Evidence-Based Approach to Cranial Cruciate Repair Surgery
Jennifer Wardlaw, DVM, MS, DACVS
Gateway Veterinary Surgery
St. Louis, MO

Current research and literature will be reviewed to encourage the audience to update and make their own decisions regarding this multi-million dollar problem in our small animal patients. This lecture gets to the heart of which procedures are the best in skilled surgeons’ hands for our canine patients.

Cranial cruciate ligament rupture is a common cause of hindlimb lameness in dogs and is seen in cats as well. Patients can be managed without surgery with exercise restrictions, body weight management and pain medications. However, a better prognosis is achieved when the patients are less than 15 kg. Also, the presence of a meniscal tear or concurrent patellar luxation makes medical management less successful.

When surgical stabilization is opted for, the veterinarian is faced with a plethora of options. The key is to find the balance of what the surgeon is comfortable with and what the best option is for the patient. If the best possible option is chosen by the veterinarian but they do not have the training or experience to perform the procedure correctly, the potential complications can be disastrous. The fact that several options are available to address the same surgical problem indicates that no one procedure is perfect for all cases and all situations. Being current on the options and the data published is necessary to make the most educated decisions for your patients.

There are innumerable intraarticular repair methods in the literature and the theory behind these is the basis for human ACL repair. However, due to the degenerative nature of CCLR in dogs, these techniques have fallen out of favor. Intracapsular techniques are degraded by the inflammatory mediators seen in stifles with osteoarthritis (OA). The result is an unstable surgical repair and a lower level of function due to lameness with progressive OA. Long term outcomes with intracapsular repair are not as good as extracapsular techniques. However, if this is the procedure you are most comfortable with, and the owner will not accept referral to a surgeon, than this may be the “best” option for that patient.

The original extracapsular prosthetic stabilization has gone through many revisions and adjustments since its inception in 1966. The current technique is usually a lateral circumfabellar-tibial suture. Bone anchors can be used on the femur instead of around the fabella if preferred. The tibial suture is typically passed through a tibial bone tunnel located at the level of the long digital extensor tendon groove. Sutures can be tied or crimped. Nylon leader, monofilament or braided sutures are currently used, while stainless steel is no longer recommended due to cycling failure. The type of knot thrown can affect structural strength of some suture materials. For instance a surgeon’s throw may weaken knot security, but a square knot where the first throw is clamped to maintain tension while the rest of the knot is tied has not shown to weaken a number of suture materials. Crimps are available for use with specific prosthetic materials but are not interchangeable with sizes or types of sutures. Crimp placement requires addition equipment and slippage is found to occur in 8% of cases. However crimp placement has less elongation and more stiffness than a clamped square knot. The loop configuration of the prosthetic material has also been shown to influence performance. But in most cases, the tension of the suture is not conserved for longer than six to eight weeks after surgery. Most commonly the strength is lost through elongation or rupture. Despite positive clinical results, these techniques do not achieve normalization of stifle biomechanics to the cruciate deficient stifle and may not be the best option especially for large or overweight dogs.

Isometry and a stiffer prosthesis are the potential benefits of the TightRope CCL®. The FiberTape (Arthrex Vet Systems) used in the system has shown significantly greater stiffness and ultimate load to failure forces. However this puts the joint at risk if the prosthesis is over-tightened or if poor isometry is created with inaccurate bone tunnels. In a recent study the TightRope CCL® resulted in outcomes similar to that of the TPLO (Tibial Plateau Leveling Osteotomy). A multicenter study has shown 94% of dogs having good to excellent outcomes with a 9% major complication rate including implant failure, infection, and meniscal tear.

The TPLO surgery has historically been promoted for use in active large breed dogs or dogs with excessive tibial plateau slope. Several studies have found similar results six months postoperatively when comparing the extracapsular suture and the TPLO. However, the extracapsular dogs tended to be lighter and begin physical rehabilitation earlier than the TPLO group. It is possible that larger dogs treated with a lateral suture may have had a worse outcome. Clinically the TPLO dogs are believed to bear more weight sooner while the extracapsular dogs hold the leg up for 1-2 weeks. The TPLO surgery involves specialized equipment and is described as having a steep learning curve. Utilizing arthroscopy or a mini-arthrotomy is proposed to minimize patient discomfort over the arthrotomy used with the lateral suture technique. Complication rates with the TPLO are lower with unilateral or staged procedures ranging from 12-21%. A less specialized version of the TPLO is the Cranial Closing Wedge (CCW) which also lessens the tibial slope to negate tibial thrust, but also alters the mechanical axis of the tibia with a forward shift. This changes the biomechanics of the tibia and may change weight distribution on the menisci. The technique utilizes a saw but does not require a specialized bone plate. It can be combined with the TPLO in cases with excessive (greater than 30°) tibial slope.

The Tibial Tuberosity Advancement (TTA) is a newer procedure that eliminates cranial tibial thrust. The mechanics place the patellar tendon force perpendicular to the weight-bearing force through the stifle. A bone graft appears to be beneficial for speeding
the healing of the bony defect created. Specialized equipment is required but the procedure is technically less challenging and
perhaps faster than the TPLO. Long term studies show similarities between the TPLO and TTA, although the TTA appears to take
longer to heal the osteotomy and cannot be used in cases with excessive tibial slope. Implant designs are still changing with regards to
fork design and available cage sizes for advancement. The overall complication rate for TTA ranges from 25-59%, including minor
complications.

All of the osteotomy techniques require strict confinement while the bone heals. This may be a deciding factor between techniques
in ill mannered dogs or outdoor-only animals. While physical rehabilitation is started early in all dogs, the postoperative care for the
osteotomy dogs can be weeks to month longer than the lateral suture technique. However, early return to function is vital for joint
health, and to rebuild muscle mass and regain lost bone density. Service or therapy dogs who are kept in a controlled manner will
likely benefit from the quick return to weight bearing of the osteotomy procedures, with their daily activities being as controlled and
calm as most rehabilitation programs.

The existence of so many variations on the same surgical problem has shown no concrete superior method for treating our
veterinary patients exists to date. Research is ongoing to illustrate the pros and cons of the newer techniques to determine the best
options. Kinematic and objective controlled multi-center prospective trials are needed. But patient needs and variation in fibrosis,
activity level, meniscal damage and age along with owner financial constraints will all play into the decision of the “right” treatment
modality.
Surgical Options for Repairing Luxating Patellas
Jennifer Wardlaw, DVM, MS, DACVS
Gateway Veterinary Surgery
St. Louis, MO

Soft tissue and orthopedic procedures are both needed to correct luxating patellas. The intricate details of both will be discussed with focus on anatomy and accuracy. Postoperative care and physical rehabilitation will also be discussed.

Patellar luxation can be congenital or traumatic. Congenital is typically the presentation for small and toy breed dogs. Although some large breed dogs can have it as well. It is typically bilateral, with medial being the most common side of the luxation. Often times one side may be more severe than the other side. Traumatic patellar luxation is typically unilateral in nature and tends to be laterally luxated.

The patella is a sesamoid bone in quadriceps mechanism and uses the straight patellar tendon to insert on the tibial tuberosity. Although the true pathogenesis is unknown, it may result from abnormal hip conformation, angle of inclination, or coxa vara. Any of these components that cause a malalignment of the quadriceps mechanism, can lead to patella luxation. After the patella has been luxated is leads to secondary changes to the limb including medial displacement of the quadriceps, lateral bowing of the femur, torsion of the distal femur, shallow trochlear groove, stifle instability and medial displacement of the tibial tuberosity.

Clinical signs are very suggestive of the disease with an intermittent lameness or “hopping”. Animals may also have a crouched stance or “bowlegged” appearance. Lameness often increases as degenerative joint disease develops. Diagnosis is based on clinical signs and physical examination. However, radiographs are needed of the entire hindlimb to assess for torsional deformity, hip conformation and other orthopedic issues as well. Radiographs will show the displacement of the patella on the craniocaudal view. Shyline views can also be used to assess the trochlear groove. Documentation of secondary arthritis is also important for prognostic goals following surgery.

The patella luxation grading system is currently used to help elicit when surgery is needed and judge the level of surgery needed to correct the limb abnormality.

Grade 1
• Intermittent luxation
• Patella can be manually luxated, but reduces spontaneously
• Rarely lame, occasionally skip
• Minimal medial tibial rotation

Grade 2
• Frequent luxation
• Patella luxates with stifle manipulation, reduces spontaneously with rotation of the tibia
• Lameness varies- occasional skip to weight bearing lameness
• Medial tibial rotation- up to 30°

Grade 3
• Patella is luxated but it can be reduced, reluxates
• Chronic lameness of varying severity
• Medial tibial rotation of 30° to 60°
• Moderate angular and torsional deformities

Grade 4
• Patella is luxated continually and cannot be manually reduced
• Limb is carried or the animal moves in a crouched stance
• Medial tibial rotation of 60° to 90°
• Marked angular and torsional deformities

Surgical repair goals are to realign the extensor apparatus, normalize the forces acting on the physes/cartilage and stabilize the patella in the trochlear groove. Soft tissue reconstruction is helpful, but not a surgical solution to this orthopedic condition by itself. More commonly used soft tissue procedures include; Imbrication of the lateral retinacular fascia, Patellar and tibial anti-rotational sutures, Medial release (desmotomy). Bone reconstruction is far more successful and should be the cornerstone of any patellar luxation surgery. Orthopedic corrections can include; Tibial tuberosity transposition, Trochleoplasty techniques, Corrective osteotomies of femur / tibia. The goal of surgery is to improve limb function in dogs with lameness. Surgery does not prevent the progression of OA.
Reluxation is common with up to 48% reluxation in one study. However relaxation is usually mild (grade 1), and clinical signs may be minimal, negating the need for further surgery.
Managing Hip Dysplasia in Young Dogs
Jennifer Wardlaw, DVM, MS, DACVS
Gateway Veterinary Surgery
St. Louis, MO

At birth most hips are normal. The femoral head and neck are cartilaginous and begin forming bone by endochondral ossification. Joint congruence and stability are dependent on periarticular soft tissues. This congruency and stability is critical for normal joint development. Disparity in the development can happen with any bony part of the joint or soft tissue including muscles, ligaments and the joint capsule. The skeleton develops rapidly and small problems can rapidly lead to a chain reaction of disease. If the hip joint is lax or unstable it leads to poor joint congruence which causes subluxation and further abnormal hip development. A dog may have normal hips at birth but through genetics, nutritional or environmental factors, develops hip dysplasia (HD).

Nutritional influences such as a high plane of nutrition or imbalance can lead to HD. A high plane of nutrition affects growth rate and can lead to rapid bone growth and weight gain. This can over-load the soft tissue support and has been shown to increase the frequency and severity of HD. Studies have shown a faster than average weight gain may lead to HD, even with exercise restriction. A dietary electrolyte imbalance may affect the synovial fluid. A low dietary anion gap (sodium + potassium –chloride) results in less subluxation while excess may increase synovial fluid amount and joint laxity. This may be due to surface tension and hydrostatic pressure.

Paying special attention to “at risk” puppies during initial examination is a key component to managing these patients. Asking pertinent questions about their normal activity, keeping them lean and on a balanced diet to avoid adverse nutritional influences are key. In puppies that are large or giant breeds, or have known familial histories perform an Ortolani and Barden exam. Also consider switching off puppy formulas at 6 months to slow the rate of growth. You may also want to consider prophylactic management.

A radiographic diagnosis of HD is more difficult in younger dogs but can be performed with various techniques. The hip extended view is used by the Orthopedic Foundation for Animals (OFA), and the Norberg Angle. Distraction radiography is used in the PennHIP Program and Dorsolateral subluxation techniques. The OFA scale does not require special equipment but identifies OA and is not a sensitive method to detect early or mild laxity. You can also not certify with OFA until they are 2 years of age making it a difficult screening test for puppies unless they are severely affected. PennHIP requires certification to submit films as well as sedation or anesthesia of the patients. You need three mandatory radiographs. The distraction index is calculated off the percent of the femoral head that is luxated out of the acetabulum. A distraction index of greater than 0.3 is considered disease susceptible, but breed variation of measurements exist. This modality has been shown to be statistically predictable at 16 weeks of age.

Once you have a diagnosis or have decided for early prevention, time is one your side since you caught it early. Medical management is 80% successful and is clinically more helpful the earlier you begin. Weight control or reduction is the cornerstone to minimize the stress of the growing active joints. A regulated exercise program should be utilized but not overdone. OA disease modifying agents or nutraceuticals can be started early. Physical rehabilitation can be tailored for a puppy and includes homework for owners that promote not only joint health but obedience and training. NSAIDs can safely be used in puppies after 2 months if pain is an issue. The key to conservative treatment or prevention of HD is the multimodal approach. Controlled exercise programs should be designed for the active playful puppy. Consider postponing strict training until they are at least 6 months of age. Excessive force even on normal joints can cause OA. Agility, flyball, sporting and rescue training should be “walk through” training to get the idea and the motions without the force. Exercise is good in moderation and will help reduce obesity as well as maintain a good range of motion. Low impact exercise can be used liberally including swimming, walking, obedience class and leash training. Studies have shown that even with radiographic evidence at a young age of HD, weight control and leash walking can dramatically increase the range of motion, exercise tolerance and long-term function for years.

Nutraceuticals have been shown to be the most beneficial in offsetting OA when given before the inflammation starts, meaning preemptively when we suspect disease. Since they have minimal if any side effects and the potential for a large impact, it is easy to prescribe them to owners who are willing. Nutraceuticals have been called disease modifying agents, disease modifying osteoarthritic drugs, supplements, additive and vitamins. The key to understanding the options are to realize the FDA does not regulate these products for efficacy or quality. It is vital you find a company you like, believe in and has research to support their products and claims. If you are using a product and not seeing results, then try a new source. Some options work better for certain cases, but generally speaking when added to a well balanced multimodal approach can make a big difference with regards to patient comfort and cartilage health. Most contain glucosamine and chondroitin sulfate in various forms. It is reported that they are absorbed by the GI tract, become incorporated into joint tissues, and provide the necessary precursors to maintain cartilage health and decrease inflammation. Anecdotal reports, in vitro studies, and published clinical trials indicate that these agents are effective in treating OA.

Glucosamine is an amino-monosaccharide nutrient that has exhibited no toxicity even at high oral doses. It is a precursor to the disaccharide unit of glycosaminoglycans, which comprise the proteoglycan ground substance of articular cartilage. Studies using radiolabeled compounds in man and animals have shown that 87% of orally administered glucosamine is absorbed. Glucosamine acts
by providing the regulatory stimulus and raw materials for synthesis of glycosaminoglycans. Since chondrocytes obtain preformed glucosamine from the circulation (or synthesize it from glucose and amino acids), adequate glucosamine levels in the body are essential for synthesis of glycosaminoglycans in cartilage. Glucosamine is also used directly for the production of hyaluronic acid by synoviocytes.

In vitro biochemical and pharmacological studies indicate that the administration of glucosamine normalizes cartilage metabolism and stimulates the synthesis of proteoglycans. In one study, glucosamine stimulated synthesis of glycosaminoglycans, proteoglycan and collagen, suggesting it not only provides raw material for their production, but may actually up-regulate synthesis. The effects of glucosamine sulfate on human chondrocyte gene expression was also evaluated, assessing its effects on type II collagen, fibronectin and proteoglycans in normal adult chondrocytes. Glucosamine modulated the expression of cartilage proteoglycans, decreased stromelysin mRNA levels in osteoarthritic chondrocytes, and preserved the constitutive expression of type II collagen and fibronectin in both normal and osteoarthritic chondrocytes.

Chondroitin Sulfate (CS) is a long chain polymer of a repeating disaccharide unit. It is the predominant glycosaminoglycan found in articular cartilage and can be purified from bovine, whale, and shark cartilage sources. Bioavailability studies in rats, dogs and humans have shown 70% absorption of CS following oral administration. Studies in rats and humans using radiolabeled CS have shown that CS does reach synovial fluid and articular cartilage.

When human articular chondrocytes were cultivated in clusters in the presence of CS, proteoglycan levels were significantly increased and collagenolytic activity was decreased. A similar study indicated that CS competitively inhibited degradative enzymes of proteoglycans in cartilage and synovium. In a study of rabbits with chymopapain-induced stifle arthritis, proteoglycan depletion was reduced by the administration of CS.

Clinical trials in humans have also found CS to be effective in reducing the symptoms of OA. In a placebo-controlled, double-blinded study of 120 patients with OA of the knees and hips, treatment with CS resulted in significant improvements in pain-scale and collagen, suggesting it not only provides raw material for their production, but may actually up-regulate synthesis. The effects of glucosamine sulfate on human chondrocyte gene expression was also evaluated, assessing its effects on type II collagen, fibronectin and proteoglycans in normal adult chondrocytes. Glucosamine modulated the expression of cartilage proteoglycans, decreased stromelysin mRNA levels in osteoarthritic chondrocytes, and preserved the constitutive expression of type II collagen and fibronectin in both normal and osteoarthritic chondrocytes.

Dasuquin® (Nutramax Laboratories, Inc.) is a joint nutraceutical marketed for management of OA in dogs and cats. It is a combination of glucosamine, chondroitin sulfate, decaffeinated tea polyphenols, and avocado/soybean unsaponifiables (ASU). Tea polyphenols may have a positive effect on cartilage health and provide oxidative balance in the body. ASU, which are biologically active lipids, have been shown to be more effective than chondroitin sulfate in inhibiting the expression of certain OA mediators responsible for cartilage breakdown. In in vitro studies, ASU has been shown to decrease the expression of COX-2 enzyme, TNF-α, IL-1β, and PGE2 in chondrocytes. It was also shown to stimulate synthesis of cartilage matrix by increasing levels of TGF-β. A 2007 study found that dogs given ASU for 3 months had elevated levels of TGF-β in their synovial fluid compared to control dogs. The combination of ASU with glucosamine and chondroitin sulfate decreased the expression of numerous pro-inflammatory mediators, including TNF-α, IL-1β, and iNOS. This decrease in pro-inflammatory mediators seen with Dasuquin® (Cosequin® with ASU) is greater than that seen with Cosequin® alone. In an in vivo study of the effects of Cosequin® on cartilage metabolism in dogs, serum samples were collected after treatment with Cosequin® and tested for circulating glycosaminoglycan content. Median serum glycosaminoglycan levels were significantly increased in treated dogs. When normal calf cartilage segments were exposed to the serum from the treated dogs, the biosynthetic activity of chondrocytes was significantly increased and proteolytic degradation of the cartilage segments cultured in serum was reduced. In vitro studies at the Nutramax laboratories also demonstrated the beneficial effect of Dasuquin® on chondrocytes from different species including equine, camelid, canine, feline and bovine. Dasuquin® inhibited the production of inflammatory mediators and signaling molecules in the inflammatory cascade.

Omega acid supplementation was discovered when dermatologic patients were experiencing relief from their OA. Maintaining a high content of the long chain omega-3 fatty acids EPA, and DHA is the key with this nutraceutical. Short chain omega-3s compete with omega-6s for conversion to long chain fatty acids and then for uptake into cell membranes. Omega-3s and omega-6s have different effects on the inflammatory response. Omega-6 arachidonic acid is the precursor to more pro-inflammatory mediators. While omega-3 EPA is a precursor to less potent inflammatory mediators. Omega-3s are readily available from several companies for veterinary as well as human products. Pet foods that contain them must be kept in a sealed bag for less than 30 days or they dry out. Fish oils will also help lubricate the skin and shine the coat. For large breed dogs I follow the human label recommendation for full grown dogs or half the dose for puppies. If you overdose the oils, they can have soft stool or diarrhea and should decrease the dose.

Some other options that are developing for easy oral administration include green-lipped mussel, methyl-sulfonylethane, duralactin and S-adenyl-L-methionine. Less research or anecdotal evidence exists for these but is continually being developed.
The use of joint nutraceuticals in dogs prior to the development of OA is controversial. No controlled studies have been reported that document the efficacy of nutraceuticals in preventing the development of OA. However, because of their reported effects on improving cartilage matrix and reducing levels of inflammatory mediators within the joint, many clinicians have advocated the prophylactic use of joint nutraceuticals, particularly in athletic and large dogs that might be susceptible to joint injury. Additional research is needed to confirm the value of prophylactic use of joint nutraceuticals.

There are also surgical options to diminish the signs of OA in puppies that have HD. The two surgical options are Juvenile Pubic Symphysiodesis (JPS) and Triple Pelvic Osteotomy (TPO). JPS is a simple procedure performed on puppies 12 to 20 weeks of age. But the optimal results are achieved on puppies less than 16 weeks old. Please note that this age is before the PennHIP certification age. The procedure fuses the pubic symphysis with electrocautery via a ventral midline incision. There are no implants and, with proper protection of the urethra and depth to avoid the colon, very few potential side effects. Electrocautery is used every 2-3 mm along the symphysis to cause thermal necrosis and premature closure. The pelvis continues to grow in all other planes while being static at the pubis, resulting in ventroversion of the acetabulum. This procedure is not readily detectable on OFA and PennHIP films and should therefore only be performed on animals that will be sterilized to avoid certifying or breeding falsely represented hip conformation. The TPO is typically performed on dogs less than 10-12 months of age without radiographic signs of OA. It is used to correct hip laxity. Three osteotomies are made on the pubis, ischium and ilium to allow reorientation of the acetabulum. Then an angled plate is placed on the ilium to secure the weight bearing axis for bone healing. The forced manual ventroversion increases dorsal coverage of the femoral head and reduces the formation of OA by improving joint stability and congruence. However bilateral surgery is not performed due to high complication rates and surgeries should be staged at least 4 weeks apart. Potential complications include a narrowed pelvic canal, sciatic neuropraxia, implant failure and an abnormal gait. Lameness improves in 92% of dogs and the progression of OA appears to be slowed with this procedure. The JPS and TPO procedures have similar effects on hip conformation, although neither eliminate laxity or completely cure HD. They can arrest or limit the progression of HD in mild to moderate cases. Both of these preventative surgeries require early puppy screening and counseling of owners about potential benefits and expected outcomes.
Managing Hip Dysplasia in Old Dogs
Jennifer Wardlaw, DVM, MS, DACVS
Gateway Veterinary Surgery
St. Louis, MO

At birth the hips are normal. The femoral head and neck are cartilaginous and begin forming bone by endochondral ossification. Joint congruence and stability are dependent on periarticular soft tissues. This congruence and stability is critical for normal joint development. Disparity in the development can happen with any boney part of the joint or soft tissue including muscles, ligaments and the joint capsule. The skeleton develops rapidly and small problems can rapidly lead to a chain reaction of disease. If the hip joint is lax or unstable it leads to poor joint congruence which causes subluxation and further abnormal hip development. A dog may have normal hips at birth but through genetics, nutritional or environmental factors, develops hip dysplasia (HD).

A presumptive diagnosis of HD can often be made based on clinical signs or physical examination. Palpating for crepitus, a luxated hip or performing the Ortolani and Barden maneuvers can all help make a correct diagnosis of HD. A radiographic diagnosis of HD is more easily made in older dogs. The hip extended view is used by the Orthopedic Foundation for Animals (OFA), and the Norberg Angle. Distraction radiography is used in the Penn HIP Program and Dorsolateral subluxation techniques. The OFA scale does not require special equipment but identifies OA yet is not a sensitive method to detect early or mild laxity.

Medical management is 80% successful and is clinically more helpful the earlier you begin. Weight control or reduction is the cornerstone to minimize the stress diseased joints. A regulated exercise program should be utilized but not overdone. OA disease modifying agents or nutraceuticals can be started early. The key to conservative treatment of HD is the multimodal approach. Excessive force even on normal joints can cause OA. Exercise is good in moderation and will help reduce obesity as well as maintain a good range of motion and comfort.

Weight loss is the easiest and perhaps most beneficial part of a multimodal approach to OA. Minimizing the work the diseased joints have to contend with should be paramount to any regime. Dogs with OA should be kept on the thin side of normal. With proper weight management, many dogs are able to stop taking pain medications until much later in the disease process. Commercially available diets are geared towards weight loss as well as joint comfort. Diets should be low calorie and low in protein while providing an otherwise balanced nutritional plane. Having truthful conversations about treats and table scraps should be geared to reveal honest habits. Caloric responsibility should be encouraged and adjustments made to account for the dogs’ favorite treats or foods. Exercise is also important to maintain a good range of motion and weight level. Minimizing concussive forces like stairs, jumping, climbing, running, and horse play should be minimized while still maintaining a good quality of life. Encourage leash walks, swimming and pay close attention to what activities make them sorer afterwards. While we don’t want to lock our patients in a box or take away their quality of life, easing their burden is important for their joints. If they love to play fetch on the weekends, make their owners aware that that will be a painful time and they should premedicate or otherwise adjust the protocol for their pet. Having thick warm bedding should also be encouraged to help aching joints. If an overweight animal prefers the hard, cold floor, suggest placing a fan near the orthopedic bed to encourage usage.

NSAIDs are readily available and widely used for OA in dogs. The important thing is to find a drug that works well for each patient and to make the owners aware of potential side effects. If one stops working for a patient, try switching to a different one. When switching NSAIDs, a wash out period of at least two half-lives is recommended. NSAIDs can be used for painful flair ups, around times of increased activity, or later in the disease, for daily maintenance pain relief. For patients with NSAID sensitivities or for patients needing additional pain medication there are other options as well. Tramadol is a synthetic mu opioid with a wide safety margin. It can be given several times a day which make it ideal for use around exercise or physical rehabilitation periods. I typically use 5 mg/kg up to 4 times daily. Gabapentin is a GABA analogue design to treat epilepsy but is widely used for neuropathic pain and OA in people. The most common side effect appears to be sedation. An accepted canine dose is 5-10 mg/kg 2-3 times daily. Acetaminophen with codeine is an additional option for OA management. Due to the limited pill size it is often times easier to dose than tramadol in larger patients. Since it is not considered a COX1 or COX2 drug, side effects should be minimal when used concurrently with NSAIDs, but should still be considered. This drug is dosed off the codeine at 1-2 mg/kg three times daily.

Nutraceuticals have been shown to be the most beneficial in offsetting OA when given before the inflammation starts, meaning preemptively when we suspect disease. Since they have minimal if any side effects and the potential for a large impact, it is easy to prescribe them to owners who are willing. Nutraceuticals have been called disease modifying agents, disease modifying osteoarthritic drugs, supplements, additives and vitamins. The key to understanding the options are to realize the FDA does not regulate these products for efficacy or quality. It is vital you find a company you like, believe in and has research to support their products and claims. If you are using a product and not seeing results, then try a new source. Some options work better for certain cases, but generally speaking when added to a well balanced multimodal approach can make a big difference with regards to patient comfort and cartilage health. Most contain glucosamine and chondroitin sulfate in various forms. It is reported that they are absorbed by the GI.
tract, become incorporated into joint tissues, and provide the necessary precursors to maintain cartilage health and decrease inflammation. Anecdotal reports, in vitro studies, and published clinical trials indicate that these agents are effective in treating OA.

Physical rehabilitation for muscle mass, range of motion and comfort are a huge component to managing an older dog with arthritis. Passive range of motion with stretching and massage can help aid in comfort while bathing the articular cartilage with nutrients from the synovial fluid. Increasing awareness with bedding, stairs, and household routines will help minimize concussive activities. While implementing therapeutic exercises during regular walks can increase muscle mass and range of motion, especially extension. Focus on increasing comfort while optimizing rear weight distribution through regular, motivated exercise.

If medical management is not an option or is not working for your patient, there are two salvage procedures; Total Hip Arthroplasty (THA) and Femoral head and neck ostectomy (FHO). THA is indicated for large and giant breed dogs but is available in sizes for small dogs and cats. Unilateral replacement is adequate for 80% of dogs. The procedure is technically challenging and expensive. There are cemented and cementless systems with templates and modular designs for a custom fit. The prognosis for a pain-free function is 95% having a good to excellent outcome. Potential complications include infection, luxation, fracture, sciatic neuropraxia or implant loosening. FHO is used to preserve limb function in severe OA when medical management is ineffective or when a THA has unreparable complications. It is typically performed in small dogs and cats but can be used for larger dogs when THA is not feasible. It is less expensive and easier to perform than a THA. The prognosis is good in smaller patients but much better if muscle atrophy is not severe. Postoperative physical therapy is important to achieve a flexible pseudoarthrosis.
Cats Get Arthritis, Too
Jennifer Wardlaw, DVM, MS, DACVS
Gateway Veterinary Surgery
St. Louis, MO

Identifying painful cats with arthritis will be discussed in-depth. Potential changes to the home environment, diet and surgical options will be discussed. Specific cat medical conditions dealing with arthritic joints will be focused on.

Feline osteoarthritis (OA) is a growing problem in our veterinary patients. We are discovering that it has been around and under diagnosed for years. While we tend to think of cats as just a small dog, they are very different from their canine counterparts. Cats come with a unique set of behaviors, personalities and diseases of their own. And OA in cats is very different from OA in dogs.

Knowing when a cat has OA is probably the hardest part of the disease. Typically dog owners report lameness or a decline in exercise tolerance or endurance. They have a specific leg they hold up or skip on. Or perhaps they are sore in the morning but once they begin running they appear comfortable. Owners are very savvy on OA in dogs and most have heard of hip dysplasia. But cat owners and veterinarians are only now learning about what an arthritic cat looks like. Cats can have a lameness or altered gait, but it is much less common than dogs to present for this complaint. Cats may also exhibit stiffness when rising, which may be hard to discern from the normal cat nap stretch, which may be normal. If a cat presents with overt or obvious pain or single leg lameness, the diagnosis may be easy. But more commonly the signs will be much more subtle.

Personality changes are one of the most common observations in a cat with OA. A normally affectionate cat may become nervous around new people or bustling activity around the house. A happy playful cat may become depressed or withdrawn. And a fun loving energetic cat may turn into an aggressive animal, biting family members or hissing at people when they are trying to pet or love on the family pet. These personality changes may have previously been dismissed as a normal aging cat mood change. But these character changes are not normal and should be addressed with the client to maintain a loving home environment and a good quality of life. These may all be signs of OA and pain in cats.

Behavioral changes are also important indicators for feline OA and discomfort. If the family cat changes from years of house training to disuse of the litter box, perhaps the box it too hard to climb into or located too far away to reach in comfort. Litter boxes should be shallow and more numerable for cats with OA. Normally you would want one extra litter box above the number of cats in a household, but with OA you will need more to be easily accessible without a long trek. If the cat normally climbs onto a sunny windowsill but has stopped sunbathing due to its height, this could be another sign of OA. It may be too hard to jump up onto, or too painful when they jump down off of the high ledge. The same can be seen for getting onto kitchen countertops or the family couch they used to love sharpening their claws on. Owners may think their pet has outgrown these less desirable habits, when in reality they may have stopped due to discomfort from these activities. Stairs also pose concussive forces on painful arthritic joints. Cats with OA are less likely to travel upstairs to family bedrooms where they used to sleep and snuggle. If litter boxes are only located downstairs, cats are more apt to eliminate upstairs due to the effort required for an OA patient to travel to the normal laundry room or bathroom downstairs. Another common behavioral change is a cat that would normally dominate the house has stopped chasing the younger cats or dogs. If they normally torment housemates but have stopped doing so, the effort may be too much with arthritic joints and may not be an indication of a mellowing older feline, but a painful one.

Feline OA is much more prevalent than we previously thought. One study of randomly selected cats showed 73% had evidence of OA. Another study showed that elbow and hip joints are the most commonly affected in cats. It also appears that most cats have at least four joints involved when they have OA. There are several other studies looking at “asymptomatic” cats and the prevalence of OA. The prevalence was 64% in older cats but still 16-22% of asymptomatic cats having radiographic signs of OA. This body of knowledge supports that we are under diagnosing and under appreciating the discomfort in our elusive feline patients.

Treatment of feline OA is similar to canine once the suspicion or diagnosis has been made. Weight loss is a large component to minimizing the work the diseased joints are battling. Keeping cats on the thin side of normal as well as a balanced nutritional plane is important. Proper exercise should be encouraged for a good range of motion and joint comfort. Their beds should warm and thick. Beds should also be lowered from windowsills and be available on all floors in the house to minimize traversing stairs. Massaging the cat and encouraging range of motion or stretching can be a bonding experience as well as helping to lubricate joints by circulating synovial fluid. “Lazy” cats should be encouraged to walk by baiting with toys, treats or other positive reinforcement. Commercial diets are now also available for cats and focus on weight loss with joint additives.

Nutraceuticals including chondroitin sulfate, glucosamine HCL, and omega acids are also available in feline friendly sizes and formulations. Many come in feline friendly flavors or sprinkle formulas you can place on their food for ease in administration. Pain medication are difficult in cats but there are options as well. NSAIDs are available for short term use but not approved for long term management of OA in cats in the United States. Tramadol and Gabapentin are used in cats with success. Cats tend to be more excitable with tramadol and when this side effect is encountered, lowering the dose not help. Also, splitting tramadol tablets to dose the smaller patients creates a very bitter taste, so it should not be added to food.
OFA will certify cats and currently has 7 breeds with significant numbers including the Main Coon, which shows 18% affected. PennHIP is currently only for dogs. Surgical options are similar for cats and include Total Hip Arthroplasty (THA) and Femoral head and neck ostectomy (FHO). THA is available in sizes for small dogs and cats. Unilateral replacement is adequate for 80% of dogs. The procedure is technically challenging and expensive. There are cemented and cementless systems with templates and modular designs for a custom fit. The prognosis for a pain-free function is 95% having a good to excellent outcome. Potential complications include infection, luxation, fracture, sciatic neuropraxia or implant loosening. FHO is used to preserve limb function in severe OA when medical management is ineffective or when a THA has unreparable complications. It is typically performed in small dogs and cats but can be used when THA is not feasible. It is less expensive and easier to perform than a THA. The prognosis is good in smaller patients but much better is muscle atrophy is not severe. Postoperative physical therapy is important to achieve a flexible pseudoarthrosis.

Cats may also have OA in their stifle due to cranial cruciate ligament rupture. The degree of lameness and age are used to guide whether surgical correction should be performed. If the cat is non-weight bearing despite medical management, surgery should be considered. If the cat is young and over 15 pounds surgery may be the better option for a quicker return to function and to minimize the progression of OA. Cruciate repair techniques available in the cat include the extracapsular repair, cranial closing wedge and tibial plateau leveling osteotomy. Medial patellar luxation may also be present in cats with stifle OA. The Devon Rex and Abyssinian are particular prone to this condition. It is also usually bilateral. If the cat has a grade II or more, recurrent or continued lameness or also has a cruciate rupture, surgery should be considered.
A fairly recent study* showed that 32% of dogs referred to a surgeon for hip dysplasia treatment had, in fact, a torn ACL.

Indeed, differentiating between a torn cruciate ligament and hip dysplasia can be tricky if not frustrating. Let's review the differences between the two conditions.

**Cranial cruciate ligament tear**

Severity of lameness depends on the severity of ligament disruption.

- In stable partial tears, lameness can be subtle and noted only after periods of strenuous activity.
- In complete tears, lameness will initially be severe and non weight-bearing. Then, moderate to severe weight-bearing lameness will occur.

In obvious cases, of course, a positive cranial drawer and a tibial thrust are the keys to diagnosing a cranial cruciate rupture. But what to do in less obvious cases?

Examination reveals various degrees of stifle pain with flexion and extension, variable crepitus, and possibly clicking associated with a meniscal tear.

In partial tears, a pain response is elicited when the joint is in full extension. In chronic cases, muscle atrophy is notable and peri-articular fibrosis (medial buttress) is evident on the medial side of the stifle. Medial buttress is almost pathognomonic for a cranial cruciate rupture. The only other condition that can present with medial buttress is a medial collateral ligament tear, which is usually seen with a deranged stifle, not a simple lameness.

Joint effusion is also a key finding: it can be palpated on the medial and lateral aspect of the patellar tendon.

Affected dogs have an abnormal "sit test," i.e. they sit with the affected leg extending out to the side, rather than sitting squarely (which they will do even with hip dysplasia). This is critical step in the evaluation. See below how this Lab does not want to flex his left knee.

In a partial tear, the cranial drawer may or may not be present. A sedated exam is needed to confirm the findings. MANY patients who don't seem to have a drawer while awake, suddenly have one once they are sedated and relaxed.

Radiographs are warranted in all cases to document stifle arthritis, to confirm pathology in challenging cases of partial tears, and to rule out other disorders (occasionally, we find a tumor).

The earliest and most consistent finding is the loss of infra-patellar fat pad shadow by a soft tissue opacity in the lateral view. This is consistent with effusion.

Caudal displacement of fat density located caudal to the joint capsule by a soft tissue opacity is also consistent with synovial distention.

In many cases, you can "see" the cranial tibial thrust on an X-ray. See below how subluxated the knee is.

Another consistent finding is osteophyte and/or enthesiophyte formation in the region of femoral trochlear ridges, tibial plateau, and at the base and apex of the patella.

Rupture of the contralateral cruciate ligament occurs in 37%-48% of dogs within 6-17 months of the initial diagnosis. However, rupture can be bilateral on presentation, often times giving them a “neurologic” crouched walk.

**Hip dysplasia**

Hip dysplasia causes joint inflammation and secondary osteoarthritis, which lead to variable degrees of pain. Clinical signs can vary from slight discomfort to severe acute or chronic pain. Although the disease onset has a linear progression over time, it can be divided into two forms.

The juvenile form typically affects dogs between 5 and 12 months of age. They present with unilateral or bilateral hind limb lameness, bunny hopping, and difficulty rising after rest, reluctance to walk, run, jump, or climb stairs, exercise intolerance, and pain on hip extension.

These clinical signs are the result of joint laxity.
The chronic form of hip dysplasia has a highly variable onset of clinical signs in old dogs. Pain is most often related to DJD and has a more chronic presentation. Clinical signs are similar to the juvenile form. Pain is elicited most notably during hip extension.

As the disease progresses, crepitus can be palpated with range of motion. A sedated exam followed by orthogonal radiographs will further support the diagnosis.

Hip dysplasia dogs have a normal "sit test," i.e. they sit with both legs flexed symmetrically.

**Hip and knee**

Of course, both conditions can be present at the same time. In the study mentioned above, 32% of dogs referred to a surgeon for hip dysplasia treatment had, in fact, a torn cranial cruciate ligament. Interestingly, 94% of those dogs with a cruciate tear had concurrent radiographic signs of hip dysplasia.

It is imperative to do a thorough orthopedic and neurologic exam to accurately localize the clinical signs to avoid inappropriate diagnosis and treatment.

My absolute best advice? If in doubt, repeat your entire exam under sedation. Let's go over the 7 magic benefits of sedation:

1. Sedation allows you to check for cranial drawer, tibial thrust, Barden and Ortolani sign.
2. Under light sedation, you may still notice a pain response: increased respiratory rate or pulling on the leg.
3. Under heavy sedation, total relaxation allows you much better joint evaluation.
4. Sedation allows you to tap the knee (arthrocentesis), which is an invaluable test.

Crudely, normal fluid = clear, tiny amount and viscous. Abnormal fluid = yellowish, large amount and watery.

1. Sedation allows you to "block" a joint, with lidocaine and/or steroids.
2. Sedation enables you to take X-rays in a perfect position (knee = TPLO position, with a quarter in the picture; hip = OFA style) without fighting or causing pain.
3. Sedation allows you to focus and take your time without fighting with your patient and alienating your technicians.

**Reference**

Panosteitis is an acquired self-limiting condition of undetermined cause that affects the diaphyseal and metaphyseal regions of the long bones of young, large breed dogs. German Shepherd, Dobermans, Goldens, Saint Bernards, Bassets and Labs are overrepresented. Dogs are typically 5-18 months of age at presentation. Males are more frequently affected than females. While the etiology is unknown, histopathology reveals an increased osteoblastic and fibroblastic activity replacing the medullary cavity with connective tissue. There are no inflammatory cells or necrosis, but instead a haphazard intramembranous ossification. Clinical signs may include lethargy, anorexia and a shifting leg lameness which can be acute or chronic, but is often intermittent. Pain can be elicited on palpation of the diaphysis of affected long bones. The humerus, femur and proximal radius/ulnar are the most common sites. The pain can be cyclic and recurrent. Radiographically you can visualize an increased density within the medullary cavity blurring the trabecular pattern, often near the nutrient foramen. However, lameness is not always associated with radiographic lesions and early in the disease, radiographic signs may not be apparent. This self-limiting disease often resolves in 1-2 weeks but can recur up to 18 months of age. Conservative therapy may include NSAIDs, exercise restriction, weight reduction and dietary correction to avoid oversupplementation. The prognosis is excellent with some dogs experiencing a shifting lameness until maturity. Rarely, clinical signs persist after maturity.

Osteochondrosis (OC) is a disturbance in the process of endochondral ossification in a focal area of developing articular surface. Cartilage fails to undergo calcification and replacement by bone and therefore becomes degenerative. Cartilage retention results in thickening of the articular epiphyseal cartilage and degrades because cartilage cannot handle high shearing forces. The etiology may involve genetics, rapid growth, calcium supplementation, hormonal influences, ischemia and trauma as potential factors. OC is seen in large breed, fast growing dogs from 4 to 7 months of age. It is most commonly seen in the shoulder, stifle, elbow and tarsus. The thickened articular-epiphyseal cartilage has poor diffusion of nutrients from the synovial fluid. This leads to necrosis at the deep portion of the thickened cartilage. The consequence is an abnormal arrangement of cells, metabolism and function of these chondrocytes. When a separation occurs between the noncalcified and calcified layers at this weakened site a cartilage flap is formed and called osteochondritis dessicans (OCD). The flap may reattach or have a vertical fracture of the articular cartilage. The vertical fracture has minimal motion during weight bearing but causes synovitis, irritation and lameness leading to osteoarthritis (OA). When synovial fluid enters the vertical fracture it prevents it from healing. The cartilage flap can also detach and become a joint mouse, which causes irritation. The free floating flap can resorb or may enlarge due to nutrition from synovial fluid. Clinical signs usually begin with an intermittent lameness that is worse after exercise. Joint effusion and pain may also be present. Muscle atrophy and OA will develop over time. Radiographic findings will show a filling “defect” or “flattening” of the subchondral bone, which is the thickened area of cartilage that is not radiopaque. A joint mouse may also be visible if the flap is mineralized. You may see radiographic signs of OA. A positive contract arthrogram may aid in identifying a cartilaginous flap or joint mouse. Ultrasound, MRI and CT have also been shown to be very sensitive and specific for OCD. Conservative therapy can be utilized if there is no clinical pain or joint mouse present and in dogs less than 7 months of age with a small lesion. Rest and a restricted diet are implemented for 6 weeks. NSAIDs and chondroprotectants, with physical rehabilitation should also be used. This may allow the cartilage defect to heal. Surgical treatment is indicated for the presence of a non-healing flap, lameness of more than 6 weeks, a dog older than 8 months or a visible joint mouse on radiographs. The surgical objectives are to excise the cartilage flap and unadhered cartilage and well as encourage healing of the defect. Healing of the defect occurs by production of fibrocartilage which requires bleeding from subchondral vessels to allow invasion of mesenchymal stem cells. After surgery, the animal must be on restricted exercise for 4 to 6 weeks to allow the scar cartilage to form. Shoulder OCD has a good to excellent prognosis with other joints being guarded due to OA.

Elbow dysplasia is an inclusive term used to describe all developmental conditions resulting in elbow arthrosis. The growth discrepancy within the antebrachial growth plates, genetics, conformation and oversupplementation are all proposed etiologies for elbow dysplasia. Elbow incongruency can occur with the trochlear notch, radial head and humeral condyle. An elliptical trochlear notch will change the contact points of the elbow joint. Ununited anconeal process is when the anconeal process fails to unite with the proximal ulna before 20 weeks of age. It is common in large dogs including the German Shepherds, Bassett hounds and Saint Bernard’s. While the etiology is undetermined it may be OCD-related, trauma or genetics. Lameness, stiffness, pain and crepitation are commonly seen and usually bilateral. Radiographically a cleavage line can be seen along with sclerosis and OA of the elbow. Conservative therapy is described but rarely efficacious. In young dogs there are variable techniques to attach the fragment, but these are technically challenging. In dogs over 6 months of age excision of the ununited fragment is recommended. The prognosis is generally fair due to the inevitable OA and early surgical intervention may help limit OA. Fragmented medial coronoid process (FCP) is the third component of elbow dysplasia. It may be a result of excessive loading of the coronoid process during abnormal development or joint incongruency, direct trauma or OCD-complex. Lameness usually begins at 4 to 9 months of age and occurs in
large breed dogs as well, with males being over represented. Elbow pain and lameness are similar with signs of OCD, and the two may occur together. Elbows may also be abducted when standing or have joint effusion localized to the medial aspect of the elbow joint. The best radiographic view is a 25 degree cranio-caudal-lateromedial oblique view flexed 30 degrees. However, CT scans are much more sensitive for diagnosing FCP. Often signs on plain films are non-specific and include periarticular osteophytes, sclerosis and rarely soft tissue swelling. Conservative therapy with restricted exercise and weight control can be tried or surgical therapy with excision of the fragment via arthrotomy or arthroscopy can be used to remove the coronoid and evaluate for “kissing lesions” on the humerus. FCP carries a fair to guarded prognosis with inevitable OA. The choice between medical and surgical management for FCP remains controversial but surgery is generally recommended in dogs under a year.

Hypertrophic osteodystrophy is an idiopathic disease that affects rapidly growing large breed dogs and involves the long bone metaphysis. Dogs generally present between 2 to 8 months of age with males being overrepresented. German Shepherds, Irish Setter, Weimeraners, Great Danes and Chesies are more commonly affected. Clinical signs may include lameness with a reluctance to walk and warm painful swelling of the metaphysis of the distal radius/ulna, and tibia bilaterally. Patients can be anorexic, run a high fever, and exhibit depression or lethargy. Radiographs show a radiolucent region in the metaphysis with neighboring sclerosis called the “double physeal line”. There may also be irregular widening of the physeal and extraperiosteal cuffing. The etiology remains unknown but theories have been proposed for vitamin-C deficiency, oversupplementation of vitamins and minerals, *E. coli*, canine distemper, genetics, vascular abnormalities, and vaccine induced. Treatment is aimed at supportive care to maintain hydration, nutrition support, NSAIDs for pain and to correct any dietary imbalances. The prognosis is good with most patients recovering in 7 to 10 days with this self-limiting disease. Possible sequelae may include growth disturbances of affected limbs, systemic illness, or death.

Legg-Calve-Perthes Disease (LCPD) is avascular necrosis of the femoral head. It has also been called ischemic necrosis, or coxa plana. LCPD typically occurs in small and miniature breeds between 4 to 11 months of age with no apparent sex predilection. Etiology may be traumatic, vascular, autosomal recessive, infectious or hormonal. But an ischemic event occurs that results in the death of the affected bone. Bone necrosis occurs and subsequent remodeling of the femoral epiphysis occurs. Hip pain and lameness with crepitus are the most common clinical signs. Muscle atrophy can be seen, especially in bilateral cases which compromise 15-18% of LCPD cases. Radiographically you can see a flattening or irregular surface of the femoral head with a moth eaten appearance. Occasionally femoral neck fractures can also be seen. Conservative therapy may be tried unless a fracture is present indicating an FHO. Overall prognosis is good to excellent.

Capital Physeal Dysplasia is an uncommon disorder of the proximal femoral metaphysis seen in overweight male neutered cats as well as Shelties. It classically appears as an atraumatic Salter Harris Type I or II. It is often called a slipped epiphysis. While the pathophysiology is unknown the disease appears to be a combination of delayed physeal closure, decreased gonadal hormones, dysplastic chondrocytes and obesity. Typically cats are young, presenting from 5 months to 2 years. OA is already present at the time of diagnosis. The treatment of choice is FHO.
Laser Use in Physical Rehabilitation and Adding it to Your Practice
Jennifer Wardlaw, DVM, MS, DACVS
Gateway Veterinary Surgery
St. Louis, MO
Karen Felsted, DVM, MS, CVPM, CPA
PantheraT Veterinary Management Consulting
Dallas, TX

Laser therapy is Light Amplification by the Stimulated Emission of Radiation (LASER). Lasers are classified into four levels depending on their potential to harm tissue. Class 1 is a laser pointer used in lectures or at a grocery store while an example of a Class 4 laser would be a surgical cutting laser. Class 3 and 4s are used for low level laser therapy or physical rehabilitation. They are advocated for many things but mostly for wound healing and pain relief.

Laser therapy cause cellular oxygen production by photons being absorbed into the mitochondria. This in turn causes a proton gradient across the cell and mitochondrial membrane. The gradients result in increased cell permeability. Laser therapy also stimulates the production of ATP, thereby stimulating DNA production. Also laser therapy increases cellular metabolism and growth. This accelerates tissue repair and cell growth in tendons, ligaments and muscles.

There are also indications in human and rodent models that laser therapy may block pain transmission through conduction latencies and selectively inhibit nociceptive neuronal activities. It may also increase endorphins. For this reason laser therapy is being used for muscle trigger points and acupuncture, called acupressure.

Laser therapy is advocated in wound healing due to its ability to stimulate fibroblasts and speed collagen production to repair tissues. It appears to accelerate angiogenesis and neovascularization. Laser is used on edema because it causes vasodilation and improves lymphatic drainage. It appears laser therapy may help with surgical incisions, open wounds and burns. The goal of wound laser therapy is to increase blood circulation, stimulate the reduction of hemoglobin, then stimulate both the reduction and immediate re-oxygenation of cytochrome c oxidase. This is the normal metabolic, wound healing process, just trying to speed it up with laser therapy.

Lasers emit energy, or joules, at a certain wavelength. This wavelength determines how deep the laser will penetrate into the tissue. The power, or watts, of a laser is the rate or speed at which it can deliver the desired energy to the tissues. There are many different lasers with different penetrating wavelengths, but the energy density or dose for square of centimeter of tissue is the critical data point. Not only does the laser light need to fully penetrate the area we want, but it needs to bring the right level of energy to the tissue. Based on the size of the tissue or area we are treating (cm2) is how we determine the total dosage (J/cm2). The power of your laser will determine if that takes you 10 seconds or 10 minutes to accomplish that treatment dose. Research is still ongoing for determining whether continuous wave, or pulsed wave lasers are better, if daily or every other day protocols are superior and what the ideal dosage is for a condition. So given all the variables in laser company styles, format and protocols, it is of paramount importance that we discuss energy density and dosages in the same concise language so we can communicate appropriately; Joules per centimeter squared.

We do know that the minimum dosage in humans to achieve a photochemical response to laser therapy is 5 J/cm2. We also know there are contraindications to laser therapy; active hemorrhage, local steroids, pregnancy, cancer, heart disease, photosensitive medications.

There are limited studies looking at laser therapy, but many are in progress. Once human study should an improvement in pain relief for 2 months and up to 1 year after a two week protocol. A canine study showed similar results with weekly sessions for four to six weeks showing 70% of patients showing some improvement in arthritic pain and gait abnormalities.

The difference between commercially available laser unties lie solely in the wavelength, power density, pulse modulation and aesthetics. The goal is to stimulate the cell, and ultimately the body, to perform its natural functions, but at an enhanced rate.