We compared the ability of three different posterior cruciate ligament (PCL) reconstructions to restore normal anteroposterior laxity to the knee from 0 to 130° of knee flexion. Cadaver knees were tested intact, after PCL rupture or after bone-patellar tendon-bone grafting. Grafts were performed isometrically or with a single bundle representing the anatomical anterior PCL fibre bulk (aPC) or with a double bundle that added the posterior PCL fibre bulk (pPC). The grafts were tensioned to restore normal knee laxity at 60° of flexion, except for the pPC which was tensioned at 130°.

The isometric graft led to overconstraint as the knee extended resulting in high graft tension in extension and excess laxity in flexion. The aPC graft matched normal laxity from 0 to 60° of flexion but was lax from 90 to 130° of flexion. Only the double-bundled graft could restore normal knee laxity across the full range of flexion.

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Recently, there has been increasing interest in reconstruction of the posterior cruciate ligament. This is stronger than the anterior cruciate ligament (ACL), 1-3 but its rupture does not always cause disability. 4-8 This has led to some reluctance to undertake reconstruction, the results of which have been variable and often poor. 8,9 The initial lack of disability may relate to the function of the ligament, which has been shown not to be the primary restraint to posterior tibial subluxation when the knee is near full extension. 10 There is some evidence to indicate that a PCL-deficient knee undergoes progressive degenerative changes. 11-14

An ‘isometric’ PCL graft placement is often the aim. It has been assumed that this prevents overloading the graft as a result of the changes in length on movement, although the restoration of acceptable anteroposterior (AP) tibial laxity may not be possible. 15

The fibres of the PCL fan out from a small tibial attachment to a femoral attachment that is typically 30 mm across16 (Fig. 1). Since the anterior and posterior fibres are aligned in different directions it has been suggested that they may have different functions. It has been shown that the anterior fibres (aPC) have significantly greater material properties than the posterior fibres (pPC). 17 The aPC is tensed in the mid-range of flexion and the pPC acts in full flexion 10 (Fig. 2). Consequently, there have been attempts to reproduce the normal anatomy of the PCL using a two-bundled reconstruction. We have therefore tested the hypothesis that this more anatomical reconstruction restores normal AP laxity better than isometric reconstruction.

Materials and Methods

Eight cadaver knee specimens were obtained within two days of death. The mean age of the specimens was 60 years (43 to 81). The knees were extracted together with the surrounding subcutaneous soft tissue and about 100 mm of the extracapsular femur and tibia. The specimens were stored until needed in sealed polyethylene bags at –20°C to prevent dehydration.

The femur was stripped of muscle and fat, taking care to preserve the ligaments and joint capsule, and then set coaxially in a stainless-steel pot using polymethylmethacrylate (PMMA). During preparation and testing the specimens were covered with a wet paper towel to prevent dehydration.

Since the distal tibiofibular joint was not present the lateral collateral ligament was compromised. We therefore fixed the fibula to the tibia using two transcortical bone screws and thus restored the function of the lateral collateral ligament. The tibia was then potted in the same manner as the femur.

The intact joints were subjected to cyclic AP loads of ±100 N at 100 mm/min using an attachment with four
degrees of freedom in an Instron materials testing machine. Force versus crosshead displacement graphs were plotted. Details of this method have been described previously. The tests were carried out at 0, 30, 60, 90 and 130° of knee flexion.

Since the kinematics of the knee change when a cruciate ligament is ruptured, we had to establish an isometric point in the specimens with the PCL intact. The tibia was clamped vertically to a bench through a vertical axis bearing to allow unrestricted internal-external rotation. A transepicondylar Kirschner wire was then drilled through the femur just anterior to the roof of the intercondylar notch and used to apply a 10 N anterior load to the femur. This produced the equivalent of a 10 N posterior-drawer force, ensuring that slackness was taken out of the PCL. Straight needles carrying a light polyester thread were securely inserted within 1 mm of each other into the bone at the femoral origin of the PCL. The thread was passed over the posterior surface of the PCL and down to a displacement transducer (LVDT) which measured the change in separation of the femoral and tibial insertions. A tension of 0.1 N in the thread was maintained by the weight of the LVDT core. The femur was flexed and extended by hand, applying minimal force.

Based on the results of previous studies, we defined ‘isometric’ as a total excursion of <1 mm from 0 to 120° of flexion. We searched the area between and proximal to the anterior and posterior fibres of the PCL for an isometric point through two small portals proximal and distal to the oblique popliteal ligament.

Rupture of the PCL was produced by applying a posterior-drawer force in the Instron machine at 1000 mm/min with the knee in 90° of flexion. The four-degrees-of-freedom attachment was adjusted to lock the tibia in neutral internal-external rotation in order to give the best approximation of a straight posterior-drawer injury. Posterior tibial displacements were applied to the specimens, starting at 10 mm and increasing in 5 mm increments, until the PCL was completely ruptured.

We used 18 mm wide bone-patellar tendon-bone grafts, as preliminary experiments using smaller grafts had resulted in bone fractures. The tibial end of the graft was split into 10 mm and 8 mm wide bundles since this bone block was the strongest. We secured the 18 mm wide patellar bone block into a trough cut into the tibial insertion area of the PCL using bone cement and a screw. We tested three different reconstructions all based on bone-patellar tendon-bone grafts: isometric, single-bundled anatomic (aPC) and double-bundled anatomic (aPC+pPC). The grafts were tensioned to restore the knee to normal AP laxity at one flexion angle and then their effect on AP laxity was measured over the range of flexion-extension.

The isometric point was used as the centre for the 10 mm ‘isometric’ femoral tunnel (Fig. 3). The 10 mm portion of the graft was passed through the joint and the femoral tunnel to be secured to a tensiometer which was then adjusted to restore the intact AP laxity at 60° of flexion. The laxity of the reconstructed joint was then measured from extension to 130° of flexion by cyclic loading as before. For each flexion angle the graft tension at AP neutral was recorded. This protocol meant that joints were retested at 60° of flexion which enabled the integrity of the graft fixations to be checked. After laxity testing the graft was removed from the femoral tunnel which was then repaired with PMMA.

The single-bundled aPC used the 10 mm portion of the graft and was centred on the middle of the anterolateral bundle of the PCL (Fig. 3). This attachment site was chosen for this reconstruction since the anterolateral bundle makes up the bulk of the PCL. The tensioning, at 60° of flexion, and AP laxity testing were repeated. The 8 mm portion of the graft was then used to provide an additional bundle centred on the posterior of the posterolateral bundle of the PCL (Figs 1 and 3). The pPC graft was tensioned to restore intact AP laxity at 130° of flexion at which it has been shown to be active. The knee was again checked for AP laxity across the range of flexion.

Results

There was considerable variation in the AP laxity of the intact specimens (Fig. 4). The PCL ruptured at 1.94 ± 0.74
Figures 2a and 2b – Lateral view of the right knee with the lateral femoral condyle and lateral meniscus removed showing the anterolateral bundle slack in extension (a) and tight in flexion (b). Figures 2c and 2d – Posterior and lateral views of the left knee with the posterior capsule removed showing the posteromedial bundle tight in extension (c) and slack in flexion (d). Figure 2e – Anterolateral view of the fully flexed right knee with the lateral femoral condyle and lateral meniscus removed. The pPC fibres are shown by the dark sutures. In full flexion, the posteromedial bundle swings round to the front of the knee where it is well aligned to resist posterior drawer (reprinted from J Biomech 27: Race A, Amis AA. The mechanical properties of the two bundles of the human posterior cruciate ligament:13-24, 1994. With permission from Elsevier Science).

Figure 3a – Through-the-notch view of the femur with the patella removed showing the two-bundled anatomical reconstruction, the repaired isometric drill hole and the intact ACL. Figure 3b – Diagram showing the dimensions used to position the anatomical drill holes. In this specimen the drill holes did not overlap; in some smaller specimens the ‘anatomical’ drill holes overlapped the repaired isometric drill hole.
Fig. 4
Graph of the mean AP laxity of the intact knees over the range of flexion in response to a ±100 N drawer force with error bars showing 1 SD and hairlines marking the maxima and minima.

Fig. 5a
Graphs of relative AP laxity with flexion after PCL rupture (a), isometric reconstruction (b), anatomic single-bundle reconstruction (c) and anatomic double-bundle reconstruction (d). Hairlines show the relative laxity of the intact joint and a ±1 mm envelope. Error bars show 1 SD and p values significant differences with respect to the intact knee.
kN, range 1.35 to 3.50 kN, at a tibial displacement of 14.6 ± 2.8 (SD) mm, range 12 to 20 mm. A wide range of graft tensions were required to restore normal AP laxity at 60° of flexion. The tension requirement for the isometric grafts was 59 ± 51 N (SD; 2 to 153) and for the single aPC grafts 21 ± 21 N (1 to 62).

Figure 5 shows the relationship between AP laxity and flexion angle. A graft was considered to have restored normal laxity if it fell within ±1 mm of the intact laxity. Figure 6 shows the residual tension at AP neutral (no external load on the knee) over the range of flexion. Isometric grafts produced significant overconstraining at 0 and 30° of flexion (p = 0.004) and significant underconstraining at 90 and 130° of flexion (p = 0.016) (Fig. 5b). Although single aPC grafts restored normal laxity from 0 to 60°, they were significantly underconstraining at 90° and 130° of flexion (p = 0.006) (Fig. 5c). Double-bundled aPC+pPC reconstructions restored normal laxity over the full range of flexion (Fig. 5d). Isometric graft loads at AP neutral were higher than those for any of the anatomical reconstructions, significantly so at 0, 30 and 60° of flexion (p = 0.023) (Fig. 6).

Discussion

Reconstructions centred on an isometric point did not reproduce normal AP laxity. When tensioned to restore normal posterior drawer at 60° of flexion the knee was overconstrained towards extension and underconstrained in greater flexion. In addition, high loads were measured in the graft in the unloaded knee (Fig. 6). The tensiometers were extra-articular and therefore underestimated true graft load owing to graft angulation and friction in the bone tunnels.

The single-bundled aPC reconstruction resulted in nearly normal (within ±1 mm) posterior constraint from extension up to 60° of flexion. Nearly normal posterior constraint over the full range of flexion was provided by a double-bundled reconstruction. We found that the addition of a pPC graft restored nearly normal constraint in full flexion, thus giving nearly normal constraint over the entire range of flexion. In both the single- and double-bundled reconstructions the graft loads in the unloaded knee near extension were much lower than for the isometric reconstruction. We have therefore confirmed the hypothesis that the anatomical reconstruction restores normal AP laxity better than the isometric reconstruction.

Our study indicates that isometric PCL grafts may give poor results because of high loads near full extension and overconstraint of the joint. This appears to be counter-intuitive since the high tension in knee extension meant that the isometric graft was elongated. The cruciates and the grafts which replace them, however, are not rigid links but arrays of elastic fibres. In order to restore normal AP laxity at 60° of flexion the isometric graft was put under tension with no external posterior-drawer force. Tensioning the graft generated an internal drawer force which moved the tibia anteriorly until it was balanced by tension generated in the ACL. Because of this anterior movement the centre of rotation in flexion-extension moved anteriorly causing the graft to become slacker in flexion and tighter in extension. Thus, the tensed graft had caused the isometric point to become non-isometric. On knee extension, the isometric graft continued to ‘fight’ the increasingly taut ACL. If the behaviour of the PCL graft does not ‘fit’ with the ACL then the kinematics of the knee will be altered. The ACL is not designed to function with an isometric PCL. Close to extension the bulk of the PCL slackens, and becomes a secondary restraint to posterior drawer (Fig. 2a).

The graft tension required to restore normal AP laxity was three times higher for an isometric graft than for an...
aPC graft. This can be explained by considering that the effect of a ligamentous structure on AP drawer depends not only on its tension but also on its inclination to the direction of the drawer force. In the conventional sagittal plane projection both the isometric and aPC grafts were oblique to the direction of the drawer force at 60° of flexion. The alignment of the aPC graft was, however, somewhat better than that of the isometric graft. In a transverse plane projection the aPC graft, placed in the roof of the femoral notch, was nearly coincident with the direction of the AP drawer force whereas the isometric graft, placed more medially on the femur, was oblique to the drawer force. The aPC graft was better aligned than the isometric graft and could resist posterior-drawer load with a lower graft tension and elongation. When the knee was returned to AP neutral the aPC graft had not elongated as much as the isometric graft. As was seen by the lower residual graft tensions for the aPC (Fig. 6), there was antagonistic tension between the ACL and the graft at higher flexion angles, but because the aPC graft slackened markedly with extension it did not ‘fight’ the ACL and thus allowed a normal AP neutral position for the tibia near extension.

As the knee approaches full flexion, the aPC graft becomes more oblique to the tibial plateau and is therefore less efficient at resisting posterior-drawer forces (Fig. 5c). It is in this posture that the pPC graft takes over; it is almost perpendicular to the tibial plateau with the knee extended (Fig. 1), but moves around the aPC graft as the knee is flexed and takes up a more efficient orientation (Fig. 2e).

Our study has shown that it is not possible to define an ‘ideal’ graft pretension for all knees. This is understandable given the wide range of AP laxity in intact knees. Correct graft tension must be determined by its effect on AP laxity; this requires intraoperative measurement of laxity and it is not known whether the AP laxity set at the time of operation will be preserved after graft remodelling has taken place. We speculate that more anatomically ‘normal’ reconstructions will result in more benign remodelling, partly because of lower graft tensions.

Our findings of the average posterior laxity for the intact knee were very similar to those published previously for unrestricted posterior drawer, minimal in extension and increasing with flexion. Another study, using a constrained test rig with one degree of freedom, found the opposite trend.

Pearsall et al tested an isometric bone-patellar tendon-bone graft. They matched posterior laxity to normal in knee extension, but then found 7 mm of excess laxity at 90° of knee flexion, findings similar to our isometric graft results. Galloway et al obtained a better match by offsetting the attachment distally from the isometric point.

In our study we used PCL ruptures caused by straight posterior drawer at 90° of knee flexion. This mechanism of injury largely spared the posterolateral and capsular structures which are crucial to posterior stability in extension. When additional structures are damaged the results of PCL reconstructions are likely to be different.

Conclusions.

1. Since the PCL fibres diverge widely as they approach the extensive femoral attachment, it was not surprising to find that a single parallel-fibred graft could not maintain normal knee laxity across the range of knee flexion.

2. Although the idea of a single ‘isometric’ graft is attractive this does not fit the physiology of the knee, which shows distinctly non-isometric characteristics for the PCL.

3. In a single-bundled PCL reconstruction the bulk of the PCL is attached to the roof of the femoral intercondylar notch. An anterior femoral graft attachment which reproduces this will give normal posterior laxity from 0 to 60° of knee flexion.

4. If stability is essential across the full range of knee flexion, as in activities such as windsurfing or snowboarding, then a double-bundled reconstruction seems to be necessary.

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References


