1 Morphology and Function of the Cruciate Ligaments

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Introduction

In contrast to the plethora of veterinary publications on cruciate surgery in dogs, only a few papers deal with the microanatomy and neurovascular anatomy of the canine cruciate ligaments. However, an understanding of the complex anatomy and function of these ligaments is imperative to elucidate the pathophysiology of cruciate ligament rupture, and to improve surgical intervention.

Morphology

Macroanatomy

The cranial cruciate ligament (CrCL) attaches to the axial aspect of the lateral femoral condyle, very close to the articular margin (Figure 1.1). It extends diagonally across the joint space and attaches to the cranial intercondyloid area of the tibial plateau (Singleton 1957; Zahm 1965; Arnoczky & Marshall 1977). The proximal attachment site is bordered cranially by the cranial meniscotibial ligament of the medial meniscus and caudally by the cranial meniscotibial ligament of the lateral meniscus (Rudy 1974; Heffron & Campbell 1978) (Figure 1.2). The CrCL is narrowest in its mid-region and fans out proximally and distally (Alm *et al*. 1975; Heffron & Campbell 1978). The length of the CrCL is positively correlated with body weight; taking the average length of its cranial and caudal borders, the mean length has been reported as 13.5–18.7 mm (Vasseur *et al*. 1985; Wingfield *et al*. 2000; Comerford *et al*. 2005). The CrCL runs cranially, medially, and distally in an outward spiral as it passes from the femur to the tibia (Zahm 1965; Haut & Little 1969). Two demonstrably separate bundles are apparent (Figure 1.1) (Arnoczky & Marshall 1977; Heffron & Campbell 1978); these components are termed craniomedial and caudolateral, based on their relative attachment sites onto the tibial plateau. The craniomedial subdivision is the most spiral and the longest, yet smaller, component, and arises more proximally from the femur and attaches more cranially on the tibial footprint area, compared with the caudolateral subdivision. The fibers of the caudolateral component arise from the most lateral and distal part of the attachment area of the lateral femoral condyle, have a straighter path, and

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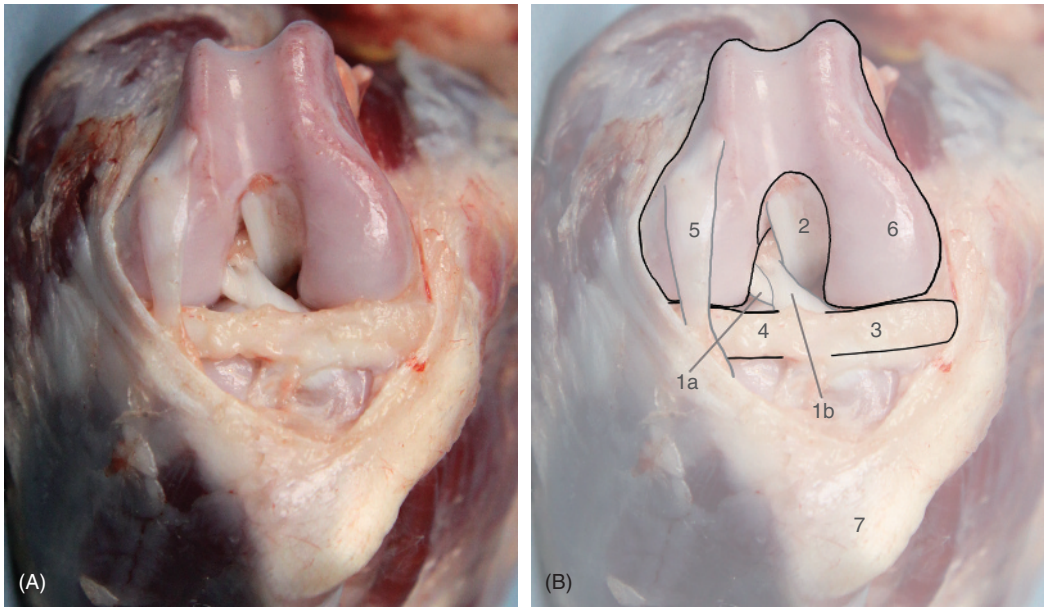

Figure 1.1 Photograph (A) and line drawing (B) of a flexed right stifle joint of a dog. Cranial view after removal of the infrapatellar fat pad. In panel B: 1a, caudolateral bundle of the cranial cruciate ligament; 1b, craniomedial bundle of the cranial cruciate ligament; 2, caudal cruciate ligament; 3, medial meniscus; 4, lateral meniscus; 5, tendon of the long digital extensor; 6, medial femoral condyle; 7, tibial tuberosity.

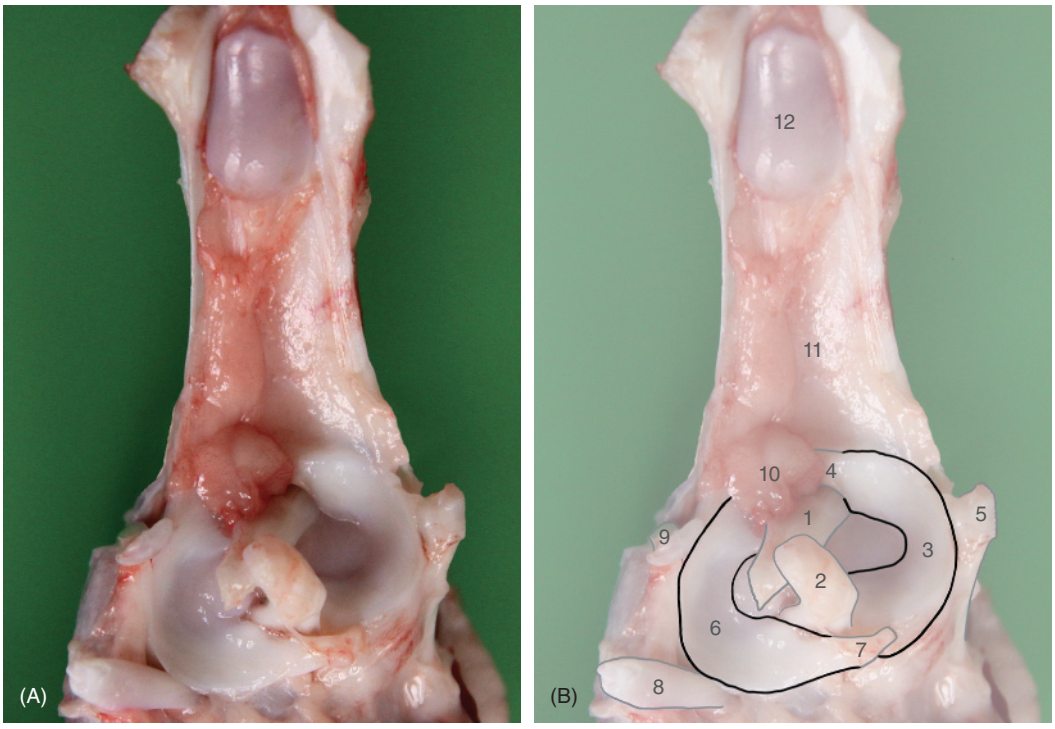

Figure 1.2 Photograph (A) and line drawing (B) of the left pelvic limb of a dog. Dorsal view on the tibial plateau after removal of the femur. In panel B: 1, cranial cruciate ligament; 2, caudal cruciate ligament; 3, medial meniscus; 4, intermeniscal ligament; 5, medial collateral ligament; 6, lateral meniscus; 7, meniscofemoral ligament; 8, popliteal tendon; 9, tendon of the long digital extensor; 10, infrapatellar fat pad; 11, patellar tendon; 12, patella.

attach on the most caudal region of the tibial footprint area (Arnoczky & Marshall 1977; Heffron & Campbell 1978).

The caudal cruciate ligament (CaCL) is slightly longer and broader than the CrCL (Rudy 1974; Arnoczky & Marshall 1977; Harari 1993). Even its collagen fibrils are thicker compared with its cranial counterpart (Brunnberg 1989). The total mid-section diameter is smallest as it fans out from the center, making the femoral and, to a lesser extent, the tibial attachments larger (Rudy 1974). In the dog the CaCL also has two components, although they are less distinct and often inseparable (Heffron & Campbell 1978; Harari 1993).

Microanatomy

The cruciate ligaments are multifascicular structures, the base unit of which is collagen, that contain many wavy fascicular subunits (Figure 1.3A). Fascicles may be composed of one up to 10 subfascicles, containing bundles of collagen fibers (Heffron & Campbell 1978; Yahia & Drouin 1989). At the osseous attachment sites of the CrCL, the collagen fibers are not arranged entirely parallel to the longitudinal axis of the ligament and, especially in younger specimens, columns of chondroid cells do penetrate into the ligament (Figure 1.3B) (Zahm 1965; Alm & Strömberg 1974). Where both cruciate ligaments are in contact, the collagen fibers are more densely packed and oriented tangential to the surface instead of parallel to the long axis (Vasseur *et al*. 1985). Fibers are formed by fibrils that are composed by organization of repeated collagen subunits (Alm & Strömberg 1974; Heffron & Campbell 1978; Vasseur *et al*. 1985). Their architecture is a combination of helical or planar, parallel, or twisted networks. The centrally located collagen fibrils are nearly straight, whereas those at the periphery are arranged in a helical wave pattern (Zahm 1965; Alm & Strömberg 1974; Yahia & Drouin 1989).

Alongside collagen, elastin fibers and bundles of microfibrils (fibrillin 1 and 2) are abundant in cruciate ligaments, both within the collagen bundles and in the interfascicular regions (Figures 1.4AB) (Smith *et al*. 2011). Those fibers are predominantly orientated parallel to the collagen bundles, and this study suggested a mechanical role for their distribution during ligament deformation.

As evident in other ligaments, the cruciate ligaments are characterized by relative hypocellularity. Where both cruciate ligaments are in contact, the epiligament is more cellular than anywhere else (Smith *et al*. 2012). In the interfascicular regions, the cells are interspersed

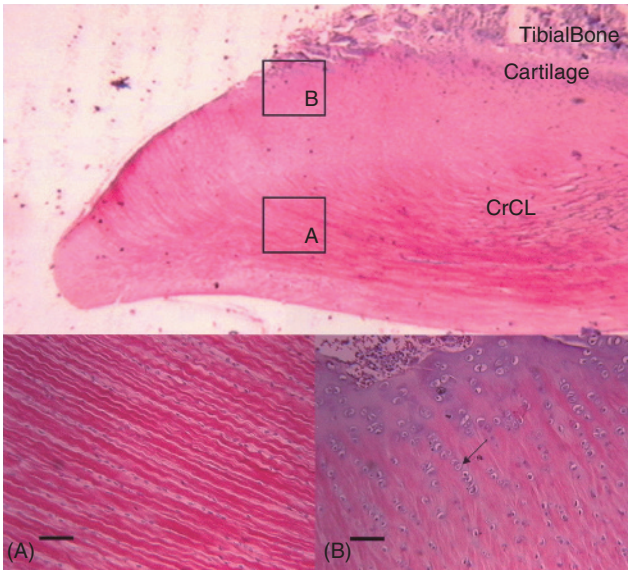

Figure 1.3 Histologic section (H&E stain) of a normal cranial cruciate ligament (CrCL) of a 4-month-old Riezenschnauzer. (A) Along the CrCL, dense collagen is aligned parallel to the long axis of the ligament. The collagen fibers have a regular accordion-like pattern. (B) At the attachment site of the CrCL, the collagen is not arranged entirely parallel to the long axis of the ligament. Columns of chondroid cells (arrow) do penetrate into the CrCL (scale bar = 100 μm). Source: de Rooster *et al*. 2006. Reproduced with permission from John Wiley & Sons, Inc.

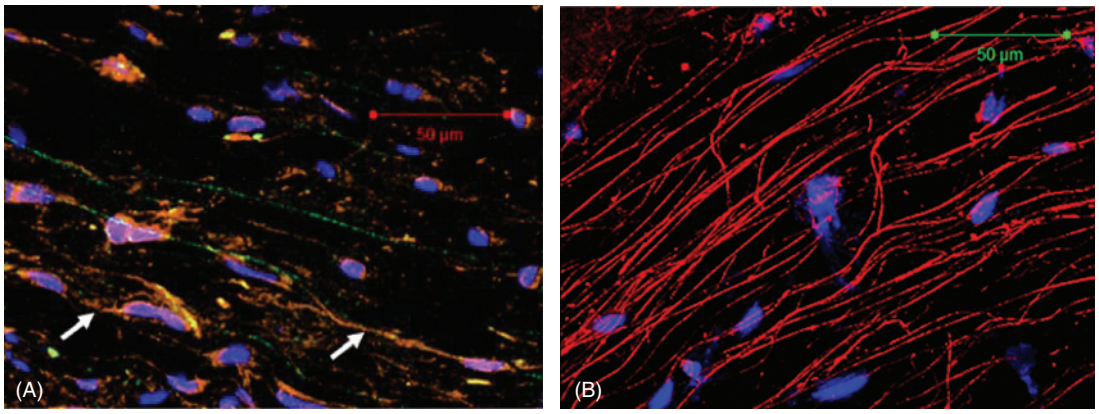

Figure 1.4 (A) Distribution of fibrillin-1 in the canine cruciate ligament complex, longitudinal section cranial cruciate ligament (CrCL), ×63 confocal laser scanning microscopy (CLSM) image, enzymatic pre-treatment. Fibrillin 1 (orange) is found pericellularly where nuclei are rounded (nuclei shown in blue). Staining of fibrillin 1 is also seen extending parallel to collagen in a fiber-like structure (arrows) from an elongated nucleus. Although some co-localization (yellow) is seen with elastin (green), elastin fibers were generally found to contain little fibrillin 1. Scale bar = 50 µm. (B) Distribution of fibrillin-2 in the canine cruciate ligament complex, longitudinal section CrCL, ×63 CLSM image from fascicular region showing fibrillin-2 (red) in long and dense fibers broadly aligned with collagen bundles, with some branching. Nuclei are stained with DAPI (blue). Scale bar = 50 µm. Source: Smith *et al*. 2011. Reproduced with permission from John Wiley & Sons, Inc.

between bundles of collagenous fibers. Most are interconnected by cytoplasmic projections, which often branch markedly, forming a three-dimensional cellular lattice around those fibers. Differences in cell morphology were also found in dog breeds with high and low risks of cruciate ligament rupture. CrCLs from low-risk breeds have longer cytoplasmic projections, while high-risk CrCLs have rounder nuclei and shorter cytoplasmic projections. These changes may be indicative of a reduced communication between cells (Smith *et al*. 2012).

Synovial envelope

Both the CrCL and the CaCL are covered by a fairly uniform fold of synovial membrane which incompletely divides the stifle joint in the sagittal plane (Arnoczky *et al*. 1979). These enveloping epiligamentous membranes consist mainly of dense connective tissue, small fibroblasts, and some adipocytes; an intima and a thin subintimal layer can be distinguished (Heffron & Campbell 1978). The intima is a single layer of synoviocytes, and the subintimal layer is areolar tissue containing small vascular structures (Vasseur *et al*. 1985). Compared with the collateral ligaments, the enveloping synovial membrane is relatively cellular (Heffron & Campbell 1978). Synovial lining does not occur on the surfaces of the cruciate ligaments that are in direct contact with each other (Vasseur *et al*. 1985). When examined with scanning electron microscopy, many small holes have been detected in the synovial membrane covering the cruciate ligaments, suggesting that the cruciate ligaments are also supplied with nutrients via the synovial fluid (Kobayashi *et al*. 2006).

Vascular supply

The major vascular contribution to the center of the stifle joint occurs from branches of the middle genicular artery, which arises from the popliteal artery, penetrates the caudal joint capsule, and passes craniodistally to the fossa intercondylaris, running cranially between the cruciate ligaments (Figures 1.5 and 1.6) (Tirgari 1978). The vascular structures in the proximal part of the CrCL are more numerous and have a larger diameter compared with those on the tibial side (Zahm 1965; Alm & Strömberg 1974).

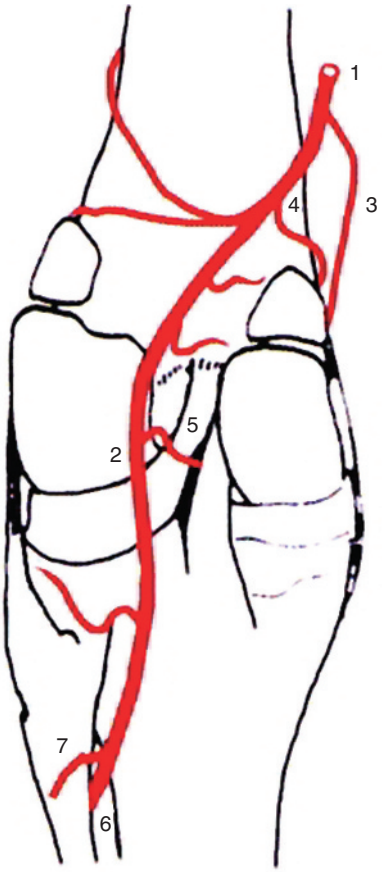

Figure 1.5 Line drawing of the major blood supply to the canine stifle joint. Caudal view. 1, femoral artery; 2, popliteal artery; 3, descending genicular artery; 4, proximal medial genicular artery; 5, middle genicular artery; 6, cranial tibial artery; 7, caudal tibial artery. Source: de Rooster *et al*. 2006. Reproduced with permission from John Wiley & Sons, Inc.

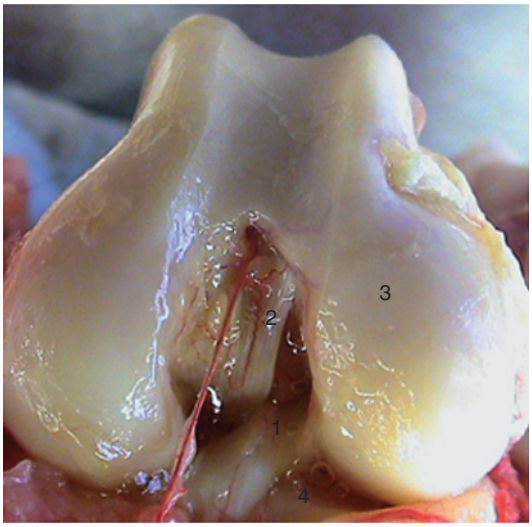

Figure 1.6 Photograph of the superficial vascularization of normal cruciate ligaments after injection of latex in an *ex vivo* specimen of an adult dog. The infrapatellar fat pad and the synovial envelope are the most important sources of vessels. 1, cranial cruciate ligament; 2, caudal cruciate ligament; 3, lateral femoral condyle; 4, tibial plateau. Source: de Rooster *et al*. 2006. Reproduced with permission from John Wiley & Sons, Inc.

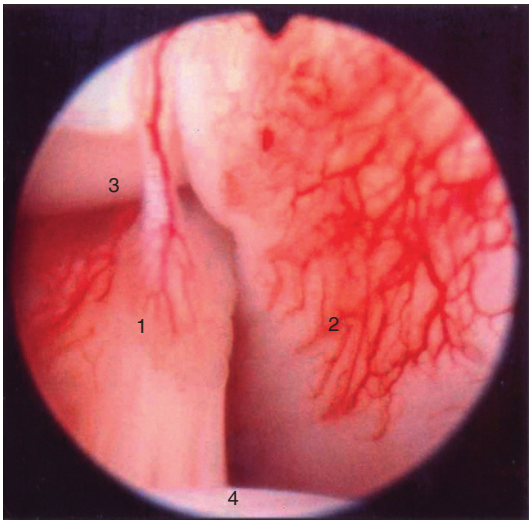

Figure 1.7 Arthroscopic view of the superficial vascularization of normal cruciate ligaments of an adult dog. The synovial vessels arborize to form a web-like network of periligamentous vessels that ensheath the cruciate ligaments. 1, cranial cruciate ligament; 2, caudal cruciate ligament; 3, lateral femoral condyle; 4, tibial plateau. Source: de Rooster *et al*. 2006. Reproduced with permission from John Wiley & Sons, Inc.

The blood supply to both cruciate ligaments is predominantly of soft tissue origin; the contribution from the osseous attachments is negligible (Arnoczky *et al*. 1979; Kobayashi *et al*. 2006). The infrapatellar fat pad and the well-vascularized synovial membranes, which form an envelope around the cruciate ligaments, are the most important sources of vessels (Alm & Strömberg 1974; Tirgari 1978; Arnoczky *et al*. 1979, Kobayashi *et al*. 2006). The synovial vessels arborize into a finely meshed network of epiligamentous vessels which ensheath the cruciate ligaments throughout their entire length (Figures 1.7 and 1.8) (Arnoczky *et al*. 1979;

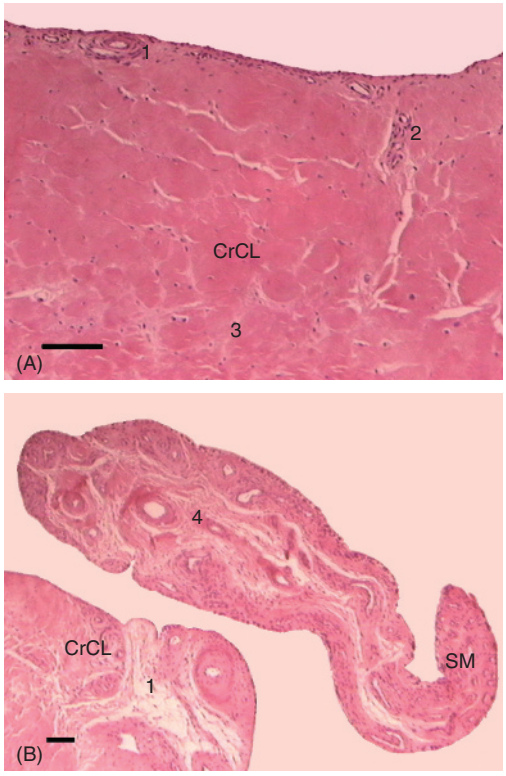

Figure 1.8 Histologic section (H&E stain) of a normal cranial cruciate ligament (CrCL) of an adult dog. (A) The CrCL is ensheathed by epiligamentous vessels (scale bar = 100 μm). (B) The well-vascularized synovial membrane (SM) forms an envelope over the CrCL (scale bar = 100 μm). 1, epiligamentous vessels; 2, anastomosis between epiligamentous and endoligamentous vessels; 3, hypovascular zone; 4, synovial vessels. Source: de Rooster *et al*. 2006. Reproduced with permission from John Wiley & Sons, Inc.

Kobayashi *et al*. 2006). In general, the vascular arrangement and structural characteristics of the vasculature inside the CrCL and the CaCL are similar (Alm & Strömberg 1974; Arnoczky *et al*. 1979; Kobayashi *et al*. 2006). In the inner part of the cruciate ligaments, around and along the bundles of collagen fibers, an endoligamentous vascular network courses in the supporting connective tissue (Alm & Strömberg 1974; Arnoczky *et al*. 1979; Hayashi *et al*. 2011). The larger vessels, usually one artery accompanied by two veins, mainly course in a longitudinal direction both proximally and distally and lie parallel to the collagen fascicles (Alm & Strömberg 1974). Some of them have a tortuous path in the interfascicular areolar tissue. Only small capillaries branching from the longitudinal endoligamentous vessels run in a transverse direction, encircling collagen bundles. The core of the mid-portion of the CrCL is less well vascularized compared with the remainder of the ligament (Zahm 1965; Tirgari 1978; Arnoczky *et al*. 1979; Vasseur *et al*. 1985; Hayashi *et al*. 2011).

Anastomoses exist between extra- and intraligamentous blood networks (Alm & Strömberg 1974; Arnoczky *et al*. 1979; Kobayashi *et al*. 2006). Epiligamentous vessels penetrate transversely into the cruciate ligaments (Figure 1.8). Their branches ramify and anastomose with the endoligamentous vessels. There are numerous endosteal vessels at the ligamentous–osseous junctions; however, communications with intrinsic endoligamentous vessels are quite poor, especially at the tibial attachment where most of the endosteal vessels seem to terminate in subchondral loops instead of crossing the ligamentous–osseous junction (Alm & Strömberg 1974; Arnoczky *et al*. 1979; Kobayashi *et al*. 2006).

Innervation

Three major articular nerves arise from the saphenous nerve, tibial nerve, and common peroneal nerve to innervate the periarticular tissues of the canine stifle joint (Figure 1.9) (O'Connor & Woodbury 1982). The main trunk of nerve bundles is found at the femoral end of the cruciate ligaments. Other nerves may contribute afferent fibers to a variable extent to the cruciate ligaments.

In dogs, the medial articular nerve, which branches from the saphenous nerve in the mid-thigh region, provides the largest contribution to stifle joint innervation (O'Connor & Woodbury 1982). Some of its branches course through the infrapatellar fat pad to terminate within the proximal or distal attachments of the cruciate ligaments or within the meniscal poles. Other branches of the medial articular nerve pass cranially through the joint capsule to

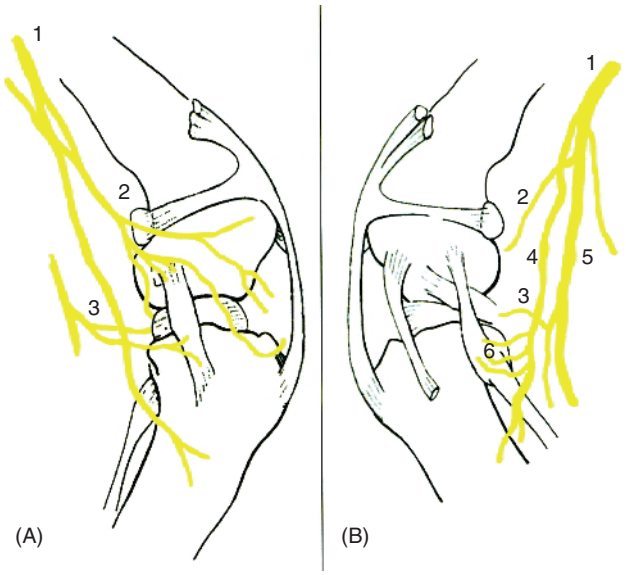

Figure 1.9 Line drawing of the major nerve supply to the canine stifle joint. (A) Medial view. (B) Lateral view. 1, saphenous nerve; 2, medial articular nerve; 3, caudal articular nerve; 4, common peroneal nerve; 5, tibial nerve; 6, lateral articular nerve. Source: de Rooster *et al*. 2006. Reproduced with permission from John Wiley & Sons, Inc.

extensively innervate the femoral attachment of the CaCL. The caudal articular nerve is variably present in dogs (O'Connor & Woodbury 1982), its branches arising either directly from the tibial nerve or from a muscular branch of the tibial nerve. The caudal articular nerve runs to the caudal aspect of the joint capsule, where it may communicate with branches of the medial articular nerve. The lateral articular nerve branches from the common peroneal nerve at the level of the fibular head, deep to the biceps femoris muscle, and supplies the lateral aspect of the stifle joint (O'Connor & Woodbury 1982).

Nerves of differing sizes are located in the richly vascularized synovial tissue covering the cruciate ligaments (Yahia *et al*. 1992). From this peripheral synovium, axons radiate towards the center of the ligaments (Yahia *et al*. 1992). Within the cruciate ligaments, most nerves course along the epiligamentous and endoligamentous blood vessels in the interfascicular areolar spaces.

Neurohistologic studies have identified various types of sensory nerve endings (receptors and free nerve endings) in the middle of the cruciate ligaments, well beneath the synovial sheath (Yahia *et al*. 1992). The highest number of mechanoreceptors was found in the proximal third of the CrCL, and the lowest in the distal third (Arcand *et al*. 2000). A high percentage of mechanoreceptors have been found in the tibia remnants of ruptured human anterior cruciate ligaments (ACLs), and it has been suggested that leaving these remnants after ACL reconstruction may be important for postoperative proprioceptive function (Sha & Zhao 2010).

Functional anatomy

The cruciate ligaments resist forces that would cause the tibia to translate cranially relative to the femur and, to a lesser degree, resist forces that would cause tibial rotation (Arnoczky & Marshall 1977). The two components of the CrCL are not isometric, the main difference being elongation of the craniomedial and shortening of the caudolateral component during flexion (Arnoczky & Marshall 1977; Heffron & Campbell 1978). The former is the major contributor to craniocaudal stability in stifle flexion, while the latter only contributes when the craniomedial band is damaged or severely stretched (Wingfield *et al*. 2000). With the stifle in extension, both components are taut, and limit cranial translation of the tibia relative to the femur (Arnoczky & Marshall 1977; Heffron & Campbell 1978).

As the stifle flexes, the cruciate ligaments are not only wrapped upon each other but also spiral on themselves (Singleton 1957; Arnoczky & Marshall 1977). The higher strain in the ligaments also limits the amount of normal internal rotation of the tibia relative to the femur (Zahm 1965; Arnoczky & Marshall 1977; Harari 1993). In extension, the medial and lateral collateral ligaments become the primary restraints of rotation, and the cruciate ligaments provide only a secondary check from the tension in both ligaments (Singleton 1957; Zahm 1965; Vasseur *et al*. 1985).

Both cruciate ligaments together provide important secondary restraints against varus and valgus angulation. The cruciate ligaments become primary restraints if there is loss of collateral ligament support (Vasseur & Arnoczky 1981).

Overextension is prevented by tension in the cruciate ligaments, where the CrCL acts as the primary restraint (Arnoczky & Marshall 1977; Heffron & Campbell 1978). The caudolateral component of the CrCL is the primary contributor to restraining hyperextension (Heffron & Campbell 1978). The slightly longer caudal component of the CaCL can only be considered a secondary restraint (Singleton 1957; Arnoczky & Marshall 1977).

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