Anterior cruciate ligament reconstruction affects proprioception in the cat's knee

Enrique Gómez-Barrena[1], Angel Nuñez[2], Rafael Ballesteros[1], Enrique Martinez-Moreno[2] and Luis Munuera[1]

To study the cat's knee after anterior cruciate ligament reconstruction, we compared its neural and muscular activity with that in the normal and the unstable knee. We recorded the electric activity in the articular nerves (posterior -PAN and medial -MAN) and periarticular muscles (quadriceps and hamstring) while performing passive flexion, extension, external and internal rotation, and also anterior translation of the tibia at 30° and 90° of flexion. The same series of maneuvers was performed in the same knees after surgical section of the anterior cruciate ligament and then after anterior cruciate reconstruction. The electric activity recorded in the reconstructed knee was compared to that in the same knee before surgery and in the same unstable knee after anterior cruciate section. We observed that the reconstructed knee, compared to the injured knee, showed a decrease in articular nerves and quadriceps activity while it regained stability. This decrease converged to the recordings in the normal knee. However, differences in MAN, PAN and hamstring activity were still present in the reconstructed knee. This suggests that, although anterior cruciate reconstruction seems beneficial for restoring articular nerve and periarticular muscle activities to a certain degree, proprioception in the reconstructed knee does not match that in the normal knee.

Departments of [1]Orthopaedic Surgery and Morphology[2], Autonomous University of Madrid. Correspondence: Dr. Enrique Gómez-Barrena, Servicio de Cirugía Ortopédica y Traumatología, Hospital "Ntra. Sra. de Gracia", C/ Ramón y Cajal 60, ES-50004 Zaragoza, Spain. Tel +34 976 44-0022. Fax -58 69. E-mail: egbarrena@correo.interlink.es
Submitted 98-08-07. Accepted 99-01-27

Most researchers and clinicians agree that both mechanical integrity and proprioception participate in normal knee function. When the knee becomes unstable and symptomatic after anterior cruciate ligament damage, mechanical restoration is based on anterior cruciate reconstruction. However, this may be insufficient to achieve normal function (Barrett 1991). In broad clinical experience (Noyes et al. 1983), supported by studies where knees without mechanical reconstruction can limit instability during activity (Vergis and Gillquist 1996), it is suggested that other structures and systems may actively compensate the anterior cruciate deficiency in some cases.

The controversial role of the knee's proprioceptive system in enhancing knee stability in the normal and anterior cruciate-deficient knee is at the center of the debate. Some authors stressed the importance of proprioception in hamstring contraction, whose latency increased after anterior cruciate rupture (Beard et al. 1993). These authors later emphasized that articular proprioception changes could be the effect, rather than the cause, of mechanical modification of the joint (Beard et al. 1996). Experimental studies focused on models where the anterior cruciate ligament was isolated, to obtain data about muscular activation after anterior cruciate loading (Solomonow et al. 1987, Pope et al. 1990, Miyatsu et al. 1993). However, the sensitivity of an experimental set-up to obtain isolated data from a single structure, such as the anterior cruciate ligament, is controversial (Cole et al. 1995). In a different direction, our group developed a model to study articular nerve output and muscular reaction to passive knee motion, considering the knee as a whole (Gómez-Barrena et al. 1997). We showed that the electric activity in the articular nerves and periarticular muscles changed when the anterior cruciate ligament was transected. This study did not show whether these changes were due to mechanical derangement, proprioceptive dysfunction or both.

To address the issue of mechanical versus proprioceptive dysfunction at the origin of electric changes in the articular nerves and periarticular muscles during passive knee motion, we designed the present study. Our experiments, in a previously used model, aimed to test the hypothesis that electric changes during passive knee motion, due to anterior cruciate damage, would differ from the normal status, if some proprioceptive dysfunction persisted af-

ter mechanical stability was recovered by an acute anterior cruciate ligament reconstruction. This hypothesis included the verification of changes between the anterior cruciate-deficient knee and the reconstructed knee, and the evaluation of any residual changes between the normal knee and the reconstructed knee.

Animals and methods

Serial experiments with electric recordings in normal, unstable and reconstructed knees were conducted on 6 knees in 4 cats, weighing 2–3 kg, which underwent intraperitoneal pentobarbital anesthesia (30 mg/kg). 2 knees were used as surgical controls, as described below. To achieve a deep anesthesia, cortical activity was recorded in an electroencephalogram (EEG) during the experiment. Supplemental doses of anesthetics were given when a decrease in the amplitude of the slow wave activity was observed in the EEG. No voluntary activity in response to stimuli was found at that point. All the experiments were performed in accordance with the Declaration of Helsinki, and following the Spanish regulations on experimental animal protection in scientific projects, and the recommendations of the European Community, to avoid animal suffering.

As in previous experiments (Gómez-Barrena et al. 1997), we exposed the medial articular nerves (MAN) and the posterior articular nerves (PAN) under a surgical microscope and placed a nichrome hook electrode over them, to record their afferent potentials. We also recorded the electromyogram (EMG) of the quadriceps muscle and the hamstring, using a pair of bluntly cut insulated nichrome wire electrodes (120 μm diameter) placed in the mid-portion of the muscle. The afferent potentials and the EMG were filtered (0.3–3 KHz), amplified and recorded on magnetic tape with the beginning of the movement as a temporal reference for off-line analysis. The start and the end of every maneuver were signaled by the examiner. To record the EEG, a macroelectrode (120 μm diameter bluntly cut insulated nichrome wire) was inserted (1.5 mm deep) into the frontal cortex. The EEG was filtered (0.3–30 Hz), amplified and continuously monitored throughout the experiment in an analog oscilloscope.

We made our recordings during the passive range of motion, in the anesthetized cat's knee, in a supine position, starting with the knee at 90° flexion and the hip at 90°. From the starting position, we performed passive maximum flexion and back, maximum extension, maximum external rotation, and maximum internal rotation. The anterior instability examination included passive anterior tibial translation with the knee at 90° of flexion (anterior drawer), and at 30° of flexion (as in the Lachman maneuver). Each movement and maneuver was repeated 10 times, at a rate of 0.2 Hz. No muscular or neural fatigue was observed.

We compared neural and muscular activity in the 6 experiments sequentially performed in 6 knees, before and after anterior cruciate transection and after anterior cruciate reconstruction. Each anterior cruciate-reconstructed knee had its own control in the same normal and unstable knee, under the same experimental conditions. Anterior cruciate section and a subsequent reconstruction were performed in the course of the same experiment. No delay was allowed, to avoid any possible muscular or proprioceptive adaptation to instability.

We obtained a first set of recordings in the normal knee, comparable to those obtained under similar conditions in previous experiments (Gómez-Barrena et al. 1997). We then harvested a bone-tendon-bone autograft from the middle third of the patellar tendon, after midline exposure. Through the gap in the patellar tendon, a miniarthrotomy was performed and the anterior cruciate ligament was surgically transected. A second set of recordings was made after anterior cruciate transection, which caused severe anterior instability of the knee. Then the same knee underwent anterior cruciate ligament reconstruction through the previously performed miniarthrotomy. After tibial and femoral bone holes had been drilled, the bone-tendon-bone graft was placed and fixed with small fragment AO screws, used as interference screws. Anterior stability and full range of motion were checked before and after securing the screws. Knee stability was manually assessed at 30° flexion, after injury and reconstruction, on a semiquantitative scale, by an experienced orthopedic surgeon. This assessment, parallel to manual assessment in a clinical setting, included five grades, with 2 mm increments, in anterior tibial translation. We defined normal knee stability as grade 0 (with no anterior tibial translation), grade I (0–2 mm), grade II (2–4 mm), grade III (4–6 mm), and grossly unstable knee after ACL section as grade IV (with an anterior tibial translation greater than 6 mm). We performed only the neurophysiological study and accepted the knee in the study if the stability obtained after anterior cruciate transection was grade IV, and if it was grade 0 or grade I after anterior cruciate reconstruction. Slight grade I laxities with a firm end-point were included in this study, as they are generally accepted in clinical practice. After the reconstruction was completed and the stability confirmed, a third set of recordings was made for each knee included in the study.

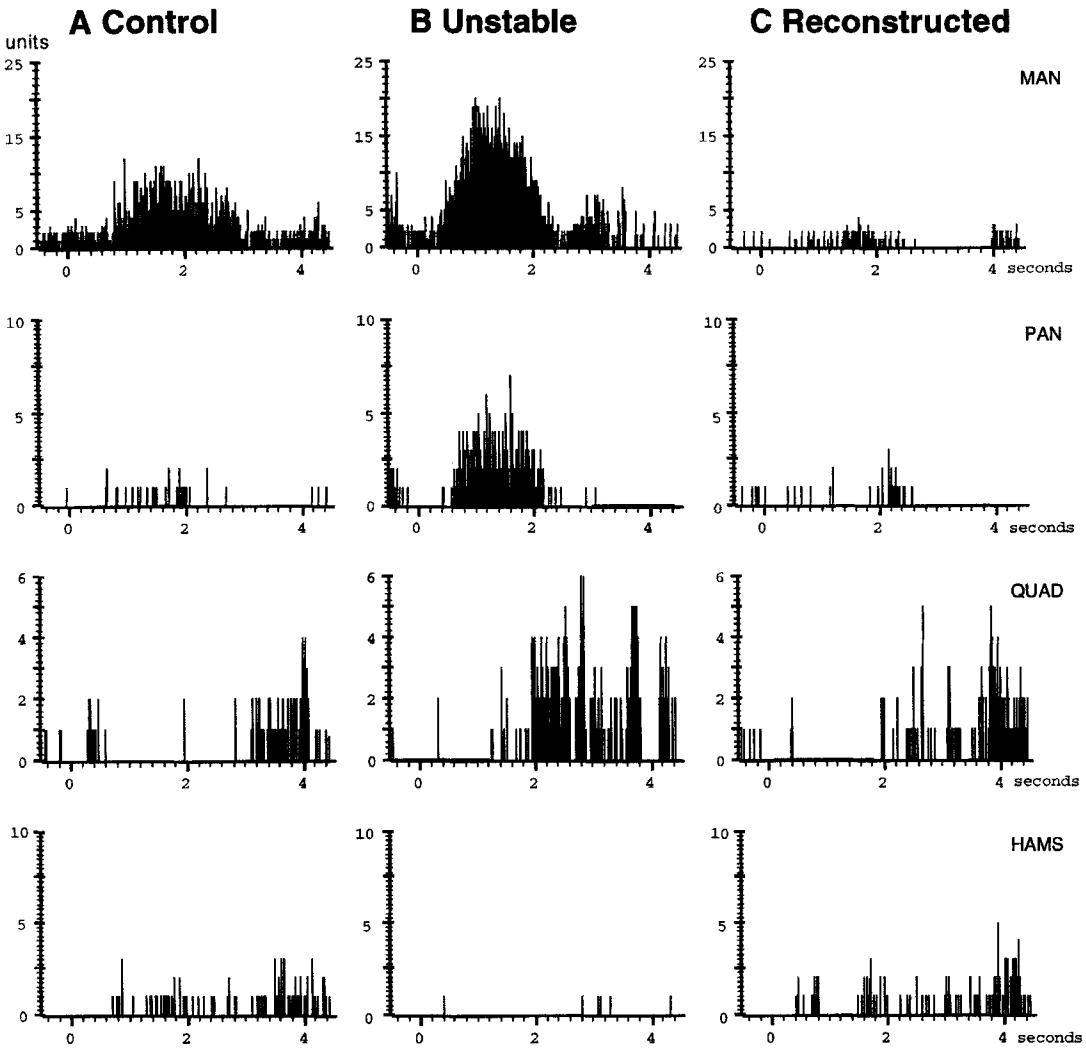

Figure 1. PSTH of afferent potentials in the MAN, PAN, and motor units in the quadriceps (QUAD) and hamstring (HAMS) in a single, representative experiment. Maneuvers of passive flexion, from 90° to maximum knee flexion, were repeated 10 times before (control), after anterior cruciate transection (unstable), and after anterior cruciate reconstruction (reconstructed).

2 knees were used as surgical controls in the stable knee. Sham operations of graft harvesting were performed after a first set of recordings, and a second set of recordings was made in the stable knee, after the autograft was harvested. 2 knees were used as surgical controls in the unstable knee. These knees were subjected to surgical section of the graft after reconstruction, and a set of recordings was performed in such knees, to be compared with previous recordings in these knees after anterior cruciate section. These controls were done to ensure that instability was reproduced with the graft section, and that the recordings previously obtained in the unstable knee were also reproduced.

The recorded data were fed into a computer at a sampling frequency of 10 KHz. Afferent multiunit potentials from the nerves and muscle motor units were detected, using a voltage threshold. Summed peristimulus time histograms (PSTH) were calculated between the afferent potentials or the motor units (Figure 1), using the beginning of the movement as a zero reference (bin width 50 msec) with Spike 2 software (C.E.D., Cambridge, UK). To study the synchronization of the neuronal and motor unit activity with the onset of the movement, a Gaussian fitting was calculated (Figure 2) on the data displayed in the PSTH. Response was defined as a mean afferent potential/bin or motor unit/bin during the movement, at least

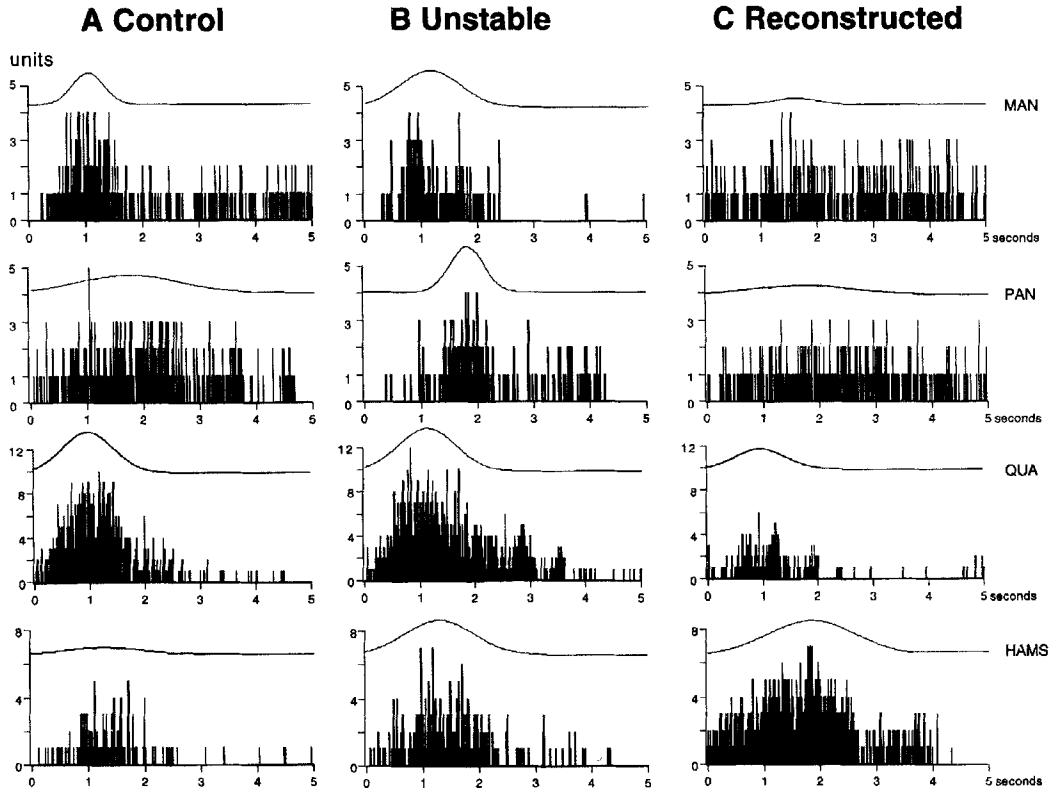

Figure 2. PSTH of afferent potentials in the MAN, PAN, and motor units in the quadriceps (QUAD) and hamstring (HAMS), with a Gaussian fit of the data in a single, representative experiment. Maneuvers of anterior tibial translation with the knee at 30° flexion (Lachman maneuver) were repeated 10 times before (control), after anterior cruciate transection (unstable), and after anterior cruciate reconstruction (reconstructed). Note loss of sequential MAN-PAN activity, and loss of any muscular parallelism.

twice as large as the spontaneous activity prior to the movement. The amplitude of the response for each movement was calculated as the mean afferent potential/bin or motor unit/bin during the movement, minus the mean spontaneous activity calculated during 200 msec recorded before the movement. Quantitative data were summarized as mean and 95% confidence intervals for the calculated units/bin. To avoid problems of multiple comparisons in serial experiments, statistical hypothesis-testing was avoided and confidence interval estimation was used instead. Statistical comparisons concerning increase or decrease in activity among serial experiments were based on 95% confidence intervals, which aimed primarily at parameter estimation. When the confidence interval obtained for an experiment did not overlap with the confidence interval for the reference status, an estimated decrease or increase was noted. This allowed comparisons among the knees in the normal, control status, with the status after anterior cruciate damage, and after subsequent anterior cruciate reconstruction.

Results

A first surgical examination was performed in stable knees, before anterior cruciate section. Sham operations in 2 animals, that included autograft harvesting and recording the stable knee without the graft, showed no differences from recordings performed in these knees before taking the graft. This proved that graft harvesting did not substantially impair proprioception in the stable knee.

A second surgical examination was performed on the unstable knee. 2 experiments proved that sectioning of the graft in the reconstructed knee allowed reproduction of recordings obtained in the unstable knee.

Anterior cruciate-deficient knees before and after reconstruction (Table)

Knee stabilization was obtained by the anterior cruciate reconstruction in all the cases, although 2 knees showed complete stability of grade 0 (no anterior tibial translation) and 4 knees showed a slight laxity of

Quantitative data on the electric activity of the normal, unstable, and reconstructed cat's knee

	MAN	PAN	Quadriceps	Hamstring
Passive motion				
Flexion				
Normal	2.9 (2.6–3.3)	2.2 (1.6–2.7)	1.5 (1.0–2.1)	1.1 (0.9–1.4)
Anterior cruciate section	6.5 (5.7–7.3)	3.3 (2.8–3.8)	3.4 (2.9–3.9)	1.2 (0.9–1.5)
Anterior cruciate reconstruction	2.0 (1.5–2.6)	2.3 (1.9–2.7)	1.4 (1.1–1.7)	1.7 (1.4–2.0)
Extension				
Normal	3.7 (3.3–4.2)	0.6 (0.4–0.8)	2.5 (2.2–2.8)	1.9 (1.7–2.1)
Anterior cruciate section	7.3 (6.5–8.1)	1.3 (1.0–1.6)	1.2 (1.0–1.4)	1.4 (1.0–1.8)
Anterior cruciate reconstruction	4.4 (3.6–5.2)	1.6 (1.2–2.1)	2.1 (1.7–2.5)	2.5 (2.2–2.9)
External rotation				
Normal	2.6 (2.4–2.8)	1.2 (1.0–1.4)	1.4 (1.2–1.7)	1.3 (1.1–1.5)
Anterior cruciate section	2.6 (2.4–2.8)	2.4 (2.2–2.6)	2.0 (1.8–2.2)	2.4 (2.2–2.6)
Anterior cruciate reconstruction	1.4 (1.1–1.6)	1.7 (1.5–1.8)	1.4 (1.2–1.6)	2.5 (2.3–2.7)
Internal rotation				
Normal	2.3 (2.0–2.6)	1.3 (1.0–1.6)	1.0 (0.8–1.2)	1.8 (1.5–2.2)
Anterior cruciate section	3.3 (2.9–3.8)	2.4 (2.1–2.8)	2.6 (2.1–3.1)	2.7 (2.2–3.2)
Anterior cruciate reconstruction	2.6 (2.3–2.8)	2.1 (1.8–2.4)	1.2 (0.9–1.4)	2.3 (2.0–2.5)
Anterior tibial translation				
At 90° flexion				
Normal	3.6 (3.1–4.0)	1.4 (1.3–1.5)	1.1 (1.0–1.2)	1.0 (0.9–1.2)
Anterior cruciate section	5.9 (5.5–6.0)	2.2 (2.0–2.4)	1.5 (1.2–1.8)	0.6 (0.5–0.6)
Anterior cruciate reconstruction	3.8 (3.2–4.4)	2.1 (1.9–2.4)	1.4 (1.2–1.5)	1.7 (1.6–1.8)
At 30° flexion				
Normal	1.7 (1.6–1.8)	1.2 (1.1–1.2)	1.4 (1.3–1.5)	1.5 (1.4–1.6)
Anterior cruciate section	3.3 (3.0–3.5)	2.0 (1.9–2.2)	2.0 (1.9–2.1)	0.9 (0.7–1.0)
Anterior cruciate reconstruction	0.9 (0.8–1.0)	1.7 (1.5–1.8)	1.5 (1.5–1.6)	1.6 (1.3–1.8)

Values express the mean units/bin (95% confidence interval) (n 6).

grade I (anterior tibial translation between 0 and 2 mm). Same-knee comparison after anterior cruciate damage, before and after reconstruction, allowed us to verify changes obtained with mechanical stabilization. The overall increase in the electric response observed in the unstable condition, as previously described (Gómez-Barrena et al. 1997), was found to decrease after anterior cruciate reconstruction. This occurred in flexion (MAN, PAN, quadriceps), extension (MAN), external rotation (MAN, PAN, quadriceps), internal rotation (MAN, quadriceps), and anterior tibial translation at 90° knee flexion (MAN), and at 30° knee flexion (MAN, PAN, quadriceps). An increase in activity was noted in extension (quadriceps and hamstring), and in anterior tibial translation at 90° and 30° flexion (hamstring).

Comparison between normal and reconstructed knees (Table)

Same-knee comparison before anterior cruciate damage and after reconstruction showed the persistence of differences that prevented a full return to the original, normal status. A marked decrease in electric activity, compared to the normal knee status, was noted in flexion (MAN), external rotation (MAN), and anterior tibial translation at 30° knee flexion (MAN). An increase in activity, compared to the basal status, was observed in flexion (hamstring), extension (PAN and hamstring), external rotation (PAN and hamstring), internal rotation (PAN), anterior tibial translation at 90° flexion (PAN and hamstring), and anterior tibial translation at 30° flexion (PAN). Apart from quantitative differences, qualitative differences were present. The reconstructed knee showed a diffuse activity in articular nerves, losing the sequential activation of MAN and PAN, and also losing any parallelism with muscular activity, as observed in the recordings of the normal and unstable knee (Figure 2).

Flexion (Figure 3)

The increase in MAN, PAN, and quadriceps electric activity noted after anterior cruciate section during passive flexion was mostly reverted after anterior cruciate reconstruction. However, MAN activity shifted to values below normal. Hamstring activity did not change substantially.

Extension (Figure 3)

The reconstruction produced a return to normal values in MAN and quadriceps activity, after the increase noted in MAN and the decrease in quadriceps due to anterior cruciate damage. The PAN activity, that was increased after anterior cruciate section, remained high after reconstruction. Hamstring activity showed a marked increase with reconstruction, and was higher than values in the damaged and normal knee.

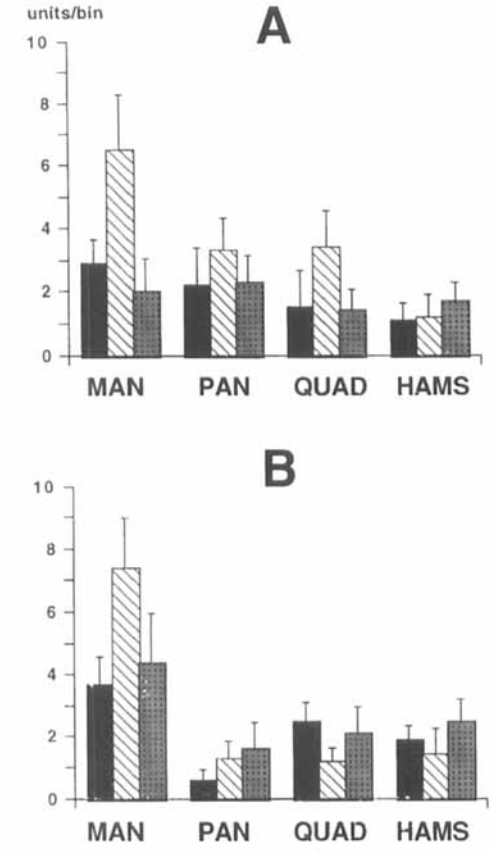

Figure 3. Plots of the response area at the MAN, PAN, quadriceps (QUAD), and hamstring (HAMS), for all the knees (n 6) during passive knee flexion (A) and extension (B), in the normal knee (solid bars), after anterior cruciate transection (hatched bars), and after anterior cruciate reconstruction (gray, dotted bars). Note the marked decrease in MAN activity in flexion, after reconstruction (dotted bar, A).

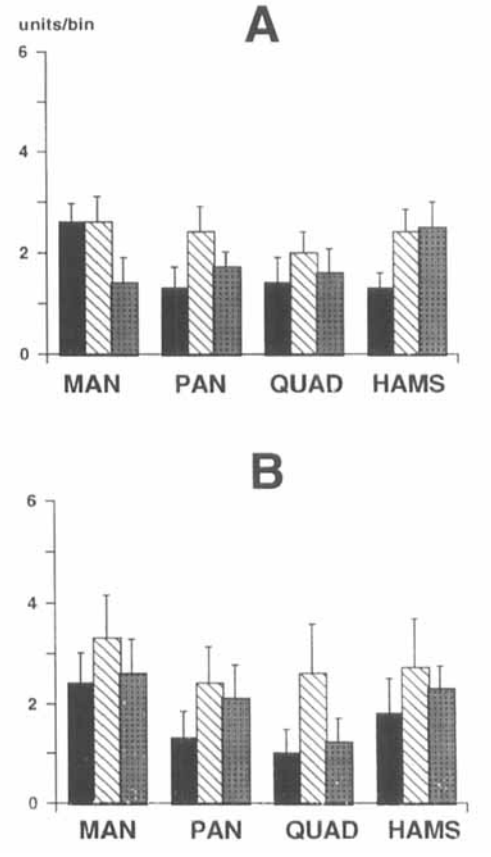

Figure 4. Plots of the response area at the MAN, PAN, quadriceps (QUAD), and hamstring (HAMS), for all the knees (n 6) during passive external (A) and internal rotation (B), in the normal knee (solid bars), after anterior cruciate transection (hatched bars), and after anterior cruciate reconstruction (gray, dotted bars). Note the marked decrease in MAN activity in external rotation, while hamstring activity remains high (A).

External rotation (Figure 4)

The activity in PAN, quadriceps, and hamstring increased after anterior cruciate section. With reconstruction, a decrease was noted in PAN and quadriceps, but also in MAN. The activity in the hamstring remained high. The decrease noted in quadriceps activity returned to normal values, but not that in PAN, where it decreased but still remained high.

Internal rotation (Figure 4)

The increase in activity noted in MAN, PAN, quadriceps, and hamstring after injury, reverted to normal values in MAN and quadriceps after reconstruction. Activity in PAN remained high, while changes in the hamstring fell between the normal and increased values.

Anterior tibial translation

When an anterior tibial translation was forced at 90° flexion (Figure 5), anterior cruciate injury caused an increase in MAN, PAN, and quadriceps activity, but a decrease in hamstring activity. Reconstruction induced a decrease in MAN activity to normal values, but no change in PAN activity, which remained high. After reconstruction, quadriceps activity fell to a level between the normal and increased values, while hamstring activity rose substantially over the injury, and even over normal values.

At 30° knee flexion (Figure 5), the activity caused by anterior tibial translation in the injured knee had increased in MAN, PAN, and the quadriceps, but had decreased in the hamstring. This activity was decreased after reconstruction in MAN, PAN, and the quadriceps, while it was increased in the hamstring.

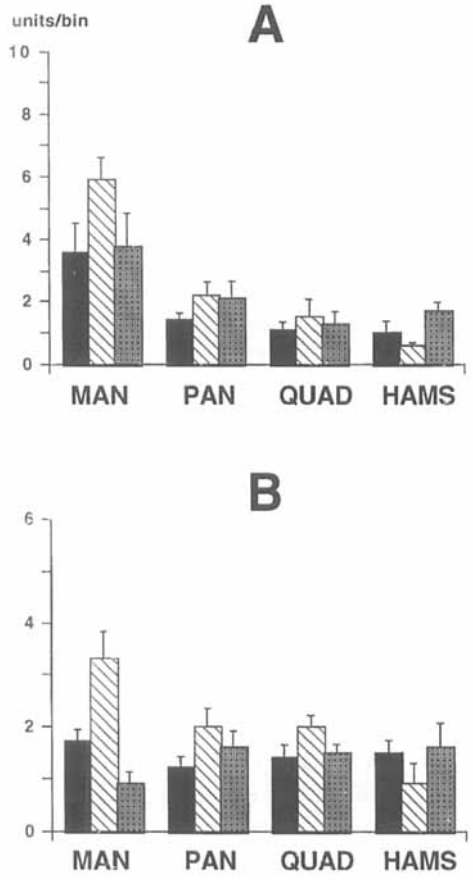

Figure 5. Plots of the response area at the MAN, PAN, quadriceps (QUAD), and hamstring (HAMS), for all the knees (n 6) during anterior tibial translation with the knee at 90° (A) and 30° flexion (B), in the normal knee (solid bars), after anterior cruciate transection (hatched bars), and after anterior cruciate reconstruction (gray, dotted bars). Note the marked increase in hamstring activity at 90° (A), and the decrease in MAN activity at 30° (B).

However, only in the quadriceps and hamstring did the activity values return to normal. A marked decrease in activity was observed in MAN, while a persistent increase was noted in PAN, in which the decrease did not return to normal values.

Discussion

Knee proprioceptive information originates in articular and periarticular neural endings. Morphologic evidence of the presence of neural fibers and endings in knee structures is widely accepted (Skoglund 1956, Freeman and Wyke 1967, Schultz et al. 1984, Schutte et al. 1987, Halata and Haus 1989, Sjölander et al. 1989, Haus and Halata 1990). The anatomic basis of the articular proprioceptive system includes not only receptors, but also articular nerves (called MAN -medial articular nerve- and PAN -posterior articular nerve- in the cat's knee, after Freeman and Wyke 1967), that lead information from the anterior cruciate and other knee structures to segmental spinal ganglia (Craig et al. 1988, Gómez-Barrena et al. 1996). This information helps to modulate muscular tone around the joint through the γ motor system, as suggested by Johansson et al. (1991). Although there is general agreement on the morphologic presence of a proprioceptive system, much debate surrounds its possible role in knee function. Experimental neurophysiologic studies can help to facilitate understanding of the role of proprioception in knee function. Rather than clarifying the neurophysiology of a single knee element, such as the anterior cruciate ligament, our study focuses on the neurophysiology of the normal knee, the anterior cruciate-deficient knee, and the knee after anterior cruciate ligament reconstruction.

In a previous study (Gómez-Barrena et al. 1997), we evaluated electric activity differences in the knee before and after anterior cruciate ligament section. In the present study and before examining the data obtained, we made recordings of the knee before surgery and after anterior cruciate section, as a control of our previous experiments. Minor differences were observed, but none of them affected activity increments. These changes were not thought to influence our previous results, but rather to support them, improving the precision of the comparisons.

When comparing the unstable and the reconstructed knee, our results showed that reconstruction caused a partial recovery from the hyperexcitable knee status that was found after acute damage of the anterior cruciate ligament. Instability after anterior cruciate damage generated increased electric activity. This was detected in articular nerves (Gómez-Barrena et al. 1997), mostly in MAN but also in PAN, and verified in the present study. However, there was a general decrease in articular nerve activity after reconstruction, mostly in MAN but also in PAN. This finding partly supports the proposal of Beard et al. (1996) that changes in proprioception after anterior cruciate reconstruction could be the effect of mechanical modification of the joint. However, when we compared the normal status with the reconstructed knee, we found that MAN activity had fallen below that expected after stabilization. Most striking was the fact that, when the knee underwent anterior tibial translation at 30° flexion (as in the Lachman maneuver), the activity detected in MAN fell below normal activity, after it had been raised in the unstable knee due to stimulation of secondary restraints such as the medial meniscus.

This decrease in MAN activity, also occurring during knee flexion and external rotation, was not present during extension or internal rotation, thus proving that MAN was functioning. In our opinion, this is a proof of some decrease in proprioception in the injured knee, even after the anterior cruciate reconstruction improved the mechanical derangement in the unstable knee.

We would expect a similar decrease in PAN activity, since we had already shown that a significant portion of anterior cruciate afferents course through this nerve (Gómez-Barrena et al. 1996). With reconstruction, we found a significant decrease in PAN activity in flexion or in anterior tibial translation at 30° (as in the Lachman test), as compared to the unstable knee, but PAN activity remained high in extension, as compared to the normal knee. Our explanation of these findings was that the possible decrease in proprioception, due to anterior cruciate damage, was hidden in the unstable knee by stimuli from different structures (posterior horns of menisci, posterolateral corner, posterior cruciate ligament). When the stability was recovered through reconstruction, the hyperexcitability decreased but a diffuse, non-sequential PAN activity was maintained (Figure 2), mostly in extension and rotation (Table). This effect may obscure the subtle decrease in quantitative proprioception, due to the anterior cruciate ligament in the PAN, a nerve through which considerable information from the posterior knee is conducted. Since the anterior cruciate reconstruction may not control rotational stability, slight residual laxity may enhance these stimuli and augment these results.

Muscular activity also differed significantly in the reconstructed knee. The increased quadriceps activity in the injured knee during flexion, external/internal rotation, or anterior tibial translation, and the decreased activity in extension, all reverted to normal values after anterior cruciate reconstruction. Therefore, no primary defect in muscle excitability was identified in the quadriceps following anterior cruciate damage and reconstruction, in our model. However, as there is a topographic relationship between MAN and the quadriceps muscle, based on retrograde axonal transport experiments (Gómez-Barrena et al. 1996), we suggest that the decreased activity found in MAN, in the reconstructed knee, may sustain quadriceps inhibition in the long-term. This may impair proprioceptive and muscle adaptation, causing persistent muscle weakness and quadriceps-related deficits (Andriacchi and Birac 1993). As this decreased activity was not observed in the stable controls, before anterior cruciate section, but after patellar tendon autograft harvesting, we cannot relate these findings to the reconstruction technique but to the anterior cruciate damage.

Most interesting results were obtained when hamstring activity was observed after reconstruction. This remained as high as in the unstable knee in external rotation. In contrast, reconstruction significantly enhanced hamstring activity over normal, in response to flexion, extension, and anterior tibial translation at 90° flexion. This hamstring effect was not observed in response to anterior tibial translation at 30° flexion. To some extent, these findings could be linked to increased PAN activity in extension, although opposite increments were found in anterior tibial translation. This divergence indicates a more complex regulation of hamstring activity, when the proprioceptive signals from the anterior cruciate are absent. The finding that mechanical stability produced enhanced hamstring activity may explain why hamstring weakness is very rare following anterior cruciate reconstructions.

We conclude that we were able to verify our hypothesis: significant changes in the electric activity in articular nerves and periarticular muscles were found between the normal and the anterior cruciate reconstructed knee, that can be related to persistent proprioceptive dysfunction. Significant changes were found between the anterior cruciate-deficient knee and the reconstructed knee, with a decrease towards the normal status. As a consequence, mechanical stabilization may be beneficial to provide a more efficient setting for proprioceptive and muscular activity. However, the decrease observed in medial proprioception was related to the absence of normal anterior cruciate ligament proprioception. Posterior proprioception that remained high after stabilization, losing the sequential activity of the normal knee, also represented a different situation from the normal status. Furthermore, hamstring activation changed from the decrease found in the unstable knee to an increase in the reconstructed knee, while quadriceps activation was diminished. These findings may explain why some abilities are not recovered in the reconstructed knees.

F.I.S. (Fondo de Investigaciones Sanitarias, Ministry of Health, Spain), grant # 95/0548.

Andriacchi T P, Birac D. Functional testing in the anterior cruciate ligament-deficient knee. Clin Orthop 1993; 288: 40-7.

Barrett D S. Proprioception and function after anterior cruciate reconstruction. J Bone Joint Surg (Br) 1991; 73: 833-7.

Beard D J, Kyberd P J, Fergusson C M, Dodd C A F. Proprioception after rupture of the anterior cruciate ligament. An objective indication of the need for surgery? J Bone Joint Surg (Br) 1993; 75: 311-5.

Beard D J, Dodd C A F, Simpson A H R W. The effect of reconstruction on proprioception in the anterior cruciate ligament-deficient knee. Trans Orthop Res Soc 1996; 21: 786.

Cole K J, Brand R A, Daley B J. The sensitivity of joint afferents to knee translation. Iowa Orthop J 1995; 15: 166-73.

Craig A D, Heppelmann B, Schaible H-G. The projection of the medial and posterior articular nerves of the cat's knee to the spinal cord. J Comp Neurol 1988; 276: 279-88.

Freeman M A R, Wyke B. The innervation of the knee joint. An anatomical and histological study in the cat. J Anat 1967; 101: 505-32.

Gómez-Barrena E, Martínez-Moreno E, Munuera L. Segmental sensory innervation of the anterior cruciate ligament and the patellar tendon of the knee. Acta Orthop Scand 1996; 67 (6): 545-52.

Gómez-Barrena E, Nuñez A, Martínez-Moreno E, Valls J, Munuera L. Neural and muscular electric activity in the cat's knee: changes when the anterior cruciate ligament is transected. Acta Orthop Scand 1997; 68 (2): 149-55.

Halata Z, Haus J. The ultrastructure of sensory nerve endings in human anterior cruciate ligament. Anat Embryol 1989; 179: 415-21.

Haus J, Halata Z. Innervation of the anterior cruciate ligament. Int Orthop (SICOT) 1990; 14: 293-6.

Johansson H, Sjölander P, Sojka P. A sensory role for the cruciate ligaments. Clin Orthop 1991; 268: 161-78.

Miyatsu M, Atsuta Y, Watakabe M. The physiology of mechanoreceptors in the anterior cruciate ligament. J Bone Joint Surg (Br) 1993; 75: 653-7.

Noyes F R, Matthews D S, Mooar P A, Grood E S. The symptomatic anterior cruciate-deficient knee. Part II. J Bone Joint Surg (Am) 1983; 65: 163-74.

Pope D F, Cole K J, Brand R A. Physiologic loading of the anterior cruciate ligament does not activate quadriceps or hamstring in the anesthetized cat. Am J Sports Med 1990; 18: 595-9.

Schultz R A, Miller D C, Kerr C S, Micheli L. Mechanoreceptors in human cruciate ligaments: A histological study. J Bone Joint Surg (Am) 1984; 69: 243-7.

Schutte M J, Dabezies E J, Zimny M L, Happel L T. Neural anatomy of the human anterior cruciate ligament. J Bone Joint Surg (Am) 1987; 66: 1072-6.

Sjölander P, Johansson H, Sojka P, Renholm A. Sensory nerve endings in the cat cruciate ligaments: A morphological investigation. Neurosci Lett 1989; 102: 33-8.

Skoglund P. Anatomical and physiological studies of the knee joint innervation in the cat. Acta Physiol Scand (Suppl 124) 1956; 34: 1-101.

Solomonow M, Baratta R, Zhou B H, Shoji H, Bose W, Beck C, D'Ambrosia R. The synergistic action of the anterior cruciate ligament and thigh muscles in maintaining joint stability. Am J Sports Med 1987; 15: 207-13.

Vergis A, Gillquist J. Sagittal plane translation of the knee during stair walking: comparison of healthy and ACL-deficient subjects. Eur Orthop Res Soc Trans 1996; 6: 12.