

T. M. Vogrin
J. Höher
A. Årøen
S. L-Y. Woo
C. D. Harner

Effects of sectioning the posterolateral structures on knee kinematics and in situ forces in the posterior cruciate ligament

Received: 5 January 1999
Accepted: 25 May 1999

Abstract The objective of this study was to determine the effects of sectioning the posterolateral structures (PLS) on knee kinematics and in situ forces in the posterior cruciate ligament (PCL) in response to external and simulated muscle loads. Ten human cadaveric knees were tested using a robotic/universal force-moment sensor testing system. The knees were subjected to three loading conditions: (a) 134-N posterior tibial load, (b) 5-Nm external tibial torque, and (c) isolated hamstring load (40 N biceps/40 N semimembranosus). The knee kinematics and in situ forces in the PCL for the intact and PLS-deficient knee conditions were determined at full extension, 30°, 60°, 90°, and 120° of knee flexion. Under posterior tibial loading posterior tibial translation with PLS deficiency increased significantly at all flexion angles by 5.5 ± 1.5 mm to 0.8 ± 1.2 mm at full extension and 90°, respectively. The corresponding in situ forces in the PCL increased by 17–19 N at full extension and 30° of

knee flexion. Under the external tibial torque, external tibial rotation increased significantly with PLS deficiency by $15.1 \pm 1.6^\circ$ at 30° of flexion to $7.7 \pm 3.5^\circ$ at 90°, with the in situ forces in the PCL increasing by 15–90 N. The largest increase occurred at 60° to 120° of knee flexion, representing forces two to six times of those in the intact knee. Under the simulated hamstring load, posterior tibial translation and external tibial and varus rotations also increased significantly at all knee flexion angles with PLS deficiency, but this was not so for the in situ forces in the PCL. Our data suggest that injuries to the PLS put the PCL and other soft tissue structures at increased risk of injury due to increased knee motion and the elevated in situ forces in the PCL.

Key words Posterior cruciate ligament · Posterolateral structure · Knee kinematics · In situ forces · Hamstring

T. M. Vogrin · J. Höher · A. Årøen
S. L-Y. Woo · C. D. Harner (✉)
Musculoskeletal Research Center,
Department of Orthopaedic Surgery,
University of Pittsburgh,
P.O. Box 71199,
Pittsburgh, PA 15213, USA
e-mail: tmvst5@pitt.edu
Tel.: +1-412-648-2000
Fax: +1-412-648-2001

Introduction

While injuries to the posterior cruciate ligament (PCL) of the knee can occur as isolated tears, the majority are accompanied by damage to one or more other structures of the knee, including the anterior cruciate ligament or medial collateral ligament, and may involve even complete knee dislocations [3, 6, 10, 14, 15]. The high incidence of

such combined injuries has been attributed to high-velocity trauma. The most common concomitant injury occurring with the PCL is that of the posterolateral structures (PLS), which has been reported to occur in up to 60% of all PCL injuries [6].

The PLS of the knee are roughly divided into two primary components, the lateral collateral ligament and the popliteus complex [12, 22, 24, 25, 27–29] (Fig. 1). The popliteus complex consists of the muscle-tendon unit and

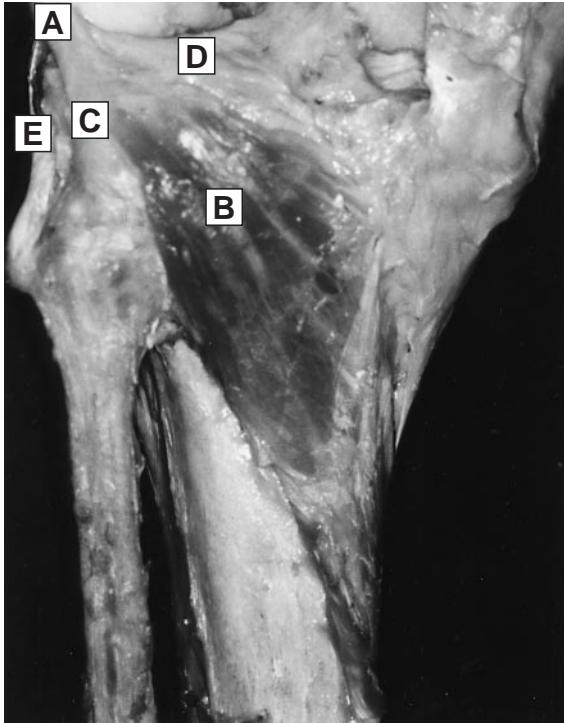


Fig.1 Photograph detailing the anatomy of the posterolateral structures. *A* Popliteus tendon, *B* popliteus muscle, *C* popliteofibular ligament, *D* popliteotibial and popliteomeniscal fascicles, *E* lateral collateral ligament

ligamentous connections between the tendon and the fibula, tibia, and meniscus, known as the popliteofibular ligament and popliteotibial and popliteomeniscal fascicles, respectively [16, 20, 24, 25]. With its tendinous and ligamentous components, the popliteus complex imparts both static and dynamic restraint to the knee [2, 11, 18, 24, 26]. The arcuate ligament complex and fabellofibular ligament are also considered part of the PLS, but the importance of these structures is believed to be relatively minor [24, 25, 27].

The PLS and the PCL work together to resist external tibial rotation and posterior tibial translation [8, 9, 24, 28]. Under a 5 Nm external tibial torque, isolated sectioning of the PLS resulted in an increase of approximately 10–15° of rotation, while sectioning of the PCL resulted in no changes in external rotation. However, with combined PCL/PLS deficiency, external tibial rotation became much larger, up to 30–40° at 60–90° of flexion [8, 9]. Similar increases are observed under posterior tibial loading [8, 9, 28].

The dynamic component of the popliteus complex has also been shown to have a significant effect on knee kinematics and on the PCL, with a simulated popliteus muscle load resulting in significant decreases in posterior tibial translation and in situ force in the PCL [11]. Previous work in our research center has also indicated that the in

situ forces in the PLS increase significantly when the PCL is absent [12]. Because of the biomechanical interaction between these two structures we hypothesize that the reverse effect would be true, that is, that PLS deficiency would significantly increase the in situ forces in the PCL. Therefore the objective of this study was to determine the effects of sectioning the PLS on knee kinematics in multiple degrees of freedom (DOF) and on the in situ forces in the PCL, when the knee is subjected to external and muscle loads. To study this we tested human cadaveric knees using a robotic/universal force-moment sensor testing system. This testing system can determine multiple DOF knee kinematics and in situ forces in knee ligaments, without making contact with any soft tissue [7, 21]. All tests were performed in the same knee, thus minimizing variability in data from testing different specimens.

Materials and methods

Ten fresh-frozen human cadaveric knees (age range 52–86 years) were tested. The knees were stored in airtight plastic bags at -20°C . At 24 h prior to testing the specimens were thawed at room temperature. All knees were radiographed and examined for any deformities or previous ligament injury; if any were detected, the specimen was excluded from the study. The femur and tibia were cut to approximately 20 cm in length from the joint line, and all muscle and soft tissue proximal and distal to the joint were dissected, enabling the exposed part of the bones to be potted in an epoxy compound for fixation in custom-designed metal clamps. Sutures were tied to the semimembranosus and biceps tendons to enable the application of simulated hamstring loads. This constituted the intact knee condition.

The intact knees were tested using a robotic/universal force-moment sensor (UFS) testing system. The femur was first rigidly fixed relative to the base of the robot, while the tibia was securely fastened through the UFS to the end-effector of the robot. The robot (Unimate, Westinghouse, Pittsburgh, Penn., USA) is capable of position control of the joint in six DOF. The UFS (JR³, Woodland Hills, Calif., USA) can measure three orthogonal forces and moments applied to its face and provides force and moment feedback to the robot, enabling it to operate in a force (or load) control mode as well. In combination the robotic/UFS testing system can measure multiple DOF knee kinematics and in situ forces in knee ligaments under externally applied loads at selected knee flexion angles without making contact with any ligament [7, 21].

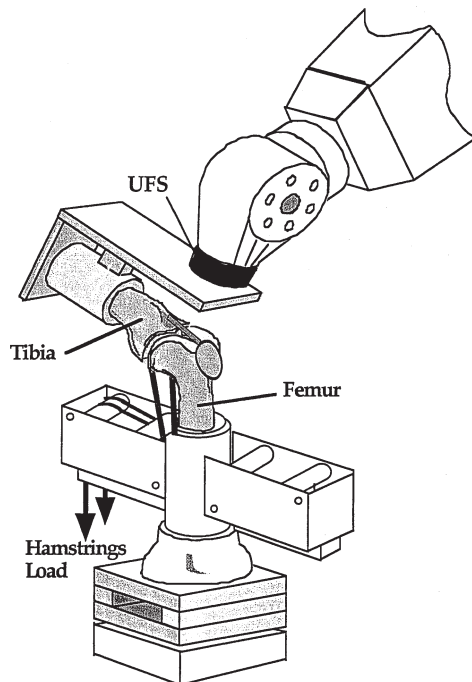
The path of passive flexion-extension of the intact knee from full extension to 120° of knee flexion was first determined. To achieve this the robotic/UFS testing system minimized all external forces and moments acting on the joint through the range of flexion-extension while operating under load control mode. This series of positions served as the reference positions for the knee specimen for the application of external loads and for the measurement of knee kinematics at selected knee flexion angles throughout the test.

The study protocol is detailed in Table 1. With the robot operating under load control, two external loads, 134 N posterior tibial load, and 5 Nm external tibial torque, were applied to the knee at full extension, 30°, 60°, 90°, and 120° of knee flexion. These loading conditions were chosen to simulate the clinical examinations for PCL and PLS deficiency, namely, the posterior drawer and external rotation tests [4, 10, 14, 24], and were in effect applied at a point 10 cm distal to the joint line. For each applied load, the resulting five DOF knee kinematics were recorded (anterior-posterior, medial-lateral, and proximal-distal tibial translations, and internal-external and varus-valgus rotations).

Table 1 Testing protocol and data obtained

Testing protocol	Data obtained
I. Intact knee	
A. 134 N posterior tibial load	Knee kinematics (A-P, M-L, P-D translation, I-E, V-V rotation) for A, B, C
B. 5 Nm external tibial torque	
C. Isolated hamstrings load	
II. PLS-deficient knee	Knee kinematics for A, B, C
Reapply A, B, C	
III. Section PCL	
IV. Repeat intact knee kinematics	In situ force in the PCL, intact knee
V. Measure new forces and moments	
VI. Repeat PLS-deficient knee kinematics	In situ force in the PCL, PLS-deficient knee
Measure new forces and moments	

The knees were further tested in response to a simulated hamstring load of 40 N each applied to the biceps and semimembranosus tendons via a customized jig with a set of pulleys and weights attached to the femoral clamp (Fig. 2) [13, 17]. This loading condition was chosen because hamstring loads have been shown to cause a posterior tibial translation and an increase in the in situ forces in the PCL [13]. The cables were oriented parallel to the femur to mimic the lines of action of the muscles in vivo. Operating under load control, the robotic/UFS testing system found the new position of the knee in response to the applied muscle load. This was defined as the position of the knee at which all external forces and moments on the joint were minimized, and the resulting five DOF knee kinematics were recorded. The muscle load

**Fig. 2** Schematic of method for application of simulated muscle loads using robotic/UFS testing system

was then removed and the UFS measured the forces and moments on the joint at the new knee position.

The PLS, including the popliteus complex and the lateral collateral ligament, were then transected using a scalpel. The previously determined kinematics of the intact knee were repeated by the robot while operating in a position-controlled mode, while the UFS measured a new set of forces and moments. The identical loading conditions were then applied to the PLS-deficient knee, and the resulting knee kinematics were recorded for each loading condition. The PCL was then sectioned arthroscopically. The previously determined kinematics of both the intact and PLS-deficient knee conditions were repeated by the robot for each loading condition. At the same time the UFS measured a new set of force and moment data. The decrease in force observed by the UFS between these two tests with identical knee positions (i.e., before and after the PCL was sectioned) can be attributed to the PCL. This is known as the principle of superposition and yields the in situ forces in the PCL [21].

The data obtained consisted of the five DOF kinematics and the in situ forces in the PCL, in both the intact and PLS-deficient knees, for each loading condition. Statistical analysis was performed using a two-factor repeated-measure analysis of variance because all tests were performed on the same specimen. The two factors investigated were knee condition (i.e., intact and PLS-deficient) and knee flexion angle. The dependent variables investigated were knee kinematics and the in situ forces in the PCL. Multiple contrasts were carried out to evaluate the effects of knee condition at specific flexion angles. Significance was set at $P < 0.05$.

Results

For ease of presentation the data are separated by the three loading conditions. Data on knee kinematics is first presented for each loading condition, followed by the data on the in situ force in the PCL.

Posterior tibial load

In response to the 134-N posterior tibial load, posterior tibial translation of the intact knee ranged from 7.3 ± 0.8 mm to 5.6 ± 1.4 mm at full extension and 90° , respectively. With PLS deficiency, these values increased to 12.8 ± 2.0 mm and 6.4 ± 1.7 mm, representing a significant increase for all knee flexion angles tested with the largest increase occurring at full extension ($P < 0.05$; Fig. 3). There was also a significant increase in the coupled external tibial rotation with PLS deficiency for all flexion angles tested. The increase ranged from $2.5 \pm 3.7^\circ$ to $16.4 \pm 5.7^\circ$ of rotation and it was most pronounced at 30° of knee flexion ($P < 0.05$). The increase in coupled varus rotation was minimal, ranging from only $1.1 \pm 1.3^\circ$ to $2.3 \pm 1.5^\circ$ ($P < 0.05$), while medial-lateral and proximal-distal translations varied by less than 1 mm. Further, the in situ forces in the PCL for the intact knee increased with knee flexion angle, ranging from 42 ± 24 N at full extension to 158 ± 9 N at 90° of knee flexion (Fig. 4). After sectioning the PLS these forces were 59 ± 30 N and 134 ± 19 N at full extension and 30° of flexion, respectively, representing a significant increase of 17–19 N ($P < 0.05$). How-

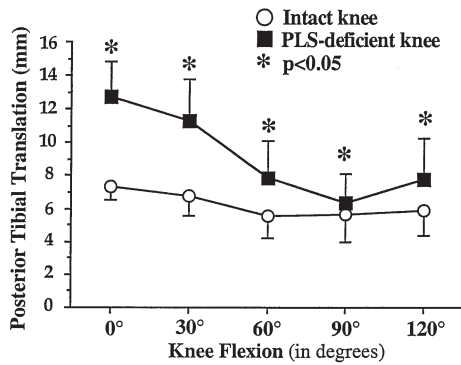


Fig.3 Posterior tibial translation (mean \pm SD) in the intact and PLS-deficient knees in response to a 134 N applied posterior tibial load

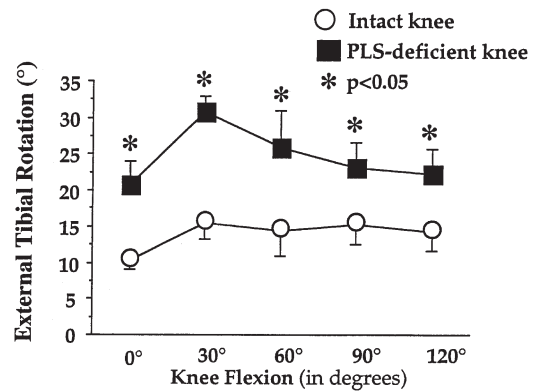


Fig.5 External tibial rotation (mean \pm SD) in response to a 5 Nm external tibial torque in the intact and PLS-deficient knees

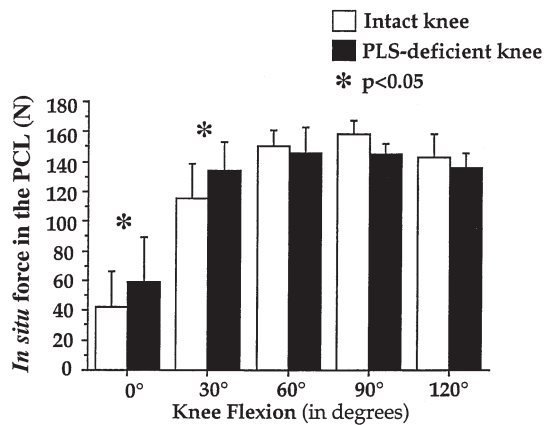


Fig.4 In situ forces in the PCL (mean \pm SD) in response to a 134-N posterior tibial load in the intact and PLS-deficient knees

ever, no significant increases occurred from 60° to 120° of knee flexion ($P > 0.05$).

External tibial torque

In response to the 5-Nm external tibial torque, external tibial rotation of the intact knee ranged from $10.3 \pm 1.2^\circ$ at full extension to $15.6 \pm 2.3^\circ$ at 30° of knee flexion. After sectioning the PLS, these values increased at all flexion angles ($P < 0.05$, Fig. 5). This increase ranged from $15.1 \pm 1.6^\circ$ to $7.7 \pm 3.5^\circ$ at 30° and 90° of flexion, respectively. There was also a significant increase in the coupled posterior tibial translation with PLS deficiency at all knee flexion angles, ranging from 9.2 ± 1.6 mm at 30° to 3.4 ± 1.8 mm at 90° ($P < 0.05$). Coupled varus rotation increased minimally by 1.5 – 4.0° at all flexion angles ($P < 0.05$), while small but significant increases of less than 2 mm were also observed in coupled medial and distal translations ($P < 0.05$). The in situ force in the PCL in the intact knee ranged from 6 ± 5 N at full extension to 43 ± 21 N at 90° of flexion and tended to increase with knee

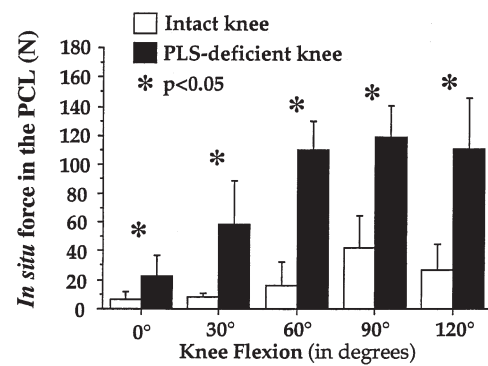


Fig.6 In situ forces in the PCL (mean \pm SD) in response to a 5 Nm external tibial torque in the intact and PLS-deficient knees

flexion up to 90°. With PLS deficiency, these forces increased significantly at all flexion angles tested and ranged from 21 ± 14 N to 116 ± 23 N ($P < 0.05$, Fig. 6). The increase in force ranged from 15 to 90 N and was largest at 60–120° of knee flexion, where the in situ force in the PCL was two to six times larger than in the intact knee.

Simulated hamstring load

With the simulated hamstring load, PLS deficiency caused significant increases in posterior tibial translation of 0.9 ± 0.7 mm to 2.4 ± 1.6 mm at 120° and 30°, respectively (Table 2). Significant changes in external tibial rotation were also observed at all flexion angles by as much as $6.5 \pm 4.2^\circ$ at 30° of knee flexion ($P < 0.05$). Small changes in medial and distal translations and varus rotation also occurred with PLS deficiency. The in situ force in the PCL ranged from 13 ± 6 N at full extension to 86 ± 19 N at 90° of knee flexion (Table 3). However, with PLS deficiency these forces decreased significantly by 11 ± 13 N at 60° to 33 ± 16 N at 90° of knee flexion ($P < 0.05$).

Table 2 Posterior tibial translation (mm, mean \pm SD) for the intact and PLS-deficient knee under a simulated hamstring load

Knee flexion ($^{\circ}$)	Intact knee	PLS-deficient knee
Full extension	1.3 \pm 0.6	2.6 \pm 1.3*
30 $^{\circ}$	2.5 \pm 0.5	4.9 \pm 1.6*
60 $^{\circ}$	2.3 \pm 1.2	3.9 \pm 1.5*
90 $^{\circ}$	2.4 \pm 1.7	3.3 \pm 1.5*
120 $^{\circ}$	2.0 \pm 1.1	3.0 \pm 1.3*

* $P < 0.05$ vs. intact**Table 3** In situ force in the PCL (N, mean \pm SD) in the intact and PLS-deficient knee under a simulated hamstring load

Knee flexion ($^{\circ}$)	Intact knee	PLS-deficient knee
Full extension	13 \pm 6	7 \pm 4
30 $^{\circ}$	28 \pm 15	24 \pm 13
60 $^{\circ}$	63 \pm 15	51 \pm 16*
90 $^{\circ}$	86 \pm 19	53 \pm 15*
120 $^{\circ}$	61 \pm 14	35 \pm 10*

* $P < 0.05$ vs. intact knee

Discussion

In this study, we determined the effects of sectioning the PLS of the knee on multiple degree of freedom knee kinematics and in situ forces in the PCL in response to several external and muscle loading conditions. We observed significant increases in posterior tibial translation and external tibial and varus rotations under a posterior tibial load and external tibial torque as well as under a simulated hamstring load. These increases were largest at full extension and at 30 $^{\circ}$ of knee flexion, which is consistent with previous studies indicating that the PLS provide the most restraint to these loading conditions when the knee is near full extension [8, 9, 24, 28].

The in situ forces in the PCL increased significantly with PLS deficiency in response to the 134 N posterior tibial load and 5 Nm external tibial torque, thus partially confirming our hypothesis. For the former, the in situ forces increased by 17–19 N at full extension and 30 $^{\circ}$ of flexion, while for the latter, the increase was up to sixfold with PLS deficiency, with the largest increases observed with the knee flexed. These findings are, again, consistent with reports that the PLS provide restraint to posterior tibial translation only when the knee is near extension, but resist external tibial rotation at all knee flexion angles [8, 9, 24, 28]. Our in situ force data are consistent with those of Markolf et al. [19], who reported similar increases from full extension to 90 $^{\circ}$ of flexion.

Surprisingly, however, under the simulated hamstring load in situ forces in the PCL actually decreased with PLS deficiency at higher knee flexion angles by up to 30 N, contrary to our hypothesis. We believe that this decrease

in the in situ force is in part due to the small but significant medial translation of the tibia which was observed after sectioning the PLS. Changes in the amount of coupled distal translation may also have contributed to this decrease in force. The isolated hamstring load tested in this study was chosen because it has been shown to result in posterior tibial translation as well as in situ forces in the PCL on the order of 100 N; addition of a quadriceps muscle load has been shown to reduce these effects, however [13]. In future studies, we will investigate the effects of other muscle groups as well, including simulated gastrocnemius and popliteus loads. Previous studies have indicated that gastrocnemius contraction increases strain in the PCL as the knee flexes [5, 23], while a popliteus load can decrease the in situ forces in the PCL [11].

It is important to note that in this study we simulated an injury to the PLS by sectioning the popliteus complex at its femoral insertion as well as the lateral collateral ligament. LaPrade and Terry [16] reported that in a series of 71 patients with PLS injuries, 75% had damage to two or more regions (i.e., lateral, posterior, and fibular head). Nevertheless, actual PLS injuries may involve rupturing only one or more of its components [1, 16]. Previous studies have found that with sectioning merely one component of the PLS (e.g., lateral collateral ligament, popliteus tendon, posterior capsule), the changes in knee kinematics would be significantly less than with sectioning two or more components [8, 9, 28]. Hence our data do represent a worst-case scenario.

We have demonstrated that deficiency of the PLS results in significant increases in posterior tibial translation and external tibial and varus rotations when the knee is subjected to external and simulated muscle loads. We also found that the in situ forces in the PCL can increase significantly, suggesting that deficiency of the PLS may elevate the risk of the PCL for injury. The additional stabilization provided by the dynamic component of the PLS would also be lost under these circumstances. Our findings further suggest that if an isolated PCL reconstruction were performed in the setting of a combined PCL/PLS injury, we can hypothesize that the PCL graft may be at a higher risk, particularly when the knee is subjected to external tibial torques. In the future, we plan to examine the effects of PLS deficiency on the PCL reconstructed knee using the robotic/UFS testing system. Additional studies designed to evaluate various reconstructions of the PCL and PLS will be needed to identify procedures that restore both knee kinematics and the in situ forces in the PCL and PLS.

Acknowledgements The authors acknowledge the assistance of Theodore W. Rudy, M.A., and Gregory J. Carlin, M.S., and the financial support of the Whitaker Foundation and the Musculoskeletal Research Center.

References

1. Baker CL Jr, Norwood LA, Hughston JC (1983) Acute posterolateral rotatory instability of the knee. *J Bone Joint Surg Am* 65:614–618
2. Basmajian JV, Lovejoy JF Jr (1971) Functions of the popliteus muscle in man. A multifactorial electromyographic study. *J Bone Joint Surg Am* 53:557–562
3. Clancy WG Jr, Sutherland TB (1994) Combined posterior cruciate ligament injuries. *Clin Sports Med* 13:629–647
4. Clancy WG Jr, Shelbourne KD, Zoellner GB, Keene JS, Reider B, Rosenberg TD (1983) Treatment of knee joint instability secondary to rupture of the posterior cruciate ligament. Report of a new procedure. *J Bone Joint Surg Am* 65:310–322
5. Dürselen L, Claes L, Kiefer H (1995) The influence of muscle forces and external loads on cruciate ligament strain. *Am J Sports Med* 23:129–136
6. Fanelli GC, Edson CJ (1995) Posterior cruciate ligament injuries in trauma patients: part II. *Arthroscopy* 11:526–529
7. Fujie H, Livesay GA, Woo SL-Y, Kashiwaguchi S, Blomstrom G (1995) The use of a universal force-moment sensor to determine in-situ forces in ligaments: a new methodology. *J Biomech Eng* 117:1–7
8. Gollehon DL, Torzilli PA, Warren RF (1987) The role of the posterolateral and cruciate ligaments in the stability of the human knee. A biomechanical study. *J Bone Joint Surg Am* 69:233–242
9. Grood ES, Stowers SF, Noyes FR (1988) Limits of movement in the human knee. Effect of sectioning the posterior cruciate ligament and posterolateral structures. *J Bone Joint Surg Am* 70:88–97
10. Harner CD, Höher J (1998) Evaluation and treatment of posterior cruciate ligament injuries. *Am J Sports Med* 26:471–482
11. Harner CD, Höher J, Vogrin TM, Carlin GJ, Woo SL-Y (1998) Effects of a popliteus muscle load on in situ forces in the PCL and knee kinematics: a cadaveric study. *Am J Sports Med* 26:669–673
12. Höher J, Harner CD, Vogrin TM, Baek GH, Carlin GJ, Woo SL (1998) In situ forces in the posterolateral structures of the knee under posterior tibial loading in the intact and posterior cruciate ligament-deficient knee. *J Orthop Res* 16:675–681
13. Höher J, Vogrin TM, Woo SL-Y, Li G, Årøen A, Carlin GJ, Harner CD (1998) In situ forces in the posterior cruciate ligament in response to muscle loads. *Trans ORS* 23:48
14. Hughston JC, Andrews JR, Cross MJ, Moschi A (1976) Classification of knee ligament instabilities. Part II. The lateral compartment. *J Bone Joint Surg Am* 58:173–179
15. Hughston JC, Jacobson KE (1985) Chronic posterolateral rotatory instability of the knee. *J Bone Joint Surg Am* 67:351–359
16. LaPrade RF, Terry GC (1997) Injuries to the posterolateral aspect of the knee. Association of anatomic injury patterns with clinical instability. *Am J Sports Med* 25:433–438
17. Li G, Rudy TW, Sakane M, Kanamori A, Ma CB, Woo SL-Y (1998) The importance of quadriceps and hamstrings muscle loading on knee kinematics and in-situ forces in the ACL. *J Biomech* 32:395–400
18. Mann RA, Hagy JL (1977) The popliteus muscle. *J Bone Joint Surg Am* 59:924–927
19. Markolf KL, Wascher DC, Finerman GA (1993) Direct in vitro measurement of forces in the cruciate ligaments. Part II: The effect of section of the posterolateral structures. *J Bone Joint Surg Am* 75:387–394
20. Maynard MJ, Deng X, Wickiewicz TL, Warren RF (1996) The popliteofibular ligament. Rediscovery of a key element in posterolateral stability. *Am J Sports Med* 24:311–316
21. Rudy TW, Livesay GA, Woo SL, Fu FH (1996) A combined robotic/universal force sensor approach to determine in situ forces of knee ligaments. *J Biomech* 29:1357–1360
22. Seebacher JR, Inglis AE, Marshall JL, Warren RF (1982) The structure of the posterolateral aspect of the knee. *J Bone Joint Surg Am* 64:536–541
23. Shelburne KB, Pandy MG (1997) A musculoskeletal model of the knee for evaluating ligament forces during isometric contractions. *J Biomech* 30:163–176
24. Stäubli HU (1994) Posteromedial and posterolateral capsular injuries associated with posterior cruciate ligament insufficiency. *Sports Med Arthrosc Rev* 2:146–164
25. Terry GC, LaPrade RF (1996) The posterolateral aspect of the knee. Anatomy and surgical approach. *Am J Sports Med* 24:732–739
26. Tria AJ Jr, Johnson CD, Zawadsky JP (1989) The popliteus tendon. *J Bone Joint Surg Am* 71:714–716
27. Veltri DM, Warren RF (1994) Posterolateral instability of the knee. *J Bone Joint Surg Am* 76:460–472
28. Veltri DM, Deng XH, Torzilli PA, Warren RF, Maynard MJ (1995) The role of the cruciate and posterolateral ligaments in stability of the knee. A biomechanical study. *Am J Sports Med* 23:436–443
29. Watanabe Y, Moriya H, Takahashi K, Yamagata M, Sonoda M, Shimada Y, Tamaki T (1993) Functional anatomy of the posterolateral structures of the knee. *J Arthrosc Relat Surg* 9:57–62