38

Posterior Cruciate Ligament: Current Concepts

Mark D. Miller, MD
Daniel E. Cooper, MD
Gregory C. Fanelli, MD
Christopher D. Harner, MD
Robert F. LaPrade, MD

Introduction

Understanding of posterior cruciate ligament (PCL) injuries of the knee has increased in large part because of a better understanding of the anatomy and biomechanics of the PCL and the posterolateral corner. More careful clinical evaluation of injury to these structures has resulted in earlier diagnosis and treatment, with new surgical techniques increasing the possibility of improved results.

Anatomy

The PCL has two functional components, an anterolateral and a posteromedial bundle (Fig. 1). The anterolateral bundle, which is approximately twice the size of the posteromedial bundle, is taut in flexion, and the posteromedial portion is taut in extension.[1] Two variable meniscofemoral ligaments (Humphry and Wrisberg) originate from the posterior horn of the lateral meniscus and contribute fibers to the PCL. The cross-sectional area of the PCL narrows midsubstance to approximately one third the area of its insertions.[2]

The posterolateral corner is perhaps best considered as consisting of superficial and deep structures. The superficial structures include the iliotibial band or tract (with the tract representing the distal third of the tendon near its insertion) and the biceps tendon. Deep structures include the lateral collateral ligament, capsular structures (midthird lateral capsular ligament, fabellofibular ligament, and posterior arcuate ligament), and the popliteus muscle complex (including the popliteofibular ligament)[3] (Fig. 2).

Biomechanics

The PCL has an ultimate load of approximately 1,600 N and a stiffness of approximately 200 N/mm.[4] The anterolateral portion of the ligament is stronger and stiffer than the other portions;[1] therefore, reconstructing this component has been the focus of PCL reconstruction techniques. Isolated sectioning of the PCL and of the posterolateral corner (as secondary stabilizer) results in posterior translation of approximately 11 mm and less than 3 mm, respectively. Sectioning of both the PCL and the posterolateral corner, however, causes posterior translation of up to 30 mm, demonstrating the synergistic relationship between these structures.[5-7] A similar effect on rotation occurs when the posterolateral corner (as primary stabilizer) and PCL (as secondary stabilizer) are sectioned individually and together.

In situ forces in the PCL increase from 36 N in full extension to 112 N at 90° of flexion.[8] These factors are reduced with axial compression (as in weight bearing) and quadriceps loading, and increased with hamstring loading.[9,10] In an intact knee, the popliteus muscle significantly reduces the in situ forces in the PCL.[11] Sectioning of the PCL causes increased in situ forces in the popliteus and the meniscofemoral ligaments.[8] PCL deficiency is associated with increased contact pressures and late arthritis of the medial femoral condyle and patella.[12-15]

Recent biomechanical studies of newer PCL reconstruction procedures have provided interesting information. The tibial inlay technique, in which the bone block of a graft is laid directly into a trough in the back of the tibia, results in less graft laxity with cyclic loading.[16] A two-bundle technique, which includes a second femoral tunnel using a separate graft or a split graft, results in improved stability in both extension and flexion.[17] Perhaps the best construct, at least from a biomechanical standpoint, would be a two-bundle inlay technique, with a split quadriceps tendon-bone graft. Clinical

Fig. 1 Clinical photograph of the anterolateral and posteromedial bundles of the PCL.

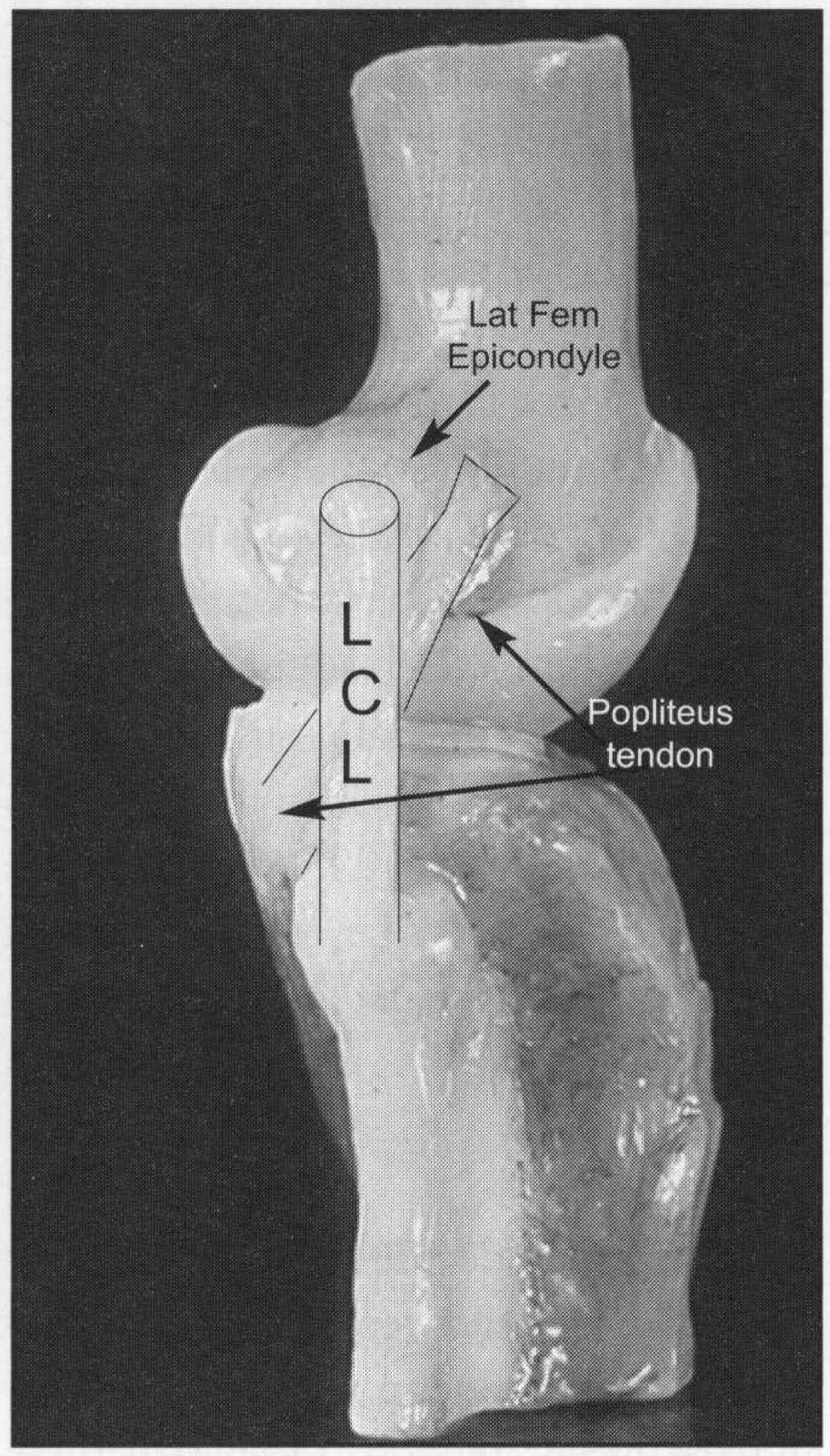


Fig. 2 The popliteus tendon runs obliquely under the lateral collateral ligament and inserts anterior and distal to the lateral femoral epicondyle.

results of these options will ultimately determine the best construct.

Combined PCL and posterolateral corner injuries are more common than initially believed, with up to 60% of significant PCL injuries having a combined posterolateral corner disruption.[18] Failure to treat this associated injury results in undue tension in the PCL graft [19] and may doom the reconstruction to failure.[20,21]

Clinical Evaluation

Physical Examination

The mechanism of PCL injury is most commonly a posterior blow to the proximal tibia with the foot in plantar flexion.[22] The foot position is important only because it changes the force vector. If the foot is dorsiflexed, the ground force contacts the patella and distal femur; with the foot plantar flexed, however, the ground force vector intersects with the proximal tibia. Other mechanisms of injury include hyperflexion and combined forces.

The posterior drawer test is the most sensitive and specific test for the diagnosis of PCL injuries.[23] The test is done with the patient supine and the knee flexed 70° to 90°. Evaluation of the tibial starting point is key. The tibia normally has an anterior step-off of approximately 10 mm in this position. If there is posterior subluxation of the tibia (with a complete PCL injury), then the starting point may be even with or posterior to the medial femoral condyle. Displacement beyond this position with a posteriorly directed force on the tibia suggests a complete injury, and posterior displacement of the tibia more than 5 mm posterior to the femur suggests a combined injury (usually involving the PCL and the posterolateral corner). The quadriceps active test[24] is done with the patient supine and the knee flexed. The patient is instructed to tense the quadriceps, and this action brings the posteriorly subluxated tibia anteriorly. The test can be useful in assessing the relative anterior and posterior instability in a knee with a chronic combined ACL and PCL deficiency.

The dial test for asymmetric external rotation is the most important test for posterolateral instability.[5,6,25] The test can be done with the patient prone or supine (an assistant holds the knees together). Both feet are passively externally rotated and the thigh-foot angle is measured. Excessive external rotation (as compared with the opposite side) of more than 10° to 15° is considered pathologic. The test is done with the knee in both 30° and 90° of flexion. If the thigh-foot angle is increased at only 30° and not 90°, the patient has an isolated PCL injury. If, however, the thigh-foot angle is increased at both 30° and 90°, this indicates a combined injury to the posterolateral corner and PCL (confirmed with a posterior drawer test). Other tests for posterolateral corner injuries (especially combined injuries) include the posterolateral drawer and external rotation recurvatum tests (with the latter being most dramatic in ACL/PCL/posterolateral corner combined injuries). With all tests for posterolateral instability, it is critical to compare the results to the contralateral normal knee.[26]

It is also important to carefully observe a patient's gait. A varus thrust gait, during which the knee shifts into varus during foot strike with associated lateral compartment opening, is common in patients with chronic posterolateral corner deficiencies. Patients may attempt to compensate by walking with a flexed knee gait with the foot in internal rota-

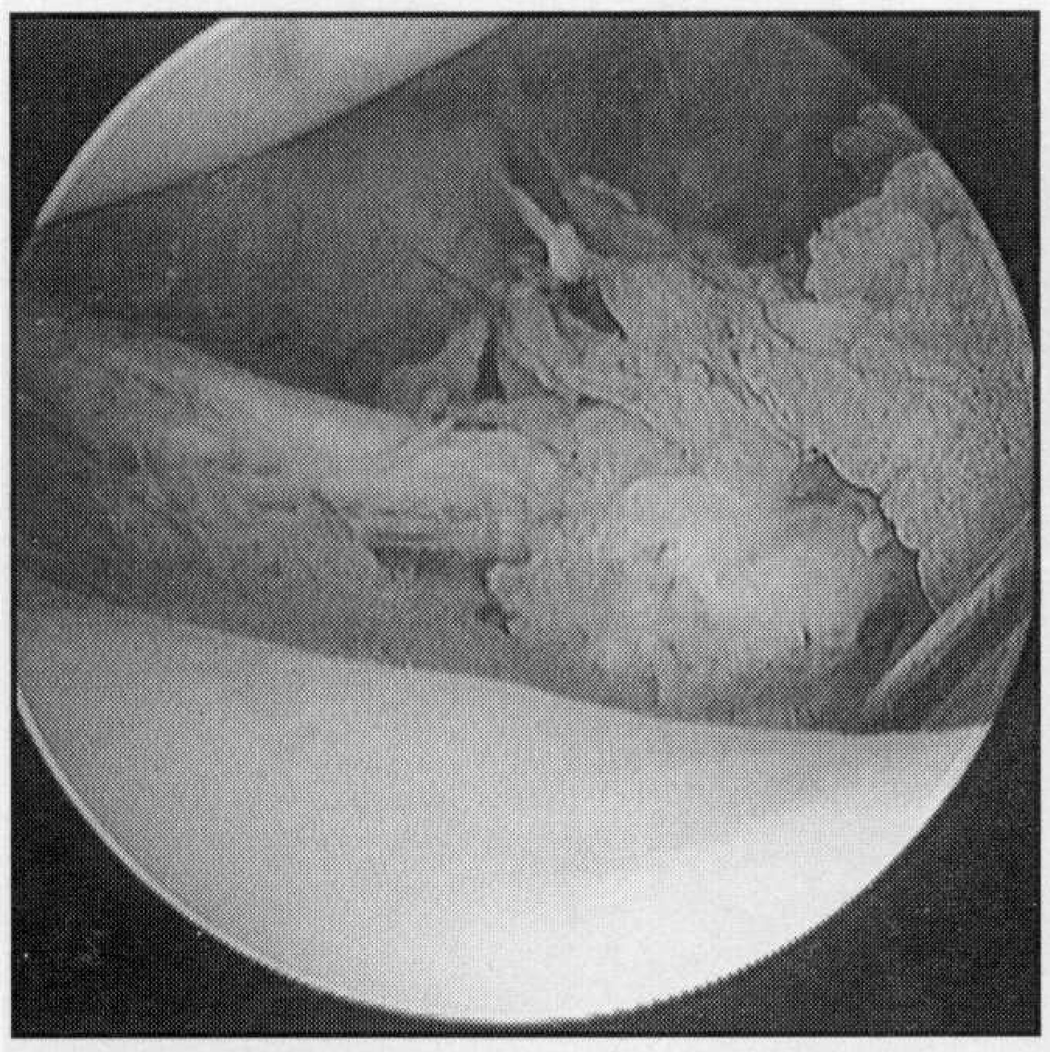


Fig. 3 Arthroscopic view of an absent popliteus tendon (empty hiatus).

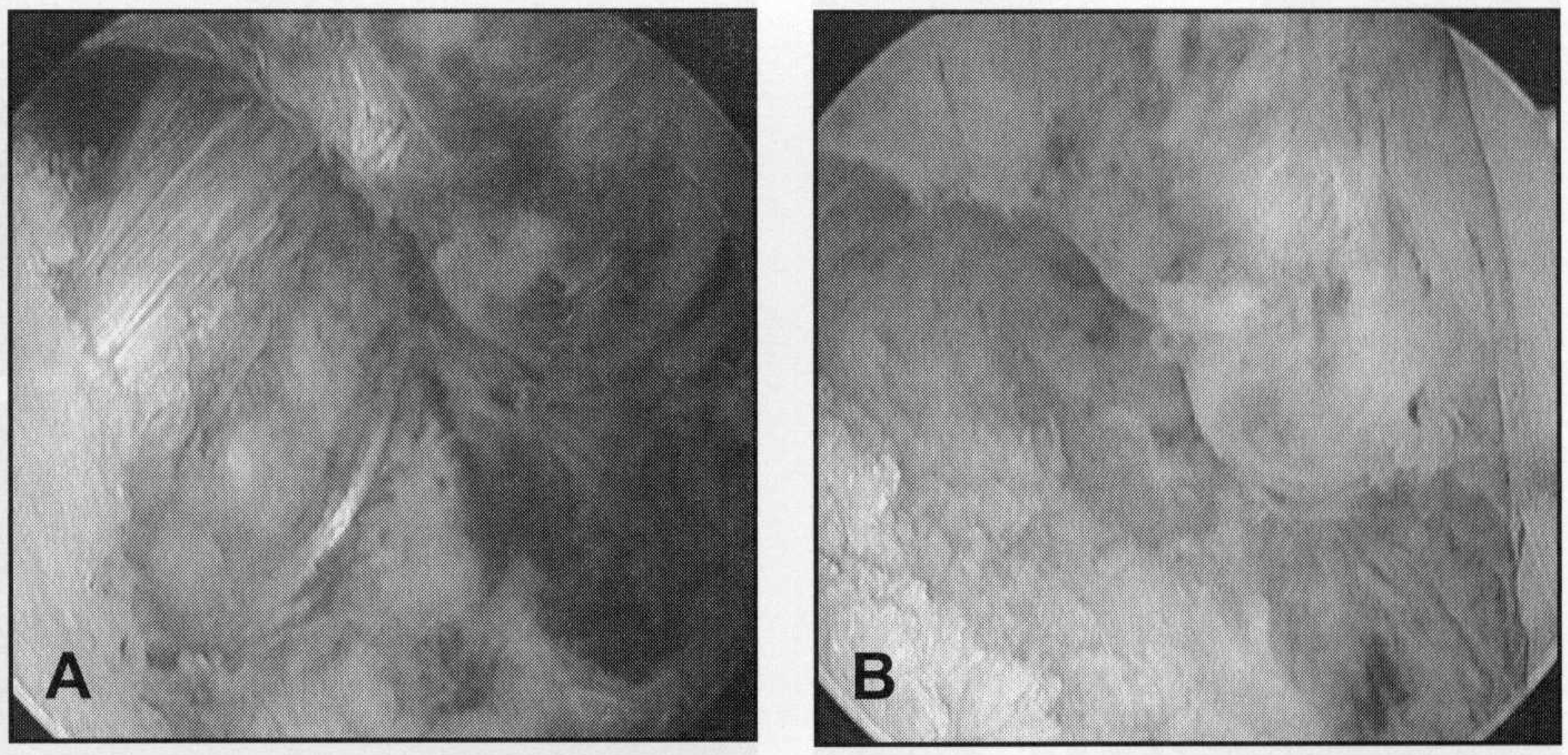


Fig. 4 Arthroscopic view of medial femoral condyle (right knee) **A,** After débridement of torn PCL. **B,** After graft passage.

tion. If these gait abnormalities are associated with an underlying varus alignment of the knee, a bony procedure (osteotomy) should precede any soft-tissue reconstruction.

Imaging

Plain radiographs are first carefully evaluated for avulsion fracture, medial Segond fracture,[27] fibular head avulsion (arcuate sign),[28] posterior sagging on the lateral view, lateral joint space widening, and, in skeletally immature patients, physeal injuries. Standing flexion weight-bearing views are mandatory, especially for chronic injuries, to evaluate for medial compartment chondrosis and to assess limb alignment. Nuclear imaging (bone scans) may be useful to detect early arthrosis; however, the clinical implications are unclear. MRI is said to be 100% sensitive and specific in detecting PCL injuries.[29] Its primary benefit is in the evaluation of combined injuries and in surgical planning. Posterolateral corner structures also can be seen with MRI;[30] however, there is significant variability in the interpretation of images. Stress radiography can be extremely beneficial and may prove to be the best way of assessing clinical results. A lateral radiograph with a posterior force allows direct measurement of posterior tibial translation.[31]

Diagnostic Arthroscopy

Although the diagnosis should be clear before surgery is begun, direct and indirect arthroscopic signs of PCL injury have been described.[32] Direct signs include torn fibers, hemorrhage, decreased tension, and laxity. Indirect signs include "sloppy ACL" (ACL pseudolaxity from posterior displacement of the tibia), degenerative changes in the patellofemoral and medial compartments, and altered contact points. The sight of a ruptured PCL often is not as dramatic as seeing the "stump" of a torn ACL, because PCL injuries commonly occur in what has been described as zone II, which is hidden by the ACL.[33]

Arthroscopic findings associated with posterolateral corner injuries, such as an unexpected amount of lateral opening during arthroscopic examination of the lateral compartment (drive-through sign), are also important to recognize.[34] The popliteus tendon origin on the femur should be inspected to determine if there is an avulsion off the femur or if it is injured (Fig. 3).

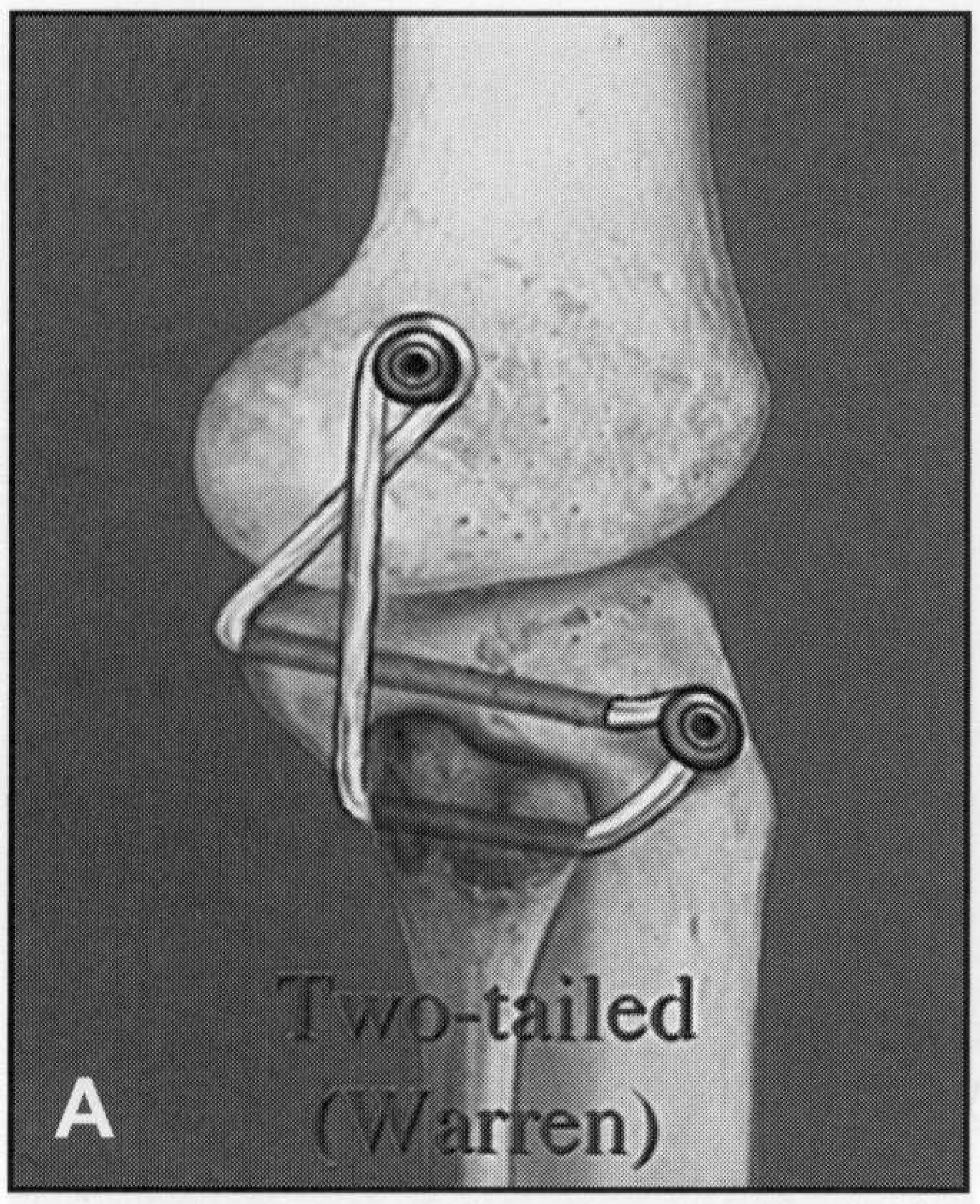
Treatment

Although some low-grade, isolated PCL injuries may do well without surgery, more severe and combined injuries have a worse outcome.[35,36] Shelbourne and associates[37] reported good results (mean Noyes score, 84.2; mean Lysholm score, 83.4) with nonsurgical management of isolated PCL injuries, but only patients with grade 2 laxity or less were included in the study group. Isolated PCL injuries with less than 10 mm of laxity (flush with the medial femoral condyle) can be treated with extension splinting, quadriceps rehabilitation, and progressive activity. Nonsurgical treatment of chronic PCL-injured knees includes quadriceps strength training, activity modification, and careful follow-up.

Although most surgeons agree that PCL bony avulsion injuries[38] and severe combined PCL injures require surgical intervention, the best management of more severe isolated PCL injuries

Table 1
Principles of Reconstruction

- Identification and treatment of all pathology
- Protection of the neurovascular structures
- Accurate tunnel placement
- Recreation of anatomic graft insertion sites
- Strong graft material
- Minimal graft bending
- Appropriate tension of the graft
- Secure primary and (if necessary) backup graft fixation
- Structured postoperative rehabilitation program



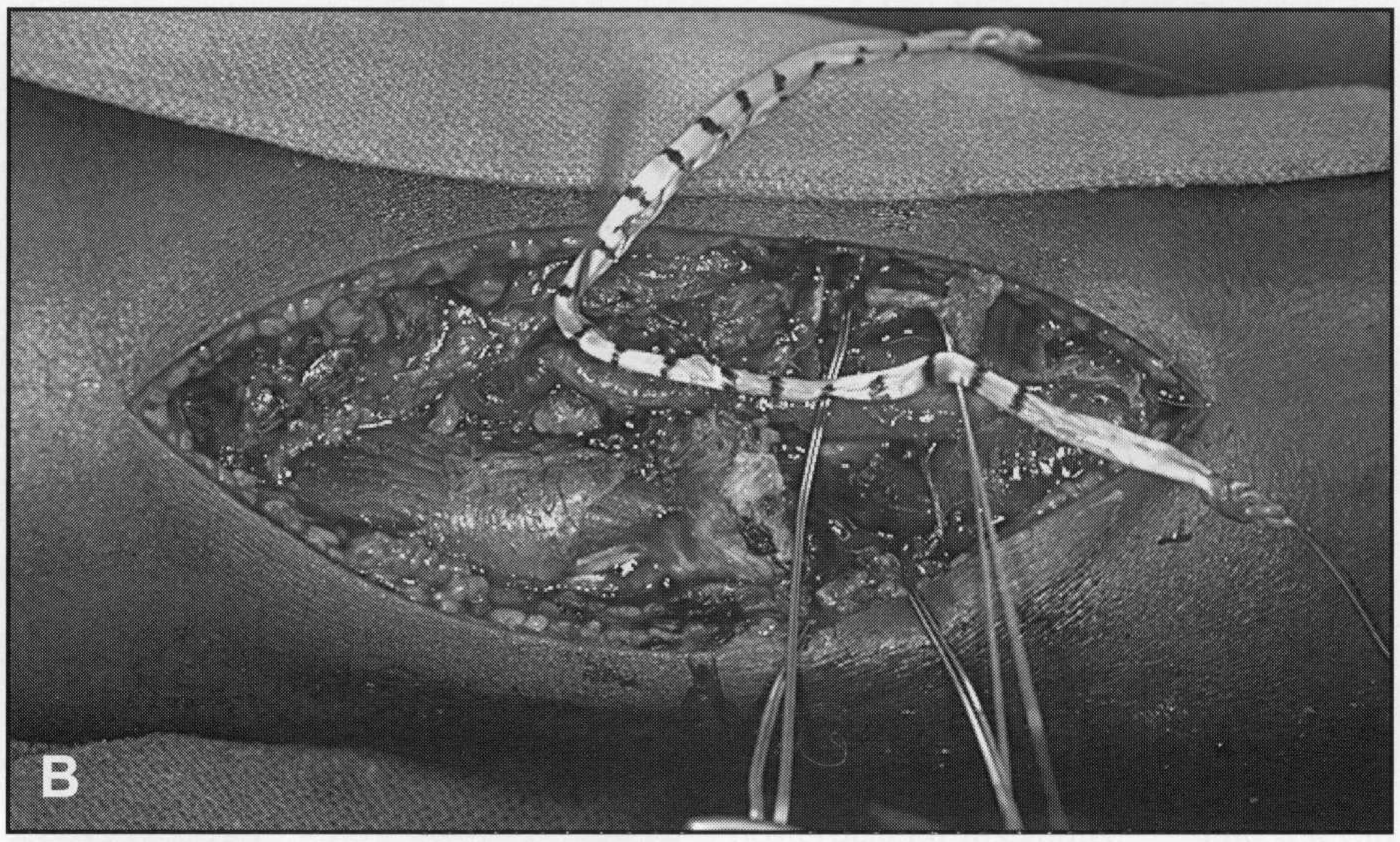


Fig. 5 A, Schematic and **B,** Clinical photograph of posterolateral corner reconstruction using the two-tailed (Warren) method.

remains controversial. Many or even most severe "isolated" PCL injuries may in fact be combined PCL/posterolateral corner injuries requiring combined reconstruction techniques. Other associated knee injuries such as meniscal tears and acute chondral injury also may benefit from surgical intervention.

Combined ligamentous injuries are best treated within 2 weeks of the injury. Combined ACL/PCL injuries represent a functional knee dislocation and all of the precautions taken in the presence of these injuries (including thorough neurovascular examination and studies) are appropriate.

The principles of reconstruction of PCL injuries are outlined in Table 1. The goal of PCL reconstruction is to reproduce the normal anterior tibial step-off and to restore the restraint to posterior displacement (Fig. 4). Likewise, the goal of posterolateral corner reconstruction is to restore injured structures. Hamstring grafts have been found to be very effective for this purpose. Although there are a variety of procedures described for posterolateral corner reconstruction, a two-tailed (through the fibula neck and tibia posterior to anterior) reconstruction is preferred. (Fig. 5).

Postoperative rehabilitation principles deserve special emphasis. Patients are initially placed in a brace locked in extension. Protected (prone) range of motion negates the effect of gravity and reduces the incidence of postoperative stiffness. Quadriceps rehabilitation is emphasized and hamstring rehabilitation is deemphasized during the early postoperative period.

References

1. Harner CD, Xerogeanes JW, Livesay GA, et al: The human posterior cruciate ligament complex: An interdisciplinary study: Ligament morphology and biomechanical evaluation. *Am J Sports Med* 1995;23:736-745.
2. Harner CD, Baek GH, Vogrin TM, Carlin GJ, Kashiwaguchi S, Woo SL: Quantitative analysis of human cruciate ligament insertions. *Arthroscopy* 1999;15:741-749.
3. Terry GC, LaPrade RF: The posterolateral aspect of the knee: Anatomy and surgical approach. *Am J Sports Med* 1996;24:732-739.
4. Prietto MP, Bain JR, Stonebrook SN, Settlage RA: Tensile strength of the human posterior cruciate ligament (PCL). *Trans Orthop Res Soc* 1988;13:195.
5. Gollehon DL, Torzilli PA, Warren RF: The role of the posterolateral and cruciate ligaments in the stability of the human knee: A biomechanical study. *J Bone Joint Surg Am* 1987;69:233-242.
6. Grood ES, Stowers SF, Noyes FR: Limits of movement in the human knee: Effect of sectioning the posterior cruciate ligament and posterolateral structures. *J Bone Joint Surg Am* 1988;70:88-97.
7. Vogrin TM, Höher J, Aroen A, Woo SL, Harner CD: Effects of sectioning the posterolateral structures on knee kinematics and in situ forces in the posterior cruciate ligament. *Knee Surg Sports Traumatol Arthrosc* 2000;8:93-98.
8. Fox RJ, Harner CD, Sakane M, Carlin GJ, Woo SL: Determination of the in situ forces in the human posterior cruciate ligament using robotic technology: A cadaveric study. *Am J Sports Med* 1998;26:395-401.
9. Harner CD, Vogrin TM, Kanamori A, Yagi M, Janaushek MA, Woo SLY: Effects of axial compression on the function of the posterior cruciate ligament. *Trans Orthop Res Soc* 2000;25:780.
10. Höher J, Harner CD, Vogrin TM, et al: Hamstring loading increases in situ forces in the PCL. *Trans ORS* 1998;23:48.
11. Harner CD, Höher J, Vogrin TM, Carlin GJ, Woo SL: The effects of a popliteus muscle load on in situ forces in the posterior cruciate ligament and on knee kinematics: A human cadaveric study. *Am J Sports Med* 1998;26:669-673.
12. Skyhar MJ, Warren RF, Ortiz GJ, Schwartz E, Otis JC: The effects of sectioning of the posterior cruciate ligament and the posterolateral complex on the articular contact pressures within the knee. *J Bone Joint Surg Am* 1993;75:694-699.
13. Clancy WG, Shelbourne KD, Zoellner GB, Keene JS, Reider B, Rosenberg TD: Treatment of knee joint instability secondary to rupture of the posterior cruciate ligament: Report of a new procedure. *J Bone Joint Surg Am* 1983;65:310-322.
14. Dejour H, Walch G, Peyrot P: The natural history of rupture of the posterior cruciate ligament. *Fr J Orthop Surg* 1988;2:112-120.
15. Geissler W, Whipple TL: Intraarticular abnormalities in association with posterior cruciate ligament injuries. *Am J Sports Med* 1993;21:846-849.
16. Bergfeld JA, McAllister DR, Parker RD: Abstract: A biomechanical comparison of posterior cruciate ligament reconstruction techniques. *Arthroscopy* 1999;15(suppl 1):S45.
17. Harner CD, Janaushek MA, Kanamori A, Yagi

M, Vogrin TM, Woo SL: Biomechanical analysis of a double-bundle posterior cruciate ligament reconstruction. *Am J Sports Med* 2000;28:144-151.

18. Fanelli GC, Edson CJ: Posterior cruciate ligament injuries in trauma patients: Part II. *Arthroscopy* 1995;11:526-529.
19. Harner CD, Vogrin TM, Höher J, Ma CB, Woo SL: Biomechanical analysis of a posterior cruciate ligament reconstruction: Deficiency of the posterolateral structures as a cause of graft failure. *Am J Sports Med* 2000;28:32-39.
20. Veltri DM, Deng XH, Torzilli PA, Warren RF, Maynard MJ: The role of the cruciate and posterolateral ligaments in the stability of the knee: A biomechanical study. *Am J Sports Med* 1995;23:436-443.
21. Sugita T, Amis AA: Anatomic and biomechanical study of the lateral collateral and popliteofibular ligaments. *Am J Sports Med* 2001;29:466-472.
22. Fowler PJ, Messieh SS: Isolated posterior cruciate ligament injuries in athletes. *Am J Sports Med* 1987;15:553-557.
23. Miller MD, Bergfeld JA, Fowler PJ, Harner CD, Noyes FR: The posterior cruciate ligament injured knee: Principles of evaluation and treatment. *Instr Course Lect* 1999;48:199-207.
24. Daniel DM, Stone ML, Barnett P, Sachs R: Use of the quadriceps active test to diagnose posterior cruciate-ligament disruption and measure posterior laxity of the knee. *J Bone Joint Surg Am* 1988;70:386-391.
25. Veltri DM, Warren RF: Isolated and combined posterior cruciate ligament injuries. *J Am Acad Orthop Surg* 1993;1:67-75.
26. Cooper DE: Tests for posterolateral instability of the knee in normal subjects: Results of examination under anesthesia. *J Bone Joint Surg Am* 1991;73:30-36.
27. Hall FM: Radiographic diagnosis and accuracy in knee joint effusions. *Skeletal Radiol* 1997;26:553-555.
28. Shindell R, Walsh WM, Connolly JF: Avulsion fracture of the fibula: The "arcuate sign" of posterolateral knee instability. *Nebr Med J* 1984;69:369-371.
29. Gross ML, Grover JS, Bassett LW, Seeger LL, Finerman GA: Magnetic resonance imaging of the posterior cruciate ligament: Clinical use to improve diagnostic accuracy. *Am J Sports Med* 1992;20:732-737.
30. LaPrade RF, Gilbert TJ, Bollom TS, Wentorf F, Chaljub G: The magnetic resonance imaging appearance of individual structures of the posterolateral knee: A prospective study of normal knees and knees with surgicall verified grade III injuries. *Am J Sports Med* 2000;28:191-199.
31. Hewett TE, Noyes FR, Lee MD: Diagnosis of complete and partial posterior cruciate ligament ruptures: Stress radiography compared with KT-1000 arthrometer and posterior drawer testing. *Am J Sports Med* 1997;25:648-655.
32. Fanelli GC, Giannotti BF, Edson CJ: The posterior cruciate ligament arthroscopic evaluation and treatment. *Arthroscopy* 1994;10:673-688.
33. Fanelli GC, Edson CJ: Posterior cruciate ligament injuries in trauma patients: Part II. *Arthroscopy* 1995;11:526-529.
34. LaPrade RF: Arthroscopic evaluation of the lateral compartment of knees with grade 3 posterolateral knee complex injuries. *Am J Sports Med* 1997;25:596-602.
35. Torg JS, Barton TM, Pavlov H, Stine R: Natural history of the posterior cruciate ligament-deficient knee. *Clin Orthop* 1989;246:208-216.
36. Keller PM, Shelbourne KD, McCarroll JR, Rettig AC: Nonoperatively treated isolated posterior cruciate ligament injuries. *Am J Sports Med* 1993;21:132-136.
37. Shelbourne KD, Davis TJ, Patel DV: The natural history of acute, isolated, nonoperatively treated posterior cruciate ligament injuries: A prospective study. *Am J Sports Med* 1999;27:276-283.
38. Meyers MH: Isolated avulsion of the tibial attachment of the posterior cruciate ligament of the knee. *J Bone Joint Surg Am* 1975;57:669-672.