Stifle joint instability is the end-stage of progressive cranial cruciate ligament (CCL) degeneration. The process of progressive CCL degeneration and the associated osteoarthritis (OA), which eventually leads to CCL rupture, is called cruciate disease. Cruciate disease begins with partial tearing of the CCL and associated OA and over time will progress to complete rupture.

CCL disease is a chronic slowly progressive degenerative condition in dogs. The initial symptoms of this disease are subtle being typical of osteoarthritis with stiffness or hind limb lameness after rest that improves as they warm up. These symptoms are slowly progressive over several months. Dogs may present apparently acutely when the degenerating ligament finally ruptures and/or when secondary tearing of the medial meniscus occurs. Evidence of chronic stifle joint change in these cases is evidence that the apparent “acute” deterioration is the final very obvious stage of a chronic insidious disease process.

Cruciate disease in dogs is quite different to anterior cruciate ligament (ACL) rupture in people, which occurs most commonly as a result of sporting trauma, with a typical acute, sport-related onset.

In dogs, once the CCL degeneration has progressed to a point where the ligament is no longer functional, weight bearing causes the tibia to move cranially relative to the femur. This movement is known as cranial tibial thrust. It causes subluxation of the stifle joint when the dog weight bears and will eventually cause meniscal damage.

Osteoarthritis is a fundamental component of cruciate disease in dogs and is present prior to the development of stifle joint instability. OA is thought to be one of the factors that contribute to the progressive degeneration of the CCL in dogs. The progression of OA has been shown to accelerate in unstable joints.

It is important to remember that stifle joint instability (positive cranial drawer sign and positive cranial tibial thrust) are the end-stage of progressive CCL degeneration. Early diagnosis of cruciate disease and early effective surgical intervention before it progresses to cruciate rupture and instability is associated with better clinical outcomes.

This image shows the right stifle joint of a dog with significant OA and a large bucket handle tear (arrowed) of the caudal horn of the medial meniscus which occurred secondary to cruciate instability.

Once stifle joint instability is present in...
dogs the risk of secondary meniscal injury increases by 160 times. Unlike people, where isolated meniscal injury can occur without ACL instability, the occurrence of an isolated or primary meniscal injury without concurrent CCL instability is a rare event.

Due to the chronic slowly progressive nature of cruciate disease in dogs and the associated OA there are symptoms and clinical signs of the problem that are apparent for several months before eventual progression to CCL instability.

**How to improve detection of cruciate disease prior to progression to cruciate rupture**

Detection of dogs with unilateral CCL rupture and consequent instability and/or meniscal injury is easy. They typically present with a “classic” marked unilateral lameness that is referable to the stifle joint with obvious pain on manipulation of the stifle. They may be non-weight-bearing or have a marked weight-bearing lameness.

While the occurrence of cruciate disease bilaterally is very common it is less common for dogs to present with bilateral CCL instability. Depending on the severity and the chronicity of the disease these dogs with bilateral cruciate instability will vary from having a markedly painful weight-bearing lameness to occasionally being non-ambulatory.

Detecting lameness in dogs with early cruciate disease before the development of stifle joint instability is more difficult. Nonetheless “early” detection of cruciate disease before it progresses to stifle joint instability is the goal and today is the standard of care that we should be aiming for.

In early cases of cruciate disease where the CCL remains intact it is not uncommon for no lameness to be apparent on examination once the animal has warmed up. Observing the stance of these dogs will usually simplify detection of lameness. In unilateral cruciate disease the dog will often have a tripod stance where they unload the lame limb and preferentially take more weight on the sound limb. Where cruciate disease is bilateral dogs will also typically shift their center of gravity further forward to take more weight on their forelimbs. This is called a forward press.

This image shows a Rottweiler dog with bilateral cruciate disease that is worse in the left stifle joint displaying both a tripod stance and a forward press.

It is important to note that these compensatory stance changes are not pathognomonic for cruciate disease. While they can occur with other causes of hind limb lameness the fact that cruciate disease is the most common cause of clinically significant hind limb lameness means that dogs presenting with these symptoms should be investigated.
further with cruciate disease one of the main differentials.

Another compensatory change that is highly suggestive of stifle joint pain and so relatively more specific than a tripod stance is a **positive sit test**. Dogs with cruciate disease will commonly avoid sitting squarely with full stifle joint flexion, as this is painful. They will sit unevenly and leave the sore stifle joint slightly extended.

This image shows the Rottweiler in the previous image displaying a positive sit test having rolled partly to the right side to avoid flexing the left stifle joint.

A positive sit test is highly suggestive of stifle joint pain and, as cruciate disease is by far the most common cause of stifle joint problems, means that cruciate disease must be the primary differential.

When performing an orthopaedic examination the standing exam comparing both hind limbs for symmetry will usually reveal signs consistent with chronic degenerative joint disease. This is obviously considerably easier in cases of unilateral disease where the abnormal joint can be directly compared against the contralateral normal joint. Detecting these changes in cases of bilateral cruciate disease requires a good familiarity with normal orthopaedic examination for that particular breed.

These signs of chronic stifle joint disease will vary with chronicity and severity and typically include

- **muscle wasting** of some degree.
- **stifle joint enlargement** – this is a combination of **joint effusion** and **periarticular fibrosis**. Palpation of the patella and patella tendon is less distinct than in the contralateral normal joint. Palpation of joint effusion is most apparent as a firm fluctuant swelling over the lateral condyle immediately adjacent to the patella. It should be noted that radiographic detection of mild joint effusion is more sensitive and reliable than detection on physical examination.
- **medial buttress** is **pathognomonic for cruciate disease** and is apparent as a hard fibrous enlargement of the proximal medial tibia. This image shows a medial buttress on the right proximal tibia of a male St Bernard dog with cruciate disease.
The recumbent part of the orthopaedic examination gives the best opportunity to examine the stifle joint for instability. Remember however that stifle joint instability in the vast majority of cases is the end stage of a chronic progressive disease. Detection of cruciate disease and effective surgical intervention before CCL rupture and instability should be the goal.

The cranial drawer sign is the “gold standard” diagnostic test for cruciate rupture however it is important to remember that this detects an end stage symptom of a chronic progressive disease. Cranial drawer is a test of passive laxity of the stifle joint and so is a “non-physiologic” test.

Testing for cranial tibial thrust can also demonstrate CCL incompetence and stifle joint instability. This is a pseudo-physiologic or “active” test of stifle joint laxity.

To get the maximum diagnostic benefit from testing for stifle joint instability it is necessary to remember that the CCL is comprised of two functional bands. The two bands are not physically separate but function slightly differently. The craniomedial band of the CCL is taut when the joint is both in flexion and in extension. The caudolateral band of the CCL is only taut when the stifle joint is in extension. So where a complete rupture of the CCL exists there will be a cranial drawer sign in both flexion and extension.

In earlier cases of cruciate disease where only a partial rupture of the CCL exists, the presence or absence of a cranial drawer sign depends on which band of the CCL is ruptured. When only the craniomedial band is torn, the caudolateral band is taut during flexion. This means that a cranial drawer sign is only present in flexion. So it is important to ensure that cranial drawer testing is done first with the stifle joint in extension and then again in flexion.

When only the caudolateral band is torn, because the craniomedial band is present in both flexion and in extension, in these cases there is no cranial drawer present. For this reason it is important to understand that cruciate disease can be present with no detectable cranial drawer.

The take home message here is that as clinicians we should not rely on detecting stifle joint instability to make a diagnosis of cruciate disease. Stifle joint instability confirms beyond any doubt the presence of end-stage cruciate disease. However the absence of stifle joint instability does not rule out the presence of cruciate disease. If lameness is present and referable to the stifle joint and there is pain, effusion and medial buttress present then, regardless of whether stifle joint instability is present or not, cruciate disease is the key differential that must be ruled out. Stifle joint exploration to confirm the cruciate disease and effective cruciate surgery is necessary in these cases.