Osteoarthritis (OA) is a chronic, progressive disease that affects both dogs and cats. It has been noted that up to 20% of adult dogs and 60% of adult cats have radiographic evidence of OA.1,2 Owners, themselves are becoming increasingly aware that bone and joint problems are and issue with their pet. Much of this increased awareness has come through the use of the Internet and social media. The overall outcome of osteoarthritis is centered on destruction of the articular cartilage and breakdown of the joint. Because of this OA must be thought of as a global disease process rather than an isolated disease entity. There is considerable cross talk among the tissues that make up a joint. For this reason the joint must be thought of as an organ and the final pathway of OA is organ failure of the joint.

OA primarily affects diarthrodial joints. A diarthrodial joint is composed of the joint capsule, synovial lining, articular cartilage, and the surrounding muscles, ligaments, tendons, and bone. The joint capsule is composed of two layers: the outer fibrous layer and the inner subsynovial layer. Both layers have a rich blood and nerve supply. One explanation of pain associated with OA is distention of the joint capsule due to joint effusion. The synovial lining covers ever structure in the joint except for the cartilage/menisci. It provides a low friction lining and is responsible for the production of synovial fluid. Two major cell populations are present in the synovial lining: type A synoviocytes and type B synoviocytes. Type A synoviocytes are macrophage-like cells that are responsible for phagocytosis. The type B synoviocytes have a more fibroblastic-like appearance and are responsible for producing hyaluronic acid (HA) and other enzymes.

Articular cartilage forms a smooth, compressible surface, which has the primary function of transmitting compressive forces onto the underlying subchondral bone. It is important to note that articular cartilage lacks blood flow, lymphatics and nerves. In fact the oxygen tension in articular cartilage is about 6-7% thus making chondrocytes survive in a hypoxic environment. Cellular destruction and apoptosis has been noted if the oxygen concentration drops below 1-2%. Because the lack of blood flow, articular cartilage must receive its nutrition from the synovial fluid. Histologically, articular cartilage is made up mainly of water (about 80%); a smaller portion is composed of the extracellular matrix (around 10-15%), and chondrocytes (around 5-10%). The chondrocytes are the only metabolically active component of articular cartilage, and are responsible for the production of the extracellular matrix (ECM). The ECM is composed of the proteoglycan matrix and type II collagen.

At the very basic level the proteoglycan matrix is composed of an aggrecan. An aggrecan is a core protein with glycosaminoglycans attached (GAGs) such as chondroitin 5 or 6 sulfate. Multiple aggrecan’s are then attached to a hyaluronic acid backbone (produced from type B synoviocytes) to form an aggrecan aggregate. These aggrecan aggregates have a gel-like consistency and are responsible for resisting compression. The proteoglycan matrix is contained within a type II collagen framework. This lattice type framework gives added support by forming interlocking loops. Having the knowledge of phenotypic make up of cartilage is important when evaluating in-vitro studies. When type II collagen from articular cartilage is taken from an in-vivo condition and placed in-vitro it tends to change its phenotypic expression and begins to produce type I collagen. Many studies utilizing OA models for various treatment modalities are in-vitro. It is very important when evaluating these studies that the study authors have proven that the collagen in-vitro has the same phenotypic expression as collagen in-vivo.

Mature cartilage is classified into 3 un-mineralized zones. Zone 1 (superficial or tangential zone) contains the highest concentration of chondrocytes. These chondrocytes are small, flat, and oriented with the long axis parallel to the joint surface. During joint compression such as with weight bearing the chondrocytes in this zone undergo tension parallel to the joint surface. Chondrocytes in Zone 2 (transitional zone) begin to become larger and more rounded. As the zones become deeper the long axis moves from being more parallel to the joint surface to becoming more perpendicular to the joint surface to compensate for the shearing and compressive forces that predominate in this area as the load on the joint increases. Chondrocytes in the deeper zone 3 (radial zone) contain larger chondrocytes that are predominately oriented perpendicular to the joint surface. This zone forms a rigid mesh and can be partially mineralized. The primary force in this area is compression. The tidemark marks the completion of cartilage maturity as it transitions to the underlying subchondral bone. In a nutshell, mature cartilage forms a pre-stressed, wear-resistant protective diaphragm in zone 1 that helps withstand tension in the plane of the articular cartilage. The middle and deeper layers have the fibrils becoming more organized perpendicular to the plane of the cartilage to withstand compressive loading.

The physiology of cartilage is important because damage to chondrocytes will not only lead to death of that particular chondrocyte but also an inflammatory response that creates problems with neighboring chondrocytes. Thus a downward, progressive spiral occurs which leads to destruction of the “work-horse” (chondrocytes) and loss of extracellular matrix production. The loss of ECM production leads to the loss of cartilage’s ability to soften and transfer loads to the underlying subchondral bone.
The pathophysiology of OA is described as a non-infectious disorder of diarthrodial joints. It is categorized by deterioration of articular cartilage, bone formation at synovial margins (osteophytes), peri-articular fibrosis, and a localized inflammatory response. For OA to develop there has to be some insult to the articular cartilage such as hip dysplasia, a cranial cruciate ligament tear, elbow dysplasia, or an articular fracture. Once the chondrocyte is damaged the inflammatory cascade begins and is followed by the release of multiple cytokines. The two main cytokines involved with OA are interleukin 1 beta (IL-1β) and tumor necrosis factor alpha (TNF-α). IL-1β is responsible for the breakdown of the matrix, while TNF-α drives the inflammatory response. Furthermore, prostaglandins are released, particular prostaglandin E2 (PGE2), which increases the release of metalloproteinases (MMPs). MMPs are responsible for the continued breakdown of the ECM.

This brings into mind one fundamental question; if inflammation is such a key driving force with OA then why don’t anti-inflammatories slow down or eliminate the problem? The answer is there must be one piece of the puzzle missing to the pathophysiology. One such puzzle piece that has been investigated is the concept of oxidative stress. Oxidative stress is an early event during the evolution of many diseases. It occurs when reactive oxygen species (ROS) outweigh the antioxidants. On a daily basis cells under go oxidative stress to help with normal cellular metabolism, which is kept in balance by the cells natural antioxidants. If at any point the ROS outweigh the antioxidants through either excessive ROS production or antioxidant depletion then oxidative stress will occur. This manifest itself as lipid peroxidation, protein, DNA or RNA oxidation. What has been shown in human medicine is that the effects of oxidative stress on chondrocytes along with MMPs are the 2 main mediators in matrix degradation.3 Furthermore, inducible nitric oxide synthase expression can occur by a single stimulation of IL-1β or TNF-α.4 This concept has also been proven in the canine where it was shown that the same inflammatory mediators that cause an inflammatory response also cause an oxidative stress response. Furthermore, oxidative stress to the cell causes a reduction to the cells natural antioxidants. With treatment of certain antioxidants the cells natural antioxidants are able to recovery and thus minimize the oxidative stress response.5

It is important to remember that OA is usually always secondary to some other disease process so it is important to eliminate the problem if one is able. Technically, any problem that involves or disrupts the joint will lead to OA. Because of this it is important to make owners aware. For example, it is common to explain to owners why their dog may rupture its cranial cruciate ligament and what treatment options exist. Its equally important to also make the owner aware that no matter what treatment option is elected the patient will still possibly develop some degree of OA.

Owners will typically complain about their pets have a reluctance to exercise, stiffness, lameness, inability to jump, or even some behavioral changes. Remember that cats are not small dogs, and they can have fewer signs. The biggest complaint from owners with cats suffering from OA is a reduction in activity, reluctance to jump, an unkempt appearance, and aggression. Orthopedically, dogs may show disuse muscle atrophy (ensure to rule out any neurogenic atrophy), a reduced range of motion, pain or discomfort on range of motion, crepitus, and joint effusion. Cats can be tricky to examine so allowing them performance tests is encouraged to see how the cat moves and interacts with its environment. One true test is to place the cat on exam table with its carrier below. Most cats will easily jump from the exam table to their carrier. Any reluctance to want to do so raises concern about possible joint pain.

Radiographs are key to aiding in the diagnosis of OA. However, just as with any diagnostic modality there are limitations. Radiographs only provide bony information, they are taken in a non-weight bearing position, and osteophytes are useful to diagnose OA but they are not pathognomonic for OA. Furthermore, the value of osteophytosis for staging OA is controversial and does not correlate with OA progression. Probably the biggest issue with radiographs is that they do not correlate with clinical signs. The radiographic key features of OA are: osteophytosis, enthesophytosis, effusion, soft tissue swelling, subchondral sclerosis, intra-articular mineralization (especially in cats), and subchondral cyst (rarely seen).

Other additional diagnostic modalities include CT, MRI, and arthroscopy. Arthroscopy is probably the most valuable means to objectively evaluate the cartilage. However, it is a surgical procedure and can be costly to perform. It does allow the evaluation of the cartilage, which can then be classified by the Modified Outerbridge score. One looming question is if you don’t perform arthroscopy and radiographs are helpful to diagnose but don’t help stage for monitoring for progression of OA is there some type of subjective based assessment? The answer is yes, the Canine Orthopedic Index (COI) was developed and validated in 2014 to provide reliable assessment of dogs with OA in terms of staging as well as response to treatment. It can be downloaded at www.canineorthopedicindex.com.

In regards to treatment there must be a multimodal approach. There is no “cook book” or one size fits all treatment plan. Treatment must be patient centered and patient specific. What works for one dog may not work for another dog. Furthermore, patients may respond initially to a treatment plan then become less responsive. In these cases the treatment plan has to be changed. In some cases it can really be trial and error. When I approach OA patients I break it into 1 of 2 categories. Am I seeing a patient that has a primary problem and has or will develop OA (such as cranial cruciate ligament rupture) or Am I seeing a patient that had a primary problem and now suffers from OA (the typical “OA consult”).

For those patients that have a primary problem and either have OA or will develop OA I give owners clear expectations for the future. If I can correct the primary problem such as fixing an articular fracture, or addressing a ruptured cruciate ligament then that is recommended. Following surgery I give owners my 4 pillars of OA management: Joint supplements, Omega-3 fatty acids (150-175
mg/kg of DHA/EPA), daily exercise and weight management. Furthermore, formal rehabilitation therapy is key for post-operative patients. Hopefully, once the primary problem is corrected and following these 4 pillars, nothing more will need to be done.

For patients that had a primary problem that either was or was not addressed but now they suffer from OA I will initially recommend joint supplements, omega-3 fatty acids, Adequan, weight management, and daily exercise. Furthermore, if the patient is having a flare-up then I will recommend formal rehabilitation the control the inflammatory response, improve range of motion and improve comfort. I only like to initially use NSAIDS at the lowest possible dose as infrequently as possible. Unfortunately, many of these patients will progress to a daily need for NSAIDS. During times of flare-ups patients will also benefit from additional analgesics such as Codeine or Tylenol 3 (both at 1 mg/kg q8-12h) or Tramadol (5 mg/kg q 8h). The biggest benefit in these patients is owner education. It is very important that owners are aware that this will be progressive and we can’t cure it. Flare-ups will occur and management must be stepwise.

If I have patients that don’t respond initially, or have more frequent flare-ups then NSAID use becomes more frequent. I will consider adding in gabapentin (5-10 mg/kg q8-12h) as well as formal rehabilitation. Other potential considerations are given to using amantadine (3-5 mg/kg q24h) with an NSAID, or intra-articular injections.

Potential intra-articular therapies include regenerative medicine (platelet rich plasma with or without stem cell treatment), hyaluronic acid, or steroids. Discussion of regenerative medicine is beyond the scope of this proceeding. HA is a viscosupplementation that restores the physiochemical properties to the joint. From a molecular standpoint it stimulates production of ECM as well as continued production of HA from resident synoviocytes. It will also inhibit inflammatory mediators. It is important to use a product that closely mimics a dog’s HA such as Evervisc from Everost (sold through Patterson). Evervisc is about 2 million Daltons in size and is made from a fermentation process rather than rooster combs. Until further research is completed it is not recommended to combine an HA injection with any other drug as this may decrease the molecular weight of the HA or could lessen its efficacy. What has been shown is that approximately 80% of dogs respond well to HA, 10% respond fair, and 10% don’t respond. The duration of response is about 4-6 months of relief. When compared to regenerative medicine a response of about 9 months is expected following a platelet rich plasma injection and about 12 months or longer following a platelet rich plasma and stem cell injection.

In summary, OA is a chronic progressive disease and the goal of management needs to be to slow and minimize the progression. Owners need to be well educated to know that it will progress and there will be flare-ups. Treatment needs to be multimodal and patient centered.

References
Patella luxation is a common cause of lameness in dogs of all sizes and can also be seen in cats. The patella is an ossification in the tendon of the quadriceps femoris muscle that acts to redirect the line of action of the quadriceps tendon in a similar function to the way a pulley redirects a cable. Thus for the patella to function properly axial alignment of the extensor apparatus is necessary. Briefly, the quadriceps mechanism is composed of the rectus femoris which originates on the ventral aspect of the ilium just cranial to the acetabulum, the vastus lateralis, vastus medialis, and vastus intermedius, which all originate on the proximal femur. All 4 tendons converge on the patella (patellar tendon), which continues on and attaches on the tibial tuberosity (patellar ligament). Contraction of the quadriceps femoris results in the patella being drawn onto a straight line that connects the proximal and distal attachments. Therefore, for the patella to be stable the extensor mechanism must align with the underlying skeleton (femoral shaft, trochlear groove, tibial tuberosity).

Medial patellar luxation (MPL)
MPL is a common cause of lameness in small breed dogs and must be differentiated from issues of the hip (avascular necrosis of the femoral head) or other stifle issues (such as a CCL rupture). In fact MPL is the most common direction of patella luxation overall, and up to 98% of patella luxations in small breed dogs are medial. In large breed dogs MPL is also the most common direction found in 67-83% of cases (Labradors being the most common). Bilateral luxations are noted in 50-65% of cases. MPL has been described as congenital; however, it is technically a developmental disorder because the MPL is not present at birth. The skeletal abnormalities that predispose an animal to an MPL are present. In other words the MPL develops after birth as a result of deformities that may be present at birth. Many of the skeletal abnormalities noted are considered inherited; therefore, affected patients should not be bred. The exact etiology is not completely understood; however, coxa vara (a decreased angle of inclination of the femoral neck) and a diminished anteverision angle (relative retroversion) have been described as the beginning underlying skeletal abnormalities. Recently coxa vara has been called into question and along with coxa vara, coxa valga has also been shown to be present in MPL. Other skeletal abnormalities noted with MPL are femoral varus, genu varum, a shallow trochlear groove (with poorly developed or absent medial trochlear ridge), medial displacement of the tibial tuberosity, internal rotation of the tibia relative to the femur, proximal tibial varus and internal rotation of the foot. In short, the thought is that coxa vara and diminished anteverision angle result in displacement of the quadriceps medially. This displacement then results in abnormal forces on the distal physis, which may retard the growth of the medial side resulting in distal femoral varus and internal rotation of the tibia. Furthermore, absence of the patella in the trochlear groove does not result in the typical pressure of the groove during growth, which results in trochlear hypoplasia.

The diagnosis of a MPL is based off of the physical examination, not the degree of skeletal deformities present. Careful palpation is needed to characterize the grade of the luxation and rule out any concurrent diseases. Gait evaluation at both the walk and trot is important. I find that it is easier for my physical examination to have the patient standing so that I can fully assess symmetry between limbs and to help judge the influence of the quadriceps muscle contraction during weight bearing. In some cases locating the patella can be challenging. At times, I will locate the patellar ligament and follow it proximally from its attachment on the tibial tuberosity until I locate the patella. Once located note if the patella is in the groove or out of the groove to begin to establish a grade. After I have located the patella I will hold it hold it between my thumb and index finger while using the other hand to grasp the tibia and lift the foot from the floor. At that point I run the stifle through range of motion including flexion, extension, internal and external rotation. While completing range of motion I will apply manual pressure in a medial and lateral direction to identify the direction and grade of luxation. For example, extending the stifle and applying medially directed pressure to the patella, internally rotating the tibia at the stifle joint, and flexing the stifle can usually cause a patella to luxate medially. When palpating the stifle pay particular attention to any joint effusion. Joint effusion is commonly associated with CCL rupture and less commonly seen with a MPL. The trochlear groove can be palpated in many cases with the patella luxated to get an idea of the trochlear groove depth. The alignment of the quadriceps can be assessed visually with the hip, stifle, and hock in a neutral alignment as the stifle is flexed and extended.

Clinical signs associated with MPL vary with the degree or grade of the luxation. MPL’s are typically based on a scale of 1-4.
A grade 1 MPL is usually an incidental finding and is noted by the patella being located in the trochlear groove, it can be luxated medially when the stifle joint is held in full extension, but returns to the groove upon release of the manual pressure. There is no crepitus noted during range of motion and bony deformities are absent. Clinical signs are typically not present.

A grade 2 luxation will cause intermittent lameness when the patella is luxated; however, when palpated the patella is typically located within the trochlear groove. These patients have the typically “skipping” lameness. This is where the dog will suddenly skip and carry the leg without apparent discomfort, flexes and extends the joint several times, and then begins to bear weight again. The
patella can be luxated with combined internal tibial rotation and joint flexion. To reduce the patella extend the stifle and externally rotate. With a grade 2 luxation mild deformities develop usually consisting of internal rotation of the tibia and abduction of the hock. It can progress to a grade 3 luxation with continued cartilage erosion of the patella and trochlear groove or with concurrent CCL rupture. I tend to further classify grade 2 patella luxations into a low grade 2 or high grade 2 depending on how lax the patella feels during examination.

With a grade 3 luxation the patella is permanently luxated but can be reduced. More severe bony deformities are present such as marked internal tibial rotation and an “S-shaped” curve of the distal femur and proximal tibia. Many times a shallow trochlear groove can be palpated. These patients will often exhibit a “crouched” gait rather than an intermittent lameness because the dog often uses the leg in a semi-flexed, internally rotated position.

With a grade 4 luxation the patella is permanently luxated and can’t be reduced. Commonly, marked femoral varus, proximal tibia valgus, and internal tibial rotation are noted. The tibia is typically rotated 60-90 degrees relative to the sagittal plan. Affected animals can be debilitated and develop a “crablike” posture necessitating them having to be carried by their owners rather than walking. Acute worsening of the lameness is often associated with rupture of the CCL. Correction of grade 4 patella luxation early in life is ideal as waiting makes surgical correction more challenging.

CCL rupture has been associated with MPL, and the thought is that the CCL is placed under increased stress due to the abnormal quadriceps mechanism. Concurrent rupture of the CCL is noted in 15-20% of middle aged dogs with MPL. It is unclear if this truly occurs secondary to the MPL or whether it is a manifestation of CCL disease. However, any acute lameness in a patient with an MPL needs to raise the concern of a concurrent CCL rupture.

Radiographs are useful to document the luxation and assess the degree of arthritic changes present. It is also important to identify and if possible quantify any skeletal abnormalities. In cases of low grade MPL or with mild skeletal abnormalities orthogonal views of the stifle are sufficient. However, in cases with more severe skeletal abnormalities or higher-grade luxations then orthogonal views of the femur, tibia, and stifle will be needed. Note that deformities such as varus or valgus can be seen on radiographs, but torsion can only be assessed with a CT scan. A CT scan will also permit a 3-D reconstruction to aid in quantifying the deformities present.

The decision for treatment is based on the combination of clinical signs and grade of the luxation. Typically, grade 1 luxations with no clinical signs will not need surgical management. In cases of grade 3 or 4 luxations surgical correction is warranted early in the course of the disease to minimize the progression of skeletal abnormalities and OA. Grade 2 luxations tend to be more a grey zone in that if it is a grade 2 with no clinical signs then continued monitoring is necessary; however, if there is a grade 2 associated with clinical signs then surgical treatment is warranted.

Conservative management consists of daily controlled activities such as leash walking or ball play, avoidance of high impact activities and formal rehabilitation therapy as needed to improve muscle mass and proprioception. I will also recommend a glucosamine/chondroitin sulfate and omega-3 fatty acid knowing that there will be some degree of OA progression. Unfortunately, formal rehabilitation therapy may help during periods of lameness or with increasing muscle mass, it will not typically improve the grade of the luxation. Unlike in people with patella mal-tracking, dogs with patella luxation have underlying skeletal abnormalities. I also always caution owners about any acute worsening of the lameness for fear of a CCL rupture. Also, owners need to be aware that if the lameness or skipping is becoming progressively worse then surgical therapy needs to be the next step.

The approach to surgical management should be approached to each individual patient. For me I start with an arthrotomy to evaluate the intra-articular structures to ensure there is no damage to the CCL. After evaluation I turn my attention to the trochlear groove. In smaller dogs I tend to find there is a very shallow to absent groove and in chronic cases of grade 3 luxations a “pseudo-groove” is forming just medial (or lateral) to the trochlear groove. In larger dogs I feel as though the groove tends to be a little deeper. If there is a shallow groove I will perform a trochleoplasty via a block recession. I find that for me performing a block recession is easier than a wedge; however, if you prefer a wedge recession then that is fine. It was shown that a trochlear block recession resulted in increased proximal patellar depth (where the patella tends to luxate commonly), increased patellar articular contact, and greater resistance to patellar luxation in an extended position when compared to the wedge recession. For a block/wedge recession one should ensure that the trochlear groove is wide enough to accommodate the patella and the goal should be to provide approximately 50% of the depth of the patella. To prevent wobble or displacement of the block/wedge it is important to ensure that the osteotomies are performed parallel to the initial osteotomy.

Following evaluation of the trochlear groove I then focus my attention to the tibial tuberosity to ensure alignment of the quadriceps mechanism. To assess patellar tracking; the hip, stifle, and hock are placed in a neutral position, the patella is centered within the trochlear groove, the pes is directed vertically, and the surgeon stands at the end of the table near the hind feet. The patellar ligament is then traced from the patella to its attachment on the tibial tuberosity. If the line of action of the patellar ligament is not centered on, and parallel, to the trochlear groove then a tibial tuberosity transposition (TTT) is needed. When performed a TTT I used an osteotome
that is as wide as the tibial crest. The osteotomy is continued distally to leave the distal periosteal attachment intact. Once the osteotomy is complete the stifle is extended to decrease the tension on the patellar ligament and the tuberosity is transposed. Once transposed the stifle is then flexed to increase the tension on the patellar ligament, which will stabilize the tuberosity in its new location. Then appropriately sized k-wires are placed. Note that 2 k-wires should be used, as using only one will not stabilize the tuberosity. In larger dogs I will use a tension-band fixation to prevent inadvertent tibial crest fractures and to aid in the stability. Use care not to damage the stifle joint. The height of the fibular head is a useful landmark such that aiming distal to the fibular head will ensure the k-wires are distal to the joint.

After correcting the skeletal abnormalities then I will perform soft tissue reconstruction techniques. Very rarely if ever, will soft tissue reconstruction alone result in a satisfactory outcome in mature dogs. In immature dogs I will perform soft tissue reconstruction with the owners knowing that if a re-luxation occurs then a second surgery will be required once the patient is mature. To complete the soft tissue reconstruction I will release the retinaculum on the side of the luxation (note that I never leave the joint capsule open as part of the “release”). I will then perform an imbrication of the retinaculum opposite the side of the luxation by placing horizontal mattress sutures. Any redundant tissue is removed.

**Lateral patellar luxation (LPL)**

LPL is most commonly seen in larger breed dogs (albeit still less than MPL’s). About 2% of small breed dogs, 17-19% of medium to large breed, and up to 33% of giant breed dogs have been noted to have LPL. Proposed skeletal deformities associated with LPL are typically opposite of what is seen with an MPL. Changes such as coxa valga and an increased angle of anteversion are noted. The grading scheme for LPL’s is similar to the classification system of MPLs.

Because of the associated skeletal abnormalities noted with LPL’s well-positioned radiographs or a CT of the femur and tibia are necessary to quantify the deformities. Treatment is similar to MPL correction, only in the opposite direction. However, femoral corrective osteotomies may be needed.

Following surgery for either an MPL or LPL I will place patients in a soft padded bandage for 24 hours. Once home I recommend passive range of motion and stretching exercises. Beginning 2 weeks post operatively I will have owners begin slow controlled leash walks along with a formal home exercise plan. At this time point I will also recommend they begin formal rehabilitation therapy. Radiographs are needed at the 4 and 8-week mark to ensure appropriate healing. Grade 2 and 3 luxations have a good prognosis with a fair prognosis for grade 4 luxations. Complications include delayed union or fixation failure of the osteotomy site, re-luxation of the patella, infection, and OA. The overall complication rate for MPL surgery is about 18% with patella re-luxation occurring about 8-48% of the time. Typically, if re-luxation does occur it is to a lower grade than initially and surgical correction is not needed. If a patient has a re-luxation with clinical signs then surgical correction is warranted and the patient may need a corrective femoral osteotomy. LPL complications are similar to that of MPL’s, and in both situations the complication rates have been noted to be higher for dogs weighing greater than 20kg.

**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.

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 is important s these are the very forces that must be overcome when choosing the appropriate fracture fixation method.

Fractures 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 interfragmentary 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 interfragmentary strain has to be <2% and the interfragmentary 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
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, epiphysial, physeal (which have their own Salter Harris classification), metaphysial, or diaphysial. 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 is 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.

Physial fractures are classified by the Salter Harris (SH) classification scheme. SH I fractures are through the physial itself, while SH II fractures are through physial and into metaphysis. SH III fractures are through physial and into the epiphysial and are considered intra-articular. SH IV fractures are through physial and into metaphysis and epiphysial as well as being considered intra-articular. SH V are compression fractures though the physial, while SH VI are compression fractures though only a portion of the physial, 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.

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 and also should I fix this fracture meaning 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 beginning with the least invasive yet least stable (external coaptation) and proceeding through the more invasive yet more stable (IM pin/kerclage wire, external skeletal fixator, interlocking nail, or bone plate and screws) 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.
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 luxate 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 acetabulum. 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, its 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, chondroproctants, 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 16 weeks.
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
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 that make up the anatomy of the stifle such as the femur, tibia, patella, the soft tissue structures, as well as the intra-articular structures. There are 3 bones that make up the stifle. The femur has 3 major articular areas with 2 condyles that are convex, while the proximal tibia has 2 condyles that are 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 menisocofemoral ligament caudally. Its motion is more coupled with the femur and therefore is less likely to be injured compared to the medial meniscus. The meniscus has a wedge shape that causes radial extrusive forces to develop from compressive forces. The primary function of the meniscus is for load bearing, load distribution, shock absorption, and joint stability. Because of its shape it acts as a spacer and bears about 40-70% of the load.

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

Physiology

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

**Pathophysiology**

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

**Diagnosis**

The diagnosis is typically straightforward and is based off the history, signalment, clinical signs, physical exam, and orthopedic exam. The history may include an acute or chronic hind limb lameness that may be mild to non-weight bearing. Interestingly, owners may report that the lameness has improved from initial injury. This usually corresponds to the timeframe from when the initial inflammatory response is ending. Regarding the signalment any age or breed can be affected. Typically we tend to see medium to large breed dogs that are around 3-8 years of age. The orthopedic exam is mainstay to diagnosing a CCL rupture. Findings may include a positive sit test where the dog will tend to sit with the affected leg projecting out to the side. Pain on hyperextension is usually the forgotten test but is very reliable. Most affected dogs will exhibit some degree of pain. Crepitus may be noted during ROM, and with chronic tears medial buttress formation may be noted. This is the peri-articular fibrosis that occurs. The classic findings for a CCL rupture are joint effusion, the cranial drawer test and the tibial compression test. A simple way to think about it, is that in an adult dog joint effusion will only be caused by a CCL rupture, septic arthritis, tick-borne disease, or immune-mediated arthritis. A medial patella luxation (MPL) will not cause the same degree of joint effusion, so if you have a patient 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 craniomedial which remains taut in both flexion and extension and the caudolateral, which is taut in extension but lax in flexion. For example if the craniomedial band is torn and the caudolateral band is intact cranial drawer is only present in flexion because in extension the caudolateral band is taut. If the caudolateral band is torn and the craniomedial band is intact no cranial drawer is present because the craniomedial band is taut in both flexion and extension. Cranial tibial thrust is a test meant to mimic active weight bearing. The goal is to hold the stifle at a standing angle (approximately 135 degrees) and while holding the stifle still flex the hock. If the CCL is ruptured there should be a cranial displacement of the tibia. As with cranial drawer, tibial thrust should be checked in both flexion and extension.

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

**Treatment**

When deciding on a treatment plan there is no one treatment fits all, but there are many, many, many options available. The reason there are so many options is because not one procedure or medical management technique is 100% perfect. I think one reason for this is because what is considered our final outcome, a stable stifle, a patient that returns to activity pain free, elimination of OA, owner satisfaction, etc.? We will never be content on cruciate disease until we figure out the goals we want to achieve for an outcome.
When I approach a dog with cruciate disease I'm going to have the same conversation with each owner; however, depending on each case I may swing my conversation in one particular direction. Factors I consider when deciding on conservative vs. surgical treatment and which procedure are the patient, owner, and veterinarian factors. I look at the breed, the size of the animal, the age, the activity level, and what is that particular 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. This can’t occur with the dog remaining active. To break it down to them I tell the owners the dog must be kept in an area where he/she can stand up, lie down, and turn around. The dog eats, drinks, and sleeps in the crate. It only goes outside to urinate and defecate on a leash then back into the crate. I also throw the disclaimer in that in my opinion OA is worse with a rapid progression as long as the stifle is unstable and usually if this is a larger dog they wont return to full function. I also really push the fact that the dog will appear to 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 can't ask the patient if he/she will tolerate the brace, I have had some dogs that don’t “mind” it at all, others take time, and some just freeze or try to chew it. The other issue is given the different shapes and sizes of dog stifles the brace MUST be custom made. This means a mold must be made and sent to the orthotist and then sent back about 2 weeks later. It’s a horrible feeling to have an owner pay the expense for a brace and then the dog won’t tolerate it.

2. **Arthritic progress:** What I can tell an owner is that with surgery we can slow down and minimize arthritic progression. Without surgery we will have continued accelerated and worsening progression OA. Along that scale is a brace; I just don’t know if the scale is closer to that of surgery or that of no-surgery?

3. **Meniscal damage:** What I can tell an owner is that with surgery we can minimize the chances of a meniscal injury. Without surgery there is a high incidence of meniscal injury. The problem is again along that scale I don’t know where a brace will fall. Will it help protect the meniscus the same as surgery, or will it not make a difference such as doing nothing? This does bring up a good point about meniscal damage. A “meniscal click” will only get you about 30-40% correct at identifying a meniscal injury. If you add in a positive McMurray test and pain on hyperflexion that may improve to about 50%. Personally, I feel as if a dog has a meniscal tear they will not benefit from a brace because it will do nothing to help with the pain and discomfort. The problem is if at best you can diagnose a meniscal injury in 50% of patients then how does one approach determining if there is meniscal injury? A MRI could be considered but is costly and requires general anesthesia, arthroscopy could be considered but personally would be below the standard of care to go to surgery to identify a meniscal injury but not treat the CCL rupture. Therefore, if I have owners that want their dog in a brace then they must undergo a stifle ultrasound. If there is evidence of meniscal damage then that dog will not be a good candidate for a brace, if they don’t appear to have meniscal damage then we can give it a shot knowing that an ultrasound is not 100%.

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
Developmental orthopedic diseases (DOD) are those, which are identified during postnatal skeletal growth. The common DOD discussed here are hypertrophic osteodystrophy (HOD), panosteitis, craniofacial osteopathy, retained ulnar cartilaginous cores, Legg-Calvé-Perthes disease, and osteochondrosis/osteoarthritis (OCD). 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.¹

**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 Airedale Terriers, Irish Setters, 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 predisposed 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

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David Dycus, DVM, MS, DACVS, CCRP
Veterinary Orthopedic and Sports Medicine Group
Annapolis Junction, MD

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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.

**Craniomandibular osteopathy**

Craniomandibular Osteopathy is also known as craniomandibular osteoarthropathy, craniomandibular osteodystrophy, 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. 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.

**Legg-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 endochondral 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 epiphyseal 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 dissecans (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 common 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 (OCD) 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 compliant 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