CCL disease, is there any superior technique?

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Cranial cruciate ligament disease is the most common cause of hindlimb lameness in the dog. The term disease is used to encompass the numerous different aetiologies present.

The cranial cruciate ligament prevents internal rotation, hyperextension and cranial drawer of the stifle. As a result hyperextension or severe internal rotation of the tibia that can occur during swift turning movements when a dog is playing can lead to damage of the ligament.

- ***Traumatic avulsion:*** Typically seen in younger skeletally immature animals when the bone is soft at the origin or insertion of the cranial cruciate ligament.
- ***Traumatic rupture of the CCL:*** Although this is the situation that is commonly observed in humans we rarely see this in dogs. Acute rupture occurs as a result of excessive hyperextension/internal rotation that excedes the ligaments inherent capacity to resist these forces.
- ***Degeneration of the CCL:*** Most common presentation. Rupture of the ligament may occur as an insidious process due to pathological weakening of the ligament by fibre degeneration.

Rupture of the cranial cruciate ligament should be considered with any dog presenting with stifle lameness. CCL injury may be associated with patellar luxation. Dogs with bilateral CCL rupture are often mistaken for having a neurological gait due to the bilateral pelvic limb lameness. Historical findings are varied, but may occur acutely or with a more insidious onset. With an acute history the lameness may occur as a result of a sudden twisting or turning movement. Degeneration of the CCL typically causes vague signs of lameness with or without concurrent stifle instability. The lameness may be gradual or there may be a sudden deterioration following complete rupture of the ligament or concurrent meniscal injury.

Clinical signs and physical examination findings
Clinical signs include:

- Muscle atrophy over the proximal thigh muscles.
- Pain on stifle manipulation especially on hyperextension of the stifle.
- Medial buttress is due to medial periarticular thickening of the soft tissues.
- Stifle effusion is identified by palpation and identification of the patellar tendon.
- Cranial drawer is a result of CCL deficiency and the resulting stifle instability detected. The figure below shows the correct hand position for performing cranial drawer. The stifle should be assessed in flexion and extension.
- Tibial thrust also detects stifle instability and mimics the force acting on the leg during normal weight bearing. The stifle joint is stabilised with the upper hand and maintained at a weight bearing angle. The index finger of the upper hand is placed on the tibial tuberosity.
- Positive sit test may be present. This is when animals keep the leg in abduction to avoid flexion of the stifle.

Cranial drawer and tibial thrust can sometimes be challenging in the conscious patient. If this is the case the animal should be examined under sedation. It is also important to recognise that young animals will have an inherent degree of laxity within the joint "puppy drawer". Animals with puppy drawer will have a distinct stop to the instability detected whereas dogs with joint instability will have a less distinct stop. The most sensitive and specific test for CCL deficiency is the presence of a stifle effusion.

1. Radiographic evaluation

Typical radiographic signs include:

- ***Joint effusion*****:** Increased soft tissue opacity within the joint and cranial displacement of the infrapatellar fat pad are early signs of stifle pathology. Caudal displacement of the intermuscular fat caudal to the stifle due to joint effusion is another consistent and early sign.
- ***Osteoarthristis*****:** An early finding is osteophytosis at the insertion site of the CCL, in the intercondylar area of the tibia, as well as in the distal pole of the patella.
- ***Distal displacement of the popliteal sesamoid bone:*** An accuracy of 99 per cent and a specificity of 100 per cent were achieved by assessing the localisation of the sesamoid bone in the diagnosis of cruciate disease.
- ***Avulsed fragments of bone:*** Avulsed fragments of bone at the origin or insertion of the CCL may be visualised with avulsion injuries.

Mediolateral and craniocaudal radiographs of both stifle joints should be taken. It is not uncommon to identify abnormalities consistent with early CCL disease in the contralateral unaffected stifle.

Numerous techniques have been reported to stabilise cranial cruciate disease in the dog and this is a highly controversial topic and undoubtedly will also be influenced by surgeon preference. Techniques can largely be divided into intra-articular, extracapsular and osteotomy techniques.

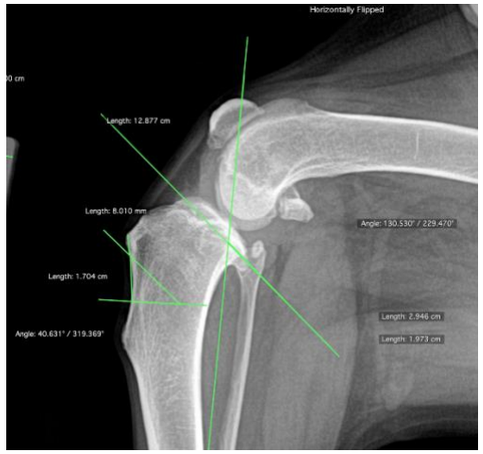
Intra-articular reconstruction was widely used in the past but are now not commonly used due to concerns regarding the inferior strength and outcome compared to other techniques.

Extracapsular or extraarticular stabilisation relies on periarticular fibrosis to achieve stability as the implants will eventually fail. Unless true isometric points are used to anchor the extacapsular implants the suture will not stabilise the stifle through a full range of movement. Whilst initial studies showed minimal differences between extracapsular sutures and TPLO procedures more recent studies have contradicted this showing poorer longer-term outcomes compared to TPLO dogs.

Several osteotomy techniques are currently available for the cruciate deficient stifle including plateau levelling procedures such as cranial closing wedge ostectomies (CCWO), tibial plateau levelling osteotomies (TPLO) and cora based levelling osteotomies (CBPLO). The compressive force across the joint is parallel to the tibial long axis, levelling of the plateau results in neutralisation of tibiofemoral shear force. In general terms the complications and outcome are similar with any of those techniques.

Advancement techniques that involve advancement of the tibial tuberosity include tibial tuberosity advancements (TTA) and various modifications of the procedures that may or may not involve a plate. The compressive force parallel to the patellar tendon in the cruciate deficient stifle results in a tibiofemoral shear force. Advancement of the patellar tendon results in neutralisation of tibiofemoral shear force and the joint force and resultant force become the same. The end result is theoretically that tibial thrust should be abolished. More recent work suggests that this may not be the case. Caution should be exercised when assuming that the original TTA procedure and more recent modifications are interchangeable. New plate-less tta modifications tend to produce more under advancement since the don't allow the tibial crest to 'jump-up' and commonly these 'plate-less TTA modifications' require a maquet hole or incomplete osteotomy to allow the advancement. Fissure propagation form this hole or incomplete osteotomies can result in complicated tibia fractures.

The body of evidence in the literature most strongly supports the ability of TPLOs in the ability to return dogs to normal function. Dogs with steep tibial plateaus (> 27 degrees) may not be candidates for tibial tuberosity advancement procedures as the amount of advancement required to neutralise tibiofemoral shear force is often excessive and they have been reported to underperform clinically. In summary TPLO or plateau levelling procedures have the greatest amount of evidence behind them, but one single procedure may not be appropriate for every dog given the variety in tibial conformation between breeds. Each case should be assessed on its individual merits and the answer is to be able to perform all osteotomies available.



Planification to execute a modified TCWO in a Labrador cross with a very narrow tibia and 40 degrees tibial plateau

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