Forelimb lameness: Diagnostic modalities
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Osteochondritis dissecans (OCD) is a manifestation of osteochondrosis in which a flap of cartilage is lifted from the articular surface. Osteochondrosis is thought to proceed OCD and is a disturbance in endochondral ossification. The disturbance in endochondral ossification leads to multiple areas of cartilage islands on the surface and within the humeral epiphysis which have not undergone normal maturation into bone. Areas of abnormal endochondral ossification of the articular surface become thickened and are susceptible to fissure and loosening (OCD) as the deeper chondrocytes undergo necrosis due to inadequate nutrition and a suboptimal microenvironment.

Large and giant-breed dogs are commonly affected and males are more often affected than females. Clinical signs often develop between 4 and 8 months of age; however, some dogs may not be presented for veterinary evaluation until they are mature. Affected animals are usually presented for examination because of unilateral forelimb lameness. Owners usually report a gradual onset of lameness that improves after rest and worsens after exercise.

Radiographs: Despite apparent lameness in only one limb, both shoulders should be radiographed because this condition is often bilateral. Sedation may be required for quality radiographs particularly in large hyper-active dogs. The earliest radiographic sign of OCD is flattening of the caudal humeral head. This is due to thickening of the articular cartilage and deviation of the subchondral bone line.

As the disease progresses, a saucer-shaped radiolucent area in the caudal humeral head may be visualized. Calcification of the flap may allow visualization of the flap either in situ or within the joint if it has detached from the underlying bone. In chronic cases, large calcified joint mice are often observed in the caudoventral joint pouch or cranially within the bicipital groove.

Forelimb lameness can be a diagnostic challenge in the athletic dog; often the lameness has been treated for months with no improvement. The only abnormal physical finding may be the observation of Grade 2 or Grade 3 lameness. The source of lameness may be
attributed to soft tissue injury, bony injury or a combination of both. In the active adult dog the most common cause of latent forelimb lameness can be attributed to pathology in the elbow and to injury of the active and passive shoulder restraints. In the author’s experience, pathology in the elbow is regularly caused by occult microfracture/fragmentation of the medial coronoid process. There is no joint effusion, loss of motion, pain, or crepitus on physical examination. Radiographs are reported as normal or may show minimal subtrochlear sclerosis of the ulna.

![Image 1](image1.png)

Modalities to facilitate an accurate diagnosis in these cases are CT, nuclear scan, and arthroscopy. Recommendations for performing CT include scanning from the point of the olecranon to 2cm distal to the radial head. Scan thickness should be 1-2mm with .5mm overlapping slice index. Transverse slices using 1500 to 3500 HU are ideal for imaging subchondral bone and fragments of the medial coronoid; transverse images at 3500HU are considered ideal for identifying ostomalacic lesions of the medial coronoid.

![Image 2](image2.png)

Nuclear scintigraphy can be used to localize the origin of the lameness and can be used to facilitate detection of subtle pathologic changes before changes are evident of radiographs. Technetium phosphonates are typically used for scintigraphy of joint tissues. Scintigraphy has high sensitivity for detection of presence of elbow pathology but is not specific for definitive diagnosis. Regulatory issues often limit the use of scintigraphy to academic institutions or large referral practices. Nevertheless it is invaluable facilitating lesion localization in dogs with forelimb lameness. Most commonly, it is used to rule in
or rule out elbow pathology. Note in the cases shown below the uptake in technetium in the involved elbow compared to the normal elbow. Each of the below cases had a long standing undiagnosed forelimb lameness. Radiographs of these cases were considered within normal limits. The use of scintigraphy localized the lesion to the elbow which then allowed application of more specific diagnostic modalities such as CT or arthroscopy.

Arthroscopy is more invasive than imaging modalities but is very specific for identification of pathology in the medial compartment of the elbow. A comparison of CT with arthroscopy showed that these procedures were complimentary for medial coronoid assessment. Care must be exercised when assessing the medial coronoid on CT and arthroscopically. Fragmentation of the articular cartilage, micro fissures and nondisplaced fragments may not be detected on CT. Likewise, with arthroscopy, thorough probing and or curettage adjacent to the radial head often will reveal abnormal bone or fragments beneath the cartilage surface not visualized on with casual observation.

Active and passive restraints of the Shoulder: Diagnosis and Treatment

Forelimb lameness attributed to shoulder instability is reported frequently in the literature. Some orthopedic centers report the presence of subtle shoulder instability frequently where as others rarely diagnose the condition. The widespread difference in
the frequency of diagnosis between clinics has led to disagreement relative to criteria for diagnosis of shoulder instability and the arthroscopic appearance normal intra-articular structures versus pathologic lesions. Stability of the shoulder is derived from passive and active restraints. Passive mechanisms include the medial and lateral glenohumeral ligaments, surrounding joint capsule, joint conformation, and synovial fluid cohesion. The medial collateral ligament (MCL) commonly appears as “y” shaped with the cranial arm coursing caudally from its origin at the medial surface of the supraglenoid tubercle. The caudal arm of the MCL originates from the medial surface of the scapular neck and joins the cranial arm to insert onto the humeral neck. The MCL and associated joint capsule is a major factor in providing joint stability; complete medial luxation occurs following transection of the MGHL. The lateral collateral ligament (LCL) originates from the lateral rim of the glenoid and extends ventrally to insert onto the humerus at the caudal region of the greater tubercle. The joint capsule originates from the periphery of the glenoid cavity. Medially, the joint capsule forms a synovial recess due to its attachment several millimeters proximal to the glenoid rim.

Dynamic active glenohumeral stability is provided by contraction of the surrounding cuff muscles. These include the biceps brachii, subscapularis, teres minor, supraspinatus, and infraspinatous muscles. Active contraction of all or selective cuff muscles induce compression across the shoulder joint as well as increasing tension in the joint capsule. When tested in neutral position, the cranial, lateral, and medial translation of the humerus was significantly increased after biceps tendon transection. In the flexed position, translation of the humerus in the cranial and lateral directions was significantly increased after biceps tendon transection. In the extended position, the medial translation of the humerus was significantly increased after biceps tendon transaction.
Examination of the shoulder for stability should be done under anesthesia or heavy sedation. Flexion, extension, abduction, craniocaudal translation, and rotational stability of the shoulder joint should be assessed. Normal range of flexion and extension are 40 degrees for flexion and 165 degrees for extension. Circumduction of the shoulder should not give rise to subluxation. Anteroposterior translation should be similar in both shoulders. A normal abduction test is approximately 23 degrees; abnormal abduction is considered present when abduction exceeds this degree and there is a difference in abduction angle between the injured side and the normal side. When performing the abduction test, it is essential to maintain the limb in extension with the elbow in neutral position; ie, one does not want the elbow externally rotated. If the elbow is externally rotated with the limb in extension, the shoulder joint will be internally rotated. The latter will give a false positive abduction test. To maintain the elbow in neutral position, the examiner should place his/her thumb on the lateral surface of the olecranon just posterior to the humeral epicondyle. Maintaining the thumb facing upward assures that the elbow remains in neutral position.

Care should be exercised in interpretation of side to side laxity difference. We have documented consistent abnormal abduction and AP translation is cases of long standing unilateral forelimb lameness. Our interpretation of shoulder laxity in these cases is that muscle and ligamentous atrophy give rise to increased shoulder laxity. In some cases, the shoulder laxity is secondary to long standing shoulder pathology such as OCD. Other cases are referred to our centers for treatment of shoulder pain. The pain is noted when the shoulder is placed in extension. By and large these cases are latent elbow dysplasia; the pain in fact arises from extension of the elbow when the shoulder is placed in extension.
Debate continues amongst surgeons as to the interpretation of the arthroscopic appearance of intra-articular structures. Some surgeons believe it is possible to detect increased intra-articular volume and redundant capsular/ligament structures within the joint while others believe this cannot be accurately detected arthroscopically. Further, the significance of intra-articular lesions remains controversial. While some surgeons believe small tears of the cranial arm of the MCL result in instability and require treatment, others believe these small tears are insignificant and most likely iatrogenic. The gutter medial to the cranial arm of the MCL adjacent to the insertion of the subscapularis is often diagnosed as a site of trauma. However, this zone often shows synovial proliferation with pathologic conditions of the shoulder such as OCD.
**Supraspinatus strain:** Dogs with strain of the supraspinatus tendon present with a chronic foreleg lameness; some dogs will exhibit periods of non-weight bearing lameness. Although uncommon, any age or breed of dog can be afflicted; however, the condition is seen more in large breeds of dogs (Labrador, Rottweiler). Radiographic views should include standard lateral projections of both shoulders. Mineralization is seen adjacent to the greater tubercle of the humerus. Patterns of mineralization are either irregular, non-homogeneous or well circumscribed and dense foci. A “skyline” view of the bicipital groove is helpful to delineate the location of dystrophic mineralization.

It is worthy to note that the mineralization of the supraspinatus insertion indicating chronic strain may be present but not be the cause of clinical dysfunction. Often mineralization is present in the shoulder in which the dog is not lame. Bilateral mineralization may be present (although different stages of mineralization can be seen between shoulders) and the dog only lame in one shoulder. Recently, MRI has been advocated as a diagnostic tool for shoulder lameness. Some surgeons believe that mineralization of the supraspinatus tendon displaces the biceps tendon causing biceps tendon pain. Although possible, ultrasound examination of the biceps tendon does not demonstrate inflammation secondary to biceps impingement. Also, the position of the biceps tendon is 3-5mm separate from the insertion of the supraspinatus tendon on the greater tubercle of the humerus. Arthrography can be used to outline the bicipital groove to determine if irregularities or filling defects suggestive of bicipital tenosynovitis are present. Diagnosis is based on clinical signs, imaging, and most importantly, ruling out other causes of forelimb lameness. Treatment is en bloc resection of the chronically inflamed section of the tendon. The tendon can be tenodesed in a position where it is exposed to less strain.

**Bicipital tenosynovitis:** Bicipital tenosynovitis is an inflammation of the biceps brachii tendon and its surrounding synovial sheath. The etiology of bicipital tenosynovitis is either direct or indirect trauma to the bicipital tendon or tendon sheath. Direct trauma due to repetitive injury may be an inciting factor and result in partial or complete tearing of the tendon. Indirect trauma secondary to proliferative fibrous connective tissue, osteophytes or adhesions between the tendon and sheath limit motion and cause pain. It has been hypothesized that mineralization of the supraspinatus tendon causes a secondary
mechanical bicipital tenosynovitis. Affected dogs are usually medium- to large-sized, and middle-aged or older. Working and active dogs are more commonly affected; there is no predisposition for either sex. Intermittent or progressive forelimb lameness, which worsens after exercise, is common. The owner may relate the lameness to trauma, but usually there is slow onset of clinical signs. Radiographically, bony resorption at the supraglenoid tuberosity is characteristic of chronic strain at the origin of the biceps tendon. Medical or surgical management have been successful in treatment of bicipital tenosynovitis.

Medical treatment consists of injecting methylprednisolone acetate into the tendon sheath and restricting activity for 3 weeks. In one report (Stobie, JAVMA 1995) reported 50% of dogs treated medically had good to excellent outcome. In the same report, all dogs treated surgically had good to excellent outcome. Given these results, initiating treatment with 1 to 2 steroid injections is a reasonable approach. If outcome is not favorable, surgical intervention is advised. The bicipital tendon may show partial tearing of tendon fibers or partial tearing of the origin of the biceps tendon at the supraglenoid tuberosity. If the biceps tendon shows evidence of partial or complete tearing, the tendon is released by transecting the tendon just distal to the torn portion. Likewise, if there is evidence of synovial proliferation, mineralization, and osteophyte formation within the bicipital groove, the tendon is released from its origin. Tendon release may be performed with either a blade instrument (banana knife, beaver blade, or 11 blade) or a radiofrequency probe. The blade is slightly faster than the radiofrequency probe; however, use of the probe will prevent problems with hemorrhage from vasculature that is often present in the center of the tendon. Upon completion of tendon release, the tendon origin should be closely inspected for small osteochondral avulsion fragments, which can be removed with a motorized shaver or graspers. Lastly, the joint is thoroughly flushed by increasing the ingress flow and allowing egress through a large instrument cannula. Inspect the joint for remaining pathology and then remove the arthroscope and instrument cannula. Suture the portals with non-reactive, non-absorbable suture.

**Elbow: Medial compartment syndrome** has been classically attributed to elbow dysplasia. The term canine elbow dysplasia is used to denote an abnormal development of the elbow joint which results in a degree of incongruence. It is used to describe developmental diseases which include ununited anconeal process, fragmentation of the medial coronoid process and osteoarthritis, all believed to be secondary to joint incongruity. The proposed incongruence results in mechanical overload of the medial coronoid/medial humeral condyle through establishment of a “short” radius or “long” ulna. The fact that
fragmentation of the medial coronoid will occur with incongruence is well noted in cases of concentric distal radial physeal injury. The latter results in a short radius, mechanical overload of the medial coronoid, and microfracture/fragmentation of the medial coronoid. Does elbow dysplasia (incongruence) exist? Yes. No question elbow dysplasia is seen in younger dogs with developmental incongruence. Not the severe cartilage change and fragmentation of the medial coronoid in the young Irish Wolfhound in the figure. However, elbow incongruence is not present in all dogs with elbow dysplasia; perhaps we do not have the imaging modality to detect subtle incongruence. Also, elbow dysplasia, a developmental condition that should be present from a juvenile age, does not account for the senior dog who has never experienced a lameness problem and presents with sudden onset lameness. The latter signalment/presentation accounts for the majority of forelimb lameness cases attributed to the elbow encountered by the author. As such, there must be other factors involved in the development of medial compartment syndrome.

Fitzpatrick (proc ACVS 2010) has shown with the use of micro CT that the pattern of trabecular fracture is consistent with acute traumatic overload (cranial tip fragment) or chronic rotational overload (radial incisures fracture). Recent work by Goodrich (proc VOS 2013) clearly has shown the weight bearing axis of the forelimb to be directed through the medial compartment. Also evident through CT of cases with medial compartment syndrome there increased bone density (sclerosis) in the medial compartment consistent with chronic repetitive mechanical overload. Acute mechanical overload directed through the medial compartment could easily account for the cranial tip fractures as described by Fitzpatrick.

9 yr old Malanois with acute onset lameness: note fragmentation, no articular cartilage pathology

9 year old Retriever with sudden onset lameness; collapse medial compartment
Repetitive mechanical overload generated by years of athletic or playful activity can account for the fragmentation and wear of articular cartilage.

Fitzpatrick has also proposed repetitive rotational overload as a possible cause of radial incisure fragmentation of the medial coronoid. The exact cause of the rotational load is not known but may be attributed to muscular forces (ex. contraction of the biceps/brachialis complex, Hulse Vet Surg 2010) or rotation of the medial humeral condyle against the radial incisure for the medial coronoid (Bottcher proc ACVS 2013). Repetitive rotation of the medial coronoid by contraction of the biceps/brachialis complex compresses the coronoid against the radial head generating a shear stress which corresponds to the fracture plane of a radial incisures fracture.

Peter Bottcher has described rotation of the medial humeral condyle on the radial incisures as a possible cause of fragmentation.

Treatment

Treatment of medial compartment syndrome includes fragment removal. This includes not only visible fragmentation but fragmentation which may be present beneath the articular surface (subtotal coronoidectomy). If surface articular pathology is present (abrasion) further treatment with medial compartment arthroplasty (abrasion arthroplasty, microfracture), decreasing medial compartment mechanical load (biceps release, sliding humeral osteotomy), and conservative modalities (weight loss, O-3 FA diet, exercise moderation, nutraceuticals, ACP, stem cell, adequan, polygylcan, NSAIDs) are indicated. If clinical lameness persists, joint replacement (CUE, Tate TER) are considered.

Fragment removal is achieved via arthroscopy or mini-arthrotomy. It is important to note that stress fractures may be present in addition to the obvious visible fragment. A limited subtotal coronoidectomy is advised. A radial incisure fragment in an adult Pittbull with sudden onset lameness. 1 yr prior to this surgery, a small fragment was removed. Dog never recovered. Repeat arthroscopy found this fragment beneath the articular surface.
Mechanical unloading of the medial compartment:

**Biceps ulnar release procedure;**
The biceps/brachialis muscles constitute a large muscular complex. The anatomic origin and insertion of the biceps and brachialis muscles are such that the muscular complex exerts considerable force on the medial compartment of the elbow. The force exerted by the biceps is continuous since it is a pennate muscle with central tendon. More importantly, because the insertion of the biceps/brachialis complex is at the ulnar tuberosity, a large polar (rotational) moment is exerted at the cranial segment of the medial coronoid. The magnitude of the polar moment is a product of the moment arm (distance from the ulnar tuberosity to the tip of the coronoid) multiplied by the force created by the biceps/brachialis muscular complex. The polar moment rotates and compresses the craniolateral segment of the medial coronoid against the radial head. The compressive force is medial to lateral transverse to the long axis of the coronoid. A compressive force generates internal shear stress at an oblique angle to the applied compressive force. In this situation, maximal internal shear stress would be oblique to the long axis of the coronoid. Under the right circumstances, the polar moment and resultant compressive force produced by the biceps/brachialis complex may produce sufficient internal shear stress to exceed the material strength of the cancellous bone in the craniolateral segment of the medial coronoid. The result would be microfracture/fragmentation adjacent to the radial head at an oblique angle to the long axis of the medial coronoid. The surgical technique involves releasing the ulnar insertion of the biceps to unload the medial compartment and prevent the rotational moment rotating the coronoid into the radial head.

**Osteotomies:** The deviation of the mechanical axis medially and the humeral varus become more apparent with the progression of medial compartment OA similar to the process in the human knee. The result of overload of the medial compartment, collapse to the medial compartment, and OA. Sliding humeral osteotomy(SHO): Sliding humeral osteotomy involves creating a midshaft transverse humeral osteotomy and translating (sliding) the diaphysis distal to the osteotomy medially. Doing so shifts the weight bearing axis through the elbow joint from the medial compartment to the lateral compartment. Owner and vet VAS scores have improved in all cases with a notable decrease in pain upon elbow manipulation

**PAUL procedure**
The PAUL procedure shifts the weight bearing axis through caudal tipping of the medial coronoid
Dynamic bi-planar ulnar osteotomy described by Noel Fitzpatrick as a method of unloading the medial compartment.

Arthroplasty

Management of articular cartilage lesions is based on the concept that providing blood with mesenchymal cell precursors access to the lesion; this encourages healing by formation of fibrocartilage. Several marrow stimulating techniques have been described to achieve this. Abrasion arthroplasty involves uniform removal of subchondral bone until bleeding is achieved. This can be accomplished in the canine elbow by use of either a curette or burr attachment on a small joint shaver. The shaver is usually more rapid and efficient and generally just as accurate. Another marrow stimulating technique is microfracture. In this technique numerous microcracks are created in the subchondral bone plate with a specialized micropick to allow bleeding at the lesion surface.

Objective evidence documenting the efficiency of abrasion arthroplasty or microfracture is not available in the dog. The figure to the right shows resurfacing of the medial coronoid in a dog 6 months after abrasion arthroplasty. In man microfracture appears to be more effective than abrasion arthroplasty and is the marrow stimulating technique of choice. The technique is highly dependent on appropriate post operative rehabilitation. In man, 4-6 weeks of non-weight bearing activity coupled with active or passive range of motion is necessary for ideal outcome. Overall, the results of abrasion arthroplasty have been unpredictable and symptoms often recur 2-3 years after surgery. Nevertheless, good to excellent results are reported in 50 – 60 % of patients.

Elbow replacement is an option in dogs which have end stage elbow OA and conservative/less invasive surgical modalities have not resolved clinical pain. A number of prosthesis are available but the most popular one today is the TATE elbow. Clinical outcome studies indicate that a mechanical lamenessness may persist but that the dogs appear
to be less painful. A prosthesis presently in clinical trial is the CUE (canine unicompartmental elbow). The concept is simple and carries little morbidity. Information concerning this technique will be forthcoming in the near future.
Osteoarthritis is the most common cause of chronic pain in dogs with approximately one in five adult dogs having OA. OA (sometimes referred to as degenerative joint disease) is a slowly progressive degenerative disease involving the entire joint: articular cartilage, subchondral bone, synovial lining, joint fluid, ligaments, and muscles. Osteoarthritis is commonly classified as primary OA or secondary OA. Primary OA is associated with aging and chronic loading and wear of the articular surface. Secondary OA (the most common form seen in dogs) has many acquired and congenital etiologies including: ligamentous injury (CCL), abnormal joint conformation (elbow dysplasia), Osteochondrosis (OCD shoulder). In general, OA can develop in any joint where abnormal stresses are imposed on a normal joint or alternatively where normal stresses are imposed on an abnormal joint. Although more senior dogs exhibit clinical signs of OA as compared to their younger counterparts, younger dogs may also exhibit signs of OA. The most common example in younger dogs being OA associated with hip dysplasia. It becomes easy to understand why OA is painful when joint innervation and the role of inflammatory cytokines are considered.

Innervation of joints includes nociceptors which are free nerve endings found in all joint tissue except articular cartilage. They are found in the subsynovial layer only two to four cell layers beneath the synoviocytes lining the joint cavity. Dogs with OA have an ongoing synovitis the severity of which varies depending upon activity and joint trauma. The synovitis is accompanied by the accumulation of increased levels of eicosinoids (prostaglandins, leukotrienes) and pro-inflammatory cytokines (IL-1, TNF, NO) in joint fluid. Additionally, synovitis is accompanied by increased vascular flow in the subsynovial tissue. These two factors, increased inflammatory mediators in the joint fluid and increased blood flow in subsynovial tissue, increases the exposure of free nerve endings (nociceptors) to inflammatory mediators. The result is sensitization of free nerve endings, increased stimulation of free nerve endings, and transmission of pain to the CNS. Inflammatory mediators also up regulate the expression of harmful mediators which play a role in catabolism of articular cartilage.

The architect of cartilage is the chondrocyte which produces the extracellular matrix. The matrix is composed of glycosaminoglycans (hyaluronan and proteoglycan) and collagens (mainly type II). The collagen forms a dense network that retains the proteoglycan. The proteoglycan is highly charged and attracts water into the tissue. Thus cartilage is 75% water. In normal cartilage there is a very slow turnover of collagens but the proteoglycan is constantly being renewed. The proteoglycans are aggregated into large molecules ("aggrecan") with a protein core and many side chains of keratan sulphate and chondroitin sulphate. This core is in turn bound to hyaluronan chains with each chain containing many proteoglycan molecules. Aggrecan and water provide the compressive stiffness to the tissue whereas collagen provides the tensile strength. The morphological changes seen in OA include: 1. cartilage loss, especially in areas of increased load, 2. subchondral bone remodelling (loss of bone initially followed by sclerosis), 3. marginal osteophytosis, 4. variable synovial inflammation. The biochemical changes in the cartilage include: 1. loss of proteoglycan, 2.upregulation in the degradative and synthetic activities of chondrocytes, 3. disruption of the collagen network, 4. increase in water content. These changes reduce the elasticity of the cartilage leading to fibrillation and fissuring of the cartilage with eventual loss of tissue. If this continues eburnation of subchondral bone may result. It is proposed that the cytokines responsible for stimulating cartilage degradation in OA are
interleukins 1 and 6 (IL-1 and IL-6) and tumor necrosis factor-α (TNF-α). However, whilst these cytokines have been shown to stimulate degradation in several species, their effect in the dog is less marked. Recent in vitro studies (Innes) on canine cartilage explants show the resistance of canine cartilage to rhIL-1, rhIL-6 and rhTNF-α. However, canine cartilage does respond readily to oncostatin M (OSM) and Leukaemia Inhibitory Factor (LIF). Catabolic cytokines can stimulate the chondrocyte to produce and release degradative enzymes. The enzymes studied in most detail in this respect are the matrix metalloproteinases (MMPs) and the new family of endopeptidases the ADAM-TS-4 and -5 (A disintegrin and metalloproteinase with a thrombospondin motif). ADAM-TS-4 and –5 are also known as aggrecanases. MMPs and aggrecanases can cleave the protein core of aggrecan so as to release the majority of the molecule from the matrix. Under normal circumstances the chondrocyte also produces a natural inhibitor of these enzymes known as tissue inhibitor of metalloproteinase (TIMP). TIMP production appears to be decreased in OA.

Osteoarthritis progresses slowly and has a gradual onset of clinical signs. Subsequently, the diagnosis of OA is often made in the later stages of the degenerative process after extensive bone and joint damage has occurred. Commonly the diagnosis of OA is made by radiographic changes characteristic of degenerative joint disease. However, by the time radiographic changes are apparent the condition has progressed considerably. Therefore, early intervention using alternative diagnostic modalities is essential for the well being of the animal. One recommendation is to establish an Osteoarthritis pain assessment screening protocol. Behaviors consistent with OA in dogs include: limping, inactivity, difficulty rising, lagging behind on walks, stopping on walks, difficulty posturing to eliminate. Managing the osteoarthritic dog is multifocal; An accurate diagnosis is essential for the management of secondary osteoarthritis since surgical intervention may be necessary to correct the underlying problem to achieve optimal outcome. In addition to appropriate surgical intervention, successful treatment of osteoarthritis is a compilation of strategies including client education, behavior modification (both client and pet), appropriate exercise activities, rest, weight control, disease modifying agents and anti-inflammatory medications. Of these, controlled exercise activity coupled with adequate rest and weight control will benefit your pet as much or more than any other modality.

Regular physical activity and rest play a key role in wellness. Episodic physical activity may also be preferable to continuous exercise by avoiding injury due to overuse. Episodic activity refers to those activities that occur for a reasonable time period multiple times throughout the day. Of considerable harm to the process of osteoarthritis is your pet having a sedentary life throughout the week only to exercise strenuously on the weekend. This lifestyle exacerbates the osteoarthritis and is very likely to result in serious injury. Treatment regimes should include regularly scheduled rest. Exercise effectively squeezes the water out of the cartilage making it less compliant and more susceptible to injury. Rest allows fluids to seep back into the cartilage restoring its mechanical efficiency and lessening the incidence of injury due to overuse. Family members must learn to recognize their pet’s body signals and know when to stop or slow down. Doing so prevents pain and injury caused by overexertion. Two types of exercise are important in osteoarthritis management. The first type, therapeutic exercises, keeps joints working as well as possible. Therapeutic exercises are low impact and designed to maintain or increase joint range of motion, proprioceptive feedback, muscle tendon unit and periarticular tissue elasticity. Examples of therapeutic exercises are passive range of motion activity, massage, aquatic therapy, and stretching. The other type of exercise, aerobic conditioning exercises, improves strength and fitness, and controls weight. Examples are brisk
walking, brisk, walking or trotting through high grass, cavaletti training, and aquatic therapy. Your veterinarian and/or a rehabilitation therapist can evaluate your pet and develop a safe, personalized exercise program to increase strength and flexibility. Each program will include a warm up period, exercise period, and cool down period. Weight and body condition are important in preventing Osteoarthritis as well as an important factor in the treatment of osteoarthritis. Heavy dogs are at increased risk of developing arthritis because their joints may be strained by excess weight. This is especially evident in weight-bearing joints such as the knees and hips, which often show the first signs of weight-related strain and injury. An investigation into the cause of cranial cruciate ligament injury and the development of secondary osteoarthritis showed a significant risk factor to be obesity. One study in man showed that an average of 10 pounds of weight loss over a 10-year period decreased the risk of osteoarthritis of the knee by 50%. Similarly, obesity accounts for up to 30% of knee OA in man, exacerbates symptoms, and is associated with more rapid progression of the disease. If your pet is overweight and you enforce a weight loss program, you will dramatically decrease the risk of your pet injuring its knee joint and developing osteoarthritis. In fact studies of dogs with hip osteoarthritis show that reaching target reduction weight increases a dogs’ ability to move in a more normal fashion as assessed by gait analysis and owner observations.

Pain control medication allows the OA dog to engage in activity; this is turn helps control body weight and improve physical condition. The drugs of first choice for controlling arthritis are NSAIDs. NSAIDs function in part by inhibiting cyclooxygenase (COX) isoenzymes. COX-1 is the constitutive isoenzyme essential for the synthesis of homeostatic PGs in the GI tract, kidney, and platlets. COX-2 is for the most part induced and results in the production of PGs associated with pain and inflammation. However, COX-2 is also constitutively expressed and has a homeostatic role in canine brain, kidney, and vascular tissues. COX-3 is constitutively expressed and plays a role in brain tissue. NSAIDs approved for use in the dog include carprofen, deracoxib, etodolac, meloxicam, tepoxalin and others. All inhibit COX -1 and COX – 2 to varying degrees. The Coxib-class may exhibit less interference with the homeostatic functions of PGs associate with COX-1. However, the clinical effect of COX 1 vs COX -2 inhibition is largely unkown (Vioxx!!)

Carprofen, a NSAID which is less ulcerogenic, is marketed by Pfizer Animal Health under their trade name Rimadyl™. Rimadyl relieves pain and clinical signs of osteoarthritis in dogs, while causing less gastrointestinal side effects. Plasma and serum concentrations of carprofen are consistent throughout the treatment period. Serum concentrations peak at 2 hours, while synovial concentrations peak between 3-6 hours. The synovial concentration of carprofen ranges between 1-10 μg/ml during the treatment period in both normal and osteoarthritic joints. A significant reduction of PGE_2 from chondrocytes occurs at all concentrations in this range, %). Recent studies have shown carprofen to have little effect on kidney and platelet function. Carprofén has been recently found to support cartilage metabolism and proteoglycan synthesis.

Etodolac (Etogesic) is a Fort Dodge product used for treatment of osteoarthritis in dogs. The drug is available as a non-chewable tablet and is administered at a dose of 10-15 mk/kg every 24 hours. Etodolac has been found to be an effective treatment for ameliorating the clinical signs of osteoarthritis. Side effects with etodolac are typical of that seen with the NSAID class of drugs, gastrointestinal ulceration being the most common problem. Gastrointestinal ulceration can be severe at dosages above the labeled dose- this is well documented in their label claim during toxicity trials. Conflicting data has been found on
etodolac’s effect on proteoglycan synthesis and cartilage metabolism. The Cox 2:Cox 1 ratio appears to be less favorable as compared to carprofen.

Meloxicam was granted USDA approval in 2003, having been available in Europe since 1993. It is indicated for the control of pain and inflammation associated with OA in dogs. It is considered to have moderate COX-2 inhibition.

Deracoxib (Deramaxx) is a recently released NSAID from Novartis Animal Health approved for use in dogs for postoperative pain and inflammation. The recommended dose is 3-4 mg/kg, po, once daily for 7 days or 1-2 mg/kg, po, sid for chronic use. Like carprofen, deracoxib has a highly favorable Cox 1:Cox 2 ratio. The expected side effects are similar to other NSAIDS, primarily gastrointestinal disturbances.

The first dual-pathway (cyclooxygenase, lipooxygenase) canine NSAID, tepoxalin, has recently been approved. It has been suggested that the reduced ulcerogenic activity of tepoxalin is due to the ability to inhibit leukotriene production.

For many years, Aspirin was the most common NSAID used in the dog. Although effective in the majority of cases, aspirin is COX-1 selective causing platelet dysfunction and GI toxicity. Nevertheless, empirical observation would suggest that as many as 40% of pet owners administer aspirin to their pets. Even low dose aspirin causes GI lesions in dogs. However, dogs develop a tolerance to aspirin and lesions do not necessarily worsen. This has recently been explained by production of endothelial cell triggered lipoxin. Aspirin triggered lipoxin (APL) appears to be anti-inflammatory and decreases PMN migration to areas of ulceration. The production of APL is mediated through the COX-2 pathway. If aspirin is followed by or given concurrently with a COX-2 inhibitor, the APL pathway is blocked. Rather than APL production, a different pathway occurs giving rise to leukotriene B4 which is a very potent inflammatory cytokine. The result is a significant increase in GI ulceration. The clinical message is that one should not administer aspirin with a COX-2 inhibitor or administer a COX-2 inhibitor without adequate washout if aspirin has been used (10 -14 days).

Chondroprotective agents are emerging as a new class of drugs used to slow progression of and treat chronic DJD. These drugs not only should be antiinflammatory; but also should support anabolic (repair) processes in cartilage, bone and synovium essential for normalization of joint function. This class of drugs include the glycosaminoglycans. Examples of these drugs include glycosaminoglycan polysulfate ester, pentosen polysulfate and sodium hyaluronate. Cosequin (Nutramax Laboratories, Baltimore, MD) is marketed as a glycosaminoglycan enhancer, capable of providing raw materials needed for the synthesis of extracellular matrix of cartilage. Unlike most nutriceuticals, Cosequin has been evaluated in a variety of studies. Cosequin contains glucosamine which has been described as the building-block of the matrix of articular cartilage. It has been described as a preferential substrate and stimulant of proteoglycan biosynthesis, including hyaluronic acid and chondroitin sulfate. Cosequin also contains chondroitin sulfate, mixed glycosaminoglycans, and manganese ascorbate for the purpose of promoting glycosaminoglycan production. Orally administered glucosamine sulfate has been associated with relief of clinical signs of DJD and chondroprotection in clinical and experimental studies in man, horse and dog. Although glucosamine has a slower onset of relief of clinical signs associated with DJD as compared to ibuprofen, two clinical trials found it to have equal long term efficacy. No significant side effects have been reported with Cosequin.
Diagnosis and Treatment of Rear Limb Disorders
Don Hulse DVM Diplomate ACVS, ECVS

Hip dysplasia is an abnormal development of the coxofemoral joint. The syndrome is characterized by subluxation or complete luxation of the femoral head in the younger patient while in the older patient mild to severe degenerative joint disease is present. Laxity in the hip joint is responsible for the early clinical signs and joint changes. Subluxation stretches the fibrous joint capsule, producing pain and lameness. When the surface area of articulation is decreased, this concentrates the stress of weight bearing over a small area through the hip joint. Subsequently, fractures of the trabecular cancellous bone of the acetabulum can occur, causing pain and lameness. The cancellous bone of the acetabulum is easily deformed by the continual dorsal subluxation of the femoral head. This piston-like action causes a wearing of the acetabular articular surface from a horizontal plane to a more vertical plane causing subluxation to worsen. The physiologic response to joint laxity is proliferative fibroplasia of the joint capsule and increased thickness of the trabecular bone. This relieves the pain associated with capsular sprain and trabecular fractures. However, the surface area of articulation is still decreased causing premature wear of articular cartilage, exposure of subchondral pain fibers and lameness. This may occur early in the pathologic process or later in life. There are two general recognizable clinical syndromes associated with hip dysplasia: (1) patients 5 to 16 months of age, (2) patients with chronic degenerative joint disease. Patients in group 1 present with lameness between 5 to 8 months of age. Symptoms include difficulty when rising after periods of rest, exercise intolerance, restlessness at night, and intermittent or continual lameness. The majority of young patients will spontaneously improve clinically around 15 to 18 months of age. This clinical improvement is due to pain relief as proliferative fibrous tissue prevents further capsular sprain, and increased thickness of the subchondral bone prevents trabecular fractures. If symptoms occur later in life, they may include difficulty in rising, exercise intolerance, lameness following exercise, atrophy of the pelvic muscle mass, and a waddling gait with the rear quarters. Physical findings in the younger group of patients include pain during external rotation and abduction of the hip joint, poorly developed pelvic muscle mass, and exercise intolerance. Hip exam performed under general anesthesia will reveal abnormal angles of reduction and subluxation reflecting excessive joint laxity. Physical findings in the older group of patients include pain during extension of the hip joint, reduced range of motion, atrophy of the pelvic musculature, and exercise intolerance. Radio graphically, there are seven grades of variation in the congruity between the femoral head and acetabulum established by the Orthopedic Foundation for Animals. Excellent, good, fair, and near normal are considered within a range of normal. Dysplastic animals fall into the categories of mild, moderate, and severe. It is important to note that clinical signs do not always correlate with radiographic findings. Recently, patients have been evaluated using a distraction index where the degree of hyperlaxity is measured and correlated with standards for each breed.

Treatment is dependent upon the age of the patient, the degree of patient discomfort, physical and radiographic findings, client expectations of patient performance, and financial capability of the client. Conservative treatment is beneficial to a large number of patients in both the young and older patient groups. Conservative management is divided into acute management and long term management. When a dog exhibiting signs of hip dysplasia enters the clinic, it is generally because they have sprained the hip joint. The dysplastic joint is either hyperlax (young dog) has a limited range of motion (mature dog). In either case, the joint is easily sprained and the dog that is presented with symptoms has generally overused (sprained) the hip joint. The
management of the case at this time period is the same as treating any other acute sprain. Rest, physical therapy, and non-steroidal analgesics will relieve signs in the majority of patients. Rest is just that!!!, controlled activity with slow walking on a leash only. There should be NO free activity for 2 weeks. Physical therapy includes cold therapy for the initial 1-4 days. Commercial cold packs are the most convenient and precise way to apply cold therapy. The application of cold should only be 5-10 minutes. The attending veterinarian must emphasize that REST and PT are the most important considerations when treating an acute sprains.

Following the acute phase of treatment, the attending veterinarian must consult with the owner regarding longterm management of the dysplastic dog. The foundation for long term management of any arthritic joint is weight control, exercise therapy, and anti-inflammatory drugs or supplements. The majority of mature dogs with hip discomfort are over weight. Studies have shown a significant improvement in function if an ideal target weight is achieved. The foundation for weight control is exercise therapy, diet, and owner behavior modification. Administration of drugs (NSAIDs, steroids, PSGAGs, Hyaluronate) or supplements (glucosamine, chondroitin sulfate, manganese) are useful to control discomfort. This is particularly true in the early stages of treatment before the benefits of weight reduction and exercise therapy are realized. The administration of drugs should be at a minimum level (dose and frequency) to achieve comfort. Supplements of glucosamine, chondroitin sulfate and manganese alone or in combination have been shown in vitro as well as in clinical studies to ameliorate discomfort or reduce the dose of drugs needed to control discomfort.

Surgical intervention also is divided into techniques useful in the younger population and those useful in mature dogs. Techniques useful in the younger population include Triple Pelvic Osteotomy (TPO), Double Pelvic Osteotomy, femoral head ostectomy, and possibly total hip replacement. My preference in this aged dog is either a TPO or DPO. The advantage of DPO is that the floor of the pelvic canal is stable is that the ischium does not undergo an ostectomy as in a TPO. This concept allows for greater patient comfort and therefore, the ability to perform a bilateral DPO at the same setting. This reduces postoperative rehabilitation time and allows more rapid return to function. Pelvic osteotomy is used in the group of younger patients to axially rotate and lateralize the acetabulum in an effort to increase dorsal coverage of the femoral head. This procedure is indicated in patients that will lead athletic lives such as the working breeds or in those patients in which the client wishes to arrest or slow the progress of osteoarthritis associated with hip dysplasia. The most favorable prognosis is in patients having minimal existing radiographic degenerative changes and an angle of reduction less than 45 degrees and angle of subluxation less than 15 degrees. The prognosis is less favorable in patients with existing degenerative changes and angles of reduction and subluxation greater than those given above. The details of the technique are beyond the scope of this handout. Briefly, the degree of axial rotation of the acetabulum is set by the previously determined angles of reduction and subluxation. The angle of reduction is the maximum degree of rotation and the angle of subluxation is the minimum degree of rotation. The most commonly used angle of acetabular axial rotation is slightly less than the measured angle of reduction. The pelvis is cut through the pubic brim and body of the ilium. The acetabulum is rotated axially, lateralized and stabilized with the appropriate osteotomy plate. The use of locking technology is an advantage that has decreased post operative implant failure. Postoperatively the patient is restricted to exercise on a leash only until radiographic healing of the osteotomies is complete.
In the older dogs, my preference is total hip replacement or conservative management. Femoral head ostectomy is an option in cases where conservative management is no longer effective and financial constraints precludes Total Hip Replacement. Advancement in Total Hip Replacement is the advent of cementless systems. Cementless systems have decreased the incidence of acetabular cup loosening and femoral stem loosening. Hybrid insertion with cementless cup and cemented stem are often used in dogs with “stove pipe”, ie, uniform diameter marrow cavity.

**Diagnosis of the CCL Deficient Stifle**

**Examination:** Perform the initial examination of the stifle with the animal standing. Simultaneously palpate both stifles to detect swelling. A swollen stifle usually indicates degenerative joint disease. The patellar ligament becomes less distinct with joint effusion and the medial aspect of the stifle enlarges because of capsular thickening and osteophyte formation. Palpate the stability of the patella with the hip joint in full extension.
Ask the animal to sit; observe the flexion of the stifle and tarsus. The earliest sign of stifle joint pathology is failure to dorsiflex the tarsus fully (compare to the opposite normal side).

**Imaging:** Early diagnosis is dependent upon radiographic presence of joint effusion. A radiolucent line adjacent to the caudal joint capsule is representative of fatty tissue in the space between the joint capsule and popliteal muscle. Caudal displacement of this line is representative of joint effusion. This is one of the earliest radiographic indications of partial anterior cruciate ligament injury. As changes progress, typical radiographic signs of DJD will be noted.

Stabilization of the CCL deficient joint can be accomplished through a variety of methods. Surgical techniques have been developed including placement of intra-articular grafts, insertion of suture material and/or advancement of periarticular structures outside the joint (extracapsular), and tibial osteotomies that alter joint mechanics. The technique of choice is based on surgeon experience. Tibial plateau leveling techniques are preferred by the author in large athletic breeds, with early partial CCL injury, and in dogs/cats with excessive slope. Recent double blind study showed that in larger breeds of dogs, the Nylon crimp technique was not as effective in all outcome parameters as a leveling osteotomy. A number of reasons why the nylon / crimp technique is ineffective have been elucidated. The placement of the nylon (attachment sites at the femur/tibia) are very non-isometric and predispose to suture elongation/breakage. The nylon material itself undergoes stress relaxation/creep, ie, elongates under continual load. Newer materials (Arthrex FiberWire/Tape) have improved structural/mechanical properties. More isometric sites have been identified; note the ligament is very complex and there is no true one isometric site. Studies in our lab at TAMU have identified near isometric sites. A discussion of the site(s) location and technique of application are presented below. Additionally, a leveling osteotomy technique is described for those who wish to apply this method based upon personal preference/indications.
Recommended sites for isometric suture placement:

**Locating the F2 site:** The F2 site is located at the level of the distal pole of the fabella. Placement of the anchor is critical. The anchor must be placed in the femoral condyle as far distal and as far caudal as is possible. An anchor placed too proximal or anterior is at risk for pull out or suture failure. To locate the correct placement site in the femoral condyle, palpate the distal pole of the fabella. Make a vertical incision through the capsular tissue to expose the joint line between the fabella and caudal margin of the femur. Locate the proper position for the anchor just distal to the fabella-femoral joint line and as far caudal as possible. A hole is pre-drilled with a 2mm drill bit (or 1/16 Steinmann pin) at the correct anchor position. The drill hole is angled directed toward the patella to cranial to eliminate the risk of entering the joint. Insert the appropriate size anchor.

**Locate the T3 site at the proximal tibia.** First locate the protuberances cranial and caudal to the long digital extensor groove. Make a vertical incision through the capsular tissue overlying the extensor groove. Palpate and locate the protuberance just caudal to the extensor groove; this is the site for placement of the first drill hole. At this site beginning as proximal as is possible without entering the joint, insert a .045 k-wire. The K-wire is directed to glide beneath the extensor groove to exit through the medial cortex of the proximal tibia. With the K-wire in
place, place a 2mm cannulated drill bit over the wire to create the first drill hole. Drill over the K-wire to exit through the medial cortex. Leave the drill bit in place and remove the K-wire. Through the cannulated hole in the drill bit, place a nytinol Arthrex suture passer such that the loop is lateral. Remove the drill bit and leave the suture passer in the drill hole.

**Passing the suture through the drill hole:** Place one of the suture ends through the loop in the nytinol suture passer. Only place about 1cm of the suture through the loop to decrease suture drag as it passes though the drill hole. Pull the suture passer medial such that the free end of the suture exits through the medial cortex. Place the free end of the suture through a two hole button such that the button will lie against the medial cortex when the suture is pulled taught. Re-insert the nytinol suture passer through the drill hole such that the loop is positioned medial. Place the free end of the suture through the nytinol loop (1cm of suture end) and pull the suture laterally. Now both free ends of the suture are lateral and ready to be tied.

**Tying the suture:** Place the limb in normal standing position (140 degrees). Place the initial double throw of a surgeons knot and check cranial drawer. Do not over constrain; there should be 2-3mm cranial translation. When satisfied with stability, complete the surgeons knot and place 4 additional half throws. Check range of motion and cranial drawer.

**Knotless Swivelock**

The 5.5mm PEEK SwiveLock is recommended for dogs weighing 50lbs or greater. One strand of 2mm Fibertape (2 limbs) is inserted for dogs up to 70 lbs or so; two strands (4 limbs) of 2mm fiberTape is recommended for dogs greater than 70 lbs.
The F2 and T3 sites described previously are used in this application.

**Concept of CORA Based Leveling Osteotomy**
Recent studies have shown joint mechanical alteration that may be contributory to articular cartilage lesions noted on 2nd look arthroscopy. One explanation for reported abnormal joint mechanics with Tibial Plateau Leveling Osteotomy is that the standard Slocum osteotomy is not based on the anatomic CORA. As such, the Axis of Correction (ACA) is not aligned with the anatomic CORA resulting in mal-alignment of the proximal and distal anatomic axis and secondary translation. The result is caudal displacement of the weight bearing axis and a focal increase in joint force. When rotated to the recommended 5 degrees, the long-term effect is loss of compliance of cranial supporting structures such as the fat pad and joint capsule. Encroachment of the cranial supporting structures (joint capsule) on the cranial articular surface of the medial/lateral femoral condyles can result in abrasion of the articular cartilage.

**Surgical Techniques for Stabilization of the Patella**

*Deepening of the Trochlear Groove.*
If the medial and lateral trochlear ridges do not constrain the patella, the trochlear groove must be deepened. This technique is generally necessary in dogs/cats with a grade III or IV luxation. Deepening the groove may be achieved with a trochlear wedge recession, trochlear block recession, or trochlear resection.
**Trochlear wedge recession** deepens the trochlear groove to restrain the patella and maintains the integrity of the patellofemoral articulation. Make a diamond shaped outline cut into the articular cartilage of the trochlea with a scalpel (use smooth arcs rather than corners on the lateral and medial sides of the diamond). The width of the cut must be sufficient at its' midpoint to accommodate the width of the patella. An osteochondral wedge of bone and cartilage is removed by following the outline previously made. Make the osteotomy so that the two oblique planes that form the free wedge intersect distally at the intercondylar notch and proximally at the dorsal edge of the trochlear articular cartilage. Use caution to avoid making the wedge too long (may affect cruciate ligament insertions) or too deep (may go through the caudal aspect of the femur).

![Trochlear wedge recession images]

**Trochlear block recession** is performed similarly to the wedge recession. Some surgeons find the block recession most appropriate for dogs that seem to luxate primarily with the stifle joint in extension, and for larger breed dogs. The advantage of the block recession is deeper placement of the patella and more advantageous proximal tracking of the patella into the trochlear groove.

![Trochlear block recession images]

**Tibial tuberosity transposition**: Tibial tuberosity or crest transposition is an effective method of treatment for grades II, III, and IV patellar luxations. **Medial release**: The medial joint capsule is thicker than normal and contracted in patients with grade III or Grade IV patella luxations. In this group of animals, the medial joint capsule and retinaculum must be released to allow lateral placement of the patella.
The pull of the sartorius muscles and vastus medialis muscle directs the patella medially, the insertions of these muscles at the proximal patella are released. Redirect the insertions and suture them to the vastus intermedius.

**Lateral reinforcement:** Reinforcement of the lateral retinaculum is accomplished with suture placement and imbrication of the fibrous joint capsule, by placement of a fascia lata graft from the fabella to the parapatellar fibrocartilage, patella sling suture, or excision of redundant retinaculum.

Femoral varus/valgus correction is required in cases where the angulation of the distal femur precludes correct alignment of the extensor mechanism. This abnormal / excessive varus/valgus is present with cases of Grade 4 patella luxation. The method of planning correction is based upon the CORA methodology. CT imaging and reconstruction are the most accurate method of determining correction. In severe cases, prototype development is ideal. Many centers do not have the ability to perform CT/reconstruction and prototype development; accurate radiographic positioning and planning will suffice except in the more severe cases. These cases can be very complex and therefore recommended only for the experienced surgeon.
Basic Principles

A) Fracture fixation and healing is essentially a “race” between failure of the implant system utilized to stabilize the fracture and the organized process of wound healing and fracture repair.

B) Although a thorough knowledge of regional anatomy, meticulous aseptic technique, respectful tissue handling, and understanding of orthopedic implant biomechanics are pre-requisites for successful fracture fixation, the majority of fracture fixation failures occur because of poor decision making which occurs prior to entering the surgical suite.

C) Why are orthopedists prone to poor decision making when treating fractures?

1. Radiographs are the standard of care diagnostic tool for most fracture patients. This imaging modality is mandatory for proper diagnosis and classification of fractures. Moreover, radiographic interpretation partially (note I did not state completely) dictates the fixation system that can be selected for fracture repair. Thus, early in the course of treating a patient for a fracture, the orthopedist begins to think in the terms of the affected bone as it is represented on a radiograph, and not of the injured bone as part of an injured patient.

2. The problem with this mindset is that focusing solely on the fractured bone often leads the orthopedist to ignore the supporting tissues around that bone and the clinical factors of the patient. Although is commonly said that orthopedists are nothing more than “frustrated carpenters”, a fractured bone is not just an inanimate biomaterial desperately in need of plates, pins, and screws. An injured bone should be thought of as an injured organ consisting of a variety of hard and soft tissues that need to be properly treated for timely healing to occur. Without a healthy, robust supporting soft tissue envelope, a fractured bone will not heal. Adequate blood supply, intact musculotendinous attachments, and patient comfort leading to early return of limb use are all mandatory for fracture healing and a clinically successful outcome. Failure to: 1) assess the health of these soft tissues, 2) respect them during surgical approach to the fracture, and 3) protect them during implant placement WILL RESULT in inadequate fracture healing and subsequent failure of the applied fixation device.

3. The third element often resulting in poor fracture decision-making involves failure to consider the veterinary-client-patient relationship. The veterinary orthopedist can make the most accurate assessment and fixation plan for an injured bone and its surrounding soft tissues, but if he/she fails to consider the client that will be caring for the injured veterinary patient, the chances for post-operative complications and subsequent failure of the fracture repair is markedly increased.
D) **Thus, the basic principles for proper fracture decision-making are to consider three factors independently for every patient: mechanical factors, biologic factors, and clinical factors (client constraints).** In fact, a numerical score, referred to as **a Fracture Assessment Score (FAS)**, should be generated for each of these 3 factors. After scoring each factor, an overall “average” score is then generated for the patient. This patient’s FAS is then used to guide choice of fixation systems.

2) **More on the Fracture-Assessment Score: Why is it important?**

A) Early, progressive consolidation of a fracture during the post-operative period causes a shift in relative load bearing toward the healing bone and away from orthopedic implants. Conversely, slower fracture healing dictates that the fracture fixation system provides stability longer, which increases the likelihood of implant-related complications. Thus, each fracture repair truly represents a “race” between implant failure and fracture healing. As stated above, poor decision-making occurs for a variety of reasons, such as observation of the fracture on radiographs or comparing how a fractured bone appears in relation to a surgical textbook diagram. Recurrent fixation failure due to poor decision-making leads surgeons (and often their clients!) to become frustrated with orthopedic case management. This frustration often occurs because of poor decision-making, and not because of the surgeon’s abilities.

3) **The fracture-assessment score (FAS) ranges from 1 to 10.** The lower end of the scale represents mechanical, biologic, and clinical factors that do not favor rapid bone union and return to function, while the upper end of the scale represents those factors that favor rapid bone union and return to function.

A) **Specific Factors:**

1. **Mechanical Factors**

   (1) **Number of limbs injured:**

   (a) Dogs and cats must bear weight on 3 limbs; weight bearing on the implant-bone construct cannot be prevented post-op when multiple limbs are injured or when pre-existing conditions exist (severe OA secondary to chronic CrCL rupture, limb missing from previous amputation).

   (2) **Patient size and activity:**

   (a) Complication rate is directly related to the stresses applied to implants during the post-operative recovery period. Excessively loaded implants have an increased rate of failure. Large/active patients subject implants to greater loads and are more prone to have implants loosen prematurely and/or fail.

   (3) **Ability to achieve load-sharing fixation between the bone and the implant:**

   (a) Ideal implant-bone load sharing occurs when a transverse or short oblique fracture is repaired using **DIRECT REDUCTION TECHNIQUES**, because restoration of the bone column allows loads
to be transmitted axially through the limb (and not solely upon the implant). In these situations loading of the implant is minimized and implant loosening/failure is less likely.

(b) Conversely, implant-bone load sharing does not occur when **INDIRECT REDUCTION TECHNIQUES** are utilized, because loads are transmitted from bone segment to bone segment COMPLETELY through the selected implants [as in a highly comminuted fracture that cannot (and should not) be anatomically reconstructed].

(i) **This does not mean that indirect reduction is inferior to direct reduction!** In fact, indirect reduction techniques have a number of advantages over direct reduction techniques. However, if indirect reduction is selected, the implant(s) must be properly sized and positioned to absorb loads throughout the healing process.

2. **Biologic Factors**
   
   (1) **Patient age:**
   
   (a) Young dogs and cats (< 6 months) are considered “healing machines” and require less robust fixation devices and these devices are typically maintained for a reduced amount of time. Conversely, geriatric patients demonstrate delayed healing, which subjects implants to increased number of cycles and an increased incidence of implant failure.

   (2) **Open vs. closed fracture:**
   
   (a) Open fractures are associated with a significant degree of soft tissue injury, resulting in longer time to bone union because soft tissues must heal first. Closed fractures tend to have less soft tissue injury and in general heal more quickly.

   (b) In addition, open fractures are often not ideal candidates for fixation systems that will remain with the patient long-term (bone plates, interlocking nail, cerclage wire), as the application of large, non-absorbable metallic implants into a contaminated environment may result in complications such as implant infection, incisonal dehiscence, long-term draining tracks, and implant failure.

   (3) **Low vs. high energy injury:**
   
   (a) Low energy fractures (fall from the bed, stepped on by client) have less soft tissue injury when compared to high-energy fractures (gunshot wounds, high-speed hit by car), which are often comminuted in nature. ***One way to think about “energy” is to consider the fracture configuration.*** Simple, two-piece fractures are typically low energy, whereas highly comminuted fractures are high-energy. If a high-energy injury was applied to the bone, that same level of injury traumatized the surrounding soft tissues.

   (4) **Choice of open vs. closed reduction:**
(a) Open reduction techniques, by their nature, damage supporting soft tissues. Conversely, closed reduction techniques such as external coaptation or closed application of an external skeletal fixator or IM pin result in minimal iatrogenic soft tissue injury. **One of the MOST POWERFUL biologic influences is the surgeon’s skill in minimizing soft tissue damage during open reduction.** Obtaining the desired fracture fragment reduction and fracture stability while causing minimal soft tissue injury in the shortest possible operative time results in the greatest success. Thus **preservation of soft tissues during open approaches is of the UTMOST importance.**

(b) This concept has led to a number of more “biologically friendly” fracture fixation techniques referred to as bridging osteosynthesis, or biologic osteosynthesis. Examples of these techniques include:

(i) Open-but-don’t-touch (OBDT) - use of a standard open approach without disturbing the fracture hematoma or the fracture fragments

(ii) Mini-approach to the fracture for reduction with closed application of external skeletal fixation – mini-approach utilized to visualize fracture and achieve reduction, but fixation system applied in a closed manner.

(iii) Minimally invasive plate osteosynthesis (MIPO) - creating small “portals” over parent bone fragments for placement of implants, followed by sliding the implants from one portal to the other beneath an intact segment of skin and soft tissue.

(5) **Amount of surrounding soft tissue:**

(a) Bones with little surrounding soft tissues, such as the radius/ulna and distal tibia result in delayed unions and complications more often than similar fractures affecting the humerus, femur, or pelvis. ***The poster-child for this phenomenon in small animal orthopedics is the distal radius/ulna fracture in toy and small breed dogs. Failure to recognize this patient population prior to initiation of treatment WILL RESULT in delayed or non-unions and a number of severe complications.***

(6) **Location on the bone:**

(a) Fractures of the diaphyseal bone heal more slowly and are inherently less stable after reduction because of the reduced blood supply and limited surface area of fracture ends. Fractures involving the metaphysis of bone heal more quickly because of improved blood supply and greater surface area for contact between fracture ends.

(7) **Concurrent diseases or medications:**

(a) Diseases that delay healing must be considered when choosing fixation systems

(i) Hypothyroidism

(ii) Hyperadrenocorticism

(iii) Iatrogenic corticosteroid or chemotherapy treatment

(b) Conditions that increase wound infection rates must also be considered when selecting internal vs. external fixation systems.
(i) Hyperadrenocorticism
(ii) Iatrogenic corticosteroid or chemotherapy
(iii) Diabetes Mellitus (little evidence to support this as a risk factor in veterinary orthopedic patients assuming the patient is well regulated)

3. **Clinical Factors**
   
   (1) **Client’s willingness/ability to participate in aftercare:**
   
   (a) A client who works 80-100 hours/week and commutes 2 hours a day is probably not the best client to task with daily external fixation pin-tract care or weekly re-check visits, whereas a retired individual or someone who works from home may be well suited for this added time commitment.

   (2) **“The bigger they are, the harder they fall!”**
   
   (a) Assuming your client has the intestinal fortitude to provide certain post-operative treatments is fraught with danger. Some of the toughest looking clients end up on the exam room floor the first time they see an external fixator attached to their beloved pet, while meek/timid clients often handle this situation well. Be certain to counsel each client on the proposed fixation system PRIOR to fracture repair.

   (3) **Compliance with post-operative exercise restriction:**
   
   (a) A client who is unlikely to confine the patient post-operatively often requires selection of more robust implants in order to cope with the increased load and cycling that will be placed on these implants if confinement instructions are not followed. Additionally, dogs or cats with clients that are unlikely to comply with post-op instructions should be treated with internal fixation systems, as external fixators and casts/splints often lead to complications in this scenario.

   (4) **Patient’s ability to cope with discomfort:**
   
   (a) Each individual patient’s “stoic nature” must be closely considered. For example, a patient from a stoic breed such as a Staffordshire terrier will often ignore external skeletal fixation devices and walk on the injured limb as if it was un-injured. Application of a similar device to a patient from a “wimpy” breed such as a Toy Poodle or Maltese will result in complete refusal to use the limb. Refusal to bear weight on the injured limb results in delayed union, disuse osteopenia, and non-union or stress-protection related fractures.

4) **Put it all together: The patient’s overall Fracture-Assessment Score (FAS)**

   A) As stated, mechanical, biologic, and clinical factors should be individually considered and scored. These 3 scores are then combined, or averaged, to provide a whole patient FAS.

   1. **Low Scores (0-3)**
      
      (1) In this category, implants often bridge large fracture gaps and therefore must have sufficient strength to prevent permanent bending or breakage. Implants must also deter excessive motion at the implant-bone interface.
Motion at this interface will cause bone resorption, premature implant loosening, and implant migration. Implants should purchase bone with raised threads (bone screws utilized to secure bone plates, positive profile transfixation pins used with external skeletal fixation (ESF).

2. **Suggested implants** include lengthening plates, bone plate/IM rod combinations, bone plate/ESF combinations, type II or III ESF, ESF/IM pin combinations, and interlocking nail fixation.

2. **Intermediate Scores (4-7)**
   1. Overlapping biologic and mechanical factors affect healing and implant selection in this category. If the implant and bone share the load following surgery, the implant will be subjected to less stress; however, healing may still be delayed due to a poor biologic score. Alternatively, biologic score may be excellent, but the implant may be subjected to high initial loads because it is buttressing (bridging) the fracture. A score toward low-center means that time to union will be long and thus the implant must purchase the bone in a way that will preserve implant-bone interface. A score toward high-center means that the stress on the implant and implant-bone interface will be high for a short period of time, thus the implant-bone interface needs maximum stability until callus formation occurs (which reduces implant stress).
   2. **Suggested implants** include bone plates, Type I or II ESF, IM pin/ESF combinations, interlocking nail fixation, and rarely IM pin/cerclage wire combinations.

3. **High Scores (8-10)**
   1. When the FAS is greater than 7, immediate load sharing between the bone-implant construct and rapid bone union is expected. Therefore the strength and stiffness of the fixation system need not be extreme, nor does the implant need to function for a long period of time.
   2. **Suggested implants** include type I ESF, IM pin/cerclage, and external coaptation. Implants that hold bone through frictional purchase (smooth tipped pin, cerclage wire) often provide adequate bone purchase.

5) **Concluding remarks: Treat the patient, and not the radiograph!**
   A) In conclusion, successful fracture fixation requires a thorough knowledge of regional anatomy, meticulous aseptic technique, respectful tissue handling, and understanding of orthopedic implant biomechanics. However, the single most important factor in successful fracture treatment is proper decision making PRIOR to incision of skin. This is best summarized by the phrase “treat the patient, not the radiograph”. After careful consideration of mechanical, biologic, and clinical factors, a patient FAS is generated. The FAS is designed to properly match the selected fracture fixation system with each patient. By considering all of the factors that result in the FAS, proper selection of fixation systems and their method of application will dramatically reduce post-operative complications and fracture fixation failure.
Intra-medullary Pins & Ancillary Stabilization Devices

Introduction
Intramedullary (IM) pins and cerclage wires are relatively easy to apply, do not require expensive equipment and, when used appropriately, can be very useful for managing many types of basic fractures in dogs and cats. IM pins and cerclage wires have gained a bad reputation, as they can be disastrous when used inappropriately. A fundamental understanding of fracture biomechanics, implant biomechanics, and fracture decision making are necessary in order for the surgeon to determine whether or not pins and wires are appropriate for individual patients.

IM Pins
Intramedullary pins (IM pins) used in animals range from ¼ inch diameter (6.3 mm) down to 5/64 inch diameter (2.0 mm). Intramedullary pins in this size range are called Steinmann pins. Smaller pins are usually referred to as Kirschner wires (K-wires), and although they may be used as intramedullary devices in very tiny animals, they are generally used as inter-fragmentary devices. K-wires are available in .035, 0.045, .054 and .062 inch diameters. Intramedullary pins and K-wires can be obtained as fully threaded, partially threaded or nonthreaded. Although partially (end) threaded pins are used as intramedullary pins with the intention of increasing rotational stability, those pins do not provide additional stability and are at risk for breakage at the thread-shaft interface. In addition, partially threaded Steinmann pins and K-wires are more difficult to remove as the bone tends to grow into the threads. For these reasons, the use of threaded pins as intramedullary devices is not recommended.

Steinmann pins and Kirschner wires are available in a variety of lengths, usually from 6-12 inches long, and may have points on one or both ends. The pins are easily cut, and there is no advantage to the veterinary surgeon in purchasing single pointed pins. Pins may be manufactured with a trocar or chisel/diamond point. Trochar points are by far the most commonly used and consists of a three-sided tip with a long bevel and good ability to penetrate cortical bone. Chisel points (also called diamond points) are broad, flat two-sided points with a short bevel and are designed to deflect the pin away from the cortex during drilling rather than engage the opposite cortex.

Intramedullary pins excel in resisting bending forces in 360 degrees, can be placed without specialized equipment, and often can be placed with a limited approach. Intramedullary pin placement, unless a very large pin is placed or reaming of the medullary cavity is performed, has limited impact on the intramedullary blood supply. Intramedullary pins are relatively easy to remove, in contrast to fixation devices such as screws, plates, or interlocking nails. Intramedullary pins do not resist rotation or axial (compression/tension) forces, and therefore are rarely used alone but combined with other types of fixation, for example cerclage wires, external fixators, or bone plates. From a mechanical standpoint, use of the largest pin possible will result in the stiffest construct and most resistance to bending. However, use of an excessively large pin has several disadvantages: difficult placement in a curved bone, for example the canine tibia and femur, damage to the intramedullary blood supply, and risk of creating additional fractures if the pin exceeds the diameter of the bone at its’ narrowest point, or isthmus. In general, a pin that is approximately 70% of the diameter of the long bone at the isthmus is chosen. If the surgeon anticipates combining the pin with another type of device, a
slightly smaller intramedullary pin is chosen. Use of a pin that is too small may result in failure by pin bending or breakage. Use of multiple small pins to fill the medullary cavity, also called “stack pinning” to increase resistance to rotational stability, has been shown to have no effect on rotational stability when compared to a single IM pin.

K-wires, while they can be used as intramedullary devices, are usually used as interfragmentary devices, often to maintain temporary fracture reduction while the primary fixation is applied. K-wires by themselves are relatively weak implants and are typically not used alone. In certain fractures, for example physeal fractures in small dogs and cats, cross-pinning with K-wires or small Steinmann pins can be sufficient when fracture healing is expected to be rapid. K-wires are also commonly utilized in combination with cerclage wire for tension-band fixations and to support full cerclage wires in areas of changing bone diameter (“skewer wires”).

**Application Techniques for Intramedullary Pins and Interfragmentary Wires**

Intramedullary pins may be inserted either from the fracture site (retrograde insertion) or from either the proximal or distal end of the bone itself (normograde insertion). The local anatomy of the bone often dictates how the pin is driven, for example, retrograde pin insertion in the tibia from a distal to proximal manner may result in damage to the articular cartilage or cruciate ligaments. An estimation of appropriate pin size (60-75%) may be made from the pre-operative radiographs and may be confirmed by observation of the pin as it is gently introduced into the fracture site if the fracture is treated with an open approach. When in doubt regarding pin diameter, a smaller pin should be used initially and replaced with a larger pin if necessary. The pin may be inserted either open or closed. Although closed pinning, based on palpation, can be performed by the experienced surgeon this can become more difficult in larger animals with soft tissue swelling, or in fractures greater than 72 hours old. The increased use of intraoperative imaging (fluoroscopy) can greatly facilitate IM pin placement in a minimally invasive fashion, with less damage to the soft tissues.

Intramedullary pins may be placed either by hand, using a Jacobs’ chuck, or with a drill. **Hand insertion** may allow the surgeon to feel whether or not the pin is advancing down the medullary cavity and whether it is up against or about to penetrate cortical bone. When placing pins by hand with a Jacobs’ chuck, the chuck should be “choked-up” as far as possible on the pin in order to prevent pin bending during cortical penetration, particularly when smaller pins are used. The chuck should be firmly tightened with the key in at least two separate places to avoid sudden loosening during pin advancement. Jacobs’ chucks are sold with protective devices that are designed to protect the surgeon’s hand from inadvertent pin penetration resulting from sudden pin loosening. It is strongly recommended that this protective device be utilized early in the learning curve. The tip of the pin should firmly engage bone, and the pin rotated back and forth with quarter turns (rather than driven consistently clockwise or counterclockwise). In larger animals with hard cortical bone, hand placement can be very difficult. In addition, the smaller the pin the more difficult it is to drill by hand, thus power insertion is suggested for interfragmentary K-wires.

When using a drill to insert an intramedullary pin or K-wire, a cannulated drill should be used to drive the pin to decrease the amount of pin bending or breakage during drilling. Ideally a high-torque, low speed drill should be used (as opposed to a high speed drill) to decrease heat
generation and subsequent bone necrosis. Saline lavage is also suggested during pin placement to decrease heating of the bone, particularly with K-wire insertion. **When driving an intramedullary pin, whether by hand or with a drill, it is critical to align the pin with the shaft of the bone in both craniocaudal and mediolateral planes.** With open reduction techniques, the surgeon may find it easiest to drive the pin with one hand and stabilize the fracture segment using a bone-holding forceps in the non-dominant hand. Having an assistant hold a second pin parallel to the shaft of the long bone may be helpful, or in certain cases, placing an “aiming pin” retrograde just a few cm into the medullary cavity so the surgeon can attempt to drive the normograde pin along the axis provided by the aiming pin. When attempting to seat an IM pin into metaphyseal bone in the distal fracture fragment, it is important not to penetrate the subchondral bone plate and cartilage. This untoward event is particularly common when placing IM pins in the femur using normograde technique. The surgeon will note an increase in resistance as the pin begins to seat into the metaphysis. In addition, the fracture fragments may begin to distract apart as the pin over-lengthens the bone as it is driven into the metaphysis. In comminuted fractures, an IM pin is an excellent tool to achieve fracture distraction and alignment in both craniocaudal and mediolateral planes. When using IM pins for this purpose, the tip of the pin should be cut as the sharp tip of the pin passes across the fracture. This will allow improved distraction of fracture fragments and prevent penetration of the joint. The distance that the pin is advanced may be judged by using a second pin of equal length and lining the two pins up after the Jacobs chuck or drill has been removed. After the pin has been seated, the proximal and distal joints should be put through a complete range of motion, as it is easy to inadvertently place a pin into a joint. Immediately prior to wound closure, the surgeon should carefully palpate the soft tissues surrounding the bone for evidence of overly long or misplaced pins, which can be difficult to feel as they unexpectedly exit cortical bone during drilling.

After the pin is determined to be in the correct position, it is cut using specialized pin cutters. Pin cutters are generally designed only to cut pins of a certain range in diameter, and inadvertent use of small pin cutters to cut a large pin may result in permanent damage to an expensive piece of equipment. The surgeon should check the range of diameters listed on the side of the cutter. It is helpful to have an autoclavable pin guide in the pack to determine the size of the pin in surgery. Pins accumulate and release substantial energy when cut and have the potential to cause serious injury to the surgeon, assistant or circulating technicians if the free end is not firmly grasped or covered with a sponge or surgical towel when cut. The fracture should be carefully observed during and after pin cutting to make sure that alignment is not disrupted.

The decision to cut a pin flush with the surface of a bone or leave the pin long for subsequent implant removal is a decision that varies among surgeons. Leaving pins long can lead to problems with iatrogenic nerve damage (proximal femur), damage to nearby articular cartilage or the patellar tendon (tibia), or soft tissue irritation with subsequent seroma formation or erosion of the tip of the pin through the soft tissues. The alternative to leaving pins long is to cut them short and countersink them, using a countersink and mallet. If countersinking is performed, the pin should be retracted approximately 1 cm, cut as short as possible, and then gently driven back into the bone with a mallet and countersink.

**Special Consideration: Cross Pinning Technique**

Cross pinning is a technique commonly employed to treat two-piece Salter-Harris (S-H) fractures
Comminuted fractures or distal long-bone fractures in middle-aged or elderly dogs and cats should not be treated with cross pinning. The most common S-H fracture amenable to cross pinning is the distal femoral S-H fracture. However, S-H fractures of the distal humerus, distal radius, and proximal tibia may also be successfully cross-pinned in select cases. Cross pinning simply denotes the use of two or more Steinman pins for stabilization of a fracture. In the case of a distal femoral fracture, the most challenging aspect of this repair is fracture reduction, owing to the small distal fracture fragment and the caudal and proximal displacement of the distal fragment. Most distal femoral fractures are successfully cross pinned via a standard lateral approach to the stifle and distal femur, but combination approaches and tibial tuberosity osteotomy approaches are occasionally employed. After lateral arthrotomy, the distal fracture fragment is identified and carefully grasped with clamshell or point-to-point bone holding forceps. Care must be taken not to injure this small fragment or the articular cartilage with forceps.

A small Hohmann retractor, periosteal elevator, or un-loaded scalpel handle can be used to lever the distal fragment into reduction. Placing the stifle in full extension facilitates this maneuver. The tendency is to partially reduce the fracture without correcting excessive pro-curvatum (femoral condyles rotated caudally). The proximal fracture lines must be identified and anatomically reduced to prevent this problem, and ensure adequate stability and proper limb use after surgery. The distal femoral physis has a “W” shape when viewed on a lateral radiograph. Anatomic reduction of the fracture results in the physis providing substantial stability to the repair. Once the fracture is reduced, Steinmann pins are driven in a distal to proximal direction across the fracture. Pins should be as large as possible without risking iatrogenic fracture. It is critical for the pins to cross each other proximal to the fracture plane, as this provides maximal implant stability. The lateral pin is started just cranial to the long digital extensor tendon sulcus, aimed and driven proximally and medially toward the medial femoral cortex. The pin is driven until the tip begins to exit the medial cortex. A second pin is started on the medial femoral condyle at the same level as the lateral pin, and aimed proximally and laterally to exit the lateral femoral cortex. The pin is driven until the tip just begins to exit the lateral cortex. Additional pins may be required in challenging cases. Stifle range of motion and fracture reduction are evaluated. Pins are backed out 5-10 mm, cut as flush to the bone as possible, then driven below the surface of the articular cartilage with a mallet and countersink. The ends of the pins must be at or below the level of the cartilage. Routine closure is performed, followed by post-operative radiography. Excessively long pin tips protruding proximally may warrant a return trip to the OR, as these pins are often impinging the quadriceps and cause substantial pain and joint immobility. Instead of opening the primary incision, a mini-approach to the offending pin is often possible and the pin is cut flush with the bone to eliminate post-operative muscle impingement. If possible, a 90-90 flexion bandage, maintained for 2-3 days after surgery, should be considered to maximize stifle range of motion.

Cerclage Wire
Cerclage refers to a wire used to encircle a bone. Cerclage wire typically is used to provide interfragmentary compression in a manner similar to that of interfragmentary screws, and is useful in situations where space is limited and screw application would be difficult, or when screws of the correct type or size are not available. Properly applied cerclage wires do not interfere with the blood supply to bone, and may be used in the immature as well as the mature animal. Types of cerclage wire application include full cerclage, where the wire completely encircles a complete cylinder of bone, and hemicerclage, where wire is passed through a hole or
holes drilled through bone (we will focus on full cerclage wire).

For the vast majority of fractures, cerclage wire is used as adjunctive, rather than primary, fixation. The surgeon should keep in mind that while properly applied cerclage wire is effective when used in appropriate cases, errors in application or case selection can be disastrous. **Cerclage wire is typically applied to long spiral or oblique fractures where the length of the fracture is roughly two and a half to three times the diameter of the bone, and a single cerclage wire should be avoided as it acts as a stress concentrator and become a fulcrum for motion of the fracture fragments.** Cerclage wire may be used both as a temporary reduction device, for example, to hold the fracture in reduction while applying a plate, external skeletal fixator or interlocking nail, or as a permanent device, often in combination with one of the above or with an intramedullary pin. **An exception to the single cerclage wire rule for long bones may be made if the intent is to prevent a non-displaced fissure fracture from propagating or fracturing further during manipulation of the fracture ends during open reduction.**

Cerclage wire is made of relatively soft (usually annealed) 316L stainless steel that is available on spools, in coils or as preformed loops. Sizes of cerclage wire typically used in cats and dogs include 16 to 22 gauge. The larger number denotes a smaller diameter wire. Wire diameter can exponentially increase load to failure (a 50% increase in diameter may increase load to failure by up to 169%), and the largest diameter wire that the surgeon can apply to the bone without technical difficulty is recommended. Method of wire application and type of knot have been extensively described in both the human and veterinary literature. When reviewing the literature, one should be aware that some studies are designed to evaluate cerclage wire used for spinal or tension band applications rather than for long bone applications. The results of spinal stabilization or tension band studies should not be directly applied to long-bone stabilization.

**Cerclage Wire Knots and Wire Tensioners**
Cerclage wire in veterinary surgery is generally applied either as a twist wire or loop wire (single or double loop). Clinical advantages of twist wires include ease of application with a wire twister (a large instrument that resembles a needle holder but is designed to grasp pins and wire), the ability to tighten and fasten the wire at the same time, and the ability to retighten the wire if it loosens during fracture reduction, as frequently occurs during the placement of multiple cerclage wires. Loop wires have the advantage of better initial tension or tightness when properly applied, and do not have a protruding twisted end that may irritate soft tissues. Loop wires that loosen during fracture reduction must be removed and replaced. There are many types of wire tensioners available or reported in the literature. They fall into categories of twist tensioners, loop tensioners and there are instruments available that can actually tie a square knot in stainless steel cerclage wire. We will focus on a wire tensioner that is used for placement of single or double loop cerclage wires.

**Application of Full Cerclage Wire**
For fixation of a long bone fracture, cerclage wire should **ONLY** be used where the fracture can be anatomically reconstructed to complete the original, 360 degree cylinder of bone. Obeying this rule will do more to limit fracture complications than any other rule or guideline. Although cerclage wires are capable of compressing bone fragments, they cannot maintain fragment position in the presence of a cortical defect. Thus, if a tiny piece of bone is missing, the fracture
will collapse as the wire is tightened resulting in a loose wire and loss of fracture reduction. In general, cerclage is reserved for two-piece long oblique or spiral fractures, although occasionally a third piece may be successfully incorporated, but only if the 3rd fragment is a large butterfly fragment with each arm of the fragment spanning two times the diameter of the bone. It is important to minimize dissection and soft tissue trauma to the musculature attached to the bone while applying cerclage wire. Cerclage wire may be passed around the bone either directly or using an instrument such as a cerclage wire passer. Soft tissue inadvertently trapped under the wire will undergo necrosis, subsequently leading to wire loosening. However, there is no need to attempt to place the wires sub-periosteally. Wires are generally spaced at least one to one-half bone diameter’s apart from each other, and should always be placed 3 wire diameters away from the proximal or distal most aspects of the fracture fragments.

After the cerclage wire has been carefully passed around the diaphysis of the bone and the fracture reduced, it must be tightened while maintaining reduction. The easiest way to maintain reduction is to place two reduction forceps at right angles to the fracture planes so that both hands may be used for wire placement. For twist wire application, the wire may be twisted by hand for the first one or two twists loosely, leaving about 0.5 to 1 cm between the bone and the twist. A wire twister should be used for applying cerclage wire, as use of an ordinary pair of pliers allows loss of tension as the wire is being twisted. Both wire ends are grasped with the twister where they intersect, and the wire pulled firmly away from the bone while at the same time twisting. This prevents one end of the wire from wrapping around the other, which drastically weakens the wire. The wire MUST be tight! The surgeon should watch as the gap between the wire and the bone disappears, and should periodically check the wire for looseness by pushing firmly on it with an instrument. If the wire is loose, tightening should continue. With practice, the operator will develop a “feel” for the mechanics of stainless steel cerclage wire. If you are an inexperienced surgeon and have not broken a few wires during the learning process, you are likely not tightening your cerclage wires appropriately. If the wire breaks between the 2nd and 3rd twist or higher and is tight, it may be left in place, otherwise, it is removed. The wire should be left without cutting or otherwise manipulating the ends until all cerclage wires have been placed, and then checked again for tightness. Care should be taken not to damage the length of wire that is going to stay in the patient, as even a small notch or defect will greatly decrease the fatigue resistance of the wire. Loose wires should either be retightened or removed and replaced. If the wires were placed for temporary fixation, for example to hold the fracture in reduction while applying a bone plate, they may be removed prior to final tightening of the plate screws. Just prior to closure, twist wires should be cut to preserve at least 2-3 twists. It has been shown that wiggling the end of the wire during cutting can substantially decrease the tension in a twist wire. In addition, wire ends should not be bent over with twist wires when they are utilized as full cerclage (as opposed to the pin and tension band technique).

Special Consideration: Pin and Tension Band Technique

The pin and tension band technique is uniquely suited to stabilize fractures that receive a distraction force (a muscle or tendon pulling one fragment away from the parent bone) during recovery. The most common fractures that experience this environment are tibia tuberosity fractures, olecranon fractures, greater trochanter fractures, and medial/lateral malleolar fractures of the distal tibia/fibula. While pin and tension band fixation is commonly employed to stabilize surgically induced osteotomies (anconeal process to access the shoulder, olecranon to access the
elbow, greater trochanter to access the hip, acetabulum, ischium), this fixation system is extremely effective for traumatic avulsion fractures of these same bones.

Proper pin and tension band fixation involves reduction of the fracture fragments, temporary stabilization with reduction forceps if possible, and placement of two K-wires or small IM pins across the fracture. The pins should be driven as perpendicular to the fracture plane as possible, and should be started at approximately the same level in the proximodistal direction. The pins are driven toward the opposite cortex until the tip of each pin just begins to exit the far cortex. A hole is next created in a caudocranial direction distal to the fracture. The hole should be 2-3 wire diameters away from the periosteum/cortex, as well as equidistant from the fracture when compared to the pins. A strand of cerclage wire with a small pre-formed loop is next passed through the tunnel, and wrapped around the ends of the IM pins in a figure-of-eight manner. The two free arms of the cerclage wire are connected and a standard wire twist is initiated. This results in a standard wire twist on one side of the figure-of-eight wire, and a second loop within the wire that is used to tension the other side of the figure-of-eight wire. Wire twisters are used to tighten each side of the tension band. Unlike long-bone cerclage wires, the ends of these wires can be gently bent flush with the bone so as not to irritate nearby tendons, muscles, and nerves. The ends of the pins are cut leaving 1-2 cm of pins. A pin-bender is next used to bend the ends of the pins in a proximal direction, and the ends of the pins are cut again so that approximately 5-8 mm of each pin remains. This final bending maneuver prevents the tension band from slipping off of the pins during the post-op period. After correct pin and tension band application, a distraction load on the avulsed bone fragment tensions the figure of eight wire, which effectively resists the distraction force. When performing pin and tension band fixation, it is important to pins and wire of appropriate size. The most common causes of pin and tension band failure are small/improperly placed pins, small/improperly tightened wire, or placement of the transverse bone tunnel too close to the periosteum/cortex of the bone, resulting in wire pull through during the recovery period.

When performing pin and tension band fixation on the tibia tuberosity, it is important to begin both pins at or above the proximal aspect of the tibia tuberosity, and aim pins toward the caudal and medial aspect of the tibia. This ensures maximal bone purchase and reduces the incidence of pin-induced fracture of the tibia tuberosity.

Contraindications of Cerclage
Cerclage wires are contraindicated in the treatment of transverse, short oblique, segmental or comminuted fractures. When evaluating pre-operative radiographs of fractures, all of the fragments, even tiny ones, should be counted and if there are more than two fragments, another method of fixation should ideally be considered. Again, the exception is a three-piece fracture in which the third fragment is a large butterfly fragment. Cerclage wires are also contraindicated if, for any reason, the full 360 shaft cannot be re-constructed, or the shape of the bone is such that they cannot be applied so that they will remain perpendicular to the long axis of the bone without slipping. Loose or damaged cerclage wires should always be removed. Finally, the surgeon must consider the risk of additional damage to the blood supply when considering cerclage wire in high-velocity, open or infected fractures. IM pin and cerclage wire fixation is contraindicated for these types of cases.
Complications and their Prevention

Properly applied cerclage wires rarely cause problems, however, *improperly applied wires almost always cause problems*. The most common complication of cerclage wiring is the development of loose wires during recovery. Loose wires occur either as a result of failure to completely reduce the fracture, improper tightening techniques, or entrapment and subsequent necrosis of soft tissues. In the author’s experience, cerclage wire complications and failures most commonly occur when utilized inappropriately on short-oblique or multi-fragmented fractures. The most common scenario is the selection of IM pin and cerclage wire stabilization for transverse, short oblique, or comminuted fractures in medium to large breed dogs. Lastly, loose wires prevent revascularization of the fracture, and sequestration of non-viable bone fragments often results. This event dramatically increases the risk for post-operative osteomyelitis, which results in lameness, swelling, and incisional draining or development of draining tracts after surgery.

Conclusion

Prevention of complications with IM pins and cerclage wire requires excellent pre-operative fracture decision making, meticulous surgical technique, and appropriate post-operative exercise restriction for the patient. A failed pin and cerclage wire fixation can be devastating for the patient - in some cases irreparable, even if referred to a specialist with access to a variety of implants and techniques. Practice using plastic bones, broom handles, and cadaver limbs is strongly suggested for the inexperienced surgeon in order to gain experience and confidence with this technique.

Bone plates have been used effectively for treatment of fractures in dogs and cats for over 30 years. Over the years, different designs of plates have become available, including DCP (dynamic compression plates), LC-DCP (limited contact dynamic compression plates) and special situation plates (veterinary cuttable plate, acetabular plates). The most recent plate design to become available is the locking compression plate. The unique feature of the locking plate is the presence of a threaded plate hole that couples with a threaded screw head or alternate locking mechanism. A number of companies have developed locking plates. One of the most recognizable locking plates is the Synthes LCP.

The Synthes LCP features a combi hole; this is is a plate hole through which the surgeon can apply compression using a standard cortical screw or apply a locking screw. Using a locking screw, threads in the head of the screw engage threads in the plate hole, locking the screw to the plate. The ability to lock the screw to the plate increases the stiffness of the construct and the pull out strength of the bone plate and screws. Standard plates do not have threaded holes; stability is achieved through compression applied between the plate and bone surface when tightening the screws. The friction between the plate and the bone provides the stability to the bone-implant construct. In contrast, the locking plate achieves stability through the concept of a fixed-angle construct. The locking plate is not pressed firmly against
the bone as the screws are tightened. The locking screws and plate function more like an external fixator. The plate functions as a connecting bar and the screw functions as a threaded fixator pin. The threads in the head of the locking screw engage the hole of the plate, similar to the clamp of an external fixator.

**EQUIPMENT AND APPLICATION**

Many of the same instruments used for application of traditional plates are used for locking plates. The 3.5 mm locking plate and screws are most commonly used in small animals. The 3.5 mm locking plate will accommodate 3.5 mm cortical screw, 4.0 mm cancellous screws or 3.5 mm locking screws. A 2.8 mm drill bit is used when applying 3.5 mm locking screws. A guide is screwed into the intended hole to center the hole when drilling. The locking screw is self-tapping and is placed with a hand or power driver.

![TraumaVet system](image)

TraumaVet system is a locking system for internal fixation. It consists of a steel support, with threaded holes where threaded inserts can be secured. The inserts are externally threaded and are to be screwed into the support. The inner hole of the insert is conical to lodge the head of the screw. The support together with the inserts forms the plate. The screw is a self-tapping with angular stability. The screw head is conical, coupling with the insert to achieve the locking mechanism.

The unique conical screw-plate locking system ensures the distribution of force throughout the structure, virtually eliminating the risk of implant breakage and screws backing out.

The locking system allows easy locking of the screw in the plate and eliminates the possibility of cross-threading the screw head in the plate.

The plate thickness ranges from 1.2mm to 2.0mm in the mini series and from 1.5mm to 3.0mm in the medium series.

The Fixin bone plate system is ideal for minimally-invasive fracture repair, direct fracture reduction, indirect fracture reduction and corrective osteotomy to treat angular limb deformities.

Traditional locking plates use a threaded screw-plate locking system. TraumaVet screws have a 2° conical head which locks into a corresponding 2° tapered cone in the bushings, which are pre-assembled on the plate. This coupling eliminates the risk of cross threading between the screw and plate and ensures a simple operative technique. It also permits
easy removal of the screws. It is an extremely stable coupling, which effectively transfers loads through the structure and reduces the risk of screws backing out or plate breakage.

**INDICATIONS for application of Locking plates**

Locking plates are particularly useful when screw pull-out is at a greater risk. Screws may be susceptible to pull-out failure in the metaphyseal region of bones where the bone cortex is thin, in osteoporotic bone, older patients, patients having slow bone healing conditions and patients that have poor compliance to restricted activity during the postoperative period. A locking plate is also useful with the presence of a limited proximal or distal target (epiphyseal or metaphyseal fracture). Minimally invasive plate osteosynthesis (MIPO) is best accomplished with the application of a sliding plate technique. MIPO is usually accomplished with small proximal and distal portals where one applies two – three screws in each fracture segment.

**Minimally invasive technique** is an open technique whereby direct or indirect fracture reduction is achieved with small exposure portals. Each portal is strategically located to allow proper reduction and application of an implant. An incision (1-2cm) is made overlying the metaphyseal-epiphyseal area of the proximal and distal parent bone. Soft tissue is reflected to expose the bone surface where the implant will be applied. An additional small portal may be necessary to expose the transverse (short oblique) fracture site. A periosteal elevator is used to create an avenue on the surface of the bone for positioning the implant. The fracture is visually aligned and the implant applied. With bone plates and screws, the implant is slid beneath the soft tissue into the previously created avenue on the surface of the bone. One proximal and distal screw is applied to hold fixation while alignment is examined. The remainder of screws is then inserted. As a generally rule, 2-3 screws are inserted into each fragment.