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Biomechanics of the PCL and related structures: posterolateral, posteromedial and meniscofemoral ligaments

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Abstract This paper reviews and updates our knowledge of the anatomy and biomechanics of the posterior cruciate ligament, and of the posterolateral, posteromedial and meniscofemoral ligaments of the knee. The posterior cruciate ligament is shown to have two functional fibre bundles that are tight at different angles of knee flexion. It is the primary restraint to tibial posterior draw at all angles of knee flexion apart from near full extension. In contrast, the posterolateral and posteromedial structures are shown to tighten as the knee extends, and to be well-aligned to resist tibial posterior draw. These structures also act as primary restraints against other tibial displacements. Tibial internal rotation is restrained by the medial and posteromedial structures, while tibial external rotation is restrained by the lateral and posterolateral structures. They are also the primary restraints against tibial abduction-adduction rotations. The meniscofemoral ligaments are shown, for the first time, to contribute significantly to resisting tibial posterior draw, and to have a strength of approximately 300 N. Taken together, this evidence shows how the posterolateral and posteromedial structures are responsible for posterior knee stability near extension, and this, along with the action of the

meniscofemoral ligaments, may explain why an isolated rupture of the posterior cruciate ligament does not often lead to knee instability

Keywords Posterior cruciate - Posterolateral - Posteromedial - Meniscofemoral - Ligament

Introduction

The overall purpose of this paper is to review and update our knowledge of the anatomy and biomechanics of the posterior cruciate ligament, and of the posterolateral, posteromedial and meniscofemoral ligaments of the knee.

The basic anatomy and function of the PCL are well-known. It has a relatively compact tibial attachment located posteriorly, on the midline between the posterior horns of the menisci and just below the joint line. The fibres are oriented in a proximal-anterior direction, but fan out as they pass from the tibia to the femur, thus giving an extensive area of femoral attachment. This femoral attachment is immediately adjacent to the condylar articular cartilage, and extends from the most anterior-distal part of the femoral intercondylar notch (that is, the notch roof when observed in a flexed knee) to the most posterior extent of the lateral surface of the medial femoral condyle. Several studies have shown that the PCL is the strongest ligament crossing the knee, and that it is the primary restraint to tibial posterior translation.

Despite this basic knowledge, the PCL remains an enigma: it is also well-known that many patients can function well following a PCL rupture, and so some surgeons choose never to operate on an isolated PCL rupture. There is clearly some difficulty in reconciling the great strength of the PCL (which, if the laws of tissue adaptation are true, implies that it transmits large forces) versus the apparent lack of functional problems following its rupture.

When the role of ACL injury and the pivot-shift instability were first appreciated, some thirty years ago, the initial response was to develop peripheral extraarticular procedures. These sought to control the excessive anterior movements of the lateral aspect of the tibial plateau. Later, as the methods for intraarticular patellar tendon graft reconstructions were refined, so the extraarticular reconstructions fell from favour. There has since been so much effort to optimise intraarticular cruciate ligament reconstructions that many surgeons have lost sight of the importance of the peripheral structures, and of the need to reconstruct these, in addition to the cruciate ligaments, if they have been damaged. One of the aims of this paper is to illuminate the roles of the peripheral structures that act in synergy with the PCL, that have received relatively little attention. Not only will this give information relating to these structures, but it will suggest why the knee can function in the absence of the PCL.

PCL biomechanics

Strength

The tensile strength of the PCL has been reported by a number of investigators: Kennedy et al. [26] 1051 N, Marinozzi et al. [31] 855 N, Prietto et al. [37] 1627 N, Trent et al. [49] 739 N. These figures may have been underestimates, because the PCL fibres act in different directions, and so a uniaxial test of the whole ligament may have caused a sequential tearing failure, as different fibres became tight. This suggestion is supported by the data of Butler et al. [6], who showed that the PCL fascicles had significantly higher strength than the whole ligament tests had suggested.

The PCL is often considered to be made up of two functional bundles of fibres, the anterolateral (aPC) and the posteromedial (pPC), where these refer to their relative positions on the femur [12, 22, 35 et seqq] (Fig. 1). Race and Amis [38] separated these bundles and found strengths of 1620 N for the aPC and 258 N for the pPC. This difference was related to significant differences in the cross-sectional areas (43 and 10 mm^2), and to the maximum stress that the tissues would withstand (35.9 and 24.4 MPa, respectively). Thus, this study showed that the aPC fibre bundle is where most of the strength of the PCL is found.

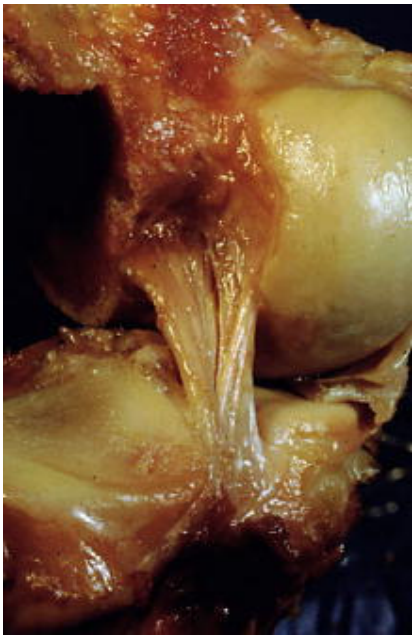

Fig. 1. Knee viewed from posteromedial aspect, after removal of the lateral femoral condyle. The PCL has been split artificially into two functional bundles: anterolateral, which attaches to the roof of the femoral intercondylar notch, and posteromedial, which attaches to the medial side of the notch (from Race and Amis [38], with permission)

These results were for specimens with a mean age of 75 years: Noyes and Grood [34] and Woo et al. [56] found that the ACL strength was approximately 2.5 times greater in young than in old knees. If this trend is also true for the PCL, this suggests that the PCL strength may be 4.5 kN (5 to 6 times body weight) in young adults.

Tibial posterior translation

Many authors have noted that isolated cutting of the PCL allows a minimal increase in posterior draw when the knee is extended, and that there is a greater increase in laxity as the knee flexes [4, 12, 35et seqq]. Several studies (e.g. [13, 15]) have produced graphs similar to Fig. 2 (from [2]), which shows clearly that PCL rupture has the greatest effect at 90 degrees knee flexion. This, therefore, is the position where the integrity of the ligament should be tested clinically. Some investigators [11, 13, 15] have found that a posterior draw force also induced a coupled tibial external rotation when the knee was intact, and that this was either eliminated or reduced after the PCL was cut. Fukubayashi et al. [11] also reported that when the coupled tibial rotation was prevented, tibial posterior laxity was reduced by 30%.

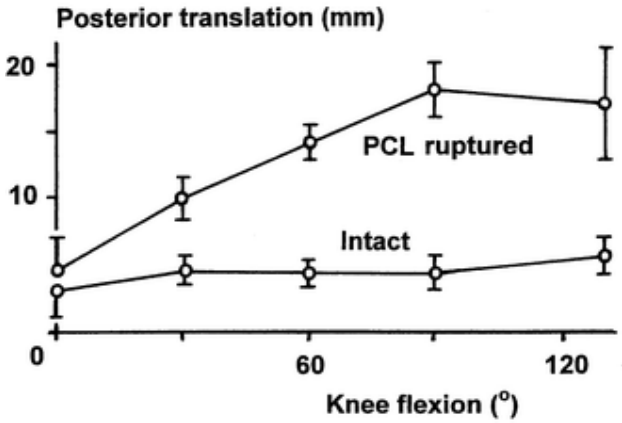

Fig. 2. Variation of tibial posterior translation with knee flexion, for a force of 100 N, for the knee when intact and after PCL rupture (from Amis [2], with permission)

The contribution of the PCL to resisting tibial posterior translation has been shown by selective cutting studies. In this method, the tibia was displaced posteriorly a known distance and the force required was recorded. When the same displacement was repeated after the PCL had been cut, the reduction in force represented the contribution that had been made by the PCL. For a 5 mm posterior displacement, Butler et al. [7] found that the PCL contributed approximately 95% at 30 and 90 degrees knee flexion. Similarly, Piziali et al. [36] found a contribution of 83% with the knee extended. However, these studies did not allow coupled tibial external rotation, and the PCL contribution would be reduced in the absence of that artificial restraint, because the load could then be shared more by the posterolateral structures.

The authors cited above who described the fibre bundles of the PCL also noted that they had different patterns of tightening and slackening as the knee flexed and extended. The aPC was noted to be slack in knee extension (and this is seen on MRI, because the fibres take a curved path from femur to tibia), and to tighten in mid flexion. The pPC fibres were tight in full knee extension and slack in the mid range. Hughston et al. [22] showed that the pPC became well-aligned to resist posterior tibial translation in deeper knee flexion. These observations suggest that the two bundles act to limit tibial posterior laxity at different angles of knee flexion.

A selective cutting study by Race and Amis [39] found that the aPC was the primary restraint to 6 mm tibial posterior translation from 30 to 120 degrees knee flexion (Fig. 3). The pPC shared the load with the aPC across this arc of flexion, and became the dominant structure in deeper flexion. Although the pPC was tight in full knee extension, it did not contribute greatly to resisting posterior draw because its fibres were aligned in a proximal-distal direction then, and so were not aligned to resist tibial posterior translation. It is important to note that structures other than the PCL were dominant from full extension to 20 degrees knee flexion. This means, in a knee with increased posterior laxity close to full extension, that structures other than the PCL have been damaged, and that an isolated PCL reconstruction will not restore the laxity to normal here.

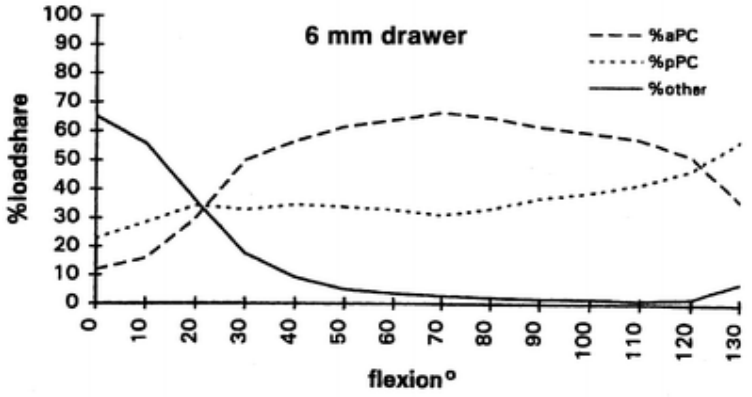

Fig. 3. Percentage contributions of the two bundles (aPC and pPC) of the PCL, and of 'other structures' to resisting 6 mm posterior tibial translation, across the range of knee flexion (from Race and Amis [39], with permission)

Tibial internal-external and abduction-adduction (valgus-varus) rotations

A number of studies have shown that the PCL is only a secondary restraint to these rotations in the intact knee. This is because the PCL, being near to the centre of the knee, has only a relatively small moment arm about the relevant axes of rotation, and so it is at a mechanical disadvantage. These effects will be discussed in sections below.

Posterolateral structures

Functional anatomy

The anatomy of the ligamentous structures around the posterolateral aspect of the knee is both complex and variable. Some structures, such as the arcuate and fabellofibular ligaments, are not always present [43, 48, 55]. In addition, the popliteofibular ligament was not recognised for some years, despite it being a direct ligamentous attachment from the femur to the fibula that is well-aligned to resist tibial external rotation [32]. The action of the popliteofibular ligament is compounded by the active tension in popliteus, which tends to internally rotate the tibia [50]. As a general rule, the lateral collateral ligament (LCL) and all other structures (i.e. the capsule, arcuate and fabellofibular ligaments) that attach to the lateral femoral condyle posterior to the LCL are tight in full knee extension, and slacken as the knee flexes. In contrast to the other structures, the popliteofibular ligament complex is isometric, and so it can stabilise the knee at all angles of flexion, while the LCL slackens significantly beyond 30 degrees knee flexion [47]. The structures listed above slant posteriorly as they pass from the femur to the tibia, and so they are well aligned to resist tibial posterior translation and also tibial external rotation. It follows that damage to these structures is associated with posterolateral rotational instability (PLRI), which results from a combination of these two components of motion (Fig. 4). In addition, the observation that these structures tighten as the knee extends fits in with the evidence from the posterior draw testing, that structures other than the PCL are most important at resisting this subluxation in the extended knee.

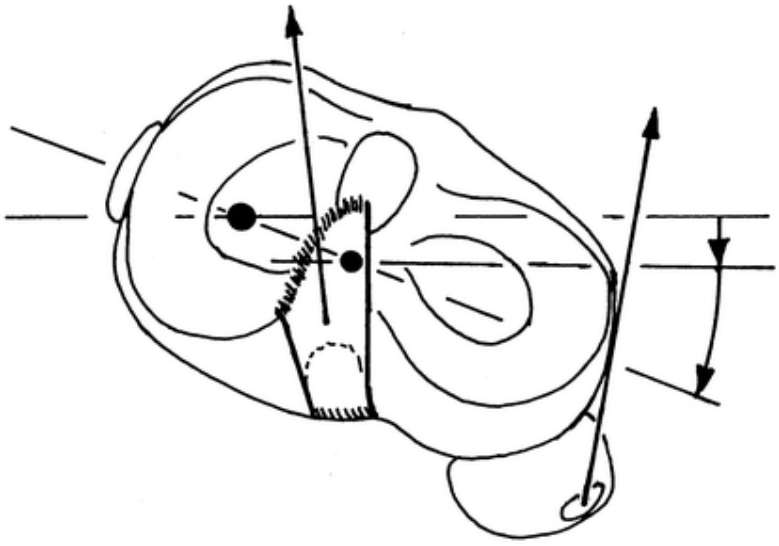

Fig. 4. Posterolateral tibial laxity arises from a combination of posterior translation, plus tibial external rotation (from Amis [2], with permission)

Strength of the posterolateral structures

The strengths of the lateral collateral ligament and popliteofibular ligament have been found in two studies: Maynard et al. [*32*] found strengths of 750 N and 425 N respectively, whilst Sugita and Amis [*47*] found strengths of 309 N and 186 N in specimens with a mean age of approximately 70 years. Thus it appears that the LCL is stronger than the popliteofibular ligament.

Posterior tibial translation

It was shown above that isolated PCL rupture leads to only a small increase in posterior tibial translation when the knee is extended (Fig. 2), and that other structures were then the primary restraint (Fig. 3). Conversely, isolated rupture of the posterolateral structures, induced by an impact onto the anteromedial aspect of the proximal tibia (which causes combined hyperextension and varus angulation) has its largest effect in the extended knee. After inducing this injury in vitro, the authors (I. Hijazi et al., unpublished) found that the intact posterior laxity of 3 mm increased to 10 mm at full knee extension, whereas this change was only from 5 mm to 6 mm at 90 degrees knee flexion, in response to 100 N posterior draw force (Fig. 5). This tendency for posterolateral damage to have the greatest effect on laxity with the knee extended is the opposite to the effect of PCL rupture, which is most evident at 90 degrees knee flexion (Fig. 2). The studies of Gollehon et al. [*13*] and Grood et al. [*15*] found that combined cutting of the PCL and the posterolateral structures led to approximately 25 mm tibial posterior translation at 90 degrees flexion. These results suggest that posterior draw tests alone are not able to show isolated posterolateral damage clearly. An associated phenomenon, tibial coupled rotation, provides the clue for diagnosis. A coupled rotation occurs as an automatic secondary motion during the draw test, resulting from the greater mobility of the lateral compartment [*2*]. Thus, tibial anterior draw induces tibial internal rotation [*3*] and posterior draw induces tibial external rotation. This coupled rotation increases significantly if the posterolateral structures have been ruptured, accentuating the motion of the fibular head during a posterior draw test (Fig. 4). This is sometimes known as a 'posterolateral draw test' if performed at 90 degrees knee flexion.

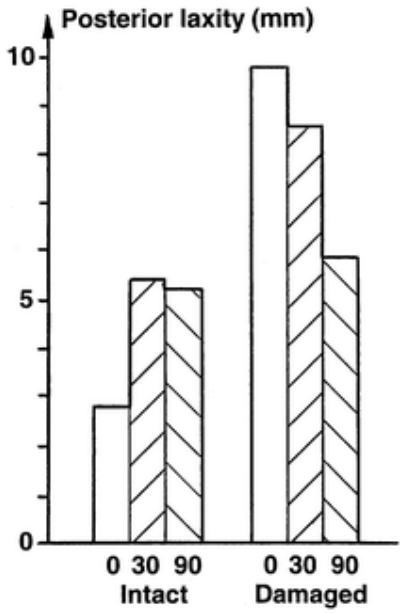

Fig. 5. Rupture of the posterolateral structures caused posterior tibial draw laxity at 100 N to increase greatly in the extended knee, but only a little at 90 degrees knee flexion (I. Hijazi et al., unpublished)

Tibial internal-external rotation

Because the posterolateral structures slant posteriorly as they pass distally, tibial internal rotation slackens them, and so posterolateral damage has no effect on internal rotation. Conversely, the posterolateral structures are dominant in resisting tibial external rotation. Isolated PCL sectioning has no measurable effect on tibial external rotation [*13*, *15*]. These studies found that posterolateral damage caused the largest increase in tibial external rotation around 30 degrees knee flexion. At 90 degrees flexion, the change in external rotation increased further when the PCL was also cut. Fig. 6 shows that posterolateral damage caused the greatest increase in tibial external rotation in the range 0 to 30 degrees knee flexion, with a smaller effect at 90 degrees flexion (I. Hijazi et al., unpublished). The studies of Veltri et al. [*51*, *52*] showed that the popliteofibular ligament complex was a primary restraint to tibial external rotation at all angles of flexion. This is because it remains well-aligned and tight throughout the range of knee flexion [*47*].

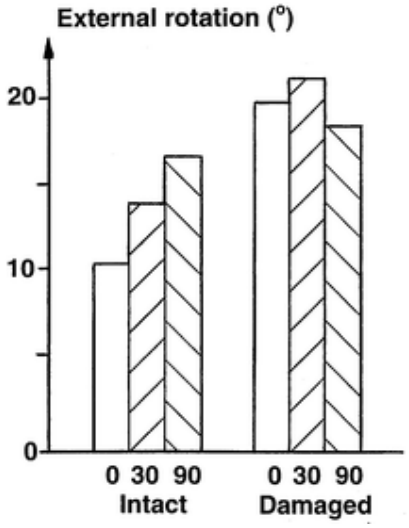

Fig. 6. Rupture of the posterolateral structures caused tibial external rotation laxity at 5 Nm torque to increase greatly in the extended knee, but only a little at 90 degrees knee flexion (I. Hijazi et al., unpublished)

"Posterolateral rotatory instability" (PLRI) describes the hypermobility of the lateral tibial plateau that is caused by damage to the posterolateral structures. If the PCL remains intact, then the tibial plateau as a whole does not fall posteriorly, and there is only an excessive tibial external rotation about a central axis. However, if the PCL is also ruptured, the tibia is then tethered medially, and there is a combination of posterior translation plus external rotation that gives excessive pathological motion of the fibular head (Fig. 4). This "coupled" rotation occurs automatically in response to a posterior translation force on the tibia.

It is clear that a significant increase in tibial external rotation at 0 to 30 degrees knee flexion is diagnostic for posterolateral damage. This is often demonstrated by the 'dial test', in which the feet are grasped and used to rotate the tibia externally: the foot of the damaged limb moves further from the 'twelve o'clock' position.

Tibial adduction (varus) rotation

The lateral collateral ligament is the primary passive restraint to tibial adduction at 5 and 25 degrees knee flexion, providing 55 and 69 percent of the resistance to 5 degrees adduction [*14*]. This corresponds to it being both tight and well-aligned here, prior to slackening in deeper knee flexion [*47*]. The posterior capsular structures, such as the arcuate and fabellofibular ligaments, contributed a further 13 percent at 5 degrees knee flexion, but slackened as the knee flexed further [*14*]. The ACL is tight near knee extension, and acts as a secondary restraint to varus joint angulation. Although the ACL is a stronger ligament than the LCL, it has a much smaller moment arm about the adduction pivot point, within the medial femoral condyle. Noting this, Grood et al. [*14*] warned that isolated rupture of the lateral structures may not cause a large

increase in adduction laxity during clinical tests, because the tensed ACL acts as a secondary restraint.

Posteromedial structures

Functional anatomy

The medial collateral ligament (MCL) consists of two distinct layers. The long, parallel-fibred superficial layer (sMCL) attaches to the femur just antero-distal to the adductor tubercle, passes distally, and attaches tangentially to the tibial periosteum over a large area approximately 6–8 cm distal to the joint line. The deep MCL (dMCL) attaches immediately distal to the femoral attachment of the sMCL, and immediately below the joint line on the medial margin of the tibial plateau, and so has much shorter fibres. En route, the deep surface attaches to the periphery of the medial meniscus [*5*, *35*, *44*, *54*].

Posterior to the parallel longitudinal fibres of the sMCL the deep and the superficial layers merge, and there is less agreement about the description of the structures. Hughston and Eilers [*23*] published diagrams of distinct fibre bundles in the posteromedial capsule and identified a distinct ligament—the posterior oblique ligament. Observations by the authors have confirmed those of Warren and Marshall [*54*], finding a wide spread of fibres in the capsule, rather than a distinct ligamentous band, so it is suggested that the whole of this structure should be described simply as the 'posteromedial capsule' (PMC). In general, these fibres are all stretched by fully extending the knee (Fig. 7), and slacken with knee flexion (Fig. 8). Their wide femoral attachment runs posteriorly from that of the sMCL, and passes proximal to the prominent posterior medial femoral condyle. The condyle causes the capsule to bulge out as the knee extends, thus tightening it. In the extended knee the PMC fibres pass posteriorly and distally towards the joint line, and so can be seen to tighten and resist tibial posterior translation and tibial internal rotation when the tibia is loaded in those directions (Fig. 7). These posteromedial capsular structures slacken and buckle as the knee flexes, and are carried deep to the posterior margin of the sMCL in deeper flexion (Fig. 8). Distal to the joint line, the PMC fibres attach to the posteromedial rim of the tibia and below this are then aligned anterodistally, converging towards the sMCL, and attaching immediately posterior to it.

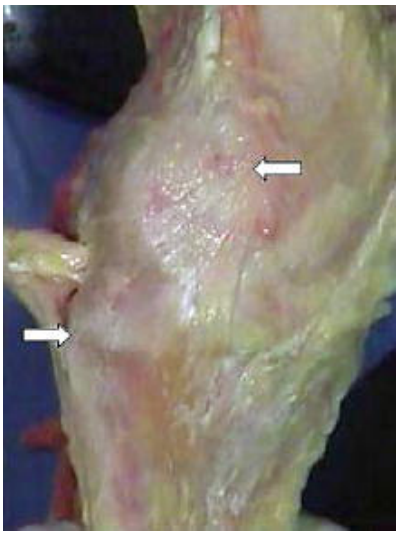

Fig. 7. Medial aspect of left knee. When the knee is extended, the fibres of the posteromedial capsule are tensed from the medial epicondyle *(top arrow)* to the posterior rim of the tibial plateau *(bottom arrow)* and are aligned to resist both tibial posterior draw and internal rotation

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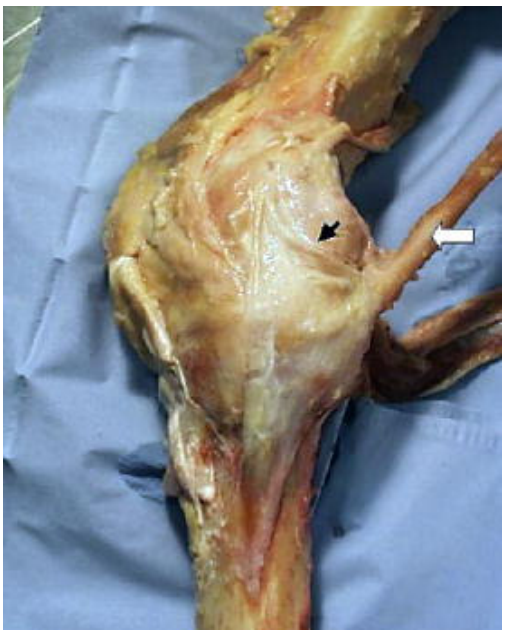

Fig. 8. Medial aspect of right knee. As the knee flexes, the posteromedial capsule slackens *(black arrow)* and is carried deep to the posterior border of the superficial MCL. The semimembranosus tendon *(white arrow)* pulls the capsule proximally, thus keeping it slack where it crosses the joint line. The long superficial MCL remains tight

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It has been suggested [46] that the PMC is tensed by the semimembranosus tendon sheath. However, because the tendon tension acts approximately parallel to the femur, it pulls the tendon sheath and PMC proximally when the knee is at a low angle of flexion. The proximal part of the capsule, that crosses the joint line, is therefore kept slack by this tendon action (Fig. 8), and so it is then unlikely to stabilise the knee.

Ligament strength

Several authors have produced data on the strength of the MCL: Kennedy et al. [26] 665 N, Marinozzi et al. [31] 465 N, Trent et al. [49] 516 N. However, none of them tested the different fibre bundles separately. It has long been known that different patterns of MCL damage can occur on the medial aspect of the knee following an abduction (valgus) injury [45]. Ligaments fail at approximately 20 percent tensile strain, and so a valgus opening injury will rupture the short dMCL before the longer fibres of the sMCL reach their ultimate strain [1]. Tensile tests of each of the structures found that the dMCL failed at 195 N at 7 mm elongation, while the sMCL and posteromedial corner both extended approximately 12 mm to reach loads of 784 N and 418 N, respectively (J.R. Robinson et al., unpublished). This means that, even in specimens with an average age of approximately 80 years, the total MCL strength was in excess of 1 kN, which is greater than has been thought previously, and higher than that of the ACL in this age range. Further, the data suggests that dMCL rupture occurs at approximately 7 degrees valgus joint opening, with complete MCL failure by 12 degrees.

Tibial posterior translation

The role of the posteromedial structures in controlling tibial posterior translation has only been studied previously in the PCL-deficient knee at 90 degrees flexion. Ritchie et al. [42] showed that posterior translation was reduced if the tibia was held in fixed internal rotation. Dividing the posteromedial capsule resulted in an increase in tibial translation from 4.1 to 4.5 mm (10%). Further dividing the sMCL resulted in an increase in tibial translation to 20.3 mm (350% increase). Thus the sMCL appeared to be the most important secondary restraint, following PCL rupture, with the knee flexed.

The posteromedial structures become tight and well-aligned to resist tibial posterior translation as the knee extends (Fig. 7). Results available at the time of writing suggest that the posteromedial structures make a significant contribution to resisting tibial posterior translation with the tibia in internal rotation, in both the intact and PCL-deficient knee. With sequential cutting, the posterior laxity increased from 6.5 mm when intact, to 10.2 mm after cutting the PCL and MFL, to 13.2 mm after cutting the PMC, at 15 degrees flexion (C.M. Gupte et al., unpublished).

Tibial internal-external rotation

Kennedy and Fowler [25] showed that a tibial external rotation of 45 degrees at 90 degrees of flexion tore the "capsular ligament" (dMCL) and that the

"superficial tibial collateral ligament" (sMCL) remained intact. Only with further rotation did this part of the medial structures fail. In contrast, Warren et al. [53] found that cutting the deep MCL in an otherwise intact knee had a "minimum effect on restraining rotation between the femur and the tibia". Cutting the long fibres of the sMCL first, however, resulted in a 2° increase in external rotation at 0° knee flexion and 4° at 90° flexion.

The authors have shown that in the intact knee both the dMCL and the sMCL restrain tibial external rotation. The dMCL provides more restraint at higher angles of flexion, with the sMCL being important throughout the arc of flexion. The fibres of the posteromedial capsule (PMC) are aligned such that they are slackened by tibial external rotation, and therefore do not contribute to restraining this movement. (J.R. Robinson et al., unpublished data). Although the dMCL is not the strongest component of the medial ligament complex, its position and short length mean that its tension rises quickly with tibial external rotation, and that is why it is sometimes ruptured beneath an intact sMCL.

The authors have found that in the intact knee both the PMC and the sMCL are important structures in restraining tibial internal rotation. The PMC acts when the knee is in extension and at low angles of flexion, with the sMCL providing an increasing contribution with progressive knee flexion. In the extended knee, cutting the sMCL and dMCL made no significant difference to tibial internal rotation, but cutting the PMC increased internal rotation from 8 to 23 degrees (J.R. Robinson et al., unpublished data).

The PMC did not at as a significant restraint to tibial internal rotation above 30 degrees knee flexion, when the sMCL appeared to become more important. Cutting the sMCL in isolation increased tibial internal rotation by approximately 7 degrees at 60 to 90 degrees knee flexion (J.R. Robinson et al., unpublished data).

Overall, the evidence shows that the primary restraint to tibial internal rotation is the PMC when the knee is at or close to extension, and the sMCL when the knee is flexed.

Tibial abduction-adduction (valgus-varus) rotation

Kennedy and Fowler [25] believed that the primary lesion of valgus instability was a tear of the capsular ligament (dMCL). Warren et al. [53] noted a significant increase in valgus laxity after sectioning the long parallel fibres of the sMCL. They were unable to demonstrate any significant valgus opening of the knee by sectioning the dMCL and posterior capsule, and believed that valgus laxity in extension was due to damage to the cruciate ligaments.

Increases in valgus laxity were measured in sequential cutting studies by Mains et al. [29] and Nielsen et al. [33]. Mains et al. cut the sMCL, dMCL, PMC, ACL and PCL in isolation and applied a 3.5 Nm abduction moment. Cutting the sMCL increased valgus rotation laxity 2.5 degrees at 0 degrees knee flexion, 5 degrees at 20–25 degrees flexion, and 4 degrees at 45 degrees flexion. Section of the dMCL also caused 4 degrees extra valgus laxity at 45

degrees knee flexion. The sMCL was described as the prime restraint to valgus rotation, with the dMCL as having a relatively minor role. The PMC appeared subjectively to resist valgus rotation with the knee in hyperextension, but this was not measured. Nielsen et al. found that maximum valgus laxity occurred at 60 degrees of knee flexion. When applying a 2 Nm torque, cutting the entire MCL increased valgus laxity only 2 degrees. Further transection of the capsule increased laxity to 9 degrees. Cutting the ACL caused a further increase to 24 degrees. However the contribution of each of the structures was not evaluated in these studies, so they did not define the primary and secondary restraints to valgus loads.

Grood et al. [*14*] found that the MCL was the primary restraint to valgus laxity when the medial aspect of the tibiofemoral joint was opened by 6 mm at 25 degrees knee flexion, resisting 78% of the load, but did not test the dMCL and sMCL separately. They noted that the PMC slackened with flexion, so it accounted for only 4 percent of the total restraint at 25 degrees flexion. With the capsule tighter at 5 degrees flexion, this contribution increased to 18 percent. The ACL, PCL and posterior capsule were found to be secondary restraints (Fig. 9) It was concluded that the capsular structures provided important attachments for the menisci but did not have primary roles in limiting valgus (abduction) laxity.

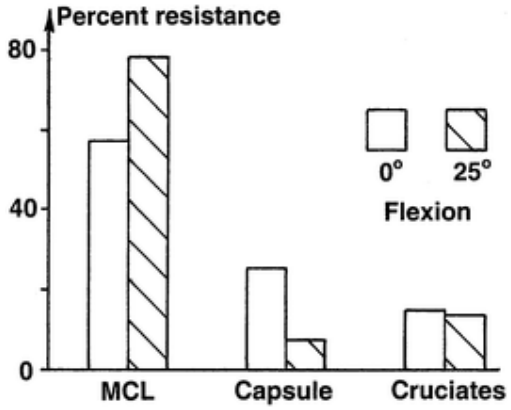

Fig. 9. Contributions to resisting tibial abduction (valgus) rotation, at 5 and 25 degrees knee flexion. Note that flexion slackened the posterior capsule, decreasing its role (from Grood et al. [*14*], with permission)

The parallel longitudinal fibres of the superficial MCL are the primary restraint to valgus laxity throughout the arc of knee flexion. Damage to this structure causes increased valgus rotation of 3 degrees in extension and 5 degrees at 30 degrees flexion. This correlates well with the clinical finding that valgus laxity due to damage of the sMCL is more demonstrable with the knee in slight flexion, because the tight posterior capsule protects the extended joint. There is a lesser contribution from the dMCL (approx 3 degrees from 15 to 90 degrees flexion). Although the sMCL was found to be the primary restraint to tibial valgus rotation at all angles of knee flexion, the PMC made a significant contribution when the knee was extended. At 0 degrees knee

flexion, the PMC resisted 29 percent of the valgus moment, which reduced to 14 percent at 30 degrees flexion and to less than 5 percent at 90 degrees flexion (J.R. Robinson et al., unpublished).

In contrast to the neat cutting studies in vitro, clinical cases will not have structures damaged in isolation. Even if only one major ligament bundle has been ruptured, other related structures will have been stretched irreversibly. This means that clinical examination will reveal larger laxities than those reported above.

Meniscofemoral ligaments

Functional anatomy

Two meniscofemoral ligaments (MFLs) may connect the posterior horn of the lateral meniscus to the intercondylar aspect of the medial femoral condyle, attaching on either side of the PCL attachment. The ligament of Humphry (aMFL) passes anterior to the PCL and attaches distally, close to the articular cartilage (Fig. 10a). The ligament of Wrisberg (pMFL) passes posterior to the PCL and attaches proximally, close to the roof of the intercondylar notch (Fig. 10b). Friederich and O'Brien [*10*] have observed that the aMFL is tense in flexion, whilst the pMFL is tense in extension. This reciprocal tightening and slackening of the MFLs may be functionally similar to the two bundles of the PCL. At least one MFL is present in 93% of specimens [*16*, *57*], whilst both ligaments co-exist more frequently in younger specimens [*18*]. The latter suggests that they may degenerate with age.

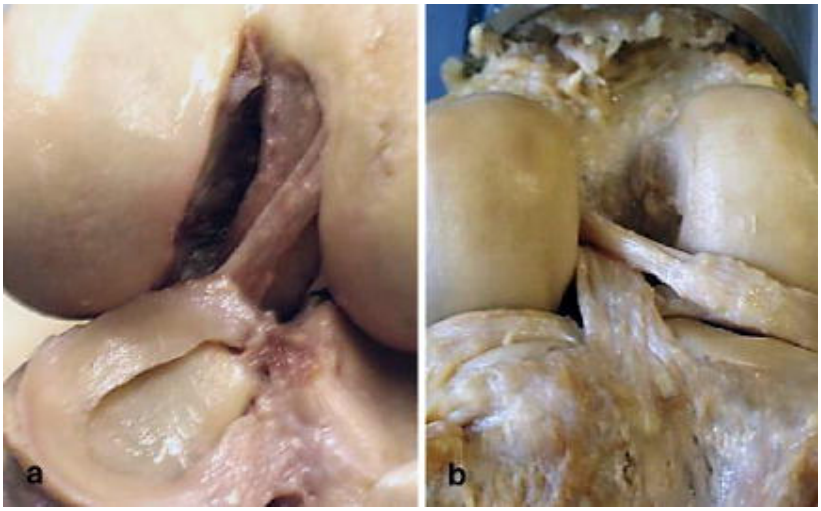

Fig. 10. a The anterior meniscofemoral ligament of Humphry viewed in the flexed right knee after ACL excision and tibia subluxed anteriorly. The aMFL fibres slant across the distal surface of the PCL, which has its fibres aligned in a sagittal plane. The MFL attaches close to the articular cartilage and posterodistally to the lateral meniscus. The distal attachment is hidden by the

ACL in the intact knee. **b** Posterior view of right knee. The posterior meniscofemoral ligament of Wrisberg slants across the posterior aspect of the PCL. It attaches proximally in the femoral intercondylar notch, and to the posterior horn of the lateral meniscus

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Strength

The aMFL is, on average, smaller than the pMFL [21]. They have a tensile strength (302.5 N and 300.5 N respectively) comparable to that of the pPC, whilst their elastic modulus is similar to that of the aPC [19]. Kusayama et al. [27] found a similar mean strength for MFLs of 297 N.

The high prevalence of the MFLs, together with their significant properties, supports the hypothesis that they may play a functional role in the knee.

Tibial posterior translation

Radioevitch [41] proposed the MFLs as "third cruciate ligaments". Others [8, 27] have suggested that they may act as secondary restraints to posterior tibial translation in the PCL-injured knee. Last [28] proposed that the MFLs are involved in controlling the motion of the lateral meniscus in conjunction with the tendon of popliteus during knee flexion, thus maintaining the congruency of the knee joint. There have previously, however, been no objective studies to confirm these hypotheses.

It has now been proven that the MFLs act as secondary restraints to tibial posterior translation [17]: there was a significant increase in laxity when the MFLs were cut, in both the intact knee and in the PCL deficient knee. This was most clear when the knee was flexed. In the intact knee at 90 degrees flexion, the MFLs contributed 28 percent of the resistance to posterior draw; this contribution rose to 70 percent in the PCL deficient knee. Thus, the MFLs act in synergy with the PCL when it is intact, and act in a similar manner to the PCL when it has been ruptured.

Tibial internal-external rotation

The oblique orientation of the MFLs suggests that they may be involved in limiting tibial internal rotation, but a selective cutting study in progress at the authors' laboratory (C.M. Gupte et al., unpublished) has not found a significant effect. This probably results from the MFLs crossing the knee close to the axis of tibial rotation, so that their tension will have little effect on rotation.

Conclusions for the surgeon

The PCL was shown to have a divergent fibre structure that could be described as consisting of two functional bundles of fibres. These were shown to have different patterns of tightening and slackening, and thus to contribute to resisting tibial posterior draw at different angles of knee flexion. This ligament

is clearly not an isometric structure, and so differs significantly from the ACL, and an isometric reconstruction would not fit in with the natural behaviour. This was shown by Race and Amis [40] who also, along with Harner et al. [20] and Mannor et al. [30], showed that a double-bundled PCL reconstruction re-established normal tibial posterior laxity significantly better than did single-bundled reconstructions. This work in vitro, however, has not yet led to reports of significant advantages in clinical results.

The posterolateral structures do not have a history of good results following reconstructive surgery, and it appears that more work is needed to optimise reconstruction methods. The treatment options were reviewed by Covey [9]. This biomechanical review has shown that the posterolateral corner is a primary restraint to tibial adduction (varus) and to tibial external rotations, as well as to posterior translation when the knee is near extension and thus in a functional load-bearing posture in gait. It may be argued, therefore, that the posterolateral structures may be more important for knee function than is the PCL, because of their multiple roles. It is hoped that better understanding of the biomechanics of the posterolateral structures will allow reconstruction methods to be improved.

The medial and posteromedial structures have been relatively neglected in recent years, and it is only now that basic data on their roles in stabilising the knee are being produced. It is clear that, in addition to the role of the superficial MCL as the primary restraint against tibial abduction (valgus) rotation, these structures also act to control tibial posterior translation, particularly when the tibia is in internal rotation. Knee extension and tibial internal rotation both tense the posteromedial structures in an efficient direction to resist both tibial posterior translation and internal rotation.

With the emergence of a role for the MFLs as secondary restraints to tibial posterior translation, it may be useful to identify these structures during arthroscopy and imaging, particularly in the PCL-deficient knee. It may also be of benefit to attempt to preserve them during PCL reconstruction. The authors now believe that intact MFLs help to stabilise the PCL-deficient knee, and so contribute to the relatively good functional status following isolated PCL rupture.

Added together, it is apparent that the combined actions of the posteromedial and posterolateral structures of the knee are capable of controlling tibiofemoral joint laxity in ways that have traditionally been thought to be the role of the PCL, particularly when the knee is extended. Noting that the bulk of the PCL is slack when the knee is extended, it appears by default that the PCL may be so strong because of needing to resist large forces when the knee is flexed. Further work is needed to gain a greater understanding of the function of the PCL and its interactions with the posterolateral, posteromedial, and meniscofemoral ligaments. This should give greater insight into the enigma of the lack of functional deficit following isolated PCL rupture, and guidance for the development of better reconstructive procedures.

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