Journal of Sport Rehabilitation, 1999, 8, 304-
© 1999 Human Kinetics Publishers, Inc.

Effects of Injury and Reconstruction of the Posterior Cruciate Ligament on Proprioception and Neuromuscular Control

Marc R. Safran, Christopher D. Harner, Jorge L. Giraldo, Scott M. Lephart, Paul A. Borsa, and Freddie H. Fu

Proprioceptive deficits have been demonstrated following anterior cruciate ligament (ACL) disruption, but little research exists evaluating proprioception in the posterior cruciate ligament (PCL)–deficient and/or –reconstructed knee. We have studied proprioception in PCL-deficient and PCL-reconstructed knees. The following summarizes our protocol and results of proprioceptive testing of kinesthesia and joint position sense in participants with isolated PCL injuries and those who underwent PCL reconstruction. We studied 18 participants with isolated ruptures of the PCL and 10 participants who underwent PCL reconstruction. Proprioception was evaluated by two tests: the threshold to detect passive motion (TTDPM) and the ability to passively reproduce passive positioning (RPP). These assess kinesthesia and joint position sense, respectively. We have shown that isolated PCL deficiency in the human knee does result in reduced kinesthesia and enhanced joint position sense. Thus, the proprioceptive mechanoreceptors in the PCL do appear to have some function. We further found that PCL reconstruction significantly improved kinesthesia at 45° of knee flexion, while 110° was not significantly different between the involved and uninvolved knee in both studies.

Key Words: proprioception, PCL injury, ligament, PCL reconstruction, knee

M.R. Safran is with the Department of Orthopaedic Surgery at Kaiser Permanente, Orange County, Anaheim, CA 92804, and the University of California, Irvine. C.D. Harner and F.H. Fu are with the Center for Sports Medicine, Department of Orthopaedic Surgery, at the University of Pittsburgh, Pittsburgh, PA 15261. J.L. Giraldo is a former Sports Medicine Research Fellow with the Neuromuscular Research Laboratory at the University of Pittsburgh. S.M. Lephart is with the Neuromuscular Research Laboratory at the University of Pittsburgh. P.A. Borsa is with the Sports Medicine Research Laboratory, Division of Kinesiology, at the University of Michigan, Ann Arbor, MI 48109-2214. This material first appeared in *Proprioception and Neuromuscular Control in Sports Injuries,* S.M. Lephart and F.H. Fu (Eds.), and is reprinted with permission from the publisher, Human Kinetics.

Our current understanding of the posterior cruciate ligament (PCL) lags behind that of the ACL by 10 years. Many reasons for this exist, including greater difficulty in making the correct diagnosis, infrequency of isolated injury to the PCL, and the presumed benign course following PCL injury. As such, previous research has concentrated on more common knee ligamentous injuries such as the medial collateral ligament (the most commonly injured knee ligament) and anterior cruciate ligament (the ligament most commonly injured resulting in residual pathological laxity). PCL injuries are being recognized more frequently, stimulating interest in the biomechanics of this ligament as well as the biomechanical and neurosensory contribution of this ligament to knee function. This is particularly true in light of the known neurosensory and mechanical contributions of the anterior cruciate ligament (ACL) in knee function. Further, the natural history, treatment, and techniques of reconstruction of the PCL are topics of great debate at this time. Critical analysis of the documented clinical results after different treatment protocols (including nonoperative) has been handicapped by heterogeneous patient populations, and the lack of uniform criteria to assess and document the results of treatment has resulted in this debate and controversy.

One of the main reasons for controversy about the treatment of isolated posterior cruciate ligament (PCL) injuries is the unclear fate of the isolated PCL-injured knee. Many authors and surgeons note that often patients function well, even in high level sports, with a torn posterior cruciate ligament. This is in direct contradistinction with the ACL-injured knee. The reason for this difference is not clear, as the PCL is a larger and stronger ligament as compared with the ACL.

For years, knee surgeons have postulated that the sensory loss associated with ACL injury may affect the results of ACL repair and reconstruction (2, 7, 34). DuToit (20), Insall and coworkers (31), and others (36, 42, 48) have all advocated certain reconstructive techniques, in part, to increase afferent preservation. A proprioceptive deficit has been demonstrated following ACL disruption (2, 4, 7, 14); however, little research exists evaluating proprioception in the PCL-deficient or reconstructed knee.

Unlike combined ligament injuries involving the PCL, there is much more debate about the natural history and treatment of the "isolated" PCL-deficient knee (10, 15, 17, 19, 22, 33, 46, 52, 58). It is well known that in the ACL-deficient knee, instability and reinjury can lead to arthritis over time (9, 23, 29, 41, 43, 49, 53, 56). Further, authors suggest that function in the ACL-deficient and reconstructed knee is more reliably predicted by proprioceptive ability than physical examination or knee test scores (4, 7, 14, 30). Several studies have shown that proprioceptive deficits that exist in ACL-deficient knees can be partially restored by surgical reconstruction (2, 4, 13). While the relationship of reduced proprioception in the arthritic knee is not known, some suggest that proprioceptive loss results in arthritis (5, 55).

Many authors note that patients with "isolated" PCL deficiency initially function well, while progression to degenerative arthritis over time is less well defined (10, 19, 46, 52). To our knowledge, the University of Pittsburgh Neuromuscular Research Laboratory is the only lab to study the potential proprioceptive deficits in the

PCL-deficient knee and assess the neuromuscular effects of PCL reconstruction. The following summarizes our protocol and the results of proprioceptive testing in subjects with isolated PCL injuries and those who underwent PCL reconstruction.

PCL-Injured Subjects

We prospectively studied 18 subjects with isolated ruptures of the PCL. These subjects ranged in age from 19 to 51 yrs (average: 32 yrs). The time from injury to examination and proprioception testing averaged 29 mo (range: 1–226 mo). No patient could have an inner ear or equilibrium disorder, previous neurological disease, or contralateral knee injury.

All subjects underwent a complete history and thorough knee examination. Examination of the posterior cruciate ligament begins with the knowledge that the medial tibial plateau normally is 10 mm anterior to the medial femoral condyle with the knee in 90° flexion. Posterior drawer testing is then graded as follows. In a grade I injury there is asymmetry side to side, but the medial tibial plateau remains anterior to the medial femoral condyle (less than 10 mm). In a grade II injury the posterior drawer at 90° pushes the tibial plateau to the level of the medial femoral condyle (approximately 10 mm). In a grade III PCL injury, the medial tibial plateau can be pushed posterior to the medial femoral condyle (more than 1 cm). The posterior sag is similarly graded: grade I is when the medial tibial plateau sits further posteriorly than the "normal" contralateral knee but still anterior to the medial femoral condyle with the knee flexed approximately 90°; in a grade II the tibial plateau is at the level of the medial femoral condyle, and a grade III step-off is when the medial tibial plateau rests posterior to the medial femoral condyle. To be an isolated PCL tear, no anterior cruciate or collateral ligament injury is noted. Further, posterior lateral rotatory instability (dial test) at 30° must be symmetric. Many so-called grade III PCL injuries have concomitant capsular and posterolateral corner involvement.

A standard radiographic knee series, including flexion weight-bearing PA view, lateral and sunrise views, and a magnetic resonance imaging scan were obtained on all subjects. KT-1000 (KT-1000, MEDmetric; San Diego, California) instrumented knee testing was also performed to assess the degree of laxity of both knees for comparison.

PCL-Reconstructed Subjects

Ten men with unilateral PCL injury, who underwent PCL reconstruction by the senior author, were evaluated. These subjects ranged in age from 23 to 54 yrs (average: 31 yrs). Five left knees and 5 right knees were involved. Seven patients had an isolated PCL rupture and no other knee disorder. One patient had concomitant medial tibiofemoral chondrosis, 1 had lateral patellar and tibiofemoral chondrosis (this patient underwent an open lateral meniscal transplantation), and another had tricompartmental arthrosis. The time from reconstruction to proprioceptive testing ranged from 6 to 44 mo (average: 27 mo). Patients were examined clinically and with the knee ligament arthrometer (KT-1000, MEDmetric; San Diego, California).

All subjects were tested on the proprioceptive testing device developed at the University of Pittsburgh (modeled after that of Barrack) (see Figure 1). Subjects with isolated PCL-deficient knees were tested for threshold to detect passive motion (kinesthesia) and the ability to passively reproduce passive positioning (joint position sense). Based on the results of this study, those patients who underwent PCL reconstruction had proprioception kinesthesia testing by studying the threshold to detect passive motion only.

A proprioceptive testing device (PTD) was used to measure kinesthesia as the threshold to detection of passive movement (TTDPM) and joint position sense by the ability to passively reproduce passive joint positioning (RPP). This device has been used previously to assess proprioceptive awareness (38). The PTD rotates the knee into flexion and extension through the axis of the joint. A rotational transducer interfaced with a digital microprocessor counter provided the angular displacement values directly. A pneumatic compression boot was placed on each foot to reduce cutaneous input. One pneumatic boot was attached to the moving bar of the PTD, the other to a stationary bar. Patients were tested in the prone position to reduce the likelihood of cutaneous input of their upper calf hitting the testing bench in higher degrees of flexion, as would occur if they were seated upright.

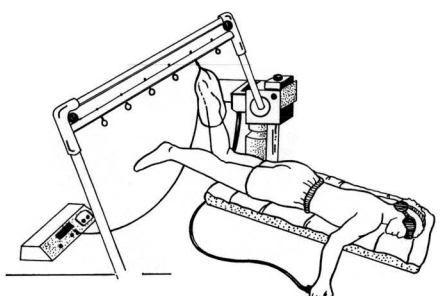


Figure **Proprioception testing device for PCL proprioception.**

The knee was tested moving into flexion and extension from two starting positions. These positions were 45° and 110° of knee flexion. The midrange of knee motion (45°) was chosen as a starting position because there, the capsule, ACL, and PCL are relatively relaxed, and, thus, the poorest proprioceptive sensory results should be noted. No difference should exist, therefore, between the injured and uninjured knees. Knee flexion of 110° was chosen, since tension on the PCL should play a role in proprioception, and thus some difference in afferent input might be identified. This is based on the findings of Wascher and coworkers, who showed a low to intermediate resultant force in the substance of the PCL in 45° flexion, and significantly greater resultant force with increasing knee flexion (60).

Two familiarity trial tests were performed prior to the subjects being blindfolded and having a headset placed over the ears to negate visual and auditory cues. Testing was performed in a single session with test order of injured and uninjured knee, starting position, and direction of movement being randomized and counterbalanced. The PTD tester was blinded as to the normal and PCL injured knee. Instrument reliability was established previously as intraclass correlations (ICC) were calculated using a fixed model and ranged from ICC = .87 to ICC = .92.

Threshold to detection of passive motion: TTDPM assessment was started with the motor and shaft of the PTD disengaged. Subjects were blindfolded and had earphones placed over their ears. The subject gave a thumbs-up to indicate readiness to perform the test. At a random point during the subsequent 20 s, knee movement was engaged by the tester. The subject disengaged the PTD by pressing a handheld switch upon perception of sensation of movement at the knee. The PTD rotated the knee at a constant angular velocity of 0.5°/s. This slow speed was chosen to minimize contribution from muscle receptors while maximally stimulating slow adapting joint mechanoreceptors. Three trials from a starting position of 45° and 110° knee flexion moving into both flexion and extension were performed. Both the injured and uninjured knees were tested. Mean TTDPM values were calculated for the 4 test conditions.

Reproduction of passive positioning: The subjects were blindfolded but permitted to communicate with the PTD tester during RPP testing. As with the TTDPM testing, 45° and 110° knee flexion were used as starting positions (reference angle). After confirmation of the subject's readiness, the knee was moved passively 10° into further flexion or extension (presented angle) by the tester. The angles were presented at variable velocities in order to reduce any time-associated cues. The limb was held in the presented angle position for 10 s and the subject was asked to concentrate on this position. The limb was then returned passively to the reference angle by the examiner. The subject was then instructed to manipulate the on/off switch to reproduce the previously presented angle at an angular velocity of 0.5°/s. This was recorded and repeated for each of the 3 starting positions moving into flexion and extension. The difference between the presented angle and the angle that was repositioned by the subject was calculated as the error of reproduction. The mean of 3 trials was calculated for the 4 test conditions.

PCL-injured and uninjured knee mean differences were analyzed using a paired t test for both TTDPM and RPP testing. A p value of .05 was considered to indicate a significant difference. Pearson product moment correlation coefficients were established between all dependent variables.

Results for PCL-Injured Subjects

Eighteen subjects met the criteria for inclusion—isolated PCL injury without contralateral knee injury, surgery, or other concomitant knee injury. These 13 males and 5 females averaged 32 yrs of age. The patients averaged 29 mo from time of injury to proprioception testing. There were 10 right knees injured and 8 left knees. All individuals except 3 (including the one who underwent PCL reconstruction 1 mo after the injury) underwent physical therapy stressing quadriceps rehabilitation.

On physical examination, 15 patients (83%) had a grade II posterior drawer test on physical examination, while the remaining 3 patients (17%) had a grade III posterior drawer test. Two subjects had minimal laxity of the medial collateral ligament, while there was no other ligamentous injury (ACL, MCL, LCL, rotatory instability) identified on physical examination of any of the subjects. Nine patients (50%) returned to their previous level of sporting activity, 5 participate in sports at a lower level, and 4 do not participate in sports at all (2 did not participate in sports prior to injury).

KT-1000 instrumented testing revealed an average corrected posterior displacement of 7.5 mm (range: 2.5–12.5 mm), manual maximum side-to-side difference at 90°, and 3.1 mm manual maximum side-to-side difference at 20° of knee flexion. Plain radiographs showed no degenerative changes in any of the 18 subjects. Magnetic resonance imaging of all 18 subjects revealed an isolated PCL tear with no meniscal or chondral injuries.

For TTDPM starting at 45°, the PCL-injured knee averaged 1.5° ± 0.2°, while the uninjured knee averaged 1.2° ± 0.1° (p = .051) as the knee was moving into extension. TTDPM at 45° moving into flexion averaged 1.9° ± 0.4° for the involved knee and 1.2° ± 0.2° for the uninvolved knee (p = .022) (see Figure 2). At 110°, TTDPM was not statistically significantly different between the PCL-injured and normal knees moving into flexion and into extension (see Figure 3).

Testing RPP at 110°, the injured knee averaged 2.3° ± 0.4° error from the true test angle, and the uninjured knee averaged 3.1° ± 0.6° error as the test angle was brought into more extension (p = .050). RPP at 110° testing into flexion showed an average error of 2.2° ± 0.3° for the involved knee and 3.0° ± 0.4° for the uninvolved knee (p = .050) (see Figure 5). Testing RPP in 45° was not significantly different between the PCL-deficient and normal knee with the test angle in more flexion or extension (see Figure 4 and Table 1).

A correlation matrix revealed a significant correlation between the time from injury and the ability to passively reproduce a joint angle at 110° flexion moving into extension (r = .687).

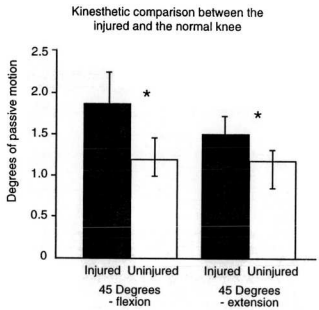


Figure 2 — Mean total magnitude of degrees before sensation was detected. Kinesthesia (angular displacement) for the PCL-injured vs. uninvolved knee from a starting position of 45° of knee flexion, moving into flexion and extension (±*SE*, $p = .02$ for flexion, $p = .05$ for extension).

Results for PCL-Reconstructed Subjects

Ten men met the criteria for inclusion: unilateral PCL injury and PCL reconstruction by the senior author. These subjects ranged in age from 23 to 54 yrs (average: 31 yrs). Five left knees and 5 right knees were involved. Seven patients had an isolated PCL rupture and no other knee disorder. One patient had concomitant medial tibiofemoral chondrosis, 1 had lateral patellar and tibiofemoral chondrosis (this patient underwent an open lateral meniscal transplantation), and another had tricompartmental arthrosis. The time from reconstruction to proprioceptive testing ranged from 6 to 44 mo (average: 27 mo). KT-1000 results identified a corrected posterior displacement of 1.2 ± .8 mm of the normal (unaffected) knee and 4.7 ± 2.6 mm laxity of the reconstructed knee ($p = .0018$). All patients remained active in sports, though four had changed to a less demanding sport, and 6 returned to their preinjury level of activity.

The results for kinesthesia from starting positions of 45° and 110° of flexion revealed no statistically significant difference between the uninvolved and reconstructed knees, as they were passively moved into flexion and extension from both starting angles. From 45° moving into flexion, the uninvolved knee averaged 1.35° prior to the

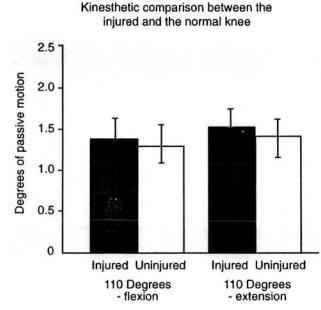


Figure 3 — Mean total magnitude of degrees before sensation was detected. Kinesthesia (angular displacement) for the PCL-injured vs. uninvolved knee from a starting position of 110° of knee flexion, moving into flexion and extension (±*SE*, $p = .29$ for flexion, $p = .12$ for extension).

subject detecting motion, while the reconstructed knee averaged 1.50° ($p = .6$). From 45° moving into extension, the uninvolved knee averaged 1.25° and the reconstructed knee averaged 1.45° ($p = .2$). (See Figure 6.) Testing from the 110° starting position and moving into flexion, the uninvolved knee averaged 1.50° and the reconstructed knee averaged 1.63° prior to the subject detecting motion ($p = .5$). Alternatively, starting at 110° and moving into extension, the uninvolved knee averaged 1.60° and the reconstructed knee averaged 1.95° ($p = .3$) (See Figure 7 and Table 2).

Discussion

Proprioception is considered a specialized variation of the sensory modality of touch and encompasses the sensations of joint movement (kinesthesia) and joint position (joint position sense). Conscious proprioception is essential for proper function in activities of daily living, sports, and occupational tasks. Unconscious proprioception modulates muscle function and initiates reflex stabilization. Much effort has been dedicated to elucidating the mechanical function of knee articular structures and the corresponding mechanical deficits that occur secondary to disruption of these structures. Knee articular structures may also have a significant

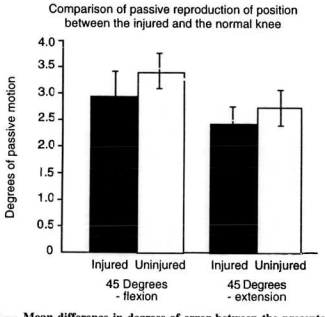
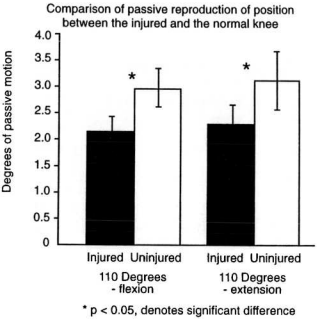


Figure 4 — Mean difference in degrees of error between the presented angle and patient's positioning. Joint position sense (angular displacement) for the PCL-injured vs. uninvolved knee from a starting position (reference angle) of 45° of knee flexion, moving into flexion and extension ($\pm SE$, $p = .16$ for flexion, $p = .22$ for extension).



* $p < 0.05$, denotes significant difference

Figure 5 — Mean difference in degrees of error between the presented angle and patient's positioning. Joint position sense (angular displacement) for the PCL-injured vs. uninvolved knee from a starting position (reference angle) of 110° of knee flexion, moving into flexion and extension ($\pm SE$, $p = .05$ for flexion, $p = .05$ for extension).

sensory function that plays a role in dynamic joint stability, acute and chronic injury, pathologic wearing, and rehabilitation training.

Extrinsic innervation of joints follows Hilton's law (61), which states that joints are innervated by articular branches of the nerves supplying the muscles that cross that joint. The afferent innervation of joints is based on peripheral receptors located in articular, muscular, and cutaneous structures. Articular receptors include nociceptive free nerve endings and proprioceptive mechanoreceptors. Ruffini endings, Pacinian corpuscles, and Golgi tendon organs are mechanoreceptors that have been histomorphologically identified in the ACL (28, 34–36, 47, 50, 51), PCL (32, 51), meniscus (40, 44, 45, 62), lateral collateral ligament (18), and infrapatellar fat pad (37).

There is considerable debate over the relative contribution of muscle receptors versus joint receptors to proprioception, with traditional views emphasizing muscle receptors (11, 16, 24–26). Recent work suggests that muscle receptors and joint receptors are probably complementary components of an intricate afferent system in which each receptor modifies the function of the other (6, 21, 27).

Functionally, kinesthesia is assessed by measuring threshold to detection of passive motion (TTDPM), and joint position sense is assessed by measuring reproduction of passive positioning (RPP). In patients with unilateral joint involvement,

Table 1 Summary of Results for Proprioception Testing in PCL-Injured Subjects

Test	Injured knee	Uninjured knee	*p* value
TTDPM 45°: ext	1.51° ± 0.19°	1.19° ± 0.10°	.05*
TTDPM 45°: flex	1.87° ± 0.35°	1.21° ± 0.22°	.02*
RPP 45°: ext	2.43° ± 0.33°	2.75° ± 0.33°	.22
RPP 45°: flex	2.97° ± 0.47°	3.42° ± 0.30°	.16
TTDPM 110°: ext	1.54° ± 0.22°	1.43° ± 0.18°	.12
TTDPM 110°: flex	1.38° ± 0.24°	1.29° ± 0.25°	.29
RPP 110°: ext	2.28° ± 0.37°	3.11° ± 0.56°	.05*
RPP 110°: flex	2.15° ± 0.28°	2.96° ± 0.36°	.05*

Proprioception results are for kinesthesia testing, which is measured as the angular displacement, in degrees, prior to sensing joint motion, and joint position sense, the angular displacement error between the true angle to which the knee was bent and the angle to which the subject moved the knee, measured in degrees. The starting knee flexion angle was 45° or 110°. (TTDPM = threshold to detect passive motion, RPP = ability to reproduce passive positioning, ext = moving into extension, flex = moving into flexion.)

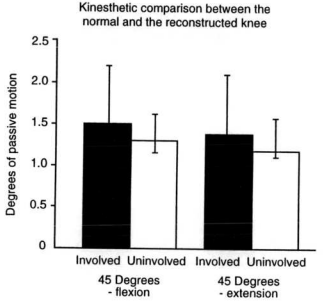


Figure 6 — Mean total magnitude of degrees before sensation was detected. Kinesthesia (angular displacement) for the PCL-reconstructed vs. uninvolved knee from a starting position of 45° of knee flexion, moving into flexion and extension ($\pm SE$, $p = .6$ for flexion, $p = .2$ for extension).

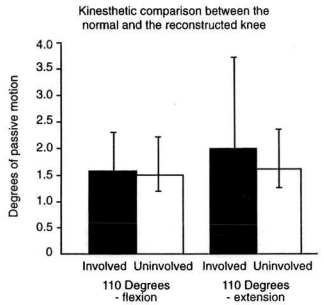


Figure 7 — Mean total magnitude of degrees before sensation was detected. Kinesthesia (angular displacement) for the PCL-reconstructed vs. uninvolved knee from a starting position of 110° of knee flexion, moving into flexion and extension ($\pm SE$, $p = .5$ for flexion, $p = .3$ for extension).

the contralateral knee serves as an internal control, and uninjured knees in a normative population serve as external controls. Using these measures in the knee, investigators have found proprioceptive deficits with aging (3, 5, 54), arthrosis (3, 5, 55), and ACL disruption (2, 4, 7, 14). These processes damage articular structures containing mechanoreceptors and are thus hypothesized to result in partial deafferentation with resultant proprioceptive deficits. Proprioceptive enhancement was found to occur in ballet dancers (1) and also with the use of an elastic knee sleeve (4, 38), suggesting that training and bracing may have proprioceptive benefits.

The use of the threshold to detect passive motion as a measure of kinesthesia has been established by previous studies. Slow, passive motion was used in this investigation, as this is thought to maximally stimulate slow adapting joint mechanoreceptors while minimally stimulating muscle receptors (2). Although we were primarily focusing on joint receptors in joint injury, muscle receptors are an integral component of a complex afferent system and may also play a role in kinesthetic awareness of slow, passive motion. In addition to reflex pathways, joint mechanoreceptors have been shown to have cortical pathways that account for conscious appreciation of joint movement and position.

As with other information about the knee ligaments, information about PCL proprioception lags behind that of ACL proprioception. We have studied the proprioceptive function of a selective group of human subjects with isolated injuries to the posterior cruciate ligament and those who have undergone PCL reconstruction. Other than studies identifying proprioceptive mechanoreceptors within the substance of the PCL, few studies have attempted to assess the clinical function of these mechanoreceptors within the PCL.

Table 2 Summary of Results for Proprioception Testing in PCL-Reconstructed Subjects

Test	Reconstructed knee	Uninjured knee	*p* value
TTDPM 45°: ext	1.45° ± 0.7°	1.25° ± 0.4°	.2
TTDPM 45°: flex	1.5° ± 0.7°	1.35° ± 0.3°	.6
TTDPM 110°: ext	1.95° ± 1.7°	1.62° ± 0.7°	.3
TTDPM 110°: flex	1.63° ± 0.4°	1.5° ± 0.4°	.5

Proprioception results are for kinesthesia testing, measured as the angular displacement, in degrees, prior to sensing joint motion. The starting knee flexion angle was 45° or 110°. (TTDPM = threshold to detect passive motion, ext = moving into extension, flex = moving into flexion.)

One previous study found better joint position sense in patients undergoing total knee arthroplasty using a PCL-retaining prosthesis as compared with a PCL-sacrificing implant (59). Joint position sense was measured by having the subjects move a handheld knee model to replicate the perceived amount of passively placed knee flexion. Twenty-five subjects tested underwent a total knee replacement with a PCL-retaining prosthesis more than 1 year prior to testing and were compared with 9 age-matched controls and 30 patients with PCL-sacrificing knee arthroplasties (59). However, Tibone and coworkers, in the only other PCL proprioception-related study in the English literature that we have found, reported no electromyographic differences between PCL-deficient (some with posterolateral corner injury) and PCL-reconstructed knees (using medial head of the gastrocnemius) during functional tasks (57). Both groups had abnormal findings during gait (57). It may be that those with PCL reconstruction, who were quite symptomatic preoperatively, may have had greater proprioceptive deficits. Thus, the discrepancy as compared with our data may be due to many possible factors such as nonanatomic reconstruction (medial head of gastrocnemius using muscles that may affect proprioceptive input versus our intraarticular reconstruction of the anterolateral bundle of the PCL), large preoperative proprioceptive deficits that may be only incompletely restored, or their testing protocol, which is functional, requiring input from muscle and other fibers as compared with our slow moving, passive model.

One published study more similar to ours studied the threshold to detect passive positioning in 8 patients with isolated PCL-deficient knees (12). Their 8 patients averaged 34 yrs of age, 7 were men, and the average time from injury to testing was 3 years (range: 8 mo to 6 yrs). These patients were tested for TTDPM in the sitting position with their knee moved at 0.5°/s into flexion or extension from 37°. These authors found statistically significantly reduced ability to detect passive motion in the PCL-injured knee as compared with the normal, contralateral knee (12). We also found statistically significant differences in the TTDPM at a similar range (45° moving into both flexion and extension), though we did not find the reduced TTDPM at 110° of knee flexion. Clark and coworkers did not study this greater degree of extension, nor did they study the ability to passively reproduce passive positioning.

Those with isolated PCL disruption in our study are, for the most part, examples of the best clinical case scenario. These are subjects who have been treated nonoperatively for isolated grade II and III posterior laxity of the knee. Subjects with more significant injury are more likely to undergo early knee ligament reconstruction. Therefore, individuals who undergo early ligament reconstruction may be expected to exhibit more significant proprioceptive differences. These subjects, by nature of not already having undergone a reconstructive procedure, are people who mostly have adapted or compensated to their PCL-deficient state. They function well enough for activities of daily living, and in many cases, sports.

Proprioceptive deficits have been identified in the ACL-deficient knee (2, 38). These proprioceptive losses are reduced more significantly at 15° than at 45° flexion in the ACL-deficient knee. This is expected since the ACL has more force at 15° flexion and thus more input in functioning mechanoreceptors. Further, in a study of ACL-reconstructed patients, Barrett (4) showed that patient satisfaction and function appeared to correlate with proprioceptive function rather than with clinical examination and knee scores.

We have shown that isolated PCL deficiency in the human knee does result in reduced kinesthesia, as tested by the threshold to detect passive positioning and enhanced ability to reproduce passive positioning. Thus, the proprioceptive mechanoreceptors within the PCL do appear to have some function. Proprioceptive deficits in studies of patients with ACL disruption reveal greater proprioceptive deficits, both in magnitude and over a greater range of motion, as compared with the findings presented here for PCL deficiency. The proprioceptive deficits in kinesthesia identified in PCL-deficient knees are eliminated (or reduced to a nonsignificant level) by PCL reconstruction.

It has been argued that proprioception may play a protective role in acute injury through reflex muscular splinting (38). The protective reflex arc initiated by mechanoreceptors and muscle spindle receptors occurs much more quickly than the reflex arc initiated by nociceptors (70–100 m/s vs. 1 m/s). Thus, proprioception may play a more significant role than pain sensation in preventing injury in the acute setting. More importantly, proprioceptive deficits may play a more significant role in the etiology of chronic injuries and reinjury. Initial knee injury results in partial deafferentation and sensory deficits that can predispose to further injury (34). Proprioceptive deficits may also contribute to the etiology of degenerative joint disease through pathological wearing of a joint with poor sensation. It is unclear whether the proprioceptive deficits that accompany degenerative joint disease are a result of the underlying pathological process or contribute to the etiology of the pathological process. It may be surmised then, that the apparent loss of proprioception over a greater range of motion in the ACL-deficient knee helps explain why the so-called isolated PCL-deficient knee has a relatively more "benign course." It has yet to be proven that PCL deficiency leads to degenerative arthritis, and yet to be reported is whether PCL reconstruction can prevent or delay the onset of degenerative change within the operative knee.

We do not attempt to explain why TTDPM is reduced only at 45° and why RPP appears to be better at 110°. There are many possible explanations, including the altered kinematics of the PCL-deficient knee, variable coordinated input between the ACL and PCL at varying degrees of flexion (the ACL may have significant resultant force when flexed) (60), or even that the PCL may still contribute proprioceptive information as it may heal in a lengthened position. Further, the effect of physical therapy following injury may enhance proprioceptive function of the remaining mechanoreceptors, or it may be that kinesthesia and joint position sense are mediated through different pathways.

Looking at the threshold to detect passive motion, PCL reconstruction appears to provide a kinesthetic pattern similar to that of the contralateral, normal

knee. The deficit seen in nonoperated knees with PCL tears does not exist in knees following PCL reconstruction. This is in light of the fact that 3 patients who underwent PCL reconstruction and proprioceptive testing had some degree of degenerative joint disease. Knees with degenerative joint disease have previously been shown to have worse proprioception than the contralateral knee and age-matched controls. Thus, it is likely that PCL reconstruction did significantly improve kinesthesia at 45° of knee flexion (at 110°, no significant difference was observed between the involved and noninvolved knee in either study).

The most plausible explanation for the restoration of proprioception following reconstructive surgery appears to be related to the ligamentous and capsular tension of the joint (8, 39). Excessive laxity of those structures that contain proprioceptive mechanoreceptors results in diminished mechanical deformation of joint structures and reduces the afferent signal that mediates proprioception.

Regardless of the many possible explanations, the fact remains that mechanoreceptors within the PCL do have proprioceptive function and may play a role in the clinical function of patients with PCL injuries, and proprioception may be normalized (or deficits reduced) by PCL reconstruction. Future research in this area will help delineate the exact clinical function of proprioceptive mechanoreceptors within the posterior cruciate ligament.

References

1. Barrack, R.L., H.B. Skinner, M.E. Brunet, and S.D. Cook. Joint kinesthesia in the highly trained knee. *J. Sports Med. Phys. Fitness* 24:18-20, 1983.
2. Barrack, R.L., H.B. Skinner, and S.L. Buckley. Joint proprioception in the anterior cruciate ligament deficient knee. *Am. J. Sports Med.* 17:1-6, 1989.
3. Barrack. R.L., H.B. Skinner, S.D. Cook, and R.J. Haddad, Jr. Effect of articular disease and total knee arthroplasty on knee joint-position sense. *J. Neurophysiol.* 50:684-687, 1983.
4. Barrett, D.S. Proprioception and function after anterior cruciate reconstruction. *J. Bone Joint Surg.* 73B:833-837, 1991.
5. Barrett, D.S., A.G. Cobb, and G. Bentley. Joint proprioception in normal, osteoarthritic and replaced knee. *J. Bone Joint Surg.* 73B:53-56, 1991.
6. Baxendale, R.A., W.R. Ferrell, and L. Wood. Responses of quadriceps motor units to mechanical stimulation of knee joint receptors in the decerebrate cat. *Brain Res.* 453:150-156, 1988.
7. Beard, D.J., P.J. Kyberd, C.M. Fergusson, and C.A.F. Dodd. Proprioception after rupture of the anterior cruciate ligament. An objective indication of the need for surgery? *J. Bone Joint Surg.* 75B:311-315, 1993.
8. Blaiser, R., J. Carpenter, and L. Huston. Shoulder proprioception. Effect of joint laxity, joint position, and direction of motion. *Orthop. Rev.* 23:45-50, 1994.
9. Chick, R.R., and R.W. Jackson. Tears of the anterior cruciate ligament in young athletes. *J. Bone Joint Surg.* 60A:970-973, 1978.
10. Clancy, W.G., Jr., K.D. Shelbourne, G.B. Zoellner, J.S. Keene, B. Reider, and T.D. Rosenberg. Treatment of knee joint instability secondary to rupture of the posterior cruciate ligament. *J. Bone Joint Surg.* 65A:310-322, 1983.
11. Clark, F.J., and P.R. Burgess. Slowly adapting receptors in cat knee joint: Can they signal joint angle? *J. Neurophysiol.* 38:1448-1463, 1975.
12. Clark, P., P.B. MacDonald, and K. Sutherland. Analysis of proprioception in the posterior cruciate ligament-deficient knee. *Knee Surg. Sports Traumatol. Arthroscopy* 4:225-227, 1996.
13. Co, F.H., H.B. Skinner, and W.D. Cannon. Proprioception of the knee following ACL reconstruction and its relation to heelstrike impulse. *Transactions of the 37th Annual Meeting of the Orthopaedic Research Society*, Anaheim, CA, 1991, p. 603.
14. Corrigan, J.P., W.F. Cashman, and M.P. Brady. Proprioception in the cruciate deficient knee. *J. Bone Joint Surg.* 74B:247-250, 1992.
15. Cross, M.J., and J.F. Powell. Long term follow-up of posterior cruciate ligament rupture. A study of 116 cases. *Am. J. Sports Med.* 12:292-297, 1984.
16. Cross, M.M., and D.I. McCloskey. Position sense following surgical removal of joints in man. *Brain* 55:443-445, 1973.
17. Dandy, D.J., and R.J. Pusey. The long-term results of unrepaired tears of the posterior cruciate ligament. *J. Bone Joint Surg.* 64B:92-94, 1982.
18. DeAvila, G.A., B.L. O'Connor, D.M. Visco, and T.D. Sisk. The mechanoreceptor innervation of the human fibular collateral ligament. *J. Anatomy* 162:1-7, 1989.
19. Dejour, H., G. Walch, J. Peyrot, and P. Eberhard. The natural history of rupture of the posterior cruciate ligament. *French J. Orthopaedic Surg.* 2:112-120, 1988.
20. DuToit, G.T. Knee joint cruciate ligament substitution. The Lindemann (Heidelberg) operation. *S. African J. Surg.* 5:25-30, 1967.
21. Ferrell, W.R. The response of slowly adapting mechanoreceptors in the cat knee joint to tetanic contraction of hind limb muscles. *Q. J. Exp. Physiol.* 70:337-345, 1985.
22. Fowler, P.J., and S.S. Messieh. Isolated posterior cruciate ligament injuries in athletes. *Am. J. Sports Med.* 15:553-557, 1987.
23. Giove, T.P., S.J. Miller III, B.E. Kent, T.L. Sanford, and J.G. Garrick. Nonoperative treatment of the torn anterior cruciate ligament. *J. Bone Joint Surg.* 65A:184-192, 1983.
24. Goodwin, G.M., D.I. McCloskey, and P.B. Matthews. The persistence of appreciable kinesthesia after paralyzing joint afferents but preserving muscle afferents. *Brain Res.* 37:326-329, 1972.
25. Goodwin, G.M., D.I. McCloskey, and P.B. Matthews. The contribution of muscle afferents to kinesthesia shown by vibration induced illusions of movement and by the effects of paralyzing joint afferents. *Brain* 95:705-748, 1972.
26. Grigg, P. Mechanical factors influencing response of joint afferent neurons from cat knee. *J. Neurophysiol.* 38:1473-1484, 1975.
27. Grigg, P. Response of joint afferent neurons in cat medial articular nerve to active and passive movements of the knee. *Brain Res.* 118:482-485, 1976.
28. Halata, Z., and J. Haus. The ultrastructure of sensory nerve endings in human anterior cruciate ligament. *Anat. Embryol.* 179:415-421, 1989.

29. Hawkins, R.J., G.W. Misamore, and T.R. Merritt. Followup of the acute nonoperated isolated anterior cruciate ligament tear. *Am. J. Sports Med.* 14:205-210, 1986.
30. Ihara, H., and A. Nakayama. Dynamic joint control training for knee ligament injuries. *Am. J. Sports Med.* 14:309-315, 1986.
31. Insall, J., D.M. Joseph, P. Aglietti, and R.D. Campbell. Bone-block iliotibial-band transfer for anterior cruciate insufficiency. *J. Bone Joint Surg.* 63A:560-569, 1981.
32. Katonis, P.G., A.P. Assimakopoulos, M.V. Agapitos, and E.I. Exarchou. Mechanoreceptors in the posterior cruciate ligament. Histologic study on cadaver knees. *Acta Orthop. Scand.* 62:276-278, 1991.
33. Keller, P.M., D. Shelbourne, J.R. McCarroll, and A.C. Rettig. Nonoperatively treated isolated posterior cruciate ligament injuries. *Am. J. Sports Med.* 21:132-136.
34. Kennedy, J.C., I.J. Alexander, and K.C. Hayes. Nerve supply of the human knee and its functional importance. *Am. J. Sports Med.* 10:329-335, 1982.
35. Kennedy, J.C., H.W. Weinberg, and A.S. Wilson. The anatomy and function of the anterior cruciate ligament as determined by clinical and morphological studies. *J. Bone Joint Surg.* 56A:223-235, 1974.
36. Krauspe, R., M. Schmidt, and H.G. Schaible. Sensory innervation of the anterior cruciate ligament. An electrophysiological study of the response of single identified mechanoreceptors in the cat. *J. Bone Joint Surg.* 74A:390-397, 1992.
37. Krenn, V., S. Hofmann, and A. Engel. First description of mechanoreceptors in the corpus adiposum infrapatellare of man. *Acta Anatomica* 137:187-188, 1990.
38. Lephart, S.M., M.S. Kocher, F.H. Fu, P.A. Borsa, and C.D. Harner. Proprioception following ACL reconstruction. *J. Sport Rehabil.* 1:188-196, 1992.
39. Lephart, S.M., J.J.P. Warner, P. Borsa, and F.H. Fu. Proprioception of the shoulder joint in healthy, unstable, and surgically repaired shoulders. *J. Shoulder Elbow Surg.* 3:371-380, 1994.
40. MacKenzie, W.G., S.S. Shim, B. Day, and G. Leung. The blood and nerve supply of the knee meniscus in man. *Anat. Rec.* 211:115A-116A, 1985.
41. McDaniel, W.J., and T.B. Dameron, Jr. The untreated anterior cruciate ligament rupture. *Clin. Orthop.* 172:158-163, 1980.
42. Noyes, F.R., D.L. Butler, L.E. Paulos, and E.S. Grood. Intra-articular cruciate reconstruction. Part I: Perspectives on graft strength, vascularization, and immediate motion after replacement. *Clin. Orthop.* 172:71-77, 1983.
43. Noyes, F.R., P.A. Mooar, D.A. Matthews, and D.L. Butler. The symptomatic anterior cruciate ligament deficient knee. Part I: The long-term functional disability in athletically active individuals. *J. Bone Joint Surg.* 65A:154-162, 1983b.
44. O'Connor, B.L. The histologic structure of dog knee menisci with comment on its possible significance. *Am. J. Anat.* 147:407-417, 1976.
45. O'Connor, B.L., and J.S. McConnaughey. The structure and innervation of cat knee menisci and their relation to a "sensory hypothesis" of meniscal function. *Am. J. Anat.* 153:431-442, 1978.
46. Parolie, J.M., and J.A. Bergfeld. Long-term results of nonoperative treatment of isolated posterior cruciate ligament injuries in the athlete. *Am. J. Sports Med.* 14:35-38, 1986.
47. Rivard, C.H., L.H. Yahia, S. Rhalmi, and N. Newman. Immunohistochemical demonstration of sensory nerve fibers and endings in human anterior cruciate ligaments. *Transactions of the 39th Annual Meeting of the Orthopaedic Research Society,* San Francisco, 1993, p. 61.
48. Safran, M.R., G.L. Caldwell, Jr., and F.H. Fu. Proprioception considerations in surgery. *J. Sports Rehabil.* 1:105-115, 1994.
49. Satku, K., V.P. Kumar, and S.S. Ngoi. Anterior cruciate ligament injuries. To counsel or to operate? *J. Bone Joint Surg.* 68B:458-461, 1986.
50. Schultz, R.A., D.C. Miller, C.S. Kerr, and L. Micheli. Mechanoreceptors in human cruciate ligaments: A histologic study. *J. Bone Joint Surg.* 66A:1072-1076, 1984.
51. Schutte, M.J., E.J. Dabezies, M.L. Zimny, and L.T. Happel. Neural anatomy of the human anterior cruciate ligament. *J. Bone Joint Surg.* 69A:243-247, 1987.
52. Shelbourne, K.D., and D.V. Patel. The natural history of acute isolated non-operatively treated posterior cruciate ligament injuries of the knee: A prospective study. *Transactions of the 64th Annual Meeting of the American Academy of Orthopaedic Surgeons,* 1997, pp. 77-78.
53. Sherman, M.F., R.F. Warren, J.L. Marshall, and G.J. Savatsky. A clinical and radiographical analysis of 127 anterior cruciate insufficient knees. *Clin. Orthop.* 227:229-237, 1988.
54. Skinner, H.B., R.L. Barrack, and S.D. Cook. Age-related decline in proprioception. *Clin. Orthop.* 184:208-211, 1984.
55. Skinner, H.B., R.L. Barrack, S.D. Cook, and R.J. Haddad, Jr. Joint position sense in total knee arthroplasty. *J. Orthop. Res.* 1:276-283, 1984.
56. Sommerlath, K., J. Lysholm, and J. Gillquist. The long-term course after treatment of acute anterior cruciate ligament ruptures. *Am. J. Sports Med.* 19:156-162, 1991.
57. Tibone, J.E., T.J. Antich, J. Perry, and D. Moynes. Functional analysis of untreated and reconstructed posterior cruciate ligament injuries. *Am. J. Sports Med.* 16:217-223, 1988.
58. Torg, J.S., T.M. Barton, H. Pavlov, and R. Stine. Natural history of the posterior cruciate ligament-deficient knee. *Clin. Orthop.* 246:208-216, 1989.
59. Warren, P.J., T.K. Olanlokun, A.G. Cobb, and G. Bentley. Proprioception after knee arthroplasty. The influence of prosthetic design. *Clin. Orthop.* 297:182-187, 1993.
60. Wascher, D.C., K.L. Markolf, M.S. Shapiro, and G.A.M. Finerman. Direct in vitro measurement of forces in the cruciate ligaments. Part I: The effects of multiplane loading in the intact knee. *J. Bone Joint Surg.* 75A:377-386, 1993.
61. Wyke, B.D. The neurology of joints. *Ann. Royal Coll. Surg.* 41:25-49, 1967.
62. Zimny, M.L., D.J. Albright, and E. Dabezies. Mechanoreceptors in the human medial meniscus. *Acta Anatomica* 133:35-40, 1988.