Hip dysplasia (HD) was originally described in 1935 by Gerry Schnelle and has become one of the most common orthopedic conditions that leads to joint inflammation and secondary osteoarthritis. Unfortunately, even after all of this time the exact etiology is unknown but considered to multi-factorial. One such factor involved in the expression of HD is genetics. It is not a simple Mendelian pattern but rather a complex inheritance. This means there are multiple genes that are combined with environmental influences that lead to the clinical expression of HD. Joint laxity is considered the initiating cause of HD which in turn leads to hip subluxation and poor congruence between the femoral head and acetabulum. Multiple causes of hip laxity have been described such as abnormal hip development, biomechanics, genetic influences, increased joint fluid, pelvic muscle mass, nutrition, weight/growth, and hormonal and environmental factors. It’s probably safe to assume that HD and the subsequent arthritis are the clinical manifestation of all of these.

At the very basic level dogs with HD have normal hips at birth. The hips will remain normal if complete congruity is maintained between the femoral head and acetabulum. However, if one or more of the previously discussed factors that leads to hip laxity is present then the dog will manifest as having HD. Laxity is typically defined by the distraction index (DI) which has been shown as a primary risk factor for the development of osteoarthritis (OA). Passive laxity as measured with various radiographic and diagnostic techniques is an estimation of functional laxity which permits hip subluxation. From a hip development standpoint the earliest dysplastic joint changes can be noted as early as 30 days where there is increased volume of the ligament of the head of the femur, and increased synovial fluid volume. The ligament of the femoral head is the primary stabilizer for the hip for the first 30 days of life. For the first 2 weeks of life the ligament is short so if the hip is forced to subluxate the femoral head will fracture at the fovea. After about 2 weeks the ligament will begin to lengthen; in dysplastic dogs this lengthening allows lateral subluxation of the hip. This subluxation allows articular cartilage to become worn and roughened on the dorsal surface of the femoral head at its point of contact with the acetabular rim. The first radiographic signs of HD can be noted at 7 weeks of age where subluxation of the femoral head with under development of the cranio-dorsal acetabular rim may be noted.

From a biomechanics standpoint in a healthy congruent hip the forces are distributed across the entire cartilaginous surface of the acetabulum. The co-contractions of the gluteals, and adductors along with the biceps femoris, semimembranosus and semitendinosus create a force to reduce and stabilize the femoral head into the acetabulum during weight bearing. During the swing phase the primary muscles used to advance the limb are the transarticular muscles of the rectus femoris, sartorius, and ilipsoas, which have long muscle bellies with lines of action more parallel to the axis of the femur. In patients with hip laxity and thus subluxation the transarticular forces must increase to compensate for lateralization of the center of rotation of the joint. Additionally, the cartilage stress is increased because the forces acting on the articular cartilage are spread over a reduced surface area. This ultimately results in two destructive events: forces crossing the joint increase while the area over which the forces are transmitted decreases. What this means is that in patients with HD that the femoral head subluxates during the swing phase of the gate and upon foot strike the larger hip extensors cause catastrophic reduction of the femoral head. Additionally, less muscle mass during development is associated with an increase in joint laxity. There has been a disparity noted between the strength of pelvic muscles and rapid weight gain, which leads to joint instability. Also, muscle mass of dysplastic breeds such as German Shepherds is less than that of non-dysplastic breeds such as Greyhounds.

Nutrition is thought to be a large contributor to joint laxity and thus HD; however, no dietary deficiencies cause HD. Dietary excesses on the other hand can contribute to the development of HD. For example, increased calcium and vitamin D lead to alterations in endochondrial ossification, and delayed bone remodeling. Diets high in excessive vitamin C can lead to hypercalcemia and diets with a high anion gap lead to increased synovial fluid production, which in and of itself has been shown to be a risk factor for hip laxity. Feeding diets to promote rapid growth have been shown to have a higher incidence of HD and also cause early fusion of the acetabular growth plates.

Increased body weight is not a cause of HD, but it certainly has very important clinical consequences in susceptible dogs. Therefore, weight reduction is an effective preventative strategy. In the lifespan study of 49 Labradors it was reported that heavier dogs (dogs allowed to eat ad lib) developed radiographic OA on an average of 6 years earlier than the dogs in the restricted fed group. Furthermore, heavier dogs required long-term treatment for OA on average 3 years earlier than their restricted fed littermates.

The diagnosis of HD is made from the signalment, clinical signs, physical exam findings, and radiographs. Affected dogs are typically large breed fast growing dogs such as German Shepherds, Rottweiler’s, Labradors, or Golden Retrievers. The age of presentation is typically biphasic and contributes to the type of treatment that may be recommended. Juvenile dogs will tend to present between 5-12 months of age with an acute onset of unilateral or bilateral hind limb lameness. These clinical signs are thought to be due to joint laxity. Histologically tearing of the joint capsule along with microfracture of the dorsal acetabular rim is seen. As dogs
become older the long-standing joint laxity causes periarticular fibrosis, which may decrease or lessen the clinical signs. This is why some dogs will tend to have improvement in clinical signs until later in maturity when they present for clinical signs that are consistent with OA.

The severity of clinical signs depends on the stage/severity of the disease. Lameness can be intermittent, progressive, and range from mild to severe. In young patients with severe laxity a “popping” noise may be heard during ambulation. Both young and older patients may exhibit exercise intolerance and difficulty rising from pain and discomfort. Disuse muscle atrophy is a common finding and the gait may be characterized as either “swaying” or hopping. It is very important to remember that a non-weight bearing lameness is rare and thus other problems should be considered such as a cranial cruciate ligament rupture. Orthopedically pain in the hips along with crepitus may be noted. Many of these patients have decreased range of motion in extension and weight shifting to the forelimb. Evidence of joint laxity is determined through the Barlow, Ortolani, and Barden’s test. The Ortolani is performed with the patient in either lateral or dorsal recumbency and sedation is required in most cases. The first part of the ortolani is the Barlow test where a force is directed through the femur through the dorsum to subluxate the hip. The Barlow test is considered a provocative test in that it creates subluxation in a lax hip. The second part of the Ortolani test is the true ortolani maneuver where the limb is abducted and a click or clunk can be heard as reduction of the hip occurs. The clunk is considered a positive ortolani and indicative of coxofemoral laxity. Some surgeons will use the angles measured during an Ortolani test as indications for a triple or double pelvic osteotomy. The Barden’s test is performed with the dog in lateral recumbency; a direct lateral force is applied to the femur without abducting the limb. In the awake dog pressure on the medial thigh can cause discomfort and this should not be mistaken for hip pain. Any movement of the greater trochanter more than ¼ of an inch suggests laxity. Unfortunately, Ortolani and Barden’s only suggest laxity and do not predict later development of clinical signs of OA.

Radiographs are mainstay for the diagnosis of HD along with the characterization of the disease and any presence of OA. There are several ways to evaluate canine hips, which vary from using the hip extended view as what is done with OFA, or developing a distraction index as what is done with PennHip. OFA style radiographs are generally used in daily practice, this involves that the pelvic limbs are fully extended and parallel, the pelvis is symmetrical and the pelvic limbs are internally rotated. Sedation and/or general anesthesia is usually required. Mal-positioned radiographs can lead to false assumptions. The two most notable and early signs with hip OA are the circumferential femoral head osteophyte (CFHO) and the caudo-lateral curvilinear osteophyte (CCO). The CFHO is a white line at the articular margin of the femoral head that may or may not extend completely around the femoral head. It is graded from I to III. The CCO is also sometimes known as a Morgan’s line, it is a well-defined linear density on the femoral neck between the greater trochanter and the capital physis in dogs greater than 18 months of age. It is different from a puppy line in that a puppy line is an indistinct radiodense line on the femoral neck in dogs less than 18 months of age, it is in a similar location to the CCO but it is more subtle, more diffuse and shorter than the CCO. A puppy line is considered self-limiting and is not clinically significant.

One big debate is between the use of OFA and PennHip for HD screening. OFA is a subjective scoring system based on the hip extended view. The problem is the hip extended view is an unnatural position for dogs and can mask subluxation because the view actually forces the femoral head into the acetabulum. It does identify OA and moderate laxity but is not a sensitive method to detect early or mild laxity. PennHip uses stress radiography to detect joint laxity and it can be predictive for the development of OA. It is a measure of hip laxity, not a certification process. A study in 2010 using the OFA database described a 1.5% increase in OFA excellent films, a 3.3% increase in OFA good films, and a 2.1% decrease in OFA fair films. To complicate matters it was found that in dogs with OFA excellent films 52% had DI >0.3 putting them into the OA susceptible range, 82% of dogs with OFA good had DI greater than 0.3, and 94% of dogs with OFA fair had a DI greater than 0.3. In other words the progress of eliminating HD is moving very slow. In fact at the current progress it will take about 44 years to move Labs from a hip score of 10 where is it currently to a hip score of 5, which is equal to an OFA excellent grade.

Treatment for HD can be broken into prevention and/or laxity improvement utilizing the juvenile pubic symphysiodesis (JPS) or triple,double pelvic osteotomy (DPO or TPO). More definitive treatment can be accomplished with medical management, a femoral head and neck ostectomy (FHNO or FHO) or a total hip replacement (THA). In immature dogs that are still growing with no evidence of OA then medical therapy can be attempted. This includes promoting weight loss, daily activity, and formal rehabilitation therapy to improve muscle mass, range of motion, and comfort. Many of these patients benefit from NSAIDS, chondroproactants, and omega-3 fatty acids. For those that are severely clinically affected or have failed medical therapy then either a JPS or DPO/TPO,FHNO, or THA can be considered. In mature dogs medical management is geared towards OA management. Older dogs that become refractory to medical management would then become candidates for either a FHNO, or THA. Regardless early detection is key, in susceptible breeds hip palpation should begin by 12 weeks of age. If they have a positive Ortolani or have a high DI after 16 weeks of age then JPS should be considered in at risk breeds. A JPS is a minimally invasive way to pre-maturely cause fusion of the pubic symphysis. This causes ventro-lateral rotation of the acetabulum with growth of the animal (resulting in ventroversion and improved femoral head coverage). To procedure is completed with a small incision to the pubic symphysis, electrocautery is then used every 2-3 mm along the symphysis at 40 watts for 12-30 seconds. Best results are achieved in patients before 16 weeks of age (20 weeks in giant breeds) resulting in about 10-15 degrees of ventroversion if done at 16 weeks. No real benefit is gained if completed in animals greater than
22-24 weeks of age. The resultant hip changes are similar to what is seen with a DPO/TPO; however, it is easier and faster with fewer complications and no implants are needed.

A DPO/TPO involves osteotomies of the ischium (only with TPO, not with DPO), pubis, and ilium. It causes reorientation of the acetabulum to increase dorsal coverage of the femoral head (thus resulting in ventroversion). In theory there should be improved joint stability and congruence and hopefully reduction in the formation of OA. A DPO/TPO is reserved for patients that have no evidence of OA. The age restriction has been discussed as being less than 10 months of age (typically 6-9 months is ideal); however, I have performed TPOs in dogs 12-14 months of age with success if there is no OA present. It is said to result in about 92% improvement in lameness and slower progression of OA. However, the complication rate approaches near 50% and includes excessive narrowing of the pelvic canal, temporary constipation, sciatic nerve injury, implant failure, screw loosening, and continued OA development.

A FHNO has typically been reserved for smaller dogs and cats; however, larger dogs can also be candidates. It involves removal of the entire femoral head and neck and relies on the formation of a pseudoarthrosis. Even though owner satisfaction is high it is a salvage procedure with 62-65% return to normal function from a gait analysis standpoint. Probably the biggest complication with a FHNO is leaving femoral neck behind, other complications include shortening of the limb, patellar luxation, muscle atrophy, limited hip extension, recurrent lameness and chronic pain. In my hospital patients are required to undergo formal rehabilitation therapy beginning 3-5 days after surgery and continuing for 6-12 weeks.

A THA or “hip replacement” is considered by most to be the gold standard treatment for severe HD that is refractory to medical management. In the past it has been reserved for larger dogs; however, it can now be completed in smaller dogs and even cats. A THA results in about 95% return to function from a gait analysis standpoint. Often unilateral THA is enough to provide adequate function in bilateral disease. The complication is less than 10% but this is very dependent on surgeon ability. Contraindications for a THA are local or systemic infection, neoplasia, concurrent cruciate disease, or neurologic dysfunction. Potential complications include aseptic loosening, implant failure, infection, femur fracture, coxofemoral luxation, and sciatic nerve damage.

In summary, HD has a complex pathophysiology with the predominant feature being joint laxity. There are many factors that contribute to joint laxity. Clinical signs will vary depending on the stage of disease, but remember an older dog that is acutely non-weight bearing will often times have a cruciate rupture with underlying HD. A thorough physical examination with good quality radiographs is needed. Early detection is key so that way a JPS can be performed.

References
Developmental orthopedic diseases (DOD) are those, which are identified during postnatal skeletal growth. The common DOD discussed here are hypertrophic osteodystrophy (HOD), panosteitis, craniomandibular osteopathy, retained ulnar cartilaginous cores, Legg-Calvé-Perthes disease, and osteochondrosis ostearthrosis. While elbow dysplasia is a type of DOD disease, given its complex nature it is beyond the scope of this proceeding to try and describe elbow dysplasia.

**Hypertrophic osteodystrophy (HOD)**

A DOD in young, rapidly growing dogs also referred to as metaphyseal osteopathy, skeletal scurvy, juvenile scurvy, infantile scurvy, Moller Barlow’s disease, and osteodystrophy II. The incidence is roughly 2.8/100,000 with patients presenting between 2 and 6 months of age. HOD is predominantly seen in large and giant breed dogs. Great Danes, Chesapeake Bay Retrievers, Irish Setters, Boxers, German Shepherd, Golden Retrievers, Labrador Retrievers, and Weimaraners are at an increased risk with males 2.3 times more likely to develop HOD than females.

There are numerous proposed causes such as vitamin C deficiency, over nutrition, heritability, infections, and vaccines; however, no single cause has been determined. More recent studies have refuted the vitamin C and over nutrition theories. Heritability has been suggested for at risk breeds and has been shown in a family of Weimaraners. Infection is thought because many of the patients have a history of systemic illness with the addition of leukocytosis, although in most studies an infectious process has not been identified. One study did document an association with HOD and canine distemper virus; however, a large multi-institutional study did not support a link between HOD and canine distemper.

Diagnosis of HOD is based on signalment, history, clinical signs, and radiographs. The distal radius, ulna, and tibia are the most commonly affected bones. Clinical signs include swelling of the metaphyseal region of the bone and the lesions are often bilateral. The swelling may be warm upon palpation with varying degrees of pain and lameness (from mildly lame to a having a reluctance to want to walk. Some patients may exhibit systemic signs of illness such as hyperthermia, depression, inappetence, anorexia, and diarrhea. The pathognomonic radiographic sign is a lucent line in the metaphysis parallel to a narrow zone of increased radiodensity just adjacent to the physis (the so called “double physeal line”). There can be varying degrees of periosteal and endosteal proliferation. Differential diagnosis includes secondary nutritional hyperparathyroidism, septic polyarthritis, retained cartilaginous cores, and hypertrophic osteopathy.

In the majority of cases HOD is self-limiting within days to weeks, but can persist for months. The prognosis is typically good to excellent; however, in very severe cases death has been reported. For mild cases, analgesics along with a balanced diet should be provided. In more severe cases supportive treatment may be needed especially if the patient is reluctant to eat. Furthermore, owners should be warned about the possibly of angular limb deformity in severe cases. In Weimaraners specifically with severe HOD without bacteremia may respond better to corticosteroids than to NSAIDS.

**Panosteitis**

Panosteitis is a self-limiting inflammatory disease of the bone marrow of long bones. It is sometimes referred to as enostosis, eosinophilic panosteitis, and shifting leg lameness with an incidence of about 2.6/1000 patients. Breeds that are at an increased risk or are predisposed breeds include: Airedale Terriers, German Shorthair Pointers, Doberman Pinschers, Afghans, Great Danes, Saint Bernard’s, Bernese Mountain Dogs, Newfoundland’s, Mastiffs, Bassett Hounds, Rottweiler’s, Cocker Spaniels, Golden Retrievers, Labrador Retrievers, and German Shepherds. Given the list of pre-disposed breeds panosteitis is primarily a disease of large to giant breed dogs; however, it has been reported in small breed dogs. Age at presentation is typically between 5–12 months of age but up to 2 years of age has been noted. Males are affected more than females with a ratio of 4:1.

Histologically, the first changes noted consist of empty spaces in the adipose bone marrow, vascular proliferation with local bone formation around the nutrient foramen. These changes are thought to lead to vascular congestion and secondary increases in intraosseous pressure. The exact origin and etiology is still unknown.

Much like HOD the diagnosis is derived from the signalment, history, and clinical signs along with radiographs. The hallmark clinical sign is a shifting leg lameness with pain on palpation of affected long bones. The degree of the lameness and pain can be variable from mild to inability to walk. Typically the owner will report an acute lameness with no history of trauma. The most commonly affected bone is the ulna (42%), followed by the radius (25%), humerus (14%), femur (11%), and tibia (8%). Radiographs are helpful to differentiate panosteitis from other conditions (such as HOD, OCD, etc.). The appearance on radiographs will depend on the stage of the disease. In the early stage of disease radiographs may be normal or have a decrease in radiodensity in the medullary cavity near the nutrient foramen. As the disease progresses, the increase in medullary opacity will develop a granular pattern with loss.
of normal trabecular pattern. Other findings include periosteal bone formation. After 4-6 weeks the densities will regress leaving a trabecular pattern that is coarser than normal.

Treatment consists of rest and analgesics; however, treatment does not influence the outcome. Recurrence is possible, but the severity will decrease over time as the dog matures.

**Cranio-mandibular osteopathy**

Cranio-mandibular osteopathy is also known as cranio-mandibular osteodystrophy, cranio-mandibular osteoarthropathy, mandibular periostitis, lion jaw, Westie jaw, and Scotty jaw with an incidence reported as 1.4/100,000 cases. There is no reported sex predisposition; however, puppies less than 6 months of age are at the highest risk. The incidence is reported to decrease with age.

West Highland White Terriers and Scottish Terriers are at an increased risk; other breeds reported to be affected are Boxers, Labrador Retrievers, Great Danes, Boston Terriers, Great Danes, and German Shepherd’s. Given the strong breed predisposition in West Highland White Terriers, a heritable etiology has been suggested, and an autosomal recessive mode of inheritance has been demonstrated in this breed.

The disease is characterized by either unilateral or bilateral symmetric irregular osseous proliferations of mainly the mandible, but the tympanic bullae can also be affected. Osteoclastic resorption of lamellar bone occurs, which is followed by the presence of primitive bone that will expand beyond the periosteum. The bone is replaced by a fibrous-type stroma and inflammatory cells invade the border of the lesion destroying adjacent connective tissue and muscle.

Clinical signs will vary from minor difficulty eating and chewing to complete inability to open the mouth and thus the inability to eat or drink. Because of the lack of nutrition additional clinical signs include weight loss, salivation, depression, and pain while eating. Physical examination will reveal enlargement or irregularity of the mandibles. The patient’s mouth may be able to be opened only partially or not at all, and the teeth are unaffected. Often times blood work and urinalysis are unremarkable. Radiographs will demonstrate bony proliferation of the mandible and/or bullae. CT can be useful to identify lesions and to better delineate the areas affected.

It can be self limiting when the dog is 11-13 months of age; however, patients may need varying levels of supportive care such as feeding soft food/gruel, syringe feeding, or placing a feeding tube. Nutrition and hydration are key parameters to monitor. Given the pain involved with trying to open or close the mouth analgesics are indicated. Over time the bony proliferations will regress completely or partially. Surgical excision of the proliferation is not recommended due to the recurrence within 3 weeks. Rostral hemimandibulectomy has been reported for a severe case that facilitated lapping of food.2 Euthanasia has been performed for patients with uncontrollable discomfort or with lesions that do not resolve and affect quality of life.

**Retained ulnar cartilaginous cores**

Also known as retained endochondrial cartilage cores, this is a cone of growth plate cartilage that projects from the distal ulnar growth plate into the distal metaphysis. Histologically, the retained cartilaginous core consists of viable hypertrophic chondrocytes. It is essentially failure of the growth plate cartilage to convert to metaphyseal bone, while some consider it a growth plate manifestation of osteochondrosis. Like most DOD it occurs predominately in large to giant breed dogs.

If the retained ulnar cartilaginous core is associated with reduced ulnar length then changes similar to premature closure of the distal ulnar growth plate are noted. These changes consist of cranial bowing of the radius, external rotation, and valgus deviation of the paw; additional subluxation of the carpus and elbow may be seen.

Radiographs are mainstay for the diagnosis, where a radiolucent core (typically triangle in shape) of cartilage is noted in the distal ulnar metaphysis. There may be an area of sclerosis surrounding the area. The core may extend up to 3-4 mm into the metaphysis. This must be differentiated from premature closure of the distal ulnar growth plate. No correlation has been noted between the size of the lesion, histopathology and severity of deformity.

Treatment is based off the degree of deformity. If no deformity is noted then no treatment is recommended, and the core may disappear spontaneously. In these cases it is recommended to closely monitor patients for the development of deformities, especially of the carpus and elbow. In cases with moderate to marked deformity the surgical correction of the deformity may be required.

**Leg-Calve perthes disease**

Avascular necrosis of the femoral head is noted as a noninflammatory localized ischemia of the femoral head and neck, which results in deformation of the femoral head and neck leading to a pelvic limb lameness. It has also been referred to as aseptic necrosis of the femoral head, coxa plana, osteochondrosis, and osteochondritis coxae juvenilis. Small breed dogs are the most commonly affected with toy breeds and Terriers predisposed. Initially, the disease is histologically characterized by necrosis of the trabeculae of the femoral head, followed next by the fragmentation phase where loading of the affected hip causes collapse of the epiphysis and secondary thickening and cleft formation of the articular surface.
Osteochondrosis/osteochondritis dessecans (OCD)

In short OCD is a disturbance of endochondrial ossification. There is failure of the cartilage matrix calcification and vascular ingrowth, which results in cartilage retention. The cartilage retention results in thickening of the articular epiphysal cartilage. There are two distinct areas of osteochondrosis: the growth plate-epiphyseal complex (GEC), and the articular-epiphyseal complex (AEC). Proposed causes of osteochondrosis include genetics (especially with large and giant breed dogs), rapid growth, calcium supplementation, hormonal influences, ischemia and trauma. The incidence of AEC osteochondrosis is 8.1/1000 patients with male dogs more affected than female dogs with a typical age at presentation of 4-9 months.

Proposed mechanisms for the pathogenesis of AEC osteochondrosis is it is a result of generalized disease; however, this does not adequately address the species and site-specific nature of osteochondrosis. The other proposed mechanism is it starts as a focal disease from vascular trauma and subsequent necrosis of the subchondral bone or necrosis of the epiphyseal cartilage canals. This necrosis may possibly lead to cartilage ischemia and necrosis. The necrosis may occur at a development stage when the vessels from the perichondrium are being replaced by vessels from the adjacent epiphyseal bone marrow. As this is occurring the vessels are susceptible to damage by conformational forces and/or microtrauma. If the vessels become damaged and thus necrotic then a cartilaginous infarct develops, which prevents endochondrial ossification.

Osteochondrosis latens is used to describe the initial cartilage necrosis, at this stage the disease process can be resolved or progress to osteochondrosis manifesta where larger areas of necrotic cartilage resists vascular invasion. This will then persist during growth and can be detectable. If the overlying articular cartilage fissures or fractures (thus developing a flap) then the commonly known osteochondrosis/osteochondritis disseccans (OCD) lesion develops.

The most commonly affected joint is the shoulder, followed by the elbow, tarsus, and stifle. The caudocentral or caudomedial aspect of the shoulder is affected the most commonly and it is bilateral in 27-68% patients, while the lesions of the medial aspect of the humeral condyle are commonly areas affected in the humerus; 96% of lesions in the stifle affect the medial femoral condyle, and in the talus the medial or lateral trochlear ridge is affected.

Clinical signs associated with osteochondrosis typically become apparent when a cartilage flap develops. One theory is the motion between the flap and the subchondral bone, or the altered loading may provoke pain. If the flap detaches it can become what is known as a “joint mouse”, which may further contribute to synovitis and OA progression. Patients tend to present from 4-9 months of age with a complaint of lameness or exercise intolerance. Many cases will show signs of a unilateral lameness; however, the disease is commonly noted bilaterally so careful examination of the contralateral joint is warranted. Once a patient presents with clinical signs of a lameness the OCD lesion is considered chronic and a defect in the subchondral bone is well developed. Radiographs are a sensitive diagnostic method that may demonstrate disruption of the subchondral bone with flattening or concavity of the normal contour. Sclerotic margins may be seen around the defect. Contrast arthrograms can be used to demonstrate unmineralized cartilage flaps and joint mice, effusion, and new bone formation. CT is also useful in demonstrating an OCD lesion. Arthroscopy is useful as both a diagnostic and therapeutic modality.

Aims of treatment need to include elimination of pain and lameness, restoration of the cartilage surface with tissue of similar nature to the native tissue, normalization of joint biomechanics, and prevention of further joint degeneration. Conservative management may be recommended with small subchondral lesions and when the patient is mildly lame or asymptomatic. This form of treatment is only recommended for dogs younger than 6 months of age. Conservative management consists of NSAIDS, exercise restriction, chondroproctants, rehabilitation therapy, and weight control. Persistence of clinical signs suggests the patient should be treated with a surgical approach.

Surgical management consists of either an arthrotomy or arthroscopy (the authors preferred method). Surgical treatment consists of flap excision and joint mouse removal. Additional treatment may consist of removing peripheral cartilage that is not firmly adhered and stimulation of fibrocartilage to the underlying subchondral bed.

OCD of the shoulder usually carries a good to excellent prognosis; however, other joints affected with OCD carry a guarded prognosis with continued progression of OA and an intermittent lameness.

References
Bone is essentially the frame that supports locomotion. It’s an amazing tissue with complex properties that are a series of lever arms that act to counteract the forces of gravity while constraining and directing the forces of muscle. In general bone follows Wolff’s law in that it adapts to loads under which it is placed. Essentially, bone is shaped for the greatest strength while at the same time minimizing bone mass that would contribute to excessive weight. Bone is considered both viscoelastic and anisotropic. Viscoelastic implies that the strength of bone depends on the rate upon which it is loaded such that a bone is stronger when loaded rapidly versus slowly. Technically, bone becomes stiffer the more rapidly it is loaded; however, if the rate of loading exceeds the yield point a bone will fracture. The anisotropic property of bone says that its strength is dependent on the direction in which it is loaded, and thus bone is stronger when loaded longitudinally versus transversely. This makes sense in that while a patient is walking bone is loaded longitudinally; however, in many cases bone fractures occur due to a transverse load.

Bone in general is subjected to many forces. A fracture occurs when the sum of the forces is greater than the ultimate strength of the bone. The 5 main forces that bone is subjected to and thus must be overcome are tension, torsion, bending, shearing, and compression. Tensile forces are a type of axial force that acts to lengthen the bone while compressive forces are a type of axial force that acts to shorten the bone. The anisotropic nature of bone suggests that it is stronger when loaded in compression versus tension. Shearing forces are difficult to conceptualize with respect to bone; however, it is a common force present within bone. Shearing forces acts parallel or tangential to the bone. Torsion acts to twist bone about its long axis. This creates a shear stress in the bone (where tension and compression are seen in oblique planes). Bending forces (also referred to as moments) makes bone convex on one side and concave on the other side. The convex side is undergoing tensile forces while the concave side is undergoing compressive forces. Understanding the forces that act on bone are important as these are the very forces that must be overcome when choosing the appropriate fracture fixation method.

Fractures occur when the sum of the forces to the bone are greater than the ultimate strength of the bone. This can occur due to trauma and the force exceeds that of normal bone, or it can occur pathologically when the bone is weakened and therefore the force does not have to be as great to allow a fracture (abnormal bone). In a load deformation curve when bone is loaded there will be slight deformation. As long as the load remains in the elastic region then failure will not occur and the shape of the bone will revert back to normal. However, if the load continues past a certain point known as the yield point then bone will cross over to the plastic region, which will result in permanent deformation. If the load continues the breaking point then the bone will fracture.

Once a fracture occurs, the goal is to allow the bone to heal with restoration of normal function with acceptable cosmetics. There are certain factors that must be taken into consideration for a bone to heal such as the biologic factors (blood supply, location of the fracture, and concurrent soft tissue injuries) and the mechanical factors (such as the degree of stability at the fracture site). The afferent blood supply to the bone is supplied through the nutrient artery, where the blood flow is centrifugal in that it progresses from the medullary cavity to the periosteum. Therefore, blood flow is from the nutrient artery to the metaphyseal arteries, and then the periosteal arteries. After a fracture the medullary circulation is disrupted, therefore we get an enhancement of existing normal blood supply. Temporarily, there is a transient extraosseous supply from the soft tissues. It is very important to preserve this blood supply and be kind to the tissues during surgery. As the bone heals the medullary circulation is reestablished. From a mechanical standpoint the fixation must counteract the forces acting on the bone while preserving the blood supply. Healing will also depend on the fracture gap and the stability.

Bone healing parallels that of most other tissue in the body such as soft tissues. It will progress through the typical inflammatory, reparative, and remodeling phases. For bone to adequately heal there has to be a stable environment in that the interfraagmentary strain is <2%. This is the deformation occurring at the fracture site relative to the size of the gap, which influences the type of tissue that will form in the gap. Secondary bone healing is considered the normal course of bone healing and is how all bones healed prior to the advent of open reduction and internal fixation (ORIF). Essentially this occurs through callus formation by progressively stiffer tissue as bone healing moves through the various phases. Initially when the bone is fractured a hematoma develops. This hematoma provides no strength but is very important in that it releases lots of growth factors. The next stage is the formation of granulation tissue, which adds very slight strength. After the formation of granulation tissue, connective tissue develops followed by cartilage formation, cartilage mineralization, and finally woven bone formation. With ORIF primary bone healing can occur which allows “skipping” of the initial secondary phases. For this to occur once again the interfraagmentary strain has to be <2% and the interfraagmentary gap must be <1 mm. Thus, even with ORIF if the bone ends are not touching it will proceed through secondary bone healing, but in a quicker time since the fracture will be stable. The 2 types of primary bone healing are gap and contact bone healing. Gap bone healing occurs when the gap is <1 mm. Granulation tissue forms first with its blood supply, then lamellar bone follows
without the cartilage phase. Initially, the lamellar bone is oriented transverse to the to long axis of the bone (think like “caulk” filling in the gap). Haversian remodeling allows new lamellar bone to be oriented longitudinally. Contact bone healing occurs when the fracture fragments are in direct contact and there is no interfragmentary motion. There is no lamellar phase, but rather Haversian remodeling occurs directly by bridging the fracture with longitudinally oriented osteons known as cutting cones.

Many bones are fractured as a result of trauma. Always remember that if there is enough force to cause a bone to break then there is certainly enough force to cause soft tissue damage. It’s important to triage these patients by checking and stabilizing vitals (such as treating hypovolemic shock). A thorough physical/orthopedic exam and a neurologic exam are needed. It does no good to repair a femoral or pelvic fracture if the sciatic nerve is transected. Baseline diagnostics including chest and abdominal radiographs, TFAST, and AFAST should be completed. Any life-threatening issues need to be addressed which may mean delaying surgery. *I love fixing fractures, but I want the patient to live more.* Fractures in and of themselves are not emergencies. Analgesia is imperative as fractured bones hurt, and thus the pain will lead to a systemic cascade so controlling this is important. Pure mu opioids are recommended such as morphine, hydromorphone, or fentanyl. Unfortunately, butorphanol does not typically provide adequate analgesia. Once the patient is stable then go back and obtain a thorough history; it is important to separate traumatic from pathologic fractures. Evaluate PE/Ortho/Neuro findings. Common signs of fractures include pain, swelling, reluctance to bear weight, crepitus, or angulation deformities. And as previously mentioned if the patient is non-ambulatory it is very important to evaluate for neurologic deficits such as with the radial or sciatic nerves.

The traditional AO classification system I have found to be confusing and not many people classify fractures based on this. Each bone has a number, then there are 3 zones, and finally the fracture is classified into the morphology and severity. More commonly fractures are classified by the anatomical location, severity, configuration, displacement, contamination, and if they are a growth plate fracture or not.

Fractures can be classified by the anatomical location such that they are articular which requires complete anatomical reconstruction with rigid internal fixation, epiphyseal, physeal (which have their own Salter Harris classification), metaphyseal, or diaphyseal. Furthermore, in particular areas special terms can be used such as condylar (as seen with distal femoral or distal humeral fractures), supracondylar (meaning above the condylar region), trochanteric (as seen around the greater trochanter), or subtrochanteric. The severity is described as incomplete meaning the fracture is only through one cortex (sometimes called a “greenstick” fracture in immature patients). There is a small fissure noted but the fracture is not complete. A complete fracture involves a fracture through both cortices. Also, please note that the term “compound fracture” is not used to describe any fracture in either human or veterinary medicine. A comminuted fracture is one with multiple fragments. A segmental fracture is one with two or more separate fractures of the same bone. Avulsion fractures are classified as an enthesis fracture, which is one that occurs at the attachment of a joint capsule, or an apophysis fracture, which is one that occurs at the origin or insertion of a tendon or ligament. The configuration of a fracture can be transverse in that it is perpendicular to the axis of the bone and the fracture equals the diameter of the bone. Or the configuration can be considered oblique. A short oblique fracture is one where the fracture is less than two times the diameter of the bone versus a long oblique where the fracture is greater than two times the diameter of the bone. A spiral fracture is a long oblique with a twist. The displacement is based on the degree of displacement of the distal segment in relation to the proximal segment. You have to have orthogonal radiographs to describe this. One can’t simply have only a lateral or only an AP, but must have both. The degree of contamination is used to classify open fractures. Type I open fractures are those with <1 cm puncture wounds where the fragment briefly penetrated the skin. A type II open fracture is one where there is >1 cm puncture wound with evidence of external trauma. A type III open fracture has extensive wounds with significant soft tissue damaged or absent. It is further subclassified into IIIa where there is adequate skin to close the wound, IIIb where there insufficient skin to close (aka degloving injuries), or IIIc where there is compromised vascular supply to the skin.

If you are presented with an open fracture cover it immediately. When any open fracture arrives in our hospital I cover it as soon as they come in the door with a sterile covering. This can be as simple as a sterile Huck towel with vet-wrap around it. Trust me, the bacteria in your hospital will be much worse then the environmental bacteria the bone may have come in contact with. Once the dog is stable then remove your dressing and flush the wound with lots of fluid. In severely contaminated wounds I have used tap water, but typically will use either saline or p-lyte. I’m not a fan of combing iodine or chlorhexidine to my flush solutions because if you are not measuring out the specific concentrations correctly you could be killing viable cells. Once I have flushed and debrided the area then I will cover the wound with a more stable covering. We then have to make the decision about fixing the fracture as well as addressing the wound and dealing with any evidence of infection.

Physseal fractures are classified by the Salter Harris (SH) classification scheme. SH I fractures are through the physis itself, while SH II fractures are through physis and into metaphysis. SH III fractures are through physis and into the epiphysis and are considered intra-articular. SH IV fractures are through physis and into metaphysis and epiphysis as well as being considered intra-articular. SH V are compression fractures though the physis, while SH VI are compression fractures though only a portion of the physis, which results in angulation deformities.
To aid in ease of communication amongst veterinarians we need to list the bone involved (remember left or right), the location, configuration, displacement, and contamination if present. This will allow the veterinarian or surgeon on the receiving end to create a visual image of the fracture to begin to decide on how best to fix the fracture. Radiographs are mainstay for diagnosing fractures. However, one must take orthogonal views to determine and evaluate the extent of the fracture. This includes at least a lateral and AP radiographs to tell the whole story. CT scans can be helpful especially with sacral fractures, spinal fractures, and articular fractures.

In summary to be able to fix a fracture requires one to be able to correctly diagnosis and classify a fracture. Remembering bone characteristics, biomechanics, and healing all play in decision making for fracture fixation. In terms of classification it is important to describe the fracture with the anatomical location, severity, configuration, displacement, contamination, and if and what type of growth plate fracture may be present. The biggest piece of advice with fracture diagnosis is to take orthogonal radiographs to create the full picture.
In part I we discussed the classification and diagnosis of fractures as well as basic bone healing. Once an understanding of the appropriate classification of fractures is understood then it is important to understand the approach and selection of fixation. Unfortunately, there is no “orthopedic cookbook” in regards to selecting the appropriate fixation for a fracture. Each fracture needs to be addressed to the individual patient by different factors.

One such factor is the patient. Issues such as size, is this a big dog or a little dog? The age, as younger dogs may heal quicker and may require implant removal versus older dogs, which may take longer to heal and thus need a more robust type of fixation. Activity level certainly plays a role, as a less active dog may not require as robust of a fixation versus a dog that is very active or is a canine athlete. Client factors play a role, as they are the ones making decision. I always find it nice to give them options, as finances will play a role in what they are able to do versus what they are not able to do. I will also never commit to a certain type of fixation as my plan may change intra-operatively so I will go over all the available options that may be possible so that if something needs to change in surgery there are no surprises for the owner. Their compliance will play a big role in my selection of fixation. For example if the dog is aggressive or the owner is unable to care for an external fixator then a bone plate with screws may be a better option. The fracture itself as discussed in part I of this series is certainly a factor. The configuration will dictate what type of fixation can be used, for example an IM pin and cerclage wire is not the best option for a transverse fracture. Remember the 5 forces that need to be counteracted with fixation. The degree of contamination will dictate as well what type of fixation may be best. For example a severely contaminated fracture may be better suited for an external fixator rather than bone plates and screws. Another large factor is of course your own ability. Having the understanding of biomechanics and healing as discussed in part I is very important. Knowledge of particular implants will help decide what type of implant will be best suited for that particular type of fracture. Experience and skill level should be considered. Always ask yourself “can I fix this fracture, and should I fix this fracture” Meaning if you have experience and skill to fix it along with available implants. “Should I fix this fracture” means if you don’t have the experience should you refer it rather than attempting the unknown. Implant availability will play a role as far as what you have in your clinic to repair a fracture. It is helpful if you do lots of fractures that you have different types of implants available, as an IM pin and cerclage fixation is not an option for every fracture. If you don’t do many fractures then know the limitations of the implants you have.

In the past fractures were approached from the “carpenter” standpoint, which means absolute anatomic reconstruction with rigid internal fixation. This will disrupt the fracture hematoma and blood flow and requires significant tissue dissection. This type of approach is needed for articular fractures and for fractures that require anatomical reconstruction. Recently, a more biologic friendly way to fix fractures has been described at the “gardener” (biologic osteosynthesis) standpoint. This approach uses minimal reconstruction and rigidity to preserve blood flow. This is accomplished by indirect fracture reduction through limited approaches such as the “open but do not touch” method meaning the fracture area is approached but no manipulation of the fracture is performed or a minimally invasive plate osteosynthesis (MIPO) approach. This is accomplished through a few stab incisions and everything is done in a closed manner. When approaching these fractures there should be minimal to no disturbance of the fracture hematoma. Bridging osteosynthesis rather than rigid fixation is typically elected with limited reliance on secondary implants such as k-wires, cerclage wires, etc. In a perfect world we need to try to find the balance between the carpenter and the gardener. The fixation needs to be something that stabilizes the fracture to allow bone healing but that it is not too rigid to delay bone healing. The fixation should preserve the blood supply to the fracture and not disrupt the fracture hematoma. Furthermore, of extreme importance is to maintain joint alignment and allow early return to function.

After consideration has been given to the various factors, we then need to consider the individual factors of the various implants themselves. I have a chart that I run through in my head (see below) for every fracture I am presented with. As I run through this chart I begin to go through the pros and cons of each type of fixation until I decide on the one or two best options for that particular patient.


<table>
<thead>
<tr>
<th>INVASIVENESS &amp; STABILITY</th>
<th>PRIMARY FIXATION</th>
<th>ANCILLARY FIXATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Least Invasive Most unstable</td>
<td>External Fixation</td>
<td>Lag screws</td>
</tr>
<tr>
<td>Internal Fixation</td>
<td>External coaptation (cast or splint)</td>
<td></td>
</tr>
<tr>
<td>IM pin and/or k-wires</td>
<td>Lag screws</td>
<td></td>
</tr>
<tr>
<td>External skeletal fixator (ESF)</td>
<td>Full cerclage</td>
<td></td>
</tr>
<tr>
<td>Interlocking nail (ILN)</td>
<td>Hemi cerclage</td>
<td></td>
</tr>
<tr>
<td>Bone plate and screws</td>
<td></td>
<td></td>
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</tbody>
</table>

Note: Even though ESF and ILN are listed above bone plates and screws, the last 3 primary fixation methods offer complete stability depending on the fracture configuration; however, in general, ESF and ILN are less invasive than bone plates and screws.
**External coaptation**

External Coaptation is defined as the use of bandages, splints, casts, etc. to aid in the stability and support for soft and osseous tissues. It is useful for the management of wounds, edema reduction, and fracture management. The central theme for any patient with external coaptation is comfort and function. External coaptation for fracture management can be as the primary fixation, temporary fixation, and ancillary fixation. For primary fixation the bandage and splint will be the sole means of fixation where as with temporary fixation the bandage/splint may be used to cover open fractures or stabilize a fracture until definitive surgical correction. Ancillary fixation with external coaptation is useful for additional support such as after bone plating to prevent implant breakdown as seen with distal radius and ulna fractures.

To utilize external coaptation for primary fixation one must be able to stabilize the fracture a joint above and below the fracture. This leaves only fractures that are distal to the elbow or stifle amendable for external coaptation. Thomas-Schroeder splints are never recommended. These are traction devices that are constructed of a wire frame and soft bandage material. The splint does not adequately immobilize the shoulder or hip, and, therefore, is considered contraindicated in humeral and femoral fractures. The advantages external coaptation for fracture management are that it does not disturb the fracture site, there is minimal risk of contamination, no risk of implant break down, it is easy to apply and there may be a decreased expense. However, there are associated costs for frequent rechecks and bandage changes which tend to add up very quickly especially if complications develop. The disadvantages for using external coaptation for primary fixation are that it is limited to fractures that are distal to the stifle or elbow. External coaptation wont counteract all the forces on a fracture and there is the possible need for eventual surgery if the fracture fails to heal. If there is fracture instability this could result in delayed, nonunions, or malunions. Frequent bandage changes will be needed which will require cost and frequent visits by the owner. Furthermore limb stiffness can occur from prolonged immobilization, which can lead to disuse atrophy and fracture disease. Probably the biggest reason I really don’t like bandages and splints is just by placing a bandage there is a 63% morbidity associated with it, which will lead to costs to the owner and potential delays in fracture healing.

When determining if external coaptation is an option certain patient factors need to be considered. For example ideal candidates for external coaptation are younger animals with green stick fractures. These are fractures that are often incomplete, minimally or non-displaced and have an intact fibula or ulna which will increase stability. Breed and confirmation are very important as chondrodystrophic breeds and obese patients can be challenging to incorporate an appropriate splint. Patients that have suffered poly-trauma may not be the best candidates, as they may need internal fixation to promote early limb use. Concurrent diseases need to be considered, as immuno-suppressed patients may take longer to heal and thus require longer immobilization. Temperament needs to be considered such as aggression. If the patient is going to require multiple bandage changes then this may prove challenging. Another consideration is patient assessment. Breed is a consideration such that small and toy breed dogs with radial/ulnar fractures are not a good candidates for external coaptation. The blood supply when compared to large breed dogs is decreased and there is a higher risk of complications. In fact in small breed dogs with radius and ulna fractures treated with external coaptation are at an 83% chance of malalignment or nonunion. Another consideration when choosing external coaptation, as a primary means of fixation is fracture assessment. External coaptation will counteract bending and rotational forces as long as the joint above and below are immobilized. The goal with external coaptation is once the splint is applied orthogonal radiographs need to be taken to assess the fracture. As has been mentioned take orthogonal radiographs after splint application to evaluate the reduction and alignment.

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External coaptation when used as a temporary means of fixation can help improve comfort and reduced swelling while the patient awaits definitive repair. It can also act as a protective covering with open fractures. No matter how clean your hospital is, hospital bugs are much worse than what the bone was exposed to in the environment. I tend to use extreme caution placing a temporary splint on humeral and femoral fractures, as it is very hard to fully stabilize it. Furthermore, the splint can act as a fulcrum and cause worsening pain or malignment of the fracture. I tend to keep these in a crate with analgesic relief while awaiting fixation. External coaptation for ancillary fixation is designed to add additional support. I tend to use this with splint management following radius/ulna fractures. Along with additional support it will also help minimize cycling of the implants to help prevent premature breakdown.

The quick and dirty technique for external coaptation is to sedate or anesthetize the patient for fracture reduction and alignment. I prefer to use custom made fiberglass splints rather than preformed plastic ones as I feel that the comfort is better improved if the splint follows the patient’s anatomy. The splint is typically applied to the lateral aspect for the hind limb and either the lateral or palmar aspect for the front limb. Remember to splint a joint above and below the fracture. It’s important to provide enough padding to prevent pressure sores and movement. Leave the middle two digits exposed. To encourage weight bearing and to minimize trauma to the articular cartilage from prolonged immobilization it is important to splint them in a functional standing angle. As has been previous mentioned take orthogonal radiographs after splint application to evaluate the reduction and alignment.

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In terms of radiographic healing ideally we will consider it completely healed with disappearance of the fracture line. Technically we want 3 of the 4 possible cortices to show evidence of bridging before returning the patient to normal activity. Ideally, within about 5-7 days there will be slight widening of the fracture gap; this is normal as the bone tries to maintain interfragmentary strain. There may be some evidence of “smudging” of the fracture edges. Around 10-12 days we can see the early stages of a bony callus beginning to form. In some cases around the 30-day mark there is the beginnings of disappearance of the fracture line, then around 90 days there is complete healing and remodeling of the callus. This is the time point when many can return to normal activity.

Complications with any type of fracture fixation or implant as much as they suck, happen to everyone, even the best. The big 3 are infection, implant failure, and poor bone healing. The easiest thing to do is blame someone else. Don’t be quick to blame the owner, the dog, or the particular plate. Many times the reason for the issue is standing right in front of you if you were to look in a mirror.

Infection can occur due to hematogenous spread, direct inoculation from an open fracture or surgical contamination or less commonly direct spread from a focal soft tissue infection. In the acute phase after a bone has fractured the vascular channels are comprised which results in ischemia. Bone ischemia is a major predisposing factor for osteomyelitis. Around the bone ischemia is a reactive hyperemia that is associated with an increase in osteoclast production. Along with increased osteoclast production there is also periosteal irritation that leads to periosteal reaction. The aggressiveness of the infection is noted to parallel that of the periosteal reaction seen on radiographs. The damage and ultimately the response to treatment of bony infections are dependent on the viability and stability of the fixation, the virulence and antibiotic sensitivity of the organism and the condition of the soft tissue envelope. The most common type of osteomyelitis is from direct inoculation, which is also known as post-traumatic osteomyelitis. The staph species dominate with S. intermedius being the most common. Some gram-negative bacteria can be associated with osteomyelitis. Fungal organisms are usually due to hematogenous spread. For an infection to occur the bacteria must contaminate and colonize the bone and surrounding tissues. It's important to note that a stable fracture will heal in the face of infection, an unstable fracture will not heal in the face of infection and will perpetuate the persistence of infection.

Poor bone healing is broken into delayed union, non-union, or malunion. Delayed union is healing of a bone that takes longer than expected to heal. The normal healing time frame for a bone is 8-12 weeks versus a nonunion, which is where the bone fails to heal regardless of healing time or if a delayed union is not addressed. A malunion is characterized by a healed fracture in an improper alignment. This may be noted as shortening of a limb, malalignment of the joint surfaces, rotational abnormalities, or varus and valgus deformities. Nonunions are further broken down into viable and nonviable. Viable nonunions can be classified as hypertrophic where there is considerable callus formation but no bone healing, this is sometimes referred to as an “elephants foot”, or it can be moderately hypertrophic where there is a lesser degree of callus known as a “horses foot”. Both types of hypertrophic viable nonunions are typically caused from motion in the fracture site; therefore more rigid fixation is needed. An oligotrophic viable nonunion is hard to distinguish from a nonviable nonunion due the fact there is no radiographic evidence of healing. Its cause is due to lack of cellular activity which are typically due to loose implants in the area of the fracture such as loose cerclage wires. Nonviable nonunions can be classified as dystrophic where there is nonviable bone on either side of the fracture, necrotic where there is an infected section of bone such as a sequestrum, defect where there is a gap at the fracture site that is too large to heal, or atrophic where there is removal of dead bone by the host with no healing and often times resorption of the bone.

References
The decision in choosing internal versus external fixation is dictated by many factors that have been discussed in parts I and II. After considering the various factors such as the fracture configuration/classification, patient, client, and veterinarian factors then remember to run through the below chart in deciding on external fixation (as discussed in part II) versus internal fixation. If internal fixation is chosen then the decision has to be made as to which type.


Internal fixation is ideal for allowing early return to function, maintenance of joint function, and counteracting all the forces on the fracture. It is especially important for fractures that are subject to compression, shearing, and tension as well as comminuted or long oblique fractures. Also, if a fracture could be treated with external coaptation can’t be reduced appropriately by following the 50/50 rule then open reduction and internal fixation (ORIF) should take place. Remember the 50/50 rule states that if 50% of the fracture ends must be in contact for fracture healing to be possible not probable. So the long skinny on internal fixation is to hold the fragments rigidly until they are healed while allowing the patient to move the limb and bear weight. The health of the limb, and the joints surrounding the fracture, is optimized when the muscles continue to function and joint motion is able to maintain cartilage nutrition. Bone will heal; however, maintaining joint function is paramount so don’t get tunnel vision on just the fracture.

However, the advantages of internal fixation over external fixation don’t go without some considerations. First it requires a surgical approach, which results in increased tissue damage. Fragment manipulation may prolong healing. Years ago where the carpenter approach was used there was significant fragment manipulation, which affected healing. With newer approaches and considering the gardener approach there is less fragment manipulation and thus we see quicker tissue healing. The implants do remain inside the body and thus can potentiate infection. Patients with implants are at risk for a surgical site infection (SSI) up to 1 year after surgery, and some may even develop infections longer than that. The advantages and disadvantages of the various approaches must be balanced to optimize an individual patient’s fracture, so remember there is no “orthopedic cookbook” of one size fits all.

When considering the types of internal fixation to utilize, several factors need to be considered. We have discussed various factors when approaching fractures and many of those factors still play a role when deciding on the type of internal fixation. We need to consider the fracture classification (think part I of this series), along with the bone affected, any concurrent injuries, and the forces acting on the bone. Furthermore, several important questions need to be asked: can we reconstruct the shape of the bone, if so then can the bone share the load of the implant, or will the implant have to bear the load of the weight?

**IM pin/Cerclage wire**

Intramedullary Pins (aka IM Pins) are typically smooth round 316L stainless steel rods. Steinmann pins are most commonly used and they are available from 1/6 to 1/4 of an inch in diameter. They come in either trocar or chisel points and the end may have threads. Note that the threads do not improve holding and in fact have a tendency to fail prematurely because the thread shaft interface creates a point of weakness. K-wires appear just as Steinmann pins but their size if much smaller ranging from 0.035-0.062 inches in diameter. IM pins do a great job of resisting bending forces, but are very poor at resisting rotational forces. They can be used as a primary or ancillary fixation with external skeletal fixation or bone plating. They should never be used alone as the only source of fixation. If using an IM pin for primary fixation then cerclage wires should be added. Cerclage wires are designed to counteract axial and rotational forces while providing interfragmentary compression. Cerclage wires do not damage the blood supply or interfere with healing unless they become loose. Cerclage is French for encircling hoops placed around wooden barrels. It is essentially orthopedic wire that is placed around bone. There are various sizes available from 22 gauge, which is used for small dogs and cats up to 18 gauge, which is used in large dogs. In general cerclage wire is used in conjunction with other fixation methods especially with IM pins. It
should only be used alone in non-weight bearing bones such as the mandible. Cerclage wire when used appropriately does not damage the blood supply. The major blood supply is the medullary artery, which is not suppressed by the wires; furthermore, the periosteal arterioles are not blocked by cerclage wires. The wires do not block venous efflux as they have very minimal contact with the bone. Also, cerclage wires don’t interfere with healing unless they become loose or have been applied inappropriately.

The advantages of IM pin and cerclage wire fixation are maintenance of axial alignment with resistance to bending and rotation. In some cases there may be less cost to the client and it is technically easier than some of the other internal fixation methods. The disadvantages as with any internal fixation are it requires an open surgical approach, as previously mentioned there is a high likelihood of failure if the IM pin is used without cerclage wire. There is limited control of axial loads. Also, there is a risk of pin migration or implant breakdown.

IM pin and cerclage wire fixation should only be considered and used with long oblique or spiral fractures of the humerus, femur, or tibia. The IM pin should be placed into the medullary cavity of long bones using either a drill or Jacobs chuck. It can be placed either normograde or retrograde. If using and IM pin for primary fixation the goal is to use an IM pin that is 70% of the medullary canal. If using the IM pin for ancillary fixation for example with a bone plate then the goal is to use an IM pin that fills about 35-45% of the medullary canal. Normograde IM pin insertion involves reducing the fracture first and starting the pin at one end of the bone. The IM pin is then driven across the fracture line and the pin is seated into the opposite bone segment. This is the preferred method in the femur and tibia to avoid injury to the sciatic nerve or penetrating the joint. Retrograde IM pin insertion involves starting the pin at the fracture site, driving the pin out one of the fracture fragments, reducing the fracture, then driving the pin back across the fracture and seating it into the opposite segment. When using cerclage wires a minimum of 2 should be used. They should be placed approximately 1 cm apart beginning at least 0.5 cm from the beginning and end of the fracture line. It is very important that the cerclage wire be placed perpendicular to the bone; otherwise they will not become tight. The cerclage wires must be tight to provide adequate interfragmentary compression. When placing cerclage wire don’t entrap soft tissue. In conical shaped bone such as the tibia a notch in the bone can be placed or a k-wire can be used to create a hemi-cerclage wire. As you twist pull the wire away from the bone, which will create equal tension to ensure an interlocking twist. Cutting the twist cerclage in and of itself will reduce the wire tension by about 21%, be sure to leave at least 3 twists. After cutting the cerclage wire don’t bend it over, this will reduce the wire tension by 70% and set up a scenario for wire loosening.

When using IM pin and cerclage wire we want to avoid the articular surfaces; when using this fixation in the tibia we also want to avoid damaging the insertion site of the cranial cruciate ligament. Never place an IM pin into the radius; there is a high likelihood of joint penetration into the carpus or articular surface of the radial head. Also, avoid stack pinning (placing multiple small pins to replace one large pin) as this does not provide any additional rotational support. Complications of IM pin and cerclage wire fixation are improper placement where damage to the joints or soft tissues occur, specifically sciatic entrapment. There can be implant failure or migration. If there is abnormal rubbing of the soft tissues then a seroma can form. Continued instability of the fracture site will lead to delayed or non-unions of the fracture site. Improper case selection is also consideration when complications arise. Placing an IM pin and cerclage wire for a transverse, short oblique, or radial fractures is a guaranteed bet for a complication. Other complications such as using undersized wire use of a single wire along with technical errors such as insufficiently tightening or bending over wires. Ensure wires are placed perpendicular to the bone. Inadequate fracture reduction will also increase chances of implant loosening and complications. Failure to check all wires is very important as many times once a second wire is placed it will interfere with healing unless it becomes loose or have been applied inappropriately.

External skeletal fixation (ESF)

ESF involves transcutaneous placement of threaded pins or wires into bone, which are secured by clamps, rods, rings, or epoxy. Linear ESF uses transfixation pins or K-wires that are attached to a linear connecting bar using a clamp. Pins can be placed in one of two ways, either half pins or full pins. Half pins penetrate the skin on one side but go through both cortices of the bone while full pins penetrate the skin on both sides and go through both cortices of the bone. The classification for linear ESF is described as Type I through Type III with a few subtypes. The strength of the ESF is stronger as the types increase. A type Ia is considered unilateral and uniplaner with 1 connecting bar and a half pin. A type Ib is considered unilateral but biplanar such that two type Ia ESF’s are 90 degrees to each other. A type Iia is considered bilateral and uniplaner consisting of 2 connecting bars and full pins. This differs from a type Iib in that there are 2 connecting bars but a combination of full and half pins. A type III ESF is the strongest of the linear ESF in that it is a combination of type I and type II. It is considered bilateral and biplanar consisting of 3 connecting bars and a mix of full and half pins.

ESF is diverse in its use in that it can be applied in a variety of fracture scenarios. It is especially useful in patients with open contaminated fractures. It will allow for concurrent wound management, it keeps implants away from the fracture site, and the implants are removed after the bone has healed thus allowing resolution of infection. Furthermore, ESF is able to counteract all the forces acting on a fracture. They can be used as either primary or ancillary stabilization. It can be used with an IM pin to help control rotation.
The advantages of ESF are that it may be applied in a closed manner thus avoiding disrupting the fracture hematoma and soft tissues. It is good for most fracture configurations being that it is versatile and well tolerated. They are able to be destabilized over time to allow the healing bone to adapt. ESF are relatively easy to apply and remove. There are numerous configurations and types so if you like to construct things then ESF is for you. The disadvantages are pin loosening that happens very frequently, pin tract sepsis, limited function of soft tissues if the pins are passed through large muscle groups. The biggest issue with ESF is the postoperative care of the frame, which will require work on the owner’s part.

When using ESF the goal is to use pins that are about 25-30% of the bone diameter. The pins are inserted through stab incisions and should penetrate both the cis and trans cortex. The minimum to have a stable ESF is to use at least 2 pins above and below the fracture. My mentors always told me that 3 is better and 4 is the best. When inserting the pins try to avoid the fracture line, nerves, and vessels. Furthermore, minimize muscle penetration to cut down on post-operative drainage. When inserting the pins use low speed power insertion to reduce thermal necrosis. Using power insertion will reduce the wobble versus when inserted by hand. Thermal necrosis is a huge contributor to pin loosening. Once finished bandage the frame postoperatively to prevent it from damaging the owners home. Scrub sponges can be used to help with bandaging and they will have some chlorhexidine residue on them. They can also be placed in the freezer and used as post op cold packs around the frame. Don’t use smooth or negative profile pins with ESF. If you must use smooth pins they should be inserted at a 70-degree angle to improve holding power. Negative profile pins have a weakness in the pin at the thread pin interface since the threads are cut into the shaft of the pin, thus the diameter of the threaded region is smaller than the diameter of the rest of the pin. I try to use positive profile pins with ESF. These pins have the threads rolled onto the diameter of the pin, such that the diameter of the pin in the threaded region is the same as the rest of the pin.

**Bone plate and screws**

In the simplest terms using a bone plate and screws involves securing a bone plate to the bone via screws. Depending on the plate type the material may be stainless steel, titanium, etc. There are two big categories of bone plates: locking or non-locking. Bone plates and screws resist all forces a bone undergoes, but is weakest in regards to bending. Because of this the plate is placed on the tension surface of the bone. Non-locking plates allow the plate to be held in close contact with the bone by the screws. Once the patient begins to walk the axial load through the bone creates a shearing force at the screw-bone interface. This shearing force is counter-acted by friction generated at the plate-bone interface. Therefore, for non-locking plates to provide the best stability they must be contoured and applied directly to the bone with no soft-tissue in-between. Locking plates are considered a fixed angle system and behave more like an ESF. It does not rely on the friction between the bone and plate. Rather, the axial force the patient creates when walking is converted to and creates a compressive force at the screw-bone interface. In theory, locking plates are stronger and stiffer. Also, they do not have to be contoured to the bone. Up to 2 mm offset is considered acceptable which may improve biologic osteosynthesis.

Bone plates can be used in 3 primary ways. A neutralization plate is such that the fracture can be anatomically reconstructed and the bone/implant will share the load of the weight. Bridging or buttress plating is where the fracture can’t be anatomically reconstructed and the bone does not share the load, the implant must withstand the forces. Bridging and buttress are commonly used interchangeably; however, buttress plating is technically reserved for metaphyseal fractures to keep articular surfaces from collapsing while bridging plating is reserved for diaphyseal fractures that can’t be reconstructed. Compression plating is used with specific plates called DCP or LCP plates. Compression plating can be used when the fracture can be anatomically reconstructed to enhance stability.

The two basic screws are cortical and cancellous. As the name implies the screw is designed for the type of bone the screw is placed in. Cortical screws have a smaller pitch and less depth to the thread, which help with engaging the dense cortical bone. Cancellous screws have a larger pitch and more depth to the thread, which are designed to engage the spongy cancellous bone. Furthermore, screws can be self-tapping, or non self-tapping. Self-tapping screws have cutting flutes on them, which cut the thread into the bone and are designed to speed insertion of a screw. While nice to have, they do have less overall surface area of the bone-screw interface so the flutes have to be driven at least 2 mm past the trans cortex. Non self-tapping screws must have the threads cut into the bone prior to inserting the screw. Furthermore, the screw diameter used should be about 25% of the bone diameter. Ideally, one must engage at least 5 cortices on either side of the fracture but try to shoot for 6. Exceptions to this rule can be made on rare occasions such as with young dogs, ilial fractures, or with use of locking plates.

The advantages of bone plates and screws are that they counteract all forces, typically allow early return to function, they can be placed in a minimally invasive way and they come in a variety of sizes and configurations so that most fractures are amenable to bone plating. The disadvantages are that it requires open fixation, there is the risk of infection and thus implant removal. Furthermore, there can be implant breakdown, as well as the cost of the implants and the cost to the owner.
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, 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 to 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 convex. The femoral condyles are separated by the intercondylar eminence and also contain the intercondylar 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 meniscofemoral 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.

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. 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 cranialmedial and the caudolateral. The cranio-medial 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 patellar luxation (MPL) will not cause the same degree of joint effusion, so if you have a patient will 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 cranio-medial which remains taut in both flexion and extension and the caudolateral, which is taut in extension but lax in flexion. For example if the cranio-medial 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 cranio-medial band is intact no cranial drawer is present because the cranio-medial 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 animals 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 3 main options. Option 1 is we do nothing. By do nothing I mean we cage confine for 6 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. To break this down 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 wont 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 its not “if” but more of a matter of “when” they tear their meniscus. Personally, I am not a fan of this approach!

Option 2 is a conservative approach with exercise restriction, formal rehabilitation therapy, and a custom made stifle orthotic. While this approach parallels that of option 1, we can in theory 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 (unpublished data) 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. 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.

My issues with stifle orthotics are as follows

1) Tolerability: I cant 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 progression: 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%.

Option 3 is surgery with various means such as an extracapsular technique, tibial plateau leveling osteotomy (TPLO), tibial tuberosity advancement (TTA), etc., etc., etc. Granted I’m a surgeon, but option 3 to me is still the best option if I have a patient that can tolerate surgery. For me I prefer the TPLO. At our institution following a TPLO our patients have about a 96-98% return to pre-injury status. Granted owners may want to avoid surgery; however, with a TPLO and formal rehabilitation therapy these patients should be back to normal activity in about 12-16 weeks time.
References