



# Ligament Injuries

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## Anterior Cruciate Ligament

### Pathology

The anterior cruciate ligament (ACL) is the most common knee ligament injured in sports [1].

Injuries to the ACL can range from mild, such as a tear/sprain, to severe, when the ligament is completely torn. The ACL courses from its origin at the medial wall of the lateral femoral condyle to its insertion into the middle of the intercondylar area of the tibia. Its function is to resist anterior tibial translation and rotational loads. The ACL has two bundles, an anteromedial bundle, which is tight in flexion, and a posterolateral bundle, which is more tighter in extension. The anteromedial bundle is more responsible for restraining anterior tibial translation, while the posterolateral bundle is more responsible for rotational stability [1].

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The incidence of ACL injuries in the USA is 1 in 3500 people and is the most injured knee ligament. Some studies suggest that ACL injuries may occur more frequently in women due to weaker hamstrings and quadriceps overuse when decelerating, which stresses the ACL, or due to estrogenic effects causing increased flexibility of tissues. However, the literature for the female demographic is controversial.

ACL injuries can occur through noncontact or contact mechanisms. A noncontact pivoting injury occurs when the tibia translates anteriorly while the knee is in slight flexion and in a valgus position, which may occur in skiers, soccer players, and basketball players. In contrast, contact injuries occur with a direct trauma to the lateral aspect of the knee with the highest risk sport being football [2]. A direct trauma may also cause medial meniscus and medial collateral ligament injury, in addition to ACL injury leading to the commonly known “unhappy triad” [2].

## Clinical Presentation

Typically on presentation, a loud pop occurs that can be heard by the individual. Individuals may also report the knee “giving out,” signifying knee instability, and will have difficulty bearing weight. Knee effusion or hemarthrosis may occur within the first 2 h. More than 50% of ACL injuries occur with a meniscal tear [1].

## Physical Exam

On examination of an acute injury, one will likely see a large effusion and patients will not be able to actively extend their knee. Provocative tests that can be used are the Lachman test, pivot shift test, and the anterior drawer test. The most sensitive exam is Lachman test, with a sensitivity of 95–99% [1].

Lachman test requires the patient to be positioned supine with their injured knee flexed to 20°–30°, and some sources encourage slightly externally rotating the injured leg to relax the iliotibial band (Fig. 2.1). The examiner then uses one hand to stabilize the



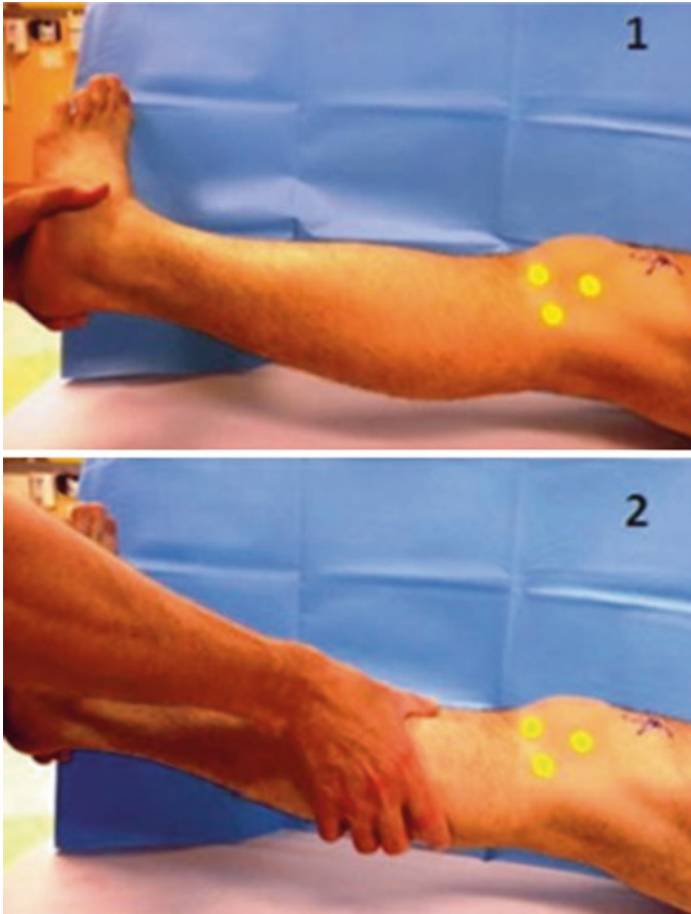
**Fig. 2.1** Lachman test [3]

distal femur, and uses their other hand to grasp the proximal tibia. Next, an anterior force is applied to the proximal tibia in an attempt to sublux the tibia forward while keeping the femur stabilized [2]. The test is considered positive if there is excessive anterior translation of the proximal tibia greater than the uninjured side and lack of a firm endpoint. ACL injuries are graded based on the amount of anterior tibial translation compared to uninjured side (Table 2.1).

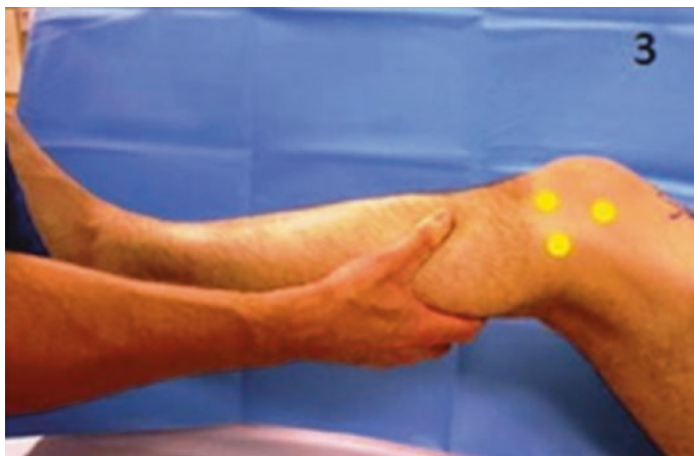
The pivot shift is not as sensitive and requires the patient to relax as much as possible. However, it is very specific (98–100%) and pathognomonic when performed under anesthesia (Fig. 2.2)

**Table 2.1** Grading ACL injuries: degree of laxity determined by amount of tibial translation compared to uninjured side [1]

Grade 1 (mild)	1–5 mm displacement
Grade 2 (moderate)	6–10 mm displacement
Grade 3 (severe)	>10 mm displacement



**Fig. 2.2** Pivot shift test [4]



**Fig. 2.2** (continued)

[1]. The examiner grasps the heel of the injured leg with the examiners opposite hand placed laterally on the proximal tibia just distal to the knee. The examiner then applies a valgus stress and an axial load while internally rotating the tibia as the knee is moved into flexion from a fully extended position. A positive test is indicated by subluxation of the tibia while the femur rotates externally followed by a reduction of the tibia at 20–30 degrees of flexion.

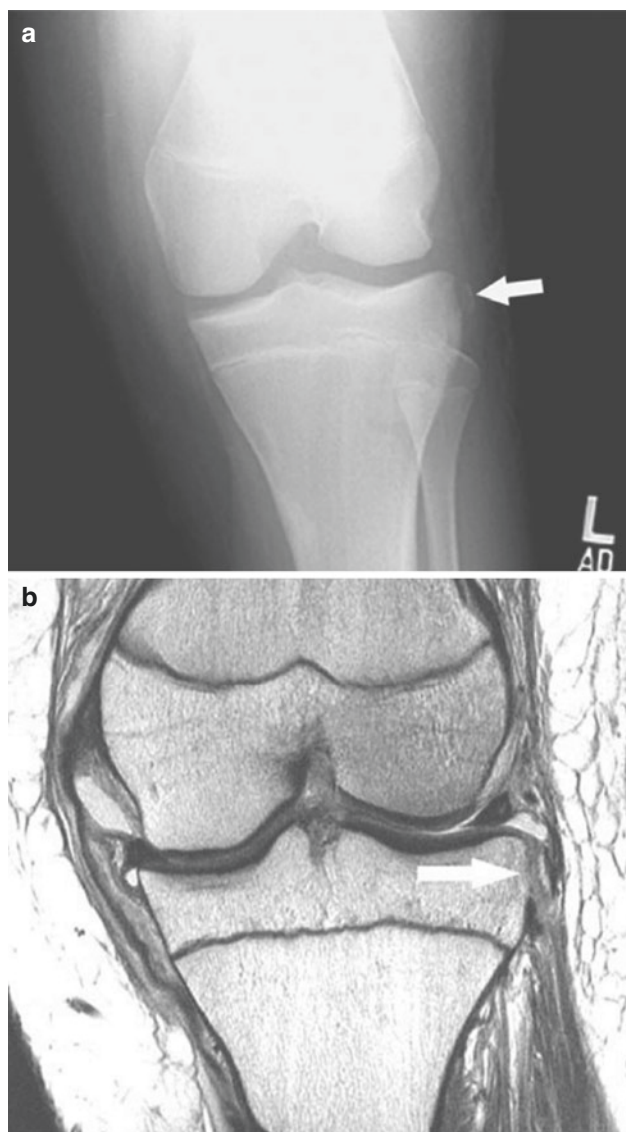
The anterior drawer test can be used, but lacks the sensitivity and specificity of the Lachman test (Fig. 2.3). The patient lies supine with their hips flexed to 45°, and the injured knee flexed to 90°. Similar to the Lachman test, the examiner then uses one hand to stabilize the distal femur, and uses their other hand to grasp the proximal tibia. Next, an anterior force is applied to the proximal tibia in an attempt to sublux the tibia forward while keeping the femur stabilized. The test is considered positive if there is excessive anterior translation relative to the contralateral side.



**Fig. 2.3** Anterior Drawer test [3]

## Diagnostic Studies

Radiographs are typically normal in ACL injuries. Radiographs can be used to evaluate for effusions and bony injuries, especially a Segond fracture. A Segond fracture is a capsular avulsion fracture of the lateral tibial plateau and it is associated with ACL tears 75–100% of the time (Fig. 2.4) [1].



**Fig. 2.4** (a) Knee X-ray anterior–posterior (AP) view showing a Second fracture in a 15-year-old girl. (b) MRI fat sat AP coronal view showing Second fracture associated with ACL tear [5]

MRIs will typically confirm clinical diagnosis of ACL rupture and evaluate for concomitant pathology with 86% sensitivity and 95% specificity. Preferred views include T2 sagittal MRIs or STIR sequences, which will show increased signal and edema of the ACL, fiber discontinuity, and change in the ACL course seen as alteration of Blumensaat's line. Blumensaat's line is the line drawn along the roof of the intercondylar notch of the femur. This line is helpful in evaluating for ACL injury as a normal ACL-Blumensaat line angle is less than  $15^\circ$ .

If the angle is greater than  $15^\circ$ , this typically indicates an ACL tear. Research has found that the sensitivity and specificity of the ACL-Blumensaat line angle to detect ACL injury is about 95% [6]. Arthroscopic evaluation is the gold standard in diagnosing ACL injury with 92–100% sensitivity and 95–100% specificity [2].

In one study, dynamic ultrasound was used to diagnose ACL tears with a sensitivity of 52% for partial tears and 79% for complete tears. Specificity was 85% for partial tears and 89% for complete tears. Ultrasound can detect complete ACL rupture but it is not currently standard practice. Overall, diagnostic musculoskeletal ultrasound can be completed at point of injury and further care [7].

## Treatment

The acute treatment of an ACL injury is rest, ice, compression, and use of a knee immobilizer or hinged knee brace to aid with pain and stability. Next steps include non-operative rehabilitation or ACL reconstruction/repair.

The ACL has a poor capacity to heal so reconstruction is preferred to repair. The ACL cannot form a fibrin-platelet clot to initiate tissue healing because clot formation is most likely inhibited by factors in the surrounding synovial fluid. Hence, the location of the ACL puts it at a disadvantage to heal when injured compared to extra-articular ligaments [8]. People who are typically recommended to undergo ACL reconstruction are active athletes/patients, have other ligamentous/repairable meniscal injuries,



and/or are experience knee instability. ACL reconstruction can be performed with patellar tendon autografts (native), quadricep tendon autografts or hamstring (semitendinosus or gracilis) autografts or allografts (cadaveric). Advantages with autografts include faster healing, lower risk of re-injury and infection. However, disadvantages include complications at the harvest site, longer surgical procedure times, and constraints around tissue selection such as size and harvest location. Cadaveric allografts are often taken from tendinous structures such as the Achilles, patellar, hamstring, and posterior tibialis tendons. However, these are preferentially used in middle aged athletes engaging in lower impact sports. While allografts perform similarly to autografts, they carry higher rates of re-injury, risk of disease transmission, immunologic reaction, and slower remodeling. Still, allografts may be the preferred choice due to decreased surgical time and less limitations on size and harvest site morbidity. Ultimately, the decision is made after weighing risks and benefits for each individual patient [9].

ACL repair was previously abandoned due to high failure rates in people of all ages, but there has been some increase in repair recently, in certain populations, especially in cases involving proximal ACL avulsions which results in separation of the ACL from the bone. There have been studies where ACL repair in this population can lead to good results, [10] but overall reconstruction is still the preferred surgical method. Current studies suggest that even modern ACL repair techniques have a failure rate of 5–10 times higher than that of ACL reconstruction [11].

If a patient decides for operative management, it is important to begin immediate weight bearing postoperatively and to be involved in early rehabilitation. There is no true standardized difference in rehabilitation protocol for different types of grafts. However, one important precaution is the avoidance of rotational stresses to the knee before initial graft incorporation to bone, which occurs approximately at 4 weeks for bone-tendon-bone grafts and 8–12 weeks for hamstring allografts [12].

There are specific modalities, muscles, and exercise types that should be targeted after a graft is placed. In the acute phase

(1–2 weeks), modalities include cryotherapy and electrical stimulation, while exercises include active assisted flexion, passive extension, isometric quadriceps contractions (between 90° and 45°), dynamic hamstring exercises, and straight leg raises.

In the recovery phase (2+ weeks), patients can start using modalities such as superficial heat, pulsed ultrasound, and electrical stimulation. Exercises are advanced to active flexion and extension training, dynamic quadriceps exercises (between 90° and 30°), and hamstring strengthening [12, 13]. These patients should also focus on closed chain exercises in early rehabilitation (2+ weeks after injury), such as leg-presses or squats. Closed chain exercises are when the hand or foot is fixed in place, whereas in open chain exercises the hand or foot is free to move. Post-ACL reconstruction patients should specifically avoid open chain quadriceps strengthening exercises and should also avoid isokinetic quadriceps strengthening from 15° to 30° during their early rehabilitation [14]. These exercises can put excess stress on the graft. Aquatic exercises, bicycling, swimming, and the elliptical trainer can also be used in the recovery phase. In the recovery phase, if the patient has full flexion and extension, symmetric quadriceps and hamstring strength, and symptom-free progression in a sports-specific program, they are considered to be advanced to the functional phase. The functional phase is the final phase and focuses on general flexibility training, strengthening, power and endurance, neuromuscular control, and proprioceptive training with a return to sport-specific participation [12].

Non-operative management consists of a physical therapy rehabilitation program. People who are considered for non-operative treatment include those who have a more sedentary lifestyle, are recreational athletes, and those without significant knee instability. Physical therapy will focus on range of motion and strengthening of the quadriceps, hamstrings, hip abductors, and core muscles. The rehabilitation protocol is slightly different from postoperative rehabilitation because there is no recommended numerical degree of movement that should be avoided, as there is no graft placement. There is more focus on range of motion in the recovery phase, otherwise the same protocol is used and the same modalities mentioned earlier can be used in non-operative management [12].

Steroid injections as a treatment for ACL injury is unclear, however, most recent literature advises against it. Dexamethasone, a steroid, was found to increase the calcification of ACL cells and caused ACL degeneration through endoplasmic reticulum stress. This suggests that long-term treatment with dexamethasone may cause adverse effects on ACL tissue and increases the risk of long-term rupture [15]. Another animal study in sheep looked at only partial ACL injuries and found that multiple repeated injections of glucocorticoids led to significant proteoglycan loss in the methylprednisolone treated knees. Proteoglycan is a component of a molecule that typically provides hydration and enables tissue to withstand compressional forces. Hence, steroid use in ACL injury is not standard of care [16].

There have been several clinical trials and case reports that demonstrate the effectiveness of prolotherapy injections for ACL injuries. While it is not standard of care, prolotherapy is defined as injection that causes growth of normal cells or tissues. Prolotherapy with intermittent dextrose injections in patients with symptomatic ACL laxity have resulted in clinically and statistically significant improvement in ACL laxity, pain, swelling, and knee range of motion. One particular study used an intraarticular injection consisting of 6–9 cc of 10% dextrose injected at intervals of 0, 2, 4, 6, and 10 months, and then injected with 6 cc of 25% dextrose at 12 months. Afterward, depending on patient preference, injection of either 10% or 25% dextrose was completed every 2–4 months through 36 months [17].

Future treatment directions may include orthobiologic techniques using platelet-rich-plasma, bio-scaffolds with tissue-engineered collagen, and mesenchymal stem cells. Animal studies show encouraging results when these regenerative medicine techniques are used for ligament healing and some preliminary literature suggests some role intra-operatively, but further research must be done to establish clinical impact, operative techniques, and validity [18].

## **Return to Activities**

The postoperative rehabilitation program lasts 6–9 months prior to full return to play [1]. There is no widely accepted specific time

to return to playing sports. It will depend on the patient's restored mobility, flexibility, strength, function, and ability to pass a series of tests that replicate specific sport activities, such as hopping and jumping on one and both legs. Psychological readiness plays a large role in returning to play. The patient must be mentally prepared and the timing of return should not be ignored [9].

Effective ACL injury prevention is a topic of research because once an athlete sustains an ACL injury and undergoes reconstructive surgery, that athlete has an increased risk of injury in both the affected extremity and the contralateral extremity [13]. Mandelbaum and colleagues started the "Prevent Injury Enhance Performance injury prevention program" (PEP). The PEP program focuses on warm-up exercises, agility exercises, plyometric exercises, and stretching and strengthening to prevent ACL injury. For example, warm-up includes jogging and shuttle runs while strengthening includes lunges, hamstring exercises, and single-leg toe raises. Plyometric exercises focus on landing techniques and knee positioning. The program is designed to be completed in 15–20 min. This study noted an 88% reduction in ACL tears in year one. Year two of the study also found significant decreases in ACL injury through training in the prevention program. This example emphasizes the importance of physical therapy and proper rehabilitation exercises months out of an ACL injury [19].

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## Posterior Cruciate Ligament

### Pathology

The posterior cruciate ligament (PCL) is less commonly injured than the ACL. The role of the PCL is to primarily resist extreme posterior translation of the tibia relative to the femur and to prevent hyperflexion. Its secondary role is to prevent excessive rotation between 90 and 120 degrees of knee flexion [20]. The PCL has two bundles, anterolateral bundle, which is tight in flexion, and posteromedial bundle, which is tight in extension. The PCL originates from the anterolateral medial femoral condyle and inserts along the posterior tibial plateau. When the PCL is injured,

other structures in the posterolateral corner including the lateral collateral ligament, popliteus tendon, and the popliteofibular ligament may be injured as well. The mechanism of a PCL injury is most commonly from an impact to the anterior tibia while the knee is in flexion; for example, in a car accident when an individual's knee hits the dashboard [1]. Another mechanism is from a noncontact hyperflexion of the knee with a plantar-flexed foot. This specific mechanism is the most common cause of isolated PCL injuries, without combined ligamentous damage [21]. A PCL injury occurs less often from knee hyperextension, but is also possible [1].

## Clinical Presentation

The patient will most likely present with minimal swelling, non-specific posterior knee pain, and inability to bear weight [1]. A common complaint is apprehension while going down stairs because of a sense of unsteadiness. However, the common complaint of buckling, such as in an ACL injury, is rarely seen in an isolated PCL injury. Thus, if a patient complains of instability, typically other ligaments are also involved representing a combined injury [7].

## Physical Exam

Provocative tests for a PCL injury include the posterior drawer test, posterior sag sign, and quadriceps activation test.

The most accurate test for determining a PCL tear is the posterior drawer test at 90°. With the knee at 90 degrees of flexion, a force directed posteriorly is applied to the proximal tibia and posterior tibial translation is then measured. Tears are divided into grade I, grade II and grade III based on the amount of posterior tibial translation during this test (Fig. 2.5, Table 2.2).

The posterior sag sign is tested with the patient lying supine with the hips and knees flexed at 90°, while the physician supports the ankles, and observes for any posterior shift of the tibia. One



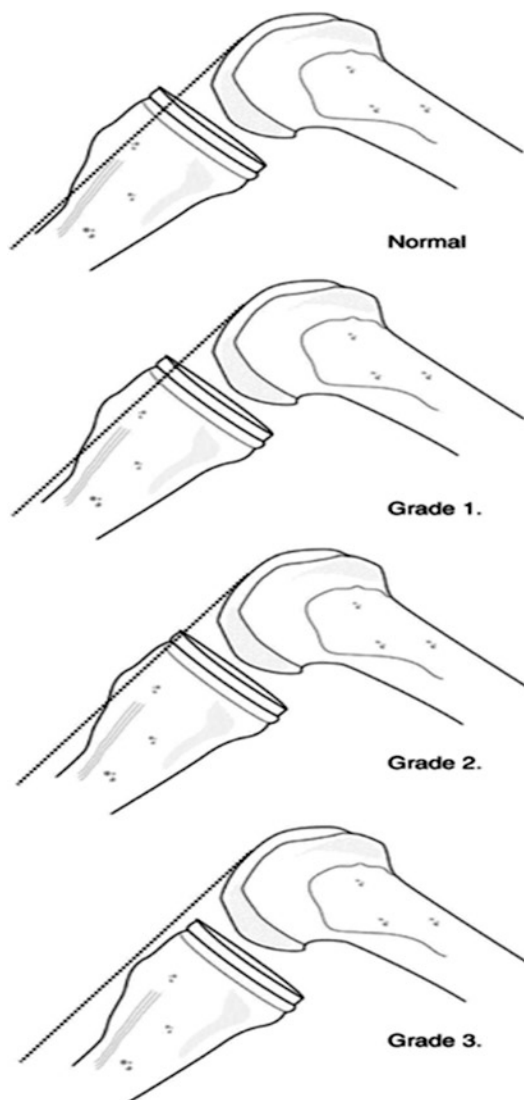
**Fig. 2.5** Posterior Drawer test [3]

**Table 2.2** Grading PCL tears based on amount of posterior tibial translation compared to uninjured side [21]

Grade I (mild)	1–5 mm partial PCL tear
Grade II (moderate)	6–10 mm complete PCL tear
Grade III (severe)	>10 mm complete PCL tear + capsuloligamentous injury [may include ACL and/or PLC (posterolateral corner) injury]

important point to note is that a quadriceps spasm may cause a false-negative in this test (Fig. 2.6).

The quadriceps activation test has a specificity of 97–100%. It is performed with the patient lying supine, knee flexed to 90° and the foot stabilized by a physician. The patient is asked to slowly



**Fig. 2.6** Posterior Sag Sign. Grade 1: Displacement of front of tibia but still in front of anterior aspect of femur. Grade 2: Displacement of front of tibia in line with anterior aspect of femur. Grade 3: Anterior aspect of tibia behind anterior aspect of femur [22]



**Fig. 2.7** Quadriceps activation test [23]

slide his or her foot down the table. In a PCL injury, the quadriceps contraction results in an anterior shift of the tibia more than 2 mm relative to the femur (Fig. 2.7) [22, 23].

The posterolateral corner (PLC) should also always be tested when suspecting a PCL injury. The posterolateral corner itself consists of three ligaments, the anterolateral ligament, the popliteofibular ligament, and the fabellofibular ligament. The dial test is a great way to differentiate PLC and PCL injury together or just isolated PLC injury.

The dial test is done with the patient prone, both knees flexed first to 30°, which best isolates the PLC, and then flexed to 90°, with external rotation applied to the tibias at each position. A 10° increase in external rotation, when compared to the contralateral side, at 30 degrees of knee flexion indicates an isolated PLC injury. If the test is positive at also 90 degrees of knee flexion, then there is a concomitant injury to the PCL (Fig. 2.8) [2].





**Fig. 2.8** Dial test to differentiate PLC and PCL injury [24]

## Diagnostic Studies

Radiographs are being used more often to evaluate PCL injuries, but are still not the gold standard for diagnosis. In an AP and supine lateral view, one can evaluate for associated avulsion fractures of the tibial insertion. The lateral stress view of an X-ray has also become increasingly used (Fig. 2.9). A lateral stress X-ray is when one applies stress to the anterior tibia with the knee flexed to  $70^\circ$ . If there is asymmetric posterior displacement of the tibia, this indicates a PCL injury. Some clinicians consider stress-radiography to be the best test to quantify posterior tibial displacement in PCL insufficiency [25]. MRIs will typically confirm clinical diagnosis of PCL rupture and evaluate for other ligamentous injuries. Arthroscopic evaluation is the gold standard for diagnosis [1].



**Fig. 2.9** Lateral stress view X-ray demonstrating PCL injury showing posterior displacement of tibia [25]

## Treatment

The acute treatment of a PCL injury would include rest, ice, and compression. One can consider a knee brace in full extension or use of crutches if there is significant functional limitation and instability [1]. Next steps of treatment include non-operative care

with rehabilitation, or less commonly, operative management with PCL reconstruction. In general, surgical treatment is only recommended if persistent instability is present and/or other concurrent meniscal/ligamentous injuries are present.

The research on steroid or prolotherapy injection use on PCL injury is minimal. One case report in a 24-year-old male soccer player who presented with a 7-year history of left posterior knee instability, grade 1 posterior drawer and grade 1 posterior sag signs, did undergo one experimental injection of prolotherapy with dextrose hyperosmolar solution. He was injected with a mixture of 1 mL of 50% dextrose, 2 mL of sterile water, and 2 mL of 1% lidocaine was injected. The patient's subjective feeling of looseness and instability resolved by 7 weeks [26].

Studies on the application of orthobiologics and regenerative medicine for PCL injury are lacking. There have been more studies with the use of regenerative techniques for the ACL, as mentioned previously, and in theory these methods could be developed and extended to other ligaments in the knee. One case series of 13 soccer players with isolated partial PCL injuries in Spain used a series of three once-weekly ultrasound-guided white blood cell-poor PRP injections to the PCL, ligament sheath, and popliteal fossa with the goal to enhance the healing of a grade I or II injured PCL. Patients also used a specific PCL brace and participated in an early rehabilitation program. In this study, the treatment regimen was effective to achieve adequate MRI-based healing in 100% of patients and a return to play in 90% of soccer players. Further well-designed studies are needed to appropriately assess this potential treatment technique [27].

The PCL suffers from partial injury more commonly than the ACL, so the grading is particularly important in terms of treatment. Another important factor to consider in PCL injury treatment is other concurrent ligamentous injury. An isolated acute PCL injury, either Grade I or Grade II, will only need 4–6 weeks of limited activity and rehabilitation for treatment. Rehabilitation exercises focus on knee extensor and quadriceps strengthening.

An isolated acute Grade III PCL injury will need 4 weeks in extension bracing to keep the knee in full extension to prevent posterior subluxation of the tibia. If the patient is a young athlete,

surgery can also be considered in this population. If the PCL is combined with injuries of the LCL, MCL or PLC, there should be PCL reconstruction within 2 weeks. PCL reconstruction options include tibial inlay or transtibial methods. Grafts include auto-graft or allograft. Allografts are typically used as there are multiple graft choices available, such as the Achilles, patellar, hamstring, or anterior tibialis tendons. The transtibial technique is when the graft passes proximally and posteriorly through the tibia and makes a 90° turn around the tibial tunnel before entering the knee joint. This 90° bend in the graft has been shown to create increased internal tendon pressures and possibly lead to graft elongation or even failure. The tibial inlay technique differs because there is arthroscopic placement of the femoral tunnel and the open creation of a bone trough in the posterior tibia. The benefit of this procedure is that the graft is secured to the anatomic tibial attachment site of the PCL, thus avoiding the 90° curve associated with the transtibial tunnel. Controversy continues to exist in PCL reconstruction regarding the optimal location of tibial fixation, ideal placement of the femoral tunnel, number of graft bundles, and appropriate graft tension [28].

Postoperatively the patient should be partially weight bearing with a hinged knee brace locked in extension for 2–4 weeks. Exercises are allowed on the first day after surgery, but should be limited to isometric quadriceps and ankle pump exercises. At 4 weeks, passive range of motion with a physical therapist can be fully performed and active range of motion is not until 8 weeks. Patients should avoid resisted hamstring strengthening exercises in early rehabilitation, such as hamstring curls. This is because the hamstrings can pull on the tibia posteriorly and cause stress on the PCL graft [20].

## **Return to Activities**

Grade I and Grade II tears typically heal quickly and most athletes can return to sports in 4–6 weeks. Return to activity following PCL reconstruction ranges, but is typically between 9–12 months following surgery, and requires sport-specific functional training [20].

## Medial Collateral Ligament

### Pathology

The incidence of medial collateral ligament (MCL) injuries may be higher than reported since low grade injuries can be missed. The MCL has two components, superficial and deep. The superficial component originates at the medial femoral condyle, inserts at the proximal tibia, and attaches 5–7 cm below the joint line. The deep component is actually contiguous with the medial meniscus and consists of the tibiomeniscal and meniscofemoral ligaments. The primary function of the MCL is to prevent joint gapping during valgus stress [1].

The most common mechanism of injury is when valgus stress is placed on the knee, especially if the knee is in slight external rotation and flexion. Injury to the MCL can be from contact or noncontact injury. A direct contact blow to the lateral knee with a medially directed force will typically result in a high grade ligamentous injury. An example in sports is a football tackle from the side. This contact injury usually causes a rupture of the MCL at its femoral insertion. Noncontact injury is less common than contact injury for the MCL. A noncontact injury can occur when performing a pivoting or cutting maneuver with valgus and external forces, such as during skiing maneuvers. These injuries are more often incomplete.

### Clinical Presentation

The clinical presentation of an MCL injury is diffuse pain over the medial knee and joint line. Medial edema will start over the next few hours after injury. The edema may increase and spread to the rest of the knee joint over the next day or two after the original injury. The most common multi-ligamentous knee injury is a combined ACL and MCL injury. After injury, the presence of hemarthrosis on presentation is highly suggestive of an associated ACL–MCL injury. This association is most often with high MCL injuries, injuries

located more superiorly on the MCL. A patient will sometimes also present with a history of a reported “pop” at the time of injury. They may complain of medial joint line pain and report instability during ambulation. Meniscal tears, medial over lateral, are also associated with MCL injury as seen in the “unhappy triad” and this can alter clinical presentation and management as well [29].

## Physical Exam

Tenderness along the medial aspect of the knee is common during palpation. Valgus stress testing is the standard physical exam test used to assess the integrity of the MCL (Fig. 2.10). It is performed with the patient supine, and by placing one hand on the outside of the knee palpating the medial joint line, while applying an abducting force at the foot and a valgus force through the knee. If medial gapping is felt compared to the opposite knee, there is MCL injury. Valgus stress testing is typically performed at both 30° and 0° degrees of knee flexion to isolate certain structures. At 30 degrees of knee flexion, the superficial MCL is isolated. Tears are divided into grade I, grade II, and grade III (Table 2.3).

If the test is performed at 0 degrees of knee extension, other structures are included in the valgus stress. When medial laxity is felt with valgus stress at 0 degrees of knee flexion, this indicates combined crucial ligament injury or posteromedial joint capsule injury [9]. Special provocative tests for the ACL, PCL, and medial meniscus should also be performed during the physical exam to evaluate for additional injuries.

## Diagnostic Studies

Radiographs are typically normal in MCL injuries but should always be taken after a knee injury to rule out other serious pathologies. Calcifications at the medial femoral insertion site can be seen in X-rays, and are from chronic MCL injury or deficiency. The combination of this calcification and medial knee pain is diagnosed as Pellegrini–Stieda syndrome (Fig. 2.11). When eval-



**Fig. 2.10** Valgus stress testing with full knee extension can elicit MCL laxity and injury [3]

**Table 2.3** Grading MCL tears based on medial joint line gap compared to uninjured side [29]

Grade I (mild)	1–4 mm gap
Grade II (moderate)	5–9 mm gap
Grade III (severe)	>10 mm gap

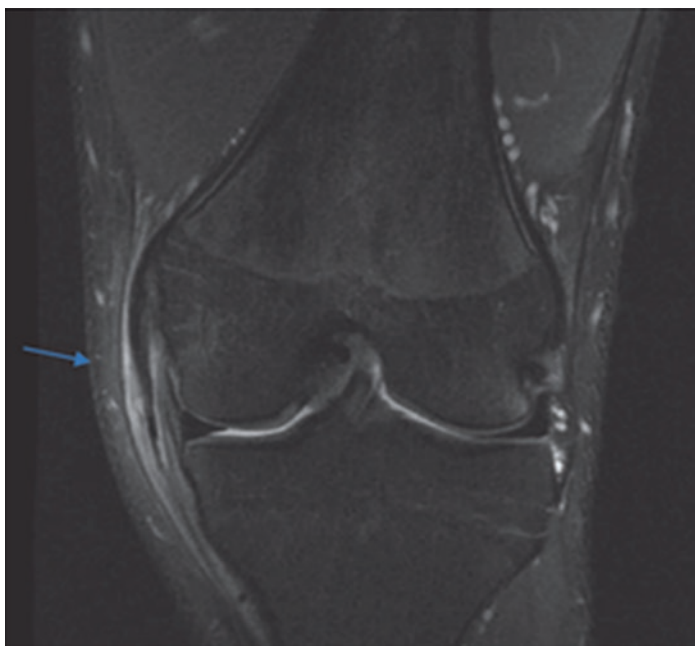


**Fig. 2.11** AP radiograph demonstrating a Pellegrini–Stieda lesion: calcification at the femoral attachment site of the medial collateral ligament [30]



uating MCL injury in adolescents, X-rays are important since valgus stress views may reveal gapping through a physeal injury [29]. An MRI is the diagnostic imaging of choice for MCL injuries, especially since an MCL injury frequently involves other ligament injuries as well. Coronal and Axial T1 and T2 MRI images are useful in distinguishing edema within the MCL fibers (Fig. 2.12).

Ultrasound is a quick, low-cost tool to assess MCL tears. Ultrasound can identify MCL pathology by abnormal sonographic appearance of the ligament (examples include heterogeneous fiber quality, hypoechoic fluid, hyperemia, cortical irregularity at



**Fig. 2.12** MRI coronal (anterior–posterior view) T2 weighted shows edema (bright signal) within the MCL indicating a Grade III tear [31]

attachment sites) and quantitative assessment of joint gapping with dynamic testing. The role of ultrasound in the assessment of suspected MCL tears is not routinely adapted and MRIs are still needed to confirm findings [32].

## Treatment

Treatment of MCL injury is divided into non-operative and operative. Non-operative treatment includes rest, physical therapy, and bracing. Grade I injuries do not need bracing, but rehabilitation is still important. Physical therapy can start with isometric quadriceps contractions and progress to isotonic exercises. Gradually, the range of motion and resistance can be increased. Grade II and Grade III injuries can benefit from bracing. Grade III will only be braced if the knee is stable with valgus stress in full extension and there is no associated cruciate injury. Bracing typically includes a knee immobilizer for comfort and a hinged knee brace for ambulation.

There have been few studies on the use of steroids in MCL injury. A total of 34 patients with chronic pain following grade I or grade II MCL injury were treated with ultrasound-guided injection of local anesthetic and steroid into the deep MCL and were allowed to return to sports immediately. While four were excluded from follow-up and four were lost all patients reported an immediate and sustained resolution of their medial knee pain. At mean follow-up of about 20 months, all were back to their pre-injury level of work. Hence, steroid injections in patients with persistent medial joint pain following grade I/II MCL sprain could be useful but not first line [33].

Similar to PCL injury, there have been case reports of prolotherapy aiding in MCL injury recovery. One case study included a rugby player, with a grade 2 MCL injury, who underwent three prolotherapy injections of 15% dextrose, in 1-week intervals. In this case, after prolotherapy and 3 weeks of physical therapy, the patient was pain free, with full knee range of motion [34].

Platelet-rich-plasma (PRP) injections have been used in MCL injury, but the evidence is limited and conflicting. One case study

published was of a 30-year-old professional wrestler with a Grade III MCL injury. Leukocyte-rich (LR) PRP injections combined with rehabilitation was used for his treatment regimen. The patient received a series of three LR-PRP injections evenly spaced 1 week apart with ultrasound guidance. In his case, the use of LR-PRP and early rehabilitation for an isolated MCL tear was beneficial and cut down his total anticipated treatment time. Further research is necessary on PRP but the evidence for rehabilitation is still strong [35].

Operative treatment consists of ligament repair or reconstruction. Repair in grade III injuries is appropriate in the setting of multi-ligamentous knee injury or if there is continued instability despite rehabilitation. Reconstruction is typically performed in chronic MCL injury or if there is loss of adequate tissue for repair.

The use of prophylactic knee bracing to prevent injury or re-injury to the MCL is controversial and studies have mixed results with benefits including stability and kinesthetic reminders to avoid pivoting motions. In some instances, however, disadvantages include weakened surrounding muscles from underuse, which may really impact an athlete depending on a player's specific position in a sport [36].

## **Return to Activities**

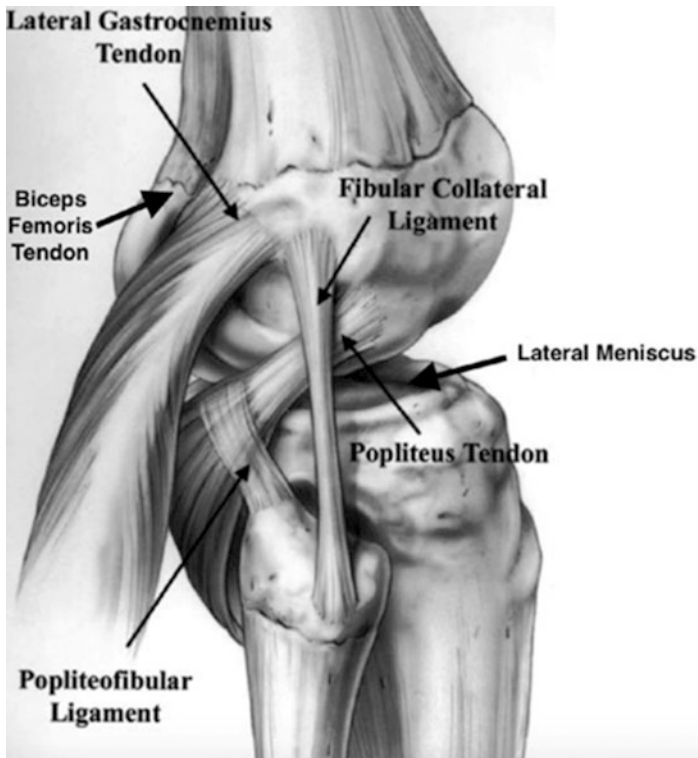
Return to activity following an isolated MCL injury can be rapid. A Grade I injury may return to play as early as 7 days, and a Grade II injury may return to play as early as 3–4 weeks.

A Grade III injury may return to play as early as 5–7 weeks, but this varies depending on if reconstruction was done or if other ligaments were also injured [37]. As previously stated, the appropriate time to resume activity after ligamentous injury varies. It is dictated by a combination of physical factors such as activity level, age, type of sport played by athletes and psychological factors such as ability to rehabilitate and cognitive ability.

## Lateral Collateral Ligament

### Pathology

Isolated lateral or fibular collateral ligament (LCL/FCL) injury is extremely rare, about less than 2% of knee injuries. Many LCL injuries are associated with injury of the posterolateral corner as well. LCL injuries can also be associated with ACL, PCL, or lateral meniscal injuries. The LCL originates at the lateral femoral condyle and inserts on the fibular head (Fig. 2.13). The primary function of the LCL is to prevent joint gapping during varus stress [1].



**Fig. 2.13** LCL/FCL and the posterolateral corner. The primary static stabilizers of the posterolateral corner include the lateral (fibular) collateral ligament, popliteofibular ligament, and popliteus tendon [38]

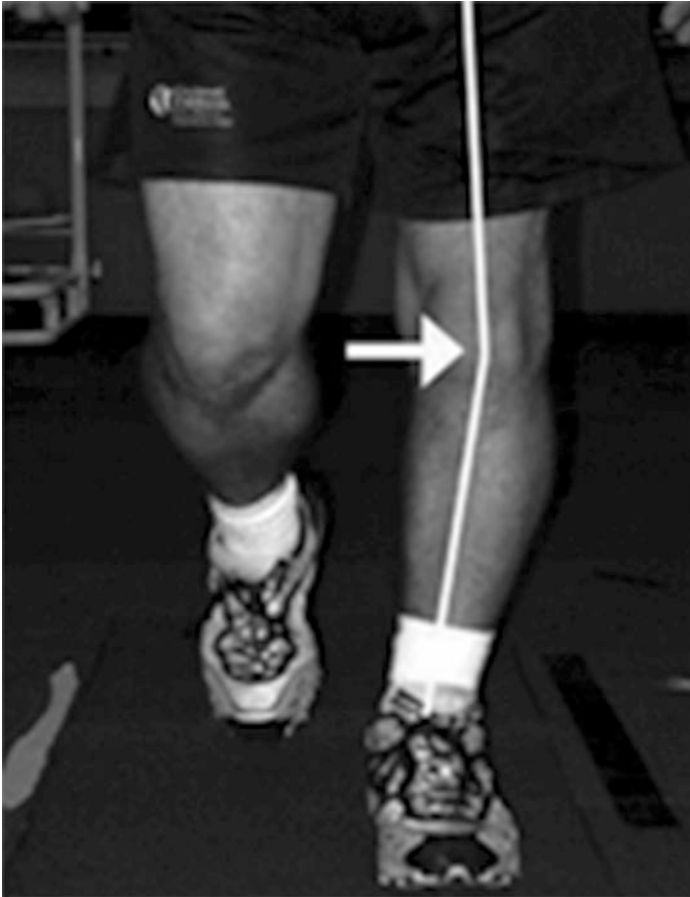
LCL injury can be from a traumatic direct blow to the medial side of the knee or excessive varus stress, excessive tibial rotation, or hyperextension. Noncontact injury to the LCL is also possible from a sudden varus moment while the knee is hyperextending. For example, weightlifters with poor lateral knee stability can endure this type of injury [39].

## Clinical Presentation

The clinical presentation of a LCL injury is pain diffusely over the lateral knee. Patients will typically complain of a sudden onset of lateral knee pain, swelling, and bruising after the injury. Swelling can happen immediately following the injury or develop up to a few hours after the injury has occurred, spreading along the rest of the knee joint. An example of a contact injury presentation is a football player suffering a blow to the medial aspect of the knee while the foot is planted and knee extended. On presentation, they will report lateral or posterolateral knee pain. Another presentation can be due to a noncontact injury when there is a sudden varus lateral movement while the knee is hyperextending, for example, during a weightlifter's heavy lift. Symptoms that patients typically complain of are instability near full knee extension, difficulty going up and down stairs, difficulty with pivoting, and lateral joint line pain. If additional ligaments are involved in the injury, one may experience additional symptoms such as instability and other areas of pain.

## Physical Exam

On initial inspection ecchymosis and lateral joint soft tissue swelling can be observed. Those with concurrent posterolateral corner injury may demonstrate a varus thrust gait (Fig. 2.14). This gait can be observed during foot strike when a gap develops in the lateral aspect of the knee. Because of this, the patient ends up shifting his/her weight during gait to reduce the knee back to normal alignment. On palpation, patients will report tenderness along the lateral knee.



**Fig. 2.14** Varus thrust gait [40]

Varus stress testing is the standard physical exam test used for the LCL (Fig. 2.15). It is performed with the patient supine, and the examiner places one hand on the lateral joint line while applying an adducting force at the foot and a varus force through the knee. If lateral gapping is felt compared to the opposite knee, there is likely an LCL injury. LCL injuries can be graded based on



**Fig. 2.15** Varus testing with knee in 30 degrees of flexion and palpation of lateral knee [38]

lateral joint line gap during varus stress compared to uninjured side (Table 2.4). Varus stress testing is typically performed at both 30° and 0° degrees of knee flexion to isolate certain structures. Varus instability at 30 degrees of flexion only would indicate an isolated LCL injury, and varus instability at both 0 and 30 degrees of flexion would indicate a combined LCL +/- ACL/PCL injury.

Another provocative test used during physical exam is the dial test (Fig. 2.16) (please see PCL section for further details).

**Table 2.4** Grading LCL tears based on lateral joint line gap seen with varus stress compared to uninjured side [38]

Grade I (mild)	0–5 mm gap
Grade II (moderate)	Partial tear 6–10 mm gap
Grade III (severe)	Complete tear >10 mm gap associated with posterolateral corner injury



**Fig. 2.16** Physical exam maneuver dial test performed to diagnose LCL injury [38]

## Diagnostic Studies

Radiographs cannot show direct damage to the LCL, but they can show small fractures that can increase suspicion for LCL injury. Radiographs can show a Second fracture, an avulsion fracture of



the lateral tibial plateau typically associated with ACL injury, but can also indicate LCL injury. Varus stress radiographs can also be helpful in the diagnosis of LCL injury. The evidence is not strong, but one may see asymmetric lateral joint line widening which is increased in the lateral joint line when placed under a varus stress. MRI is still the most useful imaging when assessing for LCL injury because it is the modality of choice to also grade severity and particular location of LCL injury.

Musculoskeletal ultrasound can also be used but is technician dependent. The LCL with a grade I or II injury can show a thickened and hypoechoic LCL. A grade III injury can show associated edema, laxity at the lateral joint line and hypoechoic thickening of the LCL with a lack of fiber continuity [39].

## Treatment

Similar to all other knee ligament injuries, acute LCL injury is treated with standard interventions, including ice, compression, rest, and analgesics. However, ice should not be applied for longer than 15 min if suspecting LCL injury because of its proximity to the common peroneal nerve. Paresthesia in the distribution of the peroneal nerve, or prolonged foot drop may occur with excessive cryotherapy.

Treatment of LCL injury is divided into non-operative and operative. Non-operative treatment is ideal for isolated grade I or grade II LCL injuries with no instability at 0 degrees of knee flexion, and consists of functional rehabilitation. Physical therapy emphasis is on quadriceps and hamstring strengthening.

Since LCL injuries are typically associated with other knee ligament injuries (such as ACL, PCL) research on isolated steroid or prolotherapy injections to the LCL is slim. However, as demonstrated earlier in this chapter, there has been some efficacy for prolotherapy injections with other knee ligaments, so a comprehensive prolotherapy treatment to the knee as whole could be used for LCL injury.

While it appears that the use of orthobiologics may be similarly applied for LCL injuries, there appears to be sparse published data with isolated LCL injuries.

Operative treatment includes LCL repair or LCL reconstruction for grade III injuries. LCL repair has higher failure rates [39]. The semitendinosus tendon autograft is the preferred graft for LCL reconstruction due to the length of the LCL. In addition, the semitendinosus tendon is closer in anatomical size as compared to other sources for grafts. If there is also a PLC injury, reconstruction may be necessary using other grafts, such as the hamstring or the Achilles tendon. PLC reconstruction involves the LCL, popliteus tendon, and the popliteofibular ligament [41].

For a Grade I injury, hinged bracing is necessary for 4–5 weeks during all weight bearing and physical therapy exercises. Quadriceps and hamstrings strengthening exercises start light and will then progress [39].

For Grade II, physical therapy is typically started on week two, with a hinged brace also worn at all times. Physical therapy range of motion exercises will start with non-weight bearing at week two and then progress to full weight bearing at week three, and continue for up to 16 weeks or as long as needed [39].

## **Return to Activities**

As mentioned throughout this chapter, return to activity is based on many factors. Criteria for return to sport after an LCL injury include full painless knee motion, absence of significant tenderness or ligamentous laxity on exam, quadriceps and hamstring strength that is at least 90% of the unaffected lower extremity, and ability to complete sports-specific warm-up exercises without pain or difficulty.

In general, grade I return to activity is about 4 weeks, grade II is about 10 weeks, and grade III is about 10–14 weeks plus surgical recovery time [39].

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