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Proprioception in the posterior cruciate ligament deficient knee

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Abstract This study was undertaken to evaluate knee proprioception in patients with isolated unilateral posterior cruciate ligament (PCL) injuries. Eighteen subjects with isolated PCL tears were studied 1–234 months after injury. The threshold to detect passive motion (TTDPM) was used to evaluate kinesthesia and the ability to passively reproduce passive positioning (RPP) to test joint position sense. Two starting positions were tested in all knees: 45° (middle range) and 110° (end range) to evaluate knee proprioception when the PCL is under different amounts of tension. TTDPM and RPP were tested as the knee moved into flexion and extension from both starting positions. A statistically significant reduction in TTDPM was identified in PCL-injured knees tested from the 45° starting position, moving into flexion and extension. RPP was statistically better in the PCL-deficient knee as tested from 110° moving into flexion and extension. No difference was identified in the TTDPM starting at 110° or in RPP with the presented angle at 45° moving into flexion or extension. These subtle but statistically significant findings suggest that proprioceptive mechanoreceptors may play a clinical role in PCL-intact and PCL-deficient patients. Further, it appears that kinesthesia and joint position sense may function through different mechanisms.

Key words Posterior cruciate ligament · Proprioception · Ligament · Knee

Introduction

The treatment of isolated posterior cruciate ligament (PCL) injuries is controversial. This is partly because the natural history of the isolated PCL injured knee is unclear. It has been noted that many patients function well, even in high level sports, with a torn PCL. This is in direct contradistinction with the ACL injured knee. The reason for this difference is not clear, as the PCL is larger and stronger than 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 [3, 7, 41]. DuToit [19], Insall et al. [34], and others [42, 47, 53] have all advocated certain reconstructive techniques due in part to increased afferent preservation. A proprioceptive deficit has been demonstrated following ACL disruption [3, 4, 7, 13]; however, no literature exists reporting proprioception in the PCL deficient 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 [9, 15, 16, 18, 22, 39, 51, 63]. It is well known that in the anterior cruciate ligament (ACL) deficient knee instability and reinjury can lead to arthritis over time [8, 23, 31, 46, 48, 54, 57, 61]. Further, authors suggest that function in the ACL-deficient and ACL-reconstructed knee is more reliably predicted by proprioceptive ability than physical examination or knee test scores [4, 7, 13, 33]. Several stud-

ies have shown that proprioceptive deficits that exist in ACL-deficient knees can be partially restored by surgical reconstruction [3, 4, 12]. Reduced proprioception has been implicated in the development of knee arthritis [5, 59].

Many authors note that patients with "isolated" PCL deficiency initially function well while progression to degenerative arthritis over time is less well defined [9, 18, 51]. To date no studies have been performed to determine the potential proprioceptive deficits in the PCL deficient knee. With this information in mind, it was the purpose of this study to characterize the proprioceptive changes in the isolated PCL-deficient knee.

Materials and methods

Eighteen subjects met the criteria for inclusion: isolated PCL injury without contralateral knee injury, surgery or other concomitant knee injury. The 13 men and 5 women averaged 32 years of age (range 19–51). These patients averaged 29 months from time of injury to proprioception testing (range 1–234). There were 10 right knees injured and 8 left knees. The mechanism of injury was hyperextension in 7, flexion/hyperflexion in 8, and unknown in 3. Many of the patients presented to Orthopaedic Sports Medicine Service after initial injury and followed with conservative management. Most of these patients were asked to return for follow-up and testing even though they remained asymptomatic at latest evaluation. All subjects underwent a complete history and thorough knee examination by one of the authors, an orthopedic surgeon specializing in sports injuries. The bilateral knee examination included assessment of knee range of motion, lower extremity alignment, presence of effusion, patellar irritability, patellar motion, anteroposterior and varus-valgus stability, and meniscal signs. All but three individuals (including the one who underwent PCL reconstruction 1 month after the injury) underwent physical therapy stressing quadriceps rehabilitation.

In the normal knee the medial tibial plateau normally is 10 mm anterior to the medial femoral condyle with the knee in 90° flexion. Posterior drawer testing for PCL insufficiency is then graded as follows: in grade I injury there is asymmetry side to side, but the medial tibial plateau remains anterior to the medial femoral condyle; in grade II injury the posterior drawer at 90° pushes the tibial plateau to the level of the medial femoral condyle; in grade III injury the medial tibial plateau can be pushed posterior to the medial femoral condyle. The posterior sag is graded similarly: in grade I 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 grade II the tibial plateau is at the level of the medial femoral condyle; in grade III the medial tibial plateau rests posterior to the medial femoral condyle. By definition an isolated PCL tear precludes inclusion of patients with injury to the anterior cruciate or collateral ligament in this study. Furthermore, posterior lateral rotatory instability, "dial test," at 30° must be symmetric. It is important to note for this study that we tested patients with truly isolated PCL tears. In the past many so-called isolated grade III PCL injuries have been included in studies as isolated injuries, although concomitant capsular and posterolateral corner involvement likely existed. In this study these patients were excluded.

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

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 joint positioning (RPP; Fig. 1). This device has been used previously to assess proprioceptive awareness and is painless to the subjects [44]. 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 and the other to a stationary bar. The potential for input from the anterior thigh cutaneous receptors exists since the anterior thigh rests on the stable (not moving) frame of the testing apparatus. The input would likely need to be through the stretching of the distal thigh skin as the knee is moved, although this is felt to be negligible since the distal one-half to one-third of the thigh was not touching the apparatus.

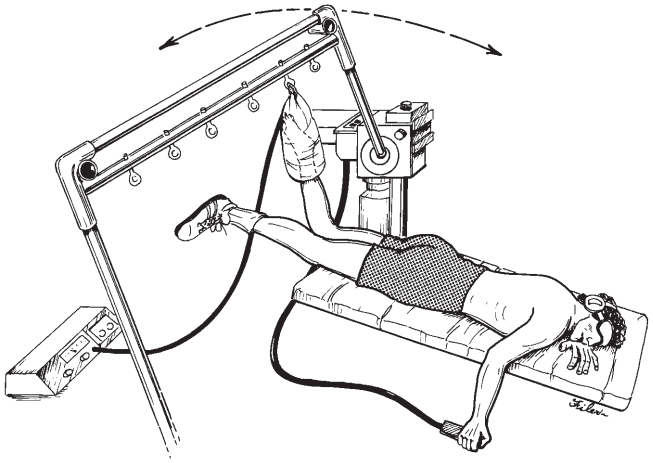


Fig. 1 PTD utilized in this study. The PTD rotates the knee into flexion and extension through the axis of the joint. A rotational transducer interfaced with a digital microprocessor counter provided the angular displacement values directly. The subject is prone. A pneumatic compression boot is placed 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. The subject is blindfolded to eliminate visual cues. For TTDPM testing, headphones with white noise are used to eliminate auditory cues. The subject shown schematically is holding an on-off switch as utilized for RPP testing to passively reproduce the presented angle

The knee was tested moving into flexion and extension from two starting positions. These positions were 45° and 110° of knee flexion. These starting positions were chosen as the middle range of knee motion (45°) where the capsule, ACL, and PCL are relatively relaxed, and thus the poorest proprioceptive sensory results should be noted. Thus no difference should exist 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.

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 were calculated using a fixed model and ranged from in value from 0.87 to 0.92.

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 thumb-up signal 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 hand held 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. Three trials from a starting position of 45° and 110° knee flexion moving into both flexion and extension were performed. The number of degrees the PTD moved the knee by the time the subject disengaged the motor was recorded as the TTDPM. Both the injured and uninjured knees were tested. Mean TTDPM values were calculated for the four 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 three trials 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 three trials was calculated for the four test conditions.

PCL injured and uninjured knee mean differences were analyzed using a paired *t* test for both TTDPM and RPP testing. Pearson product-moment correlation coefficients were established between all dependent variables.

Results

Although all of the patients complained of mechanical symptoms (locking, giving way) initially, ten (55%) of the patients noted some instability symptoms at follow-up. No subjects complained of locking of the knee. Thirteen (72%) complained of some pain in the knee at follow-up, the majority having anterior knee pain. Four (22%) subjects had instability with activities of daily living, and six had occasional giving-way with sporting activities at the most recent evaluation. Nine (50%) complained of knee pain or instability going up or down stairs, and six (33%) complained of slight recurrent effusions, primarily with a high level of activity. Nine (50%) were able to return to their previous level of activity, including four collegiate/professional athletes.

On physical examination 11 subjects had full flexion and extension of the affected knee. Six subjects had an average 9° loss of flexion (range 5–12°), and one lacked 4° of knee hyperextension. Three subjects had a small effusion, two had trace effusion. Ten subjects (56%) had crepitation of the patellofemoral joint. Twelve (67%) had tenderness of the medial facet of the patella, and six (33%) had tenderness of the lateral facet of the patella (four had tenderness of both the medial and lateral facets). Fifteen (83%) patients had a grade II posterior drawer test on physical examination, while the remaining three (17%) had a grade 3 posterior drawer test. Two subjects had minimal laxity of the medial collateral ligament (MCL), while there was no other ligamentous injury identified on physical examination (ACL, MCL, LCL, rotatory instability) of any of the subjects.

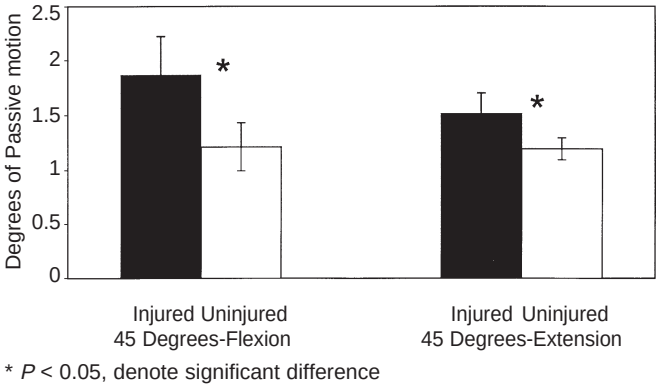


Fig. 2 Kinesthetic sensation as measured by the TTDPM of the PCL-injured and normal contralateral knees at the 45° starting angle, moving into flexion and extension. Kinesthesia was statistically significantly worse in the PCL-injured knee than in the uninjured knee, both moving into flexion and moving into extension

KT-1000 instrumented testing revealed an average 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.

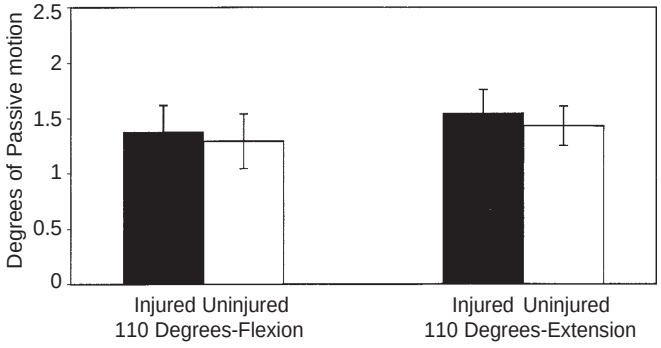
For TTDPM, starting at 45°, the PCL injured knee averaged 1.5 ± 0.2°, while the uninjured knee averaged 1.2 ± 0.1° ($P = 0.051$) as the knee was moving into extension (Fig. 2, Table 1). TTDPM at 45° moving into flexion averaged 1.9 ± 0.4° for the involved knee and 1.2 ± 0.2° for the uninvolved knee ($P = 0.022$; Fig. 2, Table 1). At 110° TTDPM values did not differ statistically significantly between PCL-injured and normal knees moving into flexion and into extension (Fig. 3, Table 1).

Testing RPP at 110°, the injured knee averaged 2.3 ± 0.4° error from the true test angle and the uninjured knee average 3.1 ± 0.6° error as the test angle was brought into more extension ($P = 0.050$; Fig. 4, Table 1). 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

Table 1 Data for each testing scenario (*45°*, *110°* starting angle, *extension* test knee moving into extension, *flexion* test knee moving into flexion)

Test	Injured knee	Uninjured knee	*P*
TTDPM			
45° extension	1.51 ± 0.19°	1.19 ± 0.10°	0.051
45° flexion	1.87 ± 0.35°	1.21 ± 0.22°	0.022
RPP			
45° extension	2.43 ± 0.33°	2.75 ± 0.33°	0.224
45° flexion	2.97 ± 0.47°	3.42 ± 0.30°	0.155
TTDPM			
110° extension	1.54 ± 0.22°	1.43 ± 0.18°	0.119
110° flexion	1.38 ± 0.24°	1.29 ± 0.25°	0.290
RPP			
110° extension	2.28 ± 0.37°	3.11 ± 0.56°	0.050
110° flexion	2.15 ± 0.28°	2.96 ± 0.36°	0.050

TTDPM values represent the average number of degrees (with standard error) before the subject sensed the knee moving. RPP values represent the average number of degrees error (with standard error) of the subjects' knee angle from the true presented angle

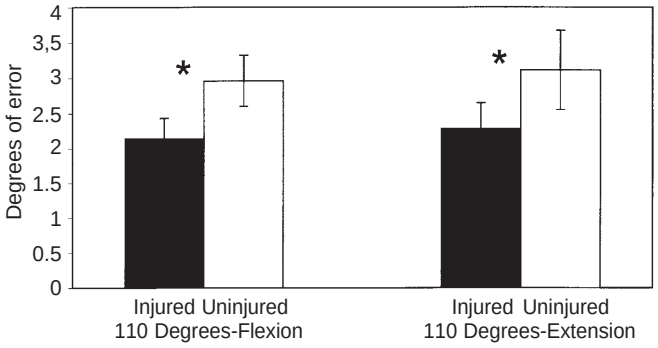


* $P < 0.05$, denote significant difference

Fig. 3 Kinesthetic sensation as measured by the TTDPM of the PCL-injured and normal contralateral knees at the 110° starting angle, moving into flexion and extension. Kinesthesia was not statistically significantly different in the PCL injured knee than in the uninjured knee either moving into flexion or moving into extension

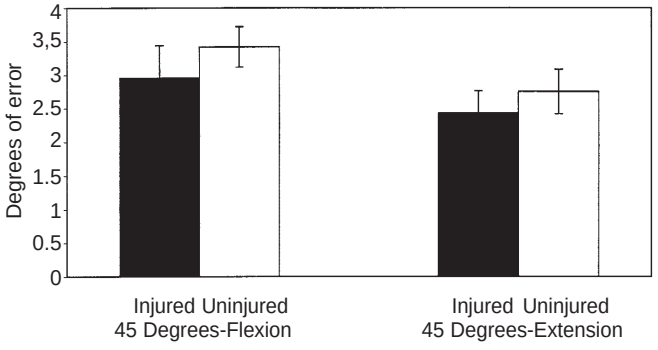
knee ($P = 0.050$; Fig. 4, Table 1). RPP values in 45° did not differ significantly between PCL-deficient and normal knee with the test angle in more flexion or extension (Fig. 5, Table 1).

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 ($r = 0.687$). Thus, the longer time from injury, the better was the subject's RPP. Knee laxity measurements, as quantified by KT-1000 knee ligament arthrometer (MedMetric, San Diego, Calif., USA), were not correlated with proprioceptive measurements.



* $P < 0.05$, denote significant difference

Fig. 4 Joint Position Sense as measured by RPP of the PCL-injured and normal contralateral knees at the 110° starting angle, moving into flexion and extension. Joint position sense was statistically significantly better in the PCL injured knee than in the uninjured knee both moving into flexion and moving into extension



* $P < 0.05$, denote significant difference

Fig. 5 Joint Position Sense as measured by RPP of the PCL-injured and normal contralateral knees at the 45° starting angle, moving into flexion and extension. Joint position sense was not statistically significantly different between the PCL-injured and the contralateral, normal knee, moving into flexion or moving into extension

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

Extrinsic innervation of joints follows Hilton's law [67], 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 [30, 40–42, 52, 55, 56], PCL [38, 56], meniscus [45, 49, 50, 68], lateral collateral ligament [17] and infrapatellar fat pad [43].

Mechanoreceptors transduce some function of mechanical deformation into a frequency modulated neural signal which is transmitted via cortical and reflex pathways. An increased stimulus of deformation is coded by an increased afferent discharge rate or an increased population of activated receptors. Grigg and Hoffman [27, 29] have correlated mechanoreceptor afferent discharge with strain energy density and have calibrated mechanoreceptors as in vivo load cells in the posterior capsule of the feline knee. Receptors demonstrate different adaptive properties based on their response to a continuous stimulus.

Quick adapting (QA) mechanoreceptors, such as the Pacinian corpuscle, decrease their discharge rate to extinction within milliseconds of the onset of a continuous stimulus. Slow-adapting (SA) mechanoreceptors, such as the Ruffini ending and the Golgi tendon organ, continue their discharge in response to a continuous stimulus. QA mechanoreceptors are very sensitive to changes in stimulation and are therefore thought to mediate the sensation of joint motion. Different populations of SA mechanoreceptors are maximally stimulated at specific joint angles, and thus a continuum of SA receptors is thought to mediate the sensation of joint position [10, 32, 36]. In animal models these mechanoreceptors respond to active or passive motion with maximal stimulation occurring at the extremes of knee motion [26–28, 37]. Stimulation of these receptors results in reflex muscle contraction about the joint [6, 20, 35, 60].

The muscle spindle receptor is a complex fusiform, SA receptor found within skeletal muscle. Via afferents and efferent to intrafusal muscle fibers, the muscle spindle receptor can detect and regulate muscle strain over a large range of extrafusal muscle length. There is considerable debate over the relative contribution of muscle receptors versus joint receptors to proprioception, with traditional views emphasizing muscle receptors [10, 14, 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].

Kinesthesia is assessed functionally by measuring TTDPM, and joint position sense by measuring RPP. In patients with unilateral joint involvement 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 [2, 5, 58], arthrosis [2, 5, 59], and ACL disruption [3, 4, 7, 13]. These processes damage articular structures containing mechanoreceptors and are thus hypothesized to result in partial deafferentation with resultant proprioceptive deficits. Proprioceptive enhancement has been found to occur in ballet dancers [1] and also with the use of an elastic knee sleeve [4, 44], suggesting that training and bracing may have proprioceptive benefits.

The use of the TTDPM as a measure of kinesthesia has been established by previous studies. Slow, painless, passive motion was used in this investigation, as this is thought to maximally stimulate slow-adapting joint mechanoreceptors while minimally stimulating muscle receptors [3]. Although we focused primarily 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.

While much research exists about ACL deficiency, including information about proprioception in the ACL deficient knee [3, 4, 7, 12, 13, 42, 44], data regarding PCL injuries are lacking [11, 62, 64]. We have studied the proprioceptive function of a selective group of human subjects with isolated injuries to the PCL. Few other studies identifying proprioceptive mechanoreceptors within the substance of the PCL have attempted to assess the clinical function of these mechanoreceptors within the PCL.

One previous study found better joint position sense in patients undergoing total knee arthroplasty using a PCL retaining prosthesis than with a PCL-sacrificing implant [64]. Joint position sense was measured by the subjects moving a hand held 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 nine age-matched controls and 30 patients with PCL-sacrificing knee arthroplasties [64]. However, Tibone et al. [62], in another PCL proprioception related study, 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 [62]. Both groups had abnormal findings during gait [62]. 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 findings may be due to many possible factors such as nonanatomic reconstruction (medial head of gastrocnemius using muscles that may affect proprioceptive input versus our intra-articular reconstruction of the anterolateral bundle of the PCL), large preoperative proprio-

ceptive deficits that may only be 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 eight patients with isolated PCL deficient knees [11]. Their eight patients averaged 34 years of age, seven were men, and the average time from injury to testing was 3 years (8 months–6 years). These patients were tested for TTDPM at 0.5°/s in the sitting position with their knee moved into flexion *or* extension from 37°. These authors found statistically significantly less ability to detect passive motion in the PCL-injured knee than in the normal, contralateral knee [11]. We also found statistically significant differences in the TTDPM at a similar range (45° moving *both* into flexion and extension), although we did not find the reduced TTDPM at 110° of knee flexion. This greater degree of extension was not studied by Clark et al. [11], nor was RPP.

We studied the proprioceptive function of a selective group of human subjects with isolated injuries to the PCL. The subjects studied are for the most part examples of the clinical best-case scenario. These are subjects who have been treated nonoperatively for isolated grade 2 and 3 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.

Proprioceptive deficits have been identified in the ACL deficient knee [3, 44]. 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, Barrett [4], in a study of ACL-reconstructed patients, found patient satisfaction and function to be correlated 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 RPP. There are many potential reasons for these findings that are beyond the scope of this study and are outlined below, although one potential reason is that the proprioceptive mechanoreceptors within the PCL have some clinical function. Proprioceptive deficits in studies of patients with ACL disruption reveal greater proprioceptive deficits, both in magnitude and over a greater range of motion, than the findings presented here for PCL deficiency.

It has been argued that proprioception may play a protective role in acute injury through reflex muscular splinting [44]. 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 which can predispose to further injury [41]. 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 may help explain why the so-called isolated PCL deficient knee has a relatively more "benign course."

This study does not attempt to explain why TTDPM is reduced only at 45°, or 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) [63], and even that the PCL still contributes proprioceptive information as it may heal in a lengthened position. Further still, the effect of physical therapy following injury may enhance proprioceptive function of the remaining mechanoreceptors. Further, it may be that kinesthesia and joint position sense are mediated through different pathways. We hypothesize that the altered proprioceptive input in the PCL-deficient knee may be due to proprioceptive function of the mechanoreceptors within the PCL, and that they may play a role in the clinical function of patients with PCL injuries.

Areas of limitations and further study include the relative importance of control design, effect of gender, length of follow-up, and effect of physical therapy. We have chosen to use the contralateral knee as the control since proprioception has been shown to be affected by training, age, injury, and other factors. Some investigators have found lower proprioceptive capabilities in the noninjured knees of ACL-deficient patients than in age- and sex-matched controls. As understanding of proprioception in the human knee, and specifically cruciate ligaments, is in its infancy, the relative strengths and limitations of both methodologies is unclear.

The number of patients in our study did not allow statistically or clinically significant evaluation of possible gender differences in proprioception following isolated PCL disruption, nor for a more meaningful understanding of the influence of time on proprioception following isolated PCL tears, although these are two of many issues that needs to be evaluated further in the future. Furthermore, due to the relatively small numbers of patients studied, a correlation of physical therapy with outcome and

proprioception could not be made. It does appear evident that a longer period from injury is correlated with enhanced proprioception (RPP only). This may be counterintuitive since the proposed natural history of isolated PCL rupture is to develop degenerative arthritis [18]. It is known that proprioception is reduced in the arthritic knee [2, 5, 58]. However, our investigation had only one patient studied nearly 20 years following PCL injury, and he had no radiographic evidence of arthritic change, as with all of the other patients (part of the exclusionary criteria). Thus the issue of the length of time after injury and the effects on proprioception are unclear. Further investigation with larger numbers of subjects with a greater range of time from injury to testing, and further follow-up of the subjects in this study to evaluate proprioceptive changes in the same individuals over time may help elucidate the effects of time on proprioception.

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