Contact Pressure and Tension in Anterior Cruciate Ligament Grafts Subjected to Roof Impingement during Passive Extension

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Summary: Contact between an anterior cruciate ligament graft and the intercondylar roof has been termed roof impingement. Grafts with impingement sustain permanent damage, and if the injury is extensive enough, then the graft may fail, causing recurrent instability. This study evaluated two mechanical factors that could be responsible for the graft injury associated with roof impingement: an increase in graft tension or elevated pressures between the graft and the roof, or both. An anterior cruciate ligament reconstruction was performed using an Achilles tendon graft in five fresh-frozen cadaveric knees. Using a six-degree-of-freedom load application system, the anterior displacement of the knee with the native anterior cruciate ligament was restored in the reconstructed knee at a flexion angle of 30° and with an anterior force of 200 N applied. Pressure between the graft and intercondylar roof, graft tension, and flexion angle were measured during passive knee extension for three tibial tunnel placements (anterior, center, and posterior). Intercondylar roof impingement increased the contact pressure between the graft and the roof but had no significant effect on graft tension. Therefore, during passive knee extension, the contact pressure between the anterior cruciate ligament graft and the intercondylar roof is a more likely cause of graft damage than increased graft tension.

Impingement of the anterior cruciate ligament graft against the intercondylar roof causes effusions, knee extension deficits, recurrent instability, or anterior knee pain (8,12,14,16,20,25). Recently reported incidences of impingement-related complications range from 2 to 6% of the patients treated (14,16,20,25). Although roof impingement complications are avoidable, they continue to occur, in part, because the mechanical factors by which impingement initiates graft injury remain unknown.

Two mechanical factors that may be responsible for graft injury are suggested from the patterns of damage observed during second-look arthroscopy of impinged anterior cruciate ligament grafts (24). Ruptured graft fibers may result from abrasion or compression caused by elevated pressure between the graft and the intercondylar roof. Alternatively, elongation of the graft without fiber rupture might be caused by elevated tension in the graft as it angulates around the intercondylar roof.

To assess both mechanical factors as potential causes of graft injury, a measure of the effect of roof impingement on each factor is required. Both graft tension (4,21,22) and contact pressure between an anterior cruciate ligament graft and the intercondylar roof (4) have been measured previously during passive knee extension. However: the effect of roof impingement on each of the two factors has not been investigated.

The purpose of this study was to test the two hypotheses that, during passive knee extension, intercondylar roof impingement causes (a) an increase in contact pressure between the graft and the roof and (b) an increase in graft tension. The results of this study are clinically relevant in that, if elevated contact pressures are observed, then graft position, graft diameter, and notch volume will have to be tailored to prevent contact between the graft and the intercondylar roof. If roof impingement causes elevated graft tension and impingement cannot be prevented, then high-strength graft material and fixation methods will be needed to avoid graft failure.

METHODS

Five fresh-frozen knee specimens obtained from five men, ranging in age from 49 to 82 years (mean: 67 years) at the time of death, were tested in a computer-controlled load application system designed and built in our research laboratory (2). The load applica-
The anterior-posterior laxity of the knee was measured at 30˚ of flexion by applying three cycles of anterior-posterior loads of 200 N to the knee, which was preconditioned by 50 N steps to incrementally increase the load from 0 to 200 N of anterior force, decrease the load to 0, increase the load to 200 N of posterior force, and decrease it again to 0. This complete loading and unloading sequence was considered one cycle and required approximately 16 seconds. Six complete loading cycles were applied at flexion angles of 0, 30, 60, 90, and 120˚. Because the goal of the preconditioning was to subject the native anterior cruciate ligament to loads that the graft would incur during laxity testing without overloading the graft, an upper load limit had to be defined. The preconditioning limit of 200 N was obtained from a pilot study that determined that an anterior load of 200 N would ensure anterior displacement of the tibia.

Using the load application system, the knee was preconditioned by 50 N steps to incrementally increase the load from 0 to 200 N of anterior force, decrease the load to 0, increase the load to 200 N of posterior force, and decrease it again to 0. This complete loading and unloading sequence was considered one cycle and required approximately 16 seconds. Six complete loading cycles were applied at flexion angles of 0, 30, 60, 90, and 120˚. Because the goal of the preconditioning was to subject the native anterior cruciate ligament to loads that the graft would incur during laxity testing without overloading the graft, an upper load limit had to be defined. The preconditioning limit of 200 N was obtained from a pilot study that determined that an anterior load of 200 N would ensure anterior displacement of the tibia.

The anterior-posterior laxity of the knee was measured at 30˚ of flexion by applying three cycles of anterior-posterior loads of 200 N (1, 9, 22). The laxity at the final cycle was chosen as the reference laxity of the joint. Thirty degrees of flexion was chosen for laxity testing because it provided a flexion angle that had minimal joint stiffness and did not cause graft-roof impingement once the anterior cruciate ligament had been excised and replaced. After the knee specimen was tested with the native anterior cruciate ligament, the knee was maximally extended (14). Proper placement of the guide wire was confirmed with a lateral roentgenogram taken with the knee in the maximally extended position. A 12 mm diameter cannulated reamer was used to drill over the guide wire, and a custom drill guide was used to drill a second 12 mm diameter tunnel centered 12 mm anterior to the center of the first tunnel. This created a tibial tunnel that was 12 mm in medial-lateral dimension and 21-25 mm in anterior-posterior dimension, depending on the angle that the reamer entered the articular surface of the tibia.

Three polytetrafluoroethylene (Teflon; DuPont, Wilmington, DE, U.S.A.) bushings, made to fit into the oversized tibial tunnel, positioned the graft in either the posterior, center, or anterior position (Fig. 1). Each bushing could be affixed within the tibial tunnel using small wedges and a fixation pin between the bushing and the bone that were all located on the distal outlet of the tunnel. Teflon minimized friction as the anterior cruciate ligament graft passed through a 9 mm diameter hole in the bushing to an external connection. The posterior placement coincided with the initial placement of the tibial guide wire. The center position was 3 mm anterior to the posterior position, and the anterior position was 3 mm anterior to the center position.

To estimate whether impingement with the intercondylar roof would occur when the anterior cruciate ligament graft was installed, a 9 mm metal rod was inserted through each of the three tunnel positions with the knee in maximal extension. If the rod did not pass into the intercondylar notch, then impingement was likely to occur when the anterior cruciate ligament graft was in place. For all five specimens, the rod indicated that impingement between the graft and the roof would occur with both the center and anterior tunnel.
The femoral tunnel location was determined by inserting a 9 mm endoscopic femoral aimer (Arthrotek) through the tibial tunnel without a Teflon bushing, with the knee in approximately 70° of flexion. A guide wire was drilled through the femoral aimer so that the guide wire entered the femoral condyle 6 mm distal to the proximal edge of the intercondylar roof at either 11 or 1 o’clock for the right or left knee, respectively. A 9 mm diameter endoscopic cannulated drill was used to create a femoral tunnel from inside the intercondylar notch to a depth of 10 mm. The femoral tunnel was completed by drilling from the lateral femur using a 12 mm diameter cannulated drill until it met the 9 mm tunnel.

The Achilles tendon was harvested from each specimen and used as an anterior cruciate ligament graft. The graft was prepared by shaping the calcaneus into a truncated cone that was 30 mm in length and tapered from 9 mm in diameter at the tendon insertion of the bone plug to 11 mm at the opposite end. The width of the tendon was trimmed until it fit snugly inside a 9 mm cylinder. The length ranged from 15 to 20 cm. The tendon end of the graft was passed through the femoral tunnel into the intercondylar notch and through one of the Teflon bushings in the tibial tunnel. The bone plug was cemented within the 12 mm diameter segment of the femoral tunnel using polymethylmethacrylate (G. C. America, Chicago, IL, USA.). This fixation ensured that graft slippage would not occur under the graft tensile forces that would be encountered.

A miniature pressure transducer (Precision Measurement, Ann Arbor, MI, U.S.A.) was used to measure pressure between the intercondylar roof and the graft (Fig. 1). The transducer was inserted through a tunnel that began at the apex of the intercondylar notch, at the junction of the intercondylar roof and articular cartilage, and exited the anterior femur just medial to the trochlear groove to avoid the patellofemoral joint. Threaded into a custom-made insertion screw with internal threads, the pressure transducer was advanced into the tunnel until the sensor face was flush with the intercondylar roof. A small straightedge was used to align the sensor face with the natural contour of the intercondylar roof. The reconstructed specimen was reinstalled in the load application system; the graft was passed through one of the Teflon bushings (selected at random) and attached to a tension load cell (A. L. Design, Buffalo, NY, U.S.A.) using a freeze clamp cooled with liquid nitrogen (Fig. 1). The reconstructed knee was preconditioned using the same protocol that had been used for the knee with the native anterior cruciate ligament, except the maximum load was changed to 250 N. A preconditioning load of 250 N was determined in a pilot study to provide a tension force to the graft that would be greater than the tension it would sustain during the passive testing sequence. The pre-tension in the graft was adjusted using a turnbuckle until the anterior laxity matched that of the knee with the native anterior cruciate ligament at 30∞ of flexion with a 200 N anterior force. The knee was cycled 10 times from hyperextension (flexion angle with 10 Nm extension moment) to 120° of flexion; then, if required, a final adjustment in the graft pre-tension was made to match the laxity. Knee flexion angle, contact pressure between the graft and the roof, and graft tension were measured at 1° increments as the knee was passively moved from hyperextension to 120° of flexion. After five cycles, the anterior laxity of the knee was remeasured at 30° of flexion with a 200 N anterior load. This testing protocol was repeated for the two remaining tibial tunnel locations.

To examine the effect of intercondylar roof impingement on the contact pressure between the graft and the roof, the data from tibial tunnel placements that resulted in roof impingement were examined. The onset of intercondylar roof impingement was defined as the flexion angle at which the slope of the contact pressure-flexion angle curve began to increase from a baseline pressure of 0 kPa. The slope for a specific flexion angle was determined using a linear estimate through the pressure data for the three flexion angles both preceding and following the flexion angle of interest. A paired t test was used to determine if the slope before and after impingement was significantly different (p < 0.05).

A two-factor repeated measures analysis of variance (ANOVA) was used to test the effect of impingement on graft tension. The two factors were tunnel placement and flexion angles at which impingement occurred. Because the mean flexion angle at which impingement occurred ranged from 5.2 ± 2.5° of flexion for the anterior tunnel to 3.6 ± 1.0° of hyperextension for the posterior tunnel, 11 levels were used for flexion angle (6, 5, 4, 3, 2, 1, 0, -1, -2, -3, and -4°). The effect of impingement on graft tension was dependent on the

FIG. 2. Contact pressure between the anterior cruciate ligament graft and the intercondylar roof for a typical specimen using three tibial tunnel placements. Intercondylar roof impingement was defined as the flexion angle at which the slope of the curve began to increase from a baseline pressure of 0 kPa. For tunnel placements that exhibited roof impingement, the slope of this curve was significantly greater after roof impingement than before (p = 0.009).
ability to detect a significant effect from the tunnel-flexion angle interaction. A significant tunnel-flexion angle interaction (p < 0.05) would indicate that roof impingement altered the graft tension-flexion angle relationship between tunnel placements. However, this effect would be valid only if the flexion angles at which impingement occurred were significantly different between the three tunnel placements. Therefore, before the analysis could be performed it was necessary to perform a single-factor repeated measures ANOVA to test the effect of tunnel placement on the flexion angle at which impingement occurred. A significant tunnel placement effect (p < 0.05) on the flexion angle at which impingement occurred would confirm the validity of the statistical test results using the tunnel-flexion angle interaction.

Since roof impingement did not occur in the posterior tunnel on two specimens or in the center tunnel on one of these same specimens, it was necessary to address these missing observations for the statistical analysis. Rather than treat these as missing observations, the maximum hyperextension angle for the respective specimen was used as a conservative approximation of the flexion angle at which impingement occurred.

### RESULTS

For each specimen, contact pressure and graft tension data for each of the five flexion-extension cycles at each tunnel placement were plotted and inspected for adequate preconditioning and repeatability. The data indicated that preconditioning had been successfully achieved prior to data collection, and all five cycles of data exhibited repeatable values.

The onset of roof impingement was identified by a significant increase in the slope of the contact pressure-flexion angle curve (p = 0.009). During the passive testing sequence, all five specimens exhibited roof impingement with an anterior tibial tunnel placement. The center tibial tunnel resulted in impingement in four of the five specimens, and three of the five specimens had roof impingement using the posterior tibial tunnel. For each specimen with tibial tunnel placements that exhibited impingement, the contact pressure between the graft and the roof began to increase at the flexion angle of impingement and continued increasing until reaching a peak pressure at the hyperextension angle corresponding to the maximum extension moment of 10 Nm (Fig. 2).

Graft tension increased during terminal extension for each of the three tibial tunnel placements (Fig. 3). However, a lack of significance in the tunnel-flexion angle interaction (p = 0.415) indicated that the slopes of the graft tension-flexion angle curves remained parallel between tunnel placements regardless of the flexion angle at which impingement occurred. Tibial tunnel placement significantly affected the flexion angle at which impingement occurred (p = 0.005), thereby confirming the use of the tunnel-flexion angle interaction as a valid statistical test.

### DISCUSSION

The first goal of this study was to investigate the effect of intercondylar roof impingement on the contact pressure between the graft and the roof, while the second goal was to investigate its effect on graft tension. To address these goals, a cadaver study was performed that quantified the effect of roof impingement on both contact pressure between the graft and the roof and graft tension during passive knee extension. Although contact pressure between the graft and the roof increased, there was no significant increase in graft tension. However, as with any study involving an in vitro investigation, certain methodological issues should be addressed because of their possible influence on results.

Although the measured tension in the anterior cruciate ligament graft may not have been the actual tension

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FIG. 3. Mean graft tension (and 1 SE) during terminal passive extension and hyperextension for five specimens using three tibial tunnel placements, Tunnel-flexion angle interaction (p = 0.415) had no significant effect on the graft tension.
nor the tension that occurs in vivo, it was differences in tension that were of interest in this study. The material and geometric properties of the graft and friction all affect the tension measured in the graft. Although various graft materials (both biologic and synthetic) have been utilized in previous research, the Achilles tendon was chosen as the replacement graft for this study because it provided both a sufficient length for attachment to an externally mounted load cell and a secure method of attachment within the femoral tunnel by utilizing a segment of its calcaneal insertion. While the 9 mm diameter of the graft was comparable with the typically used patellar tendon graft, the 15-20 cm length was longer, and therefore less stiff than the 9-11 cm patellar tendon graft. Furthermore, passage of the graft through the Teflon bushing reduced friction. A pilot study was performed to evaluate the frictional loss associated with the Teflon bushing. For the maximum graft tension that was observed during passive extension, 2.50 N, the corresponding frictional loss was 4%, or 10 N. This frictional loss was for a 40˚ angle of wrap; for 0˚ of wrap, there was no associated frictional loss. Since the conclusions were based on differences in graft tension, the interpretation of the results would not be affected if the measured graft tension was either less than the actual tension due to friction or different than in vivo tension.

Positioning of the pressure transducer was recognized to affect the measurement of contact pressure between the graft and intercondylar roof. The position chosen was believed to represent the first contact point between the graft and the intercondylar roof. The maximum pressure may not have been recorded because the pressure sensor had a contact area of only 11 mm 2 and did not measure the pressure of the entire contact area of the graft. However, the maximum pressure was not of interest in this study. Since the location of the pressure sensor was not altered between the three tibial tunnel placements and comparisons of pressure measurements were made between tunnel placements within a knee, the observation that roof impingement caused an increase in contact pressure is valid.

The pressure sensor was used to detect the flexion angle at which roof impingement occurred and was subject to the same effect from placement of the sensor as the contact pressure. Again, it was the change in the flexion angle at which impingement occurred between tibial tunnel placements that was of concern, not the absolute flexion angle. Therefore: for the same reasoning as the contact pressure, the result that tibial tunnel placement affected the flexion angle at which impingement occurred was valid.

Although the goal of anterior cruciate ligament reconstruction is to reproduce the biomechanics of the normal knee, an anterior cruciate ligament graft may not be capable of exact restoration (17,21). However, some criteria must be established as a means of minimizing the experimental variability associated with the replacement procedure. For this study, as well as others (5,22,23), the criterion was that the laxity of the reconstructed knee match the laxity of the knee with the native anterior cruciate ligament at a given flexion angle with a specified anterior load. Using a protocol that ensured that both sufficient preconditioning and appropriate pre-tensioning had been applied for each tunnel placement, the laxity of the reconstructed knee prior to passive testing was restored to within an average of 0.3 ± 0.1 mm of the laxity of the knee with the native anterior cruciate ligament. At the completion of the passive testing sequence, the laxity had increased by an average of 0.2 ± 0.1 mm. Although the protocol was successful in matching and maintaining the laxity of the knee with the native anterior cruciate ligament, the effect of the variable pre-tension between tunnel placements on the resultant graft tension must be considered.

The sensitivity of joint kinematics to graft pre-tensioning has been well documented (1,7,9,21,22). Graft pretensioning results in a posterior tibial subluxation that was observed in this study and has been reported by others (1,18,21). The average graft pre-tension required to restore the intact laxity at 30˚ of flexion was 16 N (range: 8-31 N) for each of the three tibial tunnel placements. This range of graft pre-tensions is comparable with the graft pre-tensions required in previous studies that utilized similar laxity restoration methods (6,21). The variation in pre-tension between each of the three tunnel placements for any single specimen ranged from 0 to 4 N. Although the pre-tension magnitude was consistent with previous work and the variability in pre-tension between tunnel placements was small, the effect of roof impingement on graft tension was governed by the pre-tension method utilized in this study. Another method of pre-tensioning may have produced different results than those obtained. Since the pre-tensioning methodology was based on the restoration of the laxity of the knee with the native anterior cruciate ligament (and this is a goal of anterior cruciate ligament reconstruction), it was considered the most appropriate pre-tensioning method for this study.

Although the results from this study have identified contact pressure between the graft and the roof as a possible cause of impingement-related injury during passive knee extension, the relationship between contact pressure and the graft injury process remains unknown. The possibility that graft injury from impingement may begin during the passive motion phase of repair has been suggested (5,10,20,23). Cadaver studies have reported abrasive wear to biologic grafts due to impingement against a bone tunnel during passive knee motion (5,10). Therefore,
damage to the anterior cruciate ligament graft can occur due to impingement during passive knee motion, at least in vitro. However, such a correlation between contact pressure and graft damage becomes difficult to make for the roof impingement-related injuries observed in vivo. Since the in vivo observations represent only a sample in time of the total graft injury process (24), an exact relationship between the contact pressure and the injury process cannot be confirmed.

The physical appearances of certain injury patterns that were previously reported resembled an excessive tensioning injury (24). However, no increase in graft tension during roof impingement was observed in this study. Although a contribution from impingement to the increase in graft tension did not occur when the graft was pre-tensioned to match the laxity of the knee with the native anterior cruciate ligament at 30° of flexion, this does not exclude the possibility that graft injury due to excessive graft tension might occur as a result of over-tensioning during graft fixation. Also, another factor that may affect graft tension, active knee extension, was not examined in this study.

In summary, roof impingement caused an increase in the contact pressure on the graft at the point of contact with the intercondylar roof; however, there was no associated increase in graft tension. Therefore, during passive knee extension, contact pressure between the anterior cruciate ligament graft and the intercondylar roof is one possible cause of graft injury.

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