Knee Surg Sports Traumatol Arthrosc (2006) 14: 204–213
KNEE
DOI 10.1007/s00167-005-0679-9

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Anatomy of the anterior cruciate ligament

Received: 15 February 2005
Accepted: 2 April 2005
Published online: 19 October 2005

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Abstract The anterior cruciate ligament (ACL) is a band of dense connective tissue which courses from the femur to the tibia. The ACL is a key structure in the knee joint, as it resists anterior tibial translation and rotational loads. When the knee is extended, the ACL has a mean length of 32 mm and a width of 7–12 mm. There are two components of the ACL, the anteromedial bundle (AMB) and the posterolateral bundle (PLB). They are not isometric with the main change being lengthening of the AMB and shortening of the PLB during flexion. The ACL has a microstructure of collagen bundles of multiple types (mostly type I) and a matrix made of a network of proteins, glycoproteins, elastic systems, and glycosaminoglycans with multiple functional interactions. The complex ultrastructural organization and abundant elastic system of the ACL allow it to withstand multiaxial stresses and varying tensile strains. The ACL is innervated by posterior articular branches of the tibial nerve and is vascularized by branches of the middle genicular artery.

Keywords Anterior cruciate ligament · Anteromedial bundle · Posterolateral bundle

Introduction

The anterior cruciate ligament (ACL) is a key structure in the knee joint, as it resists anterior tibial translation and rotational loads [15, 37, 46]. It is one of the most frequently injured structures during high impact or sporting activities [45]. The ACL does not heal when torn, and surgical reconstruction is the standard treatment in the field of sports medicine [9]. Such reconstruction aims at restoring the kinematics and stability of the injured knee, to prevent future degenerative changes [27]. Therefore, an adequate understanding of the complex anatomy, function, and biomechanics of the ACL is critical to elucidate the mechanisms of injury, understand the fate of chronic ACL deficiency, and to improve surgical reconstruction. The purpose of this paper is to present a systematic review of original research studies dealing with the macroanatomy and microanatomy of the ACL.

Embryology

The ACL appears as a mesenchymal condensation in the blastoma at 6.5 weeks of gestation, well before joint

cavitation [18]. It is surrounded by a mesentery-like fold of synovium that originates from the posterior capsular apparatus of the knee joint. Thus, while the ACL is located intraarticularly, it remains extra-synovial throughout its course [18].

Macroanatomy

The ACL is a band-like structure of dense connective tissues. Its femoral attachment displays a shape comparable to a vertically disposed semicircle [21]. The bony attachment is located at the posterior part of the inner surface of the lateral femoral condyle and not, as sometimes presumed, at the roof of the intercondylar notch. The ACL is lateral to the midline and occupies the superior 66% of the lateral aspect of the notch on an anterior view of the flexed knee joint. The size of the bony attachment can vary from 11 to 24 mm across [7, 44]. From its femoral attachment, the ACL runs anteriorly, medially, and distally to the tibia. Its length ranges from 22 to 41 mm (mean, 32 mm) and its width from 7 to 12 mm [6] (Fig. 1).

The cross-sectional shape of the ACL is "irregular" and not circular, elliptical or any other simple geometric form. This shape changes with the angle of flexion, but is generally larger in the anterior–posterior direction. The cross-sectional area increases from the femur to the tibia, as follows: 34 mm^2 proximally, 33 mm^2 mid-proximally, 35 mm^2 at mid-substance level, 38 mm^2 mid-distally, and 42 mm^2 distally [23] (Fig. 1). The ACL fibers fan out as they approach their tibial attachment [12]. They attach to a fossa located anterior and lateral to the medial tibial spine. This fossa is a wide, depressed area approximately 11 mm wide (range, 8–12 mm) and 17 mm (range, 14–21 mm) in the antero-posterior direction [7, 21, 22, 44]. Near its attachment, the ACL sends a variable amount of fibers anteriorly beneath the transverse intermeniscal ligament, and some extensions may blend with both the attachment of the anterior or posterior horn of the lateral meniscus. The tibial attachment is somewhat wider and stronger than the femoral attachment [7, 21, 22].

Functionally, Girgis et al. divided the ACL into two parts, the anteromedial bundle (AMB) and the posterolateral bundle (PLB) [21], while other authors have separated the ACL in three functional bundles (AMB, intermediate band, and PLB) [6, 26]. However, the two-bundle model has been generally accepted as the best representation to understand ACL function (Fig. 2).

The fascicles of the AMB originate at the most anterior and proximal aspect of the femoral attachment and insert at the anteromedial aspect of the tibial attachment [6]. Conversely, the fascicles of the PLB originate at the postero-distal aspect of the femoral attachment and insert at the posterolateral aspect of the tibial attachment [6]. A larger number of fascicles make up the PLB as compared to the AMB [50]. With the knee in extension the fascicles of the ACL run in a fairly parallel fashion when viewed sagitally. During flexion, there is a slight lateral rotation of the ligament as a whole around its longitudinal axis, and the AMB begins

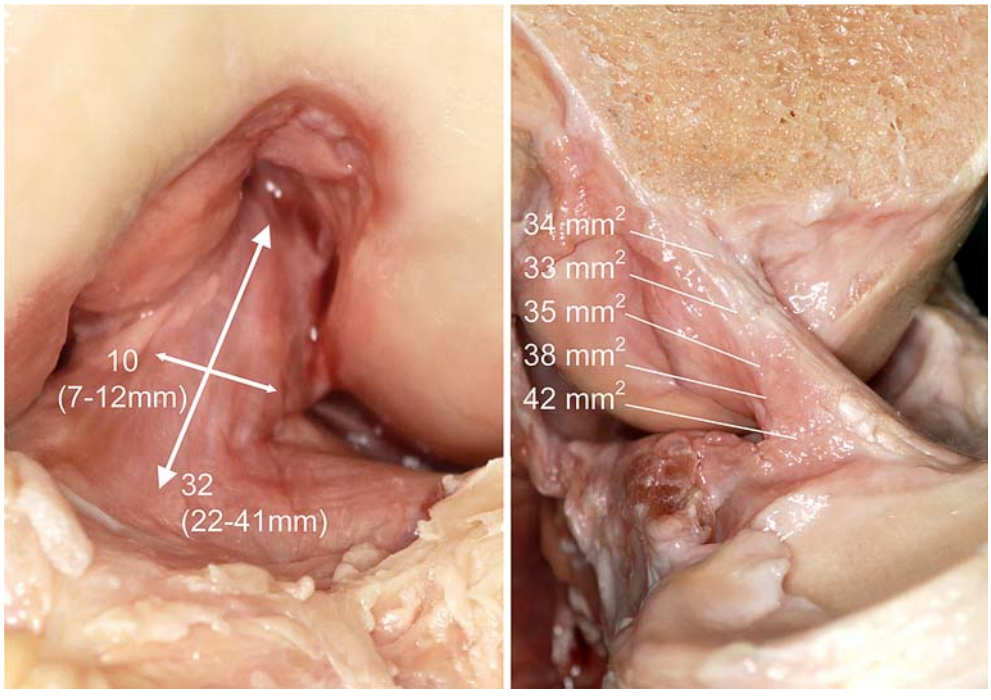


Fig. 1 Front view of a left knee showing the ACL in the femoral intercondylar notch. The mean length is 32 mm (range, 22–41 mm) (*left picture*) and the mean width is 10 mm (range, 7–12 mm). The cross-sectional area varies in size and shape from the femur to the tibia (*right picture*)

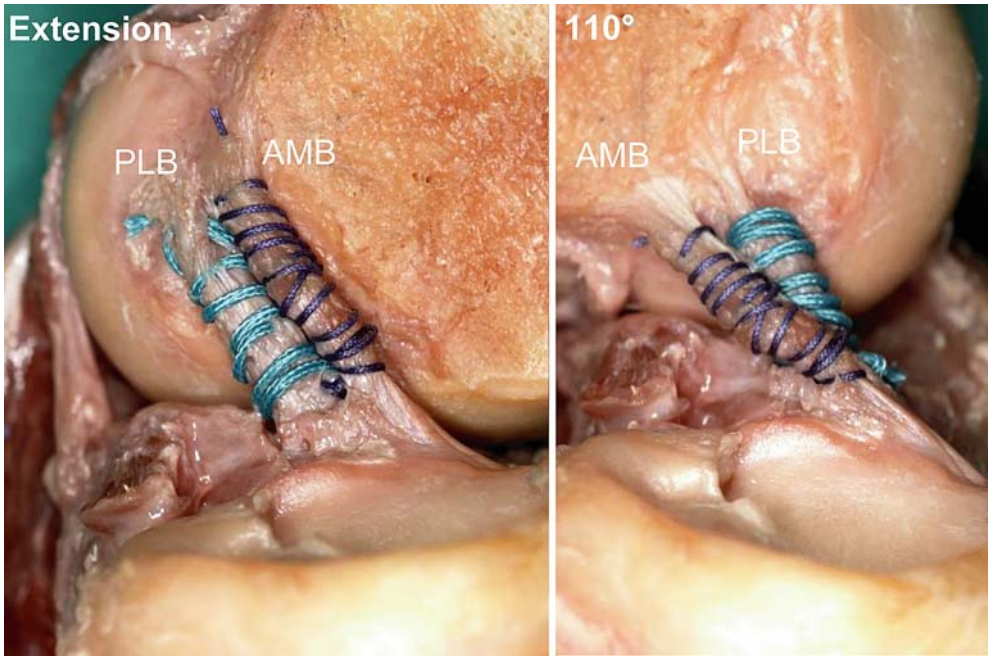


Fig. 2 The AMB and the PLB are not isometric; the AMB tightens during knee flexion while the PLB becomes slack. The AMB spirals around the rest of the ligament so that the two bundles are no longer parallel at 110° of flexion

to spiral around the rest of the ligament. This relative movement of one bundle upon the other is due to the orientation of the bony attachments of the ACL [7, 21] (Fig. 2b). In full extension there is a significant difference in length between the AMB (34 mm) and the PLB (22.5 mm) [26]. The two bundles are not isometric in flexion/extension, but experience different patterns of length changes during passive knee flexion. Hollis et al. [26] showed that the AMB lengthens and tightens in flexion, while the PLB shortens and becomes slack. According to the same authors, the AMB increases by 1.9 mm (5%) at 30° of knee flexion, and by 4 mm (12%) at 90°. Conversely, the PLB decreases by 3.2 mm (14%) when the knee is passively flexed from 0 to 30°, and by 7.1 mm (32%) at 90° of knee flexion [26]. As compared to full extension, the posterior fibers become slack in flexion, and thus leave the anteromedial fibers as the restraint to anterior tibial load. The two bundles are no longer parallel because the AMB spirals around the rest of the ligament. Amis and Dawkins [6] showed that beyond 90° of knee flexion the AMB continues to lengthen and surprisingly the PLB tightens as one approaches full flexion (Fig. 3). In contrast to Hollis et al. [26], they found that the AMB initially shortens until 30° of flexion, and then gradually elongates until it reaches maximal length at 120°. Internal rotation lengthens the ACL a little more than does external rotation, most noticeably at 30° of flexion. Indeed, tibial rotation torques of 1 N m do not cause significant ACL elongation. Twisting is resisted by a combination of capsular shearing, slanting collateral ligament action, joint surface, and meniscal geometry, while the cruciates play only a secondary role [5].

Microanatomy

Microscopically, we can distinguish three zones within the ACL:

1. The *proximal* part, which is less solid, is highly cellular, rich in round and ovoid cells, containing some fusiform fibroblasts, collagen type II and glycoproteins such as fibronectin and laminin (Fig. 4a).
2. The *middle* part, containing fusiform and spindle-shaped fibroblasts (Fig. 5a), is a high density of collagen fibers, a special zone of cartilage and fibrocartilage (especially in the anterior part where the ligament faces the anterior rim of the intercondylar notch), and elastic, and oxytalan fibers. The oxytalan fibers withstand modest multidirectional stresses, while elastic fibers absorb recurrent maximal stress [51]. The fusiform and spindle-shaped fibroblasts are prominent in this middle part, which is also named the fusiform zone, and is located in the middle part and the proximal one-quarter of the ligament. The fusiform cell zone is characterized by a high number of longitudinally oriented cells with fusiform-shaped nuclei, longitudinal blood vessels, and high crimp length. These fibroblasts show features close to the medial collateral ligament (MCL) and dermal fibroblasts [38]. The cytoplasm of the cells in this zone appears to be intimately attached to the extracellular collagen and follows the crimp waveform of the fibers. Based on our and other investigations, it appears that the mid-substance of the ACL shows a high collagen density, low cellularity, and elongated

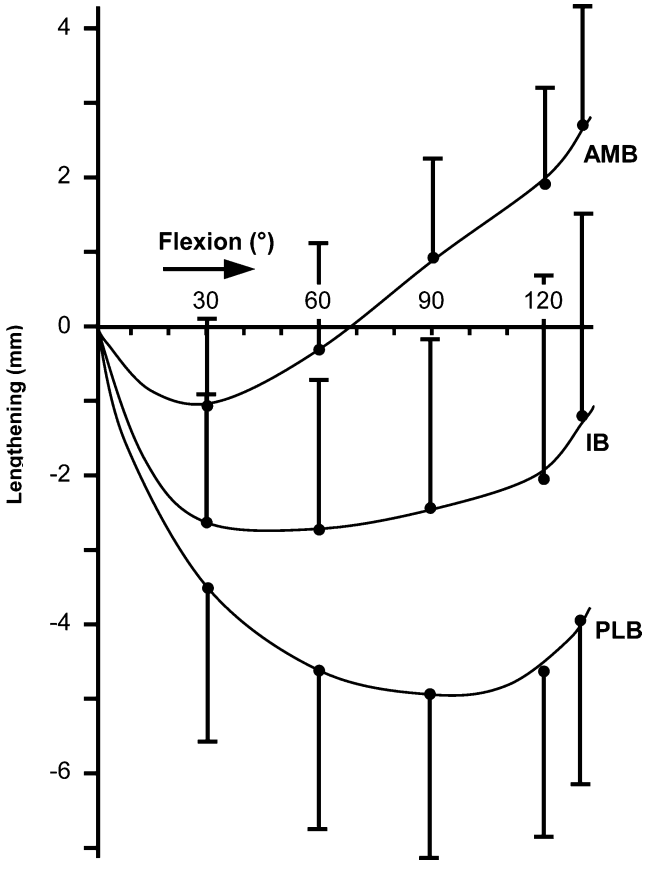


Fig. 3 Mean length change patterns for the three ACL bundles during flexion in neutral rotation (SD; $n = 9$). *AMB* anteromedial bundle, *IB* intermediate bundle, and *PLB* posterolateral bundle. (Reproduced with permission and copyright© of the British Editorial Society of Bone and Joint Surgery [6])

spindle-shape fibroblasts (Fig. 4b). Studies suggest that the ACL has different histological characteristics from the MCL or tendon, and is more cartilage-like in nature. As an example, a zone of fibrocartilage is noted in the distal third of the ligament adjacent to the roof of the intercondylar notch (see below) [38, 39].

3. The *distal* part, which is the most solid, is rich in chondroblasts and ovoid fibroblasts (Fig. 5b), and with a low density of collagen bundles. The fibroblasts, located on either side of the collagenous bundles are round to ovoid, 5–8 μm in diameter and 12–15 μm in length, and resemble the cells of articular cartilage. They have abundant cellular organelles indicating a high level of cellular activity. They also have multiple small cellular processes (microvilli), which project into a surrounding area of amorphous ground substance with reticular fibers, but not into the compact parallel collagen fibrils. Such cells are mostly seen in the distal part of the ligament [4] (Fig. 4c). In the anterior portion of the ACL, approximately 5–10 mm proximal to the tibial attachment, a layer of dense fibrous tissue surrounds the ligament instead of synovial tissue. This area corresponds to the zone where the ligament impinges on the anterior rim of the femoral intercondylar fossa in full knee extension. Within the ligament the cells resemble chondrocytes, with a round to ovoid shape and aligned in rows of 3–15 cells between the bundles of collagen. These collagen bundles are larger than elsewhere in the ligament (130–250 μm) and cross each other at sharp angles, separated by chondrocytes. In this region the ACL consists of fibrocartilage, which is mineralized and resembles bone [42]. According to the theory of "causal histogenesis" [41] the stimulus to the development of fibrocartilage within dense connective tissue is intermittent compressive and shearing forces. Compression stress on the tissue of the anterior portion of the ACL may be caused by the anterior margin of the intercondylar fossa, which serves as a bony pulley for the ACL when the knee is in full extension. Functionally, fibrocartilage with rows of chondrocytes occurs in tendons with moderate changes in angles where they pass around their pulleys [11].

The ACL has a microstructure similar to other soft connective tissues [49]. It is composed of multiple fascicles, the basic unit of which is collagen, and these fascicles range from 250 μm to several millimeters and are surrounded by a connective tissue known as the paratenon. Each fascicle is composed of 3–20 subfasciculi which are enclosed by an epitenon. The subfasciculi have an undulating course and consist of groups of subfascicular units (100–250 μm in diameter) surrounded by loose connective tissue, the endotenon,

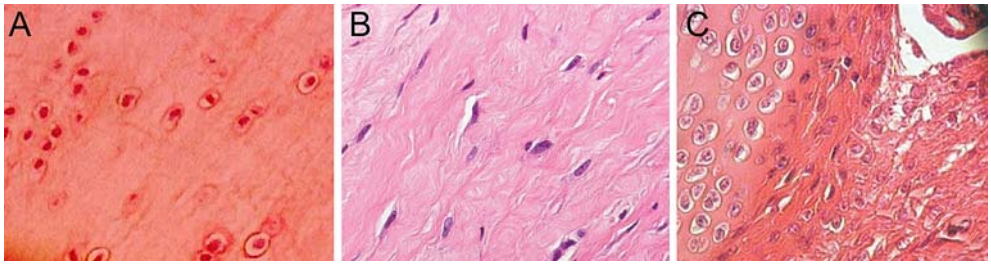


Fig. 4 The three histological zones of the ACL. The proximal part which is highly cellular with round and ovoid cells (**a**), the middle part with fusiform and spindle-shape fibroblasts and a high density of collagen bundles (**b**), and the distal part with ovoid fibroblasts and a low density of collagen bundles (**c**)

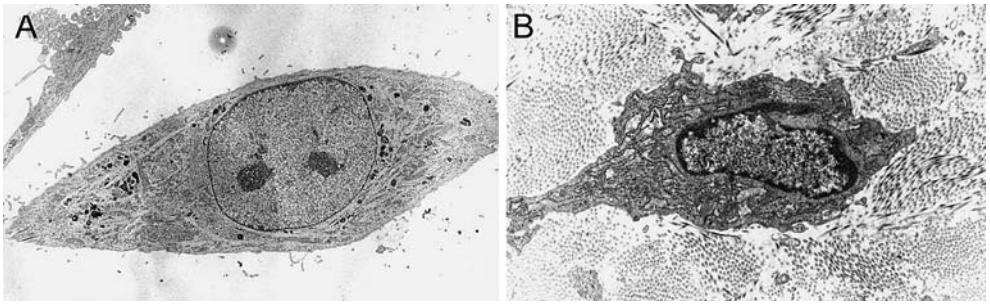


Fig. 5 Elongated spindle-shape fibroblasts (**a**), found in the mid-substance of the ACL, with their cytoplasm intimately attached to the extracellular collagen and following the crimp waveform of the fibers. The distal fibroblasts (**b**) are round to ovoid, resembling a fibro-chondrocyte, and measure about 5–8 μm in diameter and 12–15 μm in length. They have abundant cellular organelles indicating a high level of cellular activity

consisting of collagen type II. These subfascicular units are composed of fibers (1–20 μm in diameter) which are made up of collagen fibrils (25–250 nm in diameter) [51].

Strocchi et al. [51] described two types of *fibrils*:

- The first type has a variable diameter with peaks at 35, 50, and 75 nm and an irregular outline. They account for 50.3% of the entire ACL and are secreted by fibroblasts. These large inhomogeneous fibrils are specialized to resist high tensile stresses.
- The second type has a uniform diameter with smooth margins and a diameter peak at 45 nm. They account for 43.7% of the entire ACL and are secreted by fibro-chondroblasts. These small homogeneous fibrils maintain the three-dimensional organization of the ligament.

Note that the remaining 6% of ACL tissue corresponds to cells and matrix components.

The matrix of the ACL consists of four different systems:

1. *Collagen:* There are different types of collagen found in the ACL.

Type I collagen is the major collagen of ligaments and tendons. In the ACL type I collagen fibrils are oriented parallel to the longitudinal axis of the ligament and responsible for the tensile strength of the ligament [4].

Type II collagen is the typical collagen of cartilage and is normally not found in ligaments. However, it is found in the fibrocartilaginous regions of the ACL, specifically the tibial and femoral sites of attachment. This collagen is located in the pericellular matrix of the chondrocytes that lie in rows between the majority of parallel type I collagen-positive fibrils [42]. This finding indicates that this part of the ligament is exposed to pressure or shear force, as the occurrence of type II collagen in connective tissue such as ligaments or tendons is an indicator of the exposure of this part of the tendon to pressure [10, 30].

Type III collagen is a component of the reticular fibers [54]. Within the ACL, type III collagen is located in the loose connective tissue that divides the type I collagen bundles. Morphologically the fibers can be divided into a fine single-strand type, 2 μm in diameter, and a coarse multiple-strand type, 9 μm in diameter. Type III collagen has a nearly ubiquitous distribution in the ACL, but reveals maximal concentrations near the attachment zones. It is important for the pliability of the ligament, and may also serve to anchor vessels to the adjacent matrix as it bridges collagens and basement membranes [39]. Most of the newly synthesized collagen in the early phase of healing is type III [56]. It is also increased after tendon graft in a remodeling process termed "ligamentization" [3].

Type IV collagen is found in all vascular basement membranes, mainly in the proximal and distal parts of the ACL and less in the middle third which is less vascularized [39].

Type VI collagen has an orientation parallel to that of type III collagen. It serves as a gliding component between functional fibrillar units. It is present in higher amounts in the proximal and distal thirds rather than the middle part of the ACL. This distribution is due to significantly higher strains in the attachment regions in comparison with the mid-region [39].

Recently, Lee et al. [29, 34] found that estrogen directly regulates ligament structure and function by alteration of type I and III synthesis. Indeed, estrogen stimulates type I and III collagen synthesis at the mRNA level, while application of a mechanical force decreases the expression of collagen type I and III genes at all estrogen levels tested [34]. Gene expression of type I and III collagen is also stimulated by mechanical stretch in ACL cells, via up-regulation of the transforming growth factor (TGF)-β1 [29].

2. *Glycosaminoglicans:* Water comprises 60–80% of the total wet-weight of the ACL [40] and is mainly associated with the ground substance, that portion of connective tissue consisting of proteoglycans and glycosaminoglycans (GAGs). The GAGs are highly negatively charged and possess a large number of hydroxyl groups which attract water through hydrogen binding. The ACL has a high proportion of

GAGs (10 mg hexosamine/g dry tissue), two to four times that observed in tendons. The MCL also has a higher GAG concentration than tendons [2, 4]. This alters the viscoelastic properties of the ACL and represents an additional "shock-absorbing" feature in the ligament.

3. *Glyco-conjugates:* These include laminin, entactin, tenascin, and fibronectin. The fibronectins play an important role in intra- and extracellular matrix morphology, cellular adhesion, and cell migration. They function to attract and couple key elements in normal, healing, and growing tissues. They account for 2 μm/mg dry tissue [43, 51].
4. *Elastic components:* This group includes oxytalan, elaunin, mature elastic fibers, and elastic membranes. They permit the extreme distance changes during motion [39, 51].

The parallel, dense, and regular organization of ACL fibrils appears to be unique. It is a combination of helical and planar, parallel or twisted, nonlinear networks. The centrally located fascicles in the ACL are either straight or undulated in a planar wave pattern, whereas those located at the periphery are arranged in a helical wave pattern. The purpose of the wave and nonlinear pattern of the fibrils has been interpreted as "crimp" and "recruitment", respectively [49, 50]. Crimp represents a regular sinusoidal pattern in the matrix. This accordion-like pattern in the matrix provides a "buffer" in which slight longitudinal elongation may occur without fibrous damage. It also provides a mechanism for control of tension and acts as a "shock-absorber" along the length of the tissue [53]. Hence, during tensile stretch, fibril "crimp" is first straightened out by small loads, after which larger loads are needed to elongate these fibrils. As such, an increasing number of fibrils become load-bearing as larger loads are applied ("recruitment") and a gradual increase in tissue stiffness is seen, resulting in a nonlinear load–elongation curve. This phenomenon allows the ACL to rapidly provide additional protection to the joint. The periodicity and amplitude of crimp appear to be structure-specific features, and they are best evaluated under polarized light [14].

Fig. 6 Ruffini receptors (**a**) and Vater–Pacini receptors (**b**). These mechanoreceptors have proprioceptive functions and provide the afferent arc for postural changes of the knee through deformation within the ligament. Free-nerve endings (**c**) also serve as local effectors by releasing neuropeptides with vasoactive function

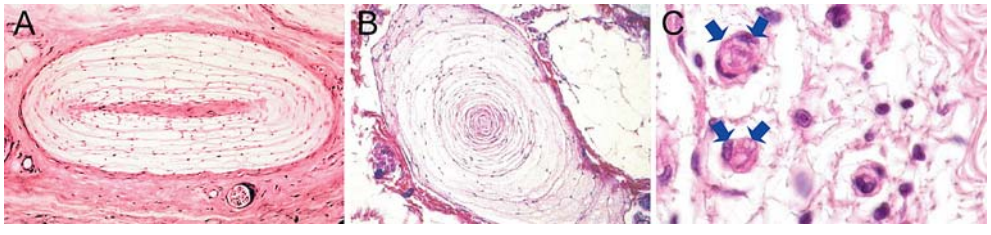
In summary, the complex ultra-structural organization, the varied orientation of the bundles in the ACL, and the abundant elastic system make it very different from other ligaments and tendons. The ACL is a unique and complex structure able to withstand multi-axial stresses and varying tensile strains [51]. This specificity and complexity may explain the difficulty in reproducing the original ACL following surgical reconstruction.

Innervation

The ACL receives nerve fibers from the posterior articular branches of the tibial nerve [28]. These fibers penetrate the posterior joint capsule and run along with the synovial and periligamentous vessels surrounding the ligament to reach as far anterior as the infrapatellar fat pad [28]. Most of the fibers are associated with the endoligamentous vasculature and have a vasomotor function. But smaller myelinated nerve fibers (2–10 μm in diameter) and unmyelinated nerve fibers (1 μm in diameter) have been observed coursing independently of the vessels, and lie alone among the fascicles of the ligament [28, 48]. The receptors of the nerve fibers mentioned are as follows (Fig. 6):

- *Ruffini receptors* which are sensitive to stretching and are located at the surface of the ligament, predominantly on the femoral portion where the deformations are the greatest [24, 57].
- *Vater–Pacini receptors* which are sensitive to rapid movements and are located at the femoral and tibial ends of the ACL [24, 57].
- *Golgi-like tension receptors* are located near the attachments of the ACL as well as at its surface, beneath the synovial membrane [28, 48].
- *Free-nerve endings* function as nociceptors, but they may also serve as local effectors by releasing neuropeptides with vasoactive function. Thus, they may have a modulatory effect in normal tissue homeostasis or in late remodeling of grafts [24, 25].

The mechanoreceptors cited above (Ruffini, Pacini, and Golgi-like receptors) have a proprioceptive function and provide the afferent arc for signaling knee postural changes. Deformations within the ligament influence the



output of muscle spindles through the fusimotor system [25, 57]. Hence, activation of afferent nerve fibers in the proximal part of the ACL influences motor activity in the muscles around the knee; a phenomenon called "ACL reflex." These muscular responses are elicited by stimulation of group II or III fibers (i.e. mechanoreceptors). Considering a reflex latency of 70 ms, at least 110 ms (reflex time + electro-mechanical delay) would pass before substantial forces could be produced by the muscles after application of load to the ACL. Therefore, this excitatory reflex cannot serve as an automatic protective mechanism for the ACL. The ACL reflex is an essential part of normal knee function and is involved in the updating of muscle programs [31, 33]. This becomes even more obvious in patients with a ruptured ACL, where the loss of feedback from mechanoreceptors in the ACL leads to quadriceps femoris weakness [31]. Indeed, this afferent feedback from the ACL has a major influence on the maximal voluntary contraction exertion of the quadriceps femoris [32]. Such patients also lose the accuracy of joint position sense since it is directly related to the number of mechanoreceptors. Therefore, preserving ACL remnants during ACL reconstruction could help to maintain proprioception after reconstruction [1].

Vascularization

The blood supply of the cruciate ligaments is provided by the middle genicular artery (MGA). The MGA originates at a right angle from the anterior aspect of the popliteal artery, most commonly at the level of the proximal contours of the femoral condyles immediately below the origin of the superior genicular artery and just above the sural artery. In its extracapsular course it is immersed in the fat of the popliteal space and is accompanied by satellite veins and the posterior articular nerve. It then pierces the posterior capsule, passing through one of the apertures existing in the oblique popliteal ligament, nearer to the lateral than the medial femoral condyle. The MGA usually crosses the posterior capsule in an oblique, almost vertical descending direction. Once within the joint the artery ramifies, providing branches to the soft tissues lodged in the intercondylar notch such as the ACL. The larger vessels mostly descend along its surface facing the PCL. Besides being supplied by nutrient arteries the ACL receives a large, posteriorly descending branch of the MGA directed to the upper tibia [7, 47] (Fig. 7a).

The synovial vessels run obliquely and longitudinally over the entire length of the ACL, beneath the synovial membrane. They arborize to form a web-like network of periligamentous vessels that ensheath the entire ligament. From the synovial sheath, blood vessels penetrate the ligament transversely and anastomose with a network of endoligamentous vessels which lie parallel to the collagen bundles within the loose connective tissue that separates the parallel collagen fibrils into bundles. These vessels do not reach the ligament through the attachment zone; no intraligamentous vessels cross the bony attachment site of the ligament to femur and tibia [7, 8, 36, 42].

The distribution of blood vessels within the substance of the ligament is not homogeneous. The proximal part of the ACL is better endowed with blood vessels than

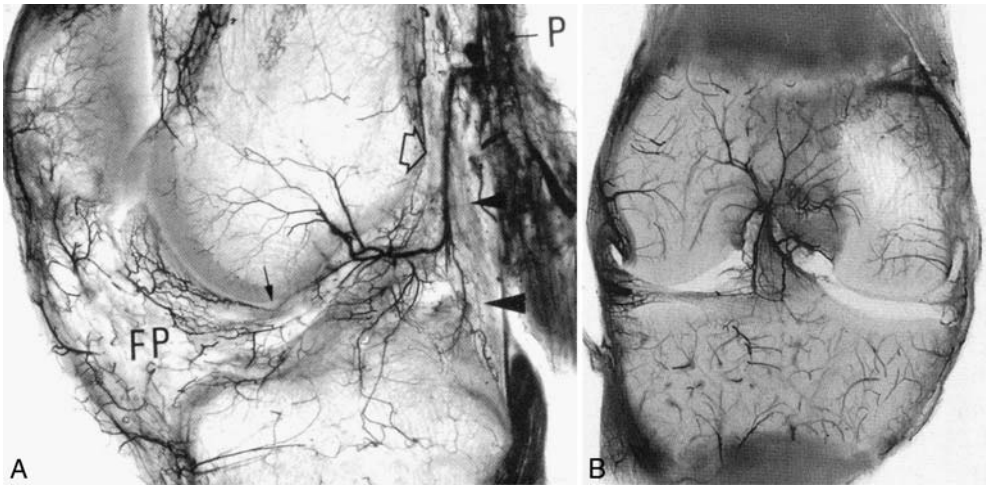


Fig. 7 The left sagittal section (**a**) shows the origin of the middle genicular artery (MGA) at a right angle (*open arrow*) to the popliteal artery (*P*), its almost vertical crossing of the posterior capsule (*arrowheads*), and its intraarticular osseous and soft tissue distribution. The descending branches for the cruciate ligaments are clearly visible. The *small arrow* in front of the ACL indicates some arterioles within the ligamentum mucosum, apparently anastomosing with the intercondylar descending branches of the MGA. FP refers to infrapatellar fat pad. The coronal section (**b**) shows the fan-like intraepiphyseal distribution of the branches of the roof of the intercondylar notch of the femur (intercondylar radiate arteries) and also the arteries descending along the ACL. (Reproduced with permission and copyright© of Wiley-Liss [47])

the distal part. The upper portion of the ACL is supplied by collateral branches of the arteries directed to the roof of the intercondylar notch and the lateral condyle of the femur. The fat pad is richly vascularized and only small arterioles penetrate the ligamentum mucosum [47]. A small amount of blood is supplied to the distal portion of the ACL by the infrapatellar branches of the inferior genicular arteries. The periligamentous fold of vessels is absent in a small zone approximately 5–10 mm proximal to the tibial attachment (Fig. 7a), and in this anterior fibrocartilaginous part the tissue is avascular [42]. The coincidence of poor vascularity and the presence of fibrocartilage is also seen in gliding tendons in areas that are subjected to compressive loads, and the coincidence of these two factors undoubtedly plays a role in the poor healing potential of the ACL [20, 52].

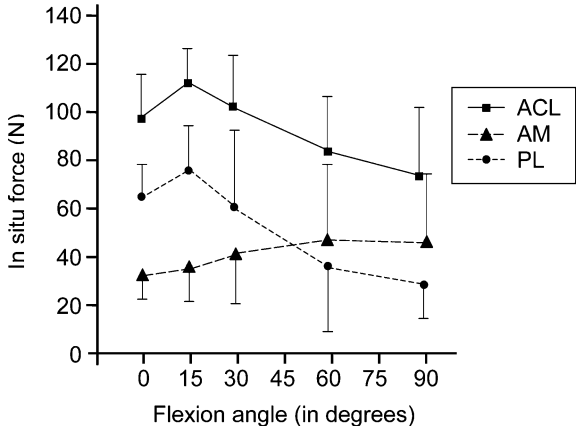


Fig. 8 Magnitude of the in situ force in the intact ACL, AMB, and PLB under 110 N of applied anterior tibial load. (Reproduced with permission and copyright© of Elsevier BV [46])

Biomechanics

The ACL plays a crucial role in joint stability. It is the primary restraint to anterior translation of the tibia relative to the femur [17]. Under normal conditions, the ACL restricts anterior neutral-position shift, but in chronic ACL-deficient knees this anterior translation of the tibia relative to the femur is four times greater than in normal knees [16]. Beynnon et al. [13, 15] showed that an anterior load of more than 50 N produced ACL strains with increasing loads up to 6%. The ACL provides an average restraint of 82–89% to the applied anterior load at 30° of flexion, but it slightly decreases to 74–85% at 90° of knee flexion [14, 55].

It has been shown in vitro that the AMB of the ACL had an in situ tension that increased, in response to a fixed anterior draw force, as the knee flexed from 20 to 90°. Conversely, the force in the PLB increased as the knee extended [19, 46]. These findings corresponded closely to the changing contributions of the fiber bundles to resisting anterior draw forces [6]. The dominance of the PLB in the extended knee provides a rationale for developing double-bundle reconstructions (Fig. 8).

The ACL also functions as a major secondary restraint to internal rotation, particularly when the joint is near full extension. In addition, the ACL functions as a minor secondary restraint to external rotation and varus–valgus angulation, particularly under weight-bearing conditions [15, 37]. Clinically, this function can be tested with the "pivot shift test" which involves applying a combined internal tibial and valgus torque throughout the range of flexion-extension [35].

The ACL can perform its function due to its unique structural properties. Woo et al. [55] studied the tensile properties of the femur–ACL–tibia complex. They found that the values of ultimate load to failure and stiffness for young specimens (22–35 years) were 2,160 (157) N and 242 (28) N/mm, respectively. However, ultimate tensile load and linear stiffness decrease significantly with age: to 658 (129) N and 180 (25) N/mm, respectively, for older specimens (60–97 years) [55].

Acknowledgements The authors would like to thank Dominique Semedo for his technical assistance and Richard Stern MD for his review of the manuscript.

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