

Cranial cruciate ligaments in dogs: why do they rupture and how can we fix it

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1 Anatomy

The cranial cruciate ligament (CrCL) runs from the caudomedial part of the lateral condyle of the femur diagonally across the intercondylar fossa to the cranial intercondyloid area of the tibia. The caudal cruciate ligament (CaCL) runs from the lateral surface of the medial femoral condyle caudodistally to the lateral edge of the popliteal notch of the tibia. The cruciate ligaments consist of two anatomically and functionally different parts. The CrCL has a stronger caudolateral and somewhat smaller craniomedial part. In extension of the stifle joint, both parts are taut. In flexion, the caudolateral band is loose whereas the craniomedial band is taut. The cruciate ligaments receive their blood supply from vessels of the synovial tissue ensheathing them.

As the stifle flexes, the lateral collateral ligament relaxes as a result of its attachments. This allows cranial displacement and internal rotation of the tibia. The reverse occurs in extension.

The lateral and medial menisci are semilunar, fibrocartilaginous discs. The lateral meniscus is slightly greater than the medial one. The transverse intermeniscal ligament is a band between their cranial horns. The lateral meniscus has a cranial and a caudal tibial ligament and a menisiofemoral ligament. This makes it more mobile with respect to the femur. The medial meniscus has a cranial and caudal tibial ligament and an attachment to the medial collateral ligament. This causes less mobility with respect to the femur and full load transmission may be placed on the caudal rim of the medial meniscus.

It is the craniomedial part of the CrCL which resists primarily against hyperextension and cranial displacement of the tibia. Secondary constraints against cranial movement are provided by the joint capsule, the menisci, the collateral ligaments, the muscle forces and the shape of the tibial plateau (Fig. 1).

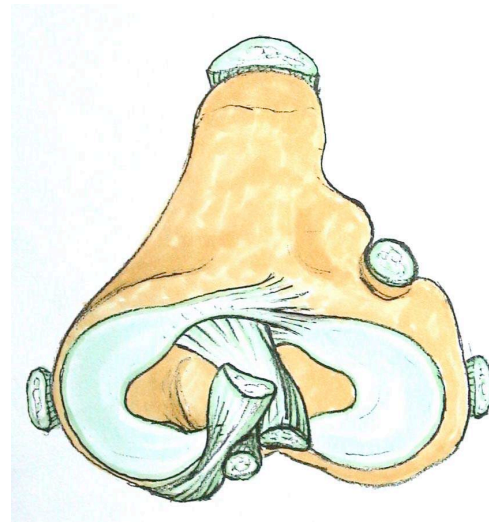


Figure 1: Illustration of the tibial plateau in a proximodistal view, showing the course of the CrCL and CaCL.

2 Pathophysiology

Traditional explanations of the pathophysiology of cranial cruciate ligament rupture in dogs included trauma or multiple traumas to the stifle joint. This was derived from human orthopedic surgery, where this correlation is obvious. However, there are several facts, which speak against it. (1) histologic examinations of ruptured CrCL showed sign of degeneration (Geyer, 1966), (2) the anamnesis mostly demonstrates first partial, then complete ruptures, (3) there is seldom a heavy impact onto the stifle joint before the rupture is diagnosed, (4) stifle biomechanics is different in humans and in dogs, (5) there are breed predilections for CrCL ruptures (Whitehair, 1993), (6) the heavier the dog, the earlier the rupture, (7) the contralateral stifle is affected in many cases, and (8) even in so called “acute ruptures”, we see osteoarthrotic signs on the stifle radiographs.

It was due to Barclay Slocum (1983), that the pathophysiology of the CrCL rupture was reassessed by the introduction of muscle forces. Further investigations and the success of the newly developed methods to treat CrCL rupture have led the following concept, which is given in a simplified form.

The quadriceps muscle, together with the body weight, is the main force acting on the stifle joint. It inserts on the tibial tuberosity by way of the patellar ligament. The angle between the direction of the quadriceps force and the tibial plateau is dependent on the standing phase of the hindlimb, but is mostly higher than 90° . According to the vector analysis, the quadriceps force can be split up into a force perpendicular to the tibial plateau and a resulting force towards cranial. This latter force is called cranial tibial thrust (CTT). It is counteracted by the CrCL and other passive elements in the stifle joint such as collateral ligaments, joint capsule or caudal horns of the menisci (Fig. 2).

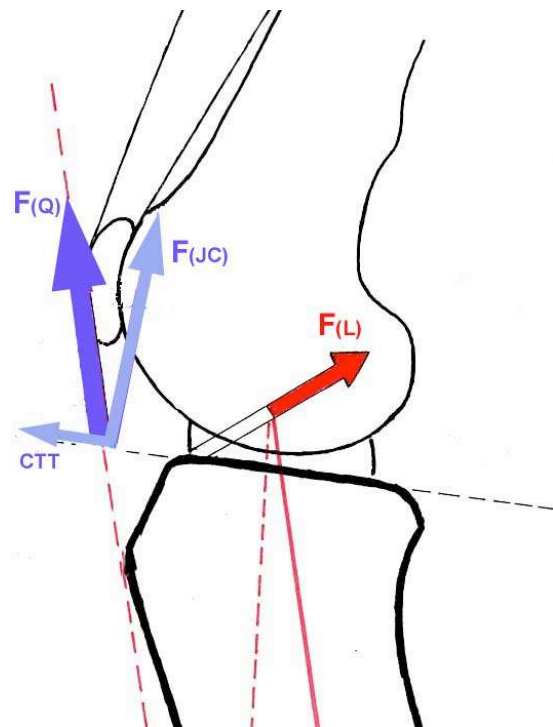


Figure 2: Schematic presentation of the main forces in the canine stifle joint. $F(Q)$ = Force of the quadriceps muscle, $F(JC)$ = Joint compressive force, CTT = cranial tibial thrust, $F(L)$ = counterforce of the CrCL. $F(Q) = F(JC) + CTT$.

In small dogs, the CTT is well balanced by the CrCL. However, there are factors, which may increase the CTT. These are overweight, large breed dogs and overextension in the stifle joints. If we look carefully to the possible candidates for a CrCL rupture, we can support these reflexions: overweight Labrador retrievers, Rottweilers, Newfoundland dogs, Mastiffs, Bullterriers. Recent research has also shown, that a relative thin tibial crest promotes the rupture of the CrCL (Inauen, Koch, Bass, 2008), thereby resulting in a unfavourable angle of insertion of the quadriceps muscle and increasing the CTT. Finally, it is only a question of time, until the CrCL is partially torn, and ends up with complete tearing. The more negative factors are present, the earlier the process begins.

One may argue, why some large breeds do not have an increased risk for CrCL ruptures (e.g. Greyhounds, Malamutes). The explanation is rather philosophic than scientific. It is the human being, which creates canine breeds, that were not foreseen to be as large or heavy, as they are forced to. Mother nature would have never allowed such a development and rather would have produced smaller and leaner dogs.

3 Diagnosis

The typical anamnesis includes initial non-weight bearing lameness, clinical improvement after some weeks and clinical signs of degenerative joint disease. Stifle laxity is palpated in upright position and lateral recumbency. A positive cranial drawer sign (Fig. 3) or tibia compression test is diagnostic for CrCL rupture. Increased internal rotation and crepitus are the most common associated findings. Incomplete CrCL ruptures do not always lead to a positive drawer sign or tibia compression test. Eventually, pain in hyperextension or a slight drawer sign in flexion may be elicited. False negative joint laxity results are obtained in dogs with heavy muscle tone or severe degenerative joint disease with capsule fibrosis. In contrast, young dogs normally have some degree of stifle laxity.

Radiographs of the stifle are taken to rule other abnormalities than a CrCL rupture. Signs associated with CrCL may be joint effusion (Fig. 4), cranial displacement of the tibia, bulging of the joint capsule, calcification after meniscal or ligament injuries, and signs of degenerative joint disease. Radiographs of the entire tibia are taken to plan the surgery and to give a prognosis concerning long term outcome. Synovial fluid assessment, scintigraphy and positive contrast arthrography add little information to the diagnosis, whereas MRI is the method of choice in human knee disorders. Partial tears of the CrCL are best diagnosed with arthroscopy or explorative arthrotomy.

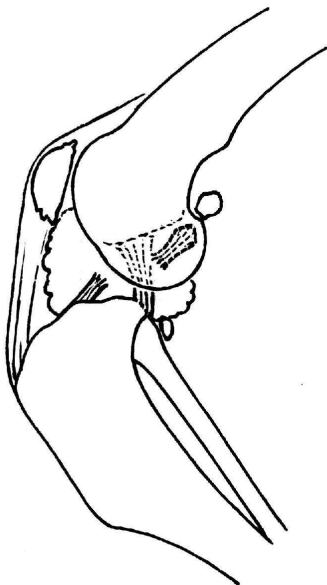


Figure 3: Illustration of the unstable stifle joint, when performing a drawer test or when the dog is walking.



Figure 4: Laterolateral radiograph of a stifle joint with a CrCL rupture. Note the joint effusion.

4 Therapy

As mentioned above, there is a biomechanical failure causing CrCL ruptures. Simple ligament prosthesis, either intra or extraarticular, will by time lead to the same catastrophic situation as it happened, when the original CrCL begun to fail. Therefore, it is strongly recommended to change biomechanics. This can be done either with a Tibia Plateau Leveling Osteotomy (TPLO according to Slocum) or with a Tibial Tuberosity Advancement (TTA). The Tibia Wedge Osteotomy (TWO) and other derived surgery proposals are less advantageous. Biomechanical corrections are performed on heavy dogs, overweight dogs and in bilateral CrCL ruptures. Dogs less than 10 kg bodyweight still can be operated with ligament prosthesis with a good rate of success.

4.1 Extracapsular suture techniques (Flo, combined with Harrison)

The approach can be performed from the medial or the lateral side. We normally approach laterally (Fig. 5). The skin is incised from the laterodistal third of the femur to the middiaphysis of the tibia. The fascia lata is incised along the cranial side of the biceps muscle, then extended to the margo cranialis tibiae. The joint capsule is incised at the same level as the fascia. Care must be taken to not sever the intraarticular tendon of the long digital extensor muscle. The incision is extended to the supratrochlear area. The patella is then luxated medially in extended stifle position und held in place by flexion of the joint. The fat pad is excised and the joint inspected under retraction of the patellar ligament.

A heavy polyester suture is placed in figure 8 configuration extracapsular around the lateral fabella and through a tunnel in the tibial tuberosity close to the proximocranial end of the tibia. One suture is placed in the same manner around the medial fabella and a separate tunnel in the tibia, which adds stability to the reconstruction (Fig. 6). The property of the suture material used leads to a periarticular inflammation and scar tissue formation. In case of suture material failure, the scar tissue should hold the stifle stable. An aponeurotic sling helps to protect the extracapsular repair and prevent meniscal damage in the early postoperative period.

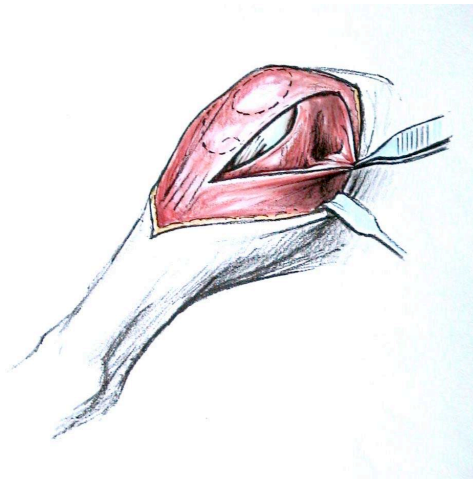


Figure 5: Lateral approach to the stifle joint through a lateral or medial skin incision and lateral incision into the fascia lata and the joint capsule.

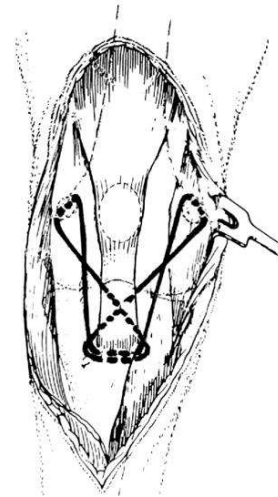


Figure 6: Flo's procedure with extracapsular prosthesis on lateral and medial side

4.2 Tibial Tuberosity Advancement (TTA according to Montavon and Tepic)

The procedure presented consists in advancing the tibial tuberosity, in order to position the patellar ligament perpendicularly to the tibial plateau, thereby reducing the tibiofemoral shear force (CTT) to zero and easing the function of the deficient CrCL. The lesser invasive technique reduces operative time and perioperative morbidity. Respecting the normal range of flexion of the stifle should make a meniscal release, hence a loss of intraarticular caudal support, not necessary. Decreased retropatellar pressure could alleviate the sulcus chondromalacia present in about 30% of the cases. These advantages should improve the short and long-time results of the surgical treatment of cranial cruciate deficient stifle.

Mediolateral radiographs of the stifle in extension, avoiding the cranial drawer phenomenon in the presence of total rupture of the cranial cruciate ligament are necessary to figure out the angle necessary to bring the patellar ligament perpendicularly to the tibial plateau. The patellar ligament is represented by its cranial border, the orientation of the tibial plateau by a line passing through both tibial origins of the cranial and caudal cruciate ligaments.

Arthroscopy or medial arthrotomy is performed in case of total cranial cruciate ligament rupture to explore the stifle joint and treat eventual meniscal lesions. There is still a great debate whether to release the medial meniscus or not. According to in-vitro studies (Thieman, 2010) and knowledge exchange between many orthopedic surgeons performing TTA (Kyon meeting, 2008), a meniscal release should not be performed, when the meniscus is intact. My personal experience is, that a good postoperative care with physiotherapy will prevent further damage to the meniscus. I therefore only release the meniscus in an abaxial position, when a poor owner compliance is expected.

Transverse osteotomy of the tibial tuberosity is carried through its distal extremity to the cranial borders of the menisci. A titanium spacer of desired size (3, 6, 9 or 12 mm) is inserted into the distracted osteotomy in order to advance the tibial tuberosity, giving its new position to the patellar ligament. The tibial tuberosity is fixed

to the tibia with a special plate (titanium plate, 2 to 8 forks) in tension band mode (Fig. 8). The wound is closed in appositional manner after mobilizing the edges in order to cover the implants.

A postoperative bandage is not necessary. Clinical results are satisfactory as documented by force plate gait analysis and many client feed-backs. Return to normal locomotion is straight forward, even in sport or hunting dogs. Recovery time is between 6 weeks and 3 months. Progression of stifle osteoarthritis is strongly slowed down.

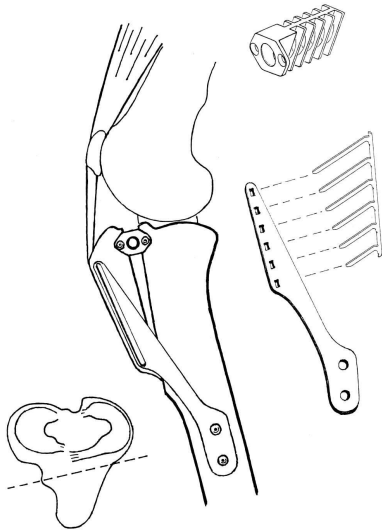


Figure 7: Schematic representation of the TTA and its implants.

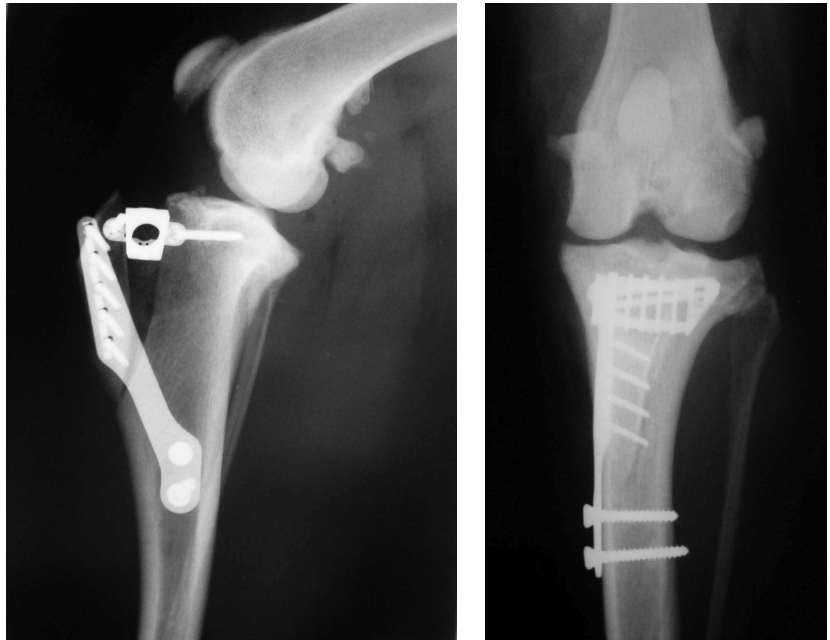


Figure 8: Postoperative radiographs of a 8 year old Labrador Retriever after TTA surgery.

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