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Am J Sports Med 2011 39: 78 originally published online October 7, 2010
DOI: 10.1177/0363546510378652

Anterior Laxity, Slippage, and Recovery of Function in the First Year After Tibialis Allograft Anterior Cruciate Ligament Reconstruction

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Background: The increase in anterior laxity and slippage is greater with metal interference screw fixation of a hamstring anterior cruciate ligament (ACL) graft than a bone-patellar tendon-bone graft.

Hypothesis: When slippage-resistant fixation is used with a soft tissue graft, early recovery of function does not result in a clinically important increase in anterior laxity and slippage

Study Design: Case series; Level of evidence, 4.

Methods: Nineteen subjects were treated with a single-tunnel, single-looped, tibialis allograft with slippage-resistant, cortical fixation. An examiner, different from the treating surgeon, used stereophotogrammetric analysis to compute the increase in anterior laxity at a 150 N anterior force and slippage between the day of surgery and each monthly follow-up interval, and determined recovery of function and motion.

Results: Anterior laxity did not increase between the day of surgery and 1 year ($P = .23$). Total slippage plateaued after 1 month, but increased 1.5 mm between the day of surgery and 1 month ($P < .05$). Extension and flexion plateaued after 2 months ($P < .0001$ and $P < .02$, respectively); activity level (Tegner score) plateaued after 3 months ($P < .05$), function (Lysholm score) plateaued after 4 months ($P < .002$), and subjective satisfaction (International Knee Documentation Committee score) plateaued after 6 months ($P < .02$).

Conclusion: Early recovery of function after ACL reconstruction with a soft tissue allograft did not result in a clinically important increase in anterior laxity and slippage at 1 year. We believe the avoidance of an increase in anterior laxity was related to the use of a transtibial technique designed to place the femoral and tibial tunnels without roof and posterior cruciate ligament impingement, the use of cortical fixation devices designed to resist slippage, the use of an aseptically harvested fresh-frozen tibialis allograft that was not irradiated or chemically processed, and the use of a self-administered rehabilitation program designed to encourage an early return of motion and function.

Keywords: anterior cruciate ligament (ACL); roentgen stereophotogrammetric analysis; anterior laxity; slippage

An increase in anterior laxity after ACL reconstruction should be avoided because it may cause recurrent instability.[5] The causes of an increase in anterior laxity are slippage at the sites of fixation, elongation of the ACL graft, and a reduction in stiffness of the ACL graft construct.[24,34,42]

An in vivo roentgen stereophotogrammetric analysis (RSA) study at monthly intervals during the first year after surgery showed that an autogenous double-looped semitendinosus and gracilis hamstring graft had a greater increase in anterior laxity and slippage at the sites of fixation than a bone-patellar tendon-bone graft when fixed with metal interference screws and a conservative rehabilitation program with use of a padded splint and protected weightbearing.[24] The increase in anterior laxity of 8.6 mm and slippage of 7.1 mm after 1 year with the autogenous hamstring graft particularly concerned us because we also prefer a soft tissue graft (ie, tibialis allograft), and because we use a self-administered rehabilitation program that is more aggressive, which might cause an even greater increase in anterior laxity and slippage.[19,21]

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One or more authors has declared a potential conflict of interest: One author (S.M.H.) is a consultant and receives royalties from Biomet Sports Medicine. A grant was received from the Musculoskeletal Transplant Foundation.

The American Journal of Sports Medicine, Vol. 39, No. 1
DOI: 10.1177/0363546510378652

This concern motivated us to investigate whether our technique for ACL reconstruction has a high increase in anterior laxity and slippage during the first year after surgery. We attempted to limit the increase in anterior laxity from biologic or incorporation causes by using an aseptically harvested, nonirradiated, nonchemically cleansed fresh-frozen tibialis allograft from a tissue bank accredited by the American Association of Tissue Banks instead of irradiated and chemically cleansed allografts, which have a higher prevalence of failure.[25,33] We attempted to limit the increase in anterior laxity from mechanical causes with the use of a wallplasty, drill guides, and arthroscopic checkpoints to eliminate graft impingement against the intercondylar roof in extension and the posterior cruciate ligament (PCL) in flexion, which stretches the graft and limits motion.[9,14,16,20,38] We attempted to limit slippage with the use of cortical fixation devices that are more slippage-resistant than interference screw fixation,[3,27,39,45] and by bone grafting the tibial tunnel to promote tendon-tunnel healing, which increases the stiffness and strength of the ACL graft construct and reduces the tensile load in the graft at the site of fixation.[18,31,39,46] These safeguards were used to justify the use of an aggressive rehabilitation program without a brace, full weightbearing, and self-administered exercises to achieve the goal of an early return of function with the keen understanding that early, cyclical tensile loading of the graft might increase anterior laxity from slippage at the sites of fixation and graft elongation.

With use of an examiner different from the treating surgeon, the present study measured the increase in anterior laxity and slippage at each monthly follow-up interval from the day of surgery and each monthly follow-up interval, and the recovery of function (Tegner, Lysholm, International Knee Documentation Committee [IKDC] scores) and motion on the day of surgery and at each monthly follow-up interval (1, 2, 3, 4, 6, and 12 months). The purpose of the study was to test the hypothesis that an early recovery of function does not have to result in a clinically important increase in anterior laxity and slippage after ACL reconstruction with a soft tissue graft.

MATERIALS AND METHODS

All patients who consented to an ACL reconstruction with a tibialis allograft between June 2007 and September 2008 were offered participation in the study. During the time frame of the study, the treating surgeon (S.M.H.) performed approximately 150 primary ACL reconstructions, of which 90% were with a tibialis allograft and 10% were with an autogenous double-looped semitendinosus gracilis hamstring graft. The inclusion criteria were a knee with a torn ACL, full motion at the time of reconstruction, and no roentgenographic evidence of degenerative arthritis. Subjects were excluded if the knee had a combined ligament injury, patellar dislocation, or prior open knee surgery. Twenty-three subjects signed a consent form approved by an institutional review board.

The single-tunnel, arthroscopically assisted ACL reconstruction was performed under general anesthesia with an aseptically harvested, fresh-frozen, nonirradiated, nonchemically treated tibialis allograft from a tissue bank accredited by the American Association of Tissue Banks (Musculoskeletal Transplant Foundation, Edison, New Jersey) with use of a previously described technique.[30] A 9-mm diameter single-looped anterior or posterior tibialis graft was used because the structural, material, and viscoelastic properties of each tendon are similar.[12] Although the exact age of the donor was unknown, studies have shown that age of the donor does not have an important effect on the mechanical properties and modulus of tendon grafts.[11,12,45] The tibial and femoral drill holes were made using a transtibial technique to fulfill the criterion of an anatomical reconstruction, which is avoiding roof impingement, avoiding PCL impingement, and matching the tension pattern in the graft to that of the intact ACL during passive motion (Figure 1). Avoiding roof and PCL impingement and matching the tension pattern of the graft to that of the intact ACL during passive motion are necessary to prevent lengthening of the graft from mechanical causes.[14,20,21,38] Roof impingement was avoided by drilling the tibial guide wire 4 to 5 mm posterior and parallel to the intercondylar roof with the knee in maximum extension (Howell 65° Tibial Guide, Biomet Sports Medicine, Inc, Warsaw, Indiana). Posterior cruciate ligament impingement was avoided by widening the notch until the space between the PCL and lateral femoral condyle was 10 mm, placing the angle of the tibial tunnel at 60° to 65° with respect to the medial joint line of the tibia in the coronal plane, and confirming that the guide wire was lateral and did not cross the PCL.[16,17]

The technique of RSA was used to measure the increase in anterior laxity and total slippage because the measurement of anterior laxity and slippage is repeatable and has no detection bias as it is quantitatively computed.[22-24,34,35,41,42] The intertest error or precision from performing serial measurements of anterior laxity with the RSA loading apparatus used in our study has been previously reported and is 0.5 mm.[40] The precision was determined by a statistical analysis of 3 sets of biplanar radiographs in 12 randomly selected subjects obtained after removing and replacing the knee in the loading apparatus 3 times at the 1-month follow-up interval.[40] The precision of 0.5 mm is a summation of all unintentional errors caused by (1) inconsistent positioning of the limb in the loading apparatus, (2) variability in the applied anterior load at the knee, (3) undetected activation of leg muscles, (4) inconsistent exposure of the radiographs, and (5) interobserver error associated with image and data processing.

Tantalum markers were injected in the femur, tibia, graft, and affixed to the femoral and tibial fixation devices to measure anterior laxity and slippage. To monitor the increase in anterior laxity, 0.8-mm diameter tantalum markers (model 20401, Tilly Medical Products AB, Lund, Sweden) were implanted in the femur (n = 6) and tibia (n = 6) with a bead injector (model 20202, Tilly Medical Products) (Figure 2). To monitor slippage of the fixation devices with respect to the bone, three 1.0-mm diameter

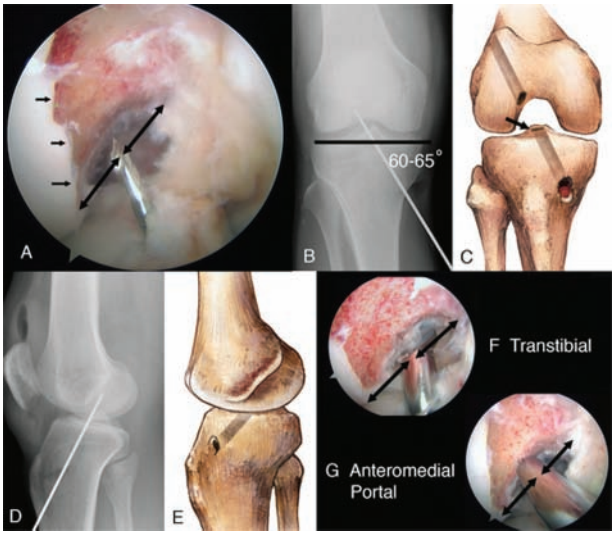

Figure 1. Composite of a right knee showing the key arthroscopic and radiographic checkpoints for placing the tibial tunnel and drilling the femoral tunnel through the tibial tunnel (transtibial technique) so that impingement of the ACL graft against the PCL in flexion and impingement against the roof in extension are avoided, and the tension pattern of the ACL graft matches that of the intact ACL throughout the flexion-extension arc.[16] Widen the notch until the space between the PCL and lateral femoral condyle exceeds the width of the graft by 1 mm (3 small arrows) (A). In the coronal plane, place the tibial guidewire so that the angle between the wire and the medial joint line is 60° to 65° (B), and place the lateral edge of the tibial tunnel so that it passes through the tip of the lateral tibial spine (arrow) (C). In the sagittal plane, place the tibial guide wire 4 to 5 mm posterior and parallel to the intercondylar roof with the knee in extension (D), which places the anterior edge of the tibial tunnel 1 to 2 mm posterior and parallel to the intercondylar roof (E). Check arthroscopically that the tip of the tibial guidewire lies midway between the apex and base of the notch (wire bisects 2 double-headed arrows) (A). If the tip lies closer to the apex of the notch (vertical placement), then the ACL graft has PCL impingement, which will limit flexion or stretch the graft, causing instability. If the tip lies closer to the bottom of the notch (posterolateral tunnel placement), then the tension in the graft will be greater than the intact ACL in extension,[29,36] which will limit extension or stretch the graft, causing instability. Once the tibial tunnel is drilled with these conditions, then the femoral tunnel can be drilled by passing the femoral aimer through the tibial tunnel (F). Alternatively, the femoral tunnel can be drilled in the same spot through the anteromedial portal (G).

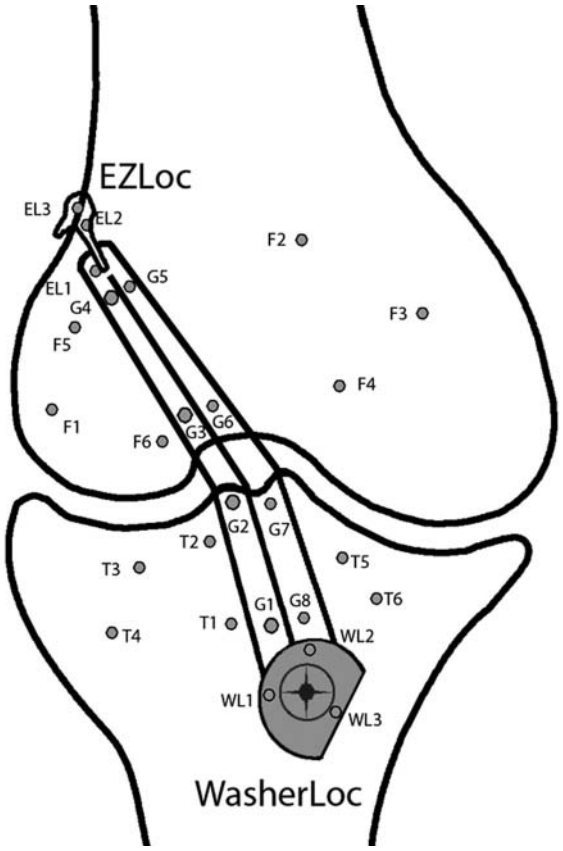

Figure 2. Schematic drawing showing the anterior-posterior view of the knee and the locations of the markers. Markers G1-G8 were inserted in the graft (n = 8), EL1-EL3 were fixed to the femoral fixation device (n = 3), WL1-WL3 were fixed to the tibial fixation device (n = 3), F1-F6 were implanted in the femur (n = 6,), and T1-T6 were implanted in the tibia (n = 6). Markers with a diameter of 1.0 mm were injected into one strand (G1-G4) and markers with a diameter of 0.8 mm were injected into the other strand (G5-G8). All the markers in the graft were inside the bone tunnels; markers G1, G4, G5, and G8 were inserted 5 mm from the fixation device, and markers G2, G3, G6, and G7 were inserted 5 mm from the entrance of the tunnel into the intercondylar notch.

tantalum markers were pressfit into holes milled into a cortical femoral fixation device (9- to 10-mm standard EZLoc, Biomet Sports Medicine) and an additional 3 markers were pressfit into a multispiked tibial fixation device (size 18 WasherLoc, Biomet Sports Medicine) (Figure 3). To monitor slippage of the soft tissue graft with respect to the fixation devices, four 0.8-mm diameter tantalum markers were injected into one strand of the tibialis allograft, and an additional four 1.0-mm diameter tantalum markers were injected into the other strand. The position of the markers within the strands was chosen so that they were confined inside the bone tunnels and with one marker within 10 mm of each fixation device.

After the markers were inserted, the tibialis allograft and femoral fixation device were fixed to the femur, tension was manually applied to the distal end of the graft, and the knee was flexed and extended for 15 cycles to seat the graft and the femoral fixation device. The anterior-posterior position of the tibia with respect to the femur and the tension applied to the graft at the time of tibial fixation were unmeasured. The position of the tibia and tension in the graft were established by allowing

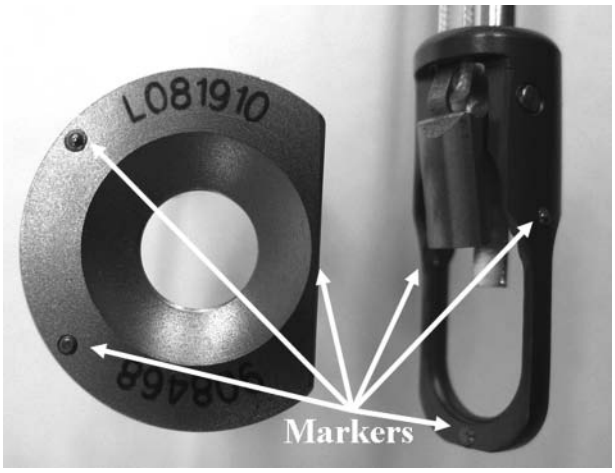

Figure 3. Locations of the three 1.0-mm diameter tantalum markers (arrows) in the multispiked tibial fixation device (left) and in the femoral fixation device (right). The markers were pressfit into milled holes made with a 0.940-mm diameter ball end mill.

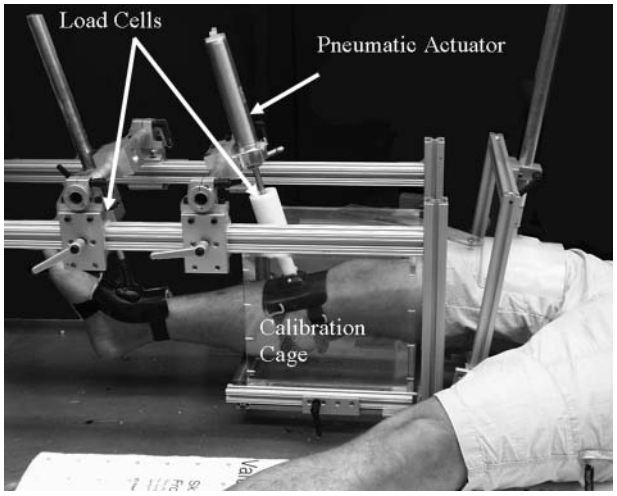

Figure 4. Limb in the loading apparatus. The knee is centered in the calibration cage and is flexed 25°. The ankle and thigh are secured in supports. The pneumatic actuator is oriented perpendicular to the shank and parallel to the knee joint at a distance of 12.5 cm distal to the joint line. The pneumatic actuator applied anterior and posterior forces to the tibia. Load cells measured the applied force and the reaction force at the ankle joint.

gravity to translate the tibia posteriorly by suspending the knee in hyperextension with the heel placed on a Mayo stand, applying a maximum unmeasured tension to the graft exiting the tibial tunnel, positioning the tibial fixation device on the cortex, applying an unmeasured posterior force to the tibial fixation device and tibia, checking that the spikes of the tibial fixation device had not engaged the graft, confirming that the maximum unmeasured tension applied to the graft was transmitted across the tibial fixation device, impacting the tibial fixation device, and inserting the compression screw. A 25-mm long, 8-mm diameter autogenous bone dowel previously harvested from the tibial tunnel was reinserted into the tibial tunnel to increase stiffness and promote circumferential tendon tunnel healing.[18,31,39] The wound was closed and 30 mL of bupivacaine with epinephrine mixed with 30 mg of ketorolac was injected in the knee and incisions. Compression and cold therapy were applied to the knee with a cuff (Knee CryoCuff, Aircast, Vista, California).

Anterior laxity was measured within 2 hours after surgery. The leg was inserted into a loading apparatus, which consisted of a calibration cage, an ankle and thigh support that restrained the movement of the leg under force, a pneumatic actuator that applied posterior and anterior forces to the shank, a load cell at the ankle support, a load cell at the pneumatic actuator, and 2 portable x-ray machines (model HF80H+, MinXray Inc., Northbrook, Illinois) (Figure 4).[42] The knee was centered in a calibration cage (model 10, Tilly Medical Products) and a goniometer was used to check that the knee was positioned in 25° of flexion. Straps secured the ankle and thigh supports. A strap secured the pneumatic actuator to the proximal tibia 12.5 cm distal to the joint line of the knee. The portable x-ray machines were positioned to expose anterior-posterior and lateral views of the knee (ie, biplanar) at a distance of 87 cm from their respective film planes. A lead shield was placed over the gonads, trunk, and thyroid to confine the radiation to the knee. Measurements were obtained of the subject with respect to the loading apparatus so that the position of the limb and x-ray machines could be re-established when the knee was tested at each monthly follow-up interval.

Surface electromyography was used to monitor muscle activation so that the exposures of the biplanar radiographs were made without muscle contraction across the knee. Surface electrodes (Bagnoli-8, DelSys, Boston, Massachusetts) were positioned over the vastus lateralis, long head of the biceps femoris, and medial head of the gastrocnemius muscles because contraction of each of these muscles affects anterior laxity.[7,8,28,44] Voluntary and involuntary activation of the muscle turned on a light that signaled both the examiner and subject that the muscles were active. The radiographs were exposed when the light was not on, ensuring that muscle contraction did not confound the measurement of anterior laxity.

On the day of surgery, 2 sets of biplanar radiographs were obtained without preconditioning the knee because preconditioning might have caused slippage, which would have underestimated the increase in anterior laxity and total slippage computed on future monthly follow-up intervals. The first set of biplanar radiographs was taken with no applied load and was used to define an anatomical coordinate system with use of a method that has been previously described.[35] The second set of biplanar radiographs was obtained with a 150-N anterior force transmitted at the knee to determine the anterior laxity and relative positions of the fixation devices and markers in the graft on the day of surgery from which the changes in anterior laxity

and slippage were computed at each monthly follow-up interval. The amount of force applied to the tibia to transmit a 150-N anterior force at the knee was calculated based on the weight and length of the subject's shank with use of a previously described technique.[42]

Rehabilitation was self-administered without a brace with the goal of walking without crutches by 1 to 2 weeks, jogging by 8 weeks, and returning to sport by 4 months.[15,19] After testing on the day of surgery, the subjects were instructed to bear weight as tolerated, perform extension and flexion exercises, and walk without crutches when they felt safe and comfortable. At 2 weeks, instructions were given to walk, swim, bike, and perform low-weight/high-repetition exercises on any exercise machine that the subject felt comfortable using. At 8 weeks, instructions were given to increase weight and resistance and begin running. Subjects were not seen by a physical therapist.

Return of function and clinical outcome measurements of extension and flexion, activity level (Tegner score), function (Lysholm score), and subjective satisfaction (IKDC score) were performed preoperatively and at 1 month (29 ± 3 days, mean ± standard deviation), 2 months (60 ± 4 days), 3 months (94 ± 4 days), 4 months (124 ± 8 days), 6 months (195 ± 24 days), and 12 months (372 ± 40 days) by an examiner (C.K.S.) different from the treating surgeon. The Lachman test (firm or soft end point) and pivot shift test (negative, glide [1+], clunk [2+], gross [3+]) were performed by the treating surgeon (S.M.H.) on the day of surgery and at each monthly interval (1, 2, 3, 4, 6, and 12 months). Knee extension and flexion were measured with a long goniometer, with a difference indicating that the treated knee lacked motion with respect to the contralateral knee. Subjects quantified their level of function, activity, and subjective satisfaction by noting the Lysholm, Tegner, and subjective IKDC scores.[1] The position of the limb with respect to the loading apparatus on the day of surgery was re-established with use of the recorded measurements. The knee was preconditioned by applying 10 cycles of a 90-N posterior force followed by a 150-N anterior force transmitted at the knee. Biplanar radiographs were exposed with a 150-N anterior force transmitted at the knee when the surface electromyography showed no muscle activation.

The RSA was used to compute the 3-dimensional coordinates of each marker from the biplanar radiographs with use of a previously described technique.[34] Each radiograph was scanned (Epson 1600, Epson America Inc, Long Beach, California) to provide a 300 dots per inch digital image. The 2-dimensional coordinates of each marker in the bone, fixation devices, and tibialis allograft were computed on each radiograph. The 3-dimensional coordinates of each marker were determined with use of custom software written with a commercial software package (Matlab version 6.0, The Mathworks Inc., Natick, Massachusetts).

To establish axes for computing anterior laxity, an anatomical coordinate system was constructed at the center of rotation of the knee.[35,42] The anterior direction (+x-axis) of the anatomical coordinate system was a line connecting 3 tantalum markers in the housing of the pneumatic actuator. The distal direction (+z-axis) was the cross-product of the x-axis and a line passing through the centers of the femoral condyles. The lateral direction (+y-axis) was the cross-product of the z-axis and x-axis. Because the choice of coordinate system has been demonstrated to affect the anterior laxity measurement, we used a center of rotation coordinate system, which has better repeatability than other coordinate systems.[35]

The increase in anterior laxity was the difference in the position of the tibia with respect to the femur along the anterior direction (ie, x-axis of the anatomical coordinate system) between the day of surgery and each monthly follow-up interval. Slippage was measured between the femur and femoral fixation, between the ACL graft and femoral fixation, between the tibia and tibial fixation, and between the ACL graft and tibial fixation. The total slippage for the construct was the sum of the slippage at these 4 interfaces. The increase in total slippage was the difference between the day of surgery and each monthly follow-up interval.[41,42]

Statistical Methods

To determine the specific monthly interval after which there were no significant increases in anterior laxity and total slippage, and to determine the month when the improvement in extension, flexion, and function (Lysholm score), activity level (Tegner score), and the subjective satisfaction (IKDC score) plateaued, the data were ranked using an n+1 ranking, and a Friedman test was performed with the independent variable being time of monthly follow-up interval (day of surgery, 1, 2, 3, 4, 6, and 12 months). The Friedman test was used with the n+1 rank because the distribution of the data was not normal and because 9 subjects missed one monthly follow-up interval, which precluded the use of a repeated measures analysis of variance. A Tukey-Kramer adjustment determined significant differences between monthly intervals.

RESULTS

Four of 23 subjects enrolled in the study were excluded for several technical reasons. Two subjects were excluded intraoperatively because the markers could not be installed in the femur and tibia as the result of a mechanical problem with the bead injector, and 2 subjects were excluded during the RSA examination on the day of surgery, one because the application of the 150-N force to the knee exceeded the comfort level of the subject and one because the loading apparatus malfunctioned. The study consisted of 19 subjects, 15 men and 4 women, with a mean age of 37 ± 10 years (range, 18-47 years). Anterior laxity was measured in all 19 subjects; however, slippage was measured in 16 subjects because one or more markers had dislodged from the graft after inserting the graft in the knee in 3 subjects.

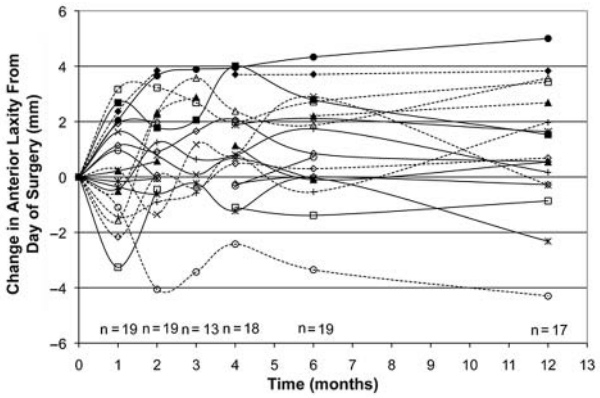

Figure 5. Line graph for each subject showing the change in anterior laxity between the day of surgery (0) and each monthly follow-up interval. The maximum increase in anterior laxity was at 6 months and averaged 1.1 ± 2 mm; however, there was no significant increase in anterior laxity from the day of surgery (P = .23).

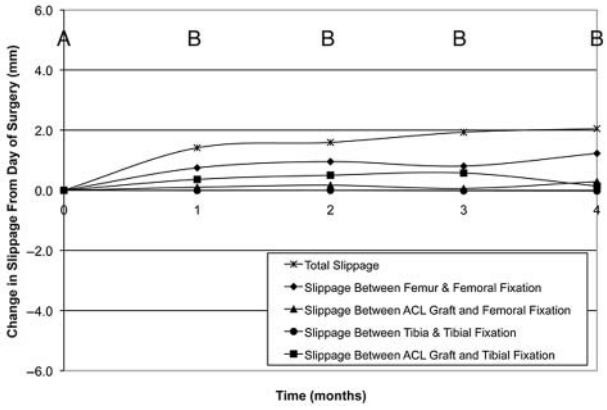

Figure 6. The change in total slippage, slippage between femur and femoral fixation, slippage between ACL graft and femoral fixation, slippage between tibia and tibial fixation, and slippage between ACL graft and tibial fixation between the day of surgery (0) and each monthly follow-up interval. Monthly follow-up intervals with dissimilar letters indicate that the difference between intervals was statistically significant ($P < .05$). Total slippage increased significantly between the day of surgery and 1 month (1.5 ± 0.8 mm) ($P < .05$), but did not increase thereafter. Slippage was not computed after 4 months because some markers in the soft tissue graft migrated. Marker migration was defined as a change in length between the 2 adjacent markers closest to the fixation device in each strand of graft between a monthly follow-up interval.[43]

Anterior laxity did not increase between the day of surgery and 1 year (P = .23). The maximum increase in anterior laxity was at 6 months, average 1.1 ± 1.9 mm, and was not different from the day of surgery (P = .23) (Figure 5). Total slippage increased between the day of surgery and 1 month (average, 1.5 ± 0.8 mm; $P < .05$) and did not increase between 1 and 4 months. The maximum total slippage was at 4 months and averaged 1.9 ± 1.4 mm (Figure 6).

Return to independent ambulation averaged 9 ± 7 days (range, 0-21) after surgery. Improvement in extension plateaued after 2 months ($P < .0001$). At 2 months the extension of the treated knee equaled that of the contralateral side in 13 of 19 subjects, and lacked 1° to 5° from the contralateral side in 6 of 19 subjects (Table 1). At 6 months the extension of the treated knee equaled that of the contralateral side in all subjects. Improvement in flexion plateaued after 2 months ($P < .0001$). At 2 months the flexion of treated knee equaled that of the contralateral side in 13 of 19 subjects, lacked 1° to 5° of flexion from that of the contralateral side in 1 of 19 subjects, lacked 6° to 10° from that of the contralateral side in 2 of 19 subjects, and lacked more than 10° of flexion from that of the contralateral side in 3 of 19 subjects. At 4 months the flexion of the treated knee equaled that of the contralateral in all subjects. Improvement in activity level (Tegner score) plateaued after 3 months ($P < .05$). At 3 months the activity level was within 1 value of the preinjury score in 69% (9 of 13 subjects), and at 12 months the Tegner score was equal to or better than the preinjury score in 89% (15 of 17 subjects) (Figure 7). Improvement in function (Lysholm score) plateaued after 4 months ($P < .002$). At 4 months, the Lysholm score was 84 or greater (ie, 84-94 indicates a "good" level of function) in 83% (15 of 18 subjects), and at 12 months the Lysholm score was 95 or greater (ie, excellent score) in 82% (14 of 17 subjects)[2] (Figure 8). Improvement in subjective satisfaction (IKDC score) plateaued after 6 months ($P < .02$). At 6 months the IKDC score was 81 or greater (ie, 81-90 indicates "good satisfaction") in 79% (15 of 19 subjects), and at 12 months the IKDC score was 90 or greater in 76% (13 of 17 subjects) (ie, excellent function)[13] (Figure 9). All the treated knees had a firm end point on Lachman test and 17 of 19 had no pivot shift at final follow-up (17 subjects at 12 months and 2 subjects at 6 months).

DISCUSSION

Although we have had a favorable experience with soft-tissue ACL grafts,[14,19,21] we became concerned when a previous RSA study with 1-year follow-up showed that metal interference screw fixation of an autogenous hamstring graft had a greater increase in anterior laxity and more total slippage than the bone-patellar tendon-bone graft. This concern prompted us to use RSA to evaluate our preferred soft tissue graft construct, which is a fresh-frozen, nonirradiated, nonchemically treated tibialis allograft fixed with cortical fixation devices that resist slippage. The present study showed that an early recovery of function does not have to cause a clinically important increase in anterior laxity and slippage after ACL reconstruction with a soft tissue graft.

Because we evaluated a specific graft construct and because we used a specific surgical technique, fixation methods, rehabilitation program, and study population,

TABLE 1
Difference in Extension and Flexion Between Treated and Contralateral Knee at Each Monthly Follow-Up Interval[a]

	Difference in Extension (deg)				Difference in Flexion (deg)			
Time	None	1–5	6–10	>10	None	1–5	6–10	>10
Day of Surgery	19 of 19	0 of 19	0 of 19	0 of 19	19 of 19	0 of 19	0 of 19	0 of 19
1 Month	3 of 19	7 of 19	7 of 19	2 of 19	3 of 19	2 of 19	2 of 19	12 of 19
2 Months	13 of 19	6 of 19	0 of 19	0 of 19	13 of 19	1 of 19	2 of 19	3 of 19
3 Months	13 of 13	0 of 13	0 of 13	0 of 19	11 of 13	0 of 13	1 of 13	1 of 13
4 Months	16 of 18	2 of 18	0 of 18	0 of 19	18 of 18	0 of 18	0 of 18	0 of 18
6 Months	19 of 19	0 of 19	0 of 19	0 of 19	19 of 19	0 of 19	0 of 19	0 of 19
12 Months	17 of 17	0 of 17	0 of 17	0 of 19	17 of 17	0 of 17	0 of 17	0 of 17

[a]The value (y of x) at each monthly follow-up interval indicates the number of subjects (y) with a specified difference in extension and flexion out of the number of subjects evaluated (x). A difference indicates the treated knee lacked motion with respect to the contralateral knee. Improvement in extension and flexion plateaued after 2 months ($P < .0001$, $P < .02$, respectively).

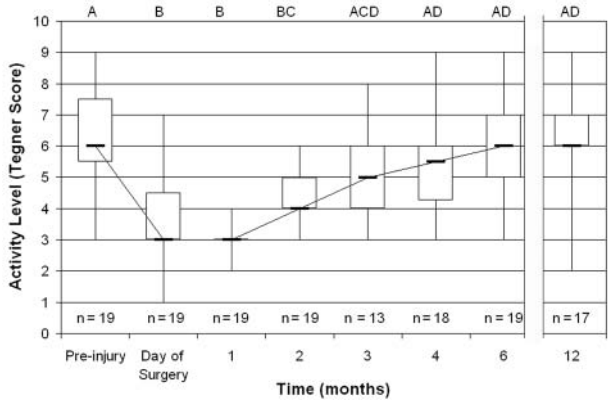

Figure 7. The distribution of activity level (Tegner score) at each monthly follow-up interval. The line across the middle of the box identifies the median value. The ends of the box identify the 25th and 75th quartiles. The 2 lines that extend from the end of each box identify the minimum and maximum value. Monthly follow-up intervals with dissimilar letters indicate that the difference between intervals was significantly different at a minimum value of $P < .05$. The Tegner score decreased significantly from a median score of 6 preinjury to 3 on the day of surgery, and increased significantly from the day of surgery to 5 at 3 months ($P < .05$); however, the improvement in the activity level plateaued between 3 and 12 months.

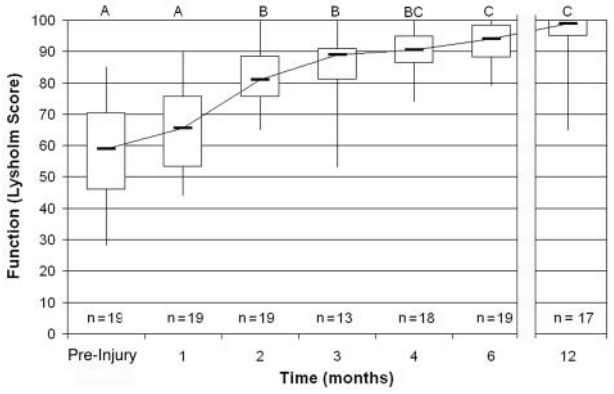

Figure 8. The distribution of function (Lysholm score) at each monthly follow-up interval. The Lysholm score increased significantly from a median of 59 on the day of surgery to 91 at 4 months ($P < .002$); however, the improvement in function plateaued between 4 and 12 months.

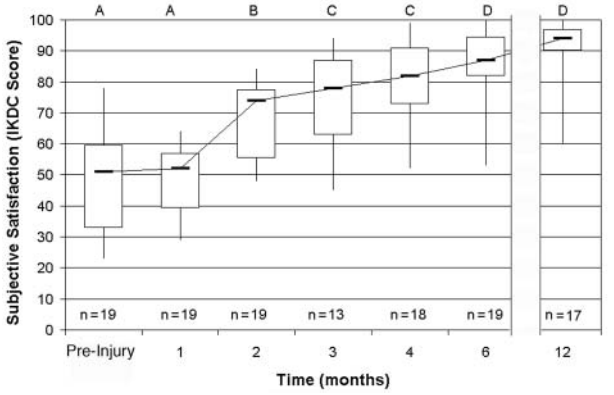

Figure 9. The distribution of subjective satisfaction (International Knee Documentation Committee [IKDC] score) at each monthly follow-up interval. The IKDC score increased significantly from a median of 51 on the day of surgery to 87 at 6 months ($P < .02$); however, the improvement in subjective satisfaction plateaued between 6 and 12 months.

any generalization of our findings should be made cautiously. We investigated an aseptically harvested, fresh-frozen, nonirradiated, nonchemically treated single-looped tibialis allograft from a tissue bank accredited by the American Association of Tissue Banks, which has a lower rate of traumatic rupture than an irradiated and chemically treated allograft.[25,33] We used a transtibial technique for placing the femoral and tibial tunnels that is designed to minimize graft lengthening by avoiding roof impingement, avoiding PCL impingement, and matching the tension pattern of the intact ACL during passive motion. Knees that have no impingement and a normal tension pattern during passive motion

have less instability and better return of flexion and extension than nonanatomical techniques that place the graft with roof impingement, PCL impingement, and an abnormal tension pattern.[14,20] We specifically chose fixation devices that grip cortical bone, which are more slippage-resistant in vivo and in vitro than interference screw fixation.[3,27,39,47] An autogenous bone dowel harvested from the tibial tunnel was reinserted into the tibial tunnel to increase stiffness and promote circumferential tendon tunnel healing.[18,31,39] We used a brace-free, self-administered rehabilitation program that one study has shown to be more effective at achieving acceptable knee range of motion at 3 months than a standard physical therapy-based program.[10] We studied older (mean, 37 years), predominantly male (15 of 19 subjects), recreational athletes, each factor of which has a lower rate of reinjury than younger, female, and more competitive athletes.[37] Finally, our results are preliminary as they represent the anterior laxity, slippage, and return of function during the first year. With these restrictions in mind, further study is needed to determine the increase in anterior laxity and slippage when a different graft construct, surgical technique, fixation method, rehabilitation program, and study population from the present study are used.

A power analysis to determine the number of subjects required to detect a clinically important increase in anterior laxity of 2 mm during the first year after surgery was not possible before the present study because we did not have any pilot or published data describing the in vivo variability of the anterior laxity measurement with our knee testing system. In the present study, the standard deviation of the anterior laxity was 2.0 mm for the maximum increase in anterior laxity of 1.1 mm at 6 months. With use of the 2.0 mm standard deviation, 19 subjects, a level of significance of .05, and the detection of a clinically important increase in anterior laxity of 2.0 mm for calculation, the power of the present study was 0.98. Hence, the probability of making a type II or beta error and wrongly concluding there was no clinically important increase in anterior laxity over time is unlikely.

One important finding of the present study is that an increase in anterior laxity during the first year after ACL reconstruction is not inevitable. Our average increase in anterior laxity of 1.0 ± 2.3 mm at 1 year was clinically unimportant and less than the 8.6 mm increase in anterior laxity of the autogenous 4-strand semitendinosus and gracilis hamstring graft and the 6-mm increase in anterior laxity of the bone-patellar tendon-bone graft at 1 year reported in the previous study.[24]

One factor that we believe contributed to the small increase in anterior laxity in the present study was the use of specific arthroscopic and radiographic checkpoints to confirm the avoidance of roof and PCL impingement,[16] which are known to cause graft elongation and failure.[14,20,38] The use of these checkpoints or other methods for avoiding roof and PCL impingement were not described in the study by Khan et al,[24] which suggests that either roof or PCL impingement might have been a factor in their increase in anterior laxity. A second factor that we believe contributed to the small increase in anterior laxity was the use of cortical fixation devices that are slippage-resistant, which resulted in less total slippage (2.1 mm at 4 months) than the total slippage of interference screw fixation of the hamstring graft (5 mm at 4 months) in the previous study. A third factor that we believe contributed to the small increase in anterior laxity was the use of a brace-free, self-administered aggressive rehabilitation that might have stress-loaded the ACL graft more and maintained the stiffness of the ACL graft better than the use of the more conservative rehabilitation in the previous study.

The small amount of slippage (1.5 ± 0.8 mm) limited to the first month implies early tendon-tunnel healing and adequate fixation with cortical devices that are slippage-resistant. The small amount of slippage as a result of early tendon-tunnel healing is consistent with the rapid rate of tendon-tunnel healing shown in in vivo animal studies.[39,46,47] In an ovine model, tendon-tunnel healing carried most of the tensile load in the graft (85%) by 4 weeks.[46] In another ovine model, all grafts failed at the tunnel entrance and not within the tunnel at 6 weeks, implying adequate tendon-tunnel healing.[47] Finally, a third ovine model showed that the type of fixation device determined whether the strength and stiffness of a tendon in a bone tunnel increases or decreases after implantation with cortical fixation devices that are slippage-resistant, providing greater stiffness and strength than an interference screw.[39]

The observation that the increase in anterior laxity was less than the total slippage between the 1 month follow-up interval and the date of surgery seems paradoxical. One explanation for this paradox is that the increase in anterior laxity at 1 month caused by lengthening of the graft construct from slippage was compensated for by a decrease in anterior laxity from stiffening of the graft construct from tendon-tunnel healing.[6,26,40] In vivo studies have shown that tendon-tunnel healing affects the stiffness of the graft construct, but whether the stiffness increases or deteriorates depends on the type of fixation device. The stiffness of the construct increases 136% at 1 month when cortical fixation is used on the tibia, and the stiffness of the construct increases 52% at 6 months when cross-pin fixation is used on the femur and tibia. In contrast, the stiffness of the construct deteriorates 40% at 1 month when an interference screw fixation is used in the tibia and deteriorates 67% when an interference screw fixation is used in the femur and tibia. The deterioration in stiffness is believed to be due to the interference screw inhibiting contact between the tendon and tunnel wall, reducing the contact area between the tendon and tunnel wall, preventing ingrowth of blood vessels, and altering the mechanism of graft-tunnel attachment to a chondral insertion that might be less effective during the early healing process.[39,47] The effect of tendon-tunnel healing on anterior laxity is substantial, as doubling the stiffness of the construct decreases the anterior laxity 50%, which can compensate for the increase in anterior laxity when total slippage is small.

A second explanation for the paradox that the increase in anterior laxity was less than the total slippage is that a portion of slippage between the femoral fixation device

TABLE 2
Clinical Findings at 12 Months[a]

Subject	Increase in Anterior Laxity (mm)	Lachman Test	Pivot Shift Test	Difference in Extension (deg)	Difference in Flexion (deg)	Lysholm	Tegner	IKDC
1	3.8	Firm	Negative	0	0	90	6	90
2	0.5	Firm	Negative	0	0	100	7	95
3	2.7	Firm	Negative	0	0	65	2	60
4	-0.3	Firm	Negative	0	0	95	9	92
5	1.6	Firm	Negative	0	0	99	6	91
6	1.5	Firm	Negative	0	0	100	9	95
7	2.0	Firm	+1	0	0	96	6	86
8	0.5	Firm	Negative	0	0	96	7	98
9	3.6	Firm	Negative	0	0	99	9	94
10	0.7	Firm	Negative	0	0	95	6	99
11	−0.3	Firm	Negative	0	0	88	6	84
12	3.4	Firm	Negative	0	0	100	6	95
13	0.2	Firm	Negative	0	0	100	7	100
14	−2.3	Firm	Negative	0	0	99	7	97
15	5.0	Firm	+1	0	0	100	9	99
16	−0.9	Firm	Negative	0	0	99	5	93
17[b]	0.7	Firm	Negative	0	0	95	6	95
18[b]	−0.1	Firm	Negative	0	0	86	6	82
19	−4.3	Firm	Negative	0	0	78	3	64

[a]Difference in extension and flexion is treated knee minus contralateral knee. IKDC, International Knee Documentation Committee.
[b]Data for these 2 patients are from 6-month, not 12-month follow-up.

and the edge of the bone tunnel might have been delayed until after early tendon-tunnel healing and therefore did not affect anterior laxity. This form of "delayed" slippage can occur from resorption of bone under the femoral fixation. Tendon-tunnel healing causes a natural decline in the contact force between the femoral fixation device,[46] and the resulting stress-shielding might cause resorption of the edge of the bone tunnel under the lever arm of the femoral fixation device, allowing the device to migrate closer to the joint line. If the migration of the device occurs after the tendon has healed so that the tendon no longer slips in the bone tunnel, then the migration of the device would not increase the anterior laxity. An in vivo study showed that tendon-tunnel healing carries 85% of the tensile load applied to the fixation device at 1 month,[46] which provides evidence that there is a substantial decline in contact force by 1 month. In the present study, the amount of slippage at 1 month between the femoral fixation device and femur was small (0.7 mm), which means that less than 1 mm of delayed resorption of the edge of the bone tunnel is needed to explain the migration of the femoral fixation device and why the migration might not have affected the anterior laxity.

In vitro studies are commonly used to test the structural properties of fixation methods, which are then used to predict the safety of their performance in vivo. However, the present and previous in vivo studies suggest that the results from in vitro studies of fixation devices should be interpreted cautiously.[39,47] For example, several in vitro studies have reported that interference screw fixation of a soft tissue graft slips minimally under cyclic load,[3,27,32] whereas the previous in vivo study showed a total slippage of 5 mm at 4 months, which is greater than the in vitro prediction[24] and similar to slippage and deterioration of stiffness seen with interference screw fixation in 2 studies in an ovine model.[39,47] Another in vitro study using porcine tibia reported that the femoral fixation device used in the present study failed in all specimens,[4] whereas no femoral fixation device failed in the present study. These inconsistencies between in vivo and in vitro performance of fixation devices underscore the importance of clinical evaluation of fixation devices using experimental methods such as RSA.

In the present study, 4 of 19 subjects (21%) had a 3 mm or greater increase in anterior laxity from the day of surgery at 12-month follow-up (range, 3.4-5.0 mm) (Table 2). However, the increase in anterior laxity was measured with respect to the treated knee on the day of surgery and not with respect to the anterior laxity of the contralateral normal knee. An anterior laxity comparison between the treated and contralateral knee was not possible in the present study and has not been performed in other studies because ethical concerns do not permit the insertion of tantalum markers in a normal knee.[9,34] We found no evidence that the increase in anterior laxity of 3 mm or greater from the day of surgery in these 4 subjects resulted in instability or a compromised clinical outcome, because each had a stable Lachman test, a median activity level (Tegner score) of 7, an average function (Lysholm score) of 97, and an average subjective satisfaction (IKDC score) of 94.5. One explanation for the acceptable subjective, functional, and objective outcomes with a 3 mm or greater increase in anterior laxity from the day of surgery is that the anterior laxity in the treated knee was less than the contralateral knee on the day of surgery.

In conclusion, RSA provided an accurate in vivo method that was not susceptible to detection bias for determining the increase in anterior laxity and slippage at the sites of fixation at specific monthly follow-up intervals after ACL reconstruction. The technique used in the present study showed that an early recovery of motion and return of function did not cause a clinically important increase in anterior laxity in the first year after ACL reconstruction with a soft tissue graft. We believe the avoidance of an increase in anterior laxity was related to the use of a transtibial technique designed to place the femoral and tibial tunnels without roof and PCL impingement and to restore the normal tension pattern of the intact ACL during passive motion, the use of cortical fixation devices designed to resist slippage, the use of a bone dowel in the tibial tunnel designed to promote tendon-tunnel healing, the use of an aseptically harvested, fresh-frozen tibialis allograft that was not irradiated or chemically processed, and the use of a self-administered rehabilitation program designed to encourage an early return of motion and function. In our study, the small amount of slippage limited to the first month implies early tendon-tunnel healing, which is consistent with the use of cortical fixation devices that are slippage-resistant in in vivo studies in the ovine model.[39,46,47]

ACKNOWLEDGMENT

We thank the operating room, recovery room, and imaging staff at Methodist Hospital for assisting us in this study.

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