



Knee Dislocation in Athletes

16

Paul Herickhoff and Marc Safran

16.1 Introduction

Tibiofemoral dislocation is an uncommon but devastating injury to athletes. Less than 0.02% of all musculoskeletal injuries are knee dislocations [1]. In high school athletes, the knee represents 16% of all dislocated joints [2]. Three of the four major ligaments of the knee are typically injured, and there is often concomitant vascular or nerve injury or fracture [3]. Due to the potentially limb-threatening nature of this injury, knee dislocation represents a true orthopedic emergency, requiring expeditious evaluation and treatment [4].

Historically, treatment was primarily limited to closed reduction and casting or cast-brace immobilization. However, with the advent of better instrumentation, an improved understanding of the anatomy of the knee, and new reconstruction techniques, combined anterior and posterior cruciate ligament (ACL/PCL) tears with medial and/or lateral collateral ligament (MCL/LCL) disruption in the athlete is almost always managed surgically [5]. Due to the relative infrequency of these injuries, and the heterogeneity of

injury patterns, there is little conclusive, high-level, scientific evidence to guide management. The purpose of this chapter is to review relevant knee anatomy, classification systems, mechanisms of injury, evaluation, treatment, rehabilitation, and outcomes of knee dislocations in athletes.

16.1.1 Anatomy

The anterior and posterior cruciate ligaments (ACL and PCL) and medial and lateral collateral ligaments (MCL and LCL) are the primary ligamentous stabilizers of the knee. The ACL is composed of two functional bundles, named anteromedial (AM) and posterolateral (PL), for their tibial attachment sites. For single-bundle ACL reconstruction (the recommended treatment for ACL tears resulting from knee dislocation), the centerpoint of the ACL tibial footprint is located 15 mm anterior to the PCL and two-fifths of the medial-lateral interspinous distance [6]. The centerpoint of ACL femoral footprint is 24.8% of the proximal to distal distance measured from the proximal femoral cortex and 28.5% of the anterior to posterior distance, measured from the top of the notch [7].

The PCL is comprised of the anterolateral (AL) and posteromedial (PM) bundles, also named for their tibial attachments. For non-repairable PCL tears resulting from knee

P. Herickhoff
Department of Orthopaedic Surgery, Pennsylvania
State University, State College, PA, USA
e-mail: pherickhoff@pennstatehealth.psu.edu

M. Safran (✉)
Department of Orthopaedic Surgery, Stanford
University, Redwood City, CA, USA
e-mail: Msafra@Stanford.edu

dislocation, single-bundle reconstruction is also the recommended treatment. The centerpoint of the PCL tibial footprint is 7.4 mm anterior to the posterior tibial cortex or “champagne drop off” point and one-third of the medial to lateral distance between the medial groove and lateral cartilage point of the lateral tibial plateau. The centerpoint of the AL bundle is typically the chosen site for the femoral tunnel in single-bundle PCL reconstruction since its cross-sectional area is twice that of the PM bundle. The center of the AL bundle on the femur is 7.9 mm proximal to the distal articular cartilage and 7.4 mm posterior from the trochlear point, where the cartilage turns medially at the roof of the intercondylar notch [8].

The MCL of the knee has superficial and deep components. The superficial MCL femoral attachment is found in a depression 3.2 mm proximal and 4.8 mm posterior to the medial epicondyle. The superficial MCL has two distinct tibial attachments. The proximal attachment is to the semimembranosus anterior arm, while the distal attachment is broad-based, lies anterior to the posteromedial crest of the tibia, and forms the floor of the pes anserine bursa, approximately 6 cm distal to the joint line. The deep MCL is a thickening of the medial joint capsule parallel to the anterior border of the superficial MCL. The deep MCL consists of distinct menisiofemoral and meniscotibial attachments. The posterior oblique ligament is the main structure of the posteromedial corner of the knee. The central arm of the posterior oblique ligament originates from the distal aspect of the main semimembranosus and attaches to the femur 1.4 mm distal and 2.9 mm anterior to the gastrocnemius tubercle [9].

The LCL attaches to the femur 1.4 mm proximal and 3.1 mm posterior to the lateral femoral epicondyle. The fibular attachment of the LCL lies 8.2 mm posterior to the anterior border of the fibular head and 28.4 mm distal to the tip of the fibular styloid process. The LCL is one of several structures comprising the posterolateral corner of the knee, which includes important secondary stabilizers of the knee, namely, the popliteus and popliteofibular ligament. The popliteus tendon

inserts into the femur 18.5 mm distal and anterior to the LCL femoral attachment. The popliteofibular ligament is composed of two bundles which attach 1.6 and 2.8 mm from the tip of the fibular styloid process, respectively [10]. Finally, the medial and lateral menisci, anterolateral capsule/ligament, and posterior capsule of the knee all provide secondary stabilization of the knee [11].

The popliteal artery is an “end artery” to the leg, with minimal collateral circulation through the superior and inferior genicular branches. It travels through the popliteal fossa, just posterior to the PCL and posterior horn of the lateral meniscus, and is the closest neurovascular structure to the knee. The distance between the popliteal artery and PCL increases from 5.4 mm with the knee in full extension to 9.3 mm with the knee at 100° of flexion [12]. The popliteal artery and vein are tethered proximally at the adductor hiatus, or Hunter’s canal, and distally at the soleus arch, which increases their risk of injury with knee dislocation.

The sciatic nerve branches into the tibial and peroneal nerves in the popliteal fossa. The peroneal nerve travels laterally around the neck of the fibula and has less excursion than the tibial nerve, placing it at increased risk of injury with knee dislocation, particularly varus injury patterns [13]. Kadiyala and colleagues also demonstrated the precarious blood supply of the common peroneal nerve caused by its lack of intraneural vessels in the region of the fibular neck [14].

16.1.2 Classification

Historically, Kennedy classified knee dislocations into five types (anterior, posterior, medial, lateral, and rotatory) based on the direction of dislocation of the tibia relative to the femur [15]. Although this information is useful when the knee remains dislocated, knee dislocations often spontaneously reduce, which is typically the case in athletes, so the true direction of dislocation may be unknown. The major limitation of the Kennedy classification, however, is the variability in injured ligaments when only accounting for the direction of dislocation.

Table 16.1 Schenck classification of knee dislocation. “C” is added for concomitant arterial injuries, while “N” is added for nerve injury

Grade	Injured structure	Intact structure
I	1 cruciate + 1 collateral	1 cruciate + 1 collateral
II	Both cruciates	Both collaterals
IIIM	Both cruciates + MCL	LCL/PLC
IIIL	Both cruciates + LCL/PLC	MCL
IV	Both cruciates, both collaterals	–
V	Fracture dislocation	

Shelbourne and colleagues classified knee dislocations as low energy or high energy based upon the mechanism of injury [16]. Knee dislocations from sporting activities, with the exception of water-skiing, were classified as low energy, while motor vehicle collisions (MVC), crush injuries, and fall from high distances were classified as high-energy dislocations. Shelbourne reported only a 4.8% rate of vascular injury in low-energy dislocations. In contrast, McCoy and colleagues reported four cases of low-energy knee dislocation, three of which sustained vascular injuries requiring surgical repair [17]. Therefore, the physician must maintain a high index of suspicion for vascular injury in knee dislocation regardless of the mechanism of injury.

The most widely used and accepted classification of knee dislocations was developed by Schenck and is based on which anatomical structures are injured [18]. The Schenck classification accounts for injured ligaments, nerve and arterial injuries, and fractures. A summary of the Schenck classification is shown in Table 16.1.

16.1.3 Mechanism of Injury

Hyperextension of the knee, from either contact or noncontact injury, classically causes anterior dislocation of the knee and rupture of both cruciate ligaments and is a high-risk pattern for neurovascular injury due to stretch on the posterior structures of the knee [13]. In a cadaver study, Kennedy demonstrated that hyperextension of the knee past 30° resulted in tearing of

the posterior capsule, while hyperextension to 50° ruptured the PCL and popliteal artery in all specimens [15].

A violent blow to the anterior proximal tibia of the flexed knee and falling directly onto a flexed knee are the typical patterns for posterior dislocations of the knee. Medial and lateral dislocations result from a varus or valgus force, often seen in contact sports such as rugby or American football, while rotatory dislocations result from twisting in combination with a varus/valgus stress or hyperextension, as may occur in skiing, wrestling, basketball, handball, or soccer [13]. In contact sports such as American football where multiple athletes are simultaneously competing with one another, the team physicians and athletic trainers may not witness the injury in real time. Therefore, asking the player “what happened to your knee” and reviewing instant replay video footage may be extremely helpful for making the diagnosis of knee dislocation.

16.1.4 Evaluation

16.1.4.1 General Considerations

The importance of immediate recognition of knee dislocation lies not with the treatment of instability but the recognition of potential vascular injury or vascular compromise [13]. Green and Allen reported an amputation rate of 86% when vascular repair associated with knee dislocations is delayed more than 8 hours after injury. Conversely, 89% of limbs remained viable when vascular repairs were performed less than 8 hours after injury [19].

On the field, a brief history is obtained from the patient, focusing on the mechanism of injury, location(s) of pain, and if there is any subjective numbness in the extremity. Socks and shoes are removed and athletic shorts pulled proximally to expose both lower extremities from the mid-thigh to the toes so that an adequate physical examination may be performed.

16.1.4.2 Physical Examination

Although the diagnosis may be obvious in cases where the knee is dislocated, signs and symptoms

may be subtle in the spontaneously reduced knee. Capsular disruption may prevent the formation of a hemarthrosis, presenting instead as soft tissue swelling or bruising [20]. As soon as the diagnosis of knee dislocation is made or suspected, emergency medical services should be alerted to coordinate transportation to the hospital for further workup.

16.1.4.3 Closed Reduction

In cases where the knee dislocation has not spontaneously reduced, the physician should expeditiously palpate the patient's dorsalis pedis and posterior tibial pulses and check tibial and peroneal nerve motor and sensory function before proceeding with a closed reduction. Gentle in-line traction with non-forceful manipulation is successful in reducing the majority of tibiofemoral knee dislocations on the field. Once the knee is reduced, a detailed physical examination is performed (below), including repeating the neurovascular exam. The extremity is then placed into a knee immobilizer or long leg splint, and the patient is transported to the hospital.

Occasionally, the knee will not completely reduce or not reduce at all. Clarke first described what is now known as the dimple sign in 1942 [20]. When the knee is gently brought into extension, a worsening skin dimple between the medial femoral condyle and the medial tibial plateau indicates that the medial femoral condyle has buttonholed through the medial joint capsule and the MCL has become entrapped within the joint. Emergent open reduction in the operating room is the appropriate treatment for irreducible knee dislocations [21].

16.1.4.4 Vascular Examination

The reported incidence of popliteal artery injury varies widely in the literature, from 3.3% to 65% [16, 19, 20, 22–25]. Anterior and posterior dislocations, higher-energy injuries, and dislocations where all four major ligaments are ruptured (Schenk classification KD IV) have been shown to be at higher risk of popliteal artery injury [13, 24].

The dorsalis pedis and posterior tibial pulses are palpated on both feet simultaneously.

Asymmetry between the injured and uninjured legs and absent or weak pulses are indicative of arterial injury. In the training room, ankle-brachial index (ABI) should be obtained for both lower extremities. In an ABI examination, the patient is placed supine, and a blood pressure cuff is placed proximal to the ankle of the injured limb. Systolic pressure is determined with a Doppler probe at either the posterior tibial artery or the dorsalis pedis artery. The same measurement is made on the ipsilateral uninjured upper extremity limb. The ABI is calculated as the systolic pressure of the injured limb divided by the systolic pressure of the uninjured limb. Although the ABI measurement may be inaccurate in patients with peripheral arterial disease or vessel calcification, these conditions are rarely seen in the athlete. ABI less than 0.9 is strongly suggestive of vascular injury [5].

Controversy exists regarding the need for advanced imaging with an arteriogram, magnetic resonance arteriography, or computed tomographic angiography versus observation with serial physical examination to detect arterial injury. Part of the reasoning for this controversy is that popliteal artery intimal injuries may occur with knee dislocation and occlude many hours after the injury, resulting in a normal examination initially, only later to have occluded, resulting in vascular compromise. Some authors advocate for selective advanced imaging only in patients with abnormal physical exam or ABI [24–27], while others recommend arteriograms be obtained in every patient regardless of physical exam findings [19, 28]. It has been our practice to obtain computed tomography (CT) angiography in all athletes with knee dislocation, regardless of the physical examination or ABI, since, in our opinion, the potential benefits far outweigh the risks.

16.1.4.5 Neurologic Examination

The reported incidence of nerve injury associated with knee dislocation varies widely in the literature from 5% to 40.0% [15, 23, 29–32]. Most commonly, the common peroneal is the injured nerve, though isolated tibial nerve palsy has been reported [33]. The reported rate of peroneal nerve recovery after knee dislocation also varies; however, a gen-

eral rule is that approximately one-third of injuries regain complete function, one-third regain partial function, and one-third do not regain any function [34, 35]. Multiple authors have demonstrated improved outcomes in patients with partial versus complete nerve injuries [36, 37]. A detailed neurologic examination includes sensation in the tibial, deep peroneal, and superficial peroneal distributions to light touch, pinprick, and temperature if available. Motor examination tests the flexor and extensor hallucis longus, tibialis anterior, and gastrocnemius to document baseline function [20].

16.1.4.6 Ligament Examination

Ligament testing in the acute multiple ligament-injured knee is often limited due to pain, and one must avoid inadvertent redislocation (e.g., by hyperextending the knee). ACL tears are diagnosed using Lachman's maneuver with the knee at 20° of flexion, while the PCL is evaluated with a posterior drawer test with the knee bent to 90°. Gentle testing of the collateral ligaments should be performed at 0 and 30° of flexion. Performing a dial test with external rotation of the feet at 30 and 90° of flexion is often difficult because of pain but is the best way to clinically evaluate the posterolateral corner [4].

16.1.4.7 Imaging

AP and lateral radiographs of the knee should be obtained to evaluate for fracture and to assess the joint reduction. Widening of the medial or lateral joint space in the AP view may be seen in cases of collateral ligament injury. PCL ruptured knees often demonstrate posterior sagging of the proximal tibia on the lateral view, which may be corrected by placing a soft, rolled blanket under the calf (with the splint or knee immobilizer in place). This maneuver helps remove tension off the posterior neurovascular structures. However, great care must be taken to ensure the rolled blanket does not migrate proximally up to the popliteal fossa, as this could cause obstruction of the popliteal artery and vein.

Magnetic resonance imaging is performed in all athletes with knee dislocation. MRI should be scrutinized not only for damage to the four major ligaments (ACL, PCL, MCL, LCL) but also for

posterior capsular tears; avulsion injuries of the iliotibial band, biceps femoris, popliteus, and posterolateral corner; and extensor mechanism injuries.

16.1.5 Treatment

16.1.5.1 Arterial Injuries

Vascular surgery is consulted for emergent evaluation and treatment of all popliteal artery injuries. A dialogue between the orthopedic and vascular surgeon is necessary so that incisions made by the vascular surgeon will be appropriately placed for later ligament reconstruction [4]. The vascular surgeon may also request knee-spanning external fixation be placed to protect the arterial repair. After the leg is prepped and draped, two self-drilling, self-tapping, 5.0-mm-diameter pins are placed bicortically into the femur through stab incisions on the anterolateral aspect of the thigh. The pins should be spaced as far apart as possible to increase the stability of the construct; however, the pin closest to the knee must be at least 7.5 cm above the superior pole of the patella to avoid the suprapatellar pouch, in addition to avoiding future skin incisions [38]. Next, two self-drilling, self-tapping, 5.0-mm-diameter pins are placed bicortically into the tibia through small stab incisions over the anteromedial tibia. Similar to the femur, the pins are maximally spaced, with the most proximal pin at least 10 cm below the knee joint line to avoid future skin incisions. Standard radiolucent bars and pin-to-bar and bar-to-bar connectors are utilized to fashion the spanning external fixation construct. Finally, a large C-arm fluoroscope is brought in to verify proper pin depth and reduction of the knee.

16.1.6 Ligament Injuries

16.1.6.1 General Considerations

Goals of treatment include restoration of knee stability, full range of motion, and return of the athlete to their pre-injury level of play. Surgical treatment is recommended for all athletes, as several studies have demonstrated superiority over

nonsurgical treatment of knee dislocations [39–41]. Optimal timing of surgery is 10–14 days after injury, when the soft tissues are amenable to repair and capsular tears have sufficiently healed to prevent fluid extravasation from the knee. It is not uncommon, however, for knee dislocations to initially be unrecognized or misdiagnosed and present several weeks out from injury, especially high school and recreational athletes without a team physician trained in musculoskeletal medicine. This becomes important in planning surgical treatment, as ligament contracture and scarring of the tissue planes prohibits repair of MCL/posteromedial corner and LCL/posterolateral corner when surgery is delayed more than 3 weeks from injury, and it becomes necessary to reconstruct these structures. The PCL and ACL are typically reconstructed regardless of time from injury to surgery, although repair of the PCL may be possible when avulsed from the femoral side or bony avulsion from the tibial side, and acute tibial eminence avulsion fractures of the ACL may also be amenable to repair. For all ligament reconstructions, allograft tissue is favored over autograft since multiple ligaments require reconstruction, autograft sources are limited, and donor site morbidity may be avoided, to minimize the duration of surgery and avoid additional trauma to the knee.

If the athlete is a high-level athlete, and good postoperative rehabilitation is available, then the senior author prefers to repair the collateral ligament(s)/corners, along with cruciate ligament reconstruction at 10–14 days post injury. There is some controversy about the outcomes of primary repair versus reconstruction acutely, as outcomes seem better for reconstruction of the collateral ligaments/corner than repair [42–44]. The senior author prefers repair and will augment the repair with allograft tissue for collateral ligament injury/corner injury, as the anatomy is more complex than just what is reconstructed with a graft. If the knee dislocation is not in an athlete, and/or rehabilitation by an experienced therapist is not available, it is the senior author's preference to reconstruct the PCL and fix/reconstruct the collateral ligament(s)/corner(s) first. Later, after the collateral ligament and corner are healed and the

patient recovered from the PCL reconstruction, the ACL can be reconstructed if the patient has instability from insufficiency of the ACL. This approach is preferred, since rehabilitation is less complicated when compared to rehabilitation after reconstructing both cruciates. The PCL is reconstructed first, with the collateral ligament and corner, to reduce the knee in its anatomic tibiofemoral relationship, taking stress off the collateral ligament and corner. Reconstructing the ACL with the PCL torn may result in problems with appropriate ACL tensioning and/or tibiofemoral relationship, and the posterior sag will result in stretching out of the repaired/reconstructed collateral ligament(s)/corner(s).

Informed consent for surgery is obtained from the patient and/or family member and includes a review of the potential risks of surgery with the patient and family members, including neurovascular injury with subsequent risk of bleeding and possible need for transfusion, infection, stiffness, recurrent instability, arthritis, and need for further surgery. Multiligament knee surgeries are scheduled as “first-start” cases, allograft tissue is ordered, and vascular surgery is contacted to ensure their availability should any question of limb perfusion arises during the case.

16.1.7 Surgical Technique

16.1.7.1 Anesthesia and Room Setup

General anesthesia is preferred in all cases, and preoperative femoral and sciatic nerve blocks are often administered to aid postoperative pain control. Surgical instrument tables are situated on the side of the operative leg, while suction, the arthroscopy fluid tower, and C-arm fluoroscopy are positioned on the opposite side of the room. Exam under anesthesia is performed to confirm the ligament injuries suspected on preoperative exam and MRI and includes palpation of the posterior tibial and dorsalis pedis pulses. Doppler ultrasound should be available and utilized when pulses are not easily palpable.

A non-sterile tourniquet is placed as high as possible on the thigh, but is not typically used during the procedure. Care is taken to not create

a venous tourniquet that may occur by wrapping the tourniquet tightly. The non-operative leg placed in an Allen stirrup with no varus or valgus stress on the knee, padding of the lateral leg to protect the peroneal nerve, and knee and hip flexion of 30–45°, each. The operative leg is placed in a circumferential thigh holder at the level of the upper to mid-thigh. Using sterile technique, the operative knee is insufflated with 60 cc of normal saline via a lateral mid-patellar approach. The foot of the bed is then lowered all the way and the table padding removed, which allows flexion of the knee to 120°.

The leg is prepped and draped using a sterile technique. Bony anatomy and skin incisions are identified with a skin marker. The anterolateral portal is just above the joint line, 5 mm lateral to the patellar tendon, while the anteromedial portal is at the same level, 1 cm medial to the patellar tendon. The tibial tubercle is identified, and the incision for ACL and PCL reconstructions is marked starting 2 cm medial to the tibial tubercle and extending distally 4 cm. This incision can be used in conjunction with a 2 cm incision centered over the medial epicondyle for MCL reconstruction or can be extended to the distal aspect of the medial epicondyle if a single incision is desired. For LCL repair or reconstruction, a hockey stick incision approximately 12 cm in length is marked out along the mid-IT band, curving distally at the lateral epicondyle and ending midway between Gerdy's tubercle and the fibular head.

16.1.7.2 Diagnostic Arthroscopy

Diagnostic arthroscopy is performed expeditiously, with gravity inflow instead of a pump to minimize the risk of fluid extravasation and compartment syndrome. Care should be taken throughout the case to ensure the calf muscle is supple; should firmness of the lower leg develop at any point during the case, fluid inflow is turned off, and dry arthroscopy is used for the remainder of the case.

A superolateral outflow cannula is placed, and the anterolateral and anteromedial portal incisions are made. Some prefer superomedial outflow cannula, particularly if a lateral collateral/posterolateral corner reconstruction is to be per-

formed. A 30-degree arthroscope with gravity inflow attached is inserted in the anterolateral portal, and an arthroscopic probe is placed in the medial portal. Pictures are taken of the patellofemoral joint, gutters, and medial and lateral compartments, to document all ligament, cartilage, and meniscus injuries. Chondral lesions are debrided to a stable base. Meniscal tears with the capacity to heal are fixed using all-inside or inside-out sutures depending on the length and pattern of the tear.

16.1.7.3 Ligament Repair or Reconstruction

We recommend addressing the PCL and ACL first by drilling the tunnels, passing the grafts, and fixing them on the femoral side before proceeding with open repair or reconstruction of the MCL/posteromedial corner and LCL/posterolateral corner. Bone patellar tendon bone allografts are preferred for ACL and PCL reconstructions, while Achilles tendon allografts are preferred for MCL and LCL reconstructions. It is important to have a trained surgical assistant begin preparing the allografts as soon as the patient enters the operating room to avoid delays in the surgical procedure.

16.1.8 Posterior Cruciate Ligament

16.1.8.1 Repair

Arthroscopic repair of femoral avulsions of the PCL is our preferred treatment strategy, since it is faster and safer than PCL reconstruction, and can better replicate the anatomy (and potentially function) as compared with PCL reconstruction, while outcomes are comparable to, or better than, PCL reconstruction [45, 46].

After debridement of the ACL stump, the femoral insertion of the PCL is debrided to bleeding bone using an arthroscopic shaver. An arthroscopic suture passing device is used to pass a high strength, nonabsorbable suture through the PCL, typically grasping the tissue just above the medial tibial spine. The suture is reloaded and passed a second time through the PCL for a locking Bunnell suture configuration. The suture is

shuttled out the anterolateral portal, and then another different-colored high-strength nonabsorbable suture is passed in similar fashion, just proximal to the first suture. A 2 cm incision is made along the posterior border of the vastus medialis, 3 cm proximal to the joint line. A PCL guide is placed through this incision and the anteromedial portal, and two bone tunnels, approximately 8–10 mm apart, are drilled retrograde using a 2.4 mm pin into the PCL femoral insertion. A Hewson suture passer is then used to shuttle each set of sutures out their respective bone tunnels, where they will eventually be tied over a button.

16.1.8.2 Reconstruction

Reconstruction of the PCL is our preferred treatment strategy for midsubstance ruptures and non-bony tibial PCL avulsions. An 11-mm-diameter bone-patellar tendon-bone allograft is fashioned on the back table with the femoral bone plug 20 mm in length and the tibial bone plug 25–30 mm in length. It is critical that the femoral bone plug be no more than 20 mm in length in order to facilitate passing the graft into the knee around the “killer turn.” After debridement of the ACL remnant and anterior fibers of the PCL, a posteromedial portal is created under direct visualization using a spinal needle for localization, and a 5.5 mm cannula is inserted. A full-radius shaver is used through the posteromedial portal to resect the PCL to its tibial insertion. Switching to a 70-degree lens helps to visualize the tibial insertion with the arthroscope in the anterolateral portal.

The previously marked 4 cm incision is made on the anteromedial proximal tibia. The PCL tibial guide is placed through the anteromedial portal with the tip toward the lateral aspect of the PCL tibial insertion 7.4 mm anterior to the posterior tibial cortex and one-third of the medial to lateral distance between the medial groove and lateral cartilage point. The angle of the PCL guide is maximally opened (usually around 65°) so that the bullet of the guide is 6–7 cm below the joint line. Opening the guide to at least 65° will ensure adequate spacing between the PCL and ACL tibial tunnels and minimizes the angle

of the so-called killer turn at the tibial tunnel aperture into the joint. A guidewire is then carefully drilled into the joint under direct visualization, choking up on the wire with the wire driver and gently drilling through the far cortex to prevent plunging into the joint and nearby neurovascular structures. Some PCL tibial guides have a flat surface to catch the guidewire, to reduce neurovascular injury by the guidewire. Fluoroscopy is brought in to check the position of the wire. After the proper position of the wire is confirmed, an 11 mm cannulated fully fluted reamer is then used to drill the tibial tunnel under arthroscopic visualization while covering the tip of the guidewire with a curette or a PCL tibial guidewire protector. Once the reamer contacts the far cortex, remove the guidewire before reaming into the joint.

The femoral PCL guide enters the joint through the anteromedial portal with the tip placed 7.9 mm proximal to the distal articular cartilage and 7.4 mm posterior from the trochlear point. A 2 cm incision is made along the posterior border of the vastus medialis, about 3 cm above the joint line, and the bullet of the guide is placed on the femur. A Beath needle is drilled into the joint outside-in, followed by retrograde drilling of the femoral tunnel with a cannulated 11 mm fully fluted reamer. To ensure that the graft will pass smoothly into the joint and femoral tunnels, the periosteum at all four apertures of the tunnels is liberally cleared with electrocautery. An 18-gauge metal wire is used to shuttle the sutures for the graft through the tunnels by bending the wire in half and pulling it retrograde through the femoral and tibial tunnels so that the looped end of the wire rests at the proximal tibia. The BPTB shuttling sutures and graft are then pulled up the tibial tunnel and through the joint until the femoral bone plug sits flush in the femoral tunnel. An 7 x 20-mm-diameter metal femoral interference screw is placed in the femoral tunnel from outside-in to fix the graft in the femur. When performing collateral ligament repair/reconstruction, PCL reconstruction is halted at this point to address these other structures. Once the ACL is fixed on the femoral side, and the collateral ligaments/corners are prepared for fixation, attention

is returned to the PCL. While performing an anterior drawer maneuver with the knee at 90° of flexion such that the proximal medial tibia is 1 cm anterior to the medial femoral condyle, a 9 × 20 mm metal interference screw is placed into the tibial tunnel under direct visualization to secure the PCL graft in place.

16.1.8.3 Anterior Cruciate Ligament Reconstruction

A 10-mm bone-patellar tendon-bone allograft is fashioned on the back table with a 20-mm femoral bone plug and 25-mm tibial bone plug. To prevent graft-tunnel mismatch, the tendinous length of the allograft should be matched to the patient's height following the algorithm of Brown et al. [47]. A notchplasty is performed to create an upside-down "U"-shaped notch and allow clear visualization of the back wall. The tibial drill guide is inserted through the anteromedial portal with the tip two-fifths of the medial-lateral interspinous distance, centered in the remaining ACL footprint. The ACL drill guide is placed on the tibia so that after reaming the tibial tunnel, there will be at least a 1 cm bone bridge between the ACL and PCL tunnels (typically 45–50°). A Beath needle is then drilled into the joint. Once the proper location of the wire is confirmed, a cannulated, fully fluted, constant diameter 10 mm reamer is used to create the tibial tunnel.

A spinal needle is placed through the anteromedial portal to visualize the trajectory toward the femoral insertion of the ACL. Frequently, it is necessary to create an accessory anteromedial portal which is more medial and distal than the original AM portal to improve the angle of drilling the femoral tunnel and prevent blowing out the back wall. A 7 mm offset drill guide is then placed through the accessory AM portal and hooked on the back wall. The knee is flexed to 120°, and a Beath pin is drilled through the femoral insertion of the ACL, 24.8% of the proximal to distal distance measured from the proximal femoral cortex, and 28.5% of the anterior to posterior distance, measured from the top of the notch. The femoral tunnel is then drilled over the Beath pin using a cannulated hemispherical reamer to a depth of 23 mm. A passing suture is

placed through the Beath pin, which is pulled out the lateral femoral cortex. The periosteum and soft tissue at the apertures of the tibial tunnel are removed with electrocautery, and then the passing suture is pulled from the joint down and out the tibial tunnel. The BPTB graft is shuttled up into the knee and then into the femoral tunnel with the cancellous surface of the graft oriented anteriorly. A nitinol wire is placed colinearly between the cancellous surface of the graft and the anterior wall of the femoral tunnel, and a 7 mm diameter metal screw with a rounded head is inserted to fix the graft on the femoral side. After the PCL and collateral ligaments have been secured, the tibial bone plug of the ACL is secured with a 9 × 20 mm metal interference screw.

16.1.8.4 Lateral Collateral Ligament/Posterolateral Corner

Repair of the lateral collateral ligament and posterolateral corner is our preferred treatment when surgery is performed less than 3 weeks out from injury, in cases of tendon avulsions from the bone, and when the tissue quality affords a robust repair. Injury patterns may vary substantially; therefore, thoughtful review of the preoperative MRI is essential for surgical planning. Care is taken to repair all damaged structures back to their anatomic insertions using double-loaded suture anchors with heavy nonabsorbable suture. Locking, Krackow suture configuration is utilized when suturing all damaged structures.

Reconstruction of the posterolateral corner is planned for all delayed surgical procedures but is also performed in acute cases (less than 3 weeks) when the tissue quality is poor, and it is not possible to obtain a robust repair. Because the final decision on repair or reconstruction will be made intraoperatively, Achilles tendon allograft must be made available for all knee dislocation surgeries regardless of the time from injury.

Our surgical technique for reconstruction of the posterolateral corner essentially mirrors the technique described by LaPrade et al., particularly for high-grade posterolateral injuries. For low-grade posterolateral corner injuries, that is, grade 3 injuries with lower degrees of laxity, Arciero's

modification of Larson's lateral reconstruction through the fibular head is utilized to minimize the extra dissection of tissues to access the posterolateral aspect of the tibia [48]. The reader is directed to the original article referenced at the end of this chapter for a detailed description of the procedure [49]; however, an abbreviated summary of the procedure is discussed in the following paragraphs.

An Achilles tendon allograft is split into two grafts, each with a 9 mm × 18 mm bone block, and the tendinous portion trimmed to fit through a 7 mm graft sizer and tubularized with #2 nonabsorbable suture. The previously marked skin incision is made, and full-thickness skin flaps are developed above the fascia. The common peroneal nerve is identified through a fascial split posterior to the biceps femoris and approximately 2 cm below the fibular head. A neurolysis is performed so that the nerve is freely mobile and the nerve is protected throughout the remainder of the case.

The interval between the lateral head of the gastrocnemius and soleus is developed bluntly to identify the posterior fibular head and posterolateral surface of the tibia. Two incisions are made in line with the fibers of the iliotibial band, the first centered at the lateral epicondyle and the second centered 1 cm proximal to the tip of the fibula. The femoral insertion of the LCL is identified through the anterior fascial incision, 1.4 mm proximal and 3.1 mm posterior to the lateral femoral epicondyle, and a Beath pin is drilled transversely across the femur and through the skin on the medial side of the knee. The femoral insertion of the popliteus tendon is then identified 18.5 mm distal and posterior to the LCL, and a Beath pin is placed across the femur parallel to the LCL pin. Twenty millimeter length blind tunnels are then drilled over the top of each wire with a 9-mm-diameter cannulated reamer. A K-wire is then drilled from the fibular insertion of the LCL, 8.2 mm posterior to the anterior border of the fibular head and 28.4 mm distal to the tip of the fibular styloid process, aiming toward the tip of the surgeon's finger which is placed on the posteromedial downslope of the fibular styloid, at the attachment of the popliteofibular ligament. A

7 mm cannulated reamer is drilled over the K-wire through the fibular head, while a posteriorly placed retractor protects the peroneal nerve. Next, an ACL guide is placed on the bone of the proximal tibia, anterior to the popliteus, with the tip approximately 1 cm medial and 1 cm proximal to the posterior aperture of the fibular tunnel, and the bullet of the guide just distal and medial to Gerdy's tubercle. The guidewire is drilled and checked with fluoroscopy, and then a 10 mm fully fluted reamer is used to drill the tibial tunnel.

After all four tunnels have been drilled, the two Achilles allograft bone plugs are secured in the femur with 7 × 20 mm metal interference screws. The tendinous portion of both grafts are shuttled under the iliotibial band. A mosquito clamp to puncture the posterolateral capsule just above the lateral meniscus and pull the popliteus graft to the posterior aspect of the knee. The popliteus graft is then shuttled through the tibial tunnel from posterior to anterior using a Hewson suture passer to shuttle the sutures. The graft from the LCL insertion is passed superficial to the popliteus allograft tendon, passed from anterior to posterior through the fibula, and then shuttled from posterior to anterior through the tibial tunnel. After the PCL graft has been fixed to the tibia, a valgus force is applied to the knee in 20° of flexion, and the LCL graft is fixed to the fibula with a 7 × 20 mm metal interference screw. Finally, with the knee placed in 60° of flexion and neutral rotation, the tendinous ends of both grafts are fixed in the tibial tunnel with a 9 × 30 mm interference screw.

16.1.8.5 Medial Collateral Ligament/Posteromedial Corner

Similar to the lateral side of the knee, repair of the medial collateral ligament and posteromedial corner with suture anchors is our preferred technique when the MCL is avulsed from its femoral origin, the surgery is performed within 3 weeks of injury, and the tissue is of good quality for a robust repair. In all other cases, reconstruction of the MCL with an Achilles allograft as described by Marx et al. is our preferred technique [50]. The reader is referred to Marx's original article

for a detailed description of the procedure; however, key points of the operation are summarized below.

An Achilles allograft is fashioned with a 9×18 mm bone block on the back Table. A 3 cm longitudinal incision is made centered over the medial epicondyle. The fascia is cut in line with the skin incision, and a subfascial tunnel is created distally toward the proximal tibial incision, under the sartorius and hamstring tendons. The origin of the superficial MCL is identified 3.2 mm proximal and 4.8 mm posterior to the medial epicondyle, and a Beath pin is driven across the femur, aiming slightly anteriorly to avoid the intercondylar notch. A nonabsorbable suture is passed through the subfascial tunnel and wrapped around the Beath pin. The isometric point on the tibia is identified by cycling the knee, typically posterior to the pes anserine insertion and marked with electrocautery. A cannulated 9 mm reamer is used to create a 20 mm blind tunnel in the femur over the Beath pin. The periosteum overlying the aperture of the femoral tunnel is removed with electrocautery. The Achilles bone block is pulled into the femoral tunnel and fixed with a 7×20 mm metal interference screw. The tendinous portion of the allograft is passed through the subfascial tunnel. After the PCL and LCL have been secured, the knee is flexed to 20° , a varus force is applied to the knee, and the graft is fixed at the previously marked isometric point with a 4.5 mm cortical screw and 17 mm spiked washer.

16.1.8.6 Postoperative Rehabilitation

At the conclusion of the surgical procedure, the patient is placed in a hinged knee brace locked in full extension after surgery and made non-weight-bearing with crutches for 6 weeks. If a standard brace is used, a couple of towels are placed between the upper calf and brace to provide an anterior directed force, to reduce the sag on the PCL. Alternatively, a brace with an anterior directed, spring-loaded force may be applied to reduce gravity forces that may result in stretching out of the PCL. Two weeks after surgery, the patient can unlock the brace to work on range of motion from 0 to 60° . At 4 weeks postoperatively, range of motion is increased to 0– 90° . After

6 weeks, the brace is unlocked to allow full range of motion, and gradual progression of weight-bearing is allowed. The brace may be discontinued after 8 weeks when the patient has demonstrated functional quadriceps control. Running is delayed until at least 6 months after surgery. Patients typically return to sports 9–12 months after surgery.

16.1.8.7 Outcomes

Knee dislocation in athletes is an uncommon injury. Most of the available literature consists of level 4 evidence from retrospective case series with small numbers of patients and a variety of injury patterns and mechanisms, including high-energy motor vehicle collisions, low-energy sports injuries, and ultralow-energy dislocations in morbidly obese patients, limiting the applicability of some of the study findings to athletes. The prognosis for knee dislocations from sports injuries may be better than high-energy and ultralow-energy mechanisms; however, the prognosis for return to sports is fair to guard at best.

Richter and colleagues retrospectively reviewed 89 knee dislocations from all mechanisms of injury managed either conservatively or with surgery, 17 (19%) of whom dislocated their knee playing sports. Lysholm and IKDC scores were noted to be higher in the sports injury patients; however, the rate of return to sports among all patients was only 45%. Of these patients, 57% returned to the same level of play, 40% to a lower level of play, and 3% to a higher level of play [39]. Engebretsen and colleagues prospectively followed 85 patients for 2–9 years after surgical treatment of knee dislocation, of which 40 were low-energy sports-related injuries. Patients with knee dislocation performed significantly better in the triple hop test but showed similar Tegner, Lysholm, and IKDC scores and no significant difference in the one-leg hop test, crossover hop test, and 6 m timed hop test. The mean age of the sports injury patients, however, was significantly greater than the high-energy knee dislocations (47 vs. 38 years old), limiting the applicability of these findings to younger athletes [36].

Two retrospective case series have specifically been limited to knee dislocations in athletes. Shelbourne et al. reported 21 knee dislocations sustained in a variety of sports, including American football, wrestling, rugby, softball, running, and hurdling. Five patients were managed conservatively, while the other 16 underwent either repair or reconstruction of the torn ligaments. Seventy-seven percent of their patients were able to return to sports, but only 19% at the same level. Improved ROM was noted in patients treated with an accelerated rehabilitation protocol. Hirschmann and colleagues retrospectively reviewed their experience surgically treating knee dislocations in 26 elite athletes, of which 13 injured their knee playing sports. Medial and lateral ligament injuries were repaired in all patients, and the ACL and PCL were reconstructed with autograft BPTB and quadriceps tendon, respectively. Twenty-four patients were available for follow-up an average of 8 years after surgery. Seventy-nine percent of patients returned to sports, and 42% of these returned to their pre-injury level of play. Patients who underwent surgery greater than 40 days after injury had worse outcome scores and lower rates of return to sports than those who underwent early surgery.

Improved surgical outcomes with early surgery for knee dislocation has been demonstrated in several other studies. A systematic review by Levy et al. found five studies comparing early surgery (less than 3 weeks) with late surgery. Early treatment resulted in higher mean Lysholm scores (90 vs. 82) and a higher percentage of excellent/good IKDC scores (47% vs. 31%), as well as higher sports activity scores (89 vs. 69) on the Knee Outcome Survey [3].

16.2 Conclusion

Knee dislocation is a rare, potentially limb-threatening injury in the athlete. The physician must have a high index of suspicion to avoid missing the diagnosis in spontaneously reduced knees. On-the-field management includes gentle closed reduction of dislocated knees, with splinting and transfer to the hospital. Magnetic resonance imaging and

angiography are recommended for all athletes to evaluate for vascular injury and assist in preoperative planning. Optimal surgical timing is 10–14 days after injury, with repair or reconstruction of all damaged structures. ACL reconstruction with allograft is recommended for all athletes. The PCL may be repaired when avulsed from the femur or bony avulsion from the tibia but is reconstructed for other tear patterns. The LCL/posterolateral corner and MCL/posteromedial corners are repaired when surgery is performed within 3 weeks of injury, the tissue quality is good, and the ligaments have avulsed from both; in all other cases, reconstruction with allograft is recommended. Nine to twelve months of rehabilitation is typically required before returning to sports. Published rates of return to play range between 50 and 80%.

References

1. Peskun CJ, et al. Diagnosis and management of knee dislocations. *Phys Sportsmed*. 2010;38(4):101–11. <http://www.ncbi.nlm.nih.gov/pubmed/21150149>
2. Kerr ZY, et al. Dislocation/separation injuries among US high school athletes in 9 selected sports: 2005–2009. *Clin J Sport Med*. 2011;21(2):101–8.
3. Levy BA, Stannard JP, et al. Decision making in the multiligament-injured knee: an evidence-based systematic review. *Arthroscopy*. 2009b;25(4):430–8. <https://doi.org/10.1016/j.arthro.2009.01.008>.
4. Cole B, Harner C. The multiple ligament injured knee. *Clin Sports Med*. 1999;18(1):241–62.
5. Levy BA, Fanelli GC, et al. Controversies in the treatment of knee dislocations and multiligament reconstruction. *J Am Acad Orthop Surg*. 2009a;17(4):197–206.
6. Hwang MD, Piefer JW, Lubowitz JH. Anterior cruciate ligament tibial footprint anatomy: systematic review of the 21st century literature. *Arthroscopy*. 2012;28(5):728–34. <https://doi.org/10.1016/j.arthro.2011.11.025>.
7. Bernard M, et al. Femoral insertion of the ACL. Radiographic quadrant method. *Am J Knee Surg*. 1997;10:14–22.
8. Anderson CJ, et al. Arthroscopically pertinent anatomy of the anterolateral and posteromedial bundles of the posterior cruciate ligament. *J Bone Joint Surg Am*. 2012;94(21):1936–45. <http://www.ncbi.nlm.nih.gov/pubmed/23138236%5Cn> <http://ovidsp.tx.ovid.com/ovftpdfs/FPDDNCGCHF1AGI00/fs046/ovft/live/gv023/00004623/00004623-201211070-00003.pdf>
9. LaPrade RF, et al. The anatomy of the medial part of the knee. *J Bone Joint Surg Am*. 2007;89(9):2000–10. <http://jbsj.org/article.aspx?doi=10.2106/JBJS.F.01176%5Cnpapers2://publication/doi/10.2106/JBJS.F.01176>

10. LaPrade RF, et al. The posterolateral attachments of the knee: a qualitative and quantitative morphologic analysis of the fibular collateral ligament, popliteus tendon, popliteofibular ligament, and lateral gastrocnemius tendon. *Am J Sports Med.* 2003;31(6):854–60. <http://www.ncbi.nlm.nih.gov/pubmed/14623649>
11. Musahl V, et al. The influence of meniscal and anterolateral capsular injury on knee laxity in patients with anterior cruciate ligament injuries. *Am J Sports Med.* 2016;20(10):1–6.
12. Matava MJ, Sethi NS, Totty WG. Proximity of the posterior cruciate ligament insertion to the popliteal artery as a function of the knee flexion angle: implications for posterior cruciate ligament reconstruction. *Arthroscopy.* 2000;16(8):796–804. <http://www.ncbi.nlm.nih.gov/pubmed/11078535>
13. Fanelli GC, Orcutt DR, Edson CJ. Current concepts. The multiple-ligament injured knee: evaluation, treatment, and results. *Arthroscopy.* 2005;21(4):471–86.
14. Kadiyala RK, et al. The blood supply of the common peroneal nerve in the popliteal fossa. *J Bone Joint Surg [Br].* 2005;87_B(3):337–42.
15. Kennedy JC. Complete dislocation of the knee joint. *J Bone Joint Surg Am.* 1963;45–A(5):889–904.
16. Shelbourne K, et al. Low-velocity knee dislocation. *Orthop Rev.* 1991;20(11):995–1004.
17. McCoy G, et al. Vascular injury associated with low-velocity dislocations of the knee. *J Bone Joint Surg Am.* 1987;69(2):285–7.
18. Schenck R. Classification of knee dislocations. *Oper Tech Sports Med.* 2003;11(3):193–8.
19. Green NE, Allen BL. Vascular injuries associated with dislocation of the knee. *J Bone Joint Surg Am.* 1977;59–A(2):236–9.
20. Lachman JR, Rehman S, Do PSP. Traumatic Knee dislocations treatment. *Orthop Clin North Am.* 2017;2015 <https://doi.org/10.1016/j.ocl.2015.06.004>.
21. Wand JS. A physical sign denoting irreducibility of a dislocated knee. *J Bone Joint Surg.* 1989;71(5):862. <http://www.ncbi.nlm.nih.gov/pubmed/2584265>
22. Natsuhara KM, et al. What is the frequency of vascular injury after knee dislocation ? *Clin Orthop Relat Res.* 2014;472:2615–20.
23. Sisto DJ, Warren RF. Complete knee dislocation. A follow-up study of operative treatment. *Clin Orthop Relat Res.* 1985;(198):94–101. <http://www.ncbi.nlm.nih.gov/pubmed/4028570>
24. Stannard JP, et al. Vascular injuries in knee dislocations: the role of physical examination in determining the need for arteriography. *J Bone Joint Surg Am.* 2004;86–A(5):910–5.
25. Treiman GS, et al. Examination of the patient with a knee dislocation. The case for selective arteriography. *Arch Surg.* 1992;127(9):1056–62. 3. <http://www.ncbi.nlm.nih.gov/pubmed/1514907>
26. Hollis JD, Daley BJ. 10-year review of knee dislocations : is arteriography. *J Trauma.* 2005;59(3):672–6.
27. Klineberg EO, et al. The role of arteriography in assessing popliteal artery injury in knee dislocations. *J Trauma.* 2004;56(4):786–90.
28. McCutchan JDS, Gillham NR. Injury to the popliteal artery associated with dislocation of the knee: palpable distal pulses do not negate the requirement for arteriography. *Injury.* 1989;20(5):307–10.
29. Liow RYL, et al. Ligament repair and reconstruction in traumatic dislocation of the knee. *J Bone Joint Surg [Br].* 2001;85–B(6):845–51.
30. Meyers M, Harvey JP. Traumatic dislocation of the knee joint. A study of eighteen cases. *J Bone Joint Surg Am.* 1971;53–A(1):16–29.
31. Shields L, Mital M, Cave E. Complete dislocation of the knee: experience at the massachusetts general hospital. *J Trauma.* 1969;9(3):192–215.
32. Twaddle BC, Bidwell TA, Chapman JR. Knee dislocations : where are the lesions ? A prospective evaluation of surgical findings in 63 cases. *J Orthop Trauma.* 2003;17(3):198–202.
33. Frassica FJ, et al. Dislocation of the knee. *Clin Orthop Relat Res.* 1991;(263):200–5.
34. Niall DM, Nutton RW, Keating JF. Palsy of the common peroneal nerve after traumatic dislocation of the knee. *J Bone Joint Surg [Br].* 2005;87–B(5):664–7.
35. Peskun CJ, Whelan DB. Outcomes of operative and nonoperative treatment of multiligament knee injuries: an evidence-based review. *Sports Med Arthrosc Rev.* 2011;19(2):167–73.
36. Engebretsen L, et al. Outcome after knee dislocations: a 2–9 years follow-up of 85 consecutive patients. *Knee Surg Sports Traumatol Arthrosc.* 2009;17:1013–26.
37. Krych AJ, et al. Is peroneal nerve injury associated with worse function after knee dislocation ? *Clin Orthop Relat Res.* 2014;472(9):2630–6.
38. Beltran MJ, et al. The safe zone for external fixator pins in the femur. *J Orthop Trauma.* 2012;26(11):643–7. <http://content.wkhealth.com/linkback/openurl?sid=WKPTLP:landingpage&an=00005131-201211000-00007%5Cnpapers2://publication/doi/10.1097/BOT.0b013e31824aed95>
39. Richter M, et al. Comparison of surgical repair or reconstruction of the cruciate ligaments versus non-surgical treatment in patients with traumatic knee dislocations. *Am J Sports Med.* 2002;30(5):718–27.
40. Ríos A, et al. Results after treatment of traumatic knee dislocations: a report of 26 cases. *J Trauma.* 2003;55(3):489–94.
41. Wong CH, et al. Knee dislocations - a retrospective study comparing operative versus closed immobilization treatment outcomes. *Knee Surg Sports Traumatol Arthrosc.* 2004;12(6):540–4.
42. Levy BA, et al. Repair versus reconstruction of the fibular collateral ligament and posterolateral corner in the multiligament-injured knee. *Am J Sports Med.* 2010;38(4):804–9.
43. Stannard JP, et al. 2012. Posteromedial corner injury in knee dislocations posteromedial corner injury in knee dislocations. (November).

44. Stannard JP, et al. 2000. The posterolateral corner of the knee repair versus reconstruction. 3–8.
45. DiFelice GS, Lissy M, Haynes P. When to arthroscopically repair the torn posterior cruciate ligament. *Clin Orthop Relat Res.* 2012;470(3):861–8.
46. Wheatley WB, et al. Arthroscopic posterior cruciate ligament repair. *Arthroscopy.* 2002;18(7):695–702.
47. Brown JA, et al. Avoiding allograft length mismatch during anterior cruciate ligament reconstruction: patient height as an indicator of appropriate graft length. *Am J Sports Med.* 2007;35(6):986–9. <http://www.ncbi.nlm.nih.gov/pubmed/17337725>
48. Rios CG, et al. Posterolateral corner reconstruction of the knee evaluation of a technique with clinical outcomes and stress radiography. *Am J Sports Med.* 2010;38(8):1564–74.
49. Laprade RF, Johansen S, Engebretsen L. Outcomes of an anatomic posterolateral knee reconstruction surgical technique. *J Bone Joint Surg AmJ.* 2011;93(92):16–22.
50. Marx RG, Hetsroni I. Surgical technique: medial collateral ligament reconstruction using achilles allograft for combined knee ligament injury. *Clin Orthop Relat Res.* 2012;470(3):798–805.