Traditional Chinese Veterinary Medicine (TCVM), although relatively new to the Western world, is a medical system that has been used in China to treat animals for thousands of years. It is an adaptation and extension of Traditional Chinese Medicine (TCM) used to treat humans. Speaking broadly, Chinese Medicine is a complete body of thought and practice grounded in Chinese Daoist philosophy. Though it can be traced back over two millennia in recorded history, it, like any medical system, continues to evolve today, and current research on acupuncture and herbal medicine is beginning to shed light on its mechanism of action.

Chinese medicine theory
Chinese Medicine is based on the Daoist worldview that the body is a microcosm of the larger, surrounding universe. As such, the cosmic laws and forces that govern the external world also govern the body’s internal environment. Just as life-energy or “Qì” is an innate force of the universe, it too is a fundamental force of the body, driving its every action and transformation. Yin-Yang theory, which is central to Daoist philosophy, also features prominently in Chinese Medicine. This theory describes how opposing forces of the universe - light and dark, hot and cold, etc.- mutually create and transform each other, and play a key role in the characterization of physiological function and disease.

The Ancient Chinese observed yearly cycles through five seasons – spring, summer, late summer, autumn, and winter, which they corresponded to the Wu Xing, or Five Elements, consisting of Wood, Fire, Earth, Metal, and Water. Just as the Earth cycles through these five seasons, the body, too, passes through the five phases in its own life cycle. In this way, a young pup is said to be in its Wood (or spring) phase of life, while an old mare is said to be in its Water (or winter) phase. Moreover, the bodily organs have also been mapped to the five phases, and the Five Element Theory is used to explain the functional relationships between organ systems. For instance, the Kidney, corresponding to the Water element, is the “mother” of the Liver, a Wood element organ, because Water generates Wood in the way that watering a tree makes it grow.

Disharmony and disease
In Chinese Medicine theory, disease is understood as an imbalance in the body, and diagnosis proceeds through identifying the underlying “pattern” of disharmony. Pattern diagnosis differs from conventional Western medical diagnosis in that it takes into account not only disease signs but how these signs relate to the individual patient. Thus, TCVM practitioners will consider the temperament, sex, age, activity, and environment of an animal along with the animal’s particular disease signs. This approach stems from the belief that the body is as an interconnected system of forces and functions so that disease and disharmony must be examined with respect to the whole patient. For this reason, Chinese Medicine is often regarded as more holistic than conventional Western Medicine.

The four branches of TCVM
Once a particular type of disharmony or disease pattern is identified, treatment often proceeds through a combination of treatment modalities. Though the terms Chinese Medicine and acupuncture are often used interchangeably in the West, acupuncture is actually only one modality or “branch” of TCM and TCVM. There are actually four branches of TCVM – Acupuncture, Herbal Medicine, Food Therapy and Tui-na (Qi-gong, a form of Chinese meditative exercise, is a fifth branch of TCM that is excluded from TCVM because it cannot be performed by animals).

1) **Acupuncture** is a treatment that involves the stimulation of points, typically achieved through the insertion of specialized needles into the body. Acupuncture points typically lie along the body’s Meridian Channels along which Qì flows. Most veterinary acupuncture points and Meridian lines are transposed to animals from humans, though knowledge of some “classical points” defined on particular species have been retained and are used to this day.

2) **Herbal Medicine** utilizes herbal ingredients listed within the Chinese Herbal Materia Medica in particular combinations or formulas to treat particular disease patterns. Herbal formulas are administered orally and are typically given in powder form to horses and other large animals and in tea pill or capsule form to cats and dogs.

3) **Food Therapy** is the use of diet to treat and prevent imbalance within the body. It utilizes knowledge of the energetics of food ingredients to tailor diets for individual animals.

4) **Tui-na** is a form of Chinese medical massage in which different manipulations are applied to acupoints and Meridians to promote the circulation of Qì and correct imbalances within the organ systems.
“Integrative” medicine: TCVM and Western veterinary practice

TCVM is often viewed as a form of complementary therapy, and is best when used in conjunction with Western Veterinary Medicine (WVM). Both TCVM and WVM have their own strengths and weaknesses. TCVM is a holistic approach that is suited to assessing the well-being of the whole patient, and treatments are generally non-invasive with few side effects. However, TCVM lacks the tools necessary to pinpoint illness to specific disease-causing agents like pathogenic bacteria or viruses, and treatments are better suited for chronic conditions than acute ones. On the other hand, WVM utilizes the tools of modern science to diagnose disease with great precision, and Western drugs and procedures are powerful and fast acting. However, its insistence on detailed diagnosis may come at the expense of getting the larger picture. In many ways, TCVM and WVM each has what the other lacks. Thus, the best medical system involves the integration of the two systems, so that the strengths of one can compensate for the weaknesses of the other.

Selected references


Dr. Xie’s Veterinary Acupuncture. Huisheng Xie, DVM PhD Vanessa Preast, DVM PhD. Published by: Blackwell Publishing.
Kinesiology in Veterinary Medicine
Matt Brunke, DVM, CCRP, CVPP, CVA
North County Veterinary Referral Practice
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Kinesiology taping was developed by Dr. Kenzo Kase in Japan during the 1970’s. Although it has been around for over 40 years now, it is just emerging as a modality for canines and felines. It has been used in people and horses, and with the advent of more effective tape it can be used on small animals.

Kinesiology tape is completely different from traditional types of sports tape in the fact that it stretches along its length (but not across its width), allowing it to contour around body parts and allows joints to move through a full range of motion. Traditional tapes have been used primarily to limit movement, but the goal of using kinesiology tape is to promote proper movement.

Most tape is woven from a blend of cotton and nylon fibers in a pattern that allows it to stretch 180% along its length, but not at all across its width. It uses an acrylic, latex-free, essentially hypoallergenic medical adhesive on one side with a paper backing that allows it to be applied by practitioners without touching the adhesive.

Tissue decompression has two primary effects on the body. First, it relieves pressure from the free nerve endings in the tissues that are responsible for nociception (pain), so it can immediately reduce perceived pain. Secondly, the decompression action of the tape allows better circulation to and from the area taped.

The second major effect of kinesiology taping is the stimulation it provides to the variety of sensory nerves in the skin and underlying tissues. The skin and the connective tissue beneath it is filled with sensory receptors that are responsible for feeling light and heavy touch, ne point discrimination, pain, temperature and pressure. Additionally, some of these receptors serve a proprioception role, meaning they contribute to the brain’s sense of where the body’s parts are in space and throughout movement.

With kinesiology tape there is an alteration of the afferent signals going from the taped area to the brain. As a result, this changes the brain’s response to the incoming information, altering the efferent signals returning to the taped area. This neurological effect of taping is responsible for many of the beneficial effects of using kinesiology tape.

Contraindications
- Ingestion of the tape – potential GI obstruction
- Local infection/open wounds of the area
- Tumors
- Cardiac decompensation

Taping for pain mitigation
Goal
To provide adjunct pain relief, after other modalities such as laser therapy, joint mobilization, massage, etc..

Procedure
A three step process is used. 1) Stretch the body part, 2) apply a stabilization strip and 3) apply a decompression strip.
- Apply maximal pain free stretch to the body part to be taped. Apply an anchor end with no stretch. Then mild stretch in a longitudinal direction, again anchoring.
- The decompression strip is applied perpendicular to the stabilization strip. This is applied with moderate stretch in the center of the tape, and then with minimal stretch in either direction.
- For stifles and shoulders, flexion of the joint is needed for initial application

Taping for inflammation/Edema
Goal
To reduce inflammation from affected area as quickly as possible, enabling faster recovery and return to function.

Procedure
Cutting strips of tape into “fingers” or “tentacles” to provide space for fluid to leave the swollen region. Creating a cross-hatch or lattice design may be needed. Work circumferentially around the area when possible (as with distal limbs). An anchor strip around the tentacle strips may be needed.

Taping for neurosensory awareness/Posture
Goal
To improve sensory awareness, improve body posture.

Procedure
Application of tape along the dorsal surface of affected metacarpal and or metatarsal areas with mild stretch. This can provide a non-painful incentive to place the foot appropriately.
Additionally, “connecting the dots” can be done and taking tape to mimic the path of the nerve that is in dysfunction.

As with any procedure, proper safety is advised. Having an assistant appropriately restrain a patient may be needed to avoid injury to the rehab practitioner.

Depending on the reason for taping, kinesiology tape should be left on 12 hours – 3 days. Reapplication for some patients will be necessary.

- Using a waterproof tape is useful for those patients then undergoing underwater treadmill therapy.
- If a patient licks or chews at the tape, an Elizabethan collar is recommended.

To remove the tape, gentle rolling of the tape in the direction of the hair can be done. Ripping the tape off will not be pleasant to the animal.

- Rubbing alcohol or acetone may be needed to release the acrylic adhesive.
- Repeating palpation of sore areas to determine the effectiveness of the tape is advised.

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Elbrond, VS, Schultz, RM, Myofascia - The unexplored tissue: Myofascial kinetic lines in horses, a model for describing locomotion using comparative dissection studies derived from human lines... Medical Research Archives, 2015, Issue 3
Management of the Down Dog
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The “down dog” can arise (pun intended) from many causes. Some are orthopedic (bilateral CCL rupture) but the majority are neurologic in nature. They can be further broken down into acute and chronic conditions.

Acute down dog examples are IVDD, FCE, tick paralysis, trauma and metabolic conditions. Chronic origin examples are lumbosacral disease, degenerative myelopathy and diskospondylitis.

A thorough history and physical examination is a requirement to further understand these patients. A minimal data base (CBC/Chem/UA, plus Thyroid or Urine Culture if indicated) should be done on all patients. Survey radiographs can be extremely helpful in providing practical information. Referral for advanced consultation, imaging (CT/MRI) or other studies (nerve conduction) and surgical intervention (if indicated) may also be needed.

The rehabilitation practitioner may encounter these patients in the peracute setting, or after specialist intervention. Regardless of timing, they must be able to provide a baseline of practical and safe care to assist the patient. General practitioners can also provide a standard of care, and should also be aware of referral to a rehabilitation practitioner (CCRP or CCRT) or boarded rehab specialist (ACVSMR).

Establishing a baseline and then proper communication with the owner is vital. These cases can be “down” for 1-2 days or 1-2 months, or permanently. Understanding prognosis, as well as financial and emotional commitment to these patients is critical. The author recommends working in 2 week intervals, and not committing anyone to a 3 month timeframe. Continuous reassessment and communication is vital.

The team approach to these patients is critical. Establishing a relationship with the patient and the owner will allow for better understanding of the case, and of each patient’s needs. Some cases may be managed as outpatient, while some may require inpatient care. With outpatient, it is recommended to have the patient left for the majority of the business day, so as to slowly work with them. Inpatient care should be discussed and the owner made aware of who will monitor the patient overnight, and care for them on weekends.

Fundamental nursing care is essential for these patients. Changing recumbency every 4 hours (and attempting to keep sternal during daytime) is vital for both physical and mental well being. Clean soft bedding, with appropriate padding (to minimize risk for pressure sores) is needed.

Progressing to standing, items such as carts, harnesses or hoists can be quite useful to save the staff members. Working through assisted standing, partial assistance and then eventually standby assisted standing exercises will depend on each individual patient.

In between standing exercises (done 4-6x a day, or more) the patient should rest. Additional modalities to be used when the patient is resting includes, but is not limited to: LASER, passive range of motion, massage, thermotherapy, cryotherapy, and electrical stimulation.

Bladder and bowel expression may be needed. If so, it should be done in a safe manner. The author recommends avoiding urinary catheters (indwelling or temporary) after the initial 72-96 hours, so that determinations can be made on patient progress. Additionally, catheters provide an access point for infection. Urinary medications (phenoxybenzamine, bethanecol) should be used carefully, and after ruling out any infection.

Bowel expression may needed as well. This can be done in a variety of ways, including manual emptying. The important thing to remember is to give the patient TIME (in a harness, hoist, etc.) to void on their OWN. The author has found that many clinicians and especially owners are not patient enough to give these patients the time they need to void on their own.

For bowels, enemas, oral lactulose and movement (walking) can help facilitate proper bowel movements.

Referral from a general practice/ER to either a rehab practitioner or boarded sports medicine-rehabilitationist should be considered early for these challenging cases. The large breed dogs themselves present unique problems that may not be readily fixable in most practice settings.

Progress can be made with these patients slowly and with proper communication to the owner, reasonable goals can be set and attempted to meet.

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Therapeutic Exercises in Veterinary Rehabilitation
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Therapeutic exercises are the real “meat and potatoes” (or tofu and potatoes for you veggie folks) of veterinary rehabilitation. They consist of a variety of focused exercises that are intended to mimic real life work. Consider this to be the occupational therapy aspect of vet rehab.

The goals of these exercises are:

- Improve active pain free ROM
- Improve muscle mass and strength, balance, performance of daily function, aerobic capacity, prevent further injury.
- Reduce weight (when indicated) and lameness
- Important method to best return of function

Remember that prior to starting any exercise, signalment, history and a complete physical examination (including assessment and plan) MUST be done. Certain patients will have limitations that you need to be aware of. This is not a “cookie cutter” approach.

You can be creative in mimicking activities, for example, if you have agility equipment available, then setting up an easier course and having the dog WALK through that is building up exercise. Cavaletti rails, leashes, weave poles, balance boards are just some of the tools that can be used for ther ex.

Remember to use your environment as well.
- Stairs (traction, not scary)
- Couches (cushions)
- Air Mattresses
- Work outside (hills, sand dunes, tall grass, snow)
- Owner limitations? Have them sit at the table

I start with introducing myself to the patient, and not being in a rush. I find it easiest to start with the “down” patient (whether from orthopedic or neurologic issues) and progress through these exercises to the fully ambulatory patient that is need of fine tuning.

For those down patients remember the fundamental aspects of down dog care (clean, dry bedding, changing recumbency, etc.)

For completely down dogs we start with assisted standing exercises. Goals here are to: strengthen the patient, aid in proprioception, improve circulation and respiration, give them a chance to eliminate, are good for their mental well-being. Maximal assistance is needed to provide support 75-100% of the patient’s body weight. They cannot independently stand, and require a team effort. Place feet appropriately and use a sling, towel, Help Em Up Harness to achieve a “normal” position. Adjust for tolerance, but start with 20-30 seconds, per stand, 15 reps per set, 2-4x a day. Slowly increase the standing from 30 second to 5 minutes per session, pending how the patient improves.

Active assisted standing exercises follow, they get stronger let them do more, requiring <75% effort from us. Just enough support to maintain standing, physiorolls great for this, as are carts, hoists, etc.

Standby assisted standing exercises follow. Now has strength and motor to support against gravity, but are still ataxic or weak. You are right by their side, only there to prevent a fall. Once they can achieve rising and holding upright on their own, they may be ready for ambulation. Remember that during ALL of the phases of standing exercises you are doing proprioceptive training. That means that EVERY standing exercise the feet are placed appropriately, providing sensory feedback to the CNS.

For those patients that did not lose ambulation, this is where most of them come in. Proprioceptive training starts with the patient standing independently – time to do it right. Exercises here include 1) weight shifting, 2) unloading of a limb, 3) balance board and 4) exercise balls and rolls. Sit to stand and sit to down to stand exercises with good form are a great home exercise at this time. The patient is actively participating in their recovery.

Dynamic ambulation (aka WALKING) comes next. All walks must be on leash, with adult supervision. The pace must be dictated by the patient, but the handler must encourage the pace (not out smelling the roses, think power-walking). The handler may need to adjust their stride, we want walking, not running. This means for small breed dogs that people must walk extremely slowly, allowing the patient to use all four legs. Otherwise they will run to catch up, and not weight bear and strength train on the limb (think about your FHO patients).

As the patient improves, variety can be taken with different exercises, based on the patient’s needs and goals. This can include:

- Egg-crates, Foam Rubber, Air Mattress
- Couch cushions (shifting balance)
- Stairs – 5-7 steps, 2-4x a day for starters (on leash)
- Pole weaving, tunnels, pulling weight
- Ankle weights
• Syringe cap (on contralateral foot)
• Cavaletti rails

Start with 3-4 simple exercises per session, and always introduce it to the patient, then the owner, before having the owner do it at home. If the patient is improving, increase EITHER the time to a particular exercise by 10-20% each week.

If they are painful? Pause, address, start back up slowly. It may be related to their surgery or be a consequence from their lack of ambulation for a prolonged period of time. Objective outcomes are key, so re-measure – girth, stance analyzer, goniometer. Smart phone apps can be used by owners to track how far/fast they are walking the dog.

A practical, multimodal approach to therapeutic exercises will result in a better patient.

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My dog doesn't have Obamacare and can't wait for the government to make up its mind: What can I do about the torn cruciate?
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My dog doesn't have Obamacare and can’t wait on the government to make up its mind; what can I do about the torn cruciate?

Cranial cruciate ligament (CCL) rupture is one of the most common orthopedic conditions encountered in the dog. In fact, over 1 billion US dollars are spent every year in dealing with the canine stifle. When dealing with hind limb lameness many dogs we see have some degree of hip dysplasia or degenerative changes in the hip; however, an acute lameness is typically not due to a hip problem. In fact 32% of dogs referred for hip problems actually have evidence of cruciate disease. About 33-50% of dogs will present with bilateral disease even if they have a unilateral lameness. Severe bilateral cruciate disease can often mimic other conditions such as severe hip dysplasia or neurologic disease. Therefore, a general rule of thumb is a hind limb lameness in a dog is cruciate disease until proven otherwise.

Personally for us, statements that we do not like are:
- All dogs that rupture their CCL must have surgery
- All dogs with CCL ruptures have joint effusion
- All surgical procedures (extra-capsular repair, TPLO, TTA, XYZ) have the same outcome
- A dog can’t return to pre-injury status following a CCL rupture
- Dogs don’t benefit from rehabilitation therapy either with a conservative approach or following surgery

Diagnosis
The diagnosis is typically straightforward and is based off the history, signalment, clinical signs, physical exam, and orthopedic exam. The history may include an acute or chronic hind limb lameness that may be mild to non-weight bearing. Interestingly, owners may report that the lameness has improved from initial injury. This usually corresponds to the timeframe from when the initial inflammatory response is ending. Regarding the signalment any age or breed can be affected. Typically we tend to see medium to large breed dogs that are around 3-8 years of age. The orthopedic exam is mainstay to diagnosing a CCL rupture. Findings may include a positive sit test where the dog will tend to sit with the affected leg projecting out to the side. Pain on hyperextension is usually the forgotten test but is very reliable. Most affected dogs will exhibit some degree of pain. Crepitus may be noted during ROM, and with chronic tears medial buttress formation may be noted. This is the peri-articular fibrosis that occurs. The classic findings for a CCL rupture are joint effusion, the cranial drawer test and the tibial compression test. A simple way to think about it, is that an adult dog joint effusion will only be caused by a CCL rupture, septic arthritis, tick-borne disease, or immune-mediated arthritis. A medial patella luxation (MPL) will not cause the same degree of joint effusion, so if you have a patient will underlying MPL that develops joint effusion be thinking about a CCL rupture.

The cranial drawer test is testing for laxity in the CCL, but this is more of a passive test and does not mimic weight bearing. To perform the test one hand is placed on the distal femur with the thumb behind the lateral condyle. The other hand is placed on the proximal tibia with the thumb behind the fabella. The goal is to move the proximal tibia cranially in relation to the femur. Always check drawer in flexion and extension. When checking for partial tears the CCL has two bands, the craniomedial which remains taut in both flexion and extension and the caudolateral, which is taut in extension but lax in flexion. For example if the craniomedial band is torn and the caudolateral band is intact cranial drawer is only present in flexion because in extension the caudolateral band is taut. If the caudolateral band is torn and the craniomedial band is intact no cranial drawer is present because the craniomedial band is taut in both flexion and extension. Cranial tibial thrust is a test meant to mimic active weight bearing. The goal is to hold the stifle at a standing angle (approximately 135 degrees) and while holding the stifle still flex the hock. If the CCL is ruptured there should be a cranial displacement of the tibia. As with cranial drawer, tibial thrust should be checked in both flexion and extension.

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

Treatment
When deciding on a treatment plan there is no one treatment fits all, but there are many, many, many options available. The reason there are so many options is because not one procedure or medical management technique is 100% perfect. I think one reason for this
is because what is considered our final outcome, a stable stifle, a patient that returns to activity pain free, elimination of OA, owner satisfaction, etc.? We will never be content on cruciate disease until we figure out the goals we want to achieve for an outcome.

When I approach a dog with cruciate disease I’m going to have the same conversation with each owner; however, depending on each case I may swing my conversation in one particular direction. Factors I consider when deciding on conservative vs. surgical treatment and which procedure are the patient, owner, and veterinarian factors. I look at the breed, the size of the animal, the age, the activity level, and what is that particular animals job. Are they a pet, an athlete, or a service dog? Regarding the owner I talk to them about their perceived outcome, their ability and willingness to follow directions post operatively, as well as finances. And then I look at my abilities such as what equipment I have available, what procedures am I comfortable doing, and what good and bad outcomes have I had with certain procedures.

When I first tell owners that their dog has a torn cruciate I try to cover 3 main options. Option 1 is we do nothing. By do nothing I mean we cage confine for 6 weeks with medical management (analgesia and NSAIDS) and (hopefully) formal rehabilitation therapy. The most important aspect here is confinement. These owners have to be aware the goal of conservative management is to allow peri-articular fibrosis to occur. This can’t occur with the dog remaining active. To break it down to them I tell the owners the dog must be kept in an area where he/she can stand up, lie down, and turn around. The dog eats, drinks, and sleeps in the crate. It only goes outside to urinate and defecate on a leash then back into the crate. I also throw the disclaimer in that in my opinion OA is worse with a rapid progression as long as the stifle is unstable and usually if this is a larger dog they wont return to full function. I also really push the fact that the dog will appear to be do “okay”; however, they have a very high chance of developing a meniscal tear. I tend to tell owners its not “if” but more of a matter of “when” they tear their meniscus. Personally, I am not a fan of this approach!

Option 2 is a conservative approach with exercise restriction, formal rehabilitation therapy, and a custom made stifle orthotic. While this approach parallels that of option 1, we can in theory attempt to help stabilize the stifle with a brace. In human medicine, knee braces are commonly used for multiple conditions. Bracing of the human knee has been shown to enhance proprioception/joint position sense, permit the injured limb to relax, reduce fatigue in injured limb, provides some mechanical protection against impact, and slow movement down to allow muscles time to react and control motion. Categories of knee braces in human medicine include the following: prophylactic (prevent or reduce severity of knee injuries in contact sports), functional (provide stability for unstable knee), rehabilitative (allow protected and controlled motion during the rehabilitation of injured knees), and patellofemoral (improve patellar tracking and relieve anterior pain). Only functional knee braces are utilized in veterinary medicine.

In theory the brace should help limit tibial subluxation. At the authors institution (unpublished data) we did find improved objective gait analysis when a custom stifle brace was worn versus when not worn; however, the gait analysis was not improved equal to that of surgery. This data reveals that a brace is not considered equal to or meant to replace surgery; furthermore, it must be worn for the duration of the pet’s life.

Option 3 is the grey zone. It may be the morbidly obese dog that needs to lose weight before surgery. It may be the family that is saving up for the TPLO, but wants to do something prior to surgery. Or it can be any other number of reasons. The focus in this area is education. Client education is critical with regards to many aspects of their pet. This includes obesity reduction (through diet and exercise), pain mitigation (through NSAIDS and other oral medications), chondroprotection (through oral supplementation of Glucosamine, Chondroitin, Omega-3 FA, etc.. Seeing the big picture with these pets is needed to achieve a favorable outcome. Without it, you eat a fat, immobile dog in chronic pain.

Obesity: Checking thyroid function on all overweight dogs is recommended. If it is normal, then using a prescription diet tailored for obesity and arthritis is recommended. (MB uses Hills Metabolic and Mobility in his practice). Once initial inflammation is reduced (usually with NSAID, 1-2 weeks after injury) a rehab program of underwater treadmill walking is initiated. The goal here is to use the buoyancy of the water to reduce stress on the unstable stifle. This will allow for reduction of muscle atrophy and caloric burn without further injuring the patient. A protocol of 10-30 minute walks 2-3x a week (progressively building) is recommended. If possible, a photobiomodulation (therapy laser) treatment protocol is also initiated at these visits, with the goal being to reduce inflammation and promote tissue healing.

An at home program can include short leash walks, but a focus here on therapeutic exercises is used instead. Once initial inflammation is reduced, working on appropriate sit to stand exercises with the stifle in appropriate position is attempted. 10 reps per set, 10-2 sets per day. Initially we aim for 5/10 reps to be square, and then each week increasing our goal by one. The affected stifle should be against a wall, so as to minimize outward rotation. The goal here is fairly quick succession throughout the set. As soon as the patient ischium touches the ground the dog should be asked to stand again and repeat the exercise. Stretching and range of motion can begin at 1-2 weeks post injury, and then continue daily through week 8. The goal here is to maintain range of motion of the joint and minimize contraction.

The walks should be at a slow pace, on leash and on flat surfaces with good traction, Hill work and turning quick corners is not added until weeks 4-6.

Around weeks 4-6 as the fibrosis is being achieved, core strengthening with balance disks and wobble boards can be done in a slow safe and professionally administered manner.
My issues with stifle orthotics are as follows:

1. **Tolerability:** I can't ask the patient if he/she will tolerate the brace, I have had some dogs that don't mind it at all, others take time, and some just freeze or try to chew it. The other issue is given the different shapes and sizes of dog stifles the brace MUST be custom made. This means a mold must be made and sent to the orthotist and then sent back about 2 weeks later. It’s a horrible feeling to have an owner pay the expense for a brace and then the dog won’t tolerate it.

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

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

Cruciate disease is a complex problem that does not have a clear cut answer. Ultimately, surgical intervention may be needed in any case that is initially managed without it. Proper education of the client is critical to establishing favorable outcomes.

**References**

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

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

Increased body weight is not a cause of HD, but it certainly has very important clinical consequences in susceptible dogs. Therefore, weight reduction is an effective preventative strategy. In the lifespan study of 49 Labradors it was reported that heavier dogs (dogs allowed to eat ad lib) developed radiographic OA on an average of 6 years earlier than the dogs in the restricted fed group. Furthermore, heavier dogs required long-term treatment for OA on average 3 years earlier than their restricted fed littermates.2

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

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

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

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

Physical rehabilitation has a multimodal approach within itself for managing HD. Physical modalities, manual therapies and therapeutic exercises can all be used to achieve relief from HD. Goals of rehab for the patient include: maintaining or improving muscle mass, building muscle support around the lax or arthritic joint (and all joints), reducing pain and weight loss (via exercise, when indicated).

Physical modalities can include thermotherapy (the use of cold and warm packs). The benefits of cryotherapy are established (pain relieving, vasoconstriction, etc.) and warm compresses can be used to relieve pain, cause vasodilation and also help to warm up stiff, tight tissues to begin other exercises.

Therapy LASERs (Light Amplification by Stimulated Emission of Radiation) have become very popular in recent years. There are different wavelengths, amplitudes, treatment times and other factors that must be considered. This process has also been called photobiomodulation. It has been proposed to activate cytokines and other tissue factors, decrease pain and inflammation and increased wound healing. Always use goggles for both the humans and patient to avoid damage to the eyes. It cannot be used over pregnancy or cancer.

Manual therapies are skilled hand movement techniques intended to: improve issue extensibility, increase range of motion (ROM), induce relaxation, mobilize or manipulate soft tissues and joints, modulate pain and reduce swelling and inflammation. These can include massage and joint mobilizations. The basic principles of joint mobilizations work from physiologic motions and accessory motions. Physiologic motions are normal active motion that is available at a joint. Examples: flexion, extension, abduction, internal rotation, etc. Accessory Motions are movements that cannot be performed actively. Examples: distraction, compression, glides, spins and rolls. There are 4 grades of mobilization, and the manipulation (used in chiropractic) is a 5th grade. Grades 1-4 are passive movements, with 1 and 2 not reaching initial resistance of the joint end feel. Grade 3 moves through the initial resistance to the end feel, but does not exceed it. Grade 4 mobilizations are compact with in the first and second resistance points. Grade 5 (manipulations) exceed the normal end feel of a joint.

Therapeutic exercises are the “meat and potatoes” of rehabilitation. These are designed to work a patient from a recumbent position back to normal (or as close as possible) activity following injury or insult. Exercises in this group can include cavalletti rails, working on balance boards, disks or other core strengthening equipment. Once walking on a flat non-slip surface is achieved, adding varying degrees of difficulty (up hills, through different traction, etc.) can be included. Sit to stand exercises and core strengthening with dancing exercises are also helpful. The key is to keep the patient moving and building.

Land treadmills – Can be useful devices for providing exercise. Small and medium dogs will work well on a human machine, but larger dogs will benefit from a canine treadmill. This is due to stride length and length of the belt. Having the dog walk on an incline will help build up the pelvic limbs.

Underwater treadmills – Can be used as both a diagnostic and therapeutic tool. The buoyancy of water will allow severely affected animals to utilize their limbs. There are also studies showing the benefit of underwater treadmill therapy for reducing obesity in dogs. With water at the level of the hock, there is a 9% reduction in perceived body weight, a 15% reduction with water at the stifte, and 62% reduction when at the greater trochanter. The non-slick, safe, contained surface an underwater treadmill provides is superior to walking in ponds, lakes or swimming in pools, in the author’s opinion.

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

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

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

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

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

The physiology of cartilage is important because damage to chondrocytes will not only lead to death of that particular chondrocyte but also an inflammatory response that creates problems with neighboring chondrocytes. Thus a downward, progressive spiral occurs which leads to destruction of the “work-horse” (chondrocytes) and loss of extracellular matrix production. The loss of ECM production leads to the loss of cartilage’s ability to soften and transfer loads to the underlying subchondral bone.

The pathophysiology of OA is described as a non-infectious disorder of diarthrodial joints. It is categorized by deterioration of articular cartilage, bone formation at synovial margins (osteophytes), peri-articular fibrosis, and a localized inflammatory response. For OA to develop there has to be some insult to the articular cartilage such as hip dysplasia, a cranial cruciate ligament tear, elbow dysplasia, or an articular fracture. Once the chondrocyte is damaged the inflammatory cascade begins and is followed by the release of multiple cytokines. The two main cytokines involved with OA are interleukin 1 beta (IL-1β) and tumor necrosis factor alpha (TNF-α). IL-1β is responsible for the breakdown of the matrix, while TNF-α drives the inflammatory response. Furthermore, prostaglandins are released, particular prostaglandin E2 (PGE2), which increases the release of metalloproteinases (MMPs). MMPs are responsible for the continued breakdown of the ECM.

In summary of OA inflammation: Osteoarthritis is a chronic progressively destructive disease that involves the entire joint. Inflammation is a key component of both joint destruction and pain. Acute pain resolves after the initial injury heals. Chronic pain involves structural changes of the dorsal horn, is more intense than acute pain and more difficult to control. Treatment considerations for osteoarthritis should address inflammation as well as pain.

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

Radiographs are key to aiding in the diagnosis of OA. However, just as with any diagnostic modality there are limitations. Radiographs only provide bony information, they are taken in a non-weight bearing position, and osteophytes are useful to diagnose OA but they are not pathognomonic for OA. Furthermore, the value of osteophytosis for staging OA is controversial and does not correlate with OA progression. Probably the biggest issue with radiographs is that they do not correlate with clinical signs. The radiographic key features of OA are: osteophytosis, enthesophytosis, effusion, soft tissue swelling, subchondral sclerosis, intra-articular mineralization (especially in cats), and subchondral cyst (rarely seen).
Other additional diagnostic modalities include CT, MRI, and arthroscopy. Arthroscopy is probably the most valuable means to objectively evaluate the cartilage. However, it is a surgical procedure and can be costly to perform. It does allow the evaluation of the cartilage, which can then be classified by the Modified Outerbridge score. One looming question is if you don’t perform arthroscopy and radiographs are helpful to diagnose but don’t help stage for monitoring for progression of OA is there some type of subjective based assessment? The answer is yes, the Canine Orthopedic Index (COI) was developed and validated in 2014 to provide reliable assessment of dogs with OA in terms of staging as well as response to treatment. It can be downloaded at www.canineorthopedicindex.com.

A multimodal approach to OA management is needed. Non-steroidal Anti-Inflammatory Drugs (NSAIDS) represent the cornerstone of therapy, but other modalities include: nutrition, chondroprotectants, additional analgesics, physical rehabilitation, weight control, exercise, an EPA rich diet and many new and emerging options. Let’s look through these individually.

Obesity is a growing issue in veterinary medicine. The effects of obesity on OA are twofold. Biomechanical stress contributes to clinical signs and progression of disease. Adipokines secreted by white fat cells contribute to the progressive inflammation of osteoarthritis. Leptin levels are elevated in obese dogs. In humans with osteoarthritis, increase leptin levels correlate with elevated MMPs and NO in synovial fluid. Adiponectin is anti-inflammatory, but levels are low in obese dogs. In human patients with knee osteoarthritis there is a significant correlation with adiponectin: leptin ratios.

Humans with increased body mass index (BMI) experience OA in non-weight bearing joints, which resolves with weight loss. Decrease in BMI, is associated with symptomatic relief from knee OA in man. Systematic review of canine studies found that preventing obesity decreases incidence of OA and weight loss reduces signs of OA. Additionally, diets rich in Omega-3 fatty acids have shown to be beneficial for both dogs and cats with OA. Additional nutritional supplements such as glucosamine, chondroitin, methylsulfonylmethane (MSM) and others have been shown potentially beneficial for our patients.

Physical rehabilitation has a multimodal approach within itself for managing OA. Physical modalities, manual therapies and therapeutic exercises can all be used to achieve relief from OA. Goals of rehab for the DJD patient include: maintaining or improving muscle mass, building muscle support around the arthritic joint (and all joints), reducing pain and weight loss (via exercise, when indicated).

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Disease modifying agents for OA are next to be discussed. Polysulfated glycosaminoglycan is FDA approved, disease modifying osteoarthritis drugs; for dogs and horses; water-based, for intramuscular injection Dosage: 2 mg/lb body weight, IM, twice weekly for up to 4 weeks (maximum of 8 injections). MOA: specific is not known; in vitro studies show; they inhibit serine proteinases; PGE2 synthesis; metalloproteases, hyaluronidases and others. Stimulate synthesis of protein, collagen, proteoglycans, and hyaluronic acid. There are studies showing it reaches feline cartilage via subcutaneous injection. This is extra label usage for this medication. Also, maintenance injections have been anecdotally reported for both dogs and cats. Clinical studies on PSGAGs showed both good efficacy and safety. Treated dogs had statistically significant improvement in range of motion and total orthopedic score over placebo treated control dogs. 2.1% of dog had adverse reactions including: transient pain at the injection site (1 incident), transient diarrhea (1 incident each in 2 dogs) and abnormal bleeding (1 incident). These effects were mild, self-limiting; did not require interruption of therapy. Do not use in dogs showing hypersensitivity to PSGAG, or in dogs with known or suspected bleeding disorders. Use with caution in dogs with renal or hepatic impairment.

Adjunct analgesics for OA are numerous. They are used in addition to or replacement for NSAIDS. Research is scant on some of them. Amantadine – only drug studied to treat canine osteoarthritis. In dogs with osteoarthritic pain refractory to an NSAID, addition of amantadine improved physical activity. Amantadine might be a useful adjunct therapy for the clinical management of canine osteoarthritic pain. It can be dosed at 3–5mg/kg SID. Gabapentin – Calcium channel modulator – 5–10mg/kg SID-TID. Amitriptyline 0.5-1.0mg/kg SID-BID – cats and dogs. Local anesthetics – Lidocaine, bupivacaine, mepivacaine. Acetaminophen can be used in dogs.
but not cats. Opioids – morphine, meperidine, methadone, oxymorphone, hydromorphone, fentanyl, fentanyl patches, butorphanol, pentazocine, nalbuphine, buprenorphine, codeine and tramadol. Tramadol’s metabolism and elimination is rapid and variable among dogs. When administered orally or intravenously to the dog, metabolism of tramadol and all metabolites is rapid. There is much variability between dogs, possibly breeds. Pain control did not necessarily correlate with plasma levels of the active metabolite (O-desmethyltramadol). Tramadol effects on α-adrenergic or serotonin receptors may contribute to analgesic effects in the dog. Regardless of mechanism of action, studies suggest oral dose should be 5 mg/kg q 6 hours or 2.5 mg/kg q 4 hours. In the author’s opinion this is a very challenging drug to utilize effectively in practice due to these variables.

Galliprant is a first-in-class non-cyclooxygenase (COX) inhibiting, non-steroidal anti-inflammatory drug (NSAID) in the piprant class. Piprants are a newly recognized drug class, established and defined by the World Health Organization in 2013 as prostaglandin receptor antagonists (PRA). Unique mechanism of action by antagonizing the prostaglandin E2 (PGE2) EP4 receptor. PGE2 its physiologic effects through binding of four different receptors, EP1, EP2, EP3 and EP4. The EP4 receptor has been identified as the primary receptor responsible for mediating pain and inflammation associated with osteoarthritis. Galliprant selectively blocks the EP4 receptor, thus blocking PGE2 elicited pain.

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

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

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