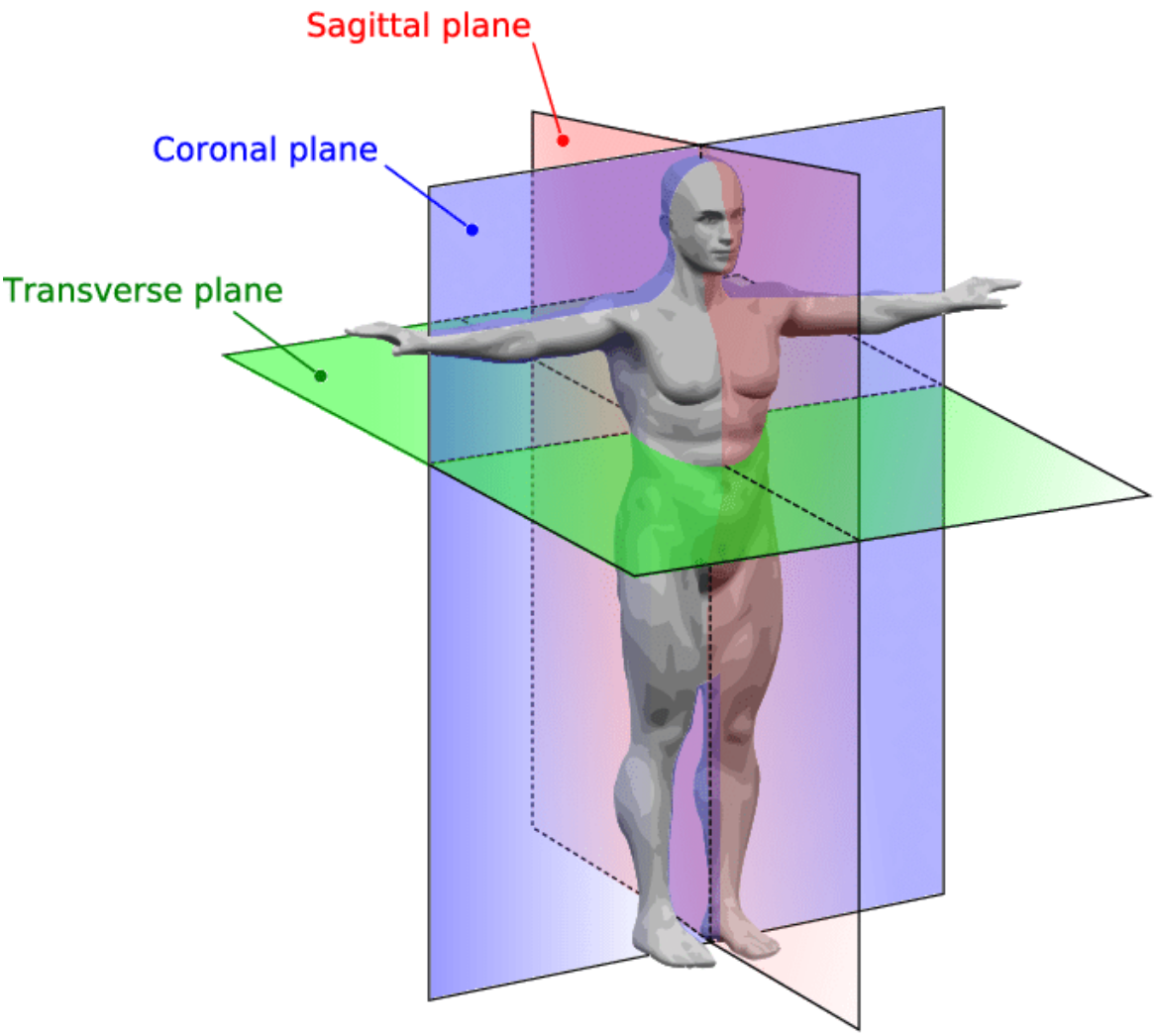


Figure 2-1: Anatomical planes of the human body used to define motions. The sagittal plane divides the body into the left and right halves. The coronal (frontal) plane divides the body into front and back sides. The transverse plane divides the body into top and bottom sections. © Yassmine Mrabet, CC-BY-SA 1.0.

In addition to these three rotations (flexion-extension, abduction/adduction, and internal-external rotation) (Figure 2-2), three translations (anterior-posterior, superior-inferior, and medial-lateral) complete the 6 degrees-of-freedom (DoF) a human joint can potentially possess. These can also be described in relation to the anatomical planes. *Anterior-Posterior (AP)* refers to positions in relation to the front and back of the body, respectively, and can be referenced by division of the frontal plane. *Inferior-Superior (IS)* describes vertical positioning in the transverse plane, with inferior referring to structures that are lower, or below, and superior referring to structures that are higher, or above. *Medial-Lateral (ML)* distinguishes between being towards the sagittal plane (medial) versus being away from the sagittal plane (lateral) (Figure 2-3).

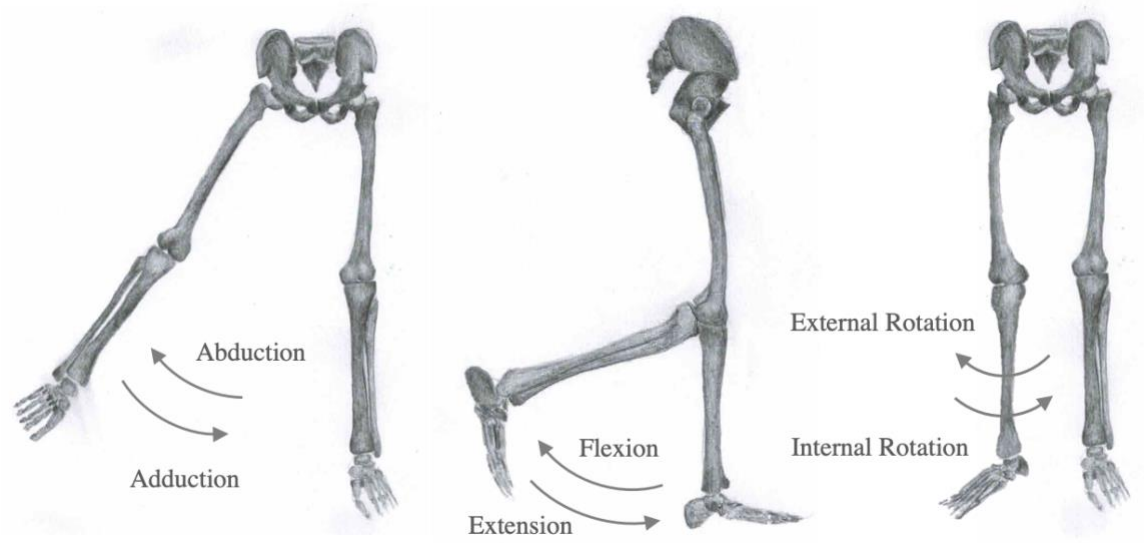


Figure 2-2: Three rotational degrees of freedom that can be defined by the anatomical planes. These are flexion-extension, abduction-adduction, and internal-external rotation.

Furthermore, *Proximal-Distal* and *Superficial-Deep* offer further description of anatomic position. Proximal indicates proximity, meaning it is used to describe closeness to the origin, specifically the torso. Distal means further from the torso (closer to an extremity, perchance). Superficial implies a position near the surface of the body, while deep refers to a position away from the surface.

By describing these anatomic planes, directions, and terminology, we lay a foundation for the exploration of the complexities that exist within biomechanics of the knee joint.

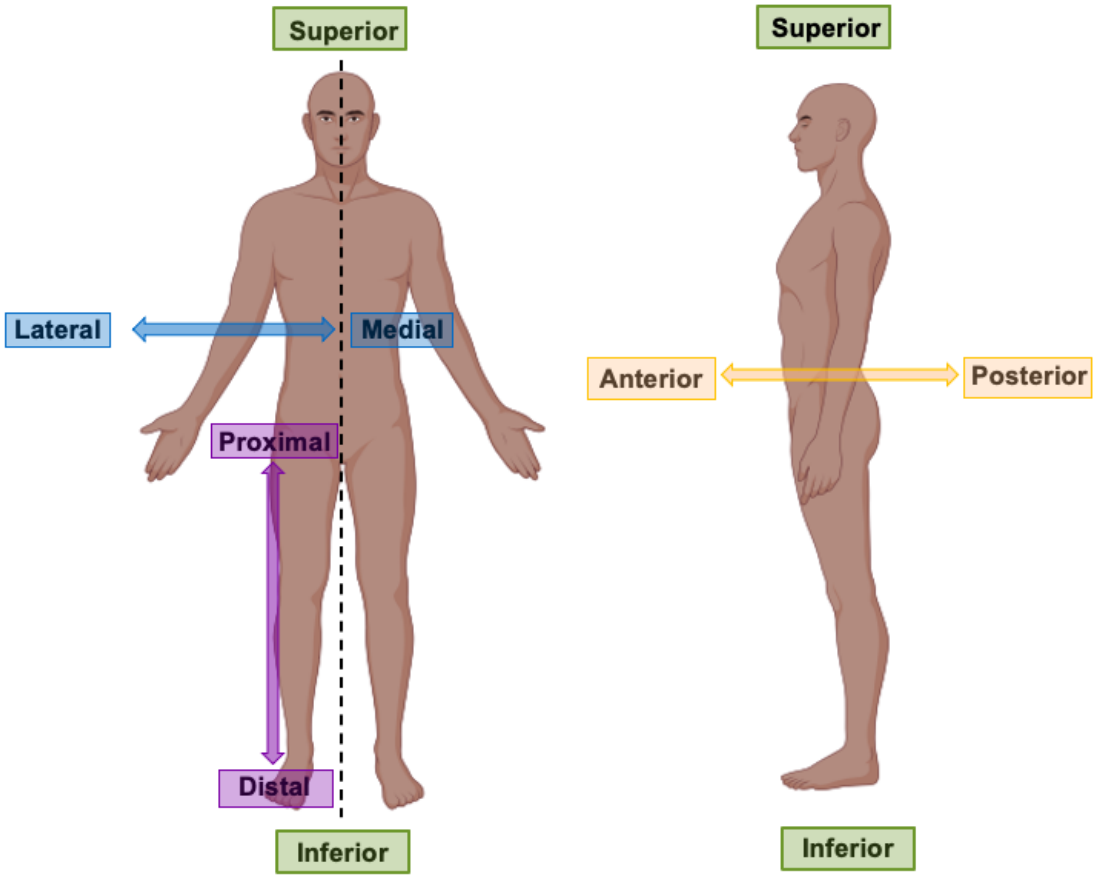


Figure 2-3: Descriptive terminology used for anatomic position. The three translational degrees-of-freedom that exist in the human joint are anterior-posterior, superior-inferior, and medial-lateral. Created in part using biorender.com.

Ligaments & Tissues & Bones

The knee joint is a very complex joint. In simplifying terms, it can be described as a synovial joint that allows flexion, extension, and internal-external rotations. The knee joint is known as a modified hinge joint because it can move in these two planes, but also possesses the ability to accommodate smaller movements [10]. Four bony components make up the knee joint: the tibia (shinbone), femur (thighbone), patella (kneecap), and fibula (Figure 2-4). Excluding the fibula (a non-weight bearing bone), these bones form the two main joints of the knee: the tibiofemoral joint and the patellofemoral joint. These

joints are what permit the knee to move in the three planes discussed previously. The patellofemoral joint is the articulation of the patella with the trochlear groove of the femur. The tibiofemoral (TF) joint is the articulation the tibia and femur. Interposed between the femoral condyles and plateaus of the tibia are two fibrocartilage rings – the medial and lateral menisci. These crescent-shaped structures deepen the articulating surfaces of the tibia, enhancing stability, and provide shock absorption by distributing loads between the medial and lateral compartments [10]. This structural framework forms the attachments sites for ligaments and muscles, which stabilize the knee joint.

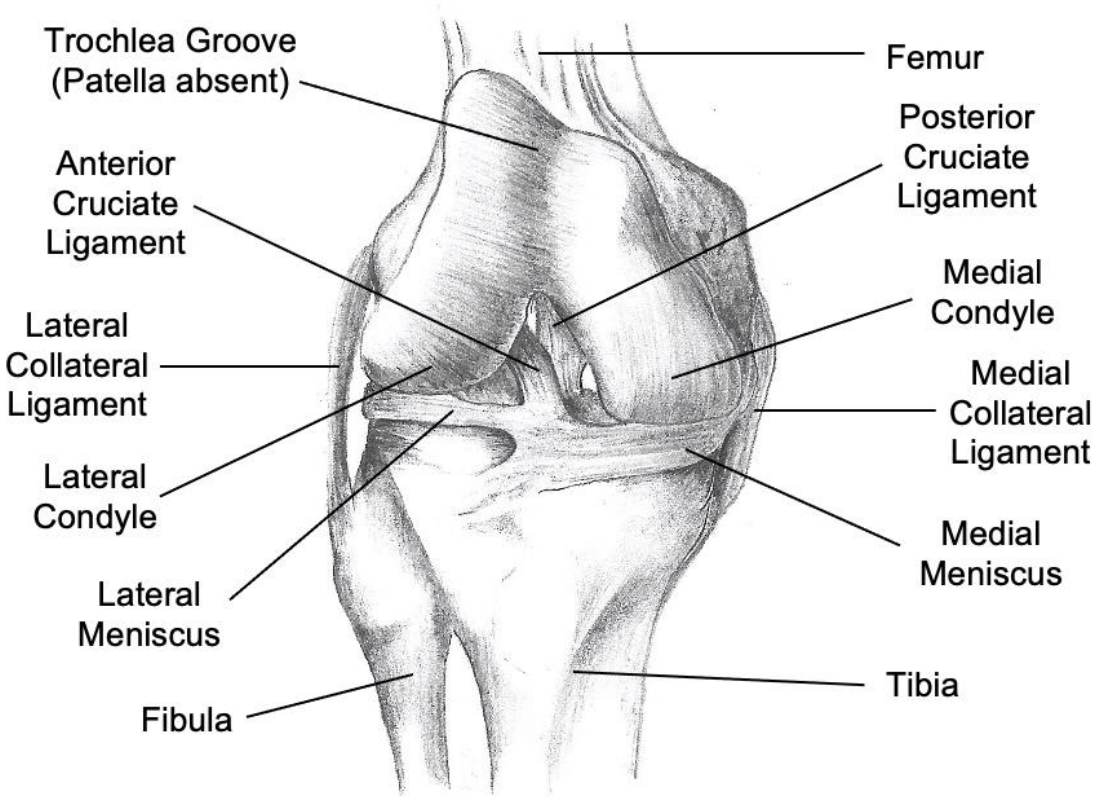


Figure 2-4: Bony and soft tissue anatomy of the knee joint. Bony structures include the femur, tibia, fibula, and patella (absent). Menisci, primary knee ligaments, and condyles are also shown.

Two main muscle groups stabilize the knee (Figure 2-5). One is the quadriceps femoris, located on the anterior aspect of the thigh. This muscle group, consisting of the vastus medialis, vastus lateralis, vastus intermedius, and rectus femoris, facilitates knee extension [11]. Conversely, the hamstring muscles, located on the posterior aspect of the

thigh, act antagonistically to the quadriceps, and enable knee flexion. The hamstrings consist of the biceps femoris, semitendinosus, and semimembranosus [12]. Muscles can provide active stabilization through contraction but can also provide passive stabilization when static loading of the joint occurs.

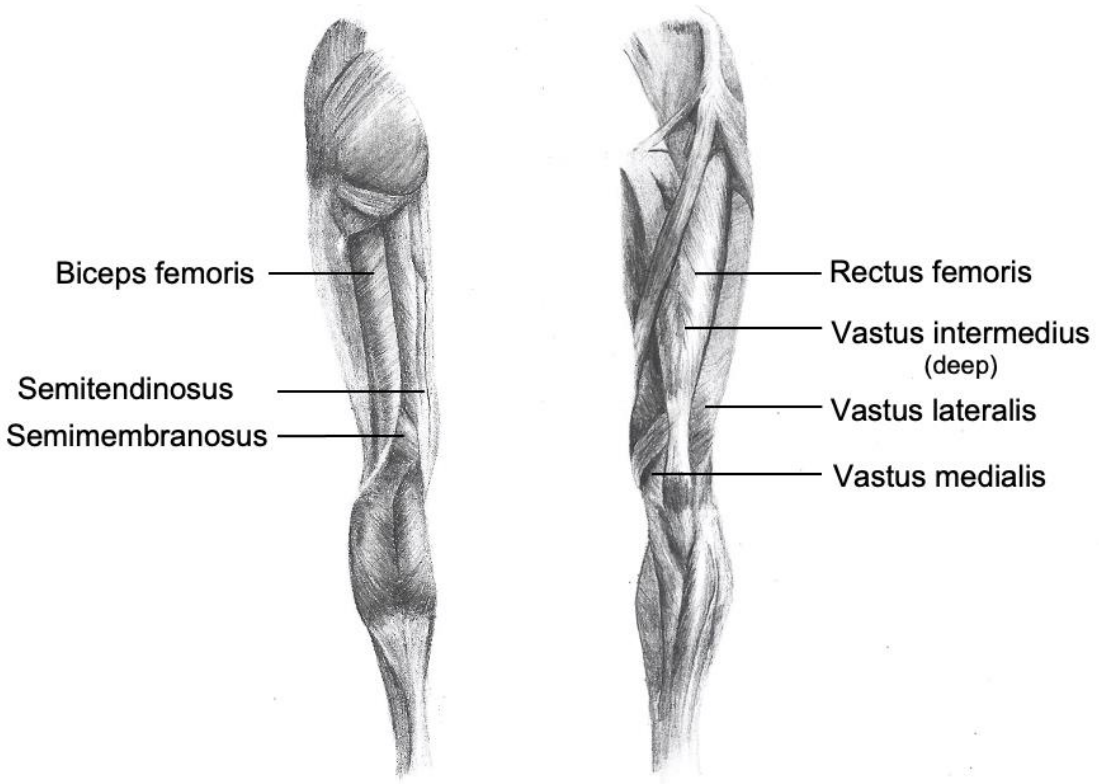


Figure 2-5: Muscle groups stabilizing the knee joint.

Ligaments also provide passive stabilization to the knee joint. A ligament is a band of fibrous connective tissue that connects bone to bone [13]. Ligaments can withstand high tensile forces, a property attributed to the strong yet flexible collagen fibrils that are organized hierarchically into fibers, then fascicles, and bundled within a connective tissue sheath [65, 66]. Only the fibrils that are taut when stretched will be load-bearing [69]. In addition to collagen, ligaments consist of elastin, proteoglycans, glycolipids, and other macromolecules [65-67]. It is the interaction of these components that provide ligaments with their viscoelastic property [67]. Viscoelasticity is a time-dependent property, evident in ligaments due to their ability to exhibit both viscous and elastic characteristics when undergoing deformation. Elasticity in a ligament is a property that allows it to stretch

when a force is applied and return to its original shape when the force is removed. As you stretch the ligament, the force (usually tension) within it increases. Once you stop pulling, the ligament naturally recoils back to its original length, and the tension gradually disappears. Viscous effects refer to the resisting force in a viscous damper that is directly related to the speed at which the damper is stretched. This force is greater when you pull quickly, and lesser if you pull slowly. If the pull is extremely slow, or if the damper is held static, the force becomes zero. Ligaments display a combination of viscous and elastic behaviours; therefore, they are referred to as viscoelastic. Viscoelastic solids display creep and stress relaxation [67, 69]. Creep is an increase in deformation over time under a constant stress [67]. Stress relaxation is a decrease in stress over time under a constant deformation [67, 69]. Together, these phenomena result in hysteresis, the energy loss observed in a material as it undergoes cyclic loading and unloading. [67, 68]. Despite the strength of ligaments, they are frequently injured due to overuse [68]. When ligaments are injured, the loading of knee is altered, and healing of ligament injuries is slow [68].

There are four main ligaments in the knee joint (Figure 2-4). There are two collateral ligaments, located on the medial and lateral sides of the tibiofemoral joint. These collateral ligaments prevent IE rotation and ML translation. The lateral collateral ligament (LCL) stabilizes the knee during varus stress and prevents posterior tibial displacement between 0° and 30° of knee flexion [18, 70]. The medial collateral ligament (MCL) stabilizes the knee during valgus stress. The MCL has a deep layer and a superficial layer. The deep layer of the MCL is a major secondary restraint to anterior translation of the tibia [71]. The proximal division of the superficial MCL primarily stabilizes against valgus stress, whereas the distal division of the superficial MCL primarily stabilizes against both internal and external rotation moments [72]. The knee also contains two cruciate ligaments, named for their attachment at the tibia. The anterior cruciate ligament (ACL) prevents anterior translation of the tibia with respect to the femur [74]. The ACL is composed of two bundles: the anteromedial bundle, which lengthens and tightens in flexion, and the posterolateral bundle, which shortens and becomes slack in flexion [76]. The ACL also functions as a major secondary restraint to internal rotation and is a minor secondary restraint to external rotation and varus-valgus

angulation [73, 75]. The posterior cruciate ligament (PCL), inserting at the posterior aspect of the proximal tibia, prevents posterior translation of the tibia with respect to the femur [64]. The PCL consists of two distinct but synergistic bundles: a larger anterolateral bundle and a smaller posteromedial bundle [64]. The function of the PCL is augmented by posterolateral structures, most notably those of the posterolateral corner complex [14]. In addition to the primary muscles and ligaments, the posterolateral corner complex plays a crucial role in knee joint stability.

2.2 Posterolateral Corner (PLC) Complex

Posterolateral Corner Complex Anatomy and Physiology

Many factors led to the PLC being regarded as the "dark side of the knee" in the late 20th century, such as its complex anatomy, difficulty to diagnosis injury accurately, and subtle presentation [9, 16, 22]. The complex anatomy of the PLC is largely the result of the evolutionary changes in the relationships between the fibular head, the popliteus tendon, and the biceps femoris muscle [16]. The understanding of the PLC has been further complicated by the inconsistent terminology used to describe its components [16]. In 1976, Hughston et al. provided the first report on the posterolateral corner (PLC) of the knee outlining its clinical significance in causing functional limitations and highlighting the discrepancies that exist in diagnosing this injury due to terminology inconsistency [15].

The PLC provides both dynamic and static stabilization. Static stabilizers include the lateral collateral ligament (LCL), popliteofibular ligament (PFL), arcuate ligament complex, fabellofibular ligament, lateral meniscus, and posterolateral capsule (Figure 2-6) [17]. Dynamic stabilizers include the biceps femoris, iliotibial band (ITB), and the lateral head of the gastrocnemius muscle (Figure 2-6) [17]. The popliteus muscle-tendon complex acts as a dynamic and static stabilizer. There are individual variations in the anatomy of the PLC among people [9]. The posterolateral corner is divided into three layers of structures, as described in a 1982 publication from Seebacher et al. [33]. The most superficial layer consists of the ITB and biceps femoris tendon [33, 77, 78]. The middle layer contains the quadriceps retinaculum, patellofemoral ligaments, and patellar

retinaculum [33, 77, 78]. The deepest layer has a superficial and deep lamina [33, 77, 78]. The superficial lamina consists of the LCL and the fabellofibular ligament [33, 77, 78]. The deep lamina contains the coronary ligament, popliteus tendon, arcuate ligament, PFL, and posterolateral capsule [33, 77, 78].

The conceptualization of the PLC outlined by Seebacher et al., in which there are many structures each divided into layers, is becoming an outdated perspective due to the compartmentalization based on anatomical proximity [77]. Contemporary literature has shifted towards a functional approach for describing the important components of the PLC, highlighting those that work both independently and synergistically to statically stabilize the knee joint [7, 77]. These key structures, documented in numerous studies that measure the effect of sectioning PLC components, include the LCL, popliteus tendon, and the PFL [7, 20, 77, 79].

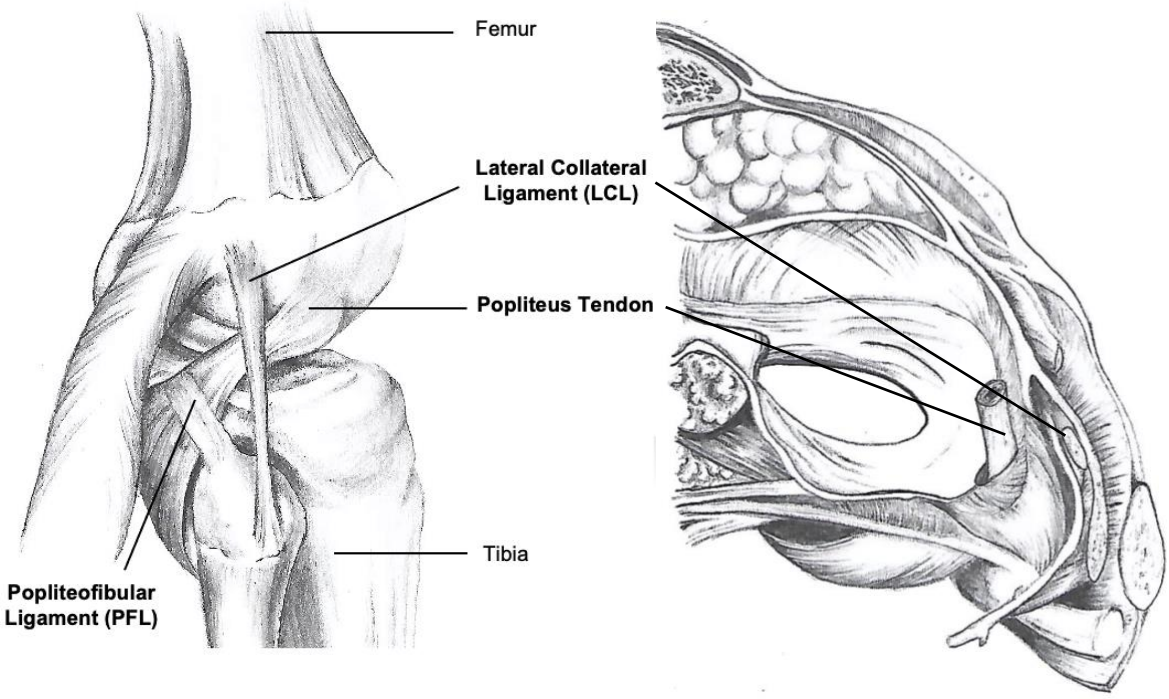


Figure 2-6: Posterolateral structures of the knee. Right: Axial depiction of the posterolateral structures. The LCL, PFL, and the popliteus tendon are the primary components of the PLC. Figure adapted from Seebacher et al. [35].

This thesis focuses on the three primary static stabilizing structures that comprise the PLC. These are identified in Figure 1-1 and 2-6. These include the LCL, PFL, and the popliteus tendon. The LCL, also referred to as the fibular collateral ligament, originates from the lateral epicondyle of the femur and inserts at the fibular head [18]. The LCL functions to stabilize the knee under varus stress and prevent tibial external rotation. The PFL originates from the inferior portion of the popliteus tendon and inserts at the styloid process of the fibula. This static stabilizer provides support against varus angulation, external rotational forces and posterior translation of the tibia [19]. The popliteus tendon arises from the popliteal muscle, which originates at the lateral surface of the lateral femoral condyle, then it gives rise to its tendon, which inserts onto the posteromedial aspect of the tibia. The popliteus tendon restrains external rotation of the tibia relative to the femur. Literature has referred to the popliteus tendon as a static stabilizer [22, 106]. While the popliteus tendon does function dynamically as a tendon, LaPrade et al. showed that the popliteus tendon also possesses ligament-like functions in contributing to the static stability of the knee [107].

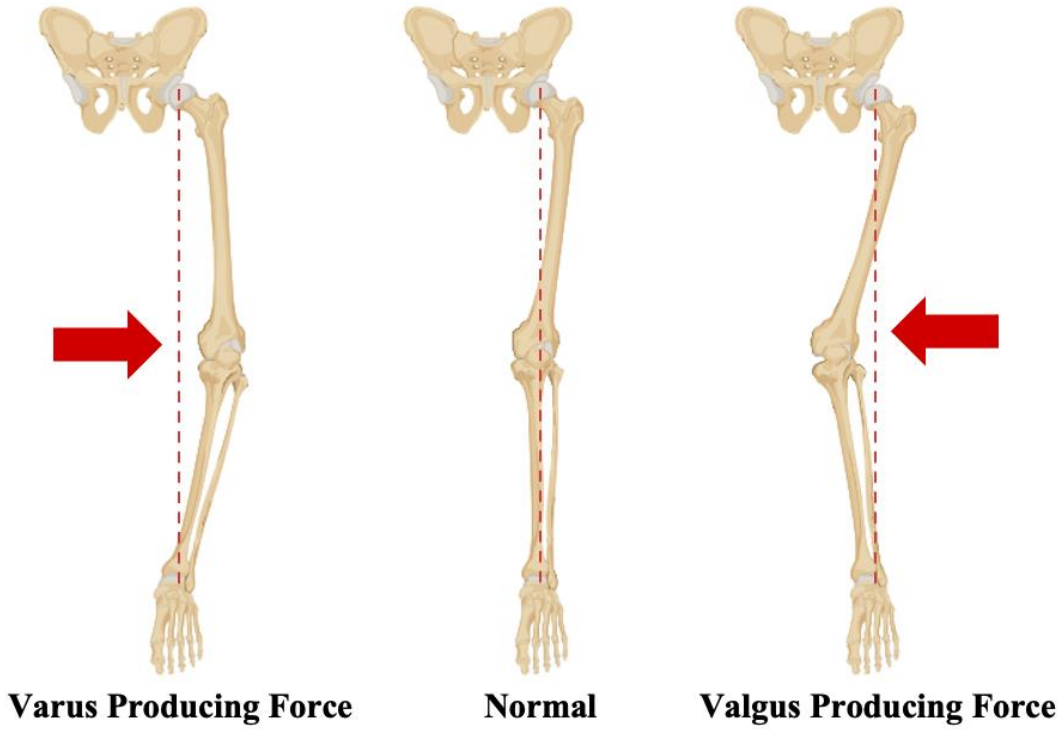


Figure 2-7: Directions of varus and valgus producing forces. A varus producing force may cause the LCL to tear. The MCL can tear under a valgus producing force. Figure generated with biorender.com.

The role of the PLC is to stabilize the knee against varus-producing forces and external rotation movements [9, 22, 78]. A varus-producing force is depicted in Figure 2-7. The LCL is the primary restraint for varus torque [18]. The PFL is important in resisting external rotation [80]. Selective sectioning studies that reconstruct some PLC components, leaving others disrupted, show that components work synergistically in addition to independently to resist these movements [21]. One selective sectioning study displayed that the popliteus tendon is also a minor stabilizer in preventing internal rotation, determined through an increase in internal rotational laxity once it was sectioned [23]. The popliteus muscle was not actively tensioned during the experiments; instead, researchers assessed passive restraint capabilities [23]. The components of the PLC have the secondary role of resisting anterior and posterior translation [22].

Posterolateral Corner Injury

The Fanelli and Larson classification system categorized PLC injuries into three types [35]. Type A PLC injury involves the PFL and popliteus tendon and a 10° increase in tibial external rotation. Type B PLC injuries affect the PFL, popliteus tendon, and the LCL. These injuries have a 10° increase in tibial external rotation *and* slight varus instability. Type C PLC injury involves the entire PLC and can include associated structures. Type C injuries display an increase in tibial external rotation plus *severe* varus instability.

Injury to the PLC occurs most commonly in cases of motor vehicle collisions, athletic-related trauma, and falls [26]. These occur through various mechanisms. LaPrade et al. published a study of 71 patients who had surgery to treat posterolateral knee injuries between 1985 and 1993 [25]. These researchers found that the most common mechanisms of injury were twisting (external rotation combined with hyperextension) (N = 21), noncontact hyperextension (N = 15), contact hyperextension (N =7), and a valgus force on a flexed knee (N = 5) [25]. DeLee et al. published a study of 12 patients that displayed isolated posterolateral instability of the knee, in which the mechanism of injury was a direct blow to the anteromedial tibia (directed posterolateral) while the knee is at or near full extension in nine cases [26]. These researchers note that the common

denominator in their patients was a force applied when the knee was near full extension [26]. These forces can produce a hyperextension and a varus moment, resulting in an isolated disruption of posterolateral structures.

An isolated injury of the PLC only comprises about 1.6% to 2.1% of all knee ligament injuries [26, 27]. Although this incidence is low, isolated injury only represents 13% of posterolateral corner injuries [26]. This statistic comes from a LaPrade et al. study of 30 patients with posterolateral knee structure injuries, in which 26 (87%) had multi-ligament damage [26]. Other studies have found this percentage of isolated injury to the PLC to reach as high as 28% [28]. Regardless, PLC injuries rarely occur in isolation. Injuries to the PLC often occur with other ligament damage, particularly with damage to the cruciate ligaments; the anterior cruciate ligament (ACL) and posterior cruciate ligament (PCL). The overall incidence of posterolateral knee injuries among all knee injuries is ultimately around 16% [26]. This includes all PLC injuries, including not only isolated tears but those that occur with multiple ligaments, as well. Conflicting literature has reported this statistic to be as high as 31.1% [29]. In a study of 61 trauma patients with acute hemarthrosis, 19 cases displayed posterolateral corner injuries [29].

The exact incidence of injury to this complex is relatively unknown. One factor that contributes to this is the difficulty in diagnosing a PLC injury. In a case report from 2016, a CrossFit athlete showed full range of motion but mild pain in the posterolateral knee, resulting in a diagnosis of a mild knee sprain [6]. Due to ongoing instability and muscle weakness, the patient saw an orthopaedic surgeon who noted mild varus angulation [6]. Further clinical tests resulted in the accurate diagnosis of a PLC deficiency, albeit a Type A injury given the lack of LCL damage [6]. In this case, the injury may have been missed due to its rare presentation of being damaged in isolation, resulting in the clinician overlooking PLC damage since no other ligaments were injured [6]. This theory is supported by a study from Pacheco et al., which found that only 28% of patients with posterolateral corner were correctly diagnosed at initial presentation, and all correctly identified cases involved multiple ligament injuries [30].

Symptoms of PLC injury include pain at the fibular head or joint line when palpated, edema, hardening, and diffuse tenderness [17, 24]. Abnormal gait is also an indicator of PLC injury [17, 30]. PLC injury is associated with a varus-thrust gait pattern (bowing-out) which involves a lift-off of the lateral compartment of the knee [6, 17]. This increases the stresses in the medial compartment; therefore, this gait pattern is associated with medial compartment osteoarthritis, rather than an indicator of PLC injury [17]. In addition to varus-thrust gait, patients may demonstrate hyperextension thrust during the stance phase of gait [24, 81]. It is typically used as an indicator of an ACL-deficient knee, adopted by patients to avoid engaging the quadriceps [24, 81]. It may also result from significant genu recurvatum instability due to PLC injury [24]. The PLC's disposition to multi-ligament damage makes it so that these injuries are frequently missed, as clinicians may prioritize and more easily recognize the damage to the major ligaments such as the ACL and neglect to consider the PLC as a potential cause of a gait abnormality [30]. Therefore, there is the potential that reported values of PLC injury are underestimated and that the injury is even more frequent than current literature may suggest [30].

Major consequences of PLC injury oversight include chronic knee instability and pain [31]. The altered loading patterns that cause these degenerative changes in the joint also cause a greater risk of graft failure in cruciate ligament reconstructions [32, 82]. O'Brien et al. conducted a retrospective review of ACL reconstructions and found in 76% of cases, laxity differed by three millimeters or less compared to the untreated knee [83]. They attributed the cases with more than three millimeters of translation to posterolateral instability, commenting that major associated ligamentous instability predisposes the reconstruction to failure [83]. This is also evident in a cadaveric study from LaPrade et al. that sectioned posterolateral components and recorded forces within an ACL graft [32]. When the LCL, PFL, and popliteus tendon were sectioned, graft forces were higher during varus loading than the joint with entirely intact posterolateral structures [32]. Early and accurate diagnosis of PLC injury is critical to preventing these complications.

A retrospective study that reviewed hospital records between 2005 and 2009 of patients referred to a specialist knee surgeon for a PLC injury found that 72% of patients had their PLC injury missed at initial presentation [30]. The most common alternative diagnosis was ACL injury [30]. Those that received correct diagnosis were those that had severe, multi-ligament injuries [30]. The lack of appropriate initial diagnosis is attributed to failure to perform appropriate clinical tests and failure to use supplementary diagnostic tools such as MRI [30]. Imaging techniques, such as MRI, can aid in diagnosing the PLC injury due to the complex anatomy and proximity of structures (Figure 2-8) [45]. An MRI can provide high accuracy in diagnosing soft tissue abnormalities like acute PLC injuries [34]. The diagnostic process should also include a neurovascular assessment, as the popliteal artery and common peroneal nerve, located between the superficial and middle layer of the posterolateral corner, are often damaged when the PLC sustains an injury. Damage to the common peroneal nerve results in numbness and weakness in the foot, a symptom of PLC injury that is often overlooked (Figure 2-6). Damage to this nerve occurs in 12.7% of posterolateral knee injuries [25].

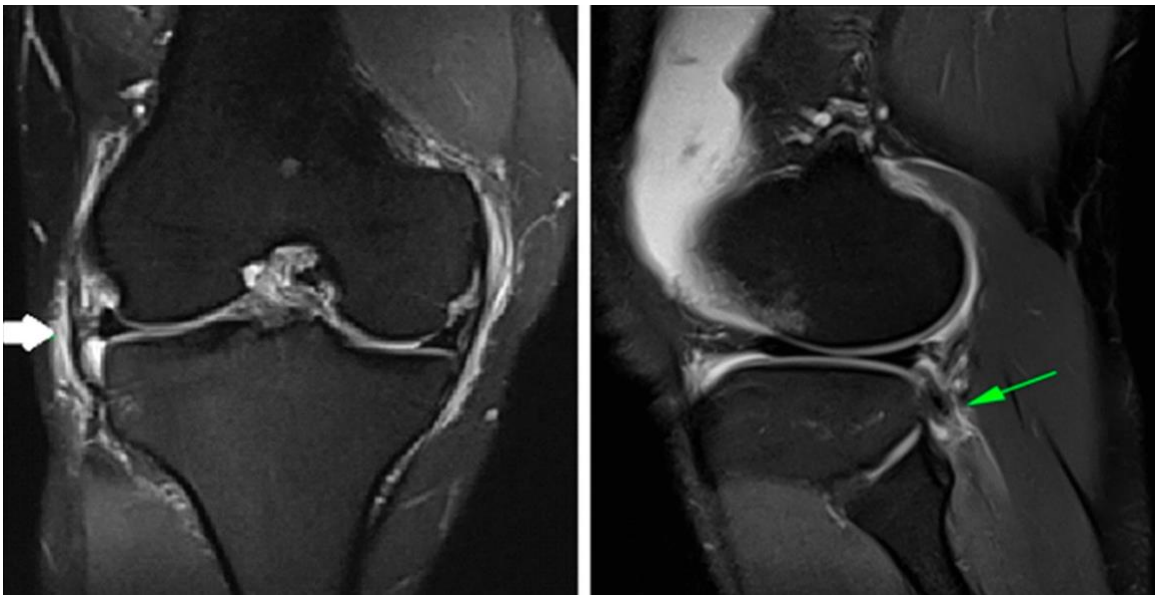


Figure 2-8: MRI can provide high accuracy in diagnosing PLC injuries [45]. MRI of 31-year-old male. Left: LCL tear (grade II). Right: Popliteo-tendinous complex tear (partial). (Image obtained from Algizawy et al. [45] under the following creative commons license: https://creativecommons.org/licenses/by/4.0/)

For accurate diagnosis, clinical tests like the dial test and the posterolateral drawer test are often utilized by physicians. The dial test is performed with the patient prone or supine. A clinician flexes the knee to 30° and 90° and externally rotates the tibia, then compares the two sides (Figure 2-9). Described by Cooper et al. in 1991, this test can determine if the injury is an isolated PLC injury or a combined PCL/PLC injury [60]. The posterolateral drawer test was described by Hughston and Norwood in 1980 [61]. It is performed supine. The knee is flexed to 90° and externally rotated by 15° (Figure 2-10). The clinician applies a posterior force to the proximal tibia. The clinician then compares laxity in the posterolateral compartment to the undamaged limb. Amongst other clinician performed tests, these tests investigate the PLC structural integrity under stress to rule out other causes of posterolateral knee pain. Combining these tests with imaging can help physicians to accurately diagnose a PLC injury – a crucial step to enable treatment planning and ensure optimal patient outcomes [17, 78].

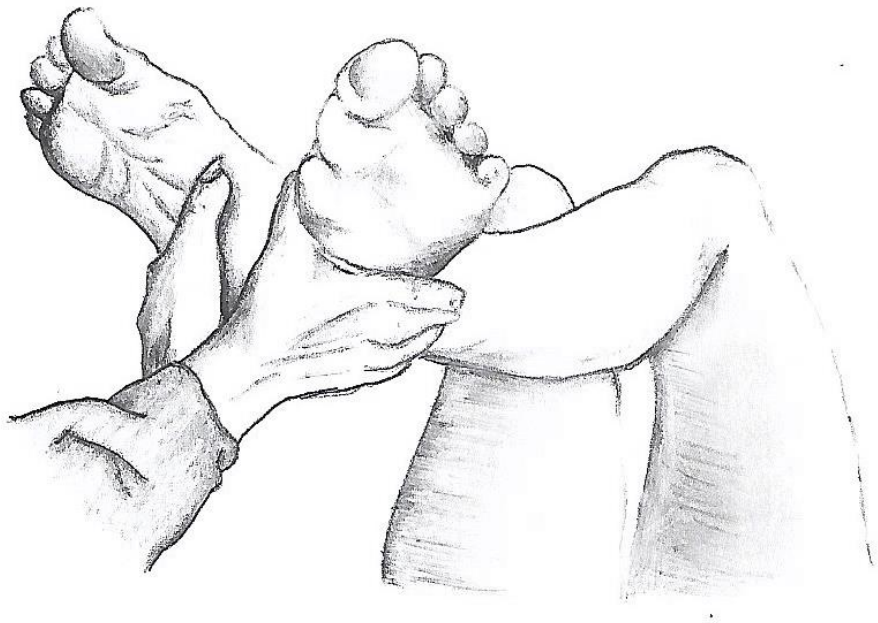


Figure 2-9: The dial test. This test, here being performed supine, consists of externally rotating the tibia and comparing each side for rotational laxity.

Figure 2-10: The posterolateral drawer test. The foot is rotated externally by about 15°, and a posterior force and external rotation torque is applied to the tibia.

Accurate diagnosis can enable the commencement of treatment planning, but determining proper treatment also depends on the classification of that injury. In addition to the Fanelli system of classification, the Hughston classification system of 1976 was the initial system developed for hierarchal organization of PLC complex injuries [15]:

Grade I: 0-5 mm of varus instability. 0°-5° rotational instability on dial test. Minor stretching or sprain of PLC structures and no abnormal motion.

Grade II: 6-10 mm of varus instability. 6°-10° rotational instability on dial test. Partial injuries to PLC structures with slight abnormal motion.

Grade III: >10 mm of varus instability. >10° rotational instability on dial test (no endpoint). Complete disruption of the ligaments and a fair amount of abnormal motion.

Classifying the grade of PLC injury helps the physicians determine appropriate management strategies specific to the severity of PLC injury. Grade I injuries are treated non-operatively, with rest, ice, and potentially rehabilitation. Grade II injuries are also treated in this way. Grade III injuries should be treated surgically, especially if the patient

presents with a concomitant cruciate ligament injury. An analysis conducted 8 years following grade III PLC injury found persisting severe or gross lateral laxity, muscle weakness, and osteoarthritis [36].

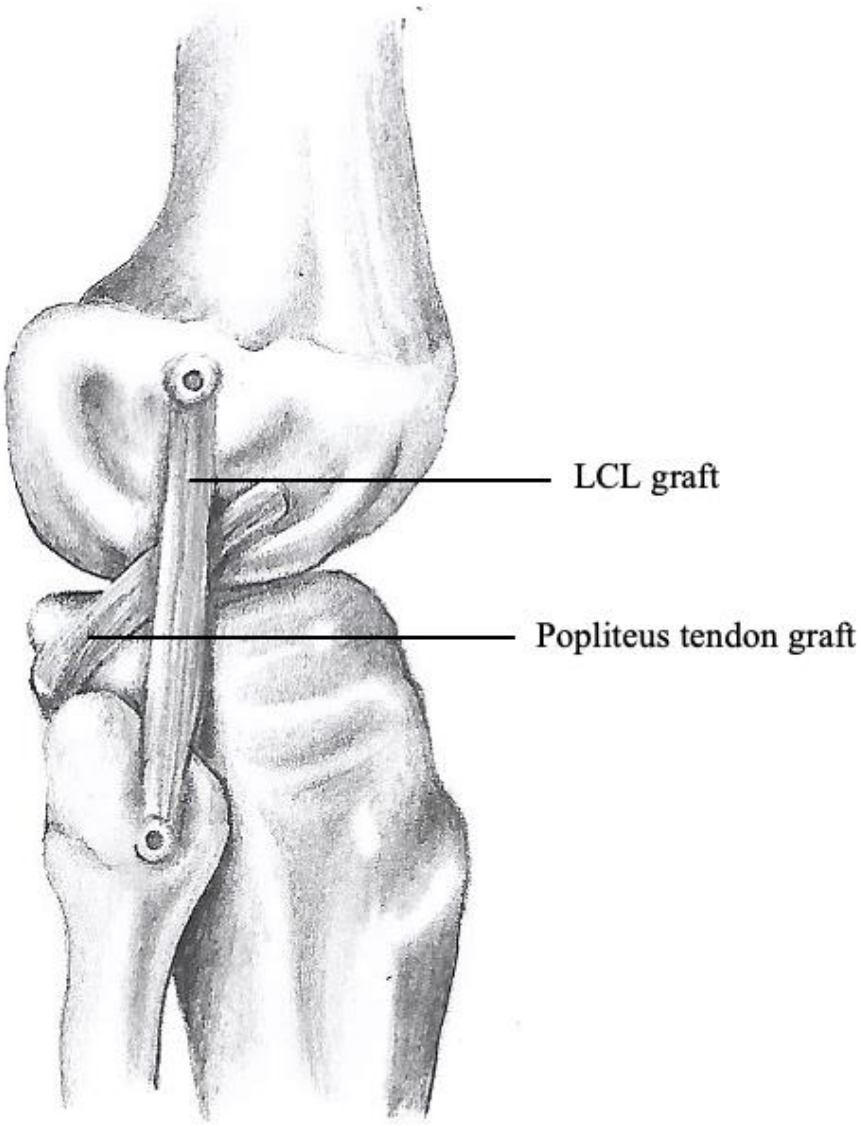


Figure 2-11: Anatomic reconstruction of the PLC. An anatomic reconstruction attempts to replicate the anatomy of PLC components using grafts. Figure adapted from van der Wal et al. [103].

Operative treatment can be divided into two categories: repair or reconstruction. Repair aims to reattach the torn ligament to its original location. Reconstruction aims to use graft tendons to reinforce the PLC components at anatomic sites (Figure 2-11). Stannard et al.

compared reconstruction and repair and reported a 9% failure rate in the reconstruction cohort, compared to a much larger 37% failure rate in the repair cohort [37]. These results agreed with a study conducted by Levy et al., that found a 6% failure rate in their reconstruction cohort, compared to a 40% failure rate in their repair cohort [38]. These studies suggest that reconstructions provide less risk of failure. Repair may be effective if accomplished within 3 weeks of the injury, however, the lower failure rates of reconstructions support it as the favoured PLC operative treatment. It is important to note that reconstructions, although shown to be more effective than repairs in these studies, will have much higher failure rates if untreated malalignment is present. An anatomic reconstruction from LaPrade et al. was compared to a modified anatomic technique proposed by Getgood et al. finding that both techniques restored varus laxity and external rotation in extension after PLC reconstruction [108].

2.3 Malalignment

The alignment of the limb is an important factor to consider prior to any reconstructive procedures of the lower limb [83, 84]. Malalignment can place additional tension on PLC grafts causing them to stretch out and fail over time, resulting in greater risk of a failed reconstruction [84]. Typically, this type of malalignment is a varus malalignment or "bow-legged" alignment [84]. Literature suggests that a high tibial opening wedge medial osteotomy should be performed prior to PLC reconstruction because of the additional stress the altered loading pattern that results from malalignment places on the grafts [39]. A high tibial opening wedge medial osteotomy allows the surgeon to make bony corrections of the knee, altering the *sagittal* and *coronal* planes. Our concern is with the alignment within the transverse plane.

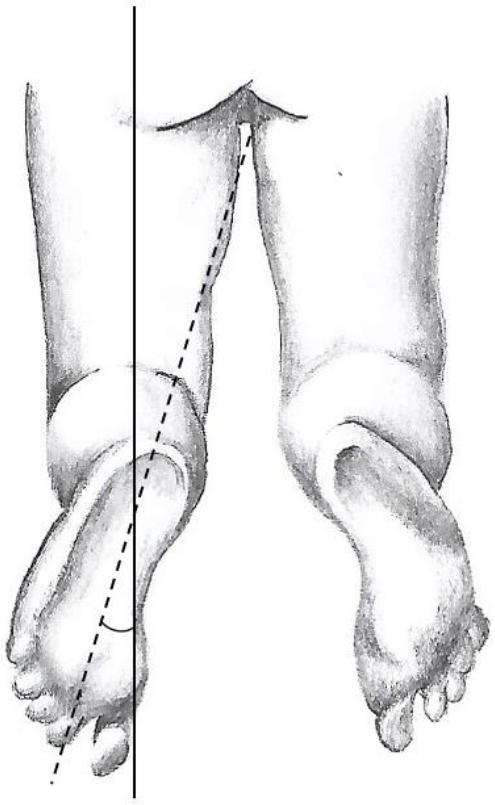


Figure 2-12: Thigh foot angle (TFA) is used to quantify rotational malalignment. The TFA is an angle between the longitudinal axis of the femur and the longitudinal axis through the 2nd metatarsal with the foot in neutral [62].

Tibial torsion is a form of tibial malalignment. The tibia is abnormally twisted along its longitudinal axis such that the rotational alignment of the planes of motion of the proximal and distal articulations are altered [40, 86]. In other words, tibial torsion is a change in the rotational alignment between the tibial plateau (the flat, top surface of the tibia) and the malleoli (the bony projections on either side of the ankle) [40]. There are two types of tibial torsion: internal and external [88]. Internal tibial torsion is the most common cause of in-toeing from age 1 to 3 years and tends to resolve with age [91]. External tibial torsion tends to increase with age and is associated with overtreatment of internal tibial torsion and neuromuscular conditions like myelodysplasia [91, 92]. Collaborators from Kliniek ViaSana (Mill, Netherlands) identified an injury pattern in a series of patients, noting that those presenting with PLC injury exhibited excessive tibial torsion, particularly external tibial torsion. External tibial torsion involves the external rotation of the foot and malleoli relative to the knee [89]. Patients with external tibial torsion will often compensate for the external rotation of their feet by adjusting their gait and posture [86, 88-90]. To maintain a more forward-facing foot alignment, various compensatory abnormalities occur across subjects and can include pelvic rotation, hip abduction/adduction; hip and ankle transverse rotations and contralateral limb compensation [86, 88-90]. External tibial torsion is a cause of patellofemoral instability, accounting for 8% of cases of patellar instability [90].

An inward rotation of the proximal tibia, characteristic of an individual with external tibial torsion, may decrease the leverage of the PLC. With reduced leverage, the PLC may require greater tension to counteract the forces of hyperextension, which can potentially strain the ligaments and soft tissues of the PLC complex. Therefore, individuals with external tibial torsion may be more susceptible to PLC injuries due to the altered biomechanics and decreased leverage of the posterolateral corner. Herein throughout this thesis, this suspected possible injury mechanism will be referred to as the **Compensatory Alignment Injury**, because of the compensatory internal rotation of the proximal tibia to yield a normal foot orientation.

This thesis also explores an alternative theory, such that if those with external tibial torsion undergo a hyperextension force, there is the potential that the knee is in a

"normal" rotational position, not in the internally rotated compensatory pattern (Figure 1-5). During a hyperextension "landing", because of the external torsional deformity, the foot may angle outward. This outward foot angle causes some of the force to be directed in a way that induces an external rotation torque on the knee. These forces cause additional stress on the PLC. The PLC can manage these forces under normal conditions, however, when the knee is subjected to abnormal external rotation due to the outward foot angle, the PLC is forced to endure greater strain. This can predispose the PLC to injury through increased tension required to counteract the abnormal forces presented by external rotation torque. Herein throughout this thesis, this suspected possible injury mechanism will be referred to as the **Non-compensatory Alignment Injury**, as it would occur when landing on an externally rotated foot, when the tibial external torsional deformity was not compensated for by internally rotating.

Tibial torsion can impact how forces are distributed across the knee joint, resulting in changes in gait [41, 85]. The foot progression angle (FPA) is the angle between the long axis of the foot from heel to 2nd metatarsal and the line in which an individual is walking [86]. An increase in external tibial torsion results in a greater FPA (toe-out gait) [40]. Rotational malalignment of the tibia can be estimated using the thigh-foot angle, the angle between the longitudinal axis of the thigh and the longitudinal axis through the 2nd metatarsal, amongst other techniques (Figure 2-12) [62, 86]. One study measured tibial torsion of patients attending an adult knee clinic and found that the average external torsion in the control group was 19° [41]. Measurements of external tibial torsion were higher in those that had unstable patellofemoral joints and Osgood Schlatter's disorder, and lower in those with osteoarthritis [41]. Hutter et al. examined 40 adult skeletons and found the average external tibial torsion to be 22.1° for the right tibia and 19.8° for the left tibia [88]. Mullaji et al. CT scanned 100 non-arthritic Indian adults, finding average tibial torsion to be 21.7° [93]. Drexler et al. suggest that an angle greater than 30° can be diagnosed as excessive external tibial torsion [94]. We simulated excessive external tibial torsion by rotating the knee by 5° and 10° internally (with the understanding that the proximal tibia will have more internal rotation to compensate for the externally rotated foot in order to attempt a more neutral lower limb alignment) [40]. We also recorded measurements for rotations of the knee by 5° and 10° externally to compare the alternate

theory that suggests that the compensatory pattern is not being utilized during a hyperextension mechanism. With the assumption that the tibia typically has an external torsion of about 20°, adding a subsequent 10° of torsion will result in the simulation of excessive external tibial torsion.

This thesis primarily investigates the role of tibial torsion on loading of the PLC components during a simulated hyperextension injury.

2.4 Overview of Protocol Techniques

It has been established that knee hyperextension can damage the components of the PLC. Studies that investigated the soft tissues that restrain hyperextension used cadaveric knee specimens, forced them into hyperextension, and analyzed which structures were damaged through gross dissection. Fornalski et al. used this methodology and hyperextended cadaveric knees to 15° and 30° of hyperextension, finding that after hyperextension to 30°, the PFL was intact in all the specimens, whereas the LCL and popliteus tendon had avulsed in some cases [42]. Schenck et al. hyperextended cadaveric knees to 45° and analyzed cruciate ligament knee injuries [43]. In 2010, Morgan et al. published a study using a different technique to examine the restraints to knee extension; selective ligament sectioning [2]. Selective ligament sectioning studies are used to examine components individually, by sequentially cutting specific ligaments and observing the resulting changes. Morgan et al. applied hyperextension torques of 14 Nm and 27 Nm before and after sectioning individual components of the posterior knee, posterolateral knee, and cruciate ligaments, recording the increase in hyperextension angle [2]. This technique is known as the principle of superposition.

Superposition

Superposition refers to the use of sequential sectioning of ligaments or soft tissues, observing the mechanical response after each ligament is cut. This can provide information on the contribution each soft tissue provides to resisting loads. In addition to the Morgan et al. study, other studies have used superposition to examine PLC structures under hyperextension stress, such as the work of Noyes et al. [3]. In brief, using this

technique, Noyes et al. found that there was no primary restraint to resisting hyperextension, and that multiple structures and the interactions between them control this motion [3]. Gollehon et al. looked at posterolateral and cruciate ligaments from zero to 90°, selectively sectioning the LCL, popliteus-arcuate (deep) ligament complex, anterior cruciate ligament and posterior cruciate ligament [49]. They found that the LCL and deep ligament complex functioned together as principal structures preventing varus rotation and external rotation of the tibia, albeit, only analyzing up to 0° of flexion [49]. In comparison to the Noyes et al. article, Morgan et al. found that the oblique popliteal ligament was the primary restraint to knee hyperextension when 27 Nm of hyperextension was applied [44]. Superposition, as utilized in these studies, is utilized in our study as well. Our study includes the addition of a rotation to replicate transverse plane malalignment. Each of the components of the posterolateral corner are sectioned selectively (e.g., the LCL, the capsule + PFL, and the popliteus tendon). The principle of superposition is applied through evaluating the change before and after the soft tissue is cut. This process is explained in more detail in the methods section of the study described in Chapter 3.

Chapter 3

3 The Role of Transverse Plane Malalignment in Posterolateral Corner Complex Injury

The motivation and introduction to this study, including a brief overview of the posterolateral corner and tibial torsion, are provided in Chapter 1 and 2. This chapter begins with the methods used to conduct this study. These include the utilization of the six degrees-of-freedom (6-DoF) joint motion simulator (VIVO, Advanced Mechanical Technology, Inc., Watertown, MA), which uses forces and displacements to investigate the role that the components of the PLC have during a simulated hyperextension injury. The study presented in this chapter will analyze the effect that a tibial deformity causing transverse plane malalignment has on PLC load distribution. The results of this study are then presented and discussed.

3.1 Methods

Specimen Preparation

Approval for this study was obtained from the institutional Research Ethics Board (REB) (REB#: 2023-124025-86735). Specimens were sourced from United Tissue Network. Donor summaries including information such as age, sex, height, weight, BMI, and other relevant medical history information were provided. Specimens with history of knee surgery or injury, arthritis, bone cancer, osteoporosis, or other pathologies that may compromise the integrity of the knee joint were excluded from the selection. Twelve fresh-frozen human cadaveric mid-femur to mid-tibia knee specimens (aged 47-61 years, 5 pairs, all male, with a mean BMI of 24) were selected from available donors and used for this study. Specimens underwent CT scanning and were assessed for any damage or disorders (arthritis, bony overgrowth, abnormalities) that could affect the biomechanical testing of the specimen. Specimens were thawed at room temperature for 24 hours prior to testing. Once thawed, soft tissues surrounding the proximal femur and distal tibia/fibula were denuded, approximately 100 mm from the knee joint center. The

exposed portion of the bones were scraped clean to ensure a dry surface for optimal adhesion into dental model stone (Modern Materials Golden Denstone Labstone, Modern Materials, Kulzer GmbH, Hanau, Germany). The dry femur can then be potted in a ~130 mm in length, 2” diameter polyvinyl chloride (PVC) pipe to mount onto the joint motion simulator, the robotic testing apparatus employed in this research.

Robotic System Overview

Biomechanical testing of each cadaveric knee specimen was conducted using a six degrees-of-freedom (6-DoF) joint motion simulator (VIVO, Advanced Mechanical Technology, Inc., Watertown, MA) (Figure 3-2). The 6-DoF joint motion simulator, or VIVO, uses Grood & Suntay coordinate conventions to express forces and motions [1]. The Grood and Suntay joint coordinate system uses spatial linkages of anatomical landmarks to describe joint position. The VIVO manipulates the joints of cadaveric specimens through two actuators. The upper (femoral) actuator of the VIVO controls flexion-extension and abduction-adduction (varus-valgus) rotations, whereas the lower (tibial) actuator translates in the anterior-posterior, medial-lateral, and proximal-distal (superior-inferior) directions and controls internal-external rotation. Closed-loop controls enable the VIVO to operate in either force or displacement control mode. In force control, loads are applied directly to the joint, producing a displacement that can be measured. In displacement control, motions are applied to the joint, resulting in reaction forces that also can be quantified. The lower actuator houses a 6-DoF load cell, which is responsible for recording measurements of these forces or moments. While not mandatory for all VIVO experiments, a stepper motor (86HSE154 RATTM-Motor, 12 Nm holding torque, RATTM-Motor, Beijing, China) was employed specifically for these tests, connected to a planetary gearhead (34SP010L-90mm, Carson MFG, Carson City, NV) so maximum allowable torque was increased to 120 Nm from the 10:1 gearbox. The stepper motor connects to the arm of the upper actuator, extending the varus-valgus range of motion beyond the mechanical limits of the VIVO.

Specimen Alignment

The potted femur can be mounted to the upper actuator of the VIVO using a custom-designed aluminum fixture. This fixture allows for the adjustment of the femur along anterior-posterior, superior-inferior, and medial-lateral axes, as well as adduction-abduction movement, to align the specimen's flexion axis with the mechanical flexion axis of the joint motion simulator. An alignment technique utilizing motion capture was used to ensure the agreement of these flexion axes. Optical motion trackers (Optotrak, Northern Digital Inc., Waterloo, CA) were installed on the femur and tibia. Three-dimensional reconstructions of each specimen's femur and tibia/fibula generated from computed tomographic (CT) images in 3D Slicer were co-registered to the actual specimen and to a tracker located on the VIVO. The femur's center and co-registration axis were defined within ParaView, by fitting spheres to the posterior medial and lateral condyles and assuming the midpoint is the femur-flexion axis. The motion capture system was then able to visualize the joint and corresponding spheres and registration axis to align the joint with respect to the coordinate system of the joint motion simulator (Figure 3-1). This was done by adjusting anterior/posterior, medial/lateral, and inferior/superior femoral mounting fixtures to align the femur-flexion axis sphere to the flexion axis of the joint-motion simulator. These adjustments were made using the custom-designed aluminum fixture. The ideal alignment of each specimen was a position within 2 mm and 1° from the VIVO's mechanical axes. Once it was concluded that femoral alignment agreed with that of the VIVO, by verifying a neutral position between the specimen and simulator, the tibia/fibula were potted. The tibia and fibula were scraped clean to ensure optimal adherence of dental stone and potted together *in situ* into a tibial pot (3" diameter polyvinyl chloride (PVC) Eaves Downspout Adapter), then affixed to the VIVO's lower actuator. Prior to cementing the tibia, the fixtures were then used to re-adjust the position of the joint to align the tibia vertically within the frontal and sagittal plane. This was achieved through adjustments of the femoral abduction/adduction fixture, and anterior/posterior position was adjusted via a custom fixture attached to the lower actuator. Taking particular care when performing the alignment protocol minimizes secondary motion and maximizes the range of motion of the VIVO. Once the specimen was aligned properly, the tibia was cemented and left to dry, following which the joint

was then subjected to 50 N of compression force. A “reference pose” or zero position for all DoF of the specimen was set at 0° flexion, with the exception of internal/external (IE) rotation. IE rotation was set to the value of IE rotation the specimen displayed when 27 Nm of hyperextension torque was applied, so that the specimen was at a “neutral” or zero position during the biomechanical testing protocol (Figure 3-1).

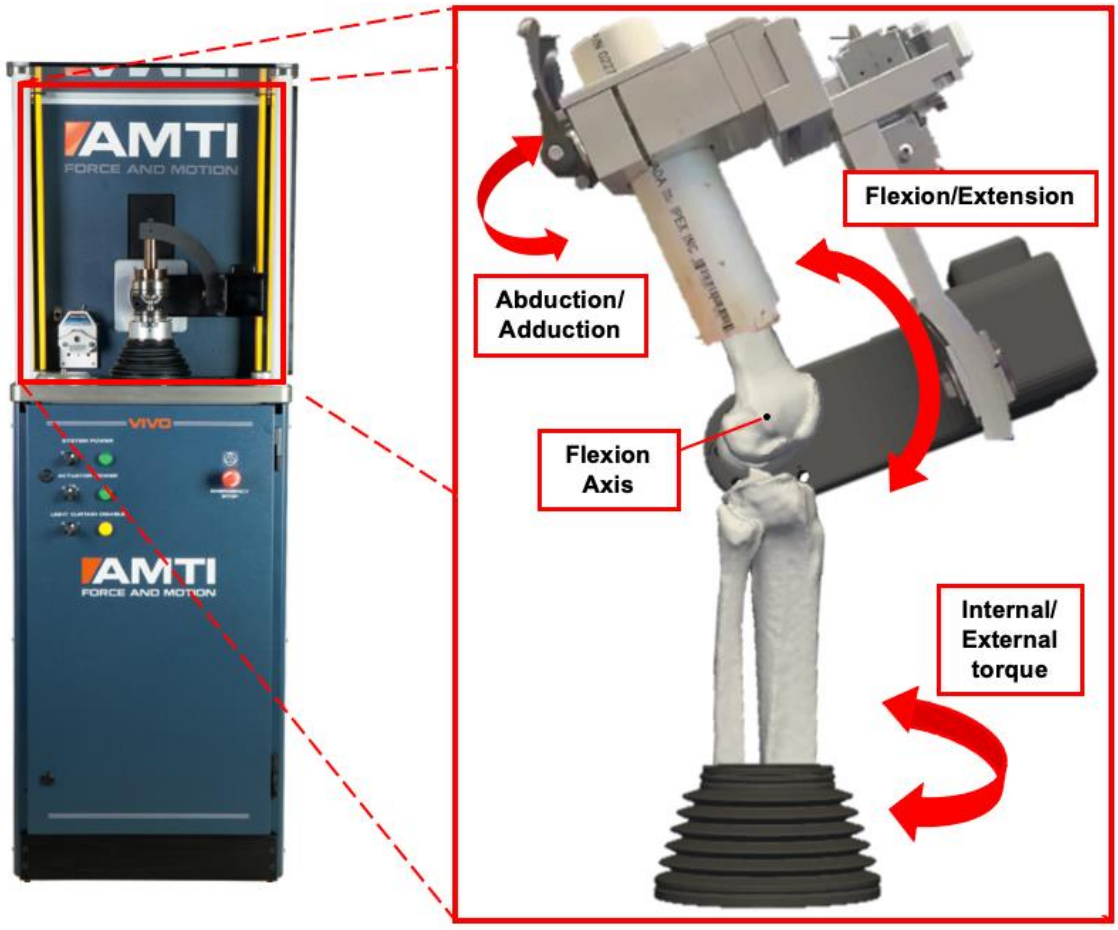


Figure 3-1: AMTI VIVO joint motion simulator alignment and rotations.

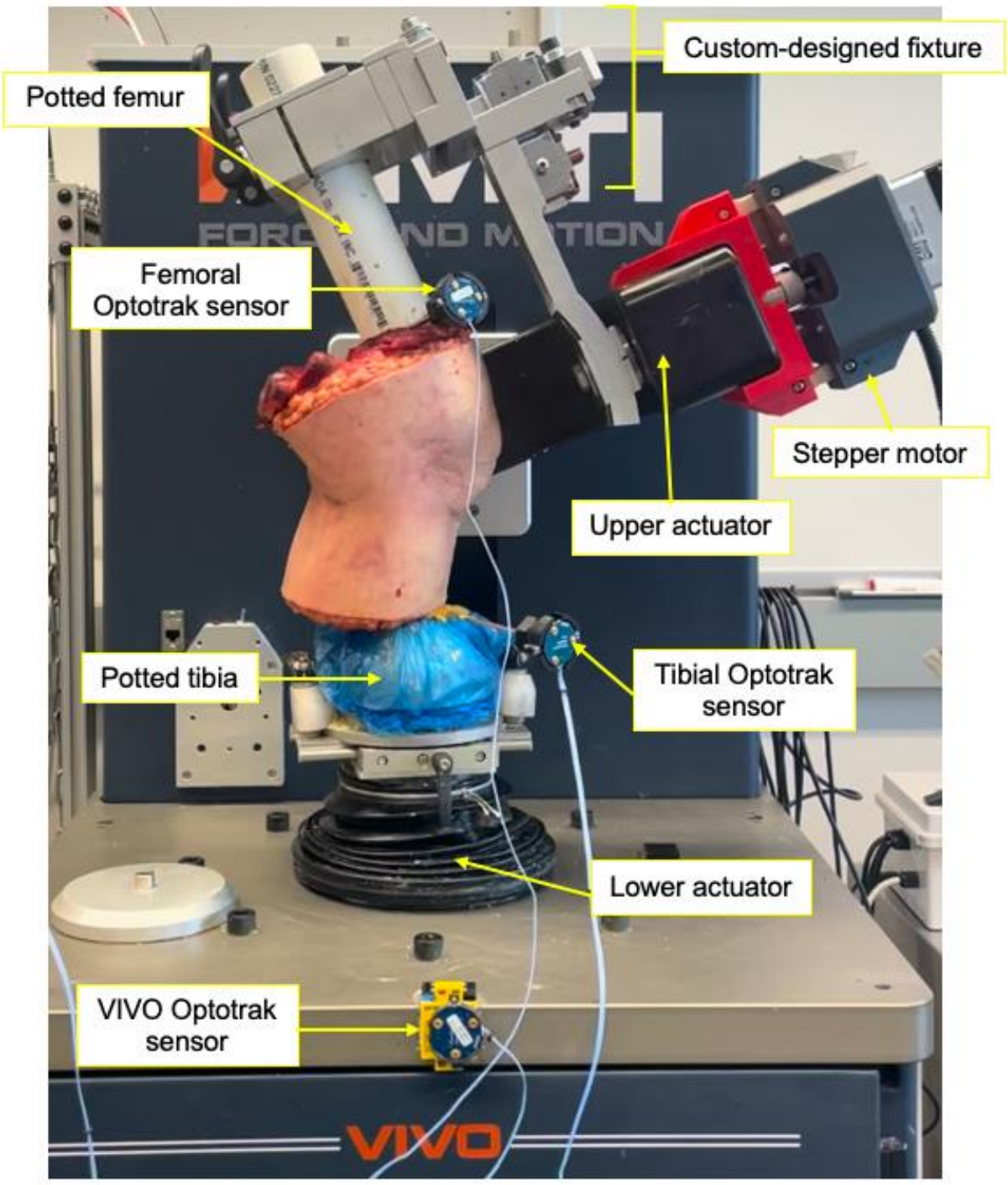


Figure 3-2: AMTI VIVO Joint Motion Simulator Experimental Setup. Experimental setup of a cadaveric specimen loaded onto the AMTI VIVO joint motion simulator. The stepper motor maximizes varus-valgus range of motion. Optotrak sensors allow for visualization of the joint for aligning with the center of the VIVO mechanical axes.

Intact Joint Testing

Once the specimen had been mounted and aligned on the VIVO joint motion simulator, 27 Nm of hyperextension torque was applied to the fresh-frozen cadaveric knee specimen using the 6-DoF VIVO joint motion simulator, with all DoF in force control. 27 Nm was derived from previous studies [2, 3]. Morgan et al. used 27 Nm in their cadaveric study that evaluated the role of the oblique popliteal ligament, posterolateral components, and cruciate ligaments during knee hyperextension [2]. These authors based this value off a study from Kerrigan et al. that explored maximum hyperextension torques in patients with and without hyperextension during gait [2, 95]. Noyes et al. used 27 Nm of hyperextension torque in their investigation of the functional interaction of cruciate ligaments and posterior knee structures in preventing hyperextension [3].

The specimen's flexion (hyperextension) angle under the hyperextension torque of 27 Nm was recorded. This was repeated with an additional 10 Nm of varus torque applied, to more accurately replicate a hyperextension injury mechanism, again recording the maximum flexion (hyperextension) angles achieved during the hyperextension moment. These values represent an intact knee that does not contain external tibial torsion; therefore, we describe this data as baseline/0° of rotation (Figure 3-3). Repetitions of this test were conducted for increments of 5° and 10° external rotation, and 5° and 10° internal rotation. Each increment aimed to mimic the tibial torsional deformity, to determine how internal and external rotational offsets influence the hyperextension limit, to ensure that limit was not exceeded and causing specimen damage when hyperextending with an IE offset.

It is important to note that each of these tests were repeated twice, utilizing the recorded value from the second repetition in all subsequent steps of the study, to have exercised the joint to minimize hysteresis effects by allowing the soft tissues to creep.

Biomechanical testing began by recording the neutral path of motion. To assess this path, the knee was put through a loading scenario involving cyclical flexion from 30° flexion to the maximum flexion angle achieved under 27 Nm of hyperextension torque (previously determined). During this loading protocol, the knee was subjected to a 50 N

of compressive force applied parallel to the longitudinal axis of the tibia. The remaining five DoF were unconstrained, allowing the specimen to move freely in those directions, under the influence of the applied compression, articular contact and soft tissue constraints. This loading scenario was repeated, except the IE rotation DoF was offset by 5° and 10° internally (to simulate the **Compensatory Alignment Injury** mechanism) and externally (to simulate the **Non-compensatory Alignment Injury** mechanism). Rotating internally aims to simulate the theory regarding external tibial torsion such that the patient experiences the hyperextension force while the feet are neutral. This would mean there is an internal rotation occurring at the proximal tibia, which we aim to simulate by 5° and 10°. The alternative theory is that a patient may experience hyperextension with the feet in an externally rotated position, therefore forces are directed such that the proximal tibia has a resulting external rotation. This is why we also tested this theory by rotating by 5° and 10° externally. Therefore, measurements of both external and internal rotations were collected. 6-DoF joint kinematics were recorded. Each test was repeated five times, recording kinematics through the 2nd and 3rd cycles. Kinematics were recorded through position encoders of the VIVO.

Recorded kinematics were applied back on the specimen, with the joint motion simulator operating in displacement control, allowing for *in situ* ligament forces to be measured. Data was acquired at a rate of 200 datasets/second with 3 cycles lasting 30 seconds each. The change in joint reaction forces, including flexion torque, valgus rotation torque, and external rotation torque is representative of the contribution of the most recently sectioned structure to the total joint reaction force, according to the principle of superposition.

Ligament and Soft Tissue Sectioning

Following the completion of biomechanical testing on the intact knee, individual components of the PLC were identified and sectioned by a fellowship trained sports medicine surgeon with experience in knee reconstruction. The joint was then loaded with the same displacement protocol as the intact knee, measuring the joint reaction forces. All displacement control tests were conducted at the same loading rate; therefore, the relative

force contributions of the ligaments should remain unchanged. Specimens either had the LCL sectioned first (N = 6), or the posterolateral capsule + popliteofibular ligament (PFL) sectioned first (N = 6) (Figure 3-3). This division aimed to investigate if potential differences in loading result from the sequence of cuts.

For the sectioning procedure, a curvilinear skin incision was made over the lateral knee, extending from the proximal lateral epicondyle toward Gerdy's tubercle on the anterolateral tibia. A horizontal incision was then made in the distal biceps bursa to identify the distal LCL, which was tagged with traction sutures to facilitate the identification of both femoral and fibular attachment sites. In the group where the LCL was sectioned first, the cut was made at the femoral attachment; in the group where the capsule + PFL were sectioned first, the LCL was cut at the fibular attachment.

For the PFL, the approach was made posterior to the biceps and anterior to the lateral head of the gastrocnemius. The PFL, running between the fibular styloid and attaching to the popliteus, was identified and sectioned. The capsulotomy was made from the lateral to posterior femoral condyle.

The LCL-deficient or Capsule-and-PFL-deficient knee was then subjected to the same kinematics and loads as the intact knee. Joint reaction forces were recorded. This process was repeated after transection of the ligament/soft tissue that was not sectioned first (either LCL or Capsule + PFL). Again, the specimen underwent the same kinematics and loads as the intact knee and joint reaction forces were recorded. The process was reiterated following transection of the final component of the PLC, the popliteus tendon.

To identify the popliteus, the iliotibial band was split at the level of the lateral epicondyle, and an anterior capsulotomy was performed to visualize the insertion of the popliteus tendon. The popliteus was then cut at its femoral attachment.

The resulting PLC-deficient knee, lacking LCL, posterolateral capsule, PFL, and popliteus tendon, was again subjected to the same loading protocol, and joint reaction forces were recorded for a fourth and final time.

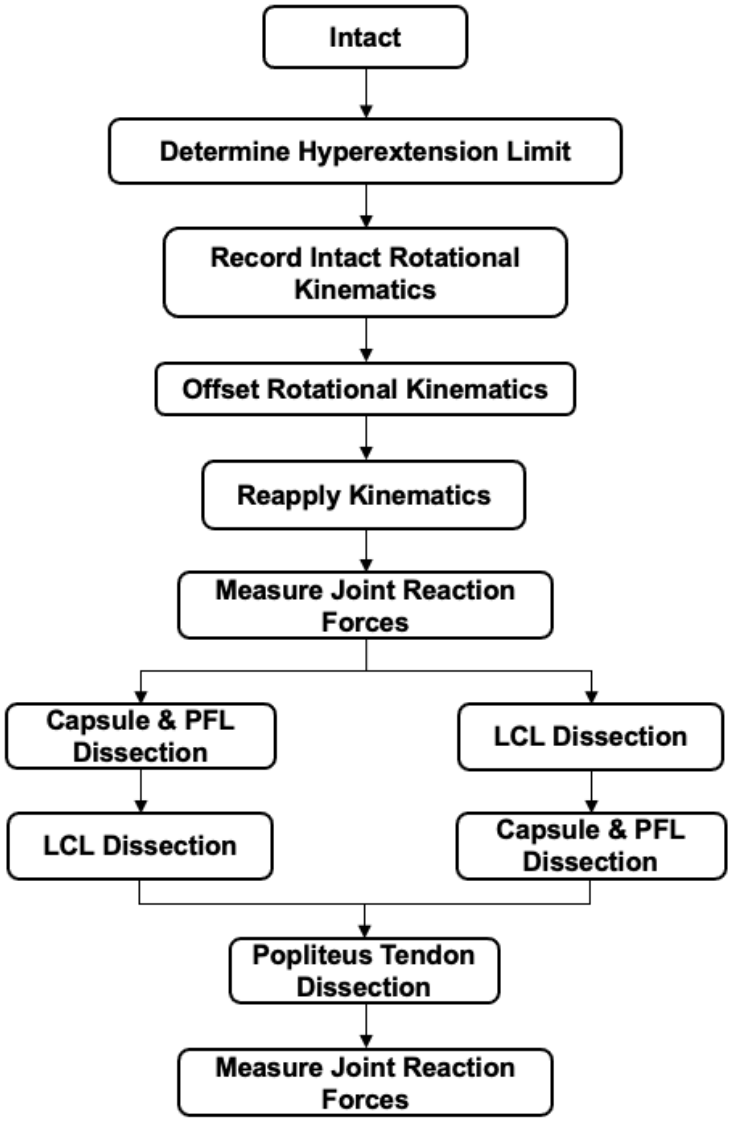


Figure 3-3: The protocol used in this study to measure the offset rotational kinematics to simulate knee loads anticipated from Compensatory and Non-compensatory Alignments.

Data Analysis

Kinematic data were filtered using a second-order Butterworth filter with a cut-off frequency of 1 Hz. Values for flexion torque, valgus/varus rotation torque, and internal/external rotation torque were recorded before and after each PLC component was sectioned. Plots of torque versus flexion angle were created, and from these plots, values

of torque at peak hyperextension were collected. This allowed for calculation of torque contributions of each individual PLC component. Values were scaled such that 100% represents an intact knee with normal hyperextension restraint. For example, if the recorded restraint after cutting the LCL showed 85% of intact, the value displayed in each figure would be 15%, displaying that cutting the LCL results in losing 15% of the knee's restraint. Examples of these plots, resulting data collection charts, and calculations performed are displayed in the appendix. A statistical comparison of peak hyperextension angles during rotation of the knee was performed using a one-way repeated-measures analysis of variance (RM-ANOVA). A paired samples t-test was performed for comparison of the structures restraining hyperextension at a neutral alignment. Statistical comparisons of Compensatory and Non-compensatory Alignments for individuals with a torsional deformity were performed using a two-way RM-ANOVA. Within-subject variables were structure (ligament(s) sectioned) and the rotation angle (10° ER, 5° ER, 0°/Baseline for external rotation analysis, and 0°/Baseline, 5° IR, 10° IR for internal rotation analysis). The structures include: (1) LCL, (2) Capsule + PFL, and (3) Popliteus Tendon. The cutting order was a between-subject variable, given that there was a cohort of LCL-cut first specimens and a cohort of capsule + PFL-cut first specimens. These were performed with a threshold of alpha = 0.05, after using a Bonferroni correction. A p value of < 0.05 was considered statistically significant. These statistical comparisons were performed using a commercial statistics software package (SPSS, v.29, IBM SPSS).

3.2 Results

3.2.1 Peak Hyperextension

At 27 Nm of hyperextension torque without an additional varus torque, peak knee hyperextension ranged from 1.1° to 26.5°, with an average of 14.7°$\pm$ 7.4° (Appendix Table 1). With the addition of 10 Nm of varus torque, peak knee hyperextension ranged from 1.1° to 27.4°, with an average of 14.9°$\pm$ 7.6° (Appendix Table 2). Relative to neutral, 5° IR of the tibia resulted in a significant decrease in hyperextension (by 0.5° $\pm$ 0.1° without varus, $p = < 0.001$; and by 0.4° $\pm$ 0.2° with varus, $p = 0.041$) (Figure 3-4). Likewise, 10° IR of the tibia resulted in a significant decrease in peak knee hyperextension relative to neutral (by 1.6° $\pm$ 0.1° without varus, $p = < 0.001$; and by

1.5° ± 0.5° with varus, $p = 0.002$) (Figure 3-4). Externally rotating the knee had a lesser effect on the peak hyperextension angle achieved when 27 Nm of hyperextension torque was applied. Relative to neutral, 5° ER of the tibia did not result in significant differences in hyperextension with respect to neutral (by 0.1° ± 0.1° without varus, $p = 0.349$; by 0.1° ± 0.2° with varus, $p = 0.407$) (Figure 3-4). A 10° ER of the tibia resulted in significant decreases in hyperextension from neutral without varus (0.5° ± 0.3°, $p = 0.007$) but did not with varus torque (0.1° ± 0.2°, $p = 0.720$) (Figure 3-4). Although very minimally, we can conclude that rotating both internally and externally caused peak hyperextension to decrease. Varus torque did not influence the peak hyperextension angle ($p = 0.357$).

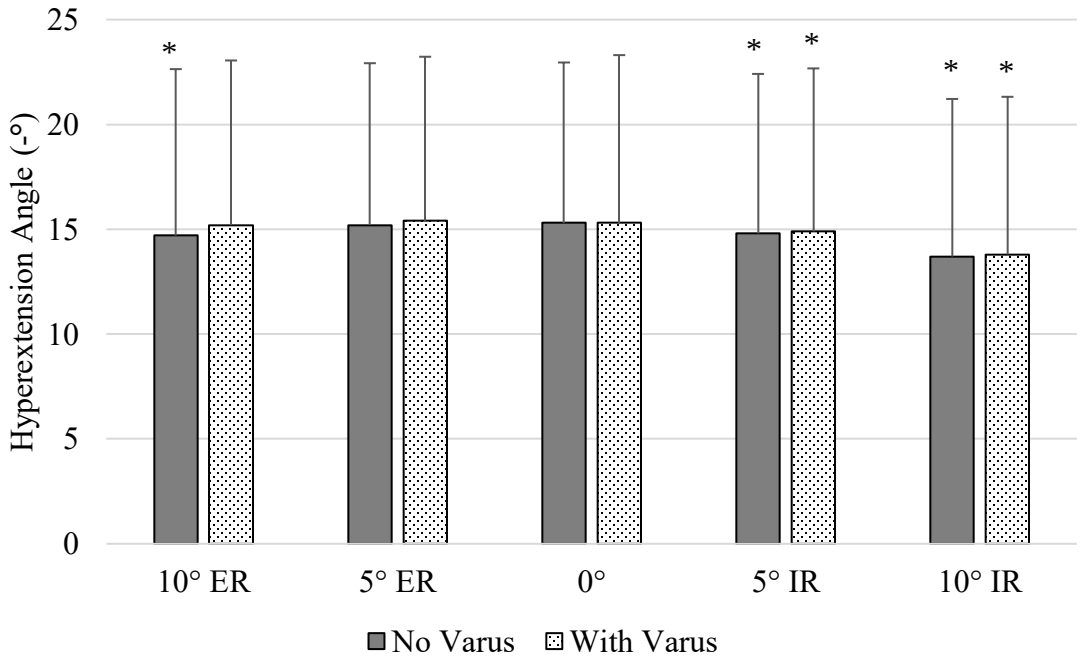


Figure 3-4: Hyperextension angle reached by specimens when 27 Nm of hyperextension torque was applied. * Indicates significant differences from neutral. These include 5° IR (with and without varus torque), 10° IR (with and without varus torque), and 10° ER (without varus torque).

3.2.2 Flexion Torque – Neutral Alignment

In the absence of varus torque, the LCL provides the greatest restraint to hyperextension at a neutral rotational alignment (15.8 ± 7.6 %; Figure 3-5). The mean contribution of the capsule + PFL was slightly less (10.8 ± 6.8 %; Figure 3-5), but this difference was not statistically significant ($p = 0.198$). The popliteus tendon provided the least restraint, only contributing 6.0 ± 3.7 %, which was 9.7 ± 1.6 % less than contribution of LCL ($p = 0.001$) (Figure 3-5). The contributions of the capsule + PFL and popliteus tendon were not significantly different ($p = 0.087$).

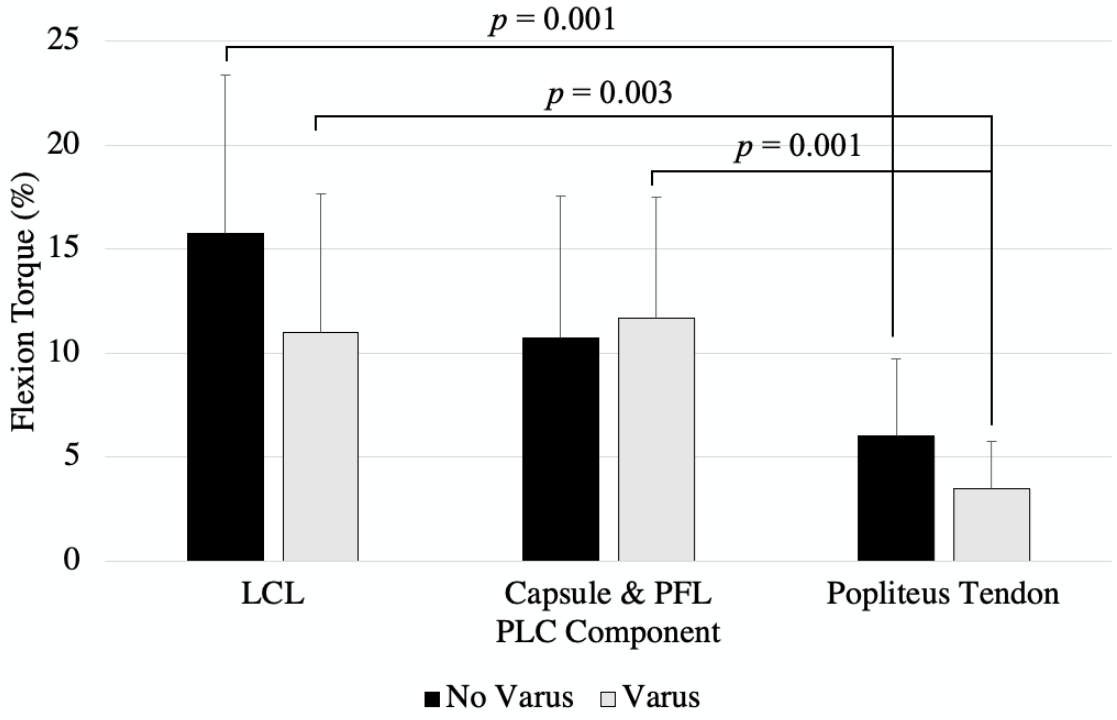


Figure 3-5: PLC restraint of 27 Nm of hyperextension, at neutral rotation.

When varus torque was added, the capsule + PFL provided the most restraint (11.7 ± 5.8 %; Figure 3-5). The LCL contributed an average of 11.0 ± 6.6 % (Figure 3-5). The contributions of the LCL and capsule + PFL were not statistically different ($p = 0.830$). Although the addition of varus torque caused the LCL and capsule + PFL to provide nearly equal amounts of hyperextension restraint, the contribution of the

popliteus tendon was still the lowest (3.5 ± 2.3 %), significantly less than the contribution of the LCL ($p = 0.003$) and capsule + PFL ($p = 0.001$) (Figure 3-5).

3.2.3 Flexion Torque – Internal Rotation

The internal rotation results examined here explore if the load distributions are influenced when the specimen is internally rotated to mimic the **Compensatory Alignment Injury** pattern evident in patients with external tibial torsion.

In the absence of varus torque, the contributions toward the total hyperextension restraint torque were significantly different between internal rotational increments ($p = 0.025$). The net restraint torque contribution of the PLC overall was greater at internal rotations of 5° than at 10° ($p = 0.006$) (Figure 3-6). Specifically, torque contributions of the LCL were significantly greater at internal rotations of 5° (14.0 ± 8.8 %) than at 10° (11.9 ± 9.7 %) ($p = 0.008$) (Figure 3-6). Compared to neutral, the mean contribution of the LCL was less at 5° IR (by 1.8 ± 1.2 %) and 10° IR (by 3.9 ± 2.1 %), however, these differences were not statistically significant ($p = 0.260$ and 0.056, respectively). The mean torque contribution of the popliteus tendon was significantly greater at internal rotations of 5° (5.2 ± 2.8 %) than at 10° (4.0 ± 2.4 %) ($p = 0.020$) (Figure 3-6). The contribution of the popliteus tendon at 5° IR was lower than neutral (by 0.9 ± 0.8 %), but this was not statistically significant ($p = 0.218$). Torque contributions of the popliteus tendon were significantly lower at 10° IR (4.0 ± 2.4 %) compared to neutral (6.0 ± 3.7%) ($p = 0.024$) (Figure 3-6). Contributions of the capsule + PFL were not statistically significant between 5° and 10° IR, nor were the torques at these IR increments different from when the joint was neutral ($p = 1.000$).

The LCL had a significantly larger contribution than the popliteus tendon after 5° IR ($p = 0.017$), and 10° IR ($p = 0.038$); similar to when the joint was neutral. At neutral alignment, the contribution of the capsule + PFL was not significantly larger than the popliteus tendon ($p = 0.265$) but became so as the knee was internally rotated by 5° IR ($p = 0.031$), and 10° IR ($p = 0.004$) (Figure 3-6).

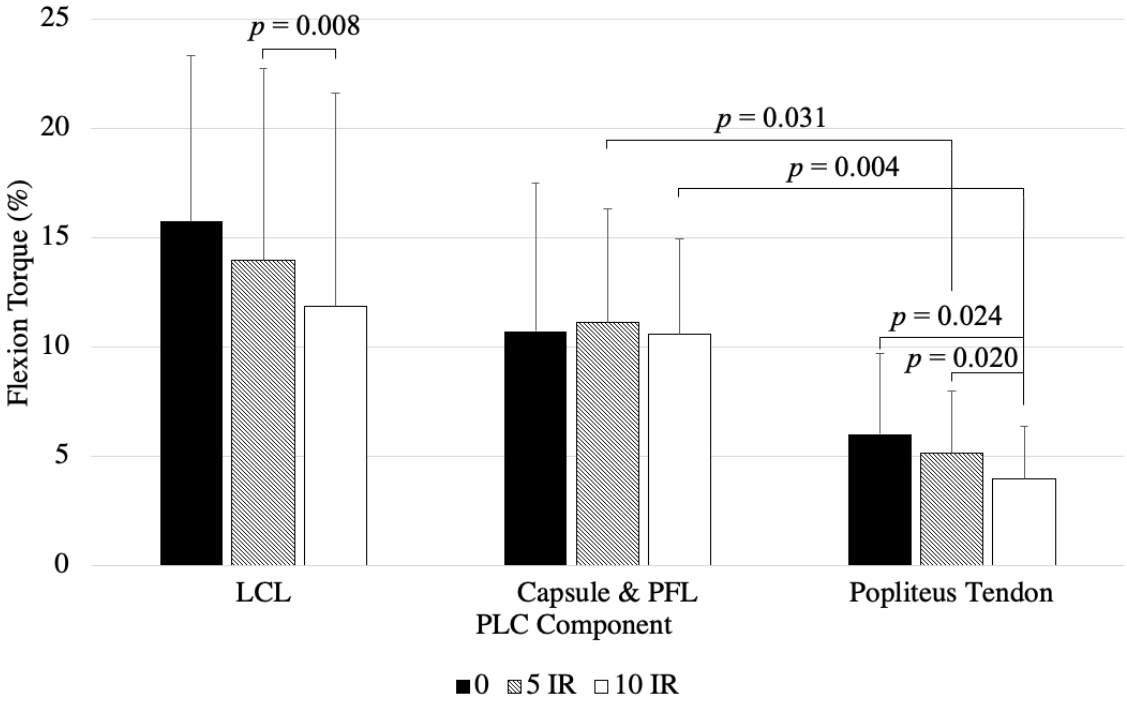


Figure 3-6: PLC restraint of 27 Nm of hyperextension during internal rotation, without a varus torque.

In tests that included a varus torque, the contributions toward the total hyperextension restraint torque were not significantly different between rotational increments ($p = 0.472$).

Contributions of the LCL were not statistically significant between 5° and 10° IR, or when these IR increments were compared to neutral ($p = 1.000$). Torque contributions of the capsule + PFL were not significantly different between neutral and internal rotations of 5° ($p = 1.000$) or 10° IR ($p = 0.708$). The popliteus tendon torque contributions were lower than neutral at 10° IR ($p = 0.885$), but these differences were only significant at 5° IR (by 0.6 ± 0.4 %, $p = 0.029$) (Figure 3-7).

The LCL had a significantly larger contribution than the popliteus tendon after internal rotations of 5° (by 8.0 ± 4.5 %, $p = 0.002$) and 10° IR (by 7.9 ± 7.5 %, $p = 0.030$), similar to when the joint was neutral (Figure 3-7). The capsule + PFL provided greater restraint to hyperextension when compared to the popliteus tendon at internal rotations of

5° (by 8.9 ± 3.2 %, p = 0.002) and 10° IR (by 7.4 ± 2.7 %, p = 0.003), also similar to when the joint was neutral (Figure 3-7).

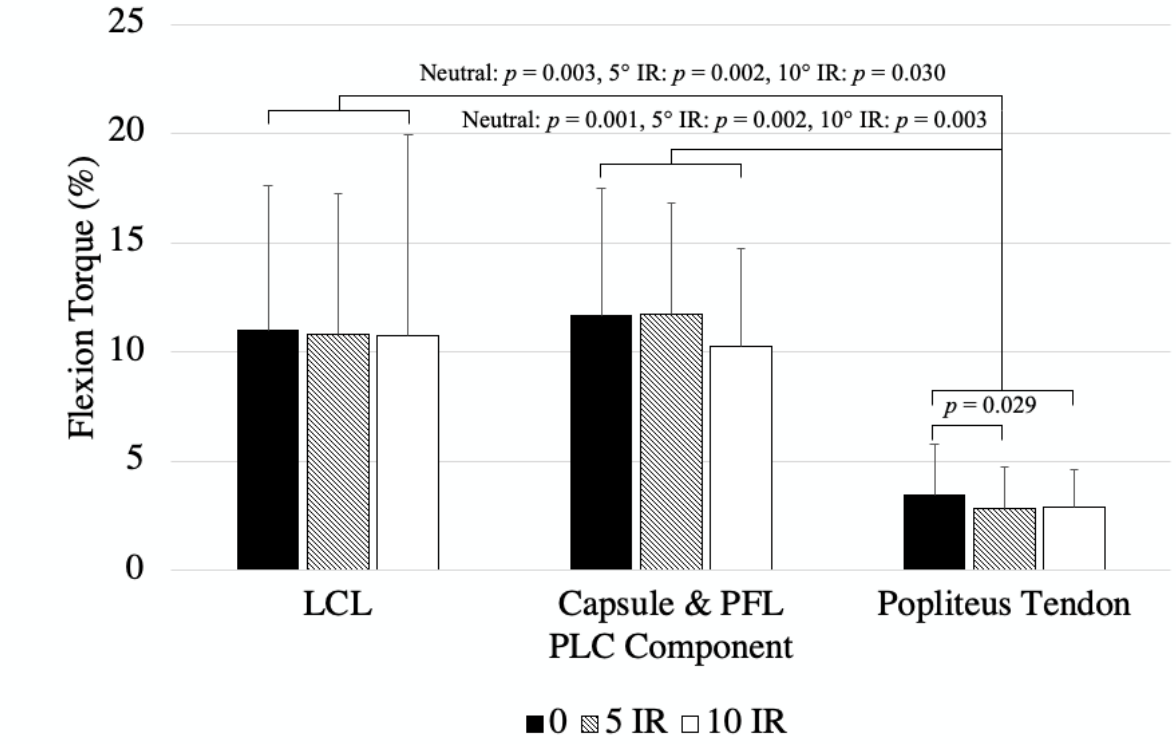


Figure 3-7: PLC restraint of 27 Nm of hyperextension, during internal rotation, with a varus torque.

Cutting sequence, in which some specimens (n = 6) had the capsule + PFL sectioned first, and some had the LCL sectioned first (n = 6), did not have a significant effect on the results obtained, either without (p = 0.822) or with (p = 0.101) varus torque.

3.2.4 Flexion Torque – External Rotation

The external rotation results presented here explore the **Non-compensatory Alignment Injury** pattern aiming to simulate excessive external tibial torsion. In the absence of varus torque, the contributions toward the total hyperextension restraint torque were not significantly different between external rotational increments (p = 0.271). Although not statistically significant, the mean torque contributions of the LCL decreased from neutral when externally rotated at 5° (by 0.8 ± 0.6 %, p = 1.000) and 10° (by 2.7 ± 1.5 %, p = 0.497). The mean torque contributions of the capsule + PFL increased from neutral

upon external rotation of 5° (by 0.3 ± 0.6 %, $p = 1.000$) and 10° ER (by 1.4 ± 0.6 %, $p = 0.430$), but neither increase was statistically significant. The popliteus tendon also did not show statistically significant results, despite the mean torque contributions decreasing from neutral when externally rotated at 5° (by 0.9 ± 0.8 %, $p = 0.557$) and 10° (by 1.9 ± 1.0 %, $p = 0.322$).

Like the neutral alignment behaviour, the LCL provided greater restraint to hyperextension when compared to the popliteus tendon at external rotations of 5° (by 9.8 ± 2.6 %, $p = 0.005$) and 10° IR (by 8.9 ± 3.6 %, $p = 0.031$) (Figure 3-8).

In tests that included a varus torque, the contributions toward the total hyperextension restraint torque were not significantly different between external rotational increments ($p = 0.385$). Although not statistically significant, unlike tests without varus torque, the mean torque contributions of the LCL increased from neutral when externally rotated by 5° (by 2.8 ± 3.1 %, $p = 0.889$) and 10° (by 2.9 ± 5.0 %, $p = 0.909$). The mean torque contributions of the capsule + PFL also increased from neutral when externally rotated by 5° (by 1.5 ± 1.7 %, $p = 0.818$) and 10° ER (by 0.7 ± 1.9 %, $p = 1.000$), although neither were statistically significant. The popliteus tendon also did not show statistically significant results, despite the mean torque contributions decreasing from neutral when externally rotated by 5° (by 0.3 ± 0.4 %, $p = 1.000$) and 10° (by 0.9 ± 1.3 %, $p = 1.000$).

Similar to the neutral alignment behaviour, the LCL provided greater contributions to hyperextension restraint when compared to the popliteus tendon at external rotations of 5° (by 10.7 ± 7.0 %, $p = 0.017$) and 10° (by 11.3 ± 8.0 %, $p = 0.034$) (Figure 3-9). Also like the neutral alignment behaviour, the capsule + PFL had significantly greater torque restraint contributions than the popliteus tendon at external rotations of 5° (by 10.1 ± 4.7 %, $p = 0.011$), and 10° (by 9.9 ± 4.1 %, $p = 0.011$) (Figure 3-9).

Cutting sequence, in which some specimens (n = 6) had the capsule + PFL sectioned first, and some had the LCL sectioned first (n = 6), did not have a significant effect on the results obtained, either without ($p = 0.157$) or with ($p = 0.452$) varus torque.

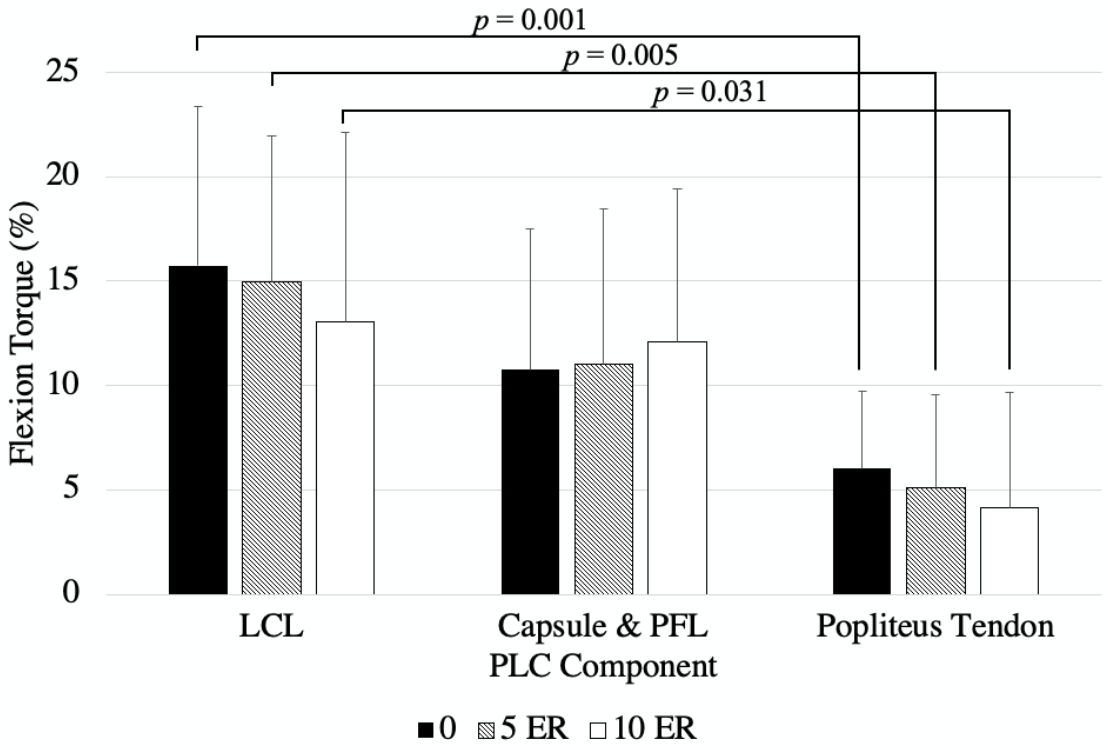


Figure 3-9: PLC restraint of 27 Nm of hyperextension during external rotation, without a varus torque.

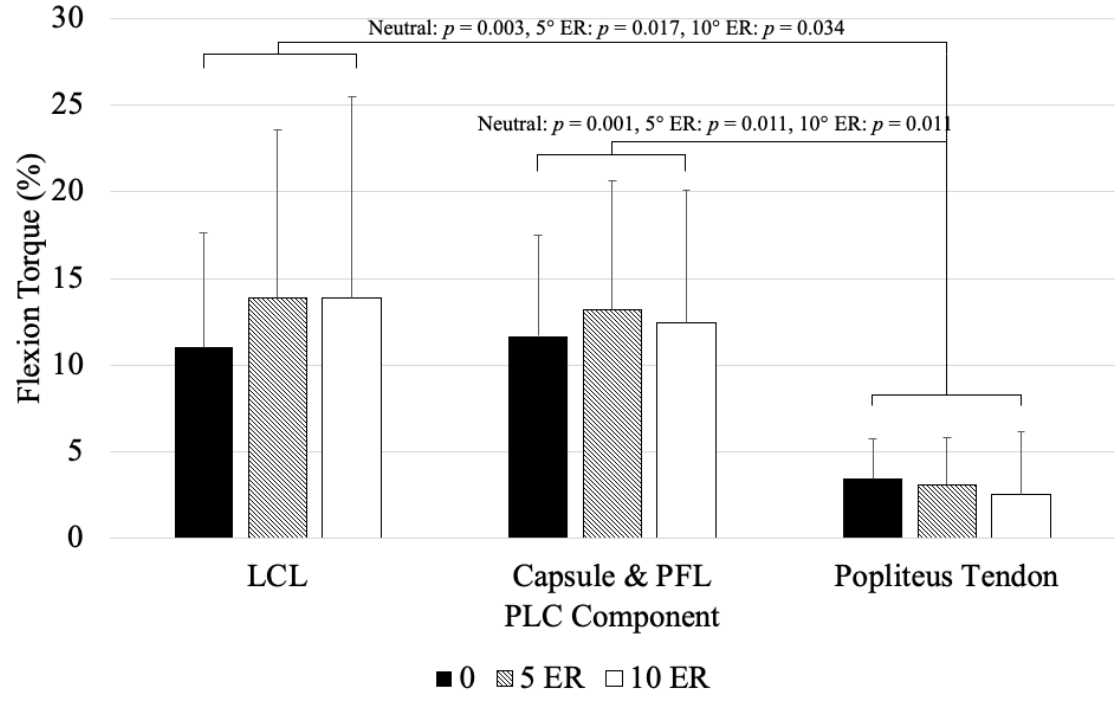


Figure 3-8: PLC restraint of 27 Nm of hyperextension during external rotation, with a varus torque.

3.3 Discussion

The main objective of this study was to compare the loads of PLC structures when the knee joint was subjected to hyperextension, a common injury mechanism. Specifically, the study aimed to observe how the applied loads are distributed throughout the soft tissues of the PLC when the transverse alignment of the tibia is offset due to a tibial deformity. The primary measure that we evaluated was flexion torque, as this would provide important information about PLC injuries when hyperextension forces occur. Data collected for varus/valgus rotation torque and internal/external rotation torque can be found in the appendix. We found minimal significant differences in contributions to valgus rotation torque restraint from the PLC components when subjected to hyperextension. Contributions towards external rotation torque restraint increased as the knee was externally rotated. This is consistent with findings of Nyland et al., who observed increased tibial external rotation after sequentially sectioning the PFL, LCL, and finally, the popliteus tendon. [110].

The concept for the **Compensatory Alignment Injury**, which suggests that an individual with external tibial torsion will compensate for the externally rotated feet resulting in an internal rotation at the knee is a concept that is validated in literature. For example, Alexander et al. discuss how compensatory hip internal rotation results in an inward-pointing knee during stance [96]. This compensatory mechanism is proven to reduce the capacity of the gluteal and soleus muscles because of the altered skeletal platform that the muscles act on [97]. If muscle capabilities are compromised, the resulting increased translations and rotations at the joint may place greater loads onto ligaments to maintain stability. This is evident in studies such as a 2018 publication from Bates et al., which demonstrated at a 4° offset of tibial internal rotation, that the peak ACL strain increased [17]. We therefore hypothesized that the inward rotation of the proximal tibia would decrease the leverage of the PLC (Figure 1-5). With reduced leverage, the PLC may require greater tension to counteract the forces of hyperextension, therefore straining the components and predisposing them to injury. Therefore, individuals with excessive external tibial torsion may be more susceptible to PLC injuries due to the altered biomechanics and decreased leverage of the posterolateral corner.

Alternatively, the **Non-compensatory Alignment Injury** investigated in this study provides results for a knee that has been exposed to hyperextension forces while rotated externally. These results are presented to support an alternative theory where the knee is not in the internally rotated compensatory position. If instead the knee is in a "normal" rotational position in a patient with an external torsional deformity, the foot is angled outward. This outward foot angle is theorized to cause some of the force to be directed in a way that induces an external rotation torque on the knee, causing a relative external rotation of the proximal tibia, therefore placing additional stress on the PLC.

This discussion explores how loads during hyperextension are distributed amongst the components of the PLC complex, both with and without varus torque, and if the loading distribution is affected by the presence of excessive external tibial torsion. This is accomplished through evaluation of the results of both injury mechanisms.

Peak Hyperextension

At 27 Nm of hyperextension torque, peak hyperextension averaged 15.3°$\pm$ 7.7° without varus torque, and 15.3°$\pm$ 8.0° with varus torque. This agreed with peak hyperextension in the Noyes et al. study, which was 14.3° $\pm$ 5.2°. Noyes et al. did not report if an additional varus torque was applied. Cadaveric research can be variable, yet the difference between peak hyperextension angles in both studies was only 1°.

Flexion Torque – Neutral Alignment

These results highlight the importance of the LCL and capsule + PFL in restraining hyperextension. In tests with varus torque, the LCL and the capsule + PFL had nearly identical contributions to restraining hyperextension, and in tests with varus torque, these structures were not contributing significantly different amounts. Therefore, we can assume that they both restrain hyperextension in similar proportions. We also found that the popliteus tendon has the smallest contribution to restraining hyperextension, of the components we explored. The popliteus tendon, functioning in part as a dynamic stabilizer, may have its functional contributions underestimated because it was not tensioned during evaluation, as it would be during normal activity.

Noyes et al. investigated cruciate ligaments and posterior knee structures resisting hyperextension [3]. They found that the posterolateral capsule primarily restrained hyperextension (18.0 ± 11.4 %), followed by the LCL (4.1 ± 3.4 %), and then the popliteus tendon (0.9 ± 1.0 %). Noyes et al. sectioned the capsule prior to the LCL. Both studies found that the popliteus tendon provided the least restraint to hyperextension, but Noyes et al. demonstrated that the posterolateral capsule had much greater contributions to hyperextension restraint compared to the LCL. Our study found that the LCL provided equal, or even greater restraint to hyperextension than the capsule + PFL when no varus torque is applied. Authors of the Noyes et al. study do not suggest a varus torque was applied.

The difference between studies may be attributed to the numerous structures sectioned by Noyes et al., such as the ACL and PCL, which were sectioned prior to the LCL and popliteus tendon. Sectioning of the cruciate ligaments, which function as major stabilizers of the knee, changes the loading distribution of the knee. We also did not evaluate the MCL, which consists of superficial (SMCL) and deep (DMCL) components. This complex played a fair role in restraining hyperextension in the Noyes et al. study (9.3 ± 7.3 %). Noyes et al. listed percentages of restraint that add up to 100%, under the assumption that all the torque resisting hyperextension were distributed among the tested structures. We cut only specific structures and reported the percentage of total torque each contributed to hyperextension restraint. This strategy acknowledges that other structures may contribute to resisting hyperextension. As we see in the Noyes et al. study, once these structures are sectioned, the LCL is less effective.

Furthermore, the capability of the capsule to restrain hyperextension was coupled with the fabellofibular ligament (FFL), which was not examined in this study, and was left intact. This ligament helps restrain hyperextension, evident in a publication that reported a 10% increase in knee hyperextension when it was sectioned after the oblique popliteal ligament and before the ACL/PCL [44]. The role of the posterolateral capsule may have been overestimated by the inclusion/contribution of the FFL.

Our results coincide with that of Gollehon et al., that tested 17 specimens with external rotation and varus torque [49]. Gollehon et al. found that the LCL and deep structures (including the posterolateral capsule + PFL) function together to stabilize the knee. Although these researchers did not explore a hyperextension injury mechanism, the conclusions that the LCL and capsule + PFL play significant roles in stabilizing the knee under varus torque conditions, and that the popliteus tendon plays a lesser role, validate the results of this study and emphasizes the critical role of the LCL and capsule + PFL.

Morgan et al. found that the oblique popliteal ligament was the primary ligamentous restraint to knee hyperextension [44]. These authors applied 27 Nm of hyperextension torque, as well, but did not separate their PLC structures, and rather grouped them as a complex to compare to the oblique popliteal ligament, the FFL, the ACL and PCL. Although the oblique popliteal ligament displayed the highest amount of hyperextension restraint, the result was not statistically significant when compared to the restraint provided by the posterolateral corner, suggesting that the PLC is largely important in hyperextension restraint.

Flexion Torque – Internal Rotation

Contributions toward the total hyperextension restraint torque were significantly different between internal rotational increments, with net restraint torque contributions of the PLC greater at internal rotations of 5° than at 10°. This suggests that as the knee is progressively internally rotated, LCL and popliteus tendon contributions to restrain hyperextension decrease. Notably, the hyperextension restraint contributed by the popliteus tendon decreased at 10° IR compared to neutral. This highlights the popliteus tendon's sensitivity to internal rotation. This finding is consistent with literature that indicates that the popliteus tendon tightens when the tibia is rotated externally [46]. In a study from Baker et al., they evaluated posterolateral injuries in 17 cadaveric knees, finding that the popliteus muscle was injured when knee hyperextension occurred with external rotation in 6 of them [47]. Therefore, when the knee is rotated internally, there is less hyperextension restraint provided by the popliteus tendon.

The contributions from the capsule + PFL were not significantly affected by internal rotations when compared to neutral, suggesting the role of these structures remains stable regardless of these rotations. At neutral alignment, the contribution of the capsule + PFL was not significantly larger than the popliteus tendon but became statistically significant as the knee was progressively internally rotated. This likely occurred because the popliteus tendon decreased in contributions to restraining hyperextension with increased internal rotation, while the capsule + PFL maintained restraint contributions.

Our primary finding in these tests of pure hyperextension, is that as the tibia is rotated internally, the net restraint torque provided by the LCL and popliteus tendon decreases. The capsule + PFL did not show any statistically significant effects at various rotational increments, which means that the concern for capsule + PFL during a hyperextension injury mechanism remains unchanged regardless of excessive external tibial torsion.

In tests that included a varus torque, the contributions toward the total hyperextension restraint torque were not significantly different between rotational increments. The torque contributions from the popliteus tendon were significantly lower than neutral at 5° IR. This finding underscores the sensitivity of the popliteus tendon to internal rotation, aligning with current literature that suggests that the popliteus tendon tightens when the tibia is rotated externally [46]. However, tests that included varus torque provided fewer significant findings, despite being more representative of a typical PLC injury mechanism.

Flexion Torque – External Rotation

In tests of pure hyperextension, the contributions of components of the PLC were not significantly altered by external rotations when compared to neutral alignment. Similar to neutral alignment results, at all external rotations the LCL provided significantly greater restraint to hyperextension compared to the popliteus tendon.

In tests that included varus torque, the contributions toward the total hyperextension restraint torque also did not significantly differ between external rotational increments. Like tests without varus torque, these tests continued to demonstrate that PLC

components were not significantly altered by external rotations when contributions of hyperextension torque restraint were compared to neutral alignment. The only difference when varus torque was applied, was that compared to the popliteus tendon, the capsule + PFL provided greater torque restraint contributions at external rotations. These results are consistent with literature that finds the sectioning of the PFL produced no significant changes in the limits of the knee movements studied, including external rotation, varus rotation, and posterior tibial translation [109]. Pasque et al., after selectively sectioning PLC components in cadaveric knees, found that external rotation was only increased when multiple ligaments were deficient, including the popliteus tendon and PFL, concluding that the posterolateral capsule, LCL, and popliteus tendon function as a unit [109].

Conclusion

In conclusion, both internal and external rotations reduced hyperextension range of motion. Significant results were obtained during internal rotations for the **Compensatory Alignment Injury**, whereas fewer significant findings were demonstrated during external rotations that represent the **Non-compensatory Alignment Injury**.

Researchers at the Kliniek ViaSana noted that patients with PLC injuries often exhibited excessive external tibial torsion. This suggested that the rotational deformity might contribute to the injury mechanism. This study's results support the theory that patients with excessive external tibial torsion are more likely to experience PLC injuries. While we are uncertain how a patient with this tibial deformity may present – whether exhibiting the **Compensatory** or **Non-compensatory Alignment Injury** – the study revealed significant differences in torque restraint contributions from PLC components when rotations were applied. Therefore, excessive external tibial torsion should not be dismissed as a potential contributor to greater risk of PLC injury.

In terms of the **Compensatory Alignment Injury** results, although the PLC ligament contributions to hyperextension restraint did not increase when internally rotated, internal rotation of the proximal tibia caused changes in loading of PLC components during hyperextension. The internally rotated position of the proximal tibia alters the alignment

and mechanical advantage of the PLC structures, potentially subjecting them to higher tensile stresses. From a clinical perspective, abnormal loading can contribute to the degeneration of the medial meniscus and articular cartilage, potentially accelerating the development of osteoarthritis. Over time, these repetitive abnormal loads and inability to effectively resist hyperextension due to decreased contributions from the PLC can lead to microtrauma and eventual macroscopic injury of PLC components and associated structures like the ACL or PCL.

Our findings suggest that a tibial malalignment affects the loads and their distribution amongst PLC components during knee hyperextension. These findings underscore the importance of PLC structures in knee stability. The interactions of the LCL, posterolateral capsule + PFL, and the popliteus tendon are important to recognize and understand to better diagnose and treat knee injuries. Understanding the specific contributions of each PLC component can lead to tailored and effective reconstruction techniques. These insights can provide clinicians with information to recognize PLC injuries efficiently, as well as inform preventive strategies for athletes and individuals at high risk for PLC injuries, particularly those with known torsional deformities.

Chapter 4

4 General Summary and Future Works

This chapter reviews the objectives of the study described in Chapter 3, summarizing the findings and conclusions to provide a brief overview of this work. This chapter also considers the clinical significance of the findings, discusses the strengths and limitations of the study, and explores potential future directions for research on the PLC and transverse plane malalignment during hyperextension injuries.

4.1 Summary

The posterolateral corner of the knee joint is a complex collection of ligaments and soft tissues, characterized by its intricate structure and interacting components. Existing literature exploring the PLC and the role of its components in stabilizing the knee is both limited, and in some cases, contradictory. The conflicts found in literature primarily regard the best surgical techniques to treat a PLC injury. Some studies suggest that best treatment outcomes are seen when injuries are treated through anatomic reconstruction, citing lower failure rates and better outcomes. Stannard et al. reported a 9% failure rate in the reconstruction cohort and a 37% failure rate in the repair cohort [37]. Others suggest that repairs or non-anatomic reconstructions could be equally as effective. In a review from LaPrade et al., albeit from 2002, they reported that patients with repairs conducted less than 3 weeks following injury have the best functional outcomes [54].

Before treatment options like reconstructions and repairs can even be considered, the PLC injury must first be recognized and properly diagnosed. However, contradictions also exist within studies that investigate PLC loading, particularly regarding which components primarily restrain specific motions and to what extent. There is further ambiguity when PLC components are explored in the context of hyperextension injuries, as most biomechanical testing is conducted between 0° and 90° of flexion. Noyes et al. found that the posterior capsular structures restrained 54.7% of total resisting moment, and that the LCL provided lower amounts of resistance [3]. In a study that evaluated torn PLC structures following hyperextension by gross dissection, researchers found that the

popliteus tendon was damaged in most cases, the LCL was damaged in some, and the PFL was damaged in none [42]. Furthermore, the inconsistent terminology used to describe the PLC complex exacerbates this knowledge gap.

The anatomical proximity of the PLC to critical knee components such as the anterior cruciate ligament (ACL) further complicates its diagnosis and treatment. This interconnected nature also means that when the PLC is damaged, other ligaments and soft tissues of the knee are also likely damaged. LaPrade et al. found that 87% of PLC injuries occurred with other ligaments, like the ACL [26]. As a result, the rare incidence of PLC injuries may be attributed to underreporting, as physicians may overlook these injuries, prioritizing damage to a primary knee ligament like the ACL. It also may be due to a perceived lack of importance of this complex, and lack of understanding of its primary injury mechanisms. When overlooked, PLC injuries can cause complications, like chronic pain, early onset of osteoarthritis, and increased risk of cruciate ligament graft failure. Therefore, researching the PLC is incredibly important to prevent these long-term complications.

Given that the most common mechanism of injury to the PLC is hyperextension, our objective was to determine the PLC components that primarily restrain this movement [25]. By identifying which components primarily restrain this movement, we can identify the most susceptible components to injury, and contribute to results reported in studies like those by Noyes, Morgan, and Fornalski et al.

In this study, we add to current literature by evaluating PLC structures under hyperextension stress while the tibia is externally and internally rotated, to replicate a PLC injury scenario in individuals with transverse plane malalignment. Individuals with tibial rotational offsets may be more vulnerable to PLC injuries due to altered knee biomechanics, causing certain variations in the structures that resist hyperextension. If tibial malalignment is proven to be an underlying factor that predisposes certain PLC components to increased loads, physicians can be better prepared to diagnose injuries in this population and customize treatment plans tailored to address the unique biomechanics evident in these individuals.

Our findings in Chapter 3 revealed that the LCL and the capsule + PFL contribute the most to resisting hyperextension, and the popliteus tendon contributes the least. Rotational increments significantly affected the contribution provided by the PLC structures to torque restraint, especially between 5° and 10° of internal rotation. Therefore, in addressing our objective, we can conclude that the capacity of these structures to resist flexion torque depends on the LCL and the capsule + PFL, and these structures require more concern in the presence of a malalignment. Significant differences were not evident between the LCL and the capsule + PFL, suggesting that the capsule + PFL and the LCL contribute proportional restraint to hyperextension. Our results agree with those from Noyes et al., that found the popliteus tendon to have the least contribution to restraining hyperextension [3]. Our results do not coincide with those of Fornalski et al., that found the popliteus tendon to have ruptured most often after hyperextension injury, yet this could be attributed to the tensile strength of the ligament as opposed to the actual loads that are restrained by this component [42]. Nonetheless, our results suggest that tibial malalignment does affect the load distribution among PLC components.

This study underscores the importance of continued research into the PLC to establish and characterize the roles of each stabilizing component. Establishing the roles of the LCL, the capsule + PFL, and the popliteus tendon for resisting various movements during hyperextension injuries can provide information clinicians need to diagnose damage. Diagnosing PLC injuries is time sensitive. A delayed diagnosis can lead to complications and different procedures must be used to treat the damage in that case. Therefore, establishing an injury pattern to recognize in those with transverse plane malalignment could provide the means for more effective diagnosis. This research also emphasizes the importance of monitoring foot positioning in high-risk individuals like those with known torsional deformities. While our findings suggest that rotating the knee in either direction did not significantly impact PLC loading, observation of foot alignment during these incidents, potentially through video, could aid in identifying landing patterns that place specific PLC structures at risk. Given the preliminary nature of this study, further research should be conducted before making concrete recommendations. Regardless, the insights provided by this study still contribute to informing clinicians. This research lays

the foundation for future work, ultimately leading to the goal of enhancing patient outcomes and reducing the incidence of long-term complications.

4.2 Limitations and Strengths

Our is study is limited by the inclusion of only male cadaveric specimens. We procured our specimens from United Tissue Network. We were provided with summaries of 22 potential cadaveric specimens from a total of 13 donors (including 9 paired knees and 4 single knees). We evaluated the donors' information to exclude specimens that may yield inaccurate results due to cancer in the bones, osteoporosis, arthritis, or previous knee surgeries. We also prioritized specimens with healthy BMIs and preferably younger age. In this process, the 12 most ideal specimens according to our criteria resulted in the selection of only male specimens. The exclusion of female cadaveric specimens limits the generalizability of our results, given that our results are primarily applicable to the male population. There are sex differences between male and female knee biomechanics [55]. Literature has suggested that tibial deformities are more striking among women, as they are accentuated by high heels [100]. These differences can affect the results of loading of the PLC components; therefore, our results must be interpreted with caution when applied to females. We also could not procure perfect specimens; two, UTN2323019 (left and right) and UTN2323018 (right) had mild arthritis, and UTN2322907 (right) had a BMI of 15.8, which is lower than ideal.

Furthermore, our sample size could be improved. We used G*Power (Heinrich Heine University, Dusseldorf, Germany) to determine our sample size. It reported that 12 specimens should detect large effects with a power of 0.8. A greater sample size could have detected small effects. Although we did find many statistically significant differences, including an additional specimen in each sectioning cohort may provide more precise and definitive results.

A notable limitation of this study is the omission of loading rate considerations in the experimental design. Due to the viscoelastic properties exhibited by ligaments, their mechanical behaviour is dependent on the rate at which they are loaded. We wanted to explore a rapid hyperextension event, such as landing from a jump, therefore, it would

have been more accurate to measure load contributions of these ligaments and soft tissues under physiological loading rates. In our experiments, we did not replicate these physiological loading rates, and consequently, our results may not fully represent the behaviour of PLC components under realistic conditions. Even though the loading rates used in our study were not entirely reflective of real-world scenarios, the relative differences in ligament contributions observed under these controlled conditions remain relevant and applicable to understanding PLC function in cases of rotational deformities.

Another limitation is that we were under the impression that the popliteus tendon had to be the final structure sectioned, however, in some literature the popliteus tendon was sectioned following the LCL [56]. We were unable to analyze how varying the cutting order in this way could affect the results.

Another limitation is attributed to errors made during the potting procedure. The cement was unable to cure properly on some specimens. Reasons for this include cutting the tibia too short, leaving less bone for the cement to adhere to, and not adequately drying the tibial canal (resulting in fluid leaking into the tibial pot). Consequently, the specimens were unable to be securely fixed in the pot. During hyperextension tests, they reached range of motion limits of the joint motion simulator because 27 Nm of hyperextension torque could not be reached. The specimens were then refrigerated overnight and repotted the following day, which may have resulted in variation in the mechanical properties of the soft tissues. Regardless, the contributions of each PLC component were evaluated relative to one another, and absolute values were of less importance.

Furthermore, the complete extent of a tibial torsional deformity could not be accurately replicated using the cadaveric specimens available to us. Excessive external tibial torsion can reach up to 30° greater than normal [97]. We used normal specimens but applied rotations to simulate the tibial deformity. A normal cadaveric specimen may rupture or tear if rotated too excessively. These specimens were not true tibial torsional deformities, but this testing procedure allowed us to have a level of control over the amount of tibial deformity.

A strength of this work is its novelty. There have yet to be publications exploring transverse plane malalignment in the context of PLC hyperextension injuries. These results will hopefully direct clinician attention to the PLC upon knee injury analysis and spark more exploration of PLC injuries in the context of tibial torsion. This research also highlights the contradictions in literature regarding the function of PLC components.

Another strength of this study is its exploration of other variables and directions than flexion torque, like external rotation torque and valgus rotation torque, along with external rotation angles.

Furthermore, exploring these variables in the context of pure hyperextension and with the addition of varus torque helps to more realistically replicate hyperextension injuries. This contributes to the increased validity of our findings.

4.3 Future Work

Future exploration of the PLC in cases of hyperextension with transverse plane malalignment should include other components of the PLC. We did not examine all the components of the PLC. We examined only the three primary stabilizing components; the PFL, the LCL, and the popliteus tendon. Other components, such as the fabellofibular ligament, discussed briefly in section 3.3, or the oblique popliteal ligament, may provide stabilizing functions. The oblique popliteal ligament, which Morgan et al. identified as the primary restraint to knee hyperextension, was not analyzed in this study [44]. Including other PLC components could provide a more comprehensive understanding of the PLC and the functional roles of its components in knee stability when torsional deformities are present.

Future studies could more closely simulate a PLC injury by also transecting cruciate ligaments such as the ACL or PCL, given that the PLC is commonly damaged alongside these ligaments during injury.

The hip joint is known to compensate for external tibial torsion by retro-torsion, therefore future studies could explore this compensatory mechanism as an indicator of tibial torsion, as opposed to at the level of the knee.

Finally, future work could include the building or validating of computer models using the results of this experiment, to explore a wider variety of scenarios than can be performed experimentally. Previous studies have shown that biomechanical evaluation of PLC structures is possible through computational models [58, 59].

4.4 Significance

To our knowledge, this study was the first to investigate if transverse plane malalignment may be an underlying cause of PLC damage during hyperextension. Our research identified a gap in the current literature and highlighted the lack of consistency in results regarding PLC hyperextension injuries. This research enhances the current understanding of PLC injuries and is important for its clinical implications. This research highlights the structures most vulnerable to damage in hyperextension injuries, providing clinicians with specific soft tissues to focus on during a knee injury assessment, in order to more efficiently diagnose PLC injury. For instance, the clinician may be inclined to assume that the LCL was damaged during a hyperextension injury and can proceed with their evaluation with that in mind. Furthermore, this research can be used by clinicians to educate patients about tibial torsion and their risk of knee injuries. If tibial torsion is suspected, patients should be mindful of partaking in activities that may lead to hyperextension injuries, and aim to maintain strong ligaments and muscles in the PLC as well as correct gait to reduce abnormal stress on the PLC. In summary, our findings aid in advancing the understanding of PLC injuries by validating and challenging existing literature and presenting practical implications for clinicians. The results of our study can be utilized to achieve more efficient and accurate diagnoses, reducing the likelihood of neglecting PLC injuries. By incorporating our research into the scientific community, healthcare providers can improve the management and outcomes of PLC injuries by selecting the most effective treatment option best suited to the patient, with careful consideration of tibial torsional deformities.

References

[1] Grood, E. S., & Suntay, W. J. (1983). A joint coordinate system for the clinical description of three-dimensional motions: application to the knee. *Journal of biomechanical engineering*, *105*(2), 136–144. https://doi.org/10.1115/1.3138397

[2] Morgan, P. M., LaPrade, R. F., Wentorf, F. A., Cook, J. W., & Bianco, A. (2010). The role of the oblique popliteal ligament and other structures in preventing knee hyperextension. *The American journal of sports medicine*, *38*(3), 550–557. https://doi.org/10.1177/0363546509318742

[3] Noyes, F. R., Clark, O., Nolan, J., & Johnson, D. J. (2023). Functional Interaction of the Cruciate Ligaments, Posteromedial and Posterolateral Capsule, Oblique Popliteal Ligament, and Other Structures in Preventing Abnormal Knee Hyperextension. *The American journal of sports medicine*, *51*(5), 1146–1154. https://doi.org/10.1177/03635465231155203

[4] Van, H. N., & Manh, K. N. (2023). Concomitantly combined anterior cruciate ligament and posterolateral corner reconstruction: A case report. *International Journal of Surgery Open*, *61*, 100706. https://doi.org/10.1016/j.ijso.2023.100706

[5] Crespo, B., James, E. W., Metsavaht, L., & LaPrade, R. F. (2014). Injuries to posterolateral corner of the knee: a comprehensive review from anatomy to surgical treatment. *Revista brasileira de ortopedia*, *50*(4), 363–370. https://doi.org/10.1016/j.rboe.2014.12.008

[6] Welsh, P., DeGraauw, C., & Whitty, D. (2016). Delayed diagnosis of an isolated posterolateral corner injury: a case report. *The Journal of the Canadian Chiropractic Association*, *60*(4), 299–304.

[7] LaPrade, R. F., Ly, T. V., Wentorf, F. A., & Engebretsen, L. (2003). The posterolateral attachments of the knee: a qualitative and quantitative morphologic analysis of the fibular collateral ligament, popliteus tendon, popliteofibular ligament, and lateral gastrocnemius tendon. *The American journal of sports medicine*, *31*(6), 854–860. https://doi.org/10.1177/03635465030310062101

[8] Bates, N. A., Nesbitt, R. J., Shearn, J. T., Myer, G. D., & Hewett, T. E. (2018). The influence of internal and external tibial rotation offsets on knee joint and ligament biomechanics during simulated athletic tasks. *Clinical biomechanics (Bristol, Avon)*, *52*, 109–116. https://doi.org/10.1016/j.clinbiomech.2018.01.019

[9] Figueroa, F., Figueroa, D., Putnis, S., Guiloff, R., Caro, P., & Espregueira-Mendes, J. (2021). Posterolateral corner knee injuries: a narrative review. *EFORT open reviews*, *6*(8), 676–685. https://doi.org/10.1302/2058-5241.6.200096

[10] Vaienti, E., Scita, G., Ceccarelli, F., & Pogliacomi, F. (2017). Understanding the human knee and its relationship to total knee replacement. *Acta bio-medica : Atenei Parmensis*, *88*(2S), 6–16. https://doi.org/10.23750/abm.v88i2-S.6507

[11] Waligora, A. C., Johanson, N. A., & Hirsch, B. E. (2009). Clinical anatomy of the quadriceps femoris and extensor apparatus of the knee. *Clinical orthopaedics and related research*, *467*(12), 3297–3306. https://doi.org/10.1007/s11999-009-1052-y

[12] Afonso, J., Rocha-Rodrigues, S., Clemente, F. M., Aquino, M., Nikolaidis, P. T., Sarmento, H., Fílter, A., Olivares-Jabalera, J., & Ramirez-Campillo, R. (2021). The Hamstrings: Anatomic and Physiologic Variations and Their Potential Relationships With Injury Risk. *Frontiers in physiology*, *12*, 694604. https://doi.org/10.3389/fphys.2021.694604

[13] Frank C. B. (2004). Ligament structure, physiology and function. *Journal of musculoskeletal & neuronal interactions*, *4*(2), 199–201.

[14] Harner, C. D., Höher, J., Vogrin, T. M., Carlin, G. J., & Woo, S. L. (1998). The effects of a popliteus muscle load on in situ forces in the posterior cruciate ligament and on knee kinematics. A human cadaveric study. *The American journal of sports medicine*, *26*(5), 669–673. https://doi.org/10.1177/03635465980260051201

[15] Hughston, J. C., Andrews, J. R., Cross, M. J., & Moschi, A. (1976). Classification of knee ligament instabilities. Part II. The lateral compartment. *The Journal of bone and joint surgery. American volume*, *58*(2), 173–179.

[16] Covey D. C. (2001). Injuries of the posterolateral corner of the knee. *The Journal of bone and joint surgery. American volume*, *83*(1), 106–118. https://doi.org/10.2106/00004623-200101000-00015

[17] Shon, O. J., Park, J. W., & Kim, B. J. (2017). Current Concepts of Posterolateral Corner Injuries of the Knee. *Knee surgery & related research*, *29*(4), 256–268. https://doi.org/10.5792/ksrr.16.029

[18] Yaras, R. J., O'Neill, N., Mabrouk, A., & Yaish, A. M. (2024). Lateral Collateral Ligament Knee Injury. In *StatPearls*. StatPearls Publishing.

[19] Pękala, P. A., Mann, M. R., Pękala, J. R., Tomaszewski, K. A., & LaPrade, R. F. (2021). Evidence-Based Clinical Anatomy of the Popliteofibular Ligament and Its Importance in Orthopaedic Surgery: Cadaveric Versus Magnetic Resonance Imaging Meta-analysis and Radiological Study. *The American journal of sports medicine*, *49*(6), 1659–1668. https://doi.org/10.1177/0363546520950415

[20] Watanabe, Y., Moriya, H., Takahashi, K., Yamagata, M., Sonoda, M., Shimada, Y., & Tamaki, T. (1993). Functional anatomy of the posterolateral structures of the knee. *Arthroscopy : the journal of arthroscopic & related surgery : official publication of the Arthroscopy Association of North America and the International Arthroscopy Association*, *9*(1), 57–62. https://doi.org/10.1016/s0749-8063(05)80344-5

[21] Shahane, S. A., Ibbotson, C., Strachan, R., & Bickerstaff, D. R. (1999). The popliteofibular ligament. An anatomical study of the posterolateral corner of the knee. *The Journal of bone and joint surgery. British volume*, *81*(4), 636–642. https://doi.org/10.1302/0301-620x.81b4.9501

[22] Chahla, J., Moatshe, G., Dean, C. S., & LaPrade, R. F. (2016). Posterolateral Corner of the Knee: Current Concepts. *The archives of bone and joint surgery*, *4*(2), 97–103.

[23] Parsons, E. M., Gee, A. O., Spiekerman, C., & Cavanagh, P. R. (2015). The Biomechanical Function of the Anterolateral Ligament of the Knee:

Response. *The American journal of sports medicine*, *43*(8), NP22. https://doi.org/10.1177/0363546515597218

[24] Lunden, J. B., Bzdusek, P. J., Monson, J. K., Malcomson, K. W., & Laprade, R. F. (2010). Current concepts in the recognition and treatment of posterolateral corner injuries of the knee. *The Journal of orthopaedic and sports physical therapy*, *40*(8), 502–516. https://doi.org/10.2519/jospt.2010.3269

[25] LaPrade, R. F., & Terry, G. C. (1997). Injuries to the posterolateral aspect of the knee. Association of anatomic injury patterns with clinical instability. *The American journal of sports medicine*, *25*(4), 433–438. https://doi.org/10.1177/036354659702500403

[26] DeLee, J. C., Riley, M. B., & Rockwood, C. A., Jr (1983). Acute posterolateral rotatory instability of the knee. *The American journal of sports medicine*, *11*(4), 199–207. https://doi.org/10.1177/036354658301100403

[27] LaPrade, R. F., Wentorf, F. A., Fritts, H., Gundry, C., & Hightower, C. D. (2007). A prospective magnetic resonance imaging study of the incidence of posterolateral and multiple ligament injuries in acute knee injuries presenting with a hemarthrosis. *Arthroscopy : the journal of arthroscopic & related surgery : official publication of the Arthroscopy Association of North America and the International Arthroscopy Association*, *23*(12), 1341–1347. https://doi.org/10.1016/j.arthro.2007.07.024

[28] Geeslin, A. G., & LaPrade, R. F. (2010). Location of bone bruises and other osseous injuries associated with acute grade III isolated and combined posterolateral knee injuries. *The American journal of sports medicine*, *38*(12), 2502–2508. https://doi.org/10.1177/0363546510376232

[29] Fanelli, G. C., Verch, D, & Malek, M.M. (1993). Posterior Cruciate Ligament Injuries in Trauma Patients. *Journal of Orthopaedic Trauma*, *7*(2), 170.

[30] Pacheco, R. J., Ayre, C. A., & Bollen, S. R. (2011). Posterolateral corner injuries of the knee: a serious injury commonly missed. *The Journal of bone and joint*

surgery. British volume, *93*(2), 194–197. https://doi.org/10.1302/0301-620X.93B2.25774

[31] Grimm, N. L., Levy, B. J., Jimenez, A. E., Bell, R., & Arciero, R. A. (2020). Open Anatomic Reconstruction of the Posterolateral Corner: The Arciero Technique. *Arthroscopy techniques*, *9*(9), e1409–e1414. https://doi.org/10.1016/j.eats.2020.05.022

[32] LaPrade, R. F., Resig, S., Wentorf, F., & Lewis, J. L. (1999). The effects of grade III posterolateral knee complex injuries on anterior cruciate ligament graft force. A biomechanical analysis. *The American journal of sports medicine*, *27*(4), 469–475. https://doi.org/10.1177/03635465990270041101

[33] Seebacher, J. R., Inglis, A. E., Marshall, J. L., & Warren, R. F. (1982). The structure of the posterolateral aspect of the knee. *The Journal of bone and joint surgery. American volume*, *64*(4), 536–541.

[34] Chahla, J., Murray, I. R., Robinson, J., Lagae, K., Margheritini, F., Fritsch, B., Leyes, M., Barenius, B., Pujol, N., Engebretsen, L., Lind, M., Cohen, M., Maestu, R., Getgood, A., Ferrer, G., Villascusa, S., Uchida, S., Levy, B. A., Von Bormann, R., Brown, C., … Gelber, P. E. (2019). Posterolateral corner of the knee: an expert consensus statement on diagnosis, classification, treatment, and rehabilitation. *Knee surgery, sports traumatology, arthroscopy : official journal of the ESSKA*, *27*(8), 2520–2529. https://doi.org/10.1007/s00167-018-5260-4

[35] Fanelli, G. C., & Larson, R. V. (2002). Practical management of posterolateral instability of the knee. *Arthroscopy : the journal of arthroscopic & related surgery : official publication of the Arthroscopy Association of North America and the International Arthroscopy Association*, *18*(2 Suppl 1), 1–8. https://doi.org/10.1053/jars.2002.31779

[36] Kannus P. (1989). Nonoperative treatment of grade II and III sprains of the lateral ligament compartment of the knee. *The American journal of sports medicine*, *17*(1), 83–88. https://doi.org/10.1177/036354658901700114

[37] Stannard, J. P., Brown, S. L., Farris, R. C., McGwin, G., Jr, & Volgas, D. A. (2005). The posterolateral corner of the knee: repair versus reconstruction. *The American journal of sports medicine*, *33*(6), 881–888. https://doi.org/10.1177/0363546504271208

[38] Levy, B. A., Dajani, K. A., Morgan, J. A., Shah, J. P., Dahm, D. L., & Stuart, M. J. (2010). Repair versus reconstruction of the fibular collateral ligament and posterolateral corner in the multiligament-injured knee. *The American journal of sports medicine*, *38*(4), 804–809. https://doi.org/10.1177/0363546509352459

[39] LaPrade, R. F., Heikes, C., Bakker, A. J., & Jakobsen, R. B. (2008). The reproducibility and repeatability of varus stress radiographs in the assessment of isolated fibular collateral ligament and grade-III posterolateral knee injuries. An in vitro biomechanical study. *The Journal of bone and joint surgery. American volume*, *90*(10), 2069–2076. https://doi.org/10.2106/JBJS.G.00979

[40] Huang, C., Chan, P. K., Chiu, K. Y., Yan, C. H., Yeung, D. S. S., Lai, C. W. K., & Fu, S. N. (2021). Knee joint loadings are related to tibial torsional alignments in people with radiographic medial knee osteoarthritis. *PloS one*, *16*(7), e0255008. https://doi.org/10.1371/journal.pone.0255008

[41] Turner M. S. (1994). The association between tibial torsion and knee joint pathology. *Clinical orthopaedics and related research*, (302), 47–51.

[42] Fornalski, S., McGarry, M. H., Csintalan, R. P., Fithian, D. C., & Lee, T. Q. (2008). Biomechanical and anatomical assessment after knee hyperextension injury. *The American journal of sports medicine*, *36*(1), 80–84. https://doi.org/10.1177/0363546507308189

[43] Schenck, R. C., Jr, Kovach, I. S., Agarwal, A., Brummett, R., Ward, R. A., Lanctot, D., & Athanasiou, K. A. (1999). Cruciate injury patterns in knee hyperextension: a cadaveric model. *Arthroscopy : the journal of arthroscopic & related surgery : official publication of the Arthroscopy Association of North America and the International Arthroscopy Association*, *15*(5), 489–495. https://doi.org/10.1053/ar.1999.v15.0150481

[44] Grood, E. S., Stowers, S. F., & Noyes, F. R. (1988). Limits of movement in the human knee. Effect of sectioning the posterior cruciate ligament and posterolateral structures. *The Journal of bone and joint surgery. American volume*, *70*(1), 88–97.

[45] Algizawy, A.A.EW.M., Sakr, H.M., Nassif, M.A., & Bassiouny, A.M. (2022). Value and imaging findings of the magnetic resonance in the posterolateral corner complex injuries of the knee. *Egyptian Journal of Radiology and Nuclear Medicine, 53(*200). https://doi.org/10.1186/s43055-022-00888-9

[46] Westrich, G. H., Hannafin, J. A., & Potter, H. G. (1995). Isolated rupture and repair of the popliteus tendon. *Arthroscopy : the journal of arthroscopic & related surgery : official publication of the Arthroscopy Association of North America and the International Arthroscopy Association*, *11*(5), 628–632. https://doi.org/10.1016/0749-8063(95)90145-0

[47] Baker, C. L., Jr, Norwood, L. A., & Hughston, J. C. (1983). Acute posterolateral rotatory instability of the knee. *The Journal of bone and joint surgery. American volume*, *65*(5), 614–618.

[48] Sugita, T., & Amis, A. A. (2001). Anatomic and biomechanical study of the lateral collateral and popliteofibular ligaments. *The American journal of sports medicine*, *29*(4), 466–472. https://doi.org/10.1177/03635465010290041501

[49] Gollehon, D. L., Torzilli, P. A., & Warren, R. F. (1987). The role of the posterolateral and cruciate ligaments in the stability of the human knee. A biomechanical study. *The Journal of bone and joint surgery. American volume*, *69*(2), 233–242.

[50] Moorman, C. T., 3rd, & LaPrade, R. F. (2005). Anatomy and biomechanics of the posterolateral corner of the knee. *The journal of knee surgery*, *18*(2), 137–145. https://doi.org/10.1055/s-0030-1248172

[51] Nielsen, S., Rasmussen, O., Ovesen, J., & Andersen, K. (1984). Rotatory instability of cadaver knees after transection of collateral ligaments and capsule. *Archives of*

orthopaedic and traumatic surgery. Archiv fur orthopadische und Unfall-Chirurgie, *103*(3), 165–169. https://doi.org/10.1007/BF00435548

[52] LaPrade, R. F., Tso, A., & Wentorf, F. A. (2004). Force measurements on the fibular collateral ligament, popliteofibular ligament, and popliteus tendon to applied loads. *The American journal of sports medicine*, *32*(7), 1695–1701. https://doi.org/10.1177/0363546503262694

[53] Vap, A. R., Schon, J. M., Moatshe, G., Cruz, R. S., Brady, A. W., Dornan, G. J., Turnbull, T. L., & LaPrade, R. F. (2017). The Role of the Peripheral Passive Rotation Stabilizers of the Knee With Intact Collateral and Cruciate Ligaments: A Biomechanical Study. *Orthopaedic journal of sports medicine*, *5*(5), 2325967117708190. https://doi.org/10.1177/2325967117708190

[54] LaPrade, R. F., & Wentorf, F. (2002). Diagnosis and treatment of posterolateral knee injuries. *Clinical orthopaedics and related research*, (402), 110–121. https://doi.org/10.1097/00003086-200209000-00010

[55] Sims, E. L., Carland, J. M., Keefe, F. J., Kraus, V. B., Guilak, F., & Schmitt, D. (2009). Sex differences in biomechanics associated with knee osteoarthritis. *Journal of women & aging*, *21*(3), 159–170. https://doi.org/10.1080/08952840903054856

[56] Plaweski, S., Belvisi, B., & Moreau-Gaudry, A. (2015). Reconstruction of the Posterolateral Corner After Sequential Sectioning Restores Knee Kinematics. *Orthopaedic journal of sports medicine*, *3*(2), 2325967115570560. https://doi.org/10.1177/2325967115570560

[57] Henke, P., Ruehrmund, L., Bader, R., & Kebbach, M. (2024). Exploration of the Advanced VIVO^{TM} Joint Simulator: An In-Depth Analysis of Opportunities and Limitations Demonstrated by the Artificial Knee Joint. *Bioengineering (Basel, Switzerland)*, *11*(2), 178. https://doi.org/10.3390/bioengineering11020178

[58] Kang, K. T., Koh, Y. G., Son, J., Jung, M., Oh, S., Kim, S. J., & Kim, S. H. (2018). Biomechanical influence of deficient posterolateral corner structures on knee joint kinematics: A computational study. *Journal of orthopaedic research : official*

publication of the Orthopaedic Research Society, 10.1002/jor.23871. Advance online publication. https://doi.org/10.1002/jor.23871

[59] Kang, K. T., Koh, Y. G., Jung, M., Nam, J. H., Son, J., Lee, Y. H., Kim, S. J., & Kim, S. H. (2017). The effects of posterior cruciate ligament deficiency on posterolateral corner structures under gait- and squat-loading conditions: A computational knee model. *Bone & joint research*, *6*(1), 31–42. https://doi.org/10.1302/2046-3758.61.BJR-2016-0184.R1

[60] Cooper D. E. (1991). Tests for posterolateral instability of the knee in normal subjects. Results of examination under anesthesia. *The Journal of bone and joint surgery. American volume*, *73*(1), 30–36.

[61] Hughston, J. C., & Norwood, L. A., Jr (1980). The posterolateral drawer test and external rotational recurvatum test for posterolateral rotatory instability of the knee. *Clinical orthopaedics and related research*, (147), 82–87.

[62] Coelho Fernandes, A. R., Sagoo, K. S., Oluku, J., & Cheema, K. S. (2021). Tibial Malrotation Following Intramedullary Nailing: A Literature Review. *Cureus*, *13*(11), e19683. https://doi.org/10.7759/cureus.19683

[63] Glyn-Jones, S., Palmer, A. J., Agricola, R., Price, A. J., Vincent, T. L., Weinans, H., & Carr, A. J. (2015). Osteoarthritis. *Lancet (London, England)*, *386*(9991), 376–387. https://doi.org/10.1016/S0140-6736(14)60802-3

[64] Fanelli G. C. (2020). Posterior Cruciate Ligament. *Sports medicine and arthroscopy review*, *28*(1), 1. https://doi.org/10.1097/JSA.0000000000000280

[65] Puetzer, J. L., Ma, T., Sallent, I., Gelmi, A., & Stevens, M. M. (2021). Driving Hierarchical Collagen Fiber Formation for Functional Tendon, Ligament, and Meniscus Replacement. *Biomaterials*, *269*, 120527. https://doi.org/10.1016/j.biomaterials.2020.120527

[66] Bobzin, L., Roberts, R. R., Chen, H. J., Crump, J. G., & Merrill, A. E. (2021). Development and maintenance of tendons and ligaments. *Development (Cambridge, England)*, *148*(8), dev186916. https://doi.org/10.1242/dev.186916

[67] Jung, H. J., Fisher, M. B., & Woo, S. L. (2009). Role of biomechanics in the understanding of normal, injured, and healing ligaments and tendons. *Sports medicine, arthroscopy, rehabilitation, therapy & technology : SMARTT*, *1*(1), 9. https://doi.org/10.1186/1758-2555-1-9

[68] Yahia, L. H., & Drouin, G. (1990). Study of the hysteresis phenomenon in canine anterior cruciate ligaments. *Journal of biomedical engineering*, *12*(1), 57–62. https://doi.org/10.1016/0141-5425(90)90116-5

[69] Ristaniemi, A., Regmi, D., Mondal, D., Torniainen, J., Tanska, P., Stenroth, L., Finnilä, M. A. J., Töyräs, J., & Korhonen, R. K. (2021). Structure, composition and fibril-reinforced poroviscoelastic properties of bovine knee ligaments and patellar tendon. *Journal of the Royal Society, Interface*, *18*(174), 20200737. https://doi.org/10.1098/rsif.2020.0737

[70] Lim, H. C., Bae, J. H., Bae, T. S., Moon, B. C., Shyam, A. K., & Wang, J. H. (2012). Relative role changing of lateral collateral ligament on the posterolateral rotatory instability according to the knee flexion angles: a biomechanical comparative study of role of lateral collateral ligament and popliteofibular ligament. *Archives of orthopaedic and trauma surgery*, *132*(11), 1631–1636. https://doi.org/10.1007/s00402-012-1591-7

[71] Andrews, K., Lu, A., Mckean, L., & Ebraheim, N. (2017). Review: Medial collateral ligament injuries. *Journal of orthopaedics*, *14*(4), 550–554. https://doi.org/10.1016/j.jor.2017.07.017

[72] Vosoughi, F., Rezaei Dogahe, R., Nuri, A., Ayati Firoozabadi, M., & Mortazavi, J. (2021). Medial Collateral Ligament Injury of the Knee: A Review on Current Concept and Management. *The archives of bone and joint surgery*, *9*(3), 255–262. https://doi.org/10.22038/abjs.2021.48458.2401

[73] Matsumoto, H., Suda, Y., Otani, T., Niki, Y., Seedhom, B. B., & Fujikawa, K. (2001). Roles of the anterior cruciate ligament and the medial collateral ligament in preventing valgus instability. *Journal of orthopaedic science : official journal*

of the Japanese Orthopaedic Association, *6*(1), 28–32. https://doi.org/10.1007/s007760170021

[74] Butler, D. L., Noyes, F. R., & Grood, E. S. (1980). Ligamentous restraints to anterior-posterior drawer in the human knee. A biomechanical study. *The Journal of bone and joint surgery. American volume*, *62*(2), 259–270.

[75] Duthon, V. B., Barea, C., Abrassart, S., Fasel, J. H., Fritschy, D., & Ménétrey, J. (2006). Anatomy of the anterior cruciate ligament. *Knee surgery, sports traumatology, arthroscopy : official journal of the ESSKA*, *14*(3), 204–213. https://doi.org/10.1007/s00167-005-0679-9

[76] Hollis, J. M., Takai, S., Adams, D. J., Horibe, S., & Woo, S. L. (1991). The effects of knee motion and external loading on the length of the anterior cruciate ligament (ACL): a kinematic study. *Journal of biomechanical engineering*, *113*(2), 208–214. https://doi.org/10.1115/1.2891236

[77] Rosas H. G. (2016). Unraveling the Posterolateral Corner of the Knee. *Radiographics : a review publication of the Radiological Society of North America, Inc*, *36*(6), 1776–1791. https://doi.org/10.1148/rg.2016160027

[78] Vinson, E. N., Major, N. M., & Helms, C. A. (2008). The posterolateral corner of the knee. *AJR. American journal of roentgenology*, *190*(2), 449–458. https://doi.org/10.2214/AJR.07.2051

[79] Watanabe, Y., Moriya, H., Takahashi, K., Yamagata, M., Sonoda, M., Shimada, Y., & Tamaki, T. (1993). Functional anatomy of the posterolateral structures of the knee. *Arthroscopy : the journal of arthroscopic & related surgery : official publication of the Arthroscopy Association of North America and the International Arthroscopy Association*, *9*(1), 57–62. https://doi.org/10.1016/s0749-8063(05)80344-5

[80] Veltri, D. M., Deng, X. H., Torzilli, P. A., Maynard, M. J., & Warren, R. F. (1996). The role of the popliteofibular ligament in stability of the human knee. A biomechanical study. *The American journal of sports medicine*, *24*(1), 19–27. https://doi.org/10.1177/036354659602400105

[81] Noyes, F. R., Dunworth, L. A., Andriacchi, T. P., Andrews, M., & Hewett, T. E. (1996). Knee hyperextension gait abnormalities in unstable knees. Recognition and preoperative gait retraining. *The American journal of sports medicine*, *24*(1), 35–45. https://doi.org/10.1177/036354659602400107

[82] Markolf, K. L., Graves, B. R., Sigward, S. M., Jackson, S. R., & McAllister, D. R. (2007). Effects of posterolateral reconstructions on external tibial rotation and forces in a posterior cruciate ligament graft. *The Journal of bone and joint surgery. American volume*, *89*(11), 2351–2358. https://doi.org/10.2106/JBJS.F.01086

[83] O'Brien, S. J., Warren, R. F., Pavlov, H., Panariello, R., & Wickiewicz, T. L. (1991). Reconstruction of the chronically insufficient anterior cruciate ligament with the central third of the patellar ligament. *The Journal of bone and joint surgery. American volume*, *73*(2), 278–286.

[84] Arthur, A., LaPrade, R. F., & Agel, J. (2007). Proximal tibial opening wedge osteotomy as the initial treatment for chronic posterolateral corner deficiency in the varus knee: a prospective clinical study. *The American journal of sports medicine*, *35*(11), 1844–1850. https://doi.org/10.1177/0363546507304717

[85] Aiona, M., Calligeros, K., & Pierce, R. (2012). Coronal plane knee moments improve after correcting external tibial torsion in patients with cerebral palsy. *Clinical orthopaedics and related research*, *470*(5), 1327–1333. https://doi.org/10.1007/s11999-011-2219-x

[86] Snow M. (2021). Tibial Torsion and Patellofemoral Pain and Instability in the Adult Population: Current Concept Review. *Current reviews in musculoskeletal medicine*, *14*(1), 67–75. https://doi.org/10.1007/s12178-020-09688-y

[87] Walton, D. M., Liu, R. W., Farrow, L. D., & Thompson, G. H. (2012). Proximal tibial derotation osteotomy for torsion of the tibia: a review of 43 cases. *Journal of children's orthopaedics*, *6*(1), 81–85. https://doi.org/10.1007/s11832-012-0384-4

[88] Hutter, C. G., Jr, & Scott, W. (1949). Tibial torsion. *The Journal of bone and joint surgery. American volume*, *31A*(3), 511–518.

[89] Bouklas P. (2020). Tibial Torsion Defects. *Deutsches Arzteblatt international*, *117*(35-36), 599. https://doi.org/10.3238/arztebl.2020.0599a

[90] Cameron, J. C., & Saha, S. (1996). External tibial torsion: an underrecognized cause of recurrent patellar dislocation. *Clinical orthopaedics and related research*, (328), 177–184.

[91] Lincoln, T. L., & Suen, P. W. (2003). Common rotational variations in children. *The Journal of the American Academy of Orthopaedic Surgeons*, *11*(5), 312–320. https://doi.org/10.5435/00124635-200309000-00004

[92] Mednick, R. E., Eller, E. B., Swaroop, V. T., & Dias, L. (2015). Outcomes of Tibial Derotational Osteotomies Performed in Patients With Myelodysplasia. *Journal of pediatric orthopedics*, *35*(7), 721–724. https://doi.org/10.1097/BPO.0000000000000373

[93] Mullaji, A. B., Sharma, A. K., Marawar, S. V., & Kohli, A. F. (2008). Tibial torsion in non-arthritic Indian adults: a computer tomography study of 100 limbs. *Indian journal of orthopaedics*, *42*(3), 309–313. https://doi.org/10.4103/0019-5413.41854

[94] Drexler, M., Dwyer, T., Dolkart, O., Goldstein, Y., Steinberg, E. L., Chakravertty, R., & Cameron, J. C. (2014). Tibial rotational osteotomy and distal tuberosity transfer for patella subluxation secondary to excessive external tibial torsion: surgical technique and clinical outcome. *Knee surgery, sports traumatology, arthroscopy : official journal of the ESSKA*, *22*(11), 2682–2689. https://doi.org/10.1007/s00167-013-2561-5

[95] Kerrigan, D. C., Deming, L. C., & Holden, M. K. (1996). Knee recurvatum in gait: a study of associated knee biomechanics. *Archives of physical medicine and rehabilitation*, *77*(7), 645–650. https://doi.org/10.1016/s0003-9993(96)90002-7

[96] Alexander, N., Wegener, R., Lengnick, H., Payne, E., Klima, H., Cip, J., & Studer, K. (2020). Compensatory gait deviations in patients with increased outward tibial

torsion pre and post tibial derotation osteotomy. *Gait & posture*, *77*, 43–51. https://doi.org/10.1016/j.gaitpost.2020.01.011

[97] Hicks, J., Arnold, A., Anderson, F., Schwartz, M., & Delp, S. (2007). The effect of excessive tibial torsion on the capacity of muscles to extend the hip and knee during single-limb stance. *Gait & posture*, *26*(4), 546–552. https://doi.org/10.1016/j.gaitpost.2006.12.003

[98] Kenawey, M., Liodakis, E., Krettek, C., Ostermeier, S., Horn, T., & Hankemeier, S. (2011). Effect of the lower limb rotational alignment on tibiofemoral contact pressure. *Knee surgery, sports traumatology, arthroscopy : official journal of the ESSKA*, *19*(11), 1851–1859. https://doi.org/10.1007/s00167-011-1482-4

[99] Kuriyama, S., Ishikawa, M., Furu, M., Ito, H., & Matsuda, S. (2014). Malrotated tibial component increases medial collateral ligament tension in total knee arthroplasty. *Journal of orthopaedic research : official publication of the Orthopaedic Research Society*, *32*(12), 1658–1666. https://doi.org/10.1002/jor.22711

[100] Stephen, J. M., Teitge, R. A., Williams, A., Calder, J. D. F., & El Daou, H. (2021). A Validated, Automated, 3-Dimensional Method to Reliably Measure Tibial Torsion. *The American journal of sports medicine*, *49*(3), 747–756. https://doi.org/10.1177/0363546520986873

[101] Markolf, K. L., Burchfield, D. M., Shapiro, M. M., Shepard, M. F., Finerman, G. A., & Slauterbeck, J. L. (1995). Combined knee loading states that generate high anterior cruciate ligament forces. *Journal of orthopaedic research : official publication of the Orthopaedic Research Society*, *13*(6), 930–935. https://doi.org/10.1002/jor.1100130618

[102] Kanamori, A., Sakane, M., Zeminski, J., Rudy, T. W., & Woo, S. L. (2000). In-situ force in the medial and lateral structures of intact and ACL-deficient knees. *Journal of orthopaedic science : official journal of the Japanese Orthopaedic Association*, *5*(6), 567–571. https://doi.org/10.1007/s007760070007

[103] van der Wal, W. A., Heesterbeek, P. J., van Tienen, T. G., Busch, V. J., van Ochten, J. H., & Wymenga, A. B. (2016). Anatomical reconstruction of posterolateral corner and combined injuries of the knee. *Knee surgery, sports traumatology, arthroscopy : official journal of the ESSKA*, *24*(1), 221–228. https://doi.org/10.1007/s00167-014-3369-7

[104] Brown, T. D., Johnston, R. C., Saltzman, C. L., Marsh, J. L., & Buckwalter, J. A. (2006). Posttraumatic osteoarthritis: a first estimate of incidence, prevalence, and burden of disease. *Journal of orthopaedic trauma*, *20*(10), 739–744. https://doi.org/10.1097/01.bot.0000246468.80635.ef

[105] Lee, S. H., Chung, C. Y., Park, M. S., Choi, I. H., & Cho, T. J. (2009). Tibial torsion in cerebral palsy: validity and reliability of measurement. *Clinical orthopaedics and related research*, *467*(8), 2098–2104. https://doi.org/10.1007/s11999-009-0705-1

[106] LaPrade R. F., Floyd, E. R., Carlson, G. B., Moatshe, G., Chahla, J., & Monson, J. (2021). The Posterolateral Corner: Explanations and Outcomes. *Journal of Arthroscopic Surgery and Sports Medicine*, *2*(2), 108 –118. https://doi.org/10.25259/JASSM_66_2020

[107] LaPrade, R. F., Wozniczka, J. K., Stellmaker, M. P., & Wijdicks, C. A. (2010). Analysis of the static function of the popliteus tendon and evaluation of an anatomic reconstruction: the "fifth ligament" of the knee. *The American journal of sports medicine*, *38*(3), 543–549. https://doi.org/10.1177/0363546509349493

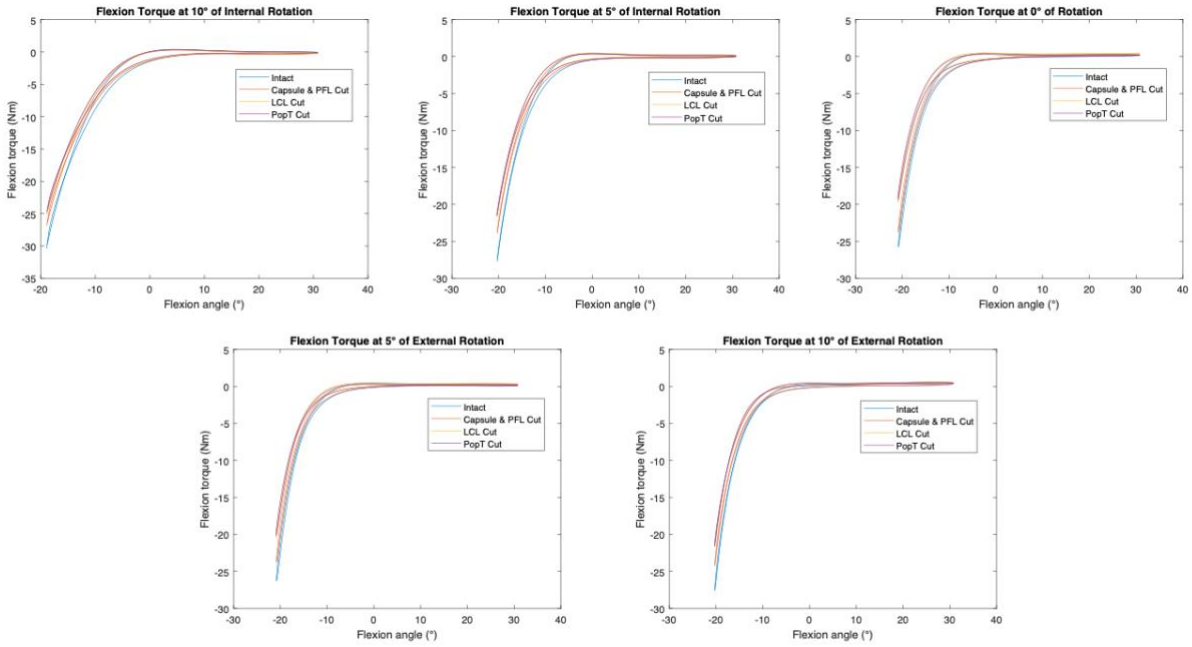
[108] Vivacqua, T., Vakili, S., Willing, R., Moatshe, G., Degen, R., & Getgood, A. M. (2022). Biomechanical Assessment of Knee Laxity After a Novel Posterolateral Corner Reconstruction Technique. *The American journal of sports medicine*, *50*(4), 962–967. https://doi.org/10.1177/03635465211070553

[109] Pasque, C., Noyes, F. R., Gibbons, M., Levy, M., & Grood, E. (2003). The role of the popliteofibular ligament and the tendon of popliteus in providing stability in the human knee. *The Journal of bone and joint surgery. British volume*, *85*(2), 292–298. https://doi.org/10.1302/0301-620x.85b2.12857

[110] Nyland, J., Lachman, N., Kocabey, Y., Brosky, J., Altun, R., & Caborn, D. (2005). Anatomy, function, and rehabilitation of the popliteus musculotendinous complex. *The Journal of orthopaedic and sports physical therapy*, *35*(3), 165–179. https://doi.org/10.2519/jospt.2005.35.3.165

Appendices

Appendix A: Example data collection.



	Flexion Torque (Nm)				
Offset (°)	**10° ER**	**5° ER**	**0°**	**5° IR**	**10° IR**
Flexion angle(°)	-20.3	-20.9	-20.9	-20.4	-18.9
Intact	-27.6	-26.3	-25.8	-27.7	-30.4
LCL Cut	-24.2	-23.8	-23.7	-23.9	-26.8
Capsule & PFL Cut	-21.6	-20.4	-19.7	-21.7	-25.1
PopT Cut	-21.6	-20.1	-19.3	-21.5	-24.8

Figure 0-1: Determining flexion torque values. Flexion torque (Nm) was determined by evaluating the point on each graph where the maximum flexion angle was achieved.

Appendix B: Flexion torque results separated by cutting sequence.

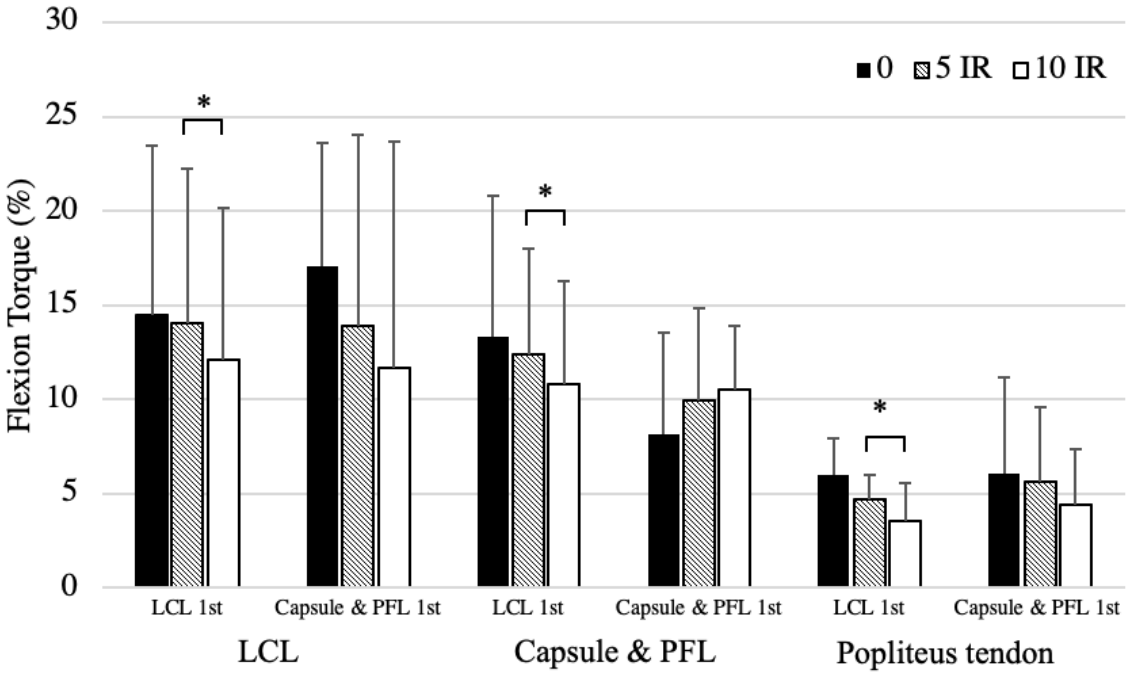


Figure 0-2: PLC restraint of 27 Nm of hyperextension at internal rotations, without a varus torque, separated by sectioning order cohort. * Indicates significantly different results between 5° and 10° IR in the sequence that had the LCL sectioned primarily.

Although cutting sequence, in which some specimens (n = 6) had the capsule + PFL sectioned first, and some had the LCL sectioned first (n = 6), did not produce evidence of influencing the loading of soft tissues during hyperextension without varus torque (p = 0.822), the LCL cut first sequence were significantly different between 5° and 10° of internal rotation (p = 0.014) (Figure 0-2).

Tests that included a varus torque also did not produce evidence of influencing the loading of soft tissues during hyperextension (p = 0.101). Within the LCL cut-first cohort, the LCL and popliteus tendon were significantly different from each other at all rotations, whereas in the capsule + PFL cut-first cohort, the LCL and popliteus tendon were not significantly different. In both sequences, the capsule + PFL had significantly greater hyperextension restraint than the popliteus tendon at 5° (LCL-cohort: p = 0.013,

capsule + PFL-cohort: p = 0.019) and at 10° IR (LCL-cohort: p = 0.027, capsule + PFL-cohort: p = 0.022) (Figure 0-3).

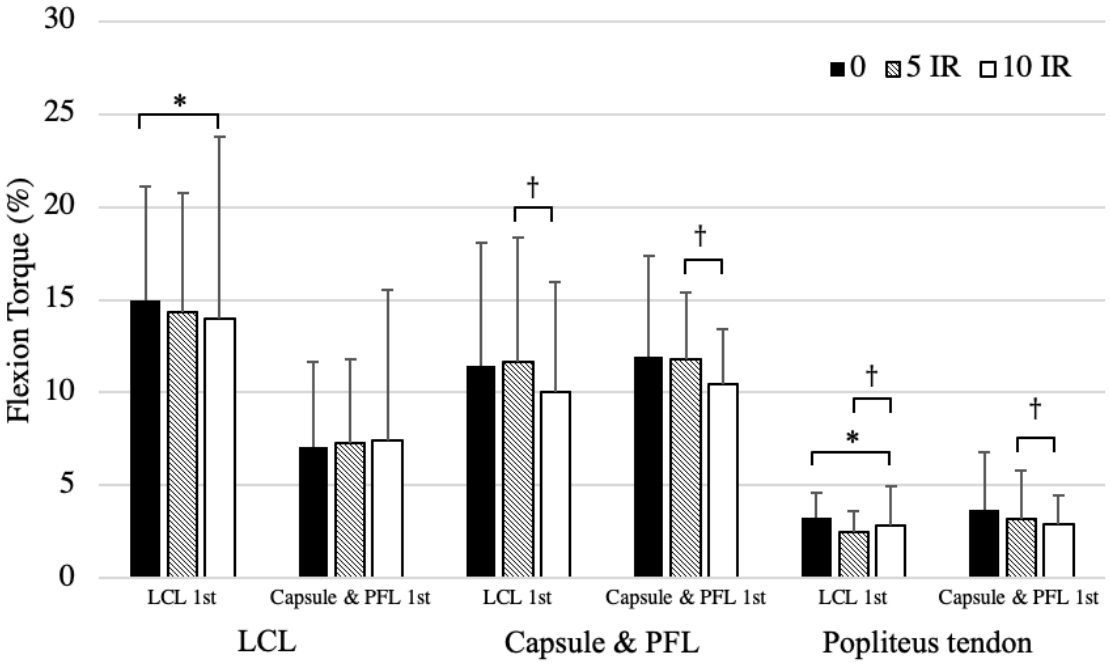


Figure 0-3: PLC restraint of 27 Nm of hyperextension at internal rotations, with a varus torque, separated by sectioning order cohort. * Indicates significantly different results between the structures at all rotational increments. † Signifies significantly different results between structures at 5° and 10°.

External rotations were also unaffected by cutting sequence variations. Cutting sequence did not show statistically significant differences both with (p = 0.452) and without (p = 0.157) varus torque. When the LCL was cut first, there are no longer significant differences between the LCL and popliteus tendon at neutral, only at 5° ER (p = 0.012), and 10° ER (p = 0.018) (Figure 0-4). When the capsule + PFL were cut first, the neutral rotation state of the LCL contributed greater hyperextension restraint than the neutral rotation state of the popliteus tendon (p = 0.023) (Figure 0-4).

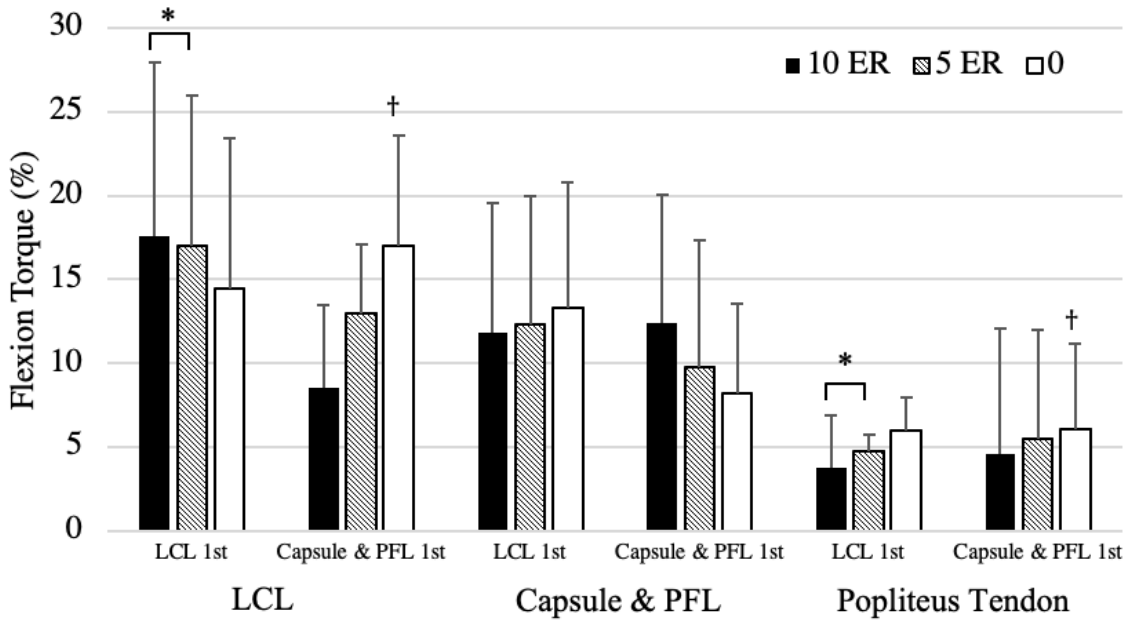


Figure 0-4: PLC restraint of 27 Nm of hyperextension at external rotations, without a varus torque, separated by sectioning order cohort. * Indicates significantly different results between the structures at 5° and 10° ER rotational increments, when the LCL is cut first. † Signifies significantly different results between structures at neutral rotation, when the capsule is cut first.

External rotations with varus torque were also unaffected by cutting sequence variations (p = 0.452). With the addition of varus torque, in LCL cut-first specimens the LCL had greater torque contributions than the popliteus tendon, at all rotational increments including neutral (p = 0.002), 5° ER (p = 0.037), and 10° ER (p = 0.033) (Figure 0-5). In capsule + PFL cut-first specimens, the capsule + PFL capsule had greater torque contributions than the popliteus tendon at 5° ER (p = 0.039) and 10° ER (p = 0.0036) (Figure 0-5).

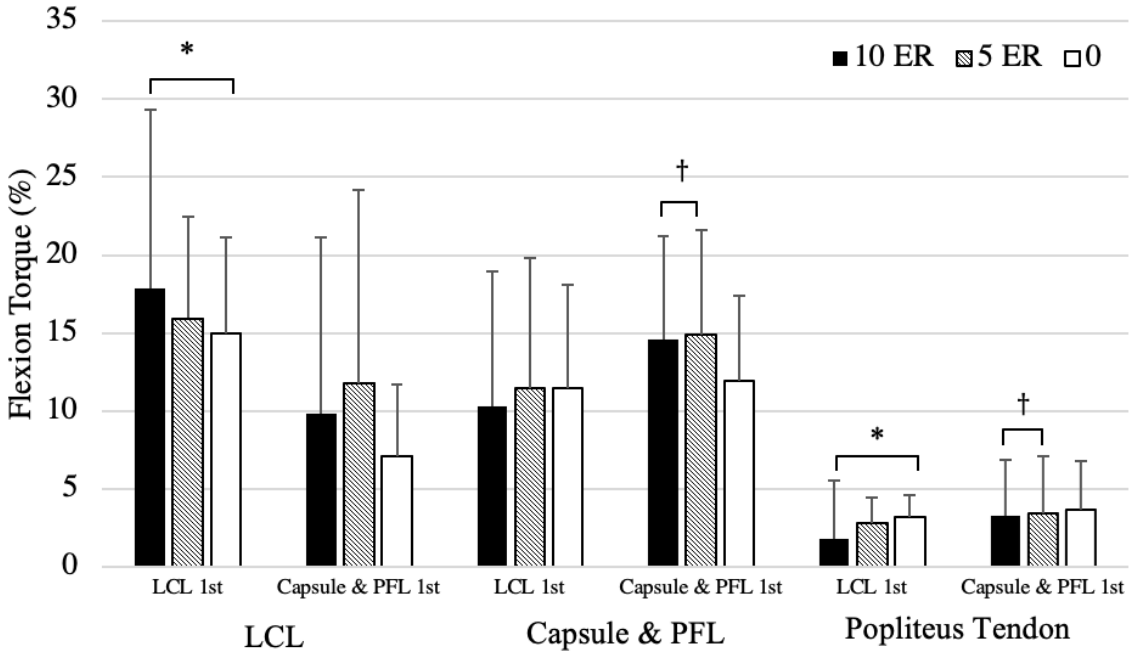


Figure 0-5: PLC restraint of 27 Nm of hyperextension at external rotations, with a varus torque, separated by sectioning order cohort. * Indicates significantly different results between the structures at all rotational increments, when the LCL is cut first. † indicates significantly different results between the structures at 5° and 10° ER rotational increments, when the capsule + PFL is cut first.

Appendix C: Peak hyperextension angles.

Table 1: Hyperextension angle reached by specimens when 27 Nm of hyperextension torque was applied, in the absence of varus torque. Specimens 1-6 had the LCL cut first, specimens 7-12 had the capsule + PFL cut first.

Specimen ID	10° ER	5° ER	0°	5° IR	10° IR
UTN2323019R	-19.3	-19.6	-19.6	-19.2	-18
UTN2322964L	-18.6	-18.7	-18.7	-18.5	-17.3
UTN2323019L	-19.6	-19.8	-19.8	-19.4	-18.2
UTN2322964R	-3.1	-3.6	-3.5	-2.7	-1.1
UTN2323172L	-8.7	-9.7	-9.8	-9.8	-9.5
UTN2323120L	-25.1	-25.2	-25.2	-24.7	-23.2
UTN2323907R	-15	-15.5	-15.5	-14.8	-13.6
UTN2323018R	-26.2	-26.5	-26.5	-25.7	-24.5
UTN2323172R	-6.8	-7	-7	-6.6	-5.4
UTN2323120R	-20.3	-20.9	-20.9	-20.4	-18.9
UTN2322986R	-4.6	-6	-6.7	-6.7	-6.1
UTN2322986L	-9.6	-10.1	-10.1	-9.3	-8
Average	-14.7	-15.2	-15.3	-14.8	-13.7

Table 2: Hyperextension angle reached by specimens when 27 Nm of hyperextension torque was applied, with the addition of a 10 Nm varus torque. Specimens 1-6 had the LCL cut first, specimens 7-12 had the capsule + PFL cut first.

Specimen ID	10° ER	5° ER	0°	5° IR	10° IR
UTN2323019R	-19.8	-20	-20	-19.5	-18.3
UTN2322964L	-19.3	-19.3	-19.3	-18.7	-17.2
UTN2323019L	-18.6	-18.7	-18.7	-18.2	-17.5
UTN2322964R	-3.1	-3.6	-3.6	-2.7	-1.1
UTN2323172L	-8.8	-9.7	-9.8	-9.8	-9.5
UTN2323120L	-25.5	-25.6	-25.6	-24.9	-23.1
UTN2323907R	-15.8	-16.2	-16.2	-15.6	-14.3
UTN2323018R	-27.4	-27.4	-27.4	-26.7	-25.3
UTN2323172R	-7.6	-7.6	-7.6	-7.2	-5.8
UTN2323120R	-20.2	-20.8	-20.9	-20.3	-18.8
UTN2322986R	-7.1	-6.5	-4.9	-6.3	-7
UTN2322986L	-9.1	-9.3	-9.3	-8.5	-7.3
Average	-15.2	-15.4	-15.3	-14.9	-13.8

Appendix D: External/Internal rotation torque results.

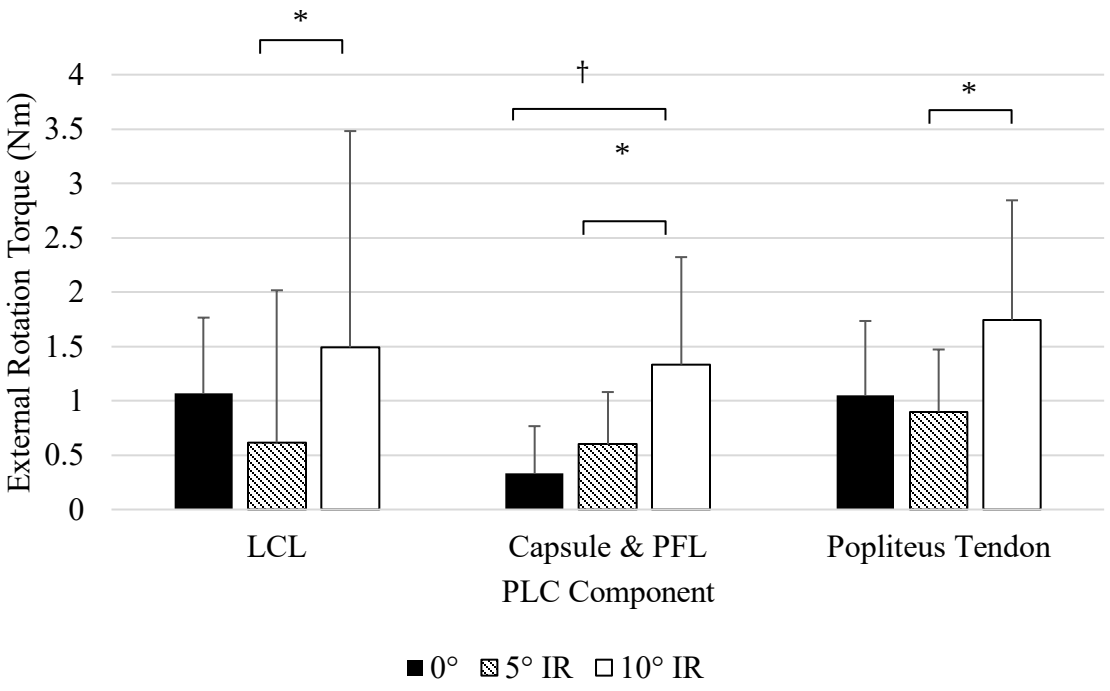


Figure 0-6: External rotation torque in PLC components exposed to 27 Nm of hyperextension torque and internally rotated, without varus torque. * Indicates statistical significance between 5° and 10° IR. † Indicates statistical significance between 10° IR and neutral.

In the absence of varus torque, mean contribution of external rotation restraint from the LCL was not statistically significantly different from neutral at 5° IR (p = 0.844) and 10° IR (p = 1.000), but 5° IR was contributing significantly less external rotation torque restraint than 10° IR (p = 0.004). The mean external rotation torque contribution of the capsule + PFL was significantly greater at 10° IR than at neutral (p = 0.037), and 5° IR (p = 0.015). The popliteus tendon did not have significantly different contributions to external rotation torque compared to neutral when the specimen was internally rotated by 5° (p = 1.000) and 10° (p = 0.473). The contributions were significantly greater when rotated internally by 10° than by 5° (p = 0.003).

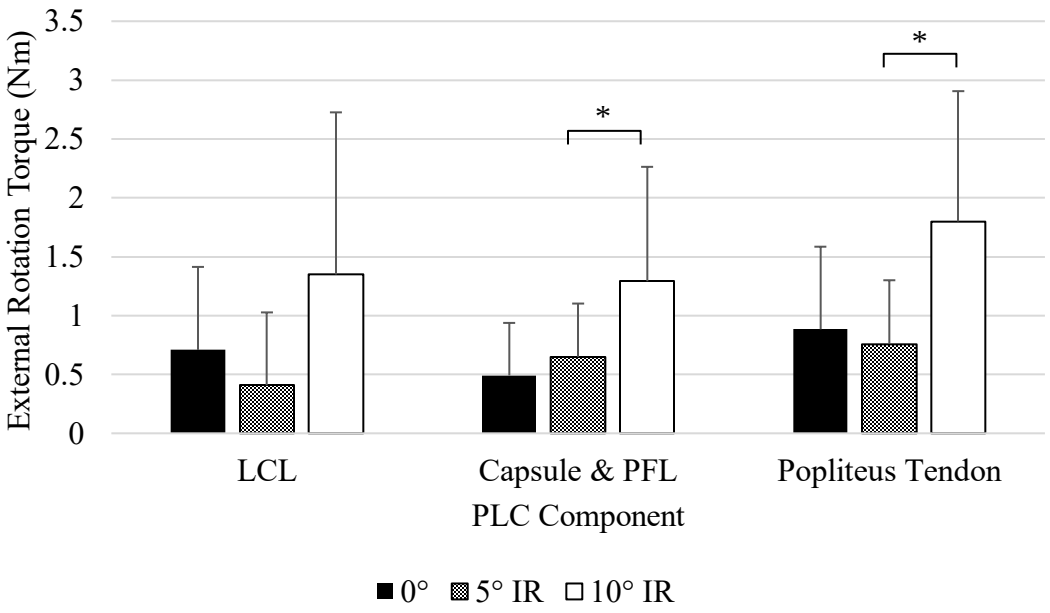


Figure 0-7: External rotation torque in PLC components exposed to 27 Nm of hyperextension torque and internally rotated, with varus torque. * Indicates statistical significance between 5° and 10° IR.

With the addition of varus torque, the mean contribution of external rotation restraint from the LCL was not significantly different from neutral at 5° IR (p = 1.000) and 10° IR (p = 0.762). The LCL was also not significantly different when comparing external rotation torque restraint at 5° IR and 10° IR (p = 0.055). The mean external rotation torque contribution of the capsule + PFL was not significantly different from neutral at 5° IR (p = 1.000) and 10° IR (p = 0.105), but 5° IR contributed significantly less external rotation torque restraint than 10° IR (p = 0.034). The popliteus tendon did not have significantly different contributions to external rotation torque compared to neutral when the specimen was internally rotated by 5° (p = 1.000) and 10° (p = 0.246). The contributions were significantly greater when rotated internally by 10° than by 5° (p = <0.001).

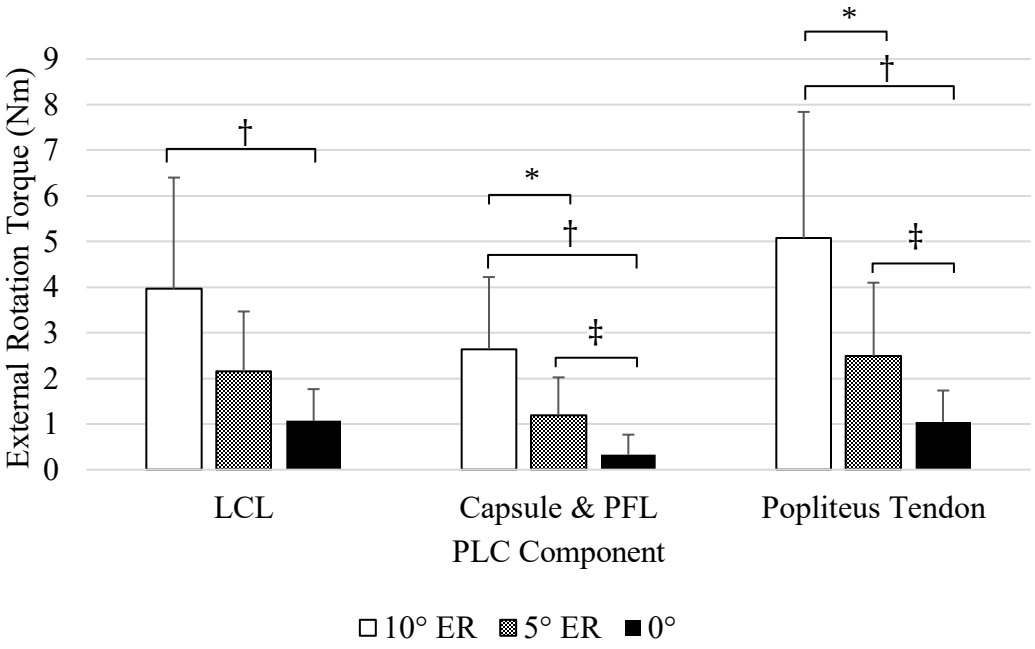


Figure 0-8: External rotation torque in PLC components exposed to 27 Nm of hyperextension torque and externally rotated, without varus torque. * Indicates statistical significance between 5° and 10° ER. † Indicates statistical significance between 10° ER and neutral. ‡ Indicates statistical significance between 5° ER and neutral.

In the absence of varus torque, the mean contribution of external rotation restraint from the LCL was significantly greater than neutral at 10° ER (p = 0.005), but not 5° ER (p = 0.070). The mean contribution of external rotation restraint from the capsule + PFL was significantly greater than neutral at 5° ER (p = 0.005) and 10° ER (p = 0.001). External rotation restraint from the capsule + PFL was significantly less at 5° ER than 10° ER (p = 0.001). The mean contribution of external rotation restraint from the capsule + PFL was significantly greater than neutral at 5° ER (p = 0.005) and 10° ER (p = <0.001). External rotation restraint from the popliteus tendon was significantly less at 5° ER than 10° ER (p = <0.001).

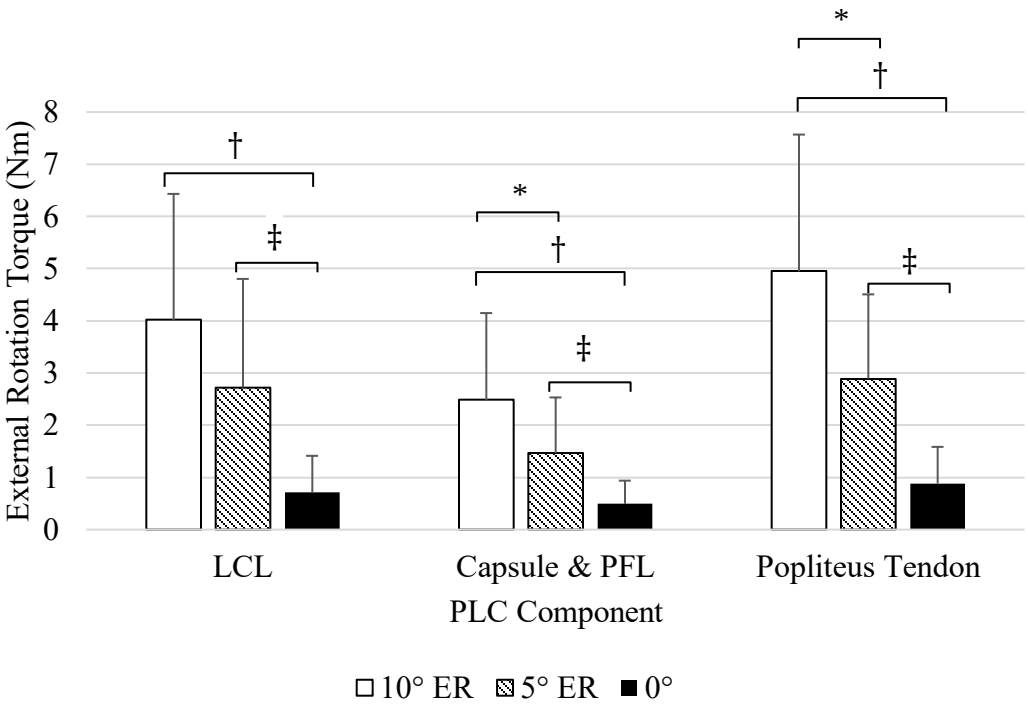


Figure 0-9: External rotation torque in PLC components exposed to 27 Nm of hyperextension torque and externally rotated, with varus torque. * Indicates statistical significance between 5° and 10° ER. † Indicates statistical significance between 10° ER and neutral. ‡ Indicates statistical significance between 5° ER and neutral.

With the addition of varus torque, the mean contribution of external rotation restraint from the LCL was significantly greater than neutral at 5° ER (p = 0.029) and 10° ER (p = 0.001). The LCL was also not significantly different when comparing external rotation torque restraint at 5° ER and 10° ER (p = 0.088). The mean external rotation torque contribution of the capsule + PFL was significantly greater than neutral at 5° ER (p = 0.004) and 10° ER (p = 0.003), and 5° ER contributed significantly less external rotation torque restraint than 10° ER (p = 0.030). The popliteus tendon had significantly greater contributions to external rotation torque compared to neutral when the specimen was externally rotated by 5° (p = 0.002) and 10° (p = <0.001). The contributions were significantly greater when rotated externally by 10° than by 5° (p = 0.012).

Appendix E: Varus/Valgus rotation torque results.

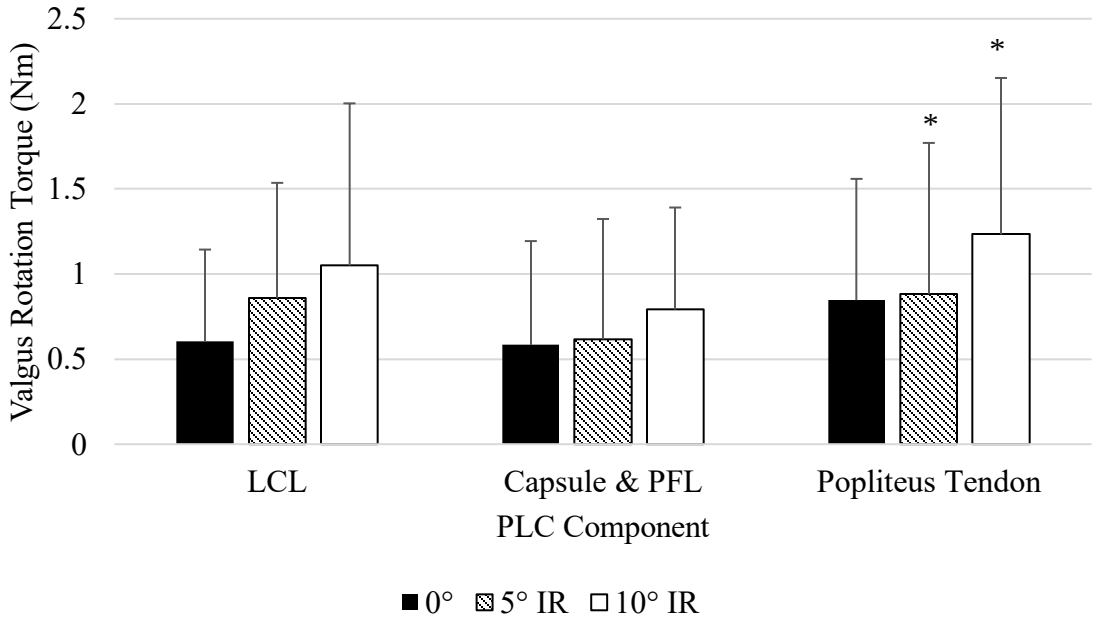


Figure 0-10: Valgus rotation torque in PLC components exposed to 27 Nm of hyperextension torque and internally rotated, without varus torque. * Indicates statistical significance between 5° and 10° IR.

In the absence of varus torque, mean contribution of valgus rotation restraint from the LCL was not statistically significantly different from neutral at 5° IR (p = 0.412) and 10° IR (p = 0.264). The mean valgus rotation torque contribution of the capsule + PFL was not statistically significantly different from neutral at 5° IR (p = 1.000) and 10° IR (p = 0.306). The popliteus tendon did not have significantly different contributions to valgus rotation torque compared to neutral when the specimen was internally rotated by 5° (p = 1.000) and 10° (p = 0.264). The contributions of the PLC were significantly greater when rotated internally by 10° than by 5° (p = 0.029).

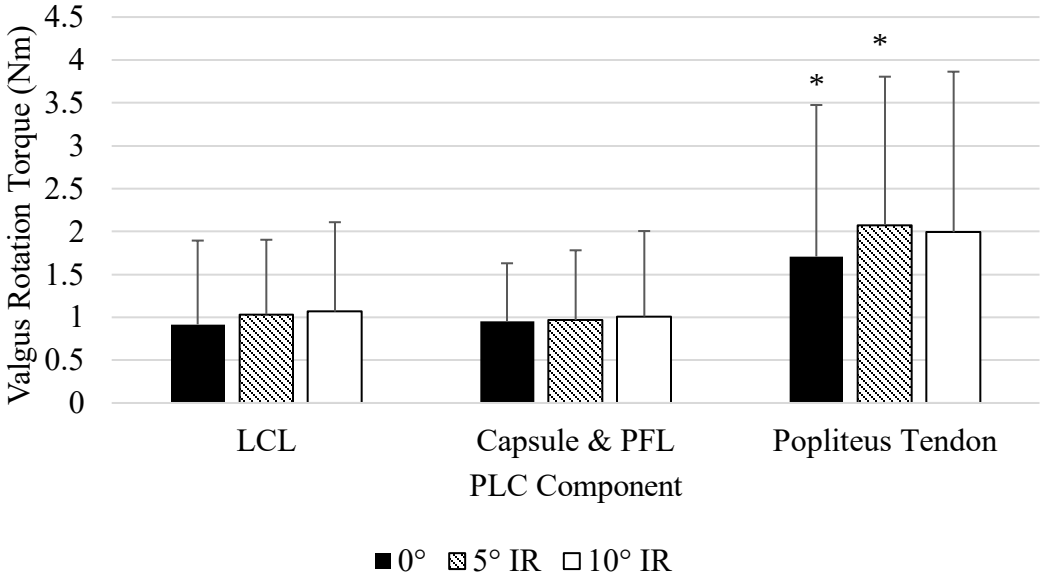


Figure 0-11: Valgus rotation torque in PLC components exposed to 27 Nm of hyperextension torque and internally rotated, with varus torque. * Indicates statistical significance between neutral and 5° IR.

With the addition of varus torque, mean contribution of valgus rotation restraint from the LCL was not statistically significantly different from neutral at 5° IR (p = 1.000) and 10° IR (p = 1.000). The mean valgus rotation torque contribution of the capsule + PFL was not statistically significantly different from neutral at 5° IR (p = 1.000) and 10° IR (p = 1.000). The popliteus tendon had significantly greater contributions to valgus rotation torque compared to neutral when the specimen was internally rotated by 5° (p = 0.034), but not when rotated by 10° (p = 0.943).

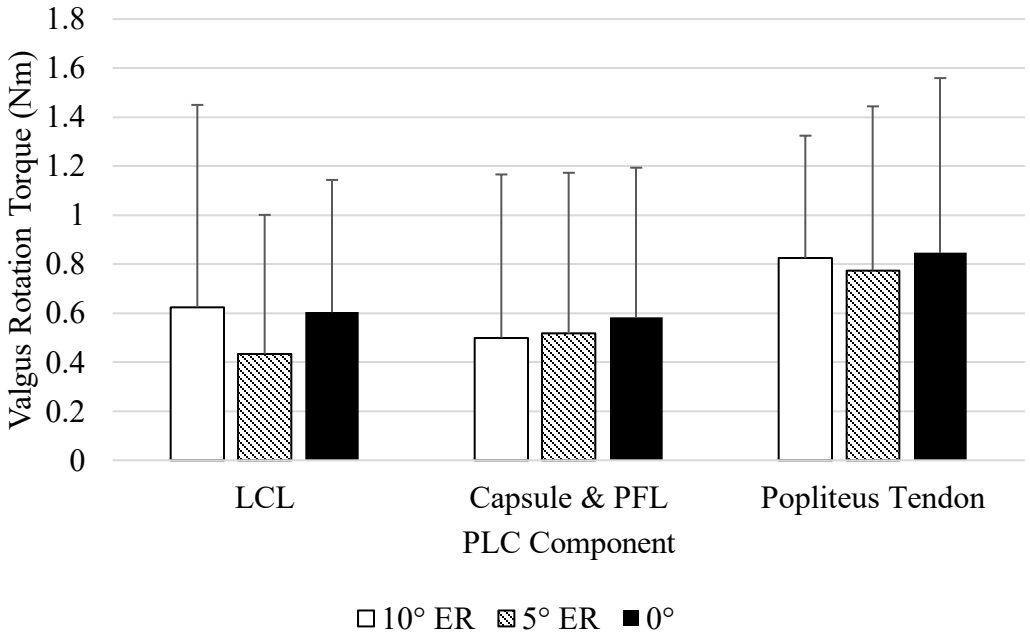


Figure 0-12: Valgus rotation torque in PLC components exposed to 27 Nm of hyperextension torque and externally rotated, with varus torque.

In the absence of varus torque, mean contribution of valgus rotation restraint from the LCL was not statistically significantly different from neutral at 5° ER (p = 0.827) and 10° ER (p = 1.000). The mean valgus rotation torque contribution of the capsule + PFL was not statistically significantly different from neutral at 5° ER (p = 1.000) and 10° ER (p = 1.000). The popliteus tendon did not have significantly different contributions to valgus rotation torque compared to neutral when the specimen was externally rotated by 5° (p = 1.000) and 10° (p = 1.000).

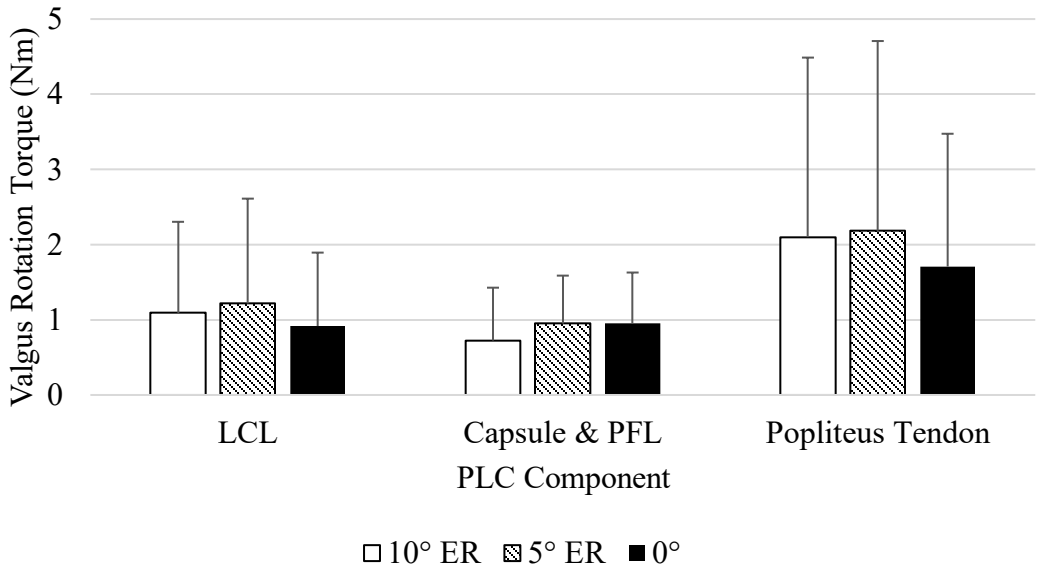


Figure 0-13: Valgus rotation torque in PLC components exposed to 27 Nm of hyperextension torque and externally rotated, with varus torque.

With the addition of varus torque, mean contribution of valgus rotation restraint from the LCL was not statistically significantly different from neutral at 5° ER (p = 0.583) and 10° ER (p = 1.000). The mean valgus rotation torque contribution of the capsule + PFL was not statistically significantly different from neutral at 5° ER (p = 1.000) and 10° ER (p = 1.000). The popliteus tendon did not have significantly different contributions to valgus rotation torque compared to neutral when the specimen was externally rotated by 5° (p = 0.457) and 10° (p = 0.515).

Curriculum Vitae

Name:	Larissa Madia
Post-secondary Education and Degrees:	The University of Western Ontario London, Ontario, Canada 2018-2022 B.MSc. The University of Western Ontario London, Ontario, Canada 2022-2024 M.E.Sc
Honours and Awards:	Dean's Honour List – The University of Western Ontario 2020-2022
Related Work Experience	Teaching Assistant The University of Western Ontario 2023-2024

Publications:
Pending