Knee Meniscal Pathology and Treatments: Ramifications for Knee Articular Cartilage

By

Kendall Henry

Submitted in partial fulfillment of the requirements for graduation as an Honors Student at Point Loma Nazarene University, San Diego, California, April, 19, 2017.

Approved By Nicole L Cosby

Nicole Cosby, Mentor

Date 4/24/2017

Committee Members:

Leon Kugler

Brandon Sawyer

ABSTRACT

In the United States, meniscal lesions represent the most common intra-articular knee injury.[1] In fact, the mean annual incidence of meniscal tears is approximately 60 to 70 per 100,000 patients.[2] Anatomically, the menisci play a major role in load bearing, load transmission of the lower extremity, shock absorption, as well as nutrition and lubrication of the articular cartilage of the tibiofemoral joint.[2] Common symptoms of a meniscal tear include: clicking, catching or locking, joint line tenderness, a feeling of "giving out" or instability, pain with squatting or pivoting motions, pain at end range of flexion and/or extension, and an overall loss of range of motion at the tibiofemoral joint.[1,2] Most often, the standard of care for meniscal tears that occur in the avascular region is arthroscopic partial meniscectomy, this surgery accounts for 10-20% of all orthopedic surgeries in the United States.[1,2] Partial meniscectomy is said to provide symptom relief, restore tibiofemoral biomechanics, and improve the quality of life for the patient. However, this has not been the case, long-term analysis has identified patients who receive partial meniscectomies as being at increased risk of developing OA in the knee joint.[1,2] Given the importance of the menisci in the knee joint and the poor prognosis that is associated with the "gold standard" treatment, it is important to explore other treatment options as a means to manage meniscal tears. Therefore, this review of literature includes an in depth study of the anatomy and biomechanics of the tibiofemoral joint when the joint is in a healthy state. Additionally, a deeper analysis of the menisci and the associated consequences for knee articular cartilage that can occur due to injury of these anatomical structures has been examined. This honors project specifically explored the current methods for diagnosis and treatments as well as new researched techniques available to treat meniscal pathologies.

Key Words: meniscus, tibiofemoral joint, osteoarthritis

LITERATURE REVIEW

KNEE ANATOMY

A thorough knowledge of the anatomy, function and structures of the knee joint is vitally important when clinicians are making decisions about treatment following a knee injury. This literature review will provide the reader with an overview of the muscular, tendinous, ligamentous, bony, cartilaginous structures that provide support to the tibiofemoral joint. Additionally, this review discussed the consequences of meniscal injury and the long-term health of the knee following surgical intervention.

MUSCLES

Anterior Compartment - Quadriceps Femoris Muscles

The muscles of the thigh are organized into three compartments by intermuscular septa that pass between the muscle groups. Those compartments are named on the basis of their location or action at the knee joint and include: the anterior or extensor, medial or adductor, and posterior or flexor compartments.[3] The quadriceps femoris (or quadriceps) forms the main bulk of the anterior thigh muscles and as a whole is the largest and most powerful muscle group in the human body.[3] The quadriceps consists of four parts: the rectus femoris (RF), vastus lateralis (VL), vastus intermedius (VI), and vastus medialis (VM). The tendons of the four quadriceps muscles unite distally to a form a single, strong band known as the quadriceps tendon.[3] The quadriceps tendon then

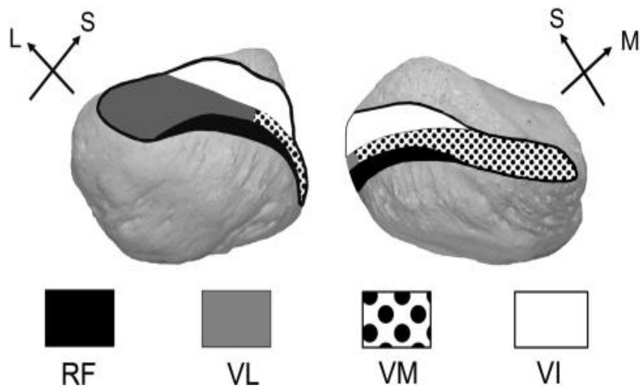


Figure 1. Image of Quadriceps Attachments on Patella[1]

continues to form the patellar ligament which attaches to the tibial tuberosity of the tibia. More specifically, the four muscular elements of the quadriceps fuse to form one quadriceps tendon 2 cm proximal to the patella (see figure 1 above for illustration on where tendons attach).[3] For the purpose of this literature review we will focus on the quadriceps function/role at the tibiofemoral joint. Concentrically, the quadriceps contract to extend the knee and have been labeled as the great extensors of the leg.[3] Perhaps their greatest function at the tibiofemoral joint is when they contract concentrically to extend the knee against gravity during tasks such as walking up stairs, rising from sitting or squatting, and during tasks that require acceleration and projection (running and jumping).[3] In addition to their concentric functions, the quadriceps also contract eccentrically to slow the rate of knee flexion during tasks such as downhill walking and descending stairs. During walking, the quadriceps muscles become active during the termination of the swing phase, which prepares the tibiofemoral joint to accept weight.[3]

The most superficial muscle of the quadriceps group is the RF. The RF muscle originates on the anterior inferior iliac spine of the ilium and inserts via common tendinous and independent attachments to the base of the patella and indirectly to the tibial tuberosity via the patellar ligament.[4] Because the RF attaches to the hip and the tibia it is considered a two joint muscle and is therefore a flexor of the hip joint and an extender of the tibiofemoral joint during concentric contraction.[3,4] The RF works with the psoas muscle during the pre-swing and initial swing phases of walking to accelerate hip flexion and then later in terminal swing to extend the knee to prepare for contact.[3] Injury to the RF can lead to a change in mechanics at the tibiofemoral joint. When there is a loss of function of the RF tibiofemoral extension has been reduced by 17%, this lack of knee extension means the tibiofemoral joint will spend more time in a knee flexed position.[3,4] Several biomechanical changes occur when the knee is not allowed to return to an extended state: 1) when the knee is in a flexed position there is an increase in contact area between the patella and the femur and an increase in compressive forces across the joint and 2) the menisci are not as mobile and stay in a deformed position, which can lead to degeneration of the menisci and subsequently degeneration of the articular cartilage of the tibiofemoral joint.[4,5]

The intermediate layer of the quadriceps group is comprised of the VL and VM. Proximally the VL attaches on the greater trochanter of the femur and the lateral lip of the linea aspera and the VM attaches to the intertrochanteric line and medial lip of the linea aspera of the femur.[4] The VL and VM unite to form a continuous aponeurosis that inserts into the base of the patella, just posterior to the insertion of the RF. Individually, the VL ends in an aponeurosis that blends with the lateral side of the suprapatellar or RF tendon. The lateral expansion of the VL then blends with the capsule of the knee, thereby forming part of the lateral patellar retinaculum.[4] Medially, the fibers of the VM end in an aponeurosis that blends with the medial side of the suprapatellar tendon. Most of the VM's distal fibers attach directly to the medial edge of the patella and extend more distally than fibers originating from any other part of the quadriceps group.[4] The deep fibers of the VM reinforce the joint capsule as a part of the medial patellar retinaculum. The most distal muscle fibers of the VM are often referred to as the VMO because their fibers pass obliquely across the patella to attach to the lateral tibial condyle.[4] The obliquely oriented fibers derived from the more superficial VMO contribute to medial patellar stability through their contribution to the superficial layer of the medial patellofemoral ligament.[4]

The deepest layer of the quadriceps muscle group consists of the vastus intermedius (VI) muscle. The VI has an intimate origin with the VL proximally and the lateral intermuscular septum distally.[4] It inserts through a broad, thin tendon into the base of the patella posterior to the VL and VM. Medially and laterally, this insertion reinforces the patellofemoral ligaments.[4] The vastus intermedius originates from the anterior and lateral surfaces of the femoral shaft.[3] All three of the vastus muscles aid in the extension of the tibiofemoral joint, but it is often difficult to isolate each of these muscles specific function.[3] Unlike the RF, the VM, VL, VI muscles do not cross two joints and therefore function only to extend the tibiofemoral joint.

Lying deep to the VI is the articularis genu (see figure 2).[3] The articularis genu is a derivative of the vastus intermedius muscle and originates from the anterior surface of the distal aspect of the femur and inserts into the proximal and posterior aspects of the suprapatellar bursa.[3,6] The articularis genu muscle retracts or elevates the suprapatellar bursa during extension of the knee, preventing entrapment of the bursa between the femur and patella.[3,6]

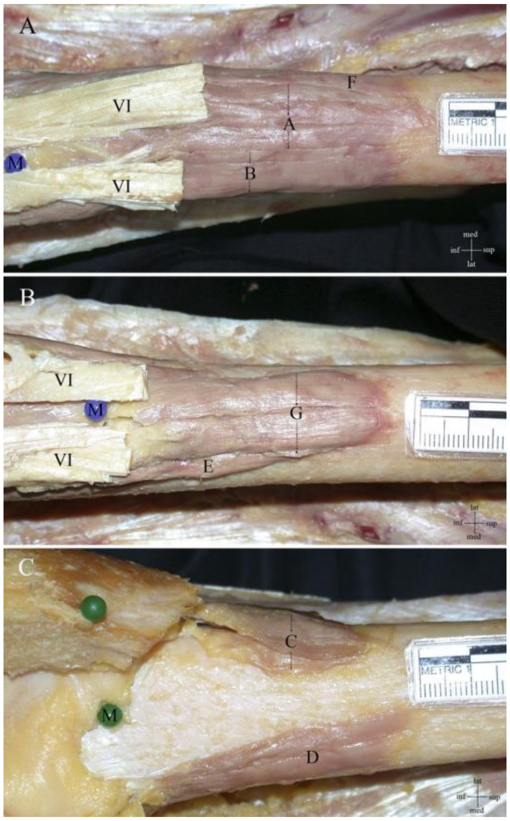


Figure 2. Cadaveric Dissection of the Articularis Genu.[4]

Posterior Compartment - Hamstring Muscle Complex

On the posterior surface of the thigh is the hamstring muscle complex, which consists of the biceps femoris, semitendinosus, semimembranosus.[3] The biceps femoris muscle is considered a double muscle, with the long head arising from the medial facet of the ischial tuberosity and the short head arising from the lateral linea aspera, lateral supracondylar line, and intermuscular septum.[7] The short head is the only muscle in the HMC that does not span two joints. Distally, the biceps femoris tendon inserts onto the fibular head, lateral condyle of the tibia, and the fascia of the leg. Additionally, the long head is innervated by the tibial portion of the sciatic nerve and the short head by the peroneal division.[7] The most tendinous hamstring muscle is the semitendinosus muscle.

The fibers of this muscle attach to the superior portion of the ischial tuberosity by way of a conjoin tendon with the long head of the biceps femoris muscle.[3,7] As the muscle spans from the ischial tuberosity to its distal attachment to the tibial flare it becomes tendinous. The muscle fibers distally insert onto the tibia with the gracilis and sartorius to form the pes anserine (see Figure 3).[5,7]

Figure 3. Cadaveric Illustration of the Pes Anserine Muscle Group

The third muscle in the HMC is the semimembranosus. The semimembranosus muscle originates on the superolateral aspect of the ischial tuberosity, deep to the proximal half of the semitendinosus muscle. The semimembranosus runs medial and anterior to the other hamstring tendons.[7] The proximal tendon of the semimembranosus is elongated and has connections to the adductor magnus tendon and the origin of the long head of the biceps femoris muscle. In contrast, the semitendinosus muscle is a thin, band like tendinous structure after its origin and for most of its course through the thigh.[3,7] Distally, the semimembranosus muscle has multiple insertions to the medial tibial condyle, posterior oblique ligament, and the posterior joint capsule and arcuate ligament. All of these attachments assist the tibial collateral ligament in providing stability to the medial side of the knee when a valgus force is applied.[3,7]

The primary function of the muscles in the HMC is to contract eccentrically, thereby absorbing kinetic energy to protect the knee and hip joints. During gait, the muscles of the HMC concentrically contract during the swing phase to actively produce extension at the hip and actively resist extension of the knee.[7] During heel strike these muscles also function to decelerate forward translation of the tibia during knee extension when foot strike occurs and the weight of the body is being shifted forward. Therefore, the HMC can be considered a dynamic stabilizer of anterior tibial transition, working alongside the ACL to stabilize the tibiofemoral joint.[7]

Medial Compartment - Adductor Muscle Group

There are several muscles that make up the medial compartment, which include the adductor longus, adductor brevis, adductor magnus, gracilis, obturator externus and pectineus. The muscles in this group as whole attach proximally to the anteroinferior external surface of the pubic bone, ischiopubic ramus, and ischial tuberosity) and distally to the linea aspera of the femur.[5] All of the muscles in this group except the hamstring portion of the adductor magnus are supplied by the obturator nerve. The hamstring part of the adductor magnus is supplied by the tibial part of the sciatic nerve. This can be better understood by observing the distal attachment of the adductor magnus hamstring portion onto the adductor tubercle of the femur.[5] Since the focus of this honors project is not on the hip and hip anatomy there will not be an in depth discussion on the structure and function of the medial compartment.[5]

LIGAMENTS

Ligaments are bands of dense, elastic connective tissue that connect one bone to another. In particular, the ligaments of the tibiofemoral joint enable it to move in six degrees of freedom: three rotations and three translations. Rotations about different axes are referred to as either internal-external rotation, flexion-extension rotation, or varus-valgus rotation.[8] There are four knee ligaments that play an important role in the providing stability at the tibiofemoral joint. Those ligaments are the anterior cruciate ligament, posterior cruciate ligament, medial collateral ligament and lateral collateral ligament. These ligaments can be defined, on the basis of their morphology, as flat (MCL) or cord-like (ACL, PCL, LCL); they can be identified as intracapsular (ACL, PCL) or extracapsular (MCL, LCL, patellar ligament), or they can they be classified as individual.[8] Each of the ligaments, with the exception of the LCL, is divided into various bundles or components. The ACL is composed of anteromedial and posterolateral bands, and the PCL is composed of anterolateral and posteromedial bands. These divisions are clinically important because each bundle has a separate function at the tibiofemoral joint. For both of the cruciate ligaments, the anterior bundle is tight during flexion and the posterior bundle is tight during extension.[9] The MCL is also divided into bundles; superficial portion and deep portion. The superficial portion of the MCL can be divided into anterior and posterior portions, with the anterior portion becoming tight between 70 to 105 degrees of knee flexion. The deep portion of the MCL on the other hand can be divided into two portions: the meniscofemoral and meniscotibial ligaments.[9] Each of these ligamentous structures will be discussed in more detail below.

EXTRA-ARTICULAR

Patellar Ligament

The first and most anterior extracapsular ligament that helps provide stability to the tibiofemoral joint is the patellar ligament.[3] The patellar ligament, also known as the distal part of the quadriceps tendon, is a strong fibrous band that passes from the apex of the patella to the tibial tuberosity of the tibia.[3] The patellar ligament is located on the anterior aspect of the tibiofemoral joint and plays an important role in maintaining alignment of the patella relative to the patellar articular surface of the femur (see figure 4).[3]

Figure 4. Image of the patellar ligament

Lateral Collateral Ligament

On the lateral aspect of the joint capsule is the lateral collateral ligament. The lateral collateral ligament (LCL), is a cord-like structure that is very strong (see figure 5).

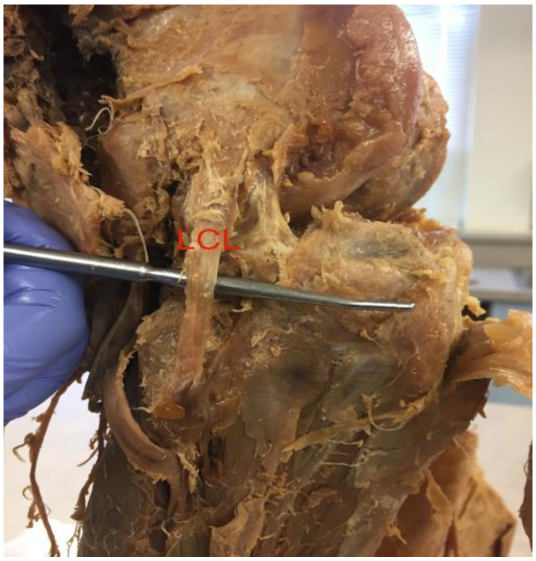


Figure 5. Lateral Collateral Ligament Observed During Cadaveric Dissection

It proximally attaches to the lateral epicondyle of the femur and distally attaches to the fibular head. The LCL is the primary restraint to excessive varus force on the tibiofemoral joint.[3] It has been reported to resist approximately 55% of the applied varus load when the tibiofemoral joint is in full extension and the cruciate ligaments (mainly ACL) resisting approximately 25% of the load at full extension. The LCL also acts to resist internal rotation forces at the tibiofemoral joint.[3] Isolated sectioning of the LCL results in an increase in varus opening, but this is a minimum increase unless the other posterolateral structures are also sectioned. Fu *et al.*[9] reported increased varus angulation when the LCL and PCL were sectioned when compared to just the LCL in isolation.

Medial Compartment Ligaments

On the medial aspect of the joint capsule there are three structures that are considered static stabilizers of the medial knee and they are: the superficial medial collateral ligament (MCL), the deep MCL, and the posterior oblique ligament.[10] The MCL is a strong, flat band that originates from the medial femoral epicondyle and inserts on the medial aspect of the proximal tibia five to seven centimeters below the joint line.[3]

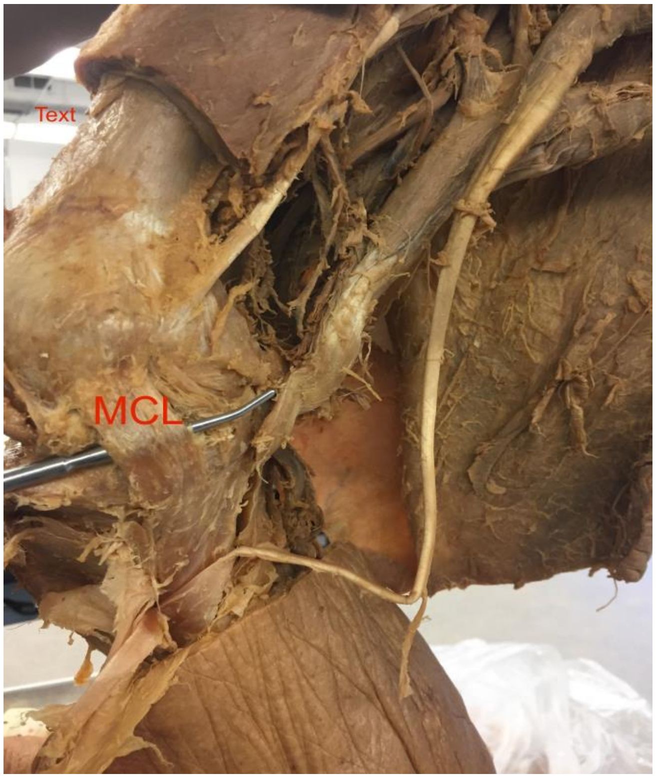


Figure 6. Medial Collateral Ligament - Superficial Bundle

As stated above, the MCL has two bundles (superficial and deep). The most superficial bundle (tibial collateral portion) provides primary restraint to valgus forces at the tibiofemoral joint. In fact, when the knee is in full extension, the MCL resists about 50% of the applied valgus load (see Figure 6).[3,9] While at 25 degrees of knee flexion the MCL provides 78% of the valgus force because there is decreased contribution from the posterior capsule.[10] What is also interesting is that isolated sectioning of the ACL, PCL, LCL, or posterolateral structures do not cause large increases in valgus angulation, however, sectioning of the MCL in isolation increases valgus instability markedly.[3] The largest increases in valgus laxity also occurred when the MCL and PCL were sectioned together.[3,9] The deep bundle of the MCL is the thick part of the middle third of the medial capsule. The deep bundle of the MCL originates from the medial epicondyle of the femur and inserts onto the tibia just below the joint line. It extends from the femur to the mid-portion of the peripheral margin of the medial meniscus as well.[3,10] Its attachments to the medial meniscus divides the deep MCL into its meniscofemoral and meniscotibial ligaments. It has been reported that injuries to the deep MCL causes increases in external rotation of the tibia.[11] The third structure that provides static stability to the medial side of the knee is the posterior oblique ligament. This ligament arises off of the adductor tubercle of the distal femur and has three distal attachments: the tibial arm which inserts on the edge of the posterior surface of the tibia and upper edge of the semimembranosus tendon, the superior or capsular arm which is continuous with the posterior capsule and inserts onto the proximal part of the oblique popliteal ligament, and distal or inferior arm which blends with the sheath covering of the semimembranosus tendon.[10] Overall, the major function of the posterior oblique ligament is to provide static restraint to valgus loads as the knee moves into full extension. It may, by way of its inferior arm act as a dynamic stabilizer to valgus force when the knee moves into flexion by way of its attachment to the semimembranosus.[10]

In addition to the three major static stabilizers of the medial joint capsule there are two structures situated posteriorly that also aid in providing stability to the tibiofemoral joint. One of those ligaments, the oblique popliteal ligament as mentioned in the discussion of the HMC muscle group is an extension of the semimembranosus tendon, which runs obliquely from the tibia proximally and laterally to its insertion on the lateral femoral condyle.[11] When there is contraction of the semimembranosus the oblique popliteal ligament is pulled medially and anteriorly which tightens the medial posterior capsule of the tibiofemoral joint.[11] The joint capsule is also strengthened posterolaterally by the arcuate popliteal ligament. The arcuate popliteal ligament arises from posterior aspect of the fibular head and spreads over the posterior surface of the tibiofemoral joint.[3] The oblique popliteal ligament and the arcuate ligament contribute to the stability and strength of the posterolateral aspect of the tibiofemoral joint.[3]

INTRA-ARTICULAR

The intra-articular ligaments of the knee joint consist of the cruciate ligaments.[3] The cruciate ligaments form a crisscross within the joint capsule, but are outside the synovial capsule. The cruciate ligaments are found at the center of the tibiofemoral joint and cross each other obliquely.[3] The cruciate ligaments wind around each other during medial rotation of the tibia on the femur, and thus limit the amount of medial rotation possible to about 10°.[3]

Anterior Cruciate Ligament

The anterior cruciate ligament consists of two major fibrous bundles, the anteromedial and posterolateral bundle, which are named based on the orientation of their tibial insertions. Source anatomists suggest the ACL has more than two fibrous bundles, but it is widely accepted that there are two major functional bundles, which will be discussed in depth below.[12] In the frontal plane, the anteromedial bundle has a more vertical direction (approximately 70 degrees to the knee baseline), while the posterolateral bundle has more horizontal orientation (approximately 55 degrees to the knee baseline). The anteromedial bundle attaches most proximally on the posterior portion of the medial surface of the lateral condyle of the femur and attaches to the tibia anteromedially on the intercondyloid eminence of the tibia.[12] Whereas the posterolateral bundles are more distally situated on the posterior portion of the medial surface of the lateral condyle of the femur and insert on the posterolateral part of the tibial insertion site. In a study conducted by Peterson *et al*[12], it was found that some of the distal fibers of the posterolateral bundle attach to posterior portion of the lateral meniscus. The general function of the ACL as a whole is to be a primary restraint against anterior tibial translation. In fact, the ACL accepts 75% of anterior force at full extension of the six degrees of freedom that occur at the tibiofemoral joint, which were discussed earlier above.[9] Although the ACL is the primary resistance against anterior forces, its two bundles play vital roles when the knee is placed in different positions.[12] When the knee is in an extended position, the posterolateral bundle is tight and the anteromedial bundle is moderately lax. In contrast, when the knee is flexed the femoral attachment of the ACL develops a more horizontal orientation the anteromedial bundle becomes taut and the posterolateral bundle becomes lax.[12] The anteromedial and posterolateral bundle also have independent functions in providing knee stability. The posterolateral bundle is the most dominant when resisting anterior translation of tibia during knee extension and the anteromedial bundle becomes most effective at 90° of flexion.[12] It was reported that when the posterolateral bundle of the ACL is transected there was substantially higher anterior tibial translation at 0 and 30 degrees of knee flexion when compared to a knee with an intact ACL.[12] These findings exemplify the importance of the posterolateral bundle in knee stabilization, especially when the knee is near full extension. The ACL also plays a major role in the restraint against the coupled movement of anterior and medial translation that occurs with internal rotation at the tibiofemoral joint.[9] Studies show that an isolated loss of the ACL results in an excessive amount of internal rotation occurring at the joint. Therefore, the ACL is essential in protecting the tibiofemoral joint from excessive internal rotation as well as anterior translation.[9]

Posterior Cruciate Ligament

The posterior cruciate ligament, like the ACL, may be split into two functional bundles; the anterolateral and the posteromedial bundle relating to their attachment on the femur. The PCL has a very compact distal attachment to the tibia.[13] The anterolateral bundle occupies a central area covering almost the entire intercondylar surface of the posterior tibial plateau.[13] The posteromedial bundle occupies a central area just superior to the plateau rim and its fibers blend with the periosteum of the tibia. The posteromedial fibers are most superficial with the anterolateral fibers deep to it on their tibial attachment site.[13] As a whole, the PCL is a very strong ligament, much stronger than the ACL. Its strength relates to a large cross-sectional area, and the fibers having an extensive femoral attachment.[13] The femoral attachment of the PCL extends across the roof and the medial aspect of the femoral intercondylar notch. The anterolateral bundle mostly attaches to the roof of the intercondylar notch while the posteromedial bundle attaches mostly to the medial side wall of the notch on the medial femoral condyle.[13] The extent of the attachment is variable, and is influenced by the presence or absence of meniscofemoral ligaments. Due to its femoral attachment site, the two bundles have different patterns of tightening and relaxing during knee flexion and extension.[13] During knee extension, the posteromedial bundle is very tight and aligned in a proximal-distal direction. Therefore, it is not aligned to restrain posterior translation of the tibia in this position, but resists hyperextension.[13] The posteromedial bundle starts to become slack once the knee starts to reach mid-flexion. However, once in deep flexion the posteromedial bundle fibers attach more anteriorly and superiorly away from the tibial plateau causing them to become tight again.[13] Thus, in deep knee flexion the posteromedial bundle is well aligned and tight in order to resist posterior tibial translation in this position.[13] The anterolateral fiber bundle is curved in the sagittal plane, which causes it to be slack when the knee is an extended position. When the knee flexes, the anterolateral fiber bundle becomes tight and takes a steeper angle from the tibial plateau.[13] This steeper angle causes the bundle to be less efficient at withstanding posterior tibial translation when in an extended position.[13]

As a whole, the PCL is the primary restraint to posterior tibial translation. It sustains approximately 90% of the posterior force at both 30 and 90 degrees of flexion.[9,13] Studies done by Fu *et al* [9,13], show that isolated loss of the posterior cruciate ligament result in an increase in posterior translation to a maximum of fifteen to twenty millimeters at 90 degrees of flexion as well as loss of coupled external rotation. The PCL also becomes taut at 90 degrees of flexion, which shows that it provides some restraint against excessive external rotation if the posterolateral structures are injured. Also, concomitant loss of the PCL with posterolateral structures and the LCL results in increased rotation laxity.[13]

Although the PCL's primary function is to prevent excessive posterior tibial translation there are other structures that aid in posterior restraint. Recent studies performed by Gupte *et al*[13] showed the importance of these other structures specifically in posterior translation as the knee reaches full extension. These structures are the ligaments of Wrisberg and Humphrey. When the ligaments of Wrisberg and Humphrey are present they attach distally to the posterior horn of the lateral meniscus and embrace the PCL anteriorly and posteriorly to attach just distally to the PCL on the femur (See Figure 7).[13] Due to their slanting arrangement from the meniscus to the femoral intercondylar notch, they are able to help withstand tibial posterior translation along with the PCL.[13] The ligaments and Wrisberg and Humphrey will be further discussed in the meniscus section of this honors project.

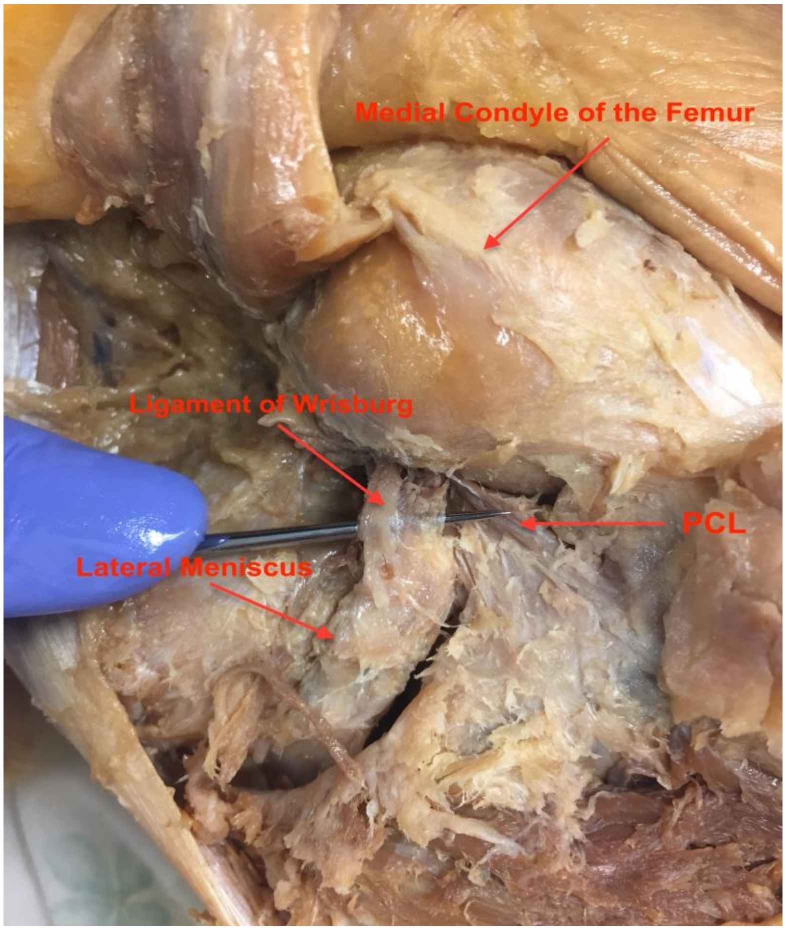


Figure 7. Cadaveric Dissection of the Ligament of Wrisberg

BONES

The tibiofemoral joint is the largest and most superficial joint in the body. It is a combination of three articulations, one between the femur and patella and two between the femoral condyles and tibial plateaus.[14] It is located between the two longest lever arms of the body and bears a majority of body weight (see figure 8).[14] It is described as

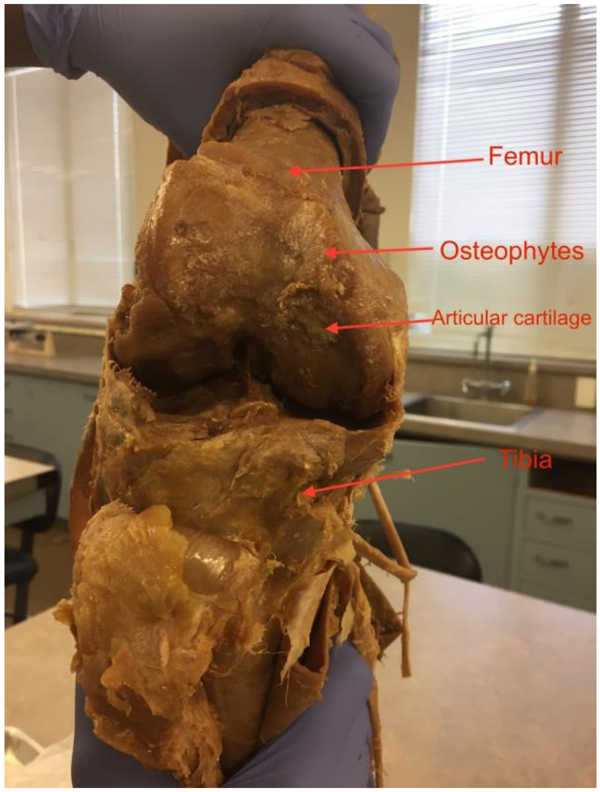


Figure 8. Boney Anatomy of the Tibiofemoral Joint

a hinge type of synovial joint with two degrees of freedom: flexion-extension and axial rotation. The amount of knee flexion and extension will vary and from 120 degrees to 160 degrees and 0 to 15 degrees, respectively.[3,14] Axial rotation also varies depending on the direction of rotation. Generally, the tibia can laterally rotate up to 40 degrees and medially rotate up to 30 degrees.[14] During the osteokinematic movements of flexion and extension of the tibiofemoral joint is a combined roll, glide, and spin that occurs between the tibia and femur, these arthrokinematic movements help maintain joint congruency.[15] During closed chain extension of the tibiofemoral joint the femoral condyles roll anteriorly and glide posteriorly on the tibial plateaus. This movement is also accompanied by medial rotation of the femur during the last 30 degrees of extension, a phenomenon known as the ‘screw home mechanism’.[14] In open chain extension, the tibial plateaus roll and glide anteriorly on the femoral condyles and in the last 30 degrees the tibia laterally rotates. During closed kinetic chain of tibiofemoral knee flexion, the femoral condyles roll posteriorly and glide anteriorly on the tibial plateaus with a conjunct lateral rotation of the femur at the beginning of flexion.[14] In open kinetic chain flexion, the tibial plateaus roll and glide posteriorly on the femoral condyles and internal rotates during the initial 30 degrees.[14]

The femur is the longest and heaviest bone in the body. It is most important in weight transference from the hip joint to the tibia during walking and standing in the human body.[3] The femur can be categorized into three distinct regions. It's proximal end, which connects to the acetabulum of the pelvic bone, the shaft, and the distal portion.[3] The distal portion of the of the femur has a medial and lateral condyle, which each have a distinct shape that is congruent with the tibial plateau. The distinct shape of the condyles is vital in the movement of the tibia in relation to the femur when walking, or performing other movements. The condyles articulate the proximal surface of the tibial plateau.[16] The proximal end of the tibia, forms a relatively flat articular surface known as the tibial plateau. The tibia is the second longest bone in the body. The tibia is characterized by the way it flares outward proximally in order to increase area for articulation with the femur, and the role it plays in weight transference in the lower limb.[3] In the middle of the tibial plateau lies the intercondylar eminence, which is formed by two intercondylar tubercles.[3] The intercondylar eminence articulates in the intercondylar fossa between the two condyles of the femur. The femoral condyles merge anteriorly to form a shallow longitudinal depression known as the patellar surface. This surface articulates with the patella.[3] The patella is located at the anterior aspect of the tibiofemoral joint, and is classified as the largest sesamoid bone in the body. It has a triangular shape along with an anterior and posterior surface. The apex of the patella is situated inferiorly, and is connected to the tibial tuberosity via the patella ligament.[17] The base of the patella forms the superior aspect, and provides the attachment site for the quadriceps tendon. The posterior surface of the patella articulates with the patellar surface of the femur.[17] The patella plays a major role in the movement of the tibiofemoral joint. The patella improves the efficiency of the extensor forces through the entire knee flexion range, and it centralizes the forces of the different quadriceps muscle bellies.[17] It also indirectly contributes to overall stability of the tibiofemoral joint, and ultimately shields the anterior aspect of the joint.[17] Each of these bones contain a cartilaginous surface on the proximal and distal ends of their bony surfaces. This cartilage, known as articular cartilage, is essential in the prevention of bone on bone contact and allows for friction reduced contact between articulations of the bones. The structure and properties of healthy articular cartilage is optimal for a joint to maintain its mechanical function.[18]

MENISCI

Histology and Structure

The menisci of the knee joint are located between the condyles of the tibia and femur.[1] Each meniscus is composed of 70% water and 30% organic matter.[19] Seventy-five percent of the organic make up is collagen, while 8-13% of the remaining dry matter consists of noncollagenous structures.[19] The collagenous make-up of the menisci is mainly Type I, which is one of the major distinctions between the menisci of the knee and articular cartilage, which is composed of mainly Type II collagen. Each meniscus is composed of three collagen fiber layers that are specifically arranged to convert compressive loads at the tibiofemoral joint into circumferential stresses.[19] This conversion of compressive loads to circumferential stresses allows for forces to be distributed more evenly across the tibiofemoral joint. In the superficial layer, the fibers travel radially, serving as ties that resist shearing and splitting forces at the tibiofemoral joint. In the middle layer, the fibers run parallel or circumferentially to reduce compressive forces during weight bearing.[19] And in the deep collagenous layer of the meniscus the fibers are aligned parallel to the periphery.[19] Proteoglycans make up the remainder of meniscal structure. In particular, the glycosaminoglycans, which make up 1% of the nonorganic matter of the menisci contribute most to its material properties.[19] They are responsible for tissue hydration, compressive stiffness and elasticity of the meniscus. It is the size of these proteoglycan macromolecules in combination with the water-retention and electrostatic properties that gives the menisci their compressive stiffness and the exudation of water from the glycosaminoglycans that provides joint lubrication as water is forced into the joint space during compressive loads.[19] Higher concentrations of glycosaminoglycans have been reported in the meniscal horns and the inner half of the menisci, this increased concentration coincides with the largest weight-bearing areas of the tibiofemoral joint.[19] Accounting for less than 1% of meniscal tissue is elastin. Elastin is believed to aid in the recovery of meniscal shape after load deformation. This biochemical make up is what allows the menisci to withstand many different forces across the tibiofemoral joint.[19] From the outer rim moving inwardly, the superior portions of the menisci are concave, which enable effective articulation with the convex femoral condyles and the inferior surfaces are flat to conform to the tibial plateaus.[19]

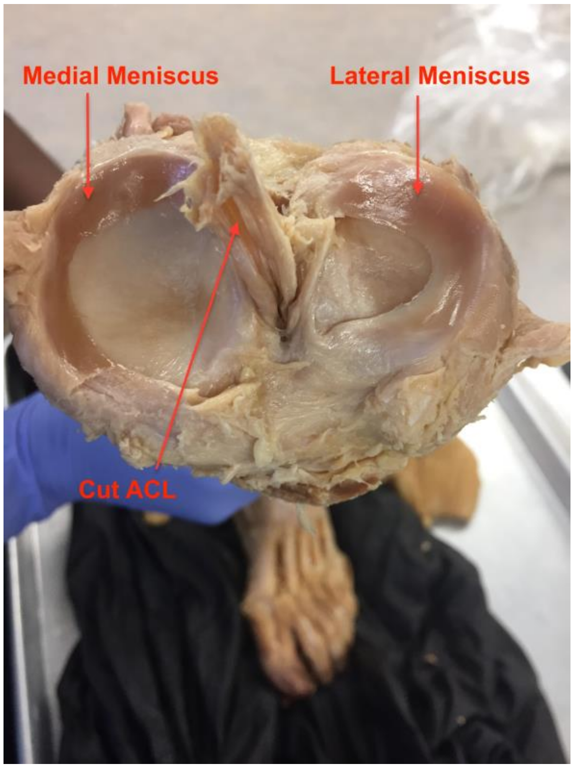


Figure 9. Cadaveric illustration of the medial and lateral meniscus

While the histology of the menisci is important to their structure and function so is their shape and size. The menisci are C-shaped or semicircular fibrocartilaginous structures (see figure 9) that play a major role in load transmission, shock absorption, proprioception, and improvement of stability and lubrication at the tibiofemoral joint.[2] In addition to the organic and nonorganic matter that provide structural integrity to the menisci of the tibiofemoral joint, the role of ligamentous support is vital to the menisci as they are constantly being loaded, deformed and returned to a resting state. The main stabilizing ligaments of the menisci are the deep medial collateral ligament, the transverse ligament (see figure 10), the meniscofemoral ligaments, and attachments at the anterior and posterior horns.[1]

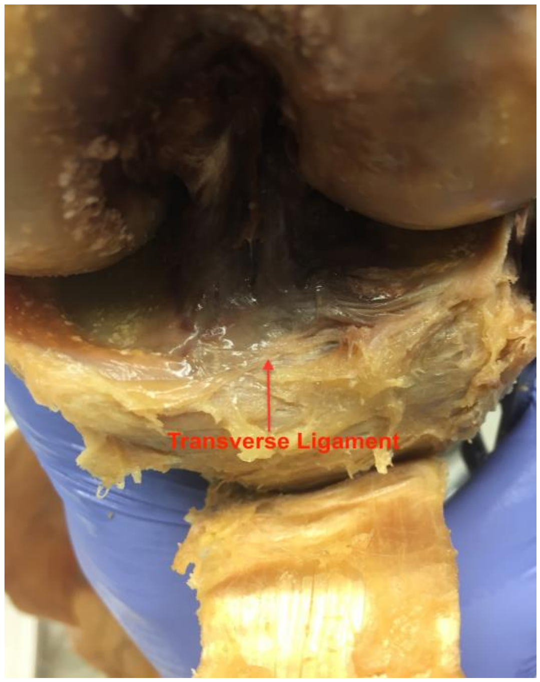


Figure 10. Cadaveric Dissection of the Transverse Ligament

In general the menisci are thicker at their periphery, and have unattached edges in the central aspect of the joint.[3] They are also wider throughout their center and are firmly attached at their anterior and posterior horns to the intercondylar area of the tibia whereas their external margins attach to the joint capsule of the tibiofemoral joint.[3] While both menisci are roughly wedge-shaped structures, the lateral meniscus displays greater variation in size, thickness, and mobility than the medial meniscus. It also covers a larger portion of the tibial plateau (75-93% laterally) compared to the medial meniscus (51-74% medially).[1] A more in depth discussion on specific attachments and shapes of each meniscus is provided below.

The medial meniscus is semicircular and almost 3.5 cm in length. The anterior horn of the medial meniscus attaches to the intercondylar fossa of the tibia just anterior to the ACL and its posterior fibers merge with the transverse intermeniscal ligament to provide a connection between the anterior horns of the two menisci.[20] The posterior horn of the medial meniscus is firmly attached to the posterior intercondylar fossa of the tibia just anterior to the PCL tibial attachment site. Its periphery is attached to the joint capsule and tibial plateau via the coronary ligament.[20] At its midpoint (most medial portion) the medial meniscus is firmly attached to the femur and tibia by way of the deep fibers of the MCL, which limits it mobility.[20]

The lateral meniscus is almost circular in gross morphology, and covers a larger portion of the tibial plateau when compared to the medial meniscus.[20] The bony attachments of the lateral meniscus are anterior and posterior to the ACL onto the tibia. Unlike the medial meniscus, the lateral meniscus has a looser peripheral attachment, which allows for greater translation and movement.[20] The main stabilizing ligaments of the lateral meniscus are the transverse ligament, the meniscofemoral ligaments, and the attachments at the anterior and posterior horns.[1] The meniscofemoral ligaments known as the ligaments of Humphrey and Wrisberg connect the posterior horn of the lateral meniscus to the medial femoral condyle.[1] It is rare for an individual to have both of the meniscofemoral ligaments. Approximately 46% of people have both, but 100% of individuals have at least one.[1] The attachments of the menisci are most secure at the posterior horns which act like a block against the femoral condyles, especially on the medial meniscus. The various ligamentous attachments increase the stability of the menisci and prevent them from being extruded out of the tibiofemoral joint during compressive loads.[19] It is the combination of the organic, nonorganic and ligamentous structures of the menisci that help to improve the efficiency of the tibiofemoral joint as a whole.[19]

Function

The meniscus is an important multifunctional component of the knee, which plays a major role in load transmission, shock absorption, proprioception, improvement of stability and lubrication of the tibiofemoral joint. The menisci are believed to carry between 40% to 70% of the load across the knee.[19] It is this function of the menisci at the tibiofemoral joint that protect the articular cartilage from compressive stresses. In order to carry such high loads across the tibiofemoral joint the menisci must be able to resist extrusion from the joint space when load is being applied.[19] This stability is provided by the multilayer orientation of the menisci, the numerous ligamentous attachments to each meniscus and the intermeniscal ligament attachments as well. Load distribution at the tibiofemoral joint is facilitated by the menisci conforming to an incongruent joint surface and moving as the femur and tibia move.[19] This meniscal movement improves joint dynamics by enhancing joint congruency, and also protects the menisci from injury between the articular surfaces of the femur and tibia.[19]

During knee flexion in the open kinetic chain, the femoral condyles glide posteriorly on the tibial plateau in conjunction with tibial internal rotation. During this movement, the lateral meniscus undergoes twice the anteroposterior translation than the medial meniscus during knee flexion.[19] Henning and Lynch[19,20] reported that the medial meniscus moves posteriorly approximately 5.1 mm and the lateral meniscus moves approximately 11.2 mm as the knee flexes from 0° to 120°. It is this translation of the menisci in an anteroposterior direction that prevents the femur from contacting the posterior margin of the tibial plateau.[19] A deeper examination into the movement of the menisci reveals that the medial condyle rolling-to-translation is 1:1, whereas the lateral condyle rolling-to-translation is 1:4. In the parasagittal plane, the medial meniscus is much more restricted in movement due to the posterior oblique fibers of the deep MCL attaching to it. Therefore, the medial meniscus is more susceptible to injuries when compared to the lateral meniscus.[20] The lateral meniscus is stabilized and motion guided by the popliteus tendon, popliteomeniscal ligaments, popliteofibular ligament, meniscofemoral ligaments, and the lateral capsule, but it still has more mobility than the medial meniscus.[20] Vedi *et al*[21] also reported that the lateral meniscus not only moves more than the medial meniscus during open kinetic knee flexion, but they also observed increased movement in the anterior horn of both menisci when compared to the posterior horn. The increased movement of the anterior horn during knee flexion is consistent with tibiofemoral arthrokinematics. During knee flexion the anterior horns of the menisci must move in order to maintain congruency of the joint surface while the femoral condyles move on the tibia.[21] While there is increased movement of the anterior horns, this is quite the opposite for the posterior horns of the menisci. The soft tissue attachments of the menisci are most substantial at the posterior horns, especially on the medial meniscus.[21] The increased soft tissue attachments on the posterior horns increases stability of the tibiofemoral joint by preventing anterior tibial translation. Since the posterior horns are less mobile they act like “wheel-blocks” against the posterior femoral condyles.[21] It is the immobility of the posterior horn that leads to the increased frequency of injury to this part of the meniscus.[21]

The menisci transmit large loads across the entire joint, and their contact areas change with different degrees of knee flexion and rotation.[20] It has been reported that the intact menisci occupy approximately 60% of the contact area between the articular cartilage of the femoral condyles and the tibial plateau, while they transmit greater than 50% of the total axial load applied in the joint.[1] These percentages are dependent on the degree of knee flexion and the health of the meniscus. Walker and Hajek[22] reported that for every 30 degrees of knee flexion, the contact surface between the femur and tibia decreases by 4%. When the knee is in an extended position the medial and lateral meniscus transmit at least 50% to 70% of the load, however, when the knee moves from extension to flexion the load transmitted through the menisci increases to 85% when the knee is at 90°.[22] Additionally, removal of the medial meniscus results in a 50-70% reduction in femoral condyle contact area and a 100% increase in contact stress. Total lateral meniscectomy causes a 40-50% decrease in contact area and increases contact stress in the lateral compartment 200% to 300%. With a decreased contact area within the joint, stresses are increased and unevenly distributed across the joint.[2] This change leads to increased compression and shearing forces across the joint and an increased risk of injury to the articular cartilage.[2]

Vascular Supply

The menisci display regional variations in vascularization as individuals mature.[1] Blood vessels and lymphatics can be found throughout the menisci from the time of birth to a child's first birthday.[23] Around 18 months, which is around the time the menisci become weight-bearing structures, the blood and lymph supply is reduced to the outer 25%-33% of the body of the menisci and the inner portion of the menisci become avascular.[23] This reduction in blood supply after the first year of life is linked to the transition of establishing a bipedal gait pattern. During the first year of an infant's life, the menisci do not experience a significant amount of weight bearing or muscular force, and therefore the inner portion of the menisci cannot rely on diffusion from the synovial fluid which is mainly responsible for providing nutrition to the inner zone of the meniscus during the earlier stages of life.[23]

Vascularization of the menisci occur via the medial, lateral and middle geniculate arteries. These arteries are the major vascular supply to the inferior and superior aspects of each meniscus.[23] Unfortunately, only 10% to 30% of the peripheral medial meniscus borders and 10% to 25% of the lateral meniscus borders receive direct blood supply. Therefore, the menisci of the knee have been divided into regions or zones based on their vascularity.[23] The peripheral or vascular region of the meniscus has been referred to as the red-red zone of the meniscus. Injuries in the peripheral region of the meniscus are capable of healing and will produce an inflammatory response.[1,23] In this region of the meniscus a fibrin clot packed with inflammatory cells is formed during injury. This attracts mesenchymal cells from the outside which can develop into fibroblasts and chondrocytes. Overall, this leads to a sealing of the meniscal defect and potential healing.[23] The second region of the meniscus is referred to as the inner or avascular region of the meniscus and has also been referred to as the white-white zone. Injuries in the avascular regions of the meniscus are incapable of repair and remodeling because there is no blood supply and therefore no inflammatory response.[1,23] Between the peripheral and inner regions is a region known as the red-white zone which displays characteristics from both the red-red and the white-white zones of the menisci.[1,23]

Ultimately, the healing capacity of the meniscus is directly correlated to the vascularity of the tissue.[1,23] Thus, injury to the white region is highly susceptible to permanent post-traumatic and degenerative lesions with very little chance of healing. Gray reported that if any healing does occur in the white-white or red-white zone, that this healing occurs as a result of vascular access channels from the periphery, abrasion of the synovium, or excision of the outer peripheral rim so that the inner portion can contact the synomium.[23]

Neural Supply

The knee joint is innervated by the posterior articular branch of the posterior tibial nerve and the terminal branches of the obturator and femoral nerves. These nerve fibers penetrate the joint capsule and supply the substance of the menisci.[19] Three types of mechanoreceptors have been identified in the tibiofemoral joint capsule and they are: type I ruffini mechanoreceptors, type II pacinian mechanoreceptors, and type III golgi mechanoreceptors.[19] Type I (Ruffini) mechanoreceptors are low threshold and slowly adapt to changes in static joint position and pressure, type II (Pacinian) mechanoreceptors are low threshold and fast adapting to tension changes, mainly signaling joint acceleration and type III (Golgi) mechanoreceptors signal when the knee joint approaches the terminal range of motion. The majority of mechanoreceptors (especially Pacinian) are most abundant in the peripheral portion of the meniscus, most specifically, the anterior and posterior horns.[2] It is the combination of the vascularity and neural elements of the menisci that play a crucial role in the type of tears that can occur to the meniscus and helps determine the treatment for the patient.[23]

MENISCAL TEARS

Mechanism of Injury, Epidemiology and Etiology

The underlying mechanism of injury to the meniscus generally include activities that involve cutting or twisting, hyperextension of the knee, and has also been reported during a twisting movement with the knee flexed and the foot planted.[1] On average, the annual occurrence of meniscal tears is 60 to 70 per 100,000 individuals per year.[2] In the United States, meniscal lesions represent the most common intra-articular knee injury, and are the most frequent cause of surgical procedures performed by orthopedic surgeons.[1] Meniscal tears are more common in males than females, with a male to female incidence ratio ranging from approximately 2.5:1 to 4:1.[2] Poehling *et al*[24] reported peak incidence of injury to the meniscus in men between the ages of 21 to 30 and in girls and women 11 to 20 years old. In general, degenerative types of meniscal tears occur most often in men during their 4th, 5th, and 6th decade of life and in women occurs after the 2nd decade of life (see table 1).[1]

Overall, the majority of meniscal tears affect the medial meniscus and tend to involve the posterior horn.[2] They are also likely to occur in conjunction with ACL tears. The reported incidence of meniscal injury varies considerably, ranging from 16% to 82% in acute ACL tears and up to 96% in chronic ACL tears. During acute traumatic ACL tears the lateral meniscus is injured more often, whereas the medial meniscus is more likely involved in chronic ACL tears.[25] Meniscal injuries are also frequently observed with a tibial plateau fracture.[2] *Vangsness et al*[26] found meniscal injuries occurred in approximately 47% of patients who had a tibial plateau fracture. These meniscal injuries were observed during arthroscopic surgery and repaired at the same time.[26] Femoral shaft fractures have also been associated with concurrent meniscal injury.[2]

Degenerative tears of the meniscus can be found in as much as 60% of the population over age 65. The majority of these tears are asymptomatic and occur in association with degenerative joint disease.[2] The reported prevalence of meniscal tears in patients with clinical and radiographic findings of degenerative joint disease is 68%-90%. These two concomitant injuries pose diagnostic problems for physicians when identifying the main pathology causing the symptoms at the tibiofemoral joint.[1] On some occasions, symptoms that may be caused by degenerative joint disease may be attributed by a physician to the presence of a meniscal tear and vice versa. This has some repercussions on the choice of proper therapy for each injury.[1] The high prevalence of meniscal injuries and degenerative joint disease most likely correlates with the normal alterations in collagen fiber orientation that occur with age.[2]

Younger patients however, are more likely to have an acute traumatic event as the cause of their meniscal pathology.[2] In this group of patients, sports-related injuries are the most common cause of meniscal injuries, accounting for more than ⅓ of all cases. In sports related injuries, 80% of meniscal injuries are also accompanied by an ACL injury.[1]

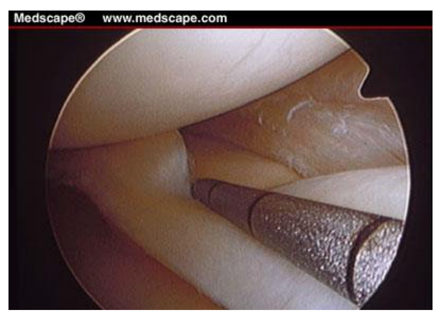
Tear Types

Meniscal injuries can be classified based on their appearance, location, shape, extent and origin. Most meniscal tears are either partial or full thickness tears and can be stable or unstable. An unstable tear is most serious, it is when the entire tear or a portion of the tear can be displaced into the joint space.[27] There it may become trapped, causing an increased amount of joint pain along with catching, locking, or effusion of the tibiofemoral joint.[1,2,27]

Additionally, meniscal injuries can be further classified based on their tear patterns. The most commonly described patterns of meniscal tears include vertical longitudinal, oblique, complex, transverse and horizontal (see Table 1 for definitions).

Table 1. Classification and Types of Meniscal Tears

Name of Tear	Definition of Tear	Image of tear
Degenerative Tear	Term used to describe tears that arise predominantly due to degenerative processes and include horizontal, flap and complex tears as well as meniscal degeneration and destruction.[33] Degenerative lesions are among the most common of the meniscal lesions.[28] Appearance at arthroscopy is highly variable and includes fraying of the free edge, central degeneration, centrally located horizontal tears, fringe tags, degenerative flap tears and extensive fibrillation of the entire meniscus.[28]	
Traumatic Tears	Term used to describe tears that are believed to arise predominantly as a result of a specific, traumatic injury event and include vertical, bucket handle and radial tears. Traumatic tears normally occur in younger sports-active people with or without associated cruciate ligament injury; the meniscus typically splits in a longitudinal direction. The central part of the meniscus may dislocate centrally and cause	

	locking of the knee. Traumatic injury is considered more frequent in the medial meniscus.[29]	
Vertical or Longitudinal tears	Vertical or longitudinal tears occur in the line with the circumferential fibres of the meniscus and parallel to the outer margin of the meniscus. Most longitudinal tears of the medial meniscus occur in the middle and posterior thirds.[32] In the lateral meniscus it is common to find acute, complete or incomplete longitudinal tears of the posterior horn when the ACL is ruptured.	
Peripheral tears	Peripheral tears are vertical or longitudinal tears, found in the peripheral one third of the meniscus. Stable vertical longitudinal tears which tend to be found in the peripheral, vascular portions of the menisci have good potential for self-healing.[29]	
Bucket handle tears	An exaggerated form of a longitudinal tear where a portion of the meniscus becomes detached from the tibia and the end result is a dislocated central part of the meniscus looking like a bucket handle. They are 3 times more common in the medial meniscus than the lateral and may be associated with acute ACL tears. Bucket handle tears are commonly seen in young adults with a history of locking, extension block or slipping of the joint.[30]	

Radial tears	Vertical tears which also occur often at the junction of the posterior and middle thirds and extend from the inner free margin toward the periphery. They are more common in the lateral meniscus and are often associated with ACL tears.[30] If they reach the periphery it transects the entire meniscus. Radial tears which occur in the avascular inner one-third of the meniscus have little potential for healing. They are commonly traumatic and occur in younger, physically active patients.[29]	
Horizontal	Cleavage or fish-mouth tears are common in older people and extend from the inner free margin to the intrameniscal substance where degeneration may be present. These tears divide the meniscus into superior and inferior flaps. They have little or no healing capacity.	
Oblique tears	Are also known as flap or parrot beak tears; they are oblique vertical cleavage tears and usually occur at the junction of the posterior and middle thirds of the meniscus.	
Displaced tears	Fragments of a torn meniscus partially attached to the meniscus and migrating to any position within the joint. Displaced meniscal fragments are often clinically significant lesions requiring surgery because of pain and knee locking. Any shape of a meniscal tear can result in a displaced fragment.[30]	

*All images used in Table one taken from Fan and Ryu[31]

Of the types of tears listed in Table 1, the most common tear pattern is the oblique or vertical longitudinal tear, which accounts for approximately 81% of meniscal injuries.[27] The following tears have been associated with a traumatic mechanism of injury: longitudinal, bucket handle and radial. Longitudinal tears can be complete, known as bucket handle tears, or incomplete and most often occur in younger individuals. These tears are most commonly associated with an ACL injury as well.[27] Bucket handle tears usually begin in the posterior horn and can vary in length from starting at approximately 1 cm to greater than two thirds of the entire meniscus. They are often unstable and cause common mechanical symptoms such as locking or stiffness of the tibiofemoral joint.[27] The medial meniscus is most susceptible to these types of tears because it has such a secure attachment to the tibial plateau, which makes it most susceptible to shear injury. Incomplete longitudinal tears also affect the posterior horn of the meniscus and can be found on both the superior and inferior surfaces of the meniscus.[27] These tears may or may not be symptomatic, and can be found during arthroscopy during probing of the meniscus. Lastly, transverse or radial tears occur in isolation or in conjunction with other tears.[27] They are typically located at the junction of the posterior and middle thirds of the medial meniscus or near the posterior attachment of the lateral meniscus. They may be asymptomatic but can span across the entire meniscus if the edges catch within the joint.[27] Complete radial tears disrupt the circumferential fibers of the meniscus and result in a loss of load-bearing function.[27]

Degenerative meniscal tears are more frequently observed with increasing age and occur most often near the posterior horns.[27] Meniscal tears that are most often associated with a degenerative process include: oblique, horizontal and complex tears. Oblique tears often referred to as flap or parrot beak tears, can occur at any location but are most often found at the junction of the posterior and middle thirds of the meniscus.[27]

The symptoms for this type of tear may result from the free torn edge of the flap catching in the joint and producing a locking or catching feeling. Horizontal tears are believed to begin near the inner margin of the meniscus and extend toward the capsule.[27] They tend to occur in the plane of the horizontally oriented middle perforating collagen fiber bundles and are thought to be the result of shear forces generated by axial compression. They are capable of occurring in all age groups, but tend to increase in frequency with increasing age.[27] A common complication associated with horizontal tears are meniscal cysts. Meniscal cysts can be symptomatic because of localized swelling and represent between 1% to 10% of meniscal pathology. They are highly correlated with meniscal tears and most often occur in the lateral meniscus.[2,27] These cysts appear directly connected to the meniscus and are filled with gel-like material biochemically similar to synovial fluid. They can be very painful and may produce symptoms that include joint line pain, and the cysts are often palpable on physical examination at or below the joint line.[2,27] Lastly, complex or degenerative tears occur in multiple planes and are more common in older age groups, approximately 40 years of age and older. They commonly occur in the posterior horn and mid body of the meniscus, and are often associated with degenerative changes of articular cartilage in the knee. They also represent part of the pathology known as degenerative arthritis.[27]

Based on the types of tears that can occur to the menisci of the tibiofemoral joint and vascularity considerations in each zone of the meniscus, possible treatment options can include: no surgery/conservative therapy only, meniscal repair, partial meniscectomy and complete meniscectomy.[27] If surgical intervention is necessary, surgeons attempt to preserve as much of the meniscal tissue as possible to reduce the chances of developing degenerative changes at the tibiofemoral joint.[27] However, before surgery is indicated a thorough evaluation of the patient is warranted.

Diagnosis of Injury

The diagnosis of a meniscal injury can be made through many different methods. Those methods include taking a careful history, a physical examination which include using clinical special tests to diagnose a tear, and imaging tests.[2] A patient with a meniscal pathology will typically present with symptoms referable to the joint line, either medially or laterally.[27] Symptoms of a meniscal injury are frequently worsened by flexing and loading the knee, and activities such as squatting and kneeling are poorly tolerated. Patients will frequently complain of a pop or clunk sensation as the knee is brought through the full range of motion.[27] An effusion in the tibiofemoral joint may or may not be present following injury. Most often if an effusion is present clinicians will observe swelling or bogginess in the area of point tenderness, particularly when the knee is in flexion.[27]

Given the extent of the signs and symptoms that may be present following a meniscal injury and incidence rate of meniscal tears in an athletic population it is important for clinicians to have a deep understanding of valid clinical special tests. There are a number of clinical tests available for clinicians to perform if a meniscal pathology is in question. These tests include the joint line tenderness test, McMurray's Test, Apley's test, and Thessaly's test.[32] One of the major issues that currently exists when using clinical special tests to diagnose a meniscal tear is that it can be difficult to diagnose as the symptoms may be nonspecific and other concomitant injuries (ACL, degenerative joint disease) can be disguised as a tear in the meniscus. Therefore, clinicians must choose clinical special tests that are accurate and have high sensitivity and specificity.[33]

The sensitivity of a clinical test refers to the proportion of patients with a meniscal tear who are correctly identified as positive by the clinical special test and the specificity of a clinical test refers the proportion of patients without a meniscal tear who are correctly identified as negative by the test.[33] Therefore, a highly sensitive test when negative will rule out the presence of a meniscal injury and a highly specific test when positive will help rule in the presence of a meniscal injury.[33] However, as the sensitivity and specificity decrease then the clinician is less sure if the special test they are choosing is truly a good clinical test to use when compared to the gold standard, which in most cases is MRI but can also be arthroscopy in patients with knee injury.[33] The sensitivity and specificity values for the most common clinical special tests are listed in Table 2.

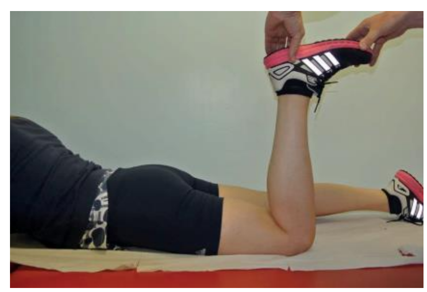
Table 2. Sensitivity and Specificity for Meniscal Special tests

Special Tests	**Sensitivity (95% CI)**	**Specificity (95% CI)**	**Gold Standard**
McMurray*	70.5 (67.4% to 73.4%)	71.1% (69.3% to 72.9%)	Arthroscopy and MRI
Apley*	60.7% (55.7% to 65.5%)	70.2% (68.0% to 72.4%)	Arthroscopy and MRI
Joint-Line Tenderness*	63.3% (60.9% to 65.7%)	77.4% (75.6% to 79.1%)	Arthroscopy and MRI
Thessaly Medial‡	89%	97%	Arthroscopy
Thessaly Lateral‡	92%	96%	Arthroscopy

*Information taken from Hegedus *et al*[34]; ‡ Information taken from Karadhalios *et al*[35]

Based on the information provided in table 2, Thessaly's special test has a high specificity and sensitivity. The results in table 2 indicate that the Thessaly's test correctly reports 97% and 96% of the patients without a medial and lateral meniscal tear and 89%-92% of patients are detected as having a meniscal tear when this test is used. Thus, if the Thessaly's test is positive then we are sure they have a meniscal tear and if it is negative we are sure they do not. This information can be applied to all of the special tests included in Table 2, however, the two clinical special tests that would be best used to diagnose a meniscal tear are Thessaly's and Joint Line Tenderness test (see special test figures and descriptions below in Table 3).

Table 3. Images and Descriptions of Common Meniscal Special Tests[33]

Special Test Name	Special Test Description	Special Test Image
McMurray's	Designed to detect tears in the posterior portion of the meniscus. A test is considered positive when a click can be heard and/or felt on joint line palpation when the knee is bent beyond 90° flexion and the tibia is rotated on the femur into full internal rotation then full external rotation (to test the lateral and medial meniscus respectively).	
Apley's	This test is carried out with the patient prone and the knee flexed to 90°. The tibia is then compressed onto the knee joint while being externally rotated. If this maneuver produces pain, this constitutes a positive test	
Joint Line Tenderness Test	Involves palpation of the joint line with the knee in 90° of flexion. The test is considered positive when there is pain along the joint line on palpation.	

Thessaly's Test	When performed at 20° of knee flexion it is reported to have a high diagnostic accuracy rate for detecting both lateral and medial meniscal tears. The Thessaly Test is a dynamic reproduction of joint loading in the knee. The examiner supports the patient by holding the patient's outstretched hands while he/she stands flat footed on the floor. The patient then rotates his or her knee and body, internally and externally, three times, keeping the knee in slight flexion (20°)	

Once a clinician performs a thorough history and physical examination the patient is usually referred to a physician to confirm the clinical diagnosis. Most often physicians rely on plain radiographs, arthrography, magnetic resonance imaging (MRI), and arthroscopy to confirm the clinical diagnosis.[2] Plain radiographs are generally not helpful in evaluating meniscal lesions other than to rule out other bony or joint pathology, thus they are not often utilized when evaluating a meniscal pathology. The major benefit to the patient having radiographs is that they allow for the defining of bony pathology and the assessment of knee joint narrowing. Most often a standard series of radiographs will include a 30° to 45° knee flexion weight bearing view, a true lateral view, and a merchant view.[23] Arthrography, which is in the injection of a contrast dye into the tibiofemoral joint has been used extensively in the past with reported accuracy rate of 60% to 97%.[26] The primary disadvantage of arthrography is its invasive nature. Arthrography today has been largely replaced by MRIs, which provides accuracy rates as high as 90% to 98%.[27] The advantages of using MRIs in patients with suspected meniscal injuries include its noninvasive nature, the ability to assess the knee in multiple planes, the absence of ionizing radiation, and the ability to assess multiple structures within the tibiofemoral joint. One of the major drawbacks to using an MRI to assess meniscal injuries is its relatively high cost.[2] The normal appearance of the meniscus on MRI is that of a uniformly low signal. Areas of increased signal are frequently seen with meniscal tears. When a physician interprets the MRI they are using a meniscus grading system (see figure11), which delineates grades 0, I, II, III.[2] Only grade III changes are consistent with meniscal tearing. While the MRI is relatively accurate diagnostic tool to use in the assessment of meniscus, the gold standard for confirming the diagnosis of meniscal tear is arthroscopic examination.[2] During arthroscopy, the tibiofemoral joint along with the menisci can be probed and the superior and inferior surfaces examined. The physician can also examine the posteromedial and posterolateral compartments to assure tears in the peripheral posterior horns are not missed. Ultimately, if the arthroscopic procedure is performed systematically, then the diagnosis of a meniscus pathology should be definitive.[2]

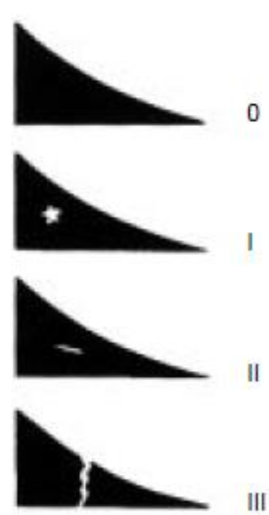


Figure 11. Grading scale for meniscal tears[2]

Non-Operative Treatment

Following a diagnosis of meniscal pathology, the patient has two treatment options: conservative or surgical treatment. No matter the chosen treatment option the ultimate goals is the same, to relieve acute symptomatology, and limit future recurrence. Most often nonoperative treatment is the mainstay of treatment following meniscal injury, while surgical procedures are reserved for patients with symptoms that are resistant to conservative management.[25] The initial focus of nonoperative therapy is the relief of knee pain. The severity of symptoms can vary from patient to patient and the pattern may change based on the type of meniscal tear. If the symptoms at the knee are infrequent and there is no severe damage to the meniscal tissue, then a clinician may implement an initial period of conservative management for the patient depending on their activity level and their individual demands.[26] There are many different conservative or nonoperative approaches that can be implemented for patients with a meniscal tear. The first form of nonoperative treatments are aimed at regulating swelling and providing some pain relief to the patient. These treatments include the use of Cryotherapy and Nonsteroidal anti-inflammatory Drugs (NSAIDs), which are most often used on an as needed basis.[25,26] If oral NSAIDs are not effective at reducing the pain and swelling associated with a meniscus injury then the physician may elect to perform a direct injection of corticosteroids into the tibiofemoral joint to control irritation and improve the patient’s activities of daily living.[26]

If the first approach to a meniscus tear is nonoperative treatment the patient is encouraged to limit activities that exacerbate the symptoms, however they must be active because complete rest is not advised.[26] If a patient becomes sedentary during the nonoperative treatment there is an increased risk for tibiofemoral joint stiffness. Since the main goal of nonoperative treatment is to restore or maintain range of motion, increase or maintain strength of the quadriceps and hip complex, improve flexibility of the hamstrings and retain knee proprioception, physical therapy and rehabilitation become the central aspect of nonoperative treatment in patients with meniscal tears.[25] It has consistently been reported that a supervised exercise regimen that includes gait therapy, quadriceps strengthening, hamstring flexibility, static cycling and dynamic neuromuscular training has led to improved pain and quality of life scores, and improved function following 8-12 weeks of a supervised and home exercise program.[25,26,27]

The success of the nonoperative treatment is most often dependent on the demands and motivation of the patient as well as the severity of the meniscal tear. While nonoperative therapy can initially be beneficial at reducing patient symptoms approximately one-third of patients will go on to have a meniscectomy.[26] This procedure is most often done if there is no symptom relief following nonoperative treatment.[26]

SURGICAL TREATMENT

Meniscus Repair

Given the functional importance of the menisci in the tibiofemoral joint, physicians have an increasing interest in avoiding meniscectomy and when possible will choose to surgically repair the meniscus, however, the location of the tear is critical when determining which surgical technique will be used for the patient. Therefore, it is the blood supply to the area being repaired that determines the success of a meniscal repair. When tears occur in the vascular region of the meniscus (red-red or red-white zones) physicians will perform meniscus repair.[27] However, current techniques have somewhat pushed this limit to allow some tears of the central and middle ⅔ of the meniscus to be repaired. Also, if vascularity of the meniscus is seen with observable bleeding in the area of the lesion, a repair should definitely be considered. Although the peripheral ⅓ of the meniscus is the general criteria for a meniscal repair, there are some new studies that have challenged this idea.[27] Attempts have been made to encourage bleeding in otherwise avascular zones by using exogenous fibrin clots to stimulate a reparative response or trephination which is the use of a needle to puncture the joint lining and the substance of the meniscus to try and stimulate healing at that location.[27] The goals of both of these treatments is to try and stimulate a reparative process by creating a blood clot in the injured area. In addition to creating blood clots in the injured area, Cannon and Vittori reported that patients with meniscal repair in conjunction with ACL reconstruction had a 93% healing rate of the meniscus when compared to 50% healing rate in patients undergoing meniscal repair alone.[36] The authors of this study attributed the increased meniscal healing rates in those with ACL reconstruction following a repair to the tibial and femoral drilling that occurs during an ACL reconstruction, which they believed resulted in the delivery of local growth and clotting factors.[36]

Although blood supply to the injured meniscus is extremely important in the decision making process for treatment, there are many other factors that must also be considered when trying to determine the best surgical intervention for the patient. The type, length, and the stability of the lesion also plays an extremely important role when considering the surgical intervention the physician will use.[37] Please see table 4 below to determine what types of tears would benefit most from surgical repair.

Table 4. Tears that require or do not require surgical intervention[38]

Requires Surgical Repair	Requires No Surgical Intervention
• Longitudinal or vertical tear • Radial tears at the posterior horn • simple acute bucket handle tears	• Stable tears less than 1 cm in length • Longitudinal tears and partial thickness tears of various types that are stable and located within the peripheral ⅔ can be left alone especially if they are less than 5 mm in length • Tears where the circumferential hoop fibers are intact • Short inner radial tears that are < 5 mm usually do not heal, but can often be left untreated because the are usually asymptomatic • Bucket handle tears that are complex with radial components

Meniscal repair can be performed either with an open or arthroscopic technique. The standard of care is closed techniques to perform a meniscal repair.[27,38] The only current indications for an open repair technique are a posterior medial meniscus tear encountered in a very tight medial compartment.[37] During an open procedure the physician makes a small incision through the capsule and synovium of the knee so that the tear can be carefully observed.[37] While we have transitioned away from an open surgical repair the long term results following the procedure have been relatively positive. Muellner *et al*[39] reported a success rate of open repaired menisci at 91% after a mean follow up of almost 13 years, and of 22 patients only two had retears. Additionally, Rockborn and Gillquist[40] compared the results of 31 patients who underwent open repair with a matched group of healthy subjects. After a 13-year follow up 80% of patients had normal knee function for daily activities, and the incidence of low-grade radiologic changes was similar between both groups.[41] Overall, the open surgical meniscal repair technique has favorable results in the long-term however, it is rarely performed due to associated neurovascular injury and high morbidity. The major advancements made by arthroscopic surgery have made this the standard of care.[27]

Arthroscopic meniscal repair has several advantages over an open repair in terms of minimal incision, early recovery, and rehabilitation. Arthroscopic repair techniques can be divided into 4 categories: inside-out techniques, outside-in techniques, all-inside techniques, and hybrid techniques that combine the multiple techniques listed above.[38] Both inside-out and outside-in repair techniques involve passing a suture from either the inside or the outside of the knee via arthroscopy tying that suture beyond the joint capsule using a small incision.[27] Both of these procedures require several incisions and also require the surgeon to tie the knot either from the inside out or outside in. With advancements in surgical procedures and tools, another surgical procedure that is being used more often is the all-inside technique. During an all-inside technique the physician uses a device to tie the incision inside the knee, therefore, additional incisions are not needed.[27] The advantages of all-inside repair with suture devices include ease of use, avoidance of an accessory incision, shorter operating time, and less risk of injury to neurovascular structures, but it also has its disadvantages.[27] The disadvantages are meniscal or chondral damage from manipulation of the devices, implant migration, foreign body reactions, and higher cost.[38] During assessment of the meniscus and radiographic evidence, the physician will generally choose their surgical technique based on the type and/or location of the meniscal tear. A meniscal tear on the mid-third horn or a peripheral capsule area can be repaired with the inside-out technique. If the tear occurs on the anterior horn or an attempt to reduction of a bucket-handle tear can be performed the best technique to use would be the outside-in technique.[38] Finally, the all inside technique can be used in a meniscal tear on the posterior horns as to reduce the risk of neurovascular compromise.[38]

In general, there is no evidence that suggests that one meniscal repair surgical technique is superior to another. Grant *et al*[41] *p*erformed a systematic review that compared 19 studies looking at different repair techniques for isolated meniscal tears and no differences in clinical failure rate or subjective outcome between inside-out and all-inside meniscus repair techniques. They found that there were complications associated with both techniques. More neurological symptoms were associated with the inside-out repair whereas patients that had the all-inside technique presented with more implant-related complications.[41] Nepple *et al*[42] found similar results in a systematic review of 13 studies with a minimum of five-year follow-up. They found a failure rate from 20.2% to 24.3% for all meniscal repair techniques.[42] When patients are not candidates for surgical repair or when the repair fails the next treatment option available if they have exhausted physical therapy is a meniscectomy.

Meniscectomy and Partial Meniscectomy

Unfortunately, many meniscal tears do not fall into the repairable or spontaneously healing categories because they occur in the avascular region of the meniscus. Therefore, most patients suffering from a meniscal tear must undergo a complete arthroscopic meniscectomy or partial meniscectomy. In fact, arthroscopic partial meniscectomy is the most common orthopedic surgical procedure performed in the United States.[2,27] During an arthroscopic partial meniscectomy, the damaged and fragmented parts of the meniscus are removed with the use of arthroscopic instruments, usually a mechanized shaver and meniscal punches, until the solid meniscal tissue is reached. The meniscus is then probed to ensure that all the loose and unstable fragments have been successfully resected with as much preservation of meniscus tissue as possible.[43] This technique has been considered the gold-standard treatment for meniscal injuries for several years. However, complete or partial loss of a meniscus can have damaging effects on the tibiofemoral joint, leading to serious and irreversible long-term effects.[1,27,42,43,44]

Baratz *et al*[44] studied the effects of a total meniscectomy on contact areas and the stresses in the knee joints of human cadavers and reported that loss of the medial meniscus led to a decrease in contact area of approximately 75% and an increase in the peak contact pressure of approximately 235%.In a long term biomechanical study, Roos *et al*[45] reported a positive correlation between increased peak contact pressure and radiographic evidence of osteoarthritis (OA) development following total meniscectomy. They also reported a greater risk of developing OA following a total meniscectomy on the lateral side when compared to the medial side. The increase in OA on the lateral side is due to greater coverage and load distribution performed by the lateral meniscus.[45] Also, increased intra-articular contact stresses within the knee after a meniscectomy and partial meniscectomy tend to overload the articular cartilage of the tibiofemoral joint, which leads to a reduction of proteoglycan, disaggregation of proteoglycan which can ultimately lead to failure of the articular cartilage causing the joint to become more susceptible to osteoarthritic changes.[46] Considering the drastic changes that occur to the tibiofemoral joint following a total meniscectomy it becomes vitally important to preserve as much meniscal tissue as is possible during arthroscopic surgery. In contrast to the total meniscectomy which completely removes all of the meniscus, a partial meniscectomy aims to only remove the piece of the meniscus that is torn while retaining as much meniscal tissue as possible.[43,44,45,46]

Several studies have been conducted to compare patient outcomes following a total and partial meniscectomy. These outcomes generally focus on patient function and satisfaction and the development of OA following their surgical procedures. It has been reported that 2-4 years following a partial meniscectomy 85%-90% of patients report satisfaction following the surgery and only 68% of patients that had a total meniscectomy reported satisfaction.[27] The patient satisfaction results presented above only give the clinician a short-term view, however, when we examine the long term implications of meniscectomies we begin to see the irreversible effects that the surgery has on the patient. While partial meniscectomies are the preferred surgical intervention of choice when compared to a total meniscectomy it has been accepted that partial meniscectomy, while in the short term improves patient satisfaction, only delays the onset of OA but does not prevent it from occurring. During an 8.5 year follow up in 131 patients who had partial meniscectomies the reoperation rate was 23% with 53% of the patients presenting with osteoarthritic radiographic changes.[47] In another long-term follow up study of 147 athletes who had partial meniscectomies 50% of the patients reported being asymptomatic at 4.5 years post-surgery while only 30% of patients reported being asymptomatic at 14 years follow up.[48] This same trend was observed during radiographic evaluation where only 40% of the patients presented with radiographic evidence of degeneration at 4.5 year follow up and 89% of the patients presented with degenerative findings at 14 year follow up. What is most alarming in this athletic population is that 46% of the patients reported giving up or reducing their participation in activity.[48] Lastly, Stein *et al*[49] examined the long-term effects of meniscal repair and meniscectomy in patients that suffered from a traumatic meniscal tear. Eighty-one patients were included in this study and assigned to repair or meniscectomy based on the location of their tear.[49] At eight year follow up only 20% of the patients in the repair group presented with radiographic evidence of OA when compared to 60% of patients in the meniscectomy group who had radiographic signs of degeneration. The Stein *et al*[49] study also reported that 96% of patients who had meniscal repair returned to pre-activity level when compared to only 50% of patients in the meniscectomy group.

Based on a complete review of the traditional treatment options available to patients who have a meniscal injury, it is important that physicians consider several factors before determining the best treatment. Mordecai *et al*[27] presented a treatment tree based on a review of the literature and outcomes following surgery. Please see Figure 12 for their proposed meniscal treatment tree. In meniscal treatment tree, treatment decisions are based on three factors: presence of degeneration during initial assessment, location of the tear, and the age of the patient. These are all factors that have been mentioned throughout this review of literature to impact the type of long-term outcomes of the surgical procedures.

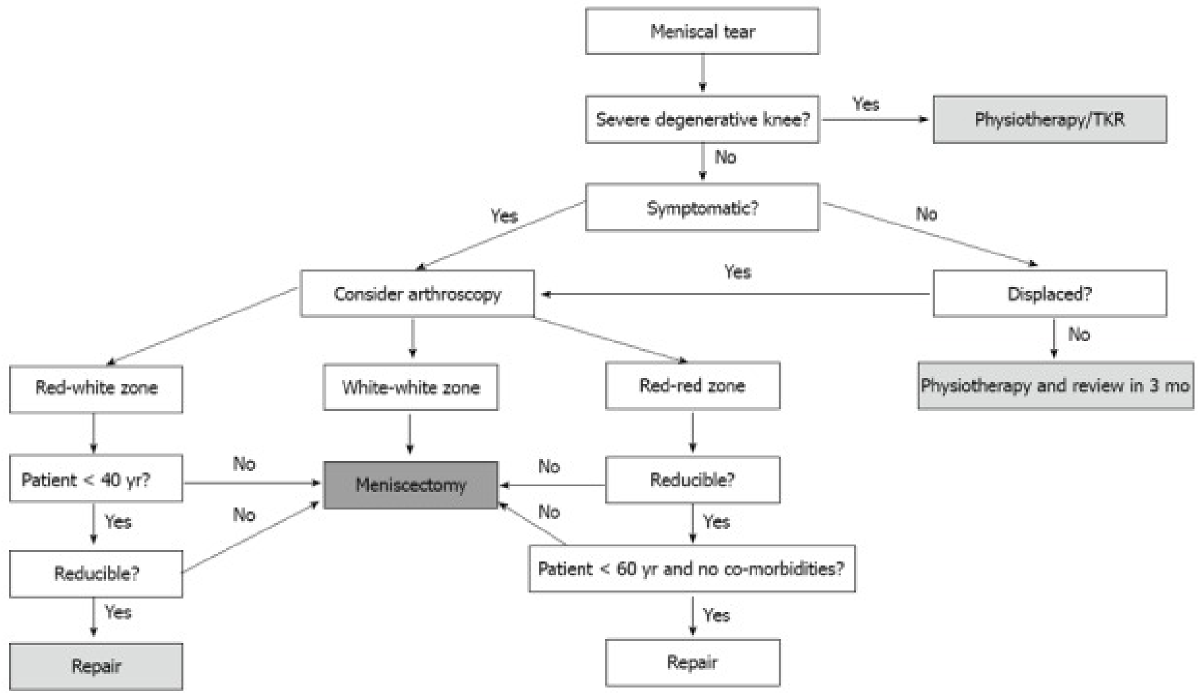


Figure 12. Meniscal Treatment Tree[27]

OSTEOARTHRITIS

Epidemiology and Pathophysiology

Osteoarthritis (OA) is the most common form of arthritis, and is a major cause of morbidity, activity limitation, physical disability, and reduced health-related quality of life in people aged 45 and above.[50] Currently, OA is the single most common cause of disability in older adults and impacts females more than males.[50] It has been estimated that by 2050, 130 million people will suffer from OA worldwide and of those 40 million will be severely disabled by the disease.[50] The most common joints affected by OA are the knee and hip, with the knee being affected most often. While OA in general has been linked to aging, it is also associated with modifiable and non-modifiable risk factors, including, obesity, lack of exercise, genetic predisposition, bone density.[50,51]

OA has been defined as a progressive disease representing the failed repair process of damaged articular cartilage due to biomechanical and biochemical changes in the joint.[52] In this disease all tissues of the joint are involved and include not only the articular cartilage, but also the subchondral bone, ligaments, periarticular structures, and menisci when present.[53] Ultimately, the results of OA are cartilage degradation and bone remolding, which are associated with increased joint stiffness, pain, and functional disability. These symptoms, which are often present in patients with OA have profound implications for treatment of the disease. While physicians have managed to treat some of the symptoms of OA with pharmacological aids, there are currently no treatments approved for slowing the structural progression of OA.[54]

Nearly half of patients with radiological features of OA have no symptoms, therefore diagnosis of the disease relies heavily on clinical and radiological features.[53] One of the major issues with OA is that it develops progressively over several years and symptoms remain stable for long periods within this time frame. One reason for the delayed response to articular damage has to do with the lack of neural and blood supply to the cartilage. Articular cartilage is non-vascularized, so this restricts the supply of oxygen and nutrients to the chondrocytes, the cells that are responsible for the maintenance of a very large amount of extracellular matrix.[52,54] At an early stage, in an attempt to initiate a repair, clusters of chondrocytes form in the damaged areas and the concentration of growth factors in the matrix rises. This attempt subsequently fails and leads to an imbalance in favor of degradation. Increased synthesis of tissue-destructive proteinases as well as increased apoptotic death of chondrocytes and inadequate synthesis of components of the extracellular matrix lead to the formation of a matrix that is unable to withstand normal mechanical stresses.[52,54] As a result, the tissue enters a vicious cycle in which breakdown dominates synthesis of the extracellular matrix. Additionally, articular cartilage is aneural so the changes that are occurring to the joint do not produce clinical signs and symptoms until the innervated tissues become involved.[52,54] The lack of neural input in the articular cartilage is one of the main reasons for the late stage diagnoses of the OA.[55]

Although the pathophysiology of osteoarthritis has long been thought to be cartilage driven, recent evidence shows an addition and integrated role of bone and synovial tissue, and patchy chronic synovitis is evident in the disease. Synovial inflammation corresponds to clinical symptoms such as joint swelling and inflammatory pain, and it is thought to be secondary to cartilage debris and catabolic mediators entering the synovial cavity. Synovial macrophages produce catabolic and proinflammatory mediators and inflammation starts negatively affecting the balance of cartilage matrix degradation and repair.[52,54,55] As a result, this process amplifies the synovial inflammation creating a vicious cycle. Synovial inflammation happens in early and late phases of osteoarthritis, and as result leads to the progressive joint degeneration.[52,54,55] Overall, the main characteristics of osteoarthritis are changes to the subchondral bone. Osteophyte formation, bone remodeling, subchondral sclerosis, and attrition are crucial for radiological diagnosis.[52] Several of these boney changes take place not only during the final stage of the disease, but also at the onset of the disease- possibly before the cartilage begins to degrade. Unfortunately, it takes time for many of these symptoms to become bothersome in patients. This is another reason that late diagnosis of OA is common and as a result treatment options are limited.[52]

Treatments

Currently there is no cure for OA and treatment is aimed at improving quality of life by targeting symptoms and improving or trying to maintain function at the joint. Therapeutic options for knee OA include nonpharmacologic, pharmacologic, and surgical interventions.[55] Nonpharmacologic measures such as weight loss, muscle-strengthening exercises, and joint protection techniques have no inherent risk and minimal costs, and are therefore advised for all patients.[55] In a passive approach to managing the disease, Zang *et al* [56,57]reported that simply providing the patient with information on the disease, its symptoms and the importance of lifestyle changes lead to improvements in symptoms. Another nonpharmacologic treatment that has been affective at reducing symptoms of knee OA include muscle strengthening, flexibility, balance and aerobic activities.[58] Other nonpharmacologic treatments that have been suggested to reduce the symptoms associated with knee OA include: knee braces, shoe insoles, electrical modalities and acupuncture, however, the evidence on the efficacy of these treatments is very scarce.[55]

Pharmacologic therapies including acetaminophen, anti-inflammatory drugs, opioid derivatives, glucosamine, chondroitin sulfate, and capsaicin, all have modest efficacy in decreasing the pain associated with knee OA. Of these treatments, NSAIDs have been deemed most effective by patients and are most often prescribed by physicians.[55] However, long term NSAID treatment has been associated with clinically important increases in the risk of cardiovascular disease- the predominant cause of death in patients with knee OA.[52,54]The use of weaker opioid analgesics (codeine, tramadol) for the treatment of OA has increased, however, patients have not reported greater improvements in OA symptoms when taking weaker opioids when compared to NSAIDs unless they were taking strong opioids (oxycodone, fentanyl). Patients have also ingested glucosamine sulfate and chondroitin in an effort to help reduce the symptoms of OA, however, there is very little evidence to draw any clinical conclusions regarding the efficacy of these drugs and the reduction of OA symptoms.[59]

New and Innovative Meniscal Treatments

Overall, meniscectomies have been proven to be beneficial in reducing short term pain and discomfort, however, removing part of the meniscus alters the biomechanical properties of the knee. This ultimately leads to chronic pain, malalignment of the tibia and femur, and joint disability. There is also a high correlation between partial meniscectomy and the development of osteoarthritis at the tibiofemoral joint.[60] The lack of a current treatment to slow down the progression of the disease has led to a shift in the scientific research and interest in the fields of biomaterials and bioengineering, where it is hoped that meniscal deterioration can be overcome through tissue engineering.[2] Tissue engineering offers a unique, interdisciplinary approach to medicine. The disciplines of biology, chemistry, physics, and engineering interact in an effort to artificially generate tissue.[60] The goal of tissue engineering is to establish new meniscal repair techniques that can restore mechanical function to the tibiofemoral joint following a meniscal injury.[2] One of the key components to successful tissue engineering is the type of tissue being engineered or created. There are currently four cell sources that have been used in tissue engineering and they include: autologous, allogeneic, xenogenic, and stem cells.

Autologous

In an autologous procedure cells from the patient's meniscus or chondrocytes from the area are biopsied and engineered and then implanted into the patient once the tissue engineering has occurred.[2] Following this autologous procedure the engineered tissue has been reported to produce more glycosaminoglycans and type II collagen.[2] One advantage to this procedure is that the cells being used for engineering come from the patient and there is less risk of tissue rejection. However, several disadvantages exist: 1) this form of tissue engineering requires two surgeries (one to remove cells and another to implant engineered tissue) and 2) the cells being used to reengineer the tissue may already be in a state of disease or degeneration depending on the stages of OA.[2]

Allogeneic and Xenogeneic Cells

Given the high risk of using unhealthy cells in an autologous procedure an allogeneic procedure offers an alternative to tissue engineering in patients following a meniscal injury. In an allogeneic procedure, meniscal and/or chondrocyte cells are taken from donor and implanted into the unhealthy meniscus.[2] Unfortunately, the current research in tissue engineering and allogeneic implants is currently in the animal literature. However, the results of these large animal studies were encouraging as to healing outcomes in the avascular zones of the meniscus of pigs.[61] Another form of allogeneic treatment is the use of xenogeneic cell sources. During tissue engineering with xenogeneic cell sources the tissue is most often reengineered using Mesenchymal stromal cells (MSCs).[61] MSCs originating from bone marrow or fat tissue have been used most often in the treatment of OA and would be specifically injected directly into the tibiofemoral joint of a patient with OA.[61] Engineering tissue using MSCs has really been a focus of scientific research because they have been observed to differentiate into many different cells: cartilage, bone, ligaments, muscle, fat, etc. and they contribute to the healing process of injured tissue.[62,63] While the xenogeneic tissue engineering research has not focused specifically on OA at the tibiofemoral joint it has been hypothesized that xenogeneic tissue engineering would be successful. In a rabbit model, Ramallal *et al*[64] created articular cartilage defects in the femoral condyle and reported regeneration of articular cartilage and integration of the cartilage with the native tissue post 24 weeks implantation of pig chondrocytes. While there are only a handful of studies that have used xenogeneic cell sources and none to date in a human model, these results do provide some insight into possible treatments for patients with meniscal tears.

Stem Cells

Human Embryonic Stem cells or Stem cell research is an emerging cell source for fibrocartilage tissue engineering. The use of Stem cells for tissue engineering has become one of the most common forms of cell research because these cells have unlimited proliferative capacity.[2] Like the MSCs, stem cells play an important role in meniscal healing by differentiating and regenerating injured tissue. Unlike MSCs, stem cells do this by producing cytokines and growth factors.[2] However, attempts towards tissue engineering the meniscus using stem cells is still in the infancy phase.[2] One such study that has investigated the potential of stem cells to differentiate into chondrocyte or fibrochondrocyte cells was conducted by Hoben *et al.*[65] In this study, stem cells were cultured with several different growth factors and chondrocyte or fibrochondrocyte cells for 3 weeks. Following three weeks of evaluation the authors of the study reported increases in cell proliferation and glycosaminoglycans. They specifically reported a 9.8-fold increase in type II collagen production, which an important finding since the menisci of the knee are mainly composed of this collagen.[63] If scientists continue to use Stem cell research for tissue engineering and the results are consistent with the Hoben *et al*[63] study, this could lead to restoration and healing of the injured meniscus, specifically in the avascular zone.

Tissue Scaffolding

As discussed in the previous tissue engineering sections of this review, growth of the meniscus can be accomplished by the biopsy of meniscal cells, fibrochrondrocytes and through scaffolding (see Figure below 13). Meniscus scaffolds are most often composed of natural or synthetic biomaterials and can be categorized into two categories: biodegradable scaffolds and permanent scaffolds.[60]

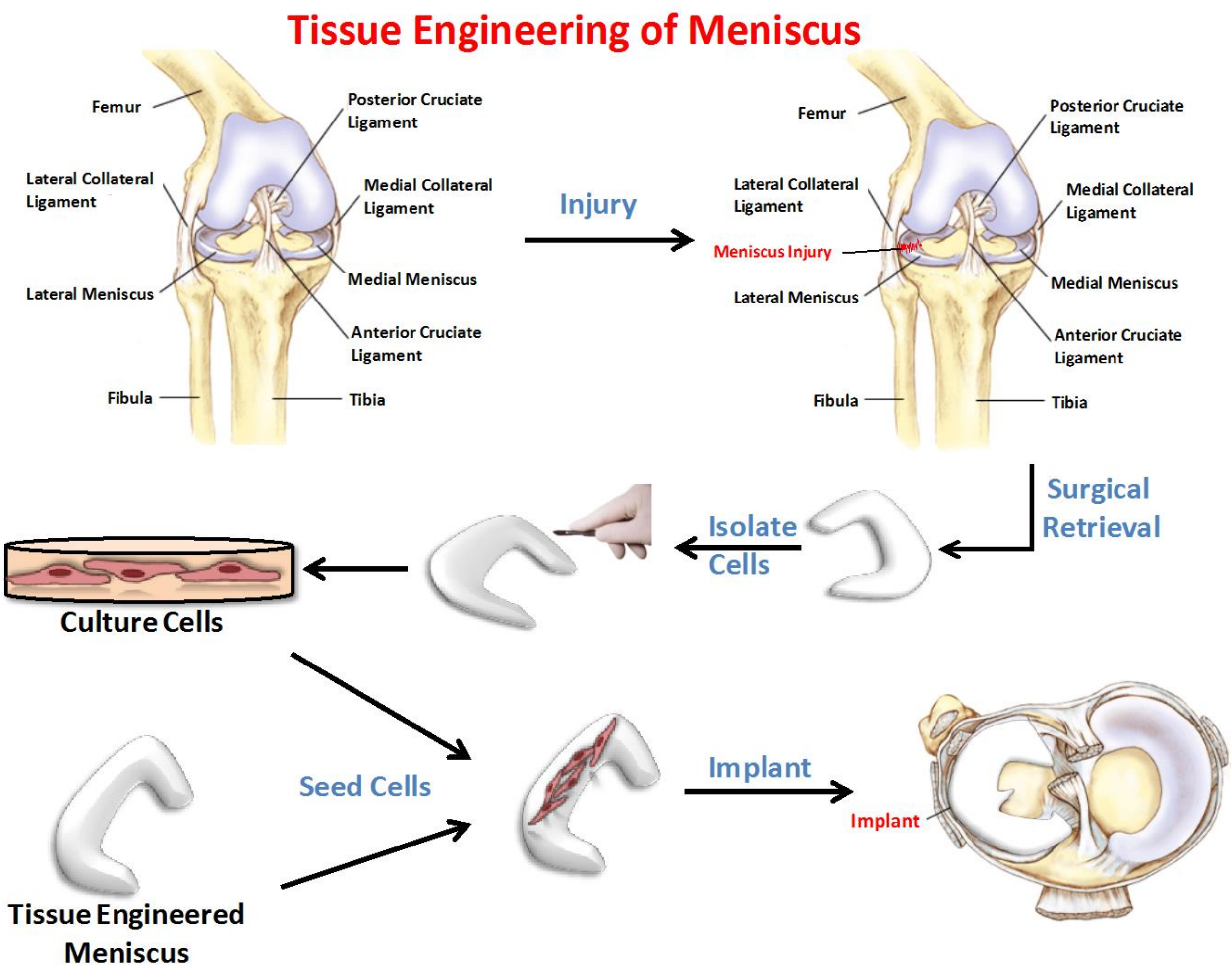


Figure 13. A working model for total meniscus replacement using an autologous seeding procedure.[58]

With biodegradable scaffolds, fibrochondrocytes attach to the natural or synthetic scaffold surface and facilitate cell growth from one tissue to another. This sort of scaffolding lays the groundwork for the production of fibrocartilage and extracellular matrix.[60] Generally, when the tissue becomes integrated and functional, the biomaterial dissolves and is absorbed by the body. Most often, biodegradable scaffolds are used for radial or longitudinal tears to the red-white region of the meniscus. Permanent scaffolds on the other hand are made using synthetic materials, natural components, or a combination of both.[60] These scaffolds act as a structural support for the joint and assume the biomechanical properties of the injured meniscal tissue.[60] Permanent scaffolds are most beneficial for patients who have undergone a complete meniscectomy because they work to relieve the force directed at the tibiofemoral joint.[60]

Natural Biodegradable Scaffolds

Naturally occurring biodegradable polymers are a great option for tissue engineering because of their availability, biocompatibility, and cost efficiency. Two of the most common natural polymers used during tissue engineering using the scaffolding method are silk fibroin and cellulose.[60] Both the natural polymers are biocompatible in the human body and have the capacity for cellular adhesion.[60] One of the major concerns in scaffolding using silk fibroin is the limited mechanical strength when compared to cellulose which is more hydrated and has a higher mechanical strength.[60]

Synthetic Biodegradable Polymer Scaffolds

Synthetic polymers hold several advantages over the natural biodegradable polymers which include fabrication under a variety of methods, limitless supply, and the potential to achieve and take on the mechanical properties of meniscal tissue.[60] One major disadvantage to using synthetic polymers for tissue engineering is they lack the intrinsic biomimetic and bioactive properties that natural polymers have.[60] Synthetic polymers, such as polyesters (polyurethane, polycaprolactone and polylactic acid) display tremendous promise in the field of tissue engineering and are approved by the FDA for medical use.[60] There are two common types of synthetic polymers that have been created and reported in the literature: polyester reinforced collagen and elastomeric polyester.

Polyester reinforced collagen is created when two polyester derivatives are seeded in a scaffold with fibrochondrocytes and cultured.[60] Following culturing this reinforced collagen is implanted into the injured area of the meniscus where it is then attached the perimeniscal tissue for fixation.[66] Following 24 weeks of implantation the scaffold displayed similar circumferential and radially aligned fibrocartilage. These results suggest that a synthetic polymer scaffold may be a viable option for patients who have meniscal tears.[66]

Another synthetic polymer that is currently being studied in tissue engineering is eastomeric polymer. Eastomers have a high stress to strain failure rate, making them a great option for meniscal tissue repair at the tibiofemoral joint. These polymers are characterized by a high elastic profile and can be tailored to match the mechanical properties of natural tissues.[67] One such eastomer is polyurethane which is highly porous and has been used to reconnect torn tissue in the vascular region of the meniscus. Polyurethane's high porosity, modifiable profile and biocompatibility are what make it an attractive material for tissue regeneration in the meniscus.[60,68]

Extracellular Matrix Component and Tissue Derived Scaffolds

Two other types of tissue engineering have been studied and they are extracellular matrix and tissue derived scaffolds. Extracellular scaffolds are materials formed primarily from a macromolecule that exists in the native matrix.[2] Two examples of extracellular matrix scaffolds include collagen meniscal implants and hyaluronan scaffolds. Since meniscus cells normally rest in a dense network of collagen and glycosaminoglycans molecules, scaffolds made from these components should provide a natural environment for them to thrive and regenerate. However, the success of the regeneration is dependent on the type of extracellular matrix molecules that are being used for scaffolding.[2] Unfortunately, not all extracellular matrix molecules are created equal.

When compared to the other scaffolding techniques, extracellular matrix scaffolds have received the most attention due to the use of collagen meniscus implants. The collagen implant is a surgical mesh composed of bovine type I collagen, cross-linked with aldehydes and molded in the shape of a lateral or medial meniscus.[69] When compared to meniscectomy patients, patients that have undergone a collagen meniscal implant have greater tissue restoration 1 year post surgery and report decreased pain and an increase in activity 7 years after implantation.[70] While the short-term and long-term outcomes for collagen meniscal implants are positive there are a few limitations. Currently, the collagen implant is not an option for patients who have undergone a complete meniscectomy, there tends to be a shape incongruency, and surgeons sometimes have difficulty suturing and fixating the implant within the tibiofemoral joint.[71,72]

Currently, a tissue engineering technique has yet to be developed that satisfies the fundamental properties of successful cartilage healing. A successful tissue engineering technique is one in which the appropriate structure-function relationship, extracellular organization, joint biomechanics have all been restored. The existing studies and research show promising results for tissue engineering but further research is still needed. It is important when developing treatments for meniscus injuries to take several variables into consideration. Some being the outline of the indications and contraindications, the selection of suitable patients for tissue repair with engineered meniscus, and the development of non-invasive assessment procedures for generated tissue, both pre-implantation and post-implantation.[2] Directions for future research should also be guided by the minimization of healthcare costs. These variables will be essential to the potential widespread clinical application of utilizing tissue-engineered meniscus as a form of treatment. Although the challenge is vast, recent scientific advances suggest that a solution to this yet unsolvable problem may be emerging from the collaborative efforts of biomedical engineers, clinicians, and industry leaders.[2]

Conclusion

This literature review has provided an in-depth study of healthy tibiofemoral joint anatomy with prominent focus on the menisci. Most importantly, it highlighted the prevalence of meniscal injuries in the general population as well as the problems associated with the treatment options currently available. Currently the gold standard treatment for meniscus injuries that occur in the avascular region is a meniscectomy. Partial meniscectomy provides symptom relief, restores tibiofemoral joint biomechanics, and improves the quality of life for the patient in the short term, however these patients are also at an increased risk of developing osteoarthritis at the tibiofemoral joint.[1,2] Osteoarthritis is an extremely painful and life altering disease so it is important to find alternative treatments that can encourage healing in the avascular zone, restore function at the tibiofemoral joint and/or replicate the function of a healthy meniscus. While meniscectomies continue to be the first choice for surgeons when treating a tear in the avascular zone of the injured meniscus, recent advances in tissue engineering have provided them with alternative treatments. This review of literature has provided evidence that tissue engineering is a promising alternative to meniscectomy however the research in this area needs to progress further before surgeons should consider it as a gold standard treatment. In the meantime, clinicians may focus on the nonpharmcologic treatments that are affective at reducing symptoms and work to help patients improve their quality of life.

REFERENCE LIST

1. Makris, E. A., Hadidi, P., & Athanasiou, K. A. (2011). The knee meniscus: structure–function, pathophysiology, current repair techniques, and prospects for regeneration. *Biomaterials*, *32*(30), 7411-7431.

2. Greis, P. E., Bardana, D. D., Holmstrom, M. C., & Burks, R. T. (2002). Meniscal injury: I. Basic science and evaluation. *Journal of the American Academy of Orthopaedic Surgeons*, *10*(3), 168-176.

3. Moore, K. L., Dalley, A. F., & Agur, A. M. (2014). *Clinically oriented anatomy*. Philadelphia: Wolters Kluwer.

4. 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.

5. Guerrero, J., & Foidart-Dessalle, M. (2008). Kinematics of normal menisci during knee flexion.

6. Woodley, S. J., Latimer, C. P., Meikle, G. R., & Stringer, M. D. (2012). Articularis genus: an anatomic and MRI study in cadavers. *J Bone Joint Surg Am*, *94*(1), 59-67.

7. Koulouris, G., & Connell, D. (2005). Hamstring muscle complex: an imaging review 1. *Radiographics*, *25*(3), 571-586.

8. Woo, S. L., Debski, R. E., Withrow, J. D., & Janaushek, M. A. (1999). Biomechanics of knee ligaments. *The American journal of sports medicine*, *27*(4), 533-543.

9. Fu, F. H., Harner, C. D., Johnson, D. L., Miller, M. D., & Woo, S. L. Y. (1993). Biomechanics of Knee ligaments: basic concepts and clinical application. *JBJS*, *75*(11), 1716-1727.

10. Phisitkul, P., James, S. L., Wolf, B. R., & Amendola, A. (2006). MCL injuries of the knee: current concepts review. *Iowa Orthopaedic Journal*, *26*, 77.

11. Jacobson, K. E., & Chi, F. S. (2006). Evaluation and treatment of medial collateral ligament and medial-sided injuries of the knee. *Sports medicine and arthroscopy review*, *14*(2), 58-66.

12. Petersen, W., & Zantop, T. (2007). Anatomy of the anterior cruciate ligament with regard to its two bundles. *Clinical orthopaedics and related research*, *454*, 35-47.

13. Amis, A. A., Gupte, C. M., Bull, A. M. J., & Edwards, A. (2006). Anatomy of the posterior cruciate ligament and the meniscofemoral ligaments. *Knee Surgery, Sports Traumatology, Arthroscopy*, *14*(3), 257-263.

14. McGinty, G., Irrgang, J. J., & Pezzullo, D. (2000). Biomechanical considerations for rehabilitation of the knee. *Clinical Biomechanics*, *15*(3), 160-166.

15. Kapandji, A. I., Kandel, M. J., & Kapandji, I. A. (1988). *Physiology of the Joints: Lower Limb: Volume 2*. Churchill Livingstone.

16. Blackburn, T. A., & Craig, E. (1980). Knee anatomy: a brief review. *Physical therapy*, *60*(12), 1556-1560.

17. Heegaard J, Leyvraz P, Curnier A, Rakotomanana L, Huiskes R. The biomechanics of the human patella during passive knee flexion. *Journal of Biomechanics*. 1995;28(11):1265-1279. doi:10.1016/0021-9290(95)00059-q.

18. Laasanen, M. S., Töyräs, J., Korhonen, R. K., Rieppo, J., Saarakkala, S., Nieminen, M. T., ... & Jurvelin, J. S. (2003). Biomechanical properties of knee articular cartilage. *Biorheology*, *40*(1, 2, 3), 133-140.

19. Brindle, T., Nyland, J., & Johnson, D. L. (2001). The meniscus: review of basic principles with application to surgery and rehabilitation. *Journal of athletic training*, *36*(2), 160.

20. Goldblatt, J. P., & Richmond, J. C. (2003). Anatomy and biomechanics of the knee. *Operative Techniques in Sports Medicine*, *11*(3), 172-186.

21. Vedi, V., Spouse, E., Williams, A., Tennant, S. J., Hunt, D. M., & Gedroyc, W. M. W. (1999). Meniscal movement. *J Bone Joint Surg Br*, *81*(1), 37-41.

22. Walker, P. S., & Hajek, J. V. (1972). The load-bearing area in the knee joint. *Journal of biomechanics*, *5*(6), 581IN3585-584IN5589.

23. Gray, J. C. (1999). Neural and vascular anatomy of the menisci of the human knee. *Journal of Orthopaedic & Sports Physical Therapy*, *29*(1), 23-30.

24. Poehling, G. G., Ruch, D. S., & Chabon, S. J. (1990). The landscape of meniscal injuries. *Clinics in sports medicine*, *9*(3), 539-549.

25. Howell, R., Kumar, N. S., Patel, N., & Tom, J. (2014). Degenerative meniscus: Pathogenesis, diagnosis, and treatment options. *World Journal of Orthopedics*, *5*(5), 597–602.

26. McCarty, E. C., Marx, R. G., & Wickiewicz, T. L. (2000). Meniscal tears in the athlete. Operative and nonoperative management. *Physical medicine and rehabilitation clinics of North America*, *11*(4), 867-880.

27. Mordecai, S. C., Al-Hadithy, N., Ware, H. E., & Gupte, C. M. (2014). Treatment of meniscal tears: An evidence based approach. *World Journal of Orthopedics*, *5*(3), 233–241.

28. Strobel MJ. Manual of arthroscopic surgery Springer-Verlag Berlin 2009;1:99-200.

29. Weiss CB, Lundberg M, Hamberg P, DeHaven KE, Gillquist J. Non-operative treatment of meniscal tears. Journal of Bone & Joint Surgery - American Volume 1989;71(6):811-22.

30. Aiello RA. Knee, meniscal tears (MRI). http://emedicine.medscape.com 12/04/10 2009.

31. Fan R, Ryu R. Meniscal lesions: Diagnosis and Treatment. http://www.medscape.com, 31/03/10 2000.

32. Mohan, B. R., & Gosal, H. S. (2007). Reliability of clinical diagnosis in meniscal tears. *International orthopaedics*, *31*(1), 57-60.

33. Blyth M, Anthony I, Francq B, et al. Diagnostic accuracy of the Thessaly test, standardised clinical history and other clinical examination tests (Apley's, McMurray's and joint line tenderness) for meniscal tears in comparison with magnetic resonance imaging diagnosis. Southampton (UK): NIHR Journals Library; 2015 Aug. (Health Technology Assessment, No. 19.62.)

34. Hegedus, E. J., Cook, C., Hasselblad, V., Goode, A., & Mccrory, D. C. (2007). Physical examination tests for assessing a torn meniscus in the knee: a systematic review with meta-analysis. *journal of orthopaedic & sports physical therapy*, *37*(9), 541-550.

35. Karachalios T, Hantes M, Zibis AH, Zachos V, Karantanas AH, Malizos KN. Diagnostic accuracy of a new clinical test (the Thessaly test) for early detection of meniscal tears. J Bone Joint Surg Am. 2005;87:955-962.

36. Cannon JR, W. D., & Vittori, J. M. (1992). The incidence of healing in arthroscopic meniscal repairs in anterior cruciate ligament-reconstructed knees versus stable knees. *The American Journal of Sports Medicine*, *20*(2), 176-181.

37. McCarty, E. C., Marx, R. G., & DeHaven, K. E. (2002). Meniscus repair: considerations in treatment and update of clinical results. *Clinical Orthopaedics and related research*, *402*, 122-134.)

38. Yoon, K. H., & Park, K. H. (2014). Meniscal Repair. *Knee Surgery & Related Research*, *26*(2), 68–76. http://doi.org/10.5792/ksrr.2014.26.2.68

39. Muellner, T., Egkher, A., Nikolic, A., Funovics, M., & Metz, V. (1999). Open meniscal repair: clinical and magnetic resonance imaging findings after twelve years. *The American journal of sports medicine*, *27*(1), 16-20.

40. Rockborn, P., & Gillquist, J. (2000). Results of open meniscus repair. Long-term follow-up study with a matched uninjured control group. *Bone & Joint Journal*, *82*(4), 494-498.

41. Grant, J. A., Wilde, J., Miller, B. S., & Bedi, A. (2012). Comparison of inside-out and all-inside techniques for the repair of isolated meniscal tears: a systematic review. *The American journal of sports medicine*, *40*(2), 459-468.

42. Nepple, J. J., Dunn, W. R., & Wright, R. W. (2012). Meniscal repair outcomes at greater than five years. *J Bone Joint Surg Am*, *94*(24), 2222-2227.

43. Sihvonen, R., Paavola, M., Malmivaara, A., Itälä, A., Joukainen, A., Nurmi, H.,& Järvinen, T. L. (2013). Arthroscopic partial meniscectomy versus sham surgery for a degenerative meniscal tear. *N Engl J Med*, *2013*(369), 2515-2524.

44. Baratz, M. E., Fu, F. H., & Mengato, R. (1986). Meniscal tears: The effect of meniscectomy and of repair on intraarticular contact areas and stress in the human knee A preliminary report. *The American Journal of Sports Medicine*, *14*(4), 270-275.

45. Roos, H., Adalberth, T., Dahlberg, L., & Lohmander, L. S. (1995). Osteoarthritis of the knee after injury to the anterior cruciate ligament or meniscus: the influence of time and age. *Osteoarthritis and Cartilage*, *3*(4), 261-267.

46. McDermott, I. D., & Amis, A. A. (2006). The consequences of meniscectomy. *Bone & Joint Journal*, *88*(12), 1549-1556.

47. Faunø, P., & Nielsen, A. B. (1992). Arthroscopic partial meniscectomy: a long-term follow-up. *Arthroscopy: The Journal of Arthroscopic & Related Surgery*, *8*(3), 345-349

48. Jorgensen, U. F. F. E., Sonne-Holm, S. T. I. G., Lauridsen, F. L. E. M. M. I. N. G., & Rosenklint, A. R. N. E. (1987). Long-term follow-up of meniscectomy in athletes. A prospective longitudinal study. *Bone & Joint Journal*, *69*(1), 80-83.

49. Stein, T., Mehling, A. P., Welsch, F., von Eisenhart-Rothe, R., & Jäger, A. (2010). Long-term outcome after arthroscopic meniscal repair versus arthroscopic partial meniscectomy for traumatic meniscal tears. *The American journal of sports medicine*, *38*(8), 1542-1548.

50. Haq I, Murphy E, Dacre J. Osteoarthritis. Postgrad Med J, 2003, 79:377–383.

51. Kaplan, W., Wirtz, V., Mantel, A., & Béatrice, P. S. U. (2013). Priority Medicines for Europe and the World Update 2013 report. *Methodology*, *2*, 7.

52. Bijlsma, J. W., Berenbaum, F., & Lafeber, F. P. (2011). Osteoarthritis: an update with relevance for clinical practice. *The Lancet*, *377*(9783), 2115-2126.

53. Hochberg, M. (2013). Osteaoarthritis: new approaches. Osteoarthritis: a story of close relationship between bone and cartilage, 35(2), 139-141.

54. Mandl, L. (2011). Treating the pain of osteoarthritis — where do we go from here? *The Journal of Rheumatology, 38*(8).

55. Fraenkel, L., Bogardus, S. T., Concato, J., & Wittink, D. R. (2004). Treatment options in knee osteoarthritis: the patient's perspective. *Archives of internal medicine*, *164*(12), 1299-1304.

56. Zhang W, Moskowitz RW, Nuki G, et al. OARSI recommendations for the management of hip and knee osteoarthritis, part II: OARSI evidence-based, expert consensus guidelines. *Osteoarthritis Cartilage* 2008; 16: 137–62.

57. Zhang W, Nuki G, Moskowitz RW, et al. OARSI recommendations for the management of hip and knee osteoarthritis: part III: Changes in evidence following systematic cumulative update of research published through January 2009. *Osteoarthritis Cartilage* 2010; 18: 476–99.

58. Roddy E, Zhang W, Doherty M. Aerobic walking or strengthening exercise for osteoarthritis of the knee? A systematic review. *Ann Rheum Dis* 2005; 64: 544–48.

59. Avouac J, Gossec L, Dougados M. Efficacy and safety of opioids for osteoarthritis: a meta-analysis of randomized controlled trials. *Osteoarthritis Cartilage* 2007; 15: 957–65.

60. Chambers, M. C., & El-Amin, S. F. (2015). Tissue engineering of the meniscus: Scaffolds for meniscus repair and replacement. *Musculoskeletal Regeneration*, *1*.

61. Weinand C, Peretti GM, Adams SB, Jr., Randolph MA, Savvidis E, Gill TJ. Healing potential of transplanted allogeneic chondrocytes of three different sources in lesions of the avascular zone of the meniscus: a pilot study. Arch Orthop Trauma Surg. 2006;126:599–605.

62. Caplan AI, Dennis JE. Mesenchymal stem cells as trophic mediators. J Cell Biochem. 2006;98:1076–84.

63. Caplan AI. Adult mesenchymal stem cells for tissue engineering versus regenerative medicine. J Cell Physiol. 2007;213:341–7.

64. Ramallal M, Maneiro E, Lopez E, Fuentes-Boquete I, Lopez-Armada MJ, Fernandez-Sueiro JL, et al. Xeno-implantation of pig chondrocytes into rabbit to treat localized articular cartilage defects: an animal model. Wound Repair Regen. 2004;12:337–45.

65. Hoben, G. M., Willard, V. P., & Athanasiou, K. A. (2009). Fibrochondrogenesis of hESCs: Growth Factor Combinations and Cocultures. *Stem Cells and Development*, *18*(2), 283–292. http://doi.org/10.1089/scd.2008.0024

66. Esposito AR, Moda M, Cattani SM, de Santana GM, Barbieri JA, Munhoz MM. PLDLA/PCL-T Scaffold for Meniscus Tissue Engineering. Biores Open Access 2013;2:138-47.

67. Verdonk R, Verdonk P, Huysse W, Forsyth R, Heinrichs E-L. Tissue ingrowth after implantation of a novel, biodegradable polyurethane scaffold for treatment of partial meniscal lesions. Am J Sports Med 2011;39:774-782.

68. Meng J, Cheng X, Kong H, Yang M, Xu H. Preparation and biocompatibility evaluation of polyurethane filled with multiwalled carbon nanotubes. J Nanosci Nanotechnol 2013;13:1467-71.

69. Stone KR, Steadman JR, Rodkey WG, Li ST. Regeneration of meniscal cartilage with use of a collagen scaffold. Analysis of preliminary data. J Bone Jt Surg Am. 1997;79:1770–7.

70. Rodkey WG, DeHaven KE, Montgomery WH, 3rd, Baker CL, Jr., Beck CL, Jr., Hormel SE, et al. Comparison of the collagen meniscus implant with partial meniscectomy. A prospective randomized trial. J Bone Jt Surg Am. 2008;90:1413–26.

71. Schoenfeld AJ, Landis WJ, Kay DB. Tissue-engineered meniscal constructs. Am J Orthop (Belle Mead NJ) 2007;36:614–20.

72. Steadman JR, Rodkey WG. Tissue-engineered collagen meniscus implants: 5- to 6-year feasibility study results. Arthroscopy. 2005;21:515–25.

Appendix A

Dissection Images

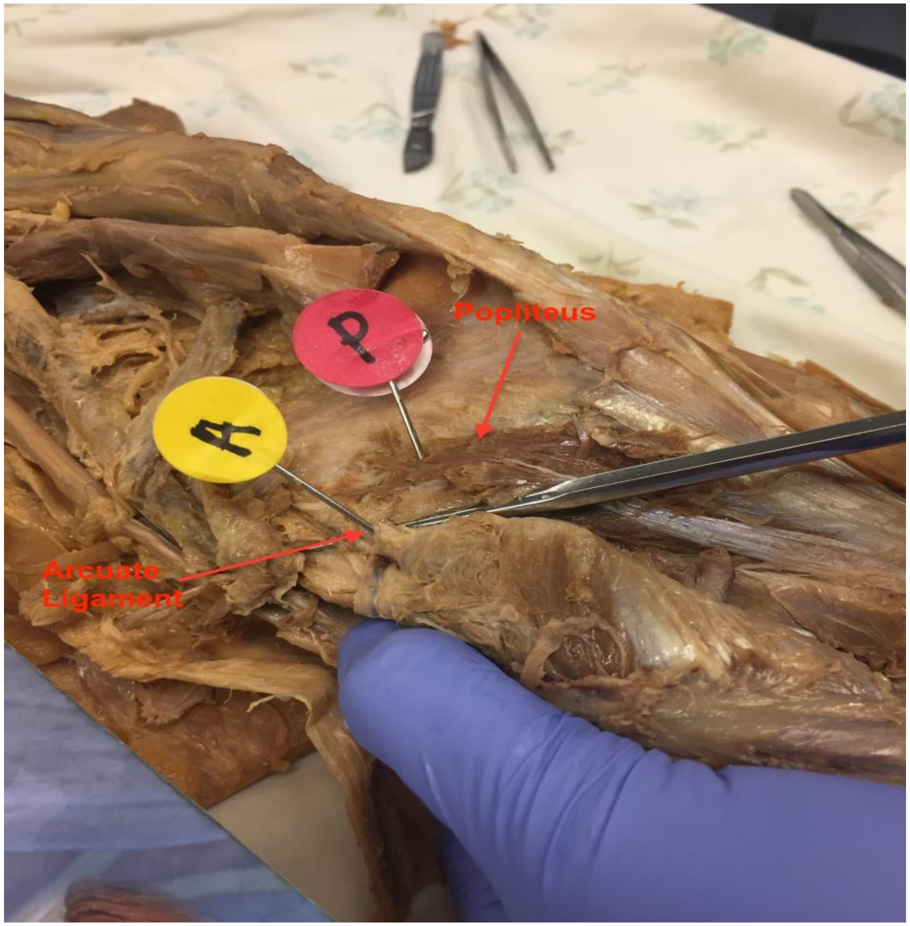


Figure 14. Cadaveric dissection of the arcuate ligament and popliteus

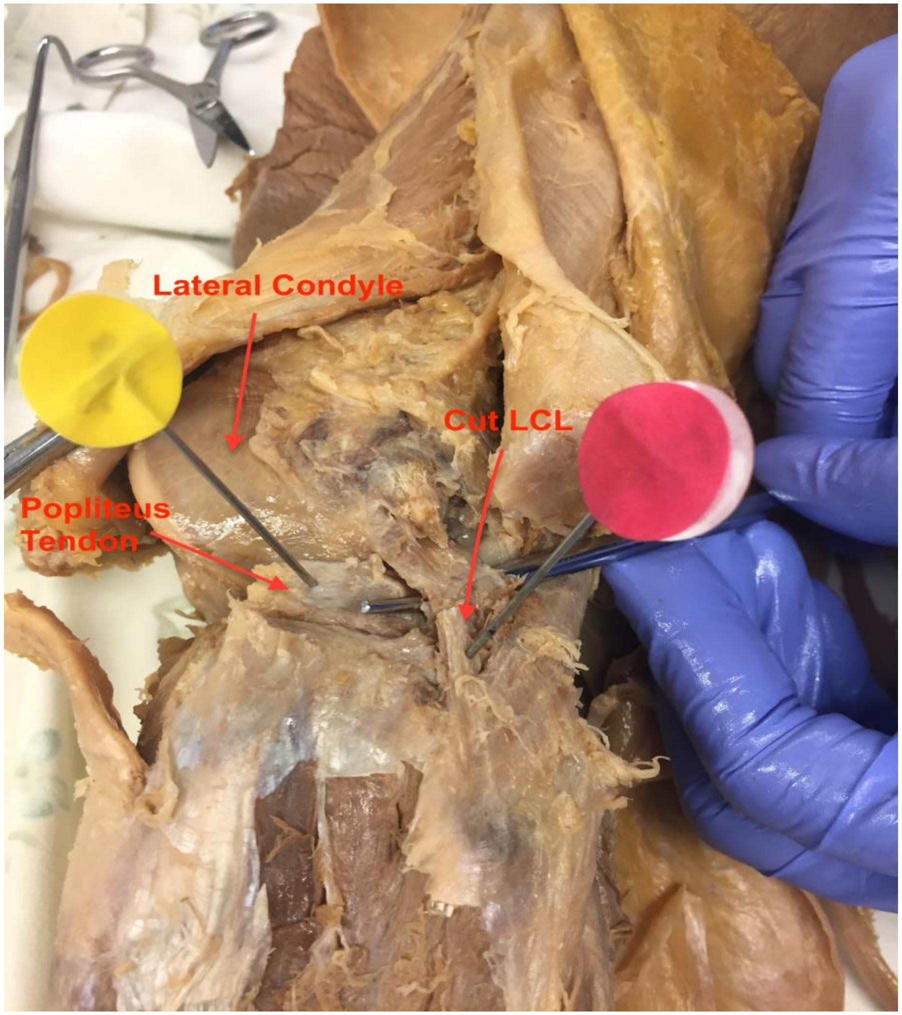


Figure 15. Cadaveric Dissection of the lateral tibiofemoral joint with lateral collateral and popliteus transected

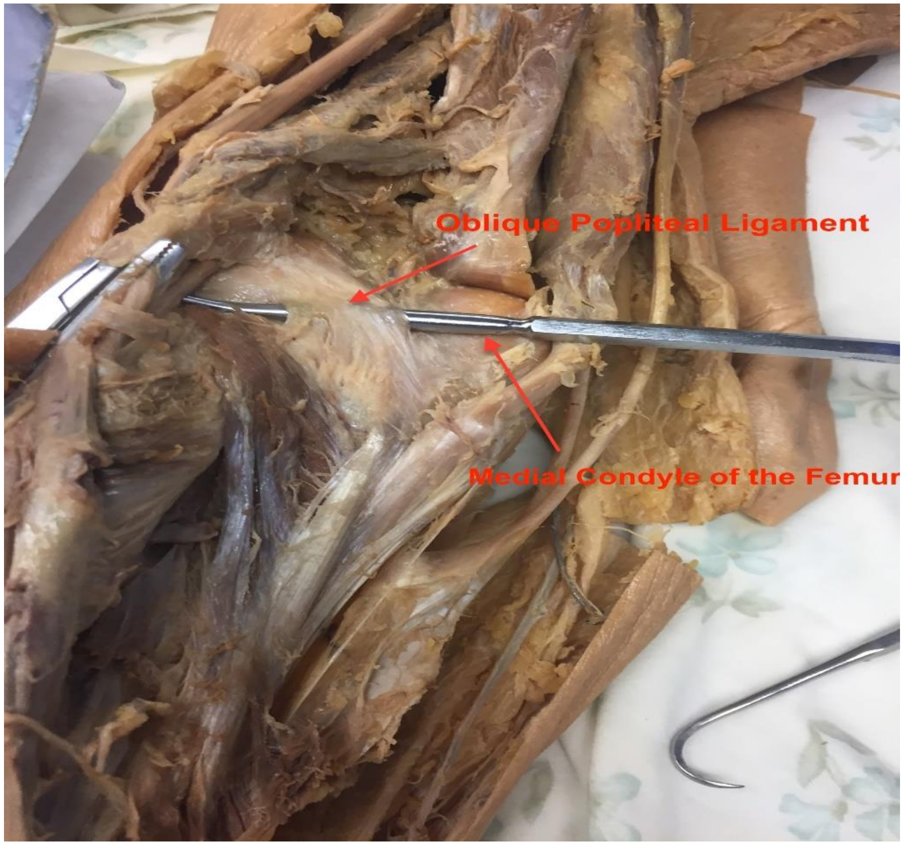


Figure 16. Cadaveric dissection of the oblique popliteal ligament