The posterolateral corner of the knee

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Abstract

The posterolateral corner (PLC) of the knee refers to a complex of structures that play a key role in knee stability. The main three structures in the PLC of the knee are the lateral collateral ligament, the popliteus and the popliteofibular ligament. These are primarily restraints to varus and external rotation forces. As such, mechanisms of injury include varus and hyperextension, and are typically high energy. These injuries are rarely isolated and are commonly associated with other injuries such as cruciate ligament injury or tibiofemoral knee dislocation. If undetected or untreated, PLC injuries can cause severe long-term disability due to instability and cartilage degeneration. Failure to recognize these injuries can also jeopardize the results of concomitant anterior or posterior cruciate ligament reconstruction. Immediate management in acute injuries involves assessment and treatment of any associated neurovascular injury, especially to the popliteal artery or peroneal nerve, and reduction of knee dislocation. Acute repairs of the damaged structures can be undertaken, but ligament reconstruction is often required, using either autograft or allograft. Graft reconstruction procedures can be fibula-based or anatomic, each having their own merits. The timing of surgery can be acute, chronic or staged, depending on the exact nature of the injury and on timing.

Keywords knee; ligament; posterolateral corner; soft tissue

Anatomy and biomechanics

The term 'posterolateral corner' (PLC) describes a complex of ligaments, muscles, tendons and joint capsule in the anatomic posterior and lateral aspects of the knee. The three main stabilizers, and therefore 'key structures', are the lateral collateral ligament (LCL), the popliteus tendon, and the popliteofibular ligament (PFL) (Figure 1).

The mean maximal force to failure of the LCL has been found to be up to 750Nm, and the PFL 425Nm.[1] Further to this are a variety of other structures that tend to have a degree of anatomical variation and that vary in reports.[2] These include the posterolateral capsule, the arcuate ligament, the fabellofibular ligament, the biceps femoris tendon and the iliotibial band (ITB).[3] The popliteus, biceps femoris and ITB are dynamic stabilizers, whereas the other structures are static stabilizers. The popliteus does, however, have both dynamic and static functions.[4]

The PLC is the primary restraint to varus and external rotation of the tibia. Specifically, the lateral collateral ligament is the primary restraint to varus force. This is seen at greatest effect at 30° of flexion; however, it is a restraint at all degrees of movement.[5] It also serves to resist external rotation, with its effect being greatest at 0–30° of flexion.

The LCL originates from just proximal and posterior to the lateral femoral epicondyle, and attaches to the anterior aspect of fibular head.[6]

The popliteus is a dynamic restraint to external rotation, particularly when the knee is extended between 20 and 130°. The popliteus also prevents posterior translation and varus between 0 and 90°.[4] The functions of the popliteus also include knee extension (weak),[2] external rotation of femur when the foot is fixed, and internal rotation of the tibia on the femur when the foot is free. In the last 15–20° of extension, the tibia externally rotates relative to the femur, 'locking' the knee in extension, which contributes to stance phase stability. This is otherwise referred to as 'the screw-home mechanism'.[7] Popliteus also resists shearing forces if the posterior cruciate ligament (PCL) is damaged.[8] Popliteus originates from the lateral epicondyle anterior to LCL, and inserts on the posterior surface of the tibia, proximal to the soleal line.[6]

The PFL ligament originates from the superior musculotendinous junction of the popliteus muscle and inserts distal and anterior to the styloid process on the fibular head.[6] It serves as a restraint to external rotation. The PFL and popliteus maximally resist external rotation at 60°.

The fabello-fibular ligament is found between the fabella (a sesamoid bone found in the lateral head of gastrocnemius) and the fibular head.[9]

The arcuate ligament is a triangular structure that runs from the posterior aspect of the fibula and that arches up medially to the popliteus tendon and attaches over the posterior capsule.[2]

The ITB arises from the gluteus maximus and tensor fascia lata, and inserts into Gerdy's tubercle.

Biceps femoris arises from two heads; the posterior aspect of the tuberosity of the ischium, and the lateral lip of the linea aspera. It inserts to the styloid process on the fibular head.

The anatomy and attachment points of these 'ancillary' posterolateral structures can be variable, and this is reflected in the many different forms of PLC reconstruction that have been described.

The anatomy has also been described in layers,[9] which is relevant surgically and in understanding the above named

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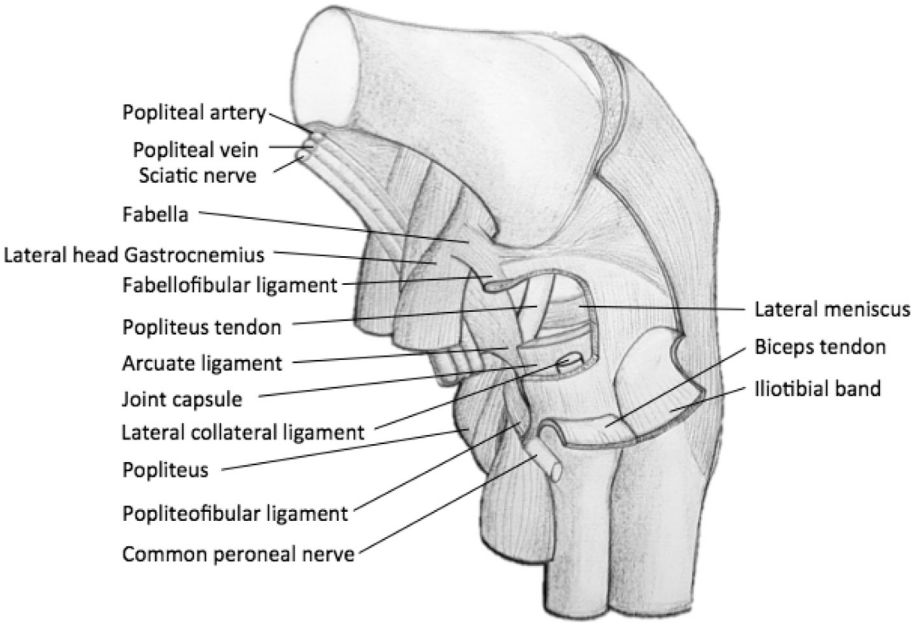

Figure 1 An illustration of the anatomy of the posterolateral corner of the knee. Reproduced from reference 1 with permission from Elsevier.

structures in more detail. This includes the superficial, middle and deep layers. There are several variations on the details of this anatomy.[9] The biceps femoris and ITB are considered to be in the superficial layer. The middle layer mainly consists of the patellar retinaculum. The deep layer has the LCL, fabellofibular ligament, arcuate ligament, coronary ligament, popliteus tendon and popliteofibular ligament.

The PLC is an important secondary stabilizer during posterior translation at all degrees of flexion. This is more pronounced in early knee flexion, at 0–60°. This is particularly important in the PCL-deficient knee.[10] The PLC is also the primary restraint to posterior translation in full extension. Thus, the reverse Lachman's test, which is performed at 30° of flexion, is often thought to be the most sensitive test for PLC damage.[11] At 90° the PCL provides the main stability in posterior translation.[12] If both the PCL and PLC are divided, the maximum translation is seen at 30° of flexion.[5]

At 0–90° the LCL provides the primary restraint to varus. The PLC is the primary restraint to external rotation of the tibia at all angles, the effect of which is more pronounced in extension.[10] Thus, the dial test of external rotation can be performed at 30° of flexion to test for PLC injury. A combined injury of the PCL and PLC leads to more pronounced external rotation of the tibia, seen greatest at 90°.[10] The dial test then becomes positive at both 30° and 90° of flexion in the combined PCL/PLC deficient knee.

Clinical assessment

Mechanism of injury

Posterolateral corner injuries can be caused by high-energy trauma, such as a motor vehicle collision, or relatively low-energy trauma, such as a sporting accident. The mechanism is typically described as a hyperextension injury and varus injury to the knee,[13] and has been demonstrated in anatomic studies. It can be associated with knee dislocation. The injury is rarely isolated, with isolated PLC injuries accounting for only 1.6% of cases.[14] Isolated injury can occur when the knee is in extension or flexion, and may be underdiagnosed due to absent radiological MRI signs. In the former, a posterolateral force is applied to the proximal tibia causing a varus-hyperextension motion. In the latter, an isolated injury can occur if the knee is in external rotation and a posterior force is applied, such as a 'dashboard injury'.

History

The history is essential is managing PLC injuries. Managing a multi-injured patient will be markedly different from treating a chronic isolated PLC injury.

In high-energy trauma concomitant injury may include damage to the vasculature, nerves, other ligaments, menisci or articular cartilage. There may be a frank dislocation. Acutely, the patient may have swelling, severe generalized pain and a limited range of motion. If neurovascular injury is present, the patient may have paraesthesia, weakness or a cold pulseless foot. Arterial injury is associated in 32% of knee dislocations.[15] The incidence of PLC injury in knee dislocation is reported at 41%.[16] Common peroneal nerve injury is found in 26.2% of cases.[17] With associated meniscal, ligamentous or cartilage injury, the patient may complain of giving way, locking or the feeling of joint instability. Associated meniscal injury is found in 37.3% and cartilage injury in 28.3% of multiligament knee injuries.[16]

An isolated injury is more likely to result from lower energy trauma, such as hyperextension/varus during sport. The patient may complain of pain in the posterolateral aspect of the knee or instability in full extension.[14] Examination may reveal only subtle signs, such as posterior Lachman's or a positive dial test at 30°, but radiology in the long-standing undetected isolated PLC injury may be normal.

Clinical examination and signs

Initial management must be guided by the Advanced Trauma Life Support protocol. Particular attention must be paid to neurovascular assessment. Specifically, lower limb pulses must be palpated together with ankle-brachial pulse index assessment. With regards to the knee, the soft tissues must be assessed carefully for potential open fractures. Further to this, there may be gross oedema or ecchymosis associated with either isolated or multiligament injury. Range of motion acutely will be reduced.

Examination of a chronic posterolateral corner injury may reveal significant recurvatum or increased hyperextension on the affected side. There may be tenderness to palpation over the posterolateral aspect of the knee in an isolated injury. Examination of other ligaments in the knee must be performed to exclude a multiligament injury. These include the anterior draw test, valgus medial collateral ligament stress test, Lachman's test, and the pivot shift test.

Whilst standing, the patient may present with varus limb alignment. Gait should be examined for evidence of varus thrust, which occurs in the stance phase. In order to avoid the pain and instability experienced in extension, some patients may walk with a slightly flexed knee.[18]

As stated, the PLC is a secondary restraint to posterior drawer of the tibia between 0° and 60° of flexion. Thus, a PLC injury may results in a 'posterior Lachman's', with increased posterior translation at 30° of flexion and a softer end point to this test than the unaffected side.[11]

The dial test is valuable in identifying PLC injuries. It is performed with the patient supine or prone. If there is more than 10° of *increased external* rotation compared to the unaffected side at 30°, this implies an injury to the PLC only. If the PCL and PLC are injured, the external rotation at 30° and 90° will be increased by more than 10°. Comparison with the contralateral limb is essential. The examiner should be aware of the false-positive dial test that results not from a PLC injury, but from an injury to the *posteromedial* corner. The subtle difference in examination here will be anterior subluxation of the medial tibia in the posteromedial corner injured knee compared to posterior subluxation of the lateral tibia in the PLC injured knee.

The external rotation recurvatum test is also valuable in identifying PLC injuries. It is performed by extending the knee by holding the second toe. With PLC injury, the affected knee will go into external rotation and varus, compared to the other side.

The posterolateral drawer test is performed with the patient supine. The knee is flexed to 90° and the hip flexed to 45°. With the tibia in 15° of external rotation, a posterolateral force is applied by the examiner. If the tibia exhibits more external rotation than the femur, this indicates a PLC injury.

The same is true for the posterior drawer test. If there is posterior translation at 30° flexion, but none at 90°, this could indicate an isolated PLC injury.[11]

Posterolateral external rotation, or the 'spin' test, has also been described for detection of posterolateral corner injuries. The knee is flexed to 30° and 90°, with application of external rotation to the tibia in both positions. Normally, the tibia is anterior to the femur. However, in the presence of PLC injury it the subluxes posteriorly. In PCL injury, the subluxation will occur at both degrees of flexion, but in an isolated PLC injury it will occur at 30° flexion only.[11]

The reverse pivot shift test is performed by reducing the subluxation mentioned in the aforementioned tests. The knee is flexed beyond 40°, and the tibia placed in external rotation. The tibia should be posterior to the femur at this stage if there is a PLC or PCL injury. Thereafter, the knee is extended and a clunk may be felt on reduction of the tibia.

The varus test can be performed by exerting a varus stress across the knee whilst at 30° of flexion. The degree of severity can be graded using Hughston[19] classification. Grade I is less than 5 mm laxity, Grade II is 5–10 mm laxity and Grade III is more than 10 mm laxity.

In general, a knee with excessive hyperextension, subtle varus laxity, a posterior Lachman's and positive dial test should raise strong suspicion for a possible PLC injury.

Imaging

The detection of a posterolateral corner problem can be difficult in an acute or chronic knee injury. Plain radiography should be performed initially, specifically with anteroposterior (AP) and lateral views. In chronic injuries, standing long-leg alignment films can be performed. The purpose of this is to assess for any fractures or opening of the lateral compartment in chronic cases. Stress radiographs should also be considered. One method describes[20] the patient being placed supine, the limb raised 35 cm from the table and externally rotated 15°. This is performed on both limbs. A line is then drawn on the lateral down the central axis of the femur. A second line is drawn perpendicular to this at the level of the femoral condyles. A third line is drawn parallel to the tibial plateau. A discrepancy of 5° implies an injury. Another technique[21] described requires a varus stress to be placed on the knee while flexed at 20°. Varus gapping of more than 4 mm can imply an injury.

If there is doubt as to varus laxity or PLC injury, stress views under image intensifier can be performed under anaesthetic when performing surgery to reconstruct other structures (e.g. the ACL). If these suggest excess lateral opening in the coronal plane (>4 mm side-to-side difference) then concomitant PLC reconstruction can be undertaken.

On plain radiograph there may be an arcuate or Segond sign, potentially indicating a PLC injury.[3] There may also be a bony avulsion at Gerdys tubercle.

Magnetic resonance imaging (MRI) is an essential adjunct in identifying PLC injuries, and has been shown to be both sensitive and specific.[22] MRI has been found to be 93% sensitive if performed within 12 weeks of injury.[23] About 9% of patients presenting with an acute haemarthrosis have been found to have a PLC injury in a radiological study.[24] More than 50% of these injuries involved more than one of the major three PLC structures. 87% will have multiligament injuries identifiable on MRI. On reviewing T2-weighted images, bony oedema at the medial aspect of the medial femoral condyle and the anterior aspect of medial tibial plateau may be present. This is a secondary sign of PLC injury. ITB swelling, anterolateral lateral meniscal disruption and biceps femoris fibular head avulsion fracture may all be signs of PLC injury. The popliteofibular ligament, lateral collateral ligament and lateral meniscus posterior horn may also show signs of injury or avulsion, indicated by increased signal. If present, femoral subluxation on the tibia is a strong indicator of associated cruciate ligament injury.

Where knee dislocation has occurred and concern over vascular status has been raised, computer tomography (CT) angiography can be performed.[25] Another option, if available, is magnetic resonance angiography, which allows for good assessment of both ligamentous and vascular structures.

Arthroscopic assessment with examination under anaesthesia is extremely useful in the diagnosis of PLC injuries.[26] It has also been found to be useful in diagnosing individual structural injury when combined with open reconstruction, and in fact can identify injury that would otherwise have been missed. The camera 'drive though sign' occurs when an excessive amount of laxity is encountered arthroscopically, which can be an indication of PLC injury. The commonest injuries found at arthroscopy are to the popliteus tendon, popliteomeniscal fascicles, lateral coronary ligament and the lateral capsular ligament.[27]

Management techniques

Treatment of PLC injury depends on the grade of injury, chronicity and degree of associated injuries.

Non-surgical management

Non-surgical management of PLC injuries is not well documented in the literature; however, it does seem to be effective in isolated Grade I and II injuries.[28–30] In higher-level injuries, non-surgical management may be appropriate in the context of the medical history of the patient, for example in a patient with multiple co-morbidities or the multiply injured patient. The patient can be placed in an extension brace initially for 2 weeks, with gradually increasing flexion angles over 6–8 weeks. If there is pain on weight-bearing, then non-weight bearing status can be implemented for 4–6 weeks.

In general, Grade III injuries of the PLC do not respond well to conservative management, with patients suffering poor outcomes.[28] Also, surgically treating patients with multiligament injuries improves outcomes.[31]

Surgical management

There is a wide array of data available in the literature regarding multiple repair and reconstruction techniques, with no consensus on the 'ideal reconstruction'. This may reflect the many structures involved and also the anatomical variability of these structures. Techniques therefore depend on the presence of a multiligament injury, chronicity and the surgeon's experience.

Surgical management can be divided into acute (less than 3 weeks since injury), subacute (3 weeks–3 months since injury) and chronic (>3 months). Acute injuries will require ATLS management. If knee dislocation is present it will require urgent reduction. Vascular injuries require intervention within 8 hours for optimal outcomes.[15] With this in mind, the most appropriate initial orthopaedic management may be external fixation, in order to protect a vascular graft. External fixation may also be utilized in the polytrauma patient, for open fractures or if the tibiofemoral joint is unstable despite simple splinting techniques. Where a spanning fixator is applied, this can be removed at 2–6 weeks[31] and then delayed reconstructive surgical management considered. Articulated external fixation has also been described as an adjunct in postoperative multiligament reconstruction, with similar outcomes to hinged knee bracing.[32]

PLC repair/reconstruction

Managing injuries acutely within 3 weeks can improve outcomes.[33,34] PLC repair can only be undertaken in acute injuries where there has been avulsion of structures such as the popliteus tendon or the LCL/biceps tendon. These can be reattached using suture anchor techniques.

The results of repair of mid-substance tears or chronic injuries are poor[35–37] probably because there has been a progressive stretching of the entire ligament during injury that does not recover.

Reconstruction using auto- or allograft should be considered in these situations.

Repair techniques

Repair refers to the suturing of injured tissue, which may involve anchorage back to the bone. It can only be performed if the tissue quality is adequate. Sutures are placed in the tendon, usually with a horizontal mattress configuration, and anchored either to the tibia or the femur. Bone anchors can be used in avulsion injury.

Hughson[38] described a technique that can be employed if the popliteus tendon is distended but not torn. This involves re-inserting the tendon antero-superior to the original site with a bone block, using a screw or staple. This technique, however, has a failure rate of up to 36%.

Reconstruction using grafts

Hamstring (gracilis, semitendinous, biceps femoris), quadriceps, patellar tendon, tendoachilles and ITB are all options for allograft or autograft.

Most PLC injuries are combined with ACL or PCL injury. Repair of acute Grade III PLC injuries in a multiligament injury with a staged cruciate reconstruction has been shown to have a failure rate of 38%, and therefore a graft reconstruction is recommended. This is associated with a failure rate of only 9%.[33] It is thought that due to the lack of cruciate support, more translatory force is applied across the repair, thus leading to higher failure rates. Many authors will recommend reconstruction in chronic injuries outright.[31]

Acute reconstruction may be associated with a higher risk of arthrofibrosis. Moatshe reported that 15.2% of patients with knee dislocation operated on acutely developed arthrofibrosis, compared to just 3.8% managed at a later stage.[16]

The failure rate for surgical reconstruction in chronic injuries is 10%.[39] The goals of surgery are to restore kinematics, reduce instability and to ultimately lower the risk of long-term osteoarthritis. Limb alignment is crucial in the management of chronic cases. This is because patients are at high risk of developing genu varum. In genu varum, high tibial osteotomy (HTO) is an option for surgery and is a powerful tool in changing sagittal and coronal alignment of the tibia. LaPrade et al. reported that 38% of patients will require no further ligamentous reconstruction[40] if osteotomy is performed. This has been shown biomechanically to decrease varus and external rotational laxity for PLC injured knees. There are fairly limited data to support this approach, however.[41] Where osteotomy is performed, it is recommended that there is evidence of full healing before proceeding to PLC reconstruction.[42]

Reconstruction in the presence of varus has been shown to increase the risk of failure over time[43] and, therefore, some authors recommend HTO prior to this. If reconstruction is performed after HTO, it is recommended 6–8 months after the initial procedure,[43] when the osteotomy has fully healed.

Multiligament versus isolated PLC injury

Surgical treatment for multiligament injury combined with PLC injury has favourable results compared to non-operative management.[34] Combined PCL injuries are the most commonly associated injury.[44] In 60% of knees with an injured PCL, there will also be a PLC injury. The ACL is involved in 10% of cases and the PCL in 27% of cases of PLC injury.[45] HTO can also be considered in patients with these injuries.[34] Reducing the posterior slope by 5° has been shown to improve sagittal instability after combined injuries.[46] Reconstruction is favoured over repair in the multiligament injured knee.[47] Laprade found increased loads on the ACL graft in multiligament injuries with coupled varus and internal rotation, and therefore recommended PLC reconstruction in this scenario.[3] Sekiya found that if both a cruciate and the PLC were reconstructed, then the kinematics of the knee were restored.[48] Some authors recommend reconstructing all ligaments in one sitting, and not staging, due to the risk of altering joint kinematics between procedures.[31]

Tunnel convergence must be avoided in multiligament reconstruction. This is a potential risk due to multiple tunnels being made for the varying ligaments. Convergence can lead to failure due to risk of fracture, damage to grafts, and insufficient bone stock for fixation techniques. The risk is higher if performing more ligament reconstructions and if the reconstruction involves multiple tunnels, such as some of the anatomic PLC techniques. Thus, the senior author prefers a one-tunnel reconstruction technique (modified Larsen, see below) in the case of multiligament reconstruction.

Graft tensioning and tensioning sequence is very important. It can result in over-constraint of the knee, leading to biomechanical and kinematic abnormalities. Conversely, under-constraint will lead to laxity and instability. Authors report varying methods of graft tensioning. Some recommend doing the PLC first, followed by the ACL, then the PLC, and finally the posteromedial corner.[44] It has been shown, however, that fixing the ACL graft first leads to increased external rotation of the tibia.[49] Some therefore fix the PCL first, then the PLC, and then finally the ACL last.[31]

No-one has studied the impact of multiple grafts, tunnels or varying anatomic fixation techniques and the clinical or kinematic effects on the knee.

Reconstruction techniques

Both non-anatomical and anatomical reconstruction methods have been described. Anatomical reconstruction refers to reconstruction of the three main PLC structures. Many authors are now using anatomic-based techniques.[36] There is also a wide variety of graft options, including auto- and allograft from varying sites. There is no consensus on this in the literature.

Non-anatomic: non-anatomic reconstruction is for the purpose of obtaining posterolateral stability by applying tension on non-injured structures. There are many types of non-anatomic reconstruction, including augmentation, femoral biceps tenodesis, arcuate ligament reconstruction and the extracapsular ITB sling.

The biceps femoris tenodesis method was described by Clancy[50] in the 1980s (first described with PCL reconstruction).[50] It involves harvesting part of the biceps femoris tendon from the fibula (Figure 2). This is passed behind the remaining tendon and attached to the lateral femoral condyle using an anchor or screw[51] with the knee at 30°. This technique, however, removes part of one of the dynamic PLC stabilizers, and is a form of LCL reconstruction. Although this technique did achieve fairly good results, it is reported that only 54% of patients could return to pre-morbid activity levels.

The arcuate ligament reconstruction was first described by Hughston in 1985.[38] This is a form of ligament tightening. This involves advancing the arcuate ligament complex and its bony attachment anteriorly and distally on the femur. If the lesion is distal, then the arcuate ligament attachment to the tibia and fibula will be loose and this must, therefore, be stabilized. Good long-term outcomes were reported in 85% of patients. The issue with tightening procedures is that isometric points of ligaments may change. In extension the laxity is controlled; however, in flexion it is not, and therefore subluxation can occur.[51]

The 'posterolateral sling' procedure was first decribed in the 1970s, and involves a graft being passed through a tibial tunnel and implanted on the distal lateral aspect of the femur. This fixation point prevents further pathological subluxation of the tibial plateau in an isometric fashion.[52] Albright popularized the use of extacapsular ITB fixation for this purpose. This procedure has been shown to have effective clinical and biomechanical results in the long-term.[53] On kinematic analysis, however, normality may not be fully restored.[54] Reverse pivot shift, hyperextension and varus laxity is reduced in the vast majority of patients; however, kinematic function may not be fully restored, as this procedure does not involve LCL or PFL reconstruction.

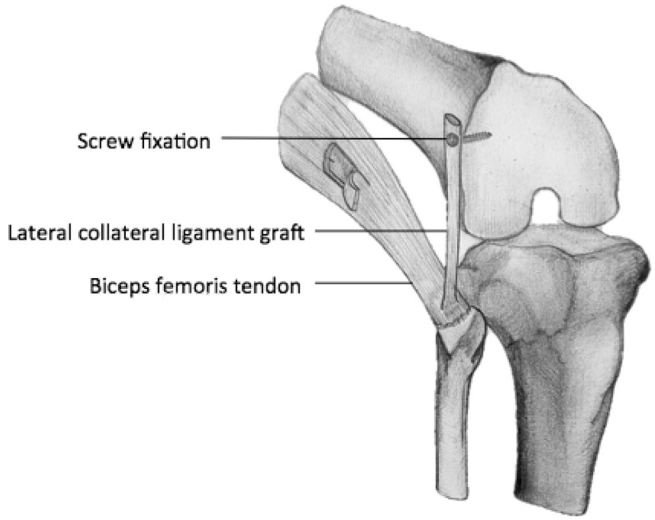

Figure 2 An illustration of a biceps femoris reconstruction of the lateral collateral ligament (the Clancy method). Illustration by Harry Hodgson.

A popular method reported by Larson[55] uses a fibular sling based technique. This method is used by the senior author. This is a form of popliteofibular reconstruction (Figure 3). A strip of allograft is passed from anterior to posterior through the fibular head. It is then passed into a tunnel in the area of the lateral epicondyle, at the isometric point of the LCL and PFL, and fixed in the femur with the knee in 30° flexion. This procedure prevents posterolateral laxity by essentially reconstructing the LCL and the PFL. This is a popular technique as it has been proven to have excellent clinical results.[56]

There are many reported variations of the Larson technique,[57–60] including two-bundle, three-bundle and four-bundle techniques. A biomechanical study has shown the four-strand technique to be stronger than the two-strand.[58] Clinically, the three-bundle technique has not been shown to be superior to two bundles.[57] There are modifications to this technique based on single/dual femoral attachments and fibular/femoral tunnel variations described.[61,62] The two femoral tunnel technique has shown greater stability compared to single tunnel on cadaveric analysis.

Anatomic: where anatomic reconstruction is performed, reconstruction of the LCL, PFL and popliteus tendon is recommended, with the use of a fibula based or tibiofibula based technique. These have been proven biomechanically to be superior to non-anatomic methods.[63] Anatomic reconstruction of the three main PLC structures has been shown to reduce varus laxity and external rotation compared to the non-anatomic PLC sling procedure.[64] Many surgeons now recommend the use of anatomic methods of reconstruction and report good results.[29,63,64]

LaPrade popularized the tibiofibular based technique for anatomical reconstruction of the three main elements of the PLC[65] (Figure 4). Graft placement in anatomical reconstruction is essential. This technique involves four bony tunnels. Two tunnels are made in the lateral femoral condyle at the isometric point of the insertion of the popliteal tendon and the LCL. An anteroposterior tibial tunnel with posterolateral exit, and a fibula head tunnel with posterior exit, are also made. Two grafts of 22 cm are then prepared and passed. Fixation on the tibia is achieved through staples and cannulated screws. Other techniques of anatomical reconstruction have been described[64,66,67] with good clinical and biomechanical results.

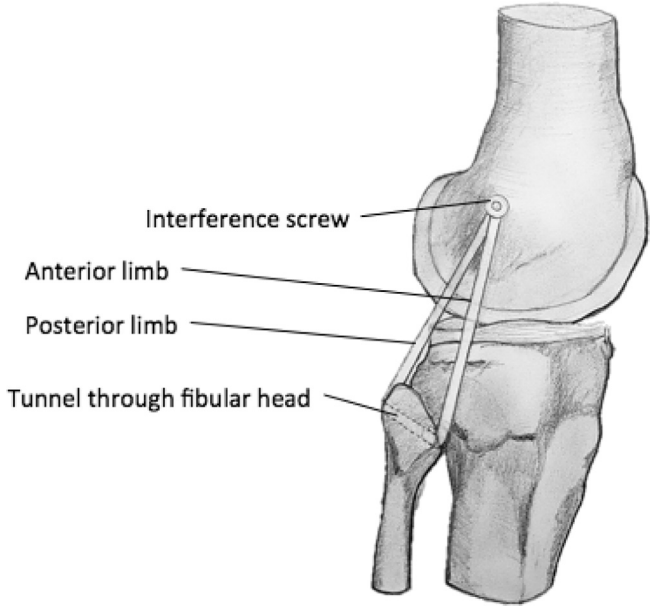

Figure 3 An illustration of one form of the Larson technique. Illustration by Harry Hodgson.

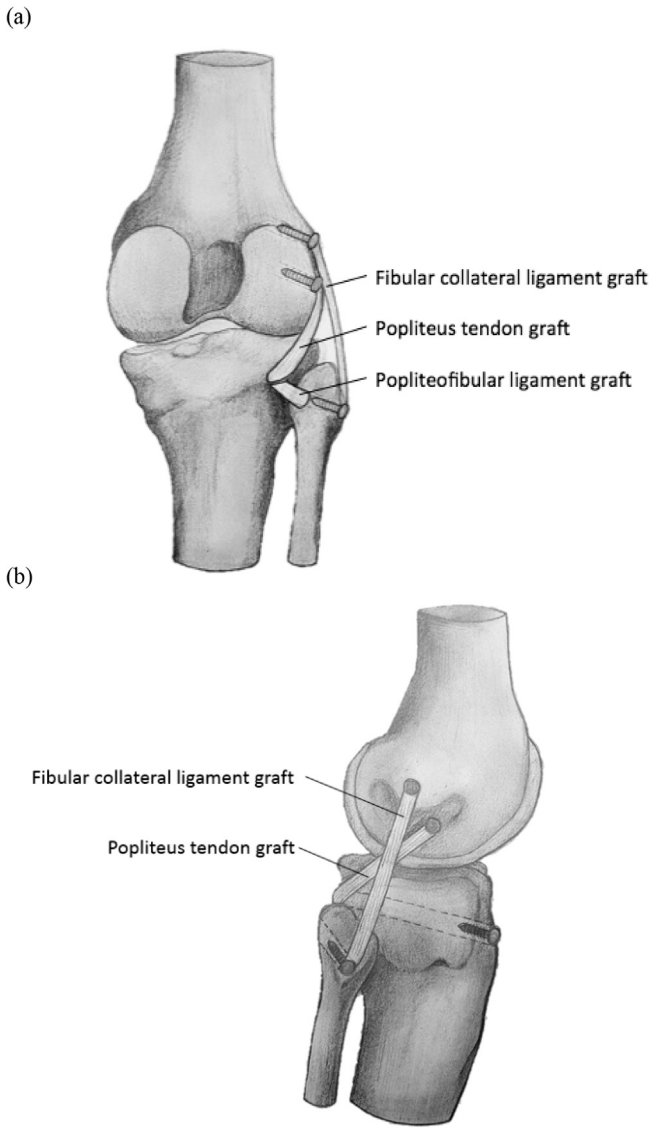

Figure 4 An illustration of the LaPrade technique of anatomical reconstruction. Illustration by Harry Hodgson.

The anatomical techniques may, however, potentially result in internal rotation over-constraint and varus.[57,68] There is also a higher potential risk of fracture with more tunnels.

Rehabilitation

The aim of rehabilitation is to protect the surgically managed ligament whilst allowing the patient to return to activities as quickly as possible. Surgical fixation should allow a rehabilitation program that encourages early mobilization, to prevent stiffness, and that protects against early graft failure. The literature describes varying rehabilitation methods, ranging from

immobilization in full extension for 6 weeks to early range of motion. A popular[36] approach, described by Laprade,[31] is to aim for 0–90° passive range of motion in the first 2 weeks after surgery. During this period, patella mobilization is focused on to prevent extensor mechanism stiffness. Protected weight-bearing is performed in multi-ligament injuries. After 6 weeks, patients can wean themselves off crutches and begin stationary bike exercises.

The senior author prefers a 'graduated flexion' rehabilitation regime, consisting of bracing at 0–30° for weeks 0–2, followed by 0–60° for weeks 2–4, followed by 0–90° at weeks 4–6. Weight-bearing status also progresses from non-weight-bearing for 3 weeks followed by 50% weight-bearing for 3 weeks, followed then by full weight-bearing.

When range of motion is restored, the focus moves to muscular endurance, strength and power. Return to sport is tailored to each patient, and usually occurs at 9–12 months. This decision is made after an assessment of range of motion, muscle strength, functional stability and clinical examination.

Further studies

There is a large volume of heterogeneous data available on the management of PLC injuries. Multiple surgical techniques have been described, with improving clinical outcomes in recent years. Due to the diverse nature of the literature and relatively low levels of evidence, the gold standard for surgical management is still debatable. Management choices should be based on patient selection, chronicity of injury, presence of multiligamentous injury and surgical experience. There are very few Level 1 studies comparing different surgical techniques. Studies comparing the biomechanics of both knees after reconstruction would be helpful. Further biomechanical and clinical studies are also required to investigate the effect of multiple graft tensioning methods. ◆

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