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The Biomechanics of the Anterior Cruciate Ligament and Its Reconstruction

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There is a vast amount of literature available discussing the movements of the knee joint and the function of the ligaments controlling these movements. (Brantigan & Voshell, 1941)

1. Introduction

The anterior cruciate ligament (ACL) continues to be a major topic in orthopaedics and biomechanics. This is due to the high incidence of injury and its disabling consequences. That the disabling consequences have not yet been completely overcome guarantees continued interest.

Injury to the ACL is associated with a range of problems, such as pain, instability, meniscal damage and osteoarthritis (Noyes et al., 1983; Segawa et al., 2001). This can be due to injury to other structures at the time of ACL injury, e.g. meniscus and articular cartilage, as well as developments secondary to the initial injury (Segawa et al., 2001). Although isolated injury to the ACL is considered uncommon, surgical transection of the ACL induces cartilage degeneration so reliably it is used to induce osteoarthritis in experimental animals (Desrochers et al., 2010).

Simple repair is effective for some ligaments, but such repair of the ACL has had poor results, and the orthopaedic world currently awaits the development of a useful scaffold that can be placed between the torn ends of the ligament to promote effective healing (Murray, 2009). In the interim a popular choice of treatment with positive results is surgical reconstruction (replacement), using a graft fashioned typically from the patellar ligament or from hamstring tendons (Woo et al., 2002). But despite positive outcomes from ACL reconstruction problems still persist after ‘successful’ surgery, especially osteoarthritis (Øiestad et al., 2010). It is only possible to speculate that kinematic differences that remain after, or are caused by, surgical reconstruction lead to osteoarthritis (Scanlan et al., 2010), but post-surgical kinematic differences may lead to cartilage loading differences, which then predispose the knee to osteoarthritis (Andriacchi et al., 2004, 2006). These loading differences may lead to cartilage areas subsequently sustaining higher or lower loads or loads of a different nature (e.g. tension, compression) (Chaudhari et al., 2008).

Development of a suitable ligament substitute is a straightforward matter: (1) determine the nature of the ligament, and (2) design a replacement that replicates this nature tolerably.
well. But straightforward is not simple, and the following review will highlight what is known, and what yet to be known, about the ACL and its substitutes.

2. Anatomy

The ACL spans the tibiofemoral joint space, and forms a cross with the posterior cruciate ligament. The proximal attachment is located posteriorly on the medial side of the lateral femoral condyle in a semilunar area with the straight border facing forward and slightly down. The ligament attaches distally on the anterior aspect of the tibia, just anterior and lateral to the anterior tibial spine (Fuss, 1989; Girgis et al., 1975). There is some variation in the description of separate parts of the ligament at the macroscopic level. The pattern with greatest acceptance shows two bundles, anteromedial and posterolateral (Furman et al., 1976; Harner et al., 1999; Inderster et al., 1993; Katouda et al., 2011; Steckel et al., 2007). Harner et al. (1999) and Steckel et al. (2007) described the anteromedial bundle as arising from the proximal part of the femoral attachment, with the posterolateral bundle arising from the distal portion. On the tibia the anteromedial bundle attaches medially and the posterolateral bundle attaches laterally.

But these findings are not unchallenged. Fuss (1989) and Odensten and Gillquist (1985) found no such subdivisions by dissection and histological examination. Clark and Sidles (1990) referred to anteromedial and posterolateral bands but found them “indistinct”, and described an anterior furrow that gave the ligament the appearance of being bifid. In the other direction, Amis and Dawkins (1991) and Norwood and Cross (1979) reported three bundles, anteromedial, intermediate and posterolateral.

The acceptance of the two bundle anatomy is so widespread today as to probably make any alternative claim go largely unnoticed. Nevertheless, without rejecting or accepting the preceding descriptions it is biomechanically meaningful to describe anteromedial and posterolateral ligament fibres, without assuming a plane of separation, as the cross-sectional shape of the ligament has an anteromedial-posterolateral long axis (Harner et al., 1995).

3. Normal function

The function of a ligament is to resist movement by resisting elongation caused by that movement, which may be rotational, translational or both. To understand the function of a ligament during a rotational movement, some consideration must be given to the relationship of the ligament to the axis of rotation. It is fundamental then to understand normal knee kinematics and ACL behaviour during knee motion.

3.1 Kinematics of the normal knee

The principal motion at the knee joint is flexion-extension in the sagittal plane, with a normal passive range of 130 to 140° and an associated 5 to 10° of hyperextension (Nordin & Frankel, 1989). The knee also has smaller ranges of motion in the horizontal and coronal planes, with passive and active ranges of approximately 70° of combined internal-external rotation in the horizontal plane, and minimal valgus-varus rotation in the coronal plane, although these figures are dependent upon the amount of flexion and extension in the joint (Hollister et al., 1993). The ranges of internal and external rotation are minimal and maximal at zero and 90° of flexion, respectively (Bull & Amis, 1998).
With reference to flexion-extension, there is still debate about the exact location of this axis of rotation (Smith et al., 2003). However, four schools of thought have emerged that present different arguments as to the nature and location of this axis.

The instantaneous axis is thought to lie in both the coronal and horizontal planes and moves in a curved or elliptical pathway as the knee undergoes rotation and translation. This approach positions the centre of mass of the joint and the movement of the joint primarily within the sagittal plane. The articular surface of the lateral femoral condyle has a greater variation than that of the medial femoral condyle. In the fully flexed position this axis is closest to the joint surface, which allows the ligaments of the joint (ACL and collateral ligaments) to slacken. In extension the axis is further away from the joint line and the same ligaments become tense (Brantigan & Voshell, 1941). The major criticism of this approach resides in the fact that condyles of the femur are varied in three dimensions rather than two dimensions, the latter of which is the only way to place the axis of rotation purely in the sagittal plane.

More recently a fixed axis of rotation offset to both the frontal and horizontal planes has been proposed (Hollister et al., 1993; Martelli & Finskerova, 2002). The first of two fixed axes, the posterior condylar axis, runs from the medial to the lateral side of the joint and is also inclined posteriorly and distal to the joint centre. This first axis is effective from 15 to 150° of knee flexion and is defined as passing through the origins of both the medial and lateral collateral ligaments and the intersection of the anterior and posterior cruciate ligaments. This axis is considered to also be inclined at an angle of 7° to the sagittal plane, thus closely equating its position to the epicondylar line of the joint (Churchill et al 1998). As the knee moves into extension this axis changes to a second fixed axis of rotation, the distal condylar axis, that is located proximal to the distal aspect of the intercondylar notch (Elias et al., 1990).

The helical axis of rotation can perhaps be more easily described and understood as simultaneous rotation around and translation along a specified axis, like the action of a nut rotating and translating along a threaded bolt. This axis defines motion of the joint as a series of displacements rather than relative positions (Bull & Amis, 1998). This approach represents an instantaneous axis that the joint can both translate and rotate about (Blankevoort et al., 1988; Soudan et al., 1979). The screw-home mechanism of the joint, where there is an internal rotation and posterior translation of the femur on the tibia during extension is an example of the need for a helical axis of rotation. The main criticism with the helical axis approach to joint motion is one associated with accuracy. For example, for the helical axis to be consistent there would be a requirement that the motion of the joint is firstly described precisely in order to determine the exact location of the helical axis. However, in order to define exact movement of a joint (flexion-extension, internal-external rotation or abduction-adduction rotation) an axis of rotation is required.

The final school of thought incorporates an infinite axis of rotation theory where the exact knee motion depends on a number of factors such as ligament and soft tissue restraints, bony architecture, passive motions and external loads and forces during weight bearing. Blankevoort et al. (1988) and La Fortune et al. (1992) found no screw-home mechanism during weight bearing in vivo, and suggested that the knee has a range of motion that is specifically dependent upon passive characteristics. For example, the screw-home mechanism can be found to be overcome by external passive forces during movement (Sanfridsson et al., 2001; Smith et al., 2003).
The choice of axis for the principal motion of knee depends largely on the degree of accuracy that is needed in whatever application it is being used. For the installation of total knee joint prosthesis the complex axis of rotation of the knee is usually replaced by a fixed axis of rotation. However, in this application careful selection of the type of prosthesis is required (Kurosawa et al., 1985; Siu et al., 1996). Alternatively, if the accuracy of determining the axes is not so stringent, as in the case of a clinical test, then the first approach, an instantaneous axis in the frontal and transverse plane, is seen as acceptable.

In the horizontal plane the knee joint rotates about a longitudinal axis that extends through the tibia, however variations in the understanding of the location of this axis still exist within the scientific literature. This axis does not intersect with the flexion-extension axis of the knee and it is firmly located within the tibia. This axis runs close to the medial intercondylar tubercle (Lehmkuhl & Smith, 1983) and also very close to the ACLs tibial attachment. Furthermore, it is directed posteromedially close to the femoral attachment site of the posterior cruciate ligament (Zatsiorski, 2002). Rotations around this axis are termed internal and external rotations of the tibia and are specified as such unless the coupled motions of the femur are also included (Smith et al., 2003). This axis of rotation is present due to the incongruence of the femoral and tibial articulating surfaces and is affected by ligamentous insufficiency. With the knee in full extension rotation around this axis is minimal as the ligaments are taut and the menisci are firmly held between the articulating surfaces. In addition, the tibial spines are lodged in the intercondylar notch (Levangie & Norkin, 2001). During knee flexion the complex arrangement changes and the condyles of the tibia and femur are free to move. At approximately 90° of knee flexion around 30° internal and 45° external rotation is present at the joint (Nordin & Frankel, 1989).

Several variations exist as to the exact location of this axis. Some research (Brantigan & Voshell, 1941; Hollister et al., 1993; Matsumoto et al., 2001; Shaw & Murray, 1974) has suggested that the axis is either fixed, passing through the medial femoral condyle or the intercondylar eminence, or as an instantaneous centre of rotation. Such wide contrasting opinion has resulted from the fact that the longitudinal axis of rotation in the knee is dependent upon many other factors than bony architecture.

3.2 ACL behaviour

The kinematics of the knee are complex and the ACL crosses the joint obliquely in three planes. There is the potential then that the function of the ACL is also complex, and this is shown by a voluminous literature. To try to impose order on a vast body of knowledge, the main movements relevant to ACL function are presented below first as separate, then combined where appropriate.

3.2.1 Flexion-extension

The ACL is not usually considered a restraint at end of range flexion or extension, but its load or strain behaviour is influenced by the knee’s position within these limits and is therefore of interest. The findings presented here do not relate to the entire ligament, as many authors have found the behaviour to differ between the anteromedial and posterolateral bands.

Several studies show reciprocal behaviour of the two bands, in that flexion causes the anteromedial band to lengthen and the posterolateral band to slacken, at least up to 120° (Amis & Dawkins, 1991; Bach et al., 1997; Hollis et al., 1991; Takai et al., 1993; Wang et al.,
1973). But this picture of simple reciprocity may be an over-simplification. Two of the above studies show that the anteromedial band length decreases in the first 10 to 30° of flexion before increasing further into flexion (Amis & Dawkins, 1991; Bach et al., 1997). And Bach and Hull (1998) showed the strains in both bundles dropped in initial flexion (15 to 30°), and then remained at zero; only after 90° did the anteromedial band strain increase. Inderster and coworkers’ (1993) results show that the length of the anteromedial band remains roughly constant to 90°, with the posterolateral band slackening from full extension.

3.2.2 Anterior translation
The ACL is the primary restraint to anterior translation, bearing 80 to 90% of the load (Abbott et al., 1944; Butler et al., 1980; Furman et al., 1976; Takai et al., 1993). Ligament strain (Ahmed et al., 1987; Bach & Hull, 1998; Berns et al., 1992; Beynnon et al., 1992; Beynnon et al., 1997; Fleming et al., 2001; Gabriel et al., 2004; Sakane et al., 1997; Takai et al., 1993; Torzilli et al., 1994) or load (Livesay et al., 1995; Markolf et al., 1995) increases with an anterior force on the tibia. When the ACL is cut anterior translation increases (Diermann et al., 2009; Fukubayashi et al., 1982; Hsieh & Walker, 1976; Markolf et al., 1976; Oh et al., 2011; Reuben et al., 1989; Wrobble et al., 1993; Yoo et al., 2005; Zantop et al., 2007), or a given translation is resisted by a lesser force (Butler et al., 1980; Li et al., 1999; Lo et al., 2008; Wu et al., 2010).

And while the function of the whole ligament is beyond question here, that of the two bands is another matter. Studies have shown that posterolateral band loading on anterior force is either greater than or equal to that in the anteromedial band in extension, and with slackening of the posterolateral band in flexion a greater proportion of load is borne by the anteromedial band (Amis & Dawkins, 1991; Gabriel et al., 2004; Sakane et al., 1997; Takai et al., 1993). In support of this concept of the posterolateral band being more functional in flexion than extension, Zantop et al. (2007) measured the anterior translation on anterior force in a series of cadaveric knees before and after resection of either the anteromedial or posterolateral band. Deficiency of the posterolateral band led to greater translation increase at 30° flexion, with greater increases seen with anteromedial band deficiency at 60 and 90°.

The findings of two other studies make the relation between the two bundles less clear. Bach and Hull (1998) found no difference in strain between bands on anterior force through flexion. Wu and colleagues (2010) calculated that ligament forces were greater in the anteromedial band than the posterolateral band at all flexion angles, with the peak anteromedial band force at 30° flexion, and the posterolateral band contribution peaking full extension. So although Wu and co-workers’ findings on the posterolateral band function reflected those above, their finding that anteromedial force was greatest at low flexion led them to conclude that the sharing of load bearing is not so much reciprocal, with the anteromedial and posterolateral bands functioning in high and low flexion, respectively, but complementary, meaning here that peaks and troughs are much more similar.

3.2.3 Internal rotation
The ACL may act as a restraint to internal rotation, but there is no widespread agreement. Studies where ACL-intact knees have been subjected to an internal rotation torque have shown increases in ACL strain, both in vitro (Ahmed et al., 1987; Bach & Hull, 1998) and in vivo (Fleming et al., 2001), and ACL force in vitro (Kanamori et al., 2000; Markolf et al., 1990). Results from an in vivo patient study, as well as transection studies in cadaver knees
have shown an increase in internal rotation range compared to the ACL-intact state (Hemmerich et al., 2011; Kanamori et al., 2000; Lipke et al., 1981; Markolf et al., 2009; Oh et al., 2011; Wroble et al., 1993). These differences though were typically small though statistically significant; Wroble et al. (1993) concluded their results were “clinically unimportant”.

However other transection studies in cadaver knees have not shown an increase in internal rotation range (Lane et al., 1994; Reuben et al., 1989; Wünschel et al., 2010), and Lo et al. (2008) showed no increase in ACL force on internal rotation in vitro. In an in vivo study, Hemmerich and colleagues (2011) found that ACL-deficient patients showed an increase in internal rotation range, but at 0° flexion and not at 30°.

So the effect of the ACL on the range of internal rotation is unclear. Lane et al. (1994) and Oh et al. (2011) concluded that the ACL is unlikely to resist rotation as its central position in the knee is too close to the rotational axis. Interestingly then, Lipke et al. (1981) transected the lateral collateral ligament and posterolateral structures in addition to the ACL. While cutting these other structures in the ACL-intact knee had no effect on internal rotation range, after they were cut in the ACL-deficient knee there was an increase in range. They concluded that cutting the ACL shifted the rotation axis medially. This is supported by other findings that show a medial shift of the rotation axis in the ACL-deficient knee (Mannel et al., 2004; Matsumoto, 1990), although not by others that show no change in position (Shaw & Murray, 1974).

Studies addressing function of the two bands also conflict. Bach and Hull (1998) found no difference in band strain on internal rotation, whereas Lorbach et al. (2010) found that transection of the posterolateral band increased internal rotation on rotational torque at 30° flexion, and was not significantly increased by transecting the anteromedial band.

### 3.2.4 Combined anterior translation/internal rotation

There is an apparent link between anterior translation and internal rotation of the tibia. Fukubayashi and co-workers (1982) found in a series of cadaver knees that an anterior force of up to 125N applied to the tibia resulted in anterior translation and internal rotation, and that the rotation ceased after the ACL was cut. When the knees were constrained to move only in anterior translation the measured translation was substantially less. In other cadaveric studies, application of a 100N anterior force produced coupled internal rotation (Hollis et al., 1991), and simulated 200N quadriceps contraction caused anterior tibial translation coupled with internal rotation (Li et al., 1999). Internal rotation due to anterior force is not a universal finding, with Lo et al. (2008) showing a 50N anterior force (with a 100N joint compressive force) did not elicit a significant increase in rotation.

Many authors have measured the opposite of this, i.e. anterior translation resulting from applied internal rotation torque; most of these also applied a valgus torque, and they will be reviewed later. Kanamori et al. (2000) measured an anterior translation on internal rotation torque (10N m) that increased significantly after ACL transection. Two studies investigated tibial translation with internal rotational torque and joint compression force. Lo and colleagues (2008) found no significant increase in anterior translation on a 5N m rotational torque, although it was at the same level as with a 50N anterior force. Oh et al. (2011) found that anterior translation upon large internal rotational torque (17N m, with an 800N compressive force) doubled after ACL transection, and concluded that internal rotation torque loads the ACL due to anterior tibial translation.
The issue of coupled anterior translation and internal rotation is clearly the major kinematic issue in ACL reconstruction research, so it is well to discuss this here. Anterior force causes anterior translation that is increased if the tibia is allowed to internally rotate. We propose that pure anterior translation is possible with a low load in the ACL, but with higher load the ACL tethers the medial tibial condyle, and if unconstrained produces internal rotation. (While Nordt et al. (1999) suggested that the increased resistance of medial compared to lateral structures gives rise to internal rotation in this case, and we agree that these structures must allow rotation, Fukubayashi et al. (1982) showed loss of internal rotation when only the ACL was cut.) An imaginary horizontal line across both tibial condyles would then translate anteriorly initially, then with a larger load may appear to translate anteriorly, but would actually diverge laterally. This case is illustrated in Figure 1(a).

Internal rotation torque causes internal rotation, and appears to cause a measurable anterior translation. But neither Kanamori et al. (2000) nor Oh et al. (2011) measured rotation with translation constrained. Fukubayashi et al. (1982) showed that pure anterior translation was restricted compared to anterior translation with unconstrained internal rotation, and it will be seen in 3.2.5 that valgus rotation is greater with internal rotation allowed than constrained. We know of no other studies that have measured rotation this way, so we hesitate to call rotation and translation coupled. Furthermore we know of no explanation of coupled translation that allows us to understand why it occurs. But the effect appears to be greater with ACL deficiency, and as such it is something better to be understood. We think the published data are consistent with a simpler interpretation provided by Lipke et al. (1981), Mannel et al. (2004) and Matsumoto (1990), in that ACL transection causes a medial shift of the rotational axis. Figure 1(a) shows the result of anterior translation and internal rotation about a central axis, but it also looks like the tibia has rotated about an axis just medial to its medial border; Figure 1(b) shows the end result of such a rotation. We ask whether it is possible to detect the difference between these two tibial movements on an actual knee. As the centre of the tibia in Figure 1(b) is travelling along an arc, it should come to rest slightly more medially than in Figure 1(a). Results of studies in vivo (DeFrate et al., 2006) and in vitro (Li et al., 2007; Seon et al., 2010; Van de Velde et al., 2009) show that transection or rupture of the ACL increases medial tibial translation. So the ACL-deficient knee appears to either combine anterior translation, internal rotation and medial translation, or the more parsimonious alternative of internally rotating about a medially shifted axis.
This also concurs with Reuben and colleagues' (1989) conclusion that ACL deficiency causes anterior translation “about a pivot point that is medially based”. Translation around a pivot, or axis, is of course rotation. We suggest that the ACL prevents the wrong sort of rotation, that being potentially any rotation about an axis to which the knee is not accustomed. In this case, the ACL appears to prevent anterior translation of the medial tibial condyle, that is rotation about a medial axis.

3.2.5 Valgus rotation
The knee joint is most restricted in motion in the coronal plane, but interesting findings have emerged from studies involving the application of torque in this plane. Inoue and co-workers (1987) subjected a series of canine knees to 0.6N·m valgus-varus torques and measured the resultant rotations with internal-external rotation and anterior-posterior translation first constrained, then unconstrained. Valgus-varus rotation was increased by 220% in the latter case, valgus being coupled with internal rotation and varus with external rotation, and anterior-posterior translation was effectively zero. The effects of ACL transection on these findings were again dependent on constraint: in the constrained state the valgus range increased by 10%, but by 186% when unconstrained, and both valgus and varus were coupled with anterior translation. These results are corroborated by those of Hollis et al. (1991), who found coupled internal and external rotation with applied valgus and varus torques, respectively.

3.2.6 Combined internal rotation/valgus rotation – the ‘pivot shift’
Internal rotation and valgus torques are commonly combined in a clinical test for ACL function called the pivot shift, which, subject to variations, consists of the application of these torques such that the lateral tibial condyle subluxes anteriorly in extension, and then with flexion the condyle abruptly slides posteriorly at around 30° (Galway & MacIntosh, 1980). Galway and Maclntosh (1980) described the subluxation as involving the whole tibia, but predominantly the lateral condyle was involved.

The usefulness of the clinical pivot shift test as an indicator of ACL deficiency is beyond the scope of this review. It continues to be of biomechanical interest due to an association between a positive test and poorer outcome after ACL reconstruction (Jonsson et al., 2004), although Noyes et al. (1991) found too much operator subjectivity in the movements produced to recommend the manual pivot shift as an objective test.

To better understand the biomechanics of the pivot shift, studies have measured knee kinematics while subjecting cadaveric knees to combined torques to simulate the manoeuvre. Markolf et al. (1995) showed that the combination of internal rotation/valgus torque (10N·m/10N·m) increased ACL force, whereas varus torque with either internal or external rotation did not. Kanamori et al. (2000) showed that anterior translation in the ACL-deficient knee resulting from a 10N·m internal rotation torque increased when that torque was combined with a 10N·m valgus torque; also ACL forces were greater with the combined torque. The most consistent finding in the ACL-deficient knee is an increase in anterior translation, where internal rotation was either not measured (Herbort et al., 2010; Yagi et al., 2002; Zantop et al., 2007), was found to be unchanged (Diermann et al., 2009; Seon et al., 2010) or increased (Kondo et al., 2010; Yamamoto et al., 2004). Studies have also sought to uncover the role of the ACL bands on combined internal rotation/valgus torque. On combined torque (5N·m/10N·m), Gabriel et al. (2004) calculated greater force borne by the
anteromedial band than the posterolateral at 15 and 30° flexion, with a greater posterolateral band contribution at 15°, and Wu et al. (2010) found no difference in load borne by either band at 0°, and greater load borne by the anteromedial band at 30°. This is consistent with a picture of lessening posterolateral band contribution to resistance to combined rotation and valgus as flexion increases, and that by 30° the force in the anteromedial band is larger. So it is interesting to note that Zantop and colleagues (2007) found greater anterior translation on combined torque (4N·m/10N·m) when the posterolateral band was cut, compared to the anteromedial band, at 0 and 30° flexion. The results of kinematic studies suggest that the function of the ACL, even in response to combined torques, is to limit anterior tibial translation. Such findings have led Diermann et al. (2009) to conclude: To assess the rotational instability of the knee joint during the clinical examination, the observer needs to quantify rather the anterior tibial translation of the tibia than the internal tibial rotation of the tibial head. So rotational instability is evinced not by rotation but by translation. These results are similar to those seen in 3.2.3. In Lane and co-workers’ (1994) cadaveric transection study, which found no increase in internal rotation, the pivot shift test was positive. This was supported by Reuben and colleagues’ (1989) findings, with pivot shift-like motion of the knee in the absence of recorded internal rotation change. Lane et al. (1994) concluded that the pivot shift reflected increased anterior translation in the presence of normal rotation. An alternative explanation is that what is observed in the pivot shift sign is internal rotation about a medi ally-shifted axis. This was concluded by Matsumoto (1990), who located the rotation axis in cadaveric ACL-deficient knees as lying at the site of the medial collateral ligament, and recalls the findings of Lipke et al. (1981) and Mannel et al. (2004) (3.2.3). Galway and MacIntosh’s (1980) description of the phenomenon (above) certainly looks to describe a rotation. Bull et al. (2002) too described a translation and rotation, but both about a medial axis. Noyes et al. (1991) measured the kinematics of an ACL- (and medial collateral ligament-) deficient cadaveric knee subjected to the pivot shift test by 11 orthopaedic surgeons. They recorded concurrent anterior translation and internal rotation, but the amount of movement depended on technique: for example, if initial internal rotation was increased then the medial tibial condyle projected less anteriorly than did the lateral. Musahl et al. (2011) showed that ACL-deficiency (1) increased lateral tibial condyle anterior translation, and (2) changed medial condyle movement from posterior translation to anterior. Why there is a seeming reluctance to describe an instability pattern where the lateral condyle subluxes anteriorly more than the medial as a rotation is unclear. We believe that measuring anterior translation and internal rotation, the latter about a central tibial axis, has led to some researchers as treating these as separate. 3.2.7 In vivo external forces – quadriceps load and body weight

Quadriceps load

Because in extension the quadriceps femoris muscles, acting via the patellar ligament, exert an anterior force on the tibia, contraction of the quadriceps is seen to load the ACL. In higher degrees of flexion the patellar ligament pulls the tibia posteriorly and this effect is lost. Studies using cadaveric knees (Bach & Hull, 1998; DeMorat et al., 2004; Renström et al., 1986) have shown that simulated quadriceps activity significantly increases ACL strain with the knee in up to 60° of flexion, and that ACL force peaks around 30° flexion (Li et al., 1999).
Li et al., 2004). This was corroborated by in vivo studies, showing that actual quadriceps contraction causes ACL strain increases to 30° flexion (Beynnon et al., 1992), and that ACL-deficient knees show increased anterior translation on quadriceps contraction in the same range (Bach & Hull, 1998; Barrance et al., 2006; DeMorat et al., 2004).

Studies examining the behaviour of the two bundles of the ACL on quadriceps contraction have shown equal band strain throughout flexion range (Bach & Hull, 1998; Wu et al., 2010).

Weight-bearing

It is clear that ligament function in the knee is sensitive to weight-bearing status. This has meaning beyond the laboratory: what obtains on the clinician's couch may not obtain on the street. Cadaveric studies have measured anterior translation with the knee unloaded and loaded with a compressive force. Hsieh and Walker (1976) found that a 98N load reduced anteroposterior translation, and Ahmed et al. (1987) showed a decreased range of anterior translation in the presence of a 900N preload, and a decrease in ACL strain with compression. In contrast, Torzilli et al. (1994) found anterior translation of the tibia in response to a joint compressive load (up to 444N), as did Oh et al. (2011), who found translation coupled with internal rotation with a compressive load of 800N. These findings were corroborated in vivo: Beynnon et al. (1997) and Fleming et al. (2001) found an increase in ACL strain with the simple change from non-weight-bearing to weight-bearing. Weight-bearing may increase ACL loading due to the posteroinferior slope of the proximal tibial surface (Hashemi et al., 2008) inducing an anterior shift on tibiofemoral compression (Fleming et al., 2001; Torzilli et al., 1994).

These findings can then help to explain the coupling of internal rotation with other movements. In 3.2.5 the application of valgus-varus torque was seen to induce coupled internal-external rotation (Inoue et al., 1987). The effect of these torques can now be seen in terms of weight-bearing through the lateral and medial condyles, respectively. Valgus torque opposes the lateral femoral and tibial condyles, and due to the slope of the tibial condyle there is relative posterior translation of the femoral condyle i.e. internal tibial rotation. Varus loading would then oppose the medial condyles, causing external tibial rotation. This is supported by Inoue's results following ACL transection: both valgus and varus torques resulted in anterior tibial translation. That the slope is greater on the lateral side (Hashemi et al., 2008) may help to explain the internal rotation found on compressing the knee joint (Meyer & Haut, 2008).

4. Anterior cruciate ligament injury and reconstruction

Loss of function of the ACL leads to kinematic changes that threaten the well-being of the knee and patient, and that may call for surgical reconstruction to combat them. But replacing a native ligament in order to replicate its function, when that function is not completely known, necessitates ongoing evaluation of the kinematics before and after surgery.

4.1 Kinematics of the ACL-deficient knee

In section 3 results of studies were discussed where the kinematics of ACL-deficient knees, both cadaveric and in living patients, were studied where external influences such as simulated weight-bearing and muscle forces were controlled. Studies have also been conducted where those influences were less controlled, in which living patients performed
weight-bearing activities closer to normal everyday function, especially gait. These studies are beyond the scope of this review, not due to relevance but complexity: the kinematics of the ACL-deficient knee in walking will be a result of both the deficiency as well as the person’s responses to that deficiency.

4.2 Kinematics of the ACL-reconstructed knee

Reconstruction of the ACL typically replaces the ligament with a graft from either a central strip of patellar ligament or one or more hamstring tendons. Studies addressing the kinematics of the ACL-reconstructed knee, comparing reconstructed knees with ACL-intact, and –deficient knees have increased our understanding of the success of attempts to restore knee function after injury. Given the lack of clarity of just what ACL deficiency causes, unsurprisingly these studies have also thrown up questions that remain unanswered.

Given the primacy of the ACL in resisting anterior translation on anterior force, this function has been a focus of ACL reconstruction research. Yoo et al. (2005) investigated the kinematics of reconstructed cadaveric knees under anterior force (130N) and simulated quadriceps load (400N). Anterior translation was restored to intact levels at flexion greater than 30°, but at 30° or less the translation was greater than the intact state.

The effects of reconstruction on knee rotation have of course been another focus. In an in vivo study, Nordt et al. (1999) measured internal and external rotation on rotational torque (5N·m) on ACL-reconstructed knees and compared values with the uninjured side. Internal rotation was decreased and external rotation increased in the reconstructed knees, leading the authors to conclude that internal rotation had been over-constrained. Yoo et al. (2005) did not apply a torsional load but they did measure rotation on anterior force (130N) and quadriceps load (400N). Interestingly internal rotation showed no difference between ACL-intact and –deficient knees, but was decreased in reconstructed compared to intact knees. This again suggested an over-constraint to internal rotation. On a combined internal rotation/valgus torque (10N·m/10N·m) in a cadaveric study, Woo et al. (2002) found that reconstructed knees showed reduced anterior translation compared to the deficient state, but increased compared to the intact state. Woo and coworkers thus suggested that reconstruction was unable to adequately limit rotational laxity, but with these few studies we see evidence of decreased and increased laxity associated with internal rotational torques.

So with problems with rotational laxity, alternative methods have been sought to improve results. One possible problem with the standard ACL reconstruction procedure is that the graft is somewhat vertical, with the femoral end just lateral to the summit of the roof of the intercondylar notch, and obliquity may affect rotational stability (Yamamoto et al., 2004). A potential improvement is a more laterally placed femoral tunnel to increase the obliquity of the graft. (As this reflects the original femoral attachment of the ligament better this has been termed an “anatomic” reconstruction (e.g. Kondo et al., 2011), but as this is also used to describe the effect of a double bundle reconstruction (e.g. Yamamoto et al., 2004) we will use the term laterally placed.) The notch with the knee in extension is open posteriorly, so the summit of the roof is anterior. With the knee in the surgical position of flexion, the summit is at the top of view, or at 12 o’clock, and the lateral wall of the notch in the right knee merges with the posterior articular cartilage at 9 o’clock (Seon et al., 2011). So the standard femoral placement is at 11 o’clock (Markolf et al., 2002); from the previous paragraph at least Woo et al. (2002) and Yoo et al. (2005) used the 11 o’clock position. Another effort to mimic
the natural anatomy is the double bundle method, where anteromedial and posterolateral bands of the original ligament are replaced separately (Zaricznyj, 1987).

**Effect of femoral tunnel site**

In a study to compare a laterally placed single bundle reconstruction with the ACL-deficient state, Lie et al. (2007) compared anterior translation and internal rotation in cadaveric knees before and after ACL transection and after reconstruction (in the half-past 10 position). They also varied the graft tension, and examined its effect on kinematics. Anterior translation on anterior force (150N) was reduced from the deficient state up to that of the intact state with increasing graft tension. The pivot shift test was simulated by applying a “specific and unique combination of loads”, meaning the results cannot be replicated but the combination of loads was kept constant for each knee. Increasing graft tension again reduced anterior translation, but not internal rotation.

Markolf and colleagues (2002) tested the laxity and ligament graft forces in a series of cadaveric knees with femoral attachment sites at the 10, 11 and 12 o’clock positions. Anterior translation on a 200N anterior force was not affected by tunnel position, and neither was graft force (on 100N anterior force or 5 and 10N m internal-external rotational and valgus-varus force), between full extension and full flexion.

Seon et al. (2011) compared single bundle in vivo reconstructions with femoral attachments at 11 o’clock (“high”) and 10 o’clock (“low”). Both methods reduced anterior translation on 100N anterior force to the same degree, agreeing with Markolf et al. (2002). The main departure from both Markolf et al. (2002) and Lie et al. (2007) was that the low group showed significantly less internal rotation on 10N·m torque than the high group, and without preinjury or contralateral knee range data, the authors declared this result “better”.

**Single (standard) versus double**

Yagi et al. (2002) compared an anatomic double bundle method with a single bundle (11 o’clock) method in a series of cadaveric knees. The double bundle method led to increased anterior translation on a 134N anterior force at 0 and 30° flexion, compared to the intact state, but this translation was significantly less than for the single bundle reconstruction. Ligament force measured was at the intact level for the double bundle method and significantly greater than for the single bundle method up to 60° flexion, and the forces measured in the single bundle were less than in the intact state up to 30°. Single and double bundle reconstructions were both unable to reduce anterior translation on combined internal rotation/valgus torque (5N·m/10N·m), but the double bundle method reduced it more. Kondo et al. (2010) performed a similar study. Both reconstructions showed significant decreases from deficient values in anterior translation on 90N anterior force, with the double bundle method showing significantly less translation, and values were not compared with the intact state. On 5N m internal rotation torque the double bundle method reduced internal rotation to intact levels, but the single bundle method did not. On combined internal rotation/valgus torque (1N·m/5N·m), both reconstructions lowered anterior translation. Under the combined torque, only the double bundle method reduced internal rotation, but to below intact levels i.e. internal rotation on pivot shift was over-constrained, but not on internal rotation torque.

**Single (lateral) versus double**

Several studies have attempted to compare the effectiveness of one development over the other i.e. laterally placed single bundle versus double bundle. Yamamoto et al. (2004)
compared a series of cadaveric knees in the ACL-intact, -deficient and -reconstructed (laterally placed [10 o’clock] single bundle and “anatomical” double bundle) states. Anterior translation on 134N anterior force was restored to normal levels in the double bundle method, and was greater than in the intact state in the single bundle method at 60 and 90° flexion. Forces borne by the two ligament grafts reflected these results, those in the single bundle graft being lower than in the intact state at 60 and 90°. Combined internal rotation/valgus torque (5N m/10N m) produced anterior translation that was reduced to intact levels by both methods, and graft forces measured were the same with each method. Combined torque produced internal rotation that was restored to intact levels by both methods at 15°, and was greater with both methods at 30°. These results suggest that a single bundle reconstruction may achieve the same rotational stability as a double bundle reconstruction, with a shift in the femoral tunnel location.

Bedi et al. (2010) compared anterior translation of the tibia on anterior force and mechanized pivot shift in cadaveric knees in the ACL-intact, -deficient (including of menisci) and reconstructed states. The reconstruction methods were double bundle and single bundle, the femoral tunnel for the latter being placed in between the tunnel sites for former. They measured movement of the lateral and tibial condyles and a central point independently. Both reconstructions restored translation to the intact level on anterior force (68N). Both reconstructions reduced lateral condyle movement during a “mechanized pivot shift” (torques not given), but the double bundle method reduced it more.

Seon et al. (2010) compared anterior and medial translation and internal rotation ranges in a series of cadaveric knees in the intact, deficient and reconstructed (single [half-past 10] and double bundle) states. They found reconstruction reduced anterior translation on anterior force (134N), simulated quadriceps load (400N) and combined internal rotation/valgus torque (5N m/10N m) from the deficient state, but the single bundle method did not reduce translation back to the intact level. Tibial medial translation was increased in the deficient state and was reduced to the intact level by both methods. Internal rotation on quadriceps load was reduced from the intact level by both reconstruction methods, more so with the double bundle, and the double bundle method reduced internal rotation on combined torque compared to the intact state. So although anterior tibial translation was improved with the double bundle method, this method also appears to have over-constrained internal rotation.

In an in vivo patient study comparing laterally placed single bundle and double bundle reconstructions, Aglietti et al. (2010) found an increase in anterior translation on 134N anterior force with the single bundle method, but only at 1- and 2-year follow-up. This reflects the findings of Seon et al. (2010) and Yamamoto et al. (2004), and is a useful reminder that the post-operative phase is only the start of an actual ACL reconstruction.

Hemmerich et al. (2011) compared internal rotation range on internal rotation torque (indexed to body weight) before and after single (10.30) or double bundle in vivo reconstruction. Although internal rotation in the deficient knee was shown to be increased at 0° (and not 30°) flexion, the only significant change in rotation after reconstruction was an over-constraint in the double bundle knees at 30°.

**Standard single versus lateral single versus double**

Kondo et al. (2011) compared translation and rotation in cadaver knees in the ACL-intact, -deficient, and -reconstructed (single bundle [11 o’clock], laterally-placed single bundle [10.30], and double bundle) states. While all reconstructions restored anterior translation on
anterior force (90N) to intact levels, there were differences in rotation. The single bundle (11 o’clock) method was unable to decrease the internal rotation range on internal rotation torque (5N·m) to intact levels, unlike the laterally-placed single bundle and double bundle methods. A mechanized pivot shift test, delivering combined internal rotation/valgus torque (1N·m/5N·m), showed no changes to rotation, even in the deficient state (presumably due to lower rotational torque). Interestingly though, the combined torque caused an anterior translation that was (a) increased in deficient knees, (b) normal in the 11 o’clock single bundle reconstruction, and (c) decreased (i.e. over-constrained) in the laterally-placed single bundle and double bundle reconstructions.

So overall the laterally placed and double bundle methods may improve anterior translation but over-constrain internal rotation, when the specific nature of the rotational function of the ACL is unclear. It is useful at this point to look more closely at some of these data. Kondo et al. (2011) showed that laterally placed single bundle and double bundle reconstructions restored anterior translation on anterior force to normal levels, but over-constrained anterior translation on simulated pivot shift. There was no general over-constraint on rotation, as internal rotation range on rotational torque was normal. If pivot shift does measure anterior translation due to internal rotation, it would appear that the ACL has a greater role in restraining this anterior translation, as there was no over-constraint in translation on anterior force. Anterior translation should therefore be greater on pivot shift after transection than an equivalent anterior force, but we know of no models that allow calculation of an anterior force resulting from an internal rotation torque in the knee.

Bedi et al. (2010) showed mean measurements of movement of different parts of the tibia, and while many of these means were not statistically significantly different, they did derive from only five knees, and the pattern of differences provides us with a possible alternative explanation to Kondo and others’ (2011) results. In the ACL-intact state, Bedi et al. (2010) showed that the pivot shift test elicited a zero mean anterior-posterior shift for the central point, with a posterior shift of the medial condyle and an anterior shift of the lateral condyle. In terminology appropriate to a situation where the degree of movement is dependent on position, the tibia rotated around a roughly central axis (Fig. 2(a)). The deficient state (again, the menisci were excised) showed anterior shift of the three measured points, lateral > central > medial, i.e. the tibia rotated around a medially placed axis (Fig. 2(b)). The change from posterior to anterior movement of the medial condyle on pivot shift was also shown in Musahl and coworkers (2011) results (3.2.6). The single bundle reconstruction showed movement of roughly equal magnitude of medial and lateral condyles, posteriorly and anteriorly respectively, but greater than in the intact state. That is, the tibia rotated about a central axis but with greater range than the intact state (Fig. 2(c)). Finally, the double bundle reconstruction led to posterior movement of the medial condyle and the central point (medial > central) and the lateral condyle moved anteriorly, i.e. rotated around a laterally placed axis (Fig. 2(d)). In this way Kondo and colleagues’ (2011) posterior translation may have been less an over-constraint than a response to a shifted axis.

5. Future research

Over the last 30 or more years there has been much written about the ACL and its function and treatment, and although results of treatment are overall very good, it is surprising that there is still so much confusion regarding the role of the ligament, and there is no meaningful consensus statement about what precisely surgery should be trying to mimic.
One issue that we believe stands in the way of such consensus is a black and white atmosphere of false dichotomies encouraged by the statistically significant/non-significant divide, almost as if, for example, any increase in rotational laxity after ACL injury is the same as any other. What is required is a series of larger high-powered studies where meaningful estimates of laxities can be made. It is quite conceivable that reconstruction can never be perfect, but that a residual laxity is tolerable following reconstruction. But a statistical test with sufficient power to show a statistically significant, yet minor, difference results in a conclusion of abnormality; one where \( n \) is low enough and/or variance is high enough to mask a larger difference indicates that the situation is normal. We believe that a goal of reconstruction research should be, after stoically accepting that there will be an intact-reconstructed difference, to estimate that difference with confidence intervals and then to determine its clinical significance. Otherwise too much influence is placed in statistical inferences, with not enough in biological ones.

The data in the literature are consistent with a change to knee joint kinematics resulting from a change in position of the axis of knee joint rotation following ACL injury, and possibly also reconstruction. Just as an increase or decrease in rotational range may oppose articular regions not accustomed to opposing each other, and thereby initiate degenerative changes, so too may a change in position of the axis of rotation. We are embarking on research to investigate the range of offset-axis rotation, and the effect on that range of ACL transection. We believe this research will extend current knowledge and thinking that is focussed on central axis rotation, and possibly registers change in axis as increase in translation.
6. Conclusion

The ACL is considered a bipartite ligament, consisting of anteromedial and posterolateral bands, a description that contradicts some sound anatomical research and will live on undaunted. But the ligament is large and complex and there are sound functional reasons for overlooking such quibbles.

A much more significant issue is the function of the ACL. There is no clear understanding of the function of the ACL beyond a role in restricting anterior motion of the tibia, and any brief statement of function either does not aspire to great accuracy or claims unwarranted certainty. Without doubt the ligament opposes anterior translation in response to an anterior force, but any effect on internal tibial rotation, especially about a centrally-placed axis, appears small and variable. Perhaps if injury to the ACL were not quite so frequent and/or disabling the discussion might end there. But less than ideal outcomes from reconstructive surgery, and an association of poor outcomes and a positive pivot shift test, indicate an issue with rotational stability.

But the nature of that instability is poorly characterized. In 1981 it was reasonably argued that ACL deficiency causes a shift in axis of internal rotation, and results since support this, yet 30 years later there is still a tacit rejection of this notion and a continued presumption that rotation always occurs around an axis passing centrally through the tibia.

Given that the ACL does restrict anterior translation and may or may not restrict internal rotation, it is interesting that post-reconstruction kinematics show increased laxity on translation and not enough on rotation. Restoration of function and avoidance of joint degeneration are important enough goals to require that our descriptions of function are as accurate as possible. But despite evidence that reconstruction may over-constrain internal rotation, the principle of less is good so even less is better is sometimes invoked in the goal to simply reduce internal rotation range.

We suggest that, just as ACL deficiency may alter the position of the axis of internal-external rotation, reconstruction may have effects on rotational axis location that we do not understand, and we provide a possible explanation for some of the kinematic changes seen after reconstructive surgery. This speculative explanation has too few data to give this confident support, but we believe that axis position on injury and reconstruction deserve more scientific investigation.

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8. References


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During last couple of years there has been an increasing recognition that problems arising in biology or related to medicine really need a multidisciplinary approach. For this reason some special branches of both applied theoretical physics and mathematics have recently emerged such as biomechanics, mechanobiology, mathematical biology, biothermodynamics. This first section of the book, General notes on biomechanics and mechanobiology, comprises from theoretical contributions to Biomechanics often providing hypothesis or rationale for a given phenomenon that experiment or clinical study cannot provide. It deals with mechanical properties of living cells and tissues, mechanobiology of fracture healing or evolution of locomotor trends in extinct terrestrial giants. The second section, Biomechanical modelling, is devoted to the rapidly growing field of biomechanical models and modelling approaches to improve our understanding about processes in human body. The last section called Locomotion and joint biomechanics is a collection of works on description and analysis of human locomotion, joint stability and acting forces.

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