

Current thoughts on canine cranial cruciate ligament disease

Eithne J. Comerford

MVB, PhD, CertVR, CertSAS, MRCVS, Liverpool, UK



INTRODUCTION

The cranial cruciate ligament (CCL) is the major contributor to craniocaudal stability of the canine stifle (Arnoczky, 1977). Disease and rupture of CCL is the most common cause of hindlimb lameness in the dog (Bennett, 1988). CCL injury occurs less frequently in the cat but is not uncommon (Scavelli, 1987). Despite this it is still a poorly understood disease and the subject of much debate amongst veterinary orthopaedic surgeons. The condition is often bilateral and, in such case, it must be differentiated from bilateral hip disease, lumbosacral disease and neurological disease.

CCL Anatomy and Physiology:

The CCL originates on the caudomedial aspect of the lateral condyle and inserts on the craniomedial aspect of the tibia just caudal and underneath the intermeniscal ligament. The CCL has two distinct functional bands - a craniomedial band and a caudolateral band. The craniomedial band is taut when the joint is in flexion and extension, whereas the caudolateral band is taut in extension only. The amount of cranial drawer movement present in a stifle will depend on the state of these two bands since a partial tear can affect them to differing degrees. When we are testing a stifle joint for instability we should try and elicit cranial drawer with the joint in flexion and extension.

Cranial tibial thrust:

The stifle is subjected to external ground reaction forces when weight bearing and internal forces such as muscle contraction. The quadriceps/gastrocnemius muscles exert a cranial force and the hamstrings/biceps femoris exert a caudal force. The combinations of these forces generate a cranially directed shear force in the tibia called cranial tibial thrust (Slocum Slocum, 1993). It has been shown that the larger the tibial plateau angle (TPA), the greater the tibial thrust (Warzee, 2001).

Aetiopathogenesis:

CCL rupture is commonly a degenerative condition (cruciate disease) rather than purely traumatic, this is important in term of the diagnosis and management of this condition. The aetiopathogenesis is still poorly understood but several theories exist such as abnormal conformation and gait, intercondylar notch abnormalities, tibial plateau angle (Macias, 2002, Read, 1982), immune-mediated disease

(Bariet *al*, 1989), obesity, collagen degradation (Comerford, 2005, Hayashiet *al*, 2003) and patellar luxation.

Diagnosis of cruciate disease:

Signalment: Any breed of dog or cat can present with this condition but medium to large breed dogs are over represented. *History and clinical signs/examination-* should indicate the affected limb and may give us a high index of suspicion of condition in the consulting room, but further diagnostic tests may be necessary. Presentation is usually acute after a period of chronic or insidious signs. Examine for atrophy of the quadriceps muscle mass, range of motion, joint click, joint effusion and medial buttress. *Other tests* include tibial thrust and cranial draw tests, radiography, arthrocentesis, arthroscopy, scintigraphy and MRI.

Treatment of cruciate disease:

Very controversial and most orthopaedic surgeons have their own preferred treatment plan. The key features that influence decision-making are lameness, bodyweight, expertise and equipment, meniscal pathology, femorotibial pathology, immune-mediated disease, response to previous treatment. In general, conservative management is advocated in dogs < 15kg (Vasseur, 1984) (but late meniscal injury (LMI) has been reported to be over-represented in small dogs (Metelman, 1995) and surgical management for dogs > 15kg.

Conservative management: Useful in small dogs < 15kg, where lameness is mild and intermittent, if there is a systemic condition which contraindicates surgery, or financial constraints. The most important aspects of management are weight restriction, exercise regulation, analgesia, and (hydrotherapy). 85% will have satisfactory outcome with conservative management (Vasseur, 1984) but may have a quicker recovery with surgical treatment.

Surgical management: The main aims of surgery are to restore stability to the femorotibial joint, to remove any damaged structures (e.g. CCL and medial meniscal tears) and to reduce tibial thrust. These aims can be achieved by loosely dividing all reported procedures into three different types of techniques.

1. Intra-articular: restores stability by replacing ligament with some type of graft (e.g. modified “over –the-top” (OTT) technique with a fascia lata graft).

2. **Extra-articular:** restores stability by using sutures or soft tissues as a sling (e.g. Fabellotibial suture with Leader line nylon).
3. **Peri-articular:** Alter local anatomy to improve stability e.g. TPLO or fibular head transposition (FHT)). The aim of the tibial wedge osteotomy or tibial plateau leveling osteotomy is to reduce the TPA and thus reduce the cranial thrust. With both techniques the TPA is reduced to 6 degrees (Warzee, 2001).

Tibial tuberosity advancement (TTA) is a procedure, which is currently being used at the University of Liverpool in some patients with CCL disease as part of an ongoing clinical trial. The procedure is based on the theory that the total joint force runs nearly parallel to the patellar ligament. Therefore advancement of the tibial insertion of the patellar ligament should reduce the tibiofemoral shear forces generated between the tibial plateau and the patellar ligament leaving the CCL unloaded. Anecdotal reports of this procedure report good short and long term outcomes but objective, long term follow-ups of patients have yet to be published.

Post-operative management: The patient should be restricted (+NSAIDs) until suture removal at 7-10 days post-op and should be lead exercised with gradual increase over the following 8-12 weeks. Recent studies advocate the use of hydrotherapy, stretching and passive range of motion exercises as well as walking in the rehabilitation of patient after surgical management of a CCL injury (Marso-lais, 2002).

Prognosis: Whilst most papers on treatment of cruciate disease will give success rates between 85% and 90%, these 'successes' will include dogs with continuing mild intermittent lameness. However, all dogs have some degree of osteoarthritis and therefore there are clearly unknown factors producing this variability in outcome. It is wise to warn owners that maximal recovery will not be evident until 12-16 weeks (or more) following surgery with most techniques.

Meniscal injuries

Anatomy: The menisci are anchored to the tibia and femur by five ligaments and to one another by the inter-meniscal ligament. The medial meniscus has an attachment to the medial collateral ligament which renders it less mobile and consequently more prone to damage associated with cranial cruciate ligament insufficiency and craniocaudal instability of the tibia with respect to the femur.

Aetiopathogenesis: It is thought that the medial meniscus becomes trapped and injured by rotation of the femoral condyles at full extension during stance. This can occur with rupture of the CCL, which is when we see most injuries to the medial meniscus. The lateral meniscus, being more mobile, is less likely to become trapped. There are a variety of meniscal injuries and these have been classified by Bennett and May (1991).

The incidence of meniscal injury in CCL varies amongst published papers (49-53%) but it seems more common in chronic cases and heavier dogs. "Late-meniscal" injury (meniscal damage to a normal meniscus following surgery) occurs a period of time after CCL surgery and can occur in 14% of cases depending on surgical technique used (Metelman, 1995).

Clinical signs: These dogs will present suddenly lame several weeks to months after initial CCL surgery. The joint may have an effusion and be unstable upon cranial draw test.

It is **imperative** that meniscal lesions are identified and treated. A partial meniscectomy should be performed. The most common injuries are bucket handle tears and a fold of the caudal horn of the medial meniscus. These injuries are currently treated with a partial meniscectomy in our patients.

References

- Arnoczky SPM, J.L. (1977) The Cruciate Ligaments of the Canine Stifle: An Anatomical and Functional Analysis. *American Journal of Veterinary Research* 38, 1807-1814
- Bari ASM, Carter SD, Bell SC, Morgan K and Bennett D (1989) Anti-type II collagen antibody in naturally occurring canine joint diseases. *British Journal of Rheumatology* 28, 480-486
- Bennett DT, B., Lewis, D.G, et al (1988) A reappraisal of anterior cruciate ligament disease in the dog. *Journal of Small Animal Practice* 29, 275-297
- Comerford EJ, Tarlton, John F., Innes, John F., Johnson, Kenneth A., Amis, Andrew A., Bailey, Allen J. (2005) Metabolism and composition of the canine anterior cruciate ligament relate to differences in knee joint mechanics and predisposition to ligament rupture. *Journal of Orthopaedic Research* 23, 61-66
- Hayashi K, Frank JD, Dubinsky C, Zhengling H, Markel MD, Manley PA and Muir P (2003) Histologic changes in ruptured canine cranial cruciate ligament. *Vet Surg* 32, 269-277.
- Macias C, McKee, W., May, C. (2002) Caudal proximal tibial deformity and cranial cruciate ligament rupture in small breed dogs. *Journal of Small Animal Practice* 43, 433-438
- Marso-lais G, Dvorak, G., Conzemius, MG. (2002) Effects of postoperative rehabilitation on limb function after cranial cruciate ligament repair in dogs. *Journal of the American Veterinary Medical Association* 222, 1325-1330
- Metelman L, Schwarz, M., Salman, M., Alvis, MR (1995) An evaluation of three different cranial cruciate ligament surgical stabilization procedures as they relate to postoperative meniscal injuries: a retrospective study of 665 stifles. *Veterinary and Comparative Orthopaedics and Traumatology* 8, 118-123
- Read RA, Robins, GM (1982) Deformity of the proximal tibia in dogs. *Veterinary Record*, 295-298
- Scavelli T, Schrader, SC. (1987) Non-surgical management of rupture of cranial cruciate ligament in 18 cats. *Journal of the American Animal Hospital Association* 23, 337-340
- Slocum Band Slocum TD (1993) Tibial plateau leveling osteotomy for repair of cranial cruciate ligament rupture in the canine. *Vet Clin North Am Small Anim Pract* 23, 777-795
- Vasseur PB (1984) Clinical results following non-operative management of rupture of the cranial cruciate ligament in dogs. *Veterinary Surgery* 13, 243-246
- Warzee C, Dejardin, LM., Arnoczky, SP., Perry, RL. (2001) Effects of tibial plateau levelling on cranial and caudal tibial thrusts in canine cranial cruciate-deficient stifles: an vitro experimental study. *Veterinary Surgery* 30, 278-286