Cranial cruciate ligament rupture and tibial tuberosity advancement

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IT is vital that the general practitioner has a good understanding of how to diagnose and, in many cases, treat the cranial cruciate ligament (CCL) deficient patient, as it is the most common cause of hindlimb lameness in the dog. This article provides a refresher in the diagnosis of CCL, and summarises a relatively new method of stifle stabilisation surgery known as tibial tuberosity advancement (TTA).

Anatomy

The cruciate ligaments are paired ligaments within the stifle and are named after their relative insertions into the tibial plateau (that is, the cranial ligament inserts cranially and vice versa). Together with the collateral ligaments, they provide stability to the joint. The paired action of the cranial and caudal cruciate ligaments act like a four-joint chain to allow both rolling and sliding of the femoral condyles over the tibial plateau (see Figure 1).

The CCL has four main functions within the joint:

• to oppose translation of the tibia with regard to the femur (“cranial draw”);

• to oppose internal rotation of the tibia;

• to oppose overextension of the femur; and

• to provide a limited degree of varus-valgus support during stifle flexion.

The CCL is further divided into cranio-medial and caudolateral bands, which, during stifle flexion, twist around each other, thereby limiting the amount of internal rotation of the tibia. The cranio-medial band is taut in all phases of stifte motion, but the caudo-lateral band is only taut during stifle extension, becoming relaxed during flexion. For this reason, stifle joints should be examined for cranial draw during both flexion and extension as partial tears of the ligament may otherwise be missed if performed only in extension.
Mechanoreceptors within the ligament itself provide feedback to prevent excessive flexion and extension of the joint. Nutrition to both cruciate ligaments comes primarily from the synovium. The natural healing potential of the ligament is very limited.

Another important structure to be aware of within the stifle is the meniscus—a fibrocartilaginous pad that acts as a “shock absorber” and compensates for incongruity between the tibial plateau and the femoral condyles. This pad is composed of two semilunar pads anchored to the tibia and femur by five ligaments, and to each other by the intermeniscal ligament. The medial menisci is further stabilised by an attachment to the medial collateral ligament—this makes it more immobile and hence more vulnerable to injury by the pinching and internal rotational forces of the CCL deficient stifle.

Blood supply to the menisci is also poor, especially to the inner portions, meaning the chance of healing is also slim. The menisci have a good nerve supply to aid with proprioception but this, in turn, can cause an extremely painful stifle when damaged.

**Aetiology**

Many different causes and theories exist to explain CCL rupture, the discussion of which are beyond the scope of this article. Possible causes are:

- **Trauma.**

  Trauma to an otherwise healthy ligament is probably a fairly uncommon cause of CCL rupture. This may come about when the CCL is tested beyond its normal breaking strain during stifle overextension or overrotation; a classic example being a foot down a rabbit hole.

- **CCL disease.** Studies suggest this is one of the major aetiologies for CCL deficient stifles in adult dogs. This occurs due to gradual degenerative and adaptive changes to the cells and matrix of the ligament associated with synovial inflammation, meaning only a small amount of trauma (or even normal force) is needed to elicit a rupture.

- **Inflammatory arthropathies.** Either immune mediated or infectious.

- **Anatomical.** Certain breeds, notably the West Highland white terrier, are often found to have steep tibial plateau angle (TPA), altering stifle mechanics and predisposing to CCL rupture. This condition can also occur with premature closure of the caudal aspect of the proximal tibial growth plate. In these cases, corrective osteotomy is often the treatment of choice. Various authors have investigated the effects of tibial plateau angle on the incidence of cruciate disease with varying results—some groups have shown no significant difference between TPA angles of CCL deficient and normal stifles of the same breed, while others have shown significant differences between greyhounds, and the stifles of affected Labradors. The mean TPA in dogs is around 23°. 


Clinical signs

Although CCL rupture is more common in large breed dogs such as Labradors, Rottweilers and mastiffs, it should be noted that any breed of dog can suffer CCL damage. Clinical signs include:

• Lameness. This can vary from non-weight bearing to mild and intermittent, depending on the nature and the chronicity of the rupture.

Acute ruptures are generally (but not exclusively) accompanied by severe non-weight bearing lameness. Chronic ruptures can vary in severity of lameness, but are often presented as weight-bearing or intermittently weight-bearing lame. Owners, on being questioned, often mention a period of acute lameness sometime previously. This is often accompanied by a difficulty in rising, increasing lameness post-exercise and after rest, and a characteristic “CCL rupture posture” when sitting, where the affected limb is held away from the body and extended to minimise the degree of flexion of the stifle and, therefore, pain. The instability in the stifle leads to the development of osteoarthritis, which is associated with chronic lameness.

Partial tears often show mild weight-bearing lameness associated with exercise, but often improving with rest; this can continue for many months before eventually completely rupturing.

All tears can be accompanied by meniscal injury, which will reduce the response rate to conservative management.

• Muscle atrophy. Over the affected limb, especially noted with chronic disease.

• Stifle effusion. This is best noted by a lack of definition of the patella tendon. It is often helpful in the unilaterally affected dog to feel the “normal” stifle in the contra lateral limb.

• Pain on flexion and extension.

• Medial buttress. This is a thickening on the medial aspect of the stifle as a result of osteoarthritis and soft tissue proliferation. It is best felt in the chronically affected stifle.

• “Clicking” or “popping”. These can often be heard or felt during stifle flexion and extension, and are due to the rolling and movement of a torn meniscus.

• Crepitus. This is due to osteophytes formed in the osteoarthritic joint.

• Joint instability. An assessment can be made while the animal is conscious, but it is far easier in the sedated or anaesthetised dog. When the animal is conscious, muscle contracture – due to apprehension and soft tissue proliferation from chronic injury – can often lead to false negatives. Two main methods are employed for this assessment and are best performed in lateral
– Cranial draw test (Figure 2): This must be done in both flexion and extension of the stifle. It is performed by positioning the index finger of the proximally placed hand over the dorsal femur (A) and the thumb on the caudal femur over the fabella (B). The distally placed hand has the index finger over the tibial crest (C) and the thumb over the caudal tibia (D). Force is then applied through the thumb (D) of the distally placed hand, pushing the tibial plateau forward. A movement of 0-2mm is expected in a normal stifle. If excessive cranial draw is noted, it indicates CCL rupture. A falsepositive result can be seen in young dogs due to an inherent laxity in the ligament that can give a normal draw movement of 4-5mm. In these young animals, a distinct endpoint to the drawing action is noted.

– Tibial compression test (Figure 3): This is best achieved with the limb in moderate flexion. It is performed by grasping the distal quadriceps in the proximally placed hand and laying the tip of one finger over the patella tendon and on to the tibial crest (A). The distally placed hand then grasps the metatarsal region and flexes the hock (B). If cranial movement of the tibial crest is noted, this indicates cruciate damage. It should be noted that both methods can show false-negative results in partial tears, particularly if performed only in extension.

**Diagnostic imaging**

A high level of suspicion of CCL rupture should be followed by further investigation. Many imaging modalities exist for the diagnosis of CCL disease, including ultrasound, MRI, and arthroscopy, but for the general practitioner, radiography continues to be the modality of choice. For this reason, it is the only method discussed in this article.

Radiography is always helpful in confirming a diagnosis of CCL rupture and should be used to rule out other possible causes of stifle lameness. It is especially useful with chronic and partial tears where other clinical signs may have been lacking or missed. It is also necessary in the planning phase of correction methods including TTA, tibial plateau levelling osteotomy (TPLO) and other corrective osteotomy techniques.

Medio-lateral and caudocranial radiographs should always be obtained, but it is the mediolateral view that is often the most helpful. “Normal” limb views can be used for comparison. Radiographic features that are often seen are shown in Figure 4, and include:

• cranial fat pad effusion and loss of definition – noted by an increase in opacity of the cranial joint space (A);

• caudal joint capsule extension due to joint effusion (B);

• distal displacement of the popliteal sesamoid, noted as a relative change during tibial
compression and particularly useful in the diagnosis of partial CCL rupture; and

- osteophyte formation, which is seen particularly in cases of chronic joint instability and partial tears. Osteophytes can be seen over the trochlear ridges of the femur (C), around the fabellae (D), the caudal surface of the tibial plateau (E), and the distal patella (F).

**Treatment**

Treatment of CCL disease can be loosely divided into medical and surgical methods. It is beyond the scope of this article to discuss all possible surgical techniques, but it should be noted that success rates of CCL surgery are reported to be more than 90 per cent, regardless of the surgical technique used. This article focuses only on the relatively new method (five to six years in general use) of tibial tuberosity advancement (TTA).

TTA works by altering the angle between the pull of the quadriceps (patella) tendon, and the tibial plateau.

To understand this, we first need to understand some of the forces acting on the stifle:

- TTA uses the proven theory that in the stifle there is a large tibiofemoral compressive force, approximately the same magnitude and direction as the patella tendon, pulling the tibia cranially ([Figure 5](#)). This force is normally counteracted by the CCL (the absence of a functional CCL allows the cranial draw action), and is best seen during stifle extension (A).

- As the stifle flexes, the force on the patella tendon becomes perpendicular to the tibial plateau and is, therefore, neutralised (B).

- At full flexion, the direction of the force changes and causes a caudal draw action (opposed by the caudal cruciate ligament; C).

- TTA works by advancing the attachment of the quadriceps mechanism cranially, thereby making the angle between the patella tendon and the tibial plateau perpendicular, or at an angle less than 90° in all phases of motion. This means there is no cranial draw force on the tibial plateau. A caudal draw force still exists.

- TTA requires specific equipment and implants, namely an oscillating saw, TTA jig, TTA “plates” and “forks” and TTA “cages” ([Figure 6](#)).

- Implant selection is made after medio-lateral radiography of the limb in a standing (approximately 135°) stance.

- The procedure is performed via a medial approach to the stifle and proximal tibia, which also
enables the surgeon to make a limited medial arthrotomy to assess the condition of the menisci.

- Using a specific jig, a number of holes are drilled into the proximal tibial crest, where the plate holding fork will eventually sit. The number of holes needed will depend on the size of plate required, which is determined by the size of the limb.

Once all holes are complete, an osteotomy of the tibial crest is made using an oscillating saw. The mobile tibial crest is then rotated forward a set distance, bringing the pull of the quadriceps perpendicular to the tibial crest. The crest is held away from the tibial body using a “cage” with two screws. The maximum distance of advancement and, therefore, the largest cage size is 12mm.

- The centre of the cage is then packed with a cancellous bone graft to aid rapid osseogenesis.

Aftercare is aimed at exercise restriction postoperatively until bone healing can be observed radiographically (Figure 7); one study put the mean time for this at 11.3 weeks, while others have reported advanced healing in 94 per cent of cases reviewed at six weeks postop.

Major complications of this procedure include postoperative meniscal trauma, tibial tuberosity fracture, tibial fracture and implant failure, and occur at a similar rate to those reported with treatment by tibial plateau levelling osteotomy (TPLO). As with any technique, there are limitations that will affect case selection. The main limitation with TTA is that in dogs with excessive tibial plateau angles (TPA), a TTA in excess of 12mm would be required in order to create the desired 90° angle. In such dogs, other techniques such as TPLO may be better suited. This cut-off angle is still up for debate and no data has been published regarding the range of TPA in dogs with TTA. However, it has been presented that successful procedures have been performed in dogs with TPA of approximately 30°.

Short-term reports of this procedure are promising, with a good to excellent outcome in 85 to 97 per cent of cases assessed subjectively by owners.

Overall, TTA is showing extremely good short-term results and provides a good alternative to other stifle stabilisation techniques. It may also have an advantage over other osteotomy techniques as technical aspects are fewer and it appears to have a shorter learning curve.

References


Figure 1. Diagram showing rolling motion of the femoral condyle over the tibial plateau during stifle flexion and the relative positions of the cranial (orange) and caudal (blue) cruciate ligaments. Based on human stifle joints. Diagram adapted from lecture material by Dr H. Schmokel, course notes in cruciate repair, Improve International Ltd.
Figure 2. Cranial draw test.
Figure 3. Tibial compression test.
Figure 4. Radiographic features often seen include: cranial fat pad effusion and loss of definition; caudal joint capsule extension; distal displacement of the popliteal sesamoid; and osteophyte formation.

Figure 5. (A) Stifle in full extension showing an overall cranial draw force (orange arrow)
when the angle of the patella tendon (white arrow) to the tibial plateau (blue) is more than 90°. (B) Stifle in mid flexion showing no draw force when the patella tendon is perpendicular to the tibial plateau. (C) Stifle in flexion showing an overall caudal draw force (orange arrow) when the patella tendon is at an angle less than 90° to the tibial plateau.

Figure 6. (left to right). 2 x 6mm cages, largest eight hole plate and fork, six hole plate and fork, three hole plate and fork, and two hole plate.
Figure 7. Postoperative radiographs.