Lateral soft-tissue structures contribute to cruciate-retaining total knee arthroplasty stability

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Running title: Lateral structures with TKA

Authors' contribution:

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Abstract

Little information is available to surgeons regarding how the lateral structures prevent instability in the replaced knee. The aim of this study was to quantify the lateral soft-tissue contributions to stability following cruciate-retaining total knee arthroplasty (CR TKA). Nine cadaveric knees were tested in a robotic system at full extension, 30°, 60°, and 90° flexion angles. In both native and CR implanted states, ±90 N anterior-posterior force, ±8 Nm varus-valgus and ±5 Nm internal-external torque were applied. The anterolateral structures (ALS, including the iliotibial band), the lateral collateral ligament (LCL), the popliteus tendon complex (Pop T) and the posterior cruciate ligament (PCL) were transected and their relative contributions to stabilising the applied loads were quantified. The LCL was found to be the primary restraint to varus laxity (an average 56% across all flexion angles), and was significant in internal-external rotational stability (28% and 26% respectively) and anterior drawer (16%). The ALS restrained 25% of internal rotation, whilst the PCL was significant in posterior drawer only at 60° and 90° flexion. The Pop T was not found to be significant in any tests. Therefore the LCL was confirmed as the major lateral structure in CR TKA stability throughout the arc of flexion, and deficiency could present a complex rotational laxity that cannot be overcome by the other passive lateral structures or the PCL.

Keywords: total knee arthroplasty; stability; lateral collateral ligament; biomechanics

Introduction

Instability in total knee arthroplasty (TKA), which is a result of soft tissue deficiency or inadequate balancing during surgery,[1-3] has been identified as the leading cause of early failure.[4] Therefore it is important to understand how ligaments and other passive structures stabilise the implanted knee to help surgeons provide a more informed peri-operative assessment. There have been many studies investigating the contributions in the native knee of anterolateral structures such as the iliotibial band (ITB), and posterolateral structures such as the lateral collateral ligament (LCL) and popliteus complex;[5-11] however their contributions in the presence of TKA is less well understood.[12]

On the anterolateral aspect of the knee, the ITB attaches to the tibia at Gerdy's tubercle and proximally splits into superficial and deep layers of the fascia lata.[13] Lying deep to the ITB are various capsular and extra-capsular structures, although there is contention regarding their anatomical description, particularly the anterolateral ligament (ALL).[14-16]

Among the posterolateral structures of the knee are the LCL and the popliteus complex. The LCL attaches on the femur just proximal and posterior to the lateral epicondyle and attaches to the lateral aspect of the fibular head close to its anterior margin.[17] The popliteus muscle originates from the posteromedial tibia, runs deep and obliquely to the LCL, and the tendon attaches to the femur distal to the lateral epicondyle.[17,18]

Lateral structures are commonly released to correct the alignment in valgus knees at time of TKA surgery, although there is no agreed standard procedure.[19] In a knee that is tight laterally in flexion, Whiteside advocated releasing the popliteus tendon (Pop T) followed by the LCL, and in a knee tight in extension releasing the ITB and lateral posterior capsule.[20] The findings from a cadaveric study by Krackow et al.[21] support the release of the LCL first to provide a more uniform joint gap space, followed by the Pop T and ITB if required in

severe deformities. By investigating the relative contributions of the different lateral structures to translational and rotational stability, complex laxity in different planes of motion caused either by intentional release or iatrogenic injury may be avoided.[22]

The objective of this study was to determine how different soft-tissue structures affect the stability in cruciate retaining (CR) TKA. Based on previous studies with native knees,[5,23-27] the hypotheses were that the LCL would be an important restraint in varus and internal-external rotation, the anterolateral structures (ALS) including the ITB, ALL and capsule would restrain internal rotation, the Pop T external rotation and the PCL posterior displacement and valgus rotation. The null hypothesis therefore was that there would be no significant difference in relative contributions of each structure to resisting tibiofemoral laxity.

Methods

Specimen Preparation

Nine fresh-frozen human cadaver knee specimens (seven male and two female) of mean age 64 years (range 47–88) were obtained from a tissue bank (five right-sided and four left-sided). Ethics approval was obtained from a local Human Tissue Authority Committee. The knees were cut 160 mm distally on the tibia/fibula and 170 mm proximally on the femur from the joint line respectively. None of the specimens had evidence of fixed flexion, misalignment or significant chondral loss.

For each specimen, the tibia/fibula and femur were skeletonized 105 mm and 110 mm from the joint line respectively, with all skin and tissue maintained within these limits. The femur and tibia were fixed in 60 mm diameter cylindrical steel pots using polymethyl-methacrylate (PMMA) bone cement (Simplex, Kemdent, UK). The tibia was aligned centrally in the bone

pot using a jig with a pointer that located the centre of the tibia as between the tips of the tibial spines.[28] The joint space was accessed through a midline incision to the skin and subcutaneous layer, and a medial parapatellar arthrotomy. The femur was cemented in the bone pot whilst in full extension, with the posterior condylar axis aligned parallel to the femoral fixture. The head of the fibula was fixed to the tibia using a transcortical bone screw to maintain its anatomical position, and the arthrotomy was sutured prior to testing.

Native Knee Kinematics

The knee was manually flexed 20 times to minimize soft tissue hysteresis, and then tested in a robotic testing system previously described.[29] The femur was fixed rigidly to the base of the robot and the tibia was mounted to the force/torque sensor on the end-effector of the manipulator. With the knee in the robot, a path of flexion from full extension to 90 ° was performed whilst the robotic system minimized forces and moments in the other five degrees of freedom (DOF) acting across the knee. This recorded the position of the knee at full extension, 30 °, 60 ° and 90 ° of flexion, which were the starting points for the following loads to the tibia: ±90 N anterior-posterior (AP) force, ±8 Nm varus-valgus (VV) torque, and ±5 Nm internal-external (IE) rotational torque. In each test, the robotic system applied the force/torque in the primary DOF, maintained the same flexion/extension DOF and minimized the loads in the four remaining DOF. These loads were chosen as being comparable to other studies of intraoperative laxity measurement [23,24,30,31] with the AP force being equivalent to that applied by a KT 1000^{TM} arthrometer (MEDmetric, CA, USA).[32] Each test was repeated three times.

Implanted Knee Kinematics

Following native knee data collection, the knee was removed from the robot and a CR TKA (PFC Sigma; DePuy Synthes Joint Reconstruction, Leeds, UK) was implanted by a consultant

orthopaedic surgeon, using a standard combination of measured resection and gap balancing performed in full extension and 90 ° of flexion. The femur was prepared using an intramedullary rod set at 5 ° of valgus, and a 9 mm resection measured from the least affected condylar side was made to the distal femur with neutral rotation referenced to the epicondylar axis. The femur was sized using an anterior down technique. The tibia was referenced using an intramedullary rod positioned with respect to the tibial anterior prominence, which corresponded to the centre of the tibial tuberosity in the specimens. A 10 mm resection with 3 ° posterior slope was then made from the least affected, most superior proximal tibial surface using a cutting guide. Gap balancing with spacers was used to confirm a rectangular space both in full extension and flexion after bone resection (but before chamfer femoral cuts). Both tibial and femoral components were cemented to the bone using PMMA. Osteophytes were removed to avoid 'tenting' of the ligaments, but no soft tissue releases were performed. A stable knee was defined as unimpeded tracking of the patella and no medial or lateral opening after implant trialling, throughout a passive flexion arc from full extension.

The knee joint with the TKA was then fixed back on the robot, and a new path of flexion from full extension to 90 ° was determined. ±90 N AP, ±8 Nm VV, and ±5 Nm IE loads were applied at the new positions of full extension, 30 °, 60 ° and 90 ° of flexion.

Lateral Soft-Tissue Contribution

With the implanted knee remaining attached to the robot, the lateral structures of the knee were sequentially transected (Fig. 1). The release of the ALS was performed whilst the knee was in full extension. The lateral margin of the patellar tendon was identified and an incision was made through the capsule at the level of Gerdy's tubercle to the anterior border of the LCL (encompassing the structures of the anterior and middle third lateral compartment as

described by Hughston et al. [33]). The structures including the ITB, ALL and capsule were then reflected off from their attachment on the proximal tibia as a single release.

The LCL and Pop T were transected separately at joint line level whilst the knee was at 90 ° flexion. Finally, at 90 ° flexion, the PCL was resected from its femoral attachment along the anterior part of the intercondylar notch to the lateral surface of the medial condyle. After each transection stage, the arthrotomy was resutured and the kinematics obtained at the implanted stage were reproduced (Table 1). Therefore using the principle of superposition,[34] the decrease in force/moment at each stage was calculated as the restraint provided by the transected structure.

Approval for this study (project code R13066) was given by the Imperial College Healthcare Tissue Bank under the Human Tissue Authority license number 12275.

Statistical Analysis

A custom Matlab (Mathworks, Natick, MA) script was used to calculate the mean peak forces/torques, translations/rotations and soft tissue contributions (defined as the drop in force/torque after the transection/release as a percentage of the original force/torque value) from the three repeats. A power calculation, based on a mean difference in AP translation of 3.5 ± 3 mm and VV rotation of 3.7 ± 3.2 ° in a prior study,[29] determined a minimum sample size of 8 would be needed to detect a significant change in laxity between the native and implanted state with 80 % power and 95 % confidence. The following statistical analysis was performed in SPSS 22 (IBM SPSS Statistics, version 22, Armonk, NY):

1. Two-way repeated-measures analysis of variance (RM-ANOVA) was performed to compare laxities to the knee state (native and implanted knee) across different flexion angles.

2. One-way RM-ANOVA was performed at each flexion angle to compare the force/torque contribution to the lateral structure cut.

For the force/torque contribution analyses, post hoc paired t-tests with Bonferroni correction at individual flexion angles were applied when differences between successive cuts were found. The p-values reported have been adjusted in SPSS by multiplying by the appropriate Bonferroni correction factor, and thus the significance level was set at $p < 0.05$. A second power analysis (based on the same previous study) determined that, given the number of specimens and expected standard deviations, soft-tissue contributions of 9 % could be detected with 80 % power and 95 % confidence.[29] Therefore it was decided that for a restraint at a given flexion angle to be deemed significant, it must have a mean resisting contribution greater than a threshold value of 10 % with $p < 0.05$.

Results

Anterior-posterior translation

Under an applied ± 90 N AP force, no significant difference was found between the native and implanted knee states (Fig. 2). The only significant restraint to anterior tibial drawer was the LCL (Fig. 3), which contributed 14 ± 10 % at 30 ° ($p = 0.024$), 15 ± 12 % at 60 ° ($p = 0.016$) and 22 ± 13 % at 90 ° ($p = 0.004$).

The main soft-tissue restraint to posterior drawer at 60 ° and 90 ° was the PCL, which restrained 36 ± 34 % ($p = 0.049$) and 50 ± 40 % ($p = 0.022$) respectively (Fig. 3). At full extension the LCL contributed 11 ± 9 % ($p = 0.034$) but was not found to be significant at any other flexion angle.

Internal-external rotation

Under an applied 5 Nm internal torque, the implanted knee state was found to be more restrained in rotation than the native knee ($p = 0.012$, < 0.001, 0.003 and 0.015 at full extension, 30 °, 60 ° and 90 ° respectively, Fig. 4). Both the ALS and the LCL's contributions were found to be significant at all flexion angles (Fig. 5). The ALS contributed 25 % on average across all flexion angles ($p < 0.05$), and the LCL was found to restrain 28 % ($p < 0.05$).

In response to 5 Nm external torque, the implanted knee start was found to be more restrained in rotation than the native knee ($p < 0.001$ at all flexion angles, Fig. 4). The LCL restrained 27 ± 17 % ($p = 0.007$), 32 ± 21 % ($p = 0.007$) and 25 ± 20 % ($p = 0.018$) at full extension, 30 ° and 60 ° respectively (Fig. 5). None of the other structures demonstrated a significant contribution over 10 %.

Varus-valgus laxity

Under an applied 8 Nm varus torque, the implanted knee state was found to be more restrained in rotation than the native knee at all flexion angles ($p = 0.045$, 0.002, 0.001 and 0.002 at full extension, 30 °, 60 ° and 90 ° respectively, Fig. 6). The LCL was the primary varus restraint at all flexion angles tested (Fig. 7). The average contribution was 56 %, ranging from 47 ± 24 % at full extension to 63 ± 12 % at 30 °. The ALS, popliteus tendon complex or the PCL were not found to be significant.

When an 8 Nm valgus torque was applied, the implanted state was more restrained than the native knee at full extension ($p = 0.003$) and 90 ° ($p < 0.001$, Fig. 6). None of the structures sectioned had a valgus contribution greater than 10 % that was found to be statistically significant (Fig. 7).

Discussion

The most important finding of the study was that in the presence of CR TKA, the LCL was a primary restraint to varus laxity and had a prominent role in internal-external rotational stability. Another major finding was that the anterolateral structures including the ITB, ALL and anterolateral capsule provided an important restraint to internal rotation. Both these findings support our hypotheses based upon prior studies on the native knee.[5,24,25] Therefore in the event of deficiency of the LCL and anterolateral structures, the results suggest that a either a more constrained implant or soft-tissue reconstruction would be required to restore stability to the knee. Care should also be taken when releasing the LCL in a valgus knee, as it may result in combined rotational instability that may not be overcome by the other passive lateral structures and the PCL.

The LCL has long been associated with restraining varus and IE rotation, which was in agreement with the results in this study. An interesting finding was that in the presence of the CR TKA, the LCL contributed to varus and IE rotation throughout all flexion angles tested, in agreement with Kanamiya et al.[35] It is known that in a native knee, the LCL slackens beyond 30 ° flexion and becomes less aligned to resist rotation,[7,36] therefore implying that the reliance on the LCL as a rotatory restraint in deeper flexion is increased in TKA compared with the native knee. The LCL was also found to contribute approximately 16 % to resisting tibial anterior translation across flexion angles, a larger role than that previously found in native knees.[24,37,38] This finding, along with a prior study into the contribution of medial soft tissues in TKA,[29] suggests that in the absence of the anterior cruciate ligament the collaterals must provide a larger anterior restraint in implants with low conforming articular surfaces.

In this study, the lateral soft-tissue anterior to the LCL (ALS) was investigated as an entire resection stage and was found to be an internal rotation restraint at all flexion angles. This

was in agreement with Wroble et al. using a similar single-stage cut in native knees, although they also found significance in anterior translation uncorroborated in this TKA study.[38] In a study separating the structures, Kittl et al. found the ITB to be a significant restraint in internal rotation in ACL-deficient native knees with less significance prescribed to the ALL and anterolateral capsule.[5] Other investigations which did not investigate the ITB found the ALL to be an important internal restraint in native knees.[39,40]

The Pop T was not found to be a significant restraint, which was not hypothesized. In the native knee, the popliteus complex has been found to be a restraint to external tibial rotation [23,38,41] as well as secondary restraint to varus rotation.[23] A possible reason could be that the geometry of the implant causes the oblique Pop T to be less well-aligned to restrain tibial external rotation than in the native knee: for example it has previously been shown that native knee femoral rollback is not replicated in implanted knees.[42-44] This result suggests that if released to correct a valgus deformity as previously suggested,[20] there would be no adverse instability in the other planes of motion. In implanted knees, Ghosh et al. found that sectioning the Pop T resulted in significant increase in IE and VV rotation only in deep flexion angles beyond 90°, which was not investigated here.[22] Kesman et al. found in a blinded in-vivo trial that Pop T resection did not subjectively alter intraoperative TKA stability.[45]

The PCL was not found to restrain valgus rotation as hypothesized, and was only found to be a significant restraint to posterior translation at 60-90° flexion. Comparatively, in native knees the PCL is the primary restraint throughout entire flexion in some studies,[24,26] although its contribution was also found to fall as the knee was extended [27]. The PCL has also been reported to be a secondary valgus restraint after the superficial medial collateral ligament.[25] Another study found that between 45-69% of the tibial PCL attachments were resected during standard CR-TKA tibial cuts,[46] which may reduce the amount of load that the PCL can carry

and therefore its usefulness post-TKA.[47] This is a potential reason why the implanted knees in this study did not meet the hypothesis stated, which was based on studies with native, non-injured PCL-intact knees.[24-27] It is clear however that if the PCL is completely ruptured there would be instability in posterior drawer especially at higher flexion angles, and a posterior-stabilised (PS) TKA with a post-cam mechanism would be needed to restore stability.

There was a difference in rotational stability observed between the native state and CR implanted knee. Decreased IE and VV laxity in TKA knees compared with the native knee were found in other previous cadaveric studies, which also included loaded hamstrings and quadriceps.[48,49] No release of tight soft-tissues during implantation was performed, to ensure the subsequent transection stages were possible. Releases were not required, due to the absence of articular erosion, and all knees demonstrated equal gap balancing in extension and flexion during implantation.

Among the limitations of cadaveric testing is that only time-zero effects can be investigated, and so healing of soft tissues back to bony attachments was not investigated. There was no axial joint compression which would increase the stability of the implanted knees. There was also no muscle tension simulation, which could potentially overestimate the contribution of the LCL relative to the other structures such as the ITB [50] and the active restraint provided by the popliteus muscle. However, both the Pop T and ITB have been found to provide static stability similar to a ligament,[5,23] and thus the study has simulated clinical evaluation of joint laxity when the patient lies supine with relaxed muscles and investigated the purely passive contributions of the lateral structures. Strengths of this study include the repeated-measures design of the protocol, which minimized any inter-specimen variability effects, and the ability of the robot testing system to apply repeatable loads and accurately measure resulting knee kinematics and forces/torques.

This study has investigated the relative contributions of lateral structures in CR TKA, and future research is required on how this varies in different implant designs. For example, a recent study found that the medial collateral ligament provided a larger valgus restraint in posterior stabilized TKA than CR TKA,[29] and this may have similar implications for the LCL. Höher et al. found higher popliteus complex in-situ forces in PCL-deficient native knees than intact knees during posterior drawer, which may manifest as a larger popliteus contribution in PCL-sacrificing TKA than found in the CR TKA in this study.[10] Ghosh et al. investigated increasing implant constraint in the face of posterolateral deficiency,[48] and extending this to investigating anterolateral deficiency could also be a focus of further work. Alternatively to increasing implant constraint, would be examining the effect of ligamentotaxis in future cadaveric studies:[51] retensioning capsular structures in an LCL-deficient knee by increasing the tibial insert thickness may restore enough stability to avoid a revision implant or ligament reconstruction.

Conclusion

This biomechanical study has delineated the relative contributions of lateral structures to stability in CR TKA, and has found that the LCL is an important restraint in varus and internal-external rotational stability. Therefore it is argued that releasing the LCL to correct a valgus deformity may introduce a complex laxity pattern that may only be stabilized by implanting a more constrained implant or reconstructing the ligament. Releasing the ITB and the ALS may introduce internal rotational laxity, however if the LCL is intact this may still be a viable option. Release of the Pop T is unlikely to cause rotational laxity and thus would be safe to perform. Complete release of the PCL may require a PS TKA to restabilise in posterior drawer at higher flexion angles.

Acknowledgments

306 This study was supported through an educational grant from Newcastle Healthcare Charities.
307 Surgical instruments and implants were supplied by DePuy Synthes Joint Reconstruction.
308 The robotic test system and Dr El Daou were supported by the Wellcome Trust and EPSRC
309 funded Centre for Medical Engineering Solutions in Osteoarthritis Centre of Excellence at
310 Imperial College London. The authors thank Piers Milner for his assistance with data
311 collection in the laboratory.

References

1. Lombardi Jr AV, Berend KR, Adams JB. 2014. Why knee replacements fail in 2013: patient, surgeon, or implant? Bone & Joint Journal 96B:101-104.
2. Sharkey PF, Hozack WJ, Rothman RH, et al. 2002. Why are total knee arthroplasties failing today? Clin Orthop Relat Res 404:7-13.
3. Mulhall KJ, Ghomrawi HM, Scully S, et al. 2006. Current etiologies and modes of failure in total knee arthroplasty revision. Clin Orthop Relat Res 446:45-50.
4. Schroer WC, Berend KR, Lombardi AV, et al. 2013. Why Are Total Knees Failing Today? Etiology of Total Knee Revision in 2010 and 2011. The Journal of Arthroplasty 28:116-119.
5. Kittl C, El-Daou H, Athwal KK, et al. 2016. The Role of the Anterolateral Structures and the ACL in Controlling Laxity of the Intact and ACL-Deficient Knee. Am J Sports Med 44:345-354.
6. Thaunat M, Pioger C, Chatellard R, et al. 2014. The arcuate ligament revisited: role of the posterolateral structures in providing static stability in the knee joint. Knee Surg Sports Traumatol Arthrosc 22:2121-2127.
7. Lim HC, Bae JH, Bae TS, et al. 2012. Relative role changing of lateral collateral ligament on the posterolateral rotatory instability according to the knee flexion angles: a biomechanical comparative study of role of lateral collateral ligament and popliteofibular ligament. Arch Orthop Traum Surg 132:1631-1636.
8. Zantop T, Schumacher T, Diermann N, et al. 2007. Anterolateral rotational knee instability: role of posterolateral structures. Arch Orthop Traum Surg 127:743-752.
9. Bonanzinga T, Signorelli C, Lopomo N, et al. 2015. Biomechanical effect of posterolateral corner sectioning after ACL injury and reconstruction. Knee Surg Sports Traumatol Arthrosc 23:2918-2924.
10. Hoher J, Harner CD, Vogrin TM, et al. 1998. In situ forces in the posterolateral structures of the knee under posterior tibial loading in the intact and posterior cruciate ligament-deficient knee. J Orthop Res 16:675-681.
11. Veltri DM, Deng XH, Torzilli PA, et al. 1995. The role of the cruciate and posterolateral ligaments in stability of the knee. A biomechanical study. Am J Sports Med 23:436-443.
12. Athwal KK, Hunt NC, Davies AJ, et al. 2014. Clinical biomechanics of instability related to total knee arthroplasty. Clinical Biomechanics (Avon) 29:119-128.
13. Fairclough J, Hayashi K, Toumi H, et al. 2006. The functional anatomy of the iliotibial band during flexion and extension of the knee: implications for understanding iliotibial band syndrome. J Anat 208:309-316.
14. Dodds AL, Halewood C, Gupte CM, et al. 2014. The anterolateral ligament: Anatomy, length changes and association with the Segond fracture. Bone Joint J 96B:325-331.
15. Claes S, Vereecke E, Maes M, et al. 2013. Anatomy of the anterolateral ligament of the knee. J Anat 223:321-328.
16. Vincent JP, Magnussen RA, Gezmez F, et al. 2012. The anterolateral ligament of the human knee: an anatomic and histologic study. Knee Surgery Sports Traumatology Arthroscopy 20:147-152.
17. LaPrade RF, Ly TV, Wentorf FA, et al. 2003. The posterolateral attachments of the knee - A qualitative and quantitative morphologic analysis of the fibular collateral ligament, popliteus tendon, popliteofibular ligament, and lateral gastrocnemius tendon. Am J Sports Med 31:854-860.
18. Takeda S, Tajima G, Fujino K, et al. 2015. Morphology of the femoral insertion of the lateral collateral ligament and popliteus tendon. Knee Surg Sports Traumatol Arthrosc 23:3049-3054.
19. Whiteside LA. 1999. Selective Ligament Release in Total Knee Arthroplasty of the Knee in Valgus. Clin Orthop Relat Res 367:130-140.
20. Whiteside LA. 2002. Soft tissue balancing - The knee. J Arthroplasty 17:23-27.

21. Krackow KA, Mihalko WM. 1999. Flexion-extension joint gap changes after lateral structure release for valgus deformity correction in total knee arthroplasty - A cadaveric study. J Arthroplasty 14:994-1004.
22. Ghosh KM, Hunt N, Blain A, et al. 2015. Isolated popliteus tendon injury does not lead to abnormal laxity in posterior-stabilised total knee arthroplasty. Knee Surg Sports Traumatol Arthrosc 23:1763-1769.
23. LaPrade RF, Wozniczka JK, Stellmaker MP, et al. 2010. Analysis of the Static Function of the Popliteus Tendon and Evaluation of an Anatomic Reconstruction: The "Fifth Ligament" of the Knee. The American Journal of Sports Medicine 38:543-549.
24. Gollehon DL, Torzilli PA, Warren RF. 1987. The role of the posterolateral and cruciate ligaments in the stability of the human knee - a biomechanical study. Journal of Bone and Joint Surgery - American Volume 69A:233-242.
25. Grood ES, Noyes FR, Butler DL, et al. 1981. Ligamentous and capsular restraints preventing straight medial and lateral laxity in intact human cadaver knees. Journal of Bone and Joint Surgery - American Volume 63:1257-1269.
26. Grood ES, Stowers SF, Noyes FR. 1988. Limits of movement in the human knee - effect of sectioning the posterior cruciate ligament and posterolateral structures. Journal of Bone and Joint Surgery - American Volume 70A:88-97.
27. Race A, Amis AA. 1996. Loading of the two bundles of the posterior cruciate ligament: an analysis of bundle function in A-P drawer. J Biomechs 29: 873-879.
28. Amis AA, Scammell BE. 1993. Biomechanics of intra-articular and extra-articular reconstruction of the anterior cruciate ligament. Journal of Bone and Joint Surgery-British Volume 75:812-817.
29. Athwal KK, El Daou H, Kittl C, et al. 2015. The superficial medial collateral ligament is the primary medial restraint to knee laxity after cruciate-retaining or posterior-stabilised total knee arthroplasty: effects of implant type and partial release. Knee Surg Sports Traumatol Arthrosc:DOI 10.1007/s00167-00015-03796-00160.
30. Petersen W, Loerch S, Schanz S, et al. 2008. The role of the posterior oblique ligament in controlling posterior tibial translation in the posterior cruciate ligament-deficient knee. Am J Sports Med 36:495-501.
31. Saeki K, Mihalko WM, Patel V, et al. 2001. Stability after medial collateral ligament release in total knee arthroplasty. Clin Orthop Relat Res 392:184-189.
32. Daniel DM, Malcom LL, Losse G, et al. 1985. Instrumented measurement of anterior laxity of the knee. Journal of Bone and Joint Surgery - American Volume 67A:720-726.
33. Hughston JC, Andrews JR, Cross MJ, et al. 1976. Classification of knee ligament instabilities part II. Lateral compartment. Journal of Bone and Joint Surgery - American Volume 58:173-179.
34. Rudy TW, Livesay GA, Woo SLY, et al. 1996. A combined robotic/universal force sensor approach to determine in situ forces of knee ligaments. J Biomech 29:1357-1360.
35. Kanamiya T, Whiteside LA, Nakamura T, et al. 2002. Ranawat Award paper. Effect of selective lateral ligament release on stability in knee arthroplasty. Clin Orthop Relat Res 404:24-31.
36. Sugita T, Amis AA. 2001. Anatomic and biomechanical study of the lateral collateral and popliteofibular ligaments. Am J Sports Med 29:466-472.
37. Butler DL, Noyes FR, Grood ES. 1980. Ligamentous restraints to anterior-posterior drawer in the human knee - biomechanical study. Journal of Bone and Joint Surgery -American Volume 62:259-270.
38. Wroble RR, Grood ES, Cummings JS, et al. 1993. The role of the lateral extraarticular restraints in the anterior cruciate ligament-deficient knee. The American Journal of Sports Medicine 21:257-263.
39. Parsons EM, Gee AO, Spiekerman C, et al. 2015. The biomechanical function of the anterolateral ligament of the knee. Am J Sports Med 43:669-674.

40. Rasmussen MT, Nitri M, Williams BT, et al. 2015. An In Vitro Robotic Assessment of the Anterolateral Ligament, Part 1: Secondary Role of the Anterolateral Ligament in the Setting of an Anterior Cruciate Ligament Injury. The American Journal of Sports Medicine:DOI 10.1177/0363546515618387.
41. Pasque C, Noyes FR, Gibbons M, et al. 2003. The role of the popliteofibular ligament and the tendon of popliteus in providing stability in the human knee. Journal of Bone and Joint Surgery-British Volume 85B:292-298.
42. Li G, Zayontz S, Most E, et al. 2001. Cruciate-retaining and cruciate-substituting total knee arthroplasty - An in vitro comparison of the kinematics under muscle loads. J Arthroplasty 16:150-156.
43. Most E, Zayontz S, Li GA, et al. 2003. Femoral rollback after cruciate-retaining and stabilizing total knee arthroplasty. Clin Orthop Relat Res 410:101-113.
44. Walker PS, Sussman-Fort JM, Yildirim G, et al. 2009. Design Features of Total Knees for Achieving Normal Knee Motion Characteristics. J Arthroplasty 24:475-483.
45. Kesman TJ, Kaufman KR, Trousdale RT. 2011. Popliteus Tendon Resection During Total Knee Arthroplasty An Observational Report. Clin Orthop Relat Res 469:76-81.
46. Matziolis G, Mehlhorn S, Schattat N, et al. 2012. How much of the PCL is really preserved during the tibial cut? Knee Surg Sports Traumatol Arthrosc 20:1083-1086.
47. Van Opstal N, Feyen H, Luyckx JP, et al. 2016. Mean tensile strength of the PCL in TKA depends on the preservation of the tibial insertion site. Knee Surg Sports Traumatol Arthrosc 24:273-278.
48. Ghosh KM, Manning WA, Blain AP, et al. 2015. Influence of increasing construct constraint in the presence of posterolateral deficiency at knee replacement: A biomechanical study. J Orthop Res:DOI 10.1002/jor.23026.
49. Hunt NC, Ghosh KM, Blain AP, et al. 2015. No statistically significant kinematic difference found between a cruciate-retaining and posterior-stabilised Triathlon knee arthroplasty: a laboratory study involving eight cadavers examining soft-tissue laxity. Bone & Joint Journal 97B:642-648.
50. Yamamoto Y, Hsu WH, Fisk JA, et al. 2006. Effect of the iliotibial band on knee biomechanics during a simulated pivot shift test. J Orthop Res 24:967-973.
51. McPherson EJ, Portugal D. 2007. Revision Total Knee Arthroplasty for Excessive Ligamentotaxis. The Journal of Arthroplasty 22:1214-1216.

453 **Figure legends**

454 **Figure 1.** Illustration demonstrating the lateral structures with cruciate-retaining total knee
455 arthroplasty (posterolateral view of a right knee).

456 **Figure 2.** Anterior-posterior translation in response to ± 90 N anterior-posterior force, with
457 standard deviation.

458 **Figure 3.** Percentage contributions of the anterolateral structures (ALS), lateral collateral
459 ligament (LCL), popliteus tendon (Pop T) and posterior cruciate ligament (PCL) in resisting
460 ± 90 N anterior-posterior force in implanted knees, with standard deviation. * indicates a
461 statistically significant contribution greater than 10 % at the specified flexion angle ($p <$
462 0.05).

463 **Figure 4.** Internal-external rotation in response to ± 5 Nm internal-external moment, with
464 standard deviation. * indicates the flexion angle at which rotation of the native knee was
465 significantly larger than the implanted state ($p < 0.05$).

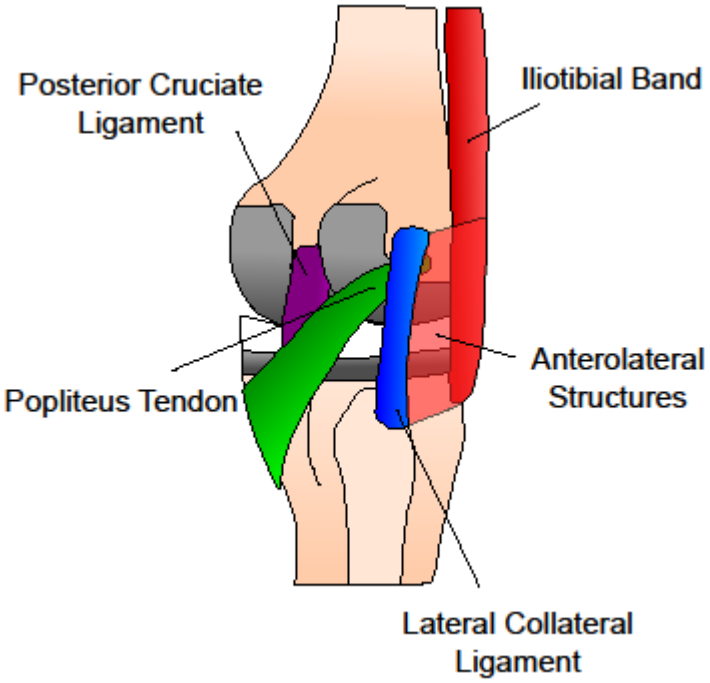
466 **Figure 5.** Percentage contributions of the anterolateral structures (ALS), lateral collateral
467 ligament (LCL), popliteus tendon (Pop T) and posterior cruciate ligament (PCL) in resisting
468 ± 5 Nm internal-external moment in implanted knees, with standard deviation. * indicates a
469 statistically significant contribution greater than 10 % at the specified flexion angle ($p <$
470 0.05).

471 **Figure 6.** Varus-valgus laxity in response to ± 8 Nm varus-valgus moment, with standard
472 deviation. * indicates the flexion angle at which rotation of the native knee was significantly
473 larger than the implanted state ($p < 0.05$).

474 **Figure 7.** Percentage contributions of the anterolateral structures (ALS), lateral collateral
475 ligament (LCL), popliteus tendon (Pop T) and posterior cruciate ligament (PCL) in resisting

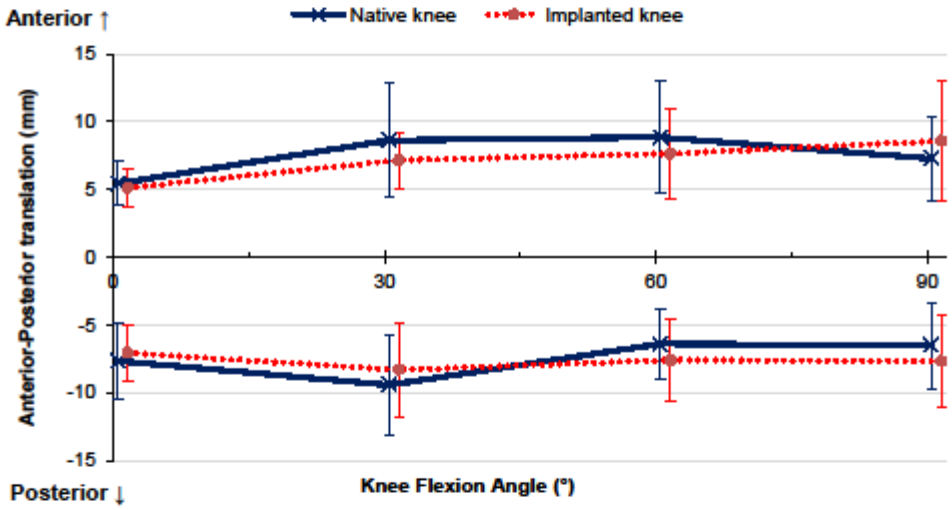
476 ± 8 Nm varus-valgus moment in implanted knees, with standard deviation. * indicates a
477 statistically significant contribution greater than 10% at the specified flexion angle ($p < 0.05$).

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Posterior Cruciate Ligament
Iliotibial Band
Popliteus Tendon
Anterolateral Structures
Lateral Collateral Ligament

479 fig 1

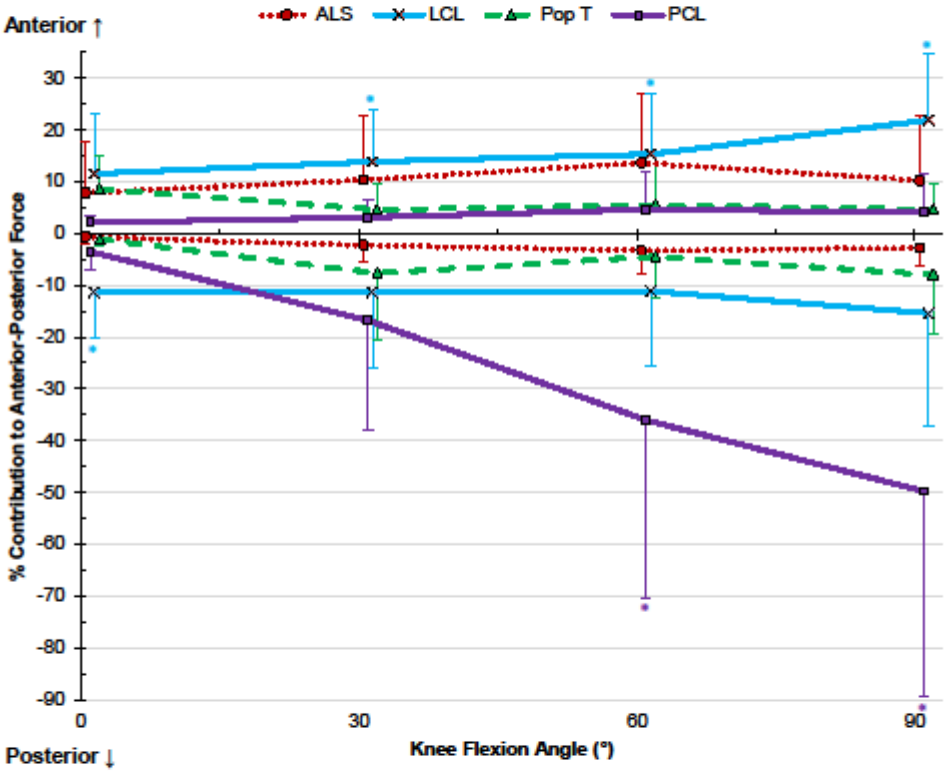
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482 fig 2

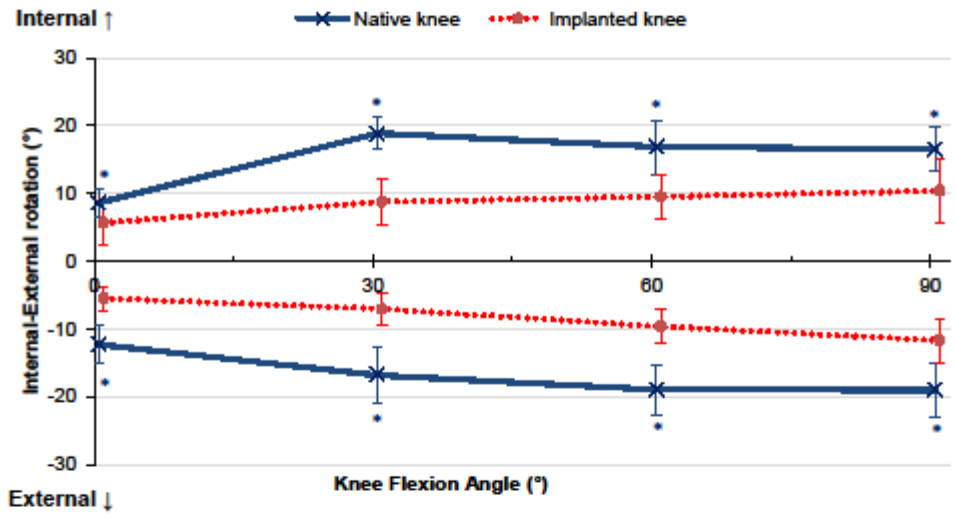
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485 fig 3

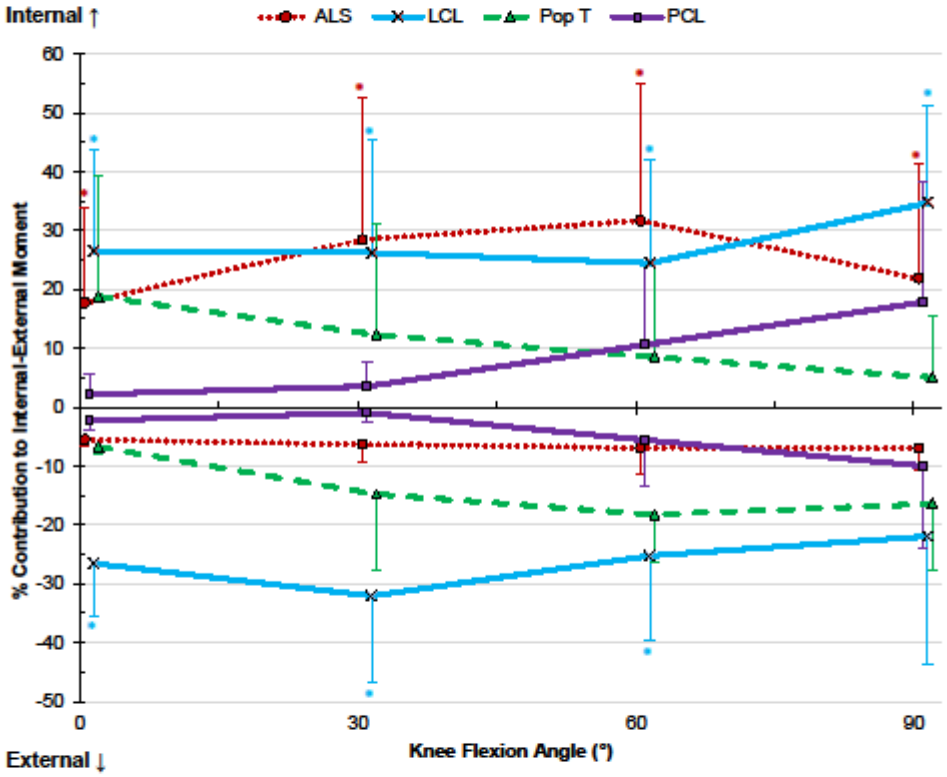
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488 fig 4

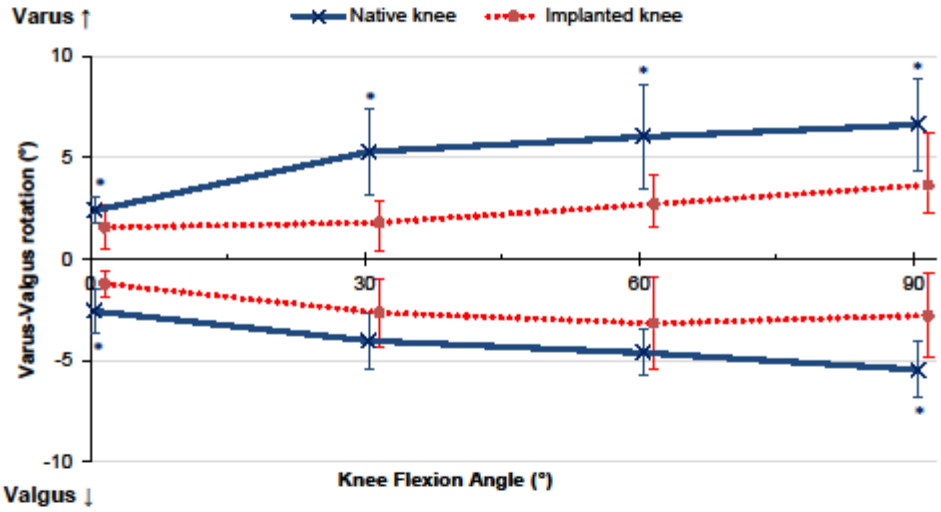
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491 fig 5

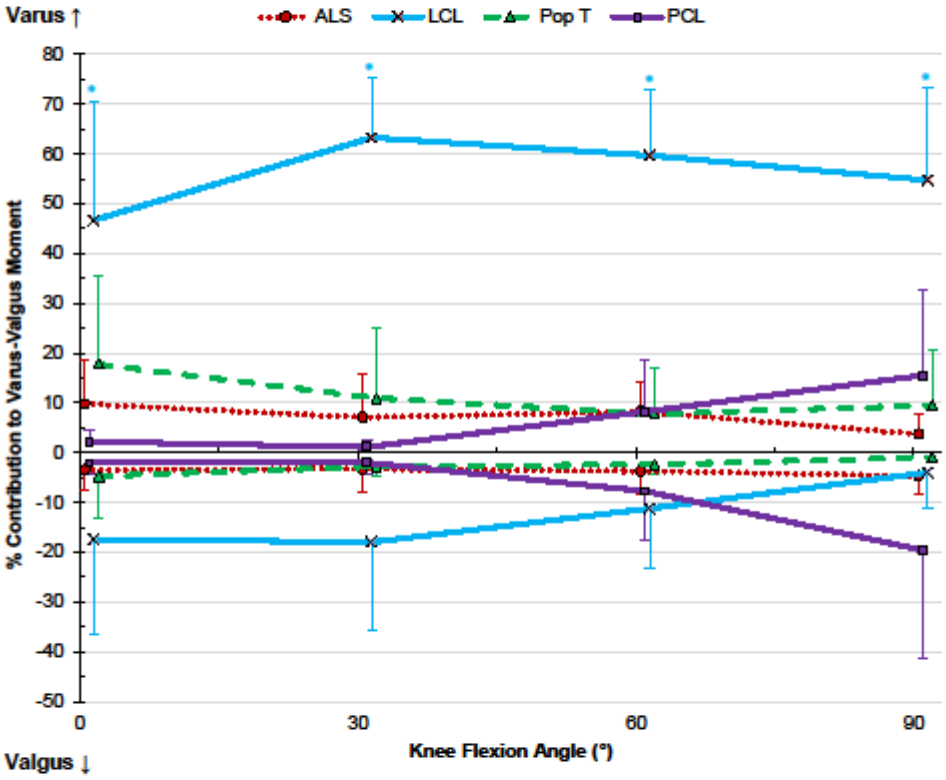
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494 fig 6

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497 fig 7

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499 Table 1.

Table 1

Tables

Table 1. Outline of the experimental protocol and data obtained.

Knee State	Kinematic Test	Data Obtained
Native knee	±90N AP, ±8Nm VV, ±5Nm IE	Native knee kinematics (I)
Implant CR TKA	±90N AP, ±8Nm VV, ±5Nm IE	TKA knee kinematics (II)
Transect anterolateral structures (ALS)	Repeat kinematics II	Restraining force/moments from ALS
Transect lateral collateral ligament (LCL)	Repeat kinematics II	Restraining force/moments from LCL
Transect popliteus tendon (Pop T)	Repeat kinematics II	Restraining force/moments from Pop T
Transect posterior cruciate ligament (PCL)	Repeat kinematics II	Restraining force/moments from PCL

Key to content: AP = anterior-posterior force, VV = varus-valgus torque, IE = internal-external torque, CR TKA = cruciate-retaining total knee arthroplasty. The anterolateral structures include the iliotibial band, anterolateral ligament and capsule.

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