

Brace Yourself: The Role of Orthotics in Cruciate Disease
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Cranial cruciate ligament (CCL) rupture is one of the most common orthopedic conditions encountered in the dog. In fact, over 1 billion US dollars are spent every year in dealing with the canine stifle. When dealing with hind limb lameness many dogs we see have some degree of hip dysplasia or degenerative changes in the hip; however, an acute lameness is typically not due to a hip problem. In fact 32% of dogs referred for hip problems actually have evidence of cruciate disease. About 33-50% of dogs will present with bilateral disease even if they have a unilateral lameness. Severe bilateral cruciate disease can often mimic other conditions such as severe hip dysplasia or neurologic disease. Therefore, a general rule of thumb is a hind limb lameness in a dog is cruciate disease until proven otherwise.

Personally for me, statements that I do not like are:

- All dogs that rupture their CCL must have surgery
- All dogs with CCL ruptures have joint effusion
- All surgical procedures (extra-capsular repair, TPLO/CBLO, TTA, XYZ) have the same outcome
- A dog can't return to pre-injury status following a CCL rupture
- Dogs don't benefit from rehabilitation therapy either with a conservative approach or following surgery

Anatomy:

The stifle is considered a complex condylar synovial joint because the articular cartilages are separated by an intra-articular fibrocartilage or the menisci. The primary functions of the stifle are flexion, extension, and rotation. There are lots of structures that work together that make up the anatomy of the stifle such as the femur, tibia, patella, the soft tissue structures, as well as the intra-articular structures. There are 3 bones that make up the stifle. The femur has 3 major articular areas with 2 condyles that are convex, while the proximal tibia has 2 condyles that are concave. The femoral condyles are separated by the intercondylar eminence and also contain the intercondyloid area, which serves as the attachment site of the CCL. The patella is the largest sesamoid bone in the body and articulates with the femoral groove. The patellar ligament is the portion of the quadriceps femoris between the patella and the tibial tuberosity, which is sometimes used interchangeably with patellar tendon. The soft tissue structures of the stifle are the medial and lateral meniscus, which are attached to the proximal tibia by paired meniscotibial ligaments. The primary ligamentous support of the stifle comes from the medial and lateral collateral ligaments as well as the cranial and caudal cruciate ligaments (CdCL). The cruciate ligaments are intra-articular but covered in synovium so they are considered extrasynovial.

The menisci are C-shaped disks of fibrocartilage that act as functional extension of the tibia. They are a true example of a specific structure function relationship. The cranial and caudal meniscal horns are attached to the bone through the cranial and caudal meniscotibial ligaments. There are 4 total: a cranial and caudal for each medial and lateral meniscus. What's important about the anatomy of the meniscus is the difference between the medial and lateral aspects. The medial meniscus is firmly attached to the medial collateral ligament and the joint capsule making it relatively immobile such that its motion is coupled with that of the tibia. On the other hand the lateral meniscus is less firmly attached to the tibia. It also has a menisiofemoral ligament caudally. Its motion is more coupled with the femur and therefore is less likely to be injured compared to the medial meniscus. The meniscus has a wedge shape that causes radial extrusive forces to develop from compressive forces. The primary function of the meniscus is for load bearing, load distribution, shock absorption, and joint stability. Because of its shape it acts as a spacer and bears about 40-70% of the load.

So why does the meniscus matter anyways? As already discussed the meniscus accepts high loads during weight bearing but also absorbs energy. It does this by undergoing elongation as a load is applied. As the joint compresses the wedge shape extrudes peripherally and the circumferentially oriented collagen fibers elongate. This is known as hoop stress. The hoop stress is then transmitted to the tibia. The meniscus also provides a concavity to the convex tibial plateau. Several studies have shown the importance of the meniscus. For example removal of the caudal horn of the medial meniscus leads to a focal area of high pressure in that area. This alteration of the articular cartilage contact may contribute to degenerative changes following a meniscectomy. Furthermore, a meniscal release causes a 140% increase in peak contact pressure and a 50% decrease in contact area.^{1,2}

Physiology:

The primary motion of the stifle in the sagittal plane is flexion and extension while secondary motion is rotation. In Labrador Retrievers the normal range of motion is 41 and 161 degrees of flexion and extension.³ During extension of the stifle the medial and lateral collateral ligaments are taut and therefore act as the primary stabilizers that limit internal and external rotation. During flexion the lateral collateral ligament relaxes while the medial remains somewhat taut. This allows the lateral femoral condyle to displace caudally and results in internal rotation. Then as the joint is extended the lateral collateral tightens up drawing the lateral condyle cranially and resulting in external rotation. In humans this is known as the screw home mechanism. The CCL functions to limit internal rotation, hyperextension, and tibial subluxation. The CCL is made up of two bands: the craniomedial and the caudolateral. The craniomedial band is primarily responsible for preventing the cranial translation of the tibia while the caudolateral band is responsible for secondary prevention of cranial translation of the tibia. The CCL and the CdCL do indeed cross themselves (hence the term cruciate which means to cross) and both the CCL and the CdCL play a partial role in preventing rotation of the stifle.

Pathophysiology:

CCL rupture is typically considered to be degenerative in nature and often bilateral. In fact 33-50% of dogs that present with a unilateral lameness will have bilateral disease. It was first described in 1926 and to this day we still don't know the exact mechanism of action. Proposed mechanisms include immune-mediated conditions, age and time of neutering, confirmation, obesity, lack of fitness, increased TPA, chronic stress, and the list goes on. Purely traumatic ruptures can occur but this is rare. It occurs when supraphysiologic loads are placed on the CCL, which results in a mid-substance "mop end" tear. In the CCL deficient stifle the limb function is altered such that the limb is more flexed throughout the gait cycle most likely as a way to minimize pain and weight bearing on the affected limb. From a kinetic standpoint the peak vertical force (PVF) and vertical impulse (VI) is decreased after a CCL tear. For example in a sound limb the PVF was found to be 70% of the static body weight (BW) of the dog. In the CCL deficient stifle the PVF was 25% at 2 weeks, 32% at 6 weeks and 37% at 12 weeks. Furthermore, tibial subluxation has been noted to be 8-12 mm and even up to 5 mm 2 years after injury. Interestingly there are not really any changes in internal rotation following a CCL rupture. There is evidence of increased meniscal damage and joint capsule fibrosis as well as progression of osteoarthritis (OA). Once the CCL is ruptured the caudal pole of the medial meniscus acts as a wedge preventing the tibia from further subluxation. However, the 2-edged sword aspect of this is that this wedge shape coupled with the anatomy of the medial meniscus also increases the risk of a meniscal tear in the untreated stifle.

Diagnosis:

The diagnosis is typically straightforward and is based off the history, signalment, clinical signs, physical exam, and orthopedic exam. The history may include an acute or chronic hind limb lameness that may be mild to non-weight bearing. Interestingly, owners may report that the lameness has improved from initial injury. This usually corresponds to the timeframe from when the initial inflammatory response is ending. Regarding the signalment any age or breed can be affected. Typically we tend to see medium to large breed dogs that are around 3-8 years of age. The orthopedic exam is mainstay to diagnosing a CCL rupture. Findings may include a positive sit

test where the dog will tend to sit with the affected leg projecting out to the side. Pain on hyperextension is usually the forgotten test but is very reliable. Most affected dogs will exhibit some degree of pain. Crepitus may be noted during ROM, and with chronic tears medial buttress formation may be noted. This is the peri-articular fibrosis that occurs. The classic findings for a CCL rupture are joint effusion, the cranial drawer test and the tibial compression test. A simple way to think about it, is that in an adult dog joint effusion will only be caused by a CCL rupture, septic arthritis, tick-borne disease, or immune-mediated arthritis. A medial patella luxation (MPL) will not cause the same degree of joint effusion, so if you have a patient with underlying MPL that develops joint effusion be thinking about a CCL rupture.

The cranial drawer test is testing for laxity in the CCL, but this is more of a passive test and does not mimic weight bearing. To perform the test one hand is placed on the distal femur with the thumb behind the lateral condyle. The other hand is placed on the proximal tibia with the thumb behind the fabella. The goal is to move the proximal tibia cranially in relation to the femur. Always check drawer in flexion and extension. When checking for partial tears the CCL has two bands, the craniomedial which remains taut in both flexion and extension and the caudolateral, which is taut in extension but lax in flexion. For example if the craniomedial band is torn and the caudolateral band is intact cranial drawer is only present in flexion because in extension the caudolateral band is taut. If the caudolateral band is torn and the craniomedial band is intact no cranial drawer is present because the craniomedial band is taut in both flexion and extension. Cranial tibial thrust is a test meant to mimic active weight bearing. The goal is to hold the stifle at a standing angle (approximately 135 degrees) and while holding the stifle still flex the hock. If the CCL is ruptured there should be a cranial displacement of the tibia. As with cranial drawer, tibial thrust should be checked in both flexion and extension.

Radiographic evaluation will help to see evidence of joint effusion with cranial displacement of the intrapatellar fat pad. With chronic CCL ruptures you may see evidence of OA and if you are lucky the stifle is sitting in drawer on the radiographs. Some people have proposed a stable stifle with joint effusion and a hind limb lameness may be evidence of a partial tear.

Treatment:

When deciding on a treatment plan there is no one treatment fits all, but there are many, many, many options available. The reason there are so many options is because not one procedure or medical management technique is 100% perfect. I think one reason for this is because what is considered our final outcome, a stable stifle, a patient that returns to activity pain free, elimination of OA, owner satisfaction, etc.? We will never be content on cruciate disease until we figure out the goals we want to achieve for an outcome.

When I approach a dog with cruciate disease I'm going to have the same conversation with each owner; however, depending on each case I may swing my conversation in one particular direction. Factors I consider when deciding on conservative vs. surgical treatment and which procedure are the patient, owner, and veterinarian factors. I look at the breed, the size of the animal, the age, the activity level, and what is that particular animal's job. Are they a pet, an athlete, or a service dog? Regarding the owner I talk to them about their perceived outcome, their ability and willingness to follow directions post operatively, as well as finances. And then I look at my abilities such as what equipment I have available, what procedures am I comfortable doing, and what good and bad outcomes have I had with certain procedures.

When I first tell owners that their dog has a torn cruciate I try to cover all options that create a patient specific approach. I fully believe that surgical fixation is the best course of action; however, not every dog is a candidate for surgery and not every owner is going to agree to surgery. For this reason, we have to have options. One option is we do nothing. By do nothing I mean we cage confine for 6-8 weeks with medical management (analgesia and NSAIDs) and (hopefully) formal rehabilitation therapy. The most important aspect here is confinement. These owners have to be aware the goal of conservative management is to allow peri-articular fibrosis to occur. This can't occur with the dog remaining active. To break it down to them I tell the owners

the dog must be kept in an area where he/she can stand up, lie down, and turn around. The dog eats, drinks, and sleeps in the crate. It only goes outside to urinate and defecate on a leash then back into the crate. I also throw the disclaimer in that in my opinion OA is worse with a rapid progression as long as the stifle is unstable and usually if this is a larger dog they won't return to full function. I also really push the fact that the dog will appear to be do "okay"; however, they have a very high chance of developing a meniscal tear. I tend to tell owners it's not "if" but more of a matter of "when" they tear their meniscus. Personally, I am not a fan of this approach! I also, really push formal rehabilitation therapy to try and at least help with development of peri-articular fibrosis, muscle building, and compensatory changes.

Another option is a conservative approach with exercise restriction, formal rehabilitation therapy, and a custom made stifle orthotic. The hope is that in theory we can attempt to help stabilize the stifle with a brace. In human medicine, knee braces are commonly used for multiple conditions. Bracing of the human knee has been shown to enhance proprioception/joint position sense, permit the injured limb to relax, reduce fatigue in injured limb, provides some mechanical protection against impact, and slow movement down to allow muscles time to react and control motion. Categories of knee braces in human medicine include the following: prophylactic (prevent or reduce severity of knee injuries in contact sports), functional (provide stability for unstable knee, rehabilitative (allow protected and controlled motion during the rehabilitation of injured knees), and patellofemoral (improve patellar tracking and relieve anterior pain). Only functional knee braces are utilized in veterinary medicine.

In theory the brace should help limit tibial subluxation. At the authors institution we did find improved objective gait analysis when a custom stifle brace was worn versus when not worn; however, the gait analysis was not improved equal to that of surgery.⁴ This data reveals that a brace is not considered equal to or meant to replace surgery; furthermore, it must be worn for the duration of the pet's life. A recent retrospective study revealed that 46% of dogs placed in a custom stifle orthotic will develop skin lesions and 32% of those require medical care for the lesions that develop. Furthermore, 37% only tolerated the brace "fairly", "did not wear it" or "did not tolerate it". When owners were asked why a stifle orthotic was chosen over surgery the answers were cost, convenience, personal preference, and **veterinarian recommendation**. This further reiterates the need for veterinarians to have a full understanding of what a stifle orthotic can and can't accomplish.⁵

The conversation I have with owners regarding stifle orthotics is as follows:

- 1) Tolerability: I can't ask the patient if he/she will tolerate the brace, I have had some dogs that don't mind it at all, others take time, and some just freeze or try to chew it. The other issue is given the different shapes and sizes of dog stifles the brace **MUST** be custom made. This means a mold must be made and sent to the orthotist and then sent back about 2 weeks later. It's a horrible feeling to have an owner pay the expense for a brace and then the dog won't tolerate it.
- 2) Arthritic progress: What I can tell an owner is that with surgery we can slow down and minimize arthritic progression. Without surgery we will have continued accelerated and worsening progression OA. Along that scale is a brace; I just don't know if the scale is closer to that of surgery or that of no-surgery?
- 3) Meniscal damage: What I can tell an owner is that with surgery we can minimize the chances of a meniscal injury. Without surgery there is a high incidence of meniscal injury. The problem is again along that scale I don't know where a brace will fall. Will it help protect the meniscus the same as surgery, or will it not make a difference such as doing nothing? This does bring up a good point about meniscal damage. A "meniscal click" will only get you about 30-40% correct at identifying a meniscal injury. If you add in a positive McMurray test and pain on hyperflexion that may improve to about 50%. Personally, I feel as if a dog has a meniscal tear they will not benefit from a brace because it will do nothing to help with the pain and discomfort. The problem is if at best you can diagnose a meniscal injury in 50% of patients then how does one approach determining if there is meniscal injury? A MRI could be considered but is costly and requires general anesthesia, arthroscopy could be considered but personally would be below the standard of care to go to surgery to identify a meniscal injury but not treat the CCL rupture. Therefore, if I

have owners that want their dog in a brace then they must undergo a stifle ultrasound. If there is evidence of meniscal damage then that dog will not be a good candidate for a brace, if they don't appear to have meniscal damage then we can give it a shot knowing that an ultrasound is not 100%.

There is also a subset of patients that will undergo surgery, but maybe from a timing standpoint it needs to be delayed. These patients fall into the "pre-hab" category. A stifle orthotic could be considered for these patients; however, from a financial standpoint I would rather have them use the funds for rehabilitation and/or surgery. For example, it may be the morbidly obese dog that needs to lose weight before surgery. It may be the family that is saving up for the TPLO/CBLO/TTA, but wants to do something prior to surgery. Or it can be any other number of reasons. The focus here is education. Client education is critical with regards to many aspects of their pet. For cruciate disease and the resultant secondary osteoarthritis this includes obesity reduction (through diet and exercise), pain mitigation (through NSAIDs and other oral medications), chondroprotection (through oral supplementation of Glucosamine, Chondroitin, Omega-3 FA, etc. Seeing the big picture with these pets is needed to achieve a favorable outcome. Without it, you have a fat, immobile dog in chronic pain. Checking thyroid function on all overweight dogs is recommended. If it is normal, then using a prescription diet tailored for obesity and arthritis is recommended.

The rehabilitation program as part of "pre-hab" or in conjunction with a stifle orthotic begins with first improving comfort (pharmaceuticals, manual therapies, and isometric exercises). Once the patient is comfortable (usually 1-2 weeks post injury) then underwater treadmill walking is initiated. The goal here is to use the buoyancy of the water to reduce stress on the unstable stifle. This will allow for reduction of muscle atrophy and caloric burn without further injuring the patient. A protocol of 10-30 minute walks 2-3x a week (progressively building) is recommended. If possible, a photobiomodulation (therapy laser) treatment protocol is also initiated at these visits, with the goal being to reduce inflammation and promote tissue healing.

An at home program can include short leash walks, but a focus here on therapeutic exercises is used instead. Once initial inflammation is reduced, working on appropriate sit to stand exercises with the stifle in appropriate position is attempted. 10 reps per set, 1-2 sets per day. Initially we aim for 5/10 reps to be square, and then each week increasing our goal by one. The affected stifle should be against a wall, so as to minimize outward rotation. The goal here is fairly quick succession throughout the set. As soon as the patient ischium touches the ground the dog should be asked to stand again and repeat the exercise. Stretching and range of motion can begin at 1-2 weeks post injury, and then continue daily through week 8. The goal here is to maintain range of motion of the joint and minimize contraction,

The walks should be at a slow pace, on leash and on flat surfaces with good traction. The goal is to achieve 20 minutes comfortable walking on level ground twice daily (in the orthotic). Once this is achieved then inclines, declines, and stairs can be added. As the patient continues to improve then straight line trotting is initiated on leash followed by supervised off-leash activity. Around weeks 4-6 as the fibrosis is being achieved, core strengthening with balance disks and wobble boards can be done in a slow safe and professionally administered manner. Return to normal activity in the orthotic is permitted around weeks 8-10 if the patient will tolerate.

Cruciate disease is a complex problem that does not have a clear-cut answer. Ultimately, surgical intervention may be needed in any case that is initially managed without it. Proper education of the client is critical to establishing favorable outcomes.

References:

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