The physics of water are important to understand in order to utilize hydrotherapy. Topics covered here will be relative density, buoyancy, hydrostatic pressure, viscosity, resistance, surface tension and water temperature.

**Relative density**
Ratio of the weight of the object to the weight of an equal volume of water. The specific gravity (just like in urinalysis) of the different types of tissues is important. Water Usg 1.0. Fat (0.8), Muscle (1.0), Bone (1.5-2). This will determine how well a pet will float. Fat dogs will float easier, while muscular dogs will be neutral. Very thin dogs (primarily made of bone) will sink.

**Buoyancy**
Will provide the upward thrust on the patient and is dependent on how much is submerged. Gravity and Buoyancy need to be in the same vertical plane otherwise get all tipsy. Studies have shown that water at the level of the malleolus in dogs will have a 9% buoyancy effect, while at the stifle will have a 15% effect, and at the level of the greater trochanter a 62% effect. This means that a 100 lb dog will “feel” like they weight 91 pounds, 85 pounds and 38 pounds respectively with water at those heights.

**Hydrostatic pressure**
The deeper a tissue is submerged, the more pressure the water will have on it. SCUBA diving is the classic example here. Why do we care? More pressure is GOOD for edematous tissues and swollen joints. This may decrease pain during exercise – stimuli of the skin sensory receptors that decreases their hypersensitivity. It may be HARMFUL for the pet with decreased cardiovascular output!! (Chest submerged). Consulting a cardiologist for any animals with compromised cardiovascular output is recommended prior to hydrotherapy.

**Viscosity**
The viscosity of the water can allow for improved sensory awareness and some resistance to keep a weak patient upright (vs. falling to one side or the other).

**Resistance**
Movement through water (either it not flowing, or more so flowing OPPOSITE the direction of movement) can cause resistance, which will lead to increased cardiovascular output and increased muscular strength.

**Surface tension**
Having to break the surface of the water requires more energy than working below the water. Therefore targeting a joint to repeatedly break the surface of the water will require increased force and therefore a harder workout.

**Water temperature**
Cold water can cause vasoconstriction and put more pressure on the heart. Hot water will cause vasodilation and decrease blood pressure. This can also be problematic for patients with cardiovascular disease. Warm water – increase tissue elasticity, improve blood flow, increase cell metabolism, pain relief and relaxation. It is a fine balance – we do not want heat stroke and some arthritic joints flare up in warmer temps. Too cold will tighten tissues and not allow for improved range of motion.

**Equipment**
Towels, life jackets, trout waders, wetsuits, bathing suits could all be needed before getting a patient in the water. Additionally pool chemicals, pool skimmer, water wings and pool noodles can be needed.

**Water**
Water can be provided in oceans, lakes, streams, ponds, pools. Some of these will have variable surfaces that may or may not provide appropriate traction. The temperature can also be quite variable with these options. Seasonal availability can also change with these. They can be challenging to provide a consistent and safe workout for the patient. An underwater treadmill, in the author’s opinion, provides the safest and most consistent option for patients.
Underwater treadmill benefits
They are safe – the patient is contained with their feet always touching floor which is provides traction. It is easy to acclimate – tech, life jacket, treats, 5 minutes. Many of the variables in hydrotherapy can be controlled in an underwater treadmill such as: depth of water, speed of belt, temperature of water (and environment). All of this information must be tracked in the medical record. Some underwater treadmills have resistance jets, can run at an incline, and different belt lengths are available. Some use different amounts of electricity to run and heat to water.

Who can benefit?
Many different patients can benefit from working in an underwater treadmill. Examples are OA, obese, orthopedic cases (CCL, MPL, FHO, THR, amputees), neurologic patients (FCE, IVDD, vestibular), athletes (agility, law enforcement) along with dogs that need exercise in general. Clients will see the benefit and seek out your practice above others in the area. Your practice will benefit by adding a new service, setting yourself apart from other practices in the area, and creating a profit center that has the ability to generate referrals from other general and specialty practices in the area.

What are the benefits/risks?
- Benefits: Increase in joint flexion/extension – H2O at target joint
- Decrease in pain
- Increase blood circulation
- Increase sodium excretion by kidneys – help reduce edema
- Increase heart rate and oxygen uptake
- Increase in metabolic requirements – need regular workouts for weight loss
- Muscle strengthening
- Improvement in proprioception
- Useful transition to land based exercise

Contraindications/precautions
- Severe pain – “wind up” alldynia, hyperalgesia
- Surgical incisions or open wounds – infections
- Skin infections – maybe make worse?
- Cardiovascular problems – increase in CV output, compression of water (less strenuous than swimming)
- Incontinent or GI problems – makes a mess in the UWT!

How do you get started?
Discussing with the various vendors the cost, space and availability of their treadmills is step one. Allocation of appropriate space for both the treadmill and the storage tank will be needed. Purchasing of the needed accessory equipment (towels, bathing suits, life jackets) should be done as well. Designating staff and having them complete appropriate training to diagnose, prescribe and monitor the workouts will be needed. Generating a caseload can come from both internal and external sources. Internal referrals should always exceed external. Patients in the clinic currently diagnosed with OA or obesity are good starting points.

Selected references
Bockstahler B, Levine D, Millis D, Essential Facts of Physiotherapy in Dogs and Cats: Rehabilitation and Pain Management, BE VetVerlag, Germany, 2004
Osteoarthritis (OA) also known as degenerative joint disease (DJD) is the number one cause of chronic pain in dogs in the US. It has been concluded that 20% of dogs have some degree of OA/DJD. This results in decreased activity, weight gain, negative impact for the patient, a change in the human animal bond (negatively) and an increase in euthanasia in dogs that become refractory to treatment.

It is critical to have options to treat this disease. Identifying patients within your practice that have OA/DJD can be done quite easily. A complete, thorough physical exam and proper history on each and every patient coming into an office call should be done. Asking questions about mobility (Trouble going up the stairs? Not playing as much? Slowing down?) should be included. Proper flexion and extension of all the joints of the limbs and palpation of the spine, flexion of the neck, checking for joint effusion and instability of joints can easily clue the clinician to finding potential OA patients. Signs such as muscle wasting, asymmetry, lameness should also be looked for. Cartilage damage occurs well before radiographic changes.

Initial trauma = fibrillation of cartilage surface, damage to chondrocytes, release of:

- Cartilage degradation products (CDPS), Matrix metalloproteinases (MMPs), Nitric oxide (NO) and inflammatory cytokines.
- MMPs/CDPS are engulfed by synoviocytes, which release: Inflammatory mediators: PGE2, TNF, IL1β and MMPs. All contribute to cycle of inflammation, degradation and pain of osteoarthritis.

Other changes that occur in the process are thickening of the joint capsule, remodeling of subchondral bone which leads to sclerosis. Osteophyte formation occurs via synoviocytes releasing Bone Morphogenetic Proteins (BMPs). Periosteum mesenchymal stem cell to differentiate into chondrocytes; initiate osteophyte formation from which a joint mouse or the osteophyte impinges on periosteum. Bottom line: OA becomes a vicious cycle. The joint is less able to bear stress and forces leading to further joint damage and the initiation of clinical signs.

This ongoing inflammation is the source of both the progressive nature and pain of OA. Prostaglandin E2 (PGE) and other inflammatory cytokines lead to: progressive degradation of the joint and stimulation of nociceptors in synovium, joint capsule and subchondral bone. PGE is also released in dorsal horn and contributes to the pain signal.

The neural aspect of the joint is also a source of pain. Cartilage is an aneural; cartilage damage alone is not painful. Inflammation stimulates nociceptors found in: joint capsule, synovium, subchondral bone and the periosteum. The joint nociceptors intimate pain.

Neurogenic inflammation is an additional step in the nociceptive pathway. Involve the antidromic release of neurotransmitters near the joint. Neurotransmitters such as Substance P trigger inflammatory mediators within the joint. This contributes to joint pain and inflammation.

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.

A multimodal approach to OA management is needed. Non-Steroidal Anti Inflammatory Drugs (NSAIDS) represent the cornerstone of therapy, but other modalities include: nutrition, chondroproteoctants, 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).
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 cavalette 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. Other modalities in this group can include walking on treadmills or underwater treadmills.

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; metalloproteinases, 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 analgesic 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 NSAIDs, addition of amantadine improved physical activity. Amantadine might be a useful adjunct therapy for the clinical management of canine osteoarthritis 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.

Intraarticular medications can provide targeted therapy for OA. These can be easy to learn with practice. In the author’s practice it is done under sedation, and after radiographs to verify the OA process. This also allows for elimination of infection or neoplasia as 464
mimics of OA pain. Diagnostic arthrocentesis could be done if indicated. The same approaches to joints for diagnostic purposes can be utilized for treatment. Removal of some of the joint fluid allows space for the infusion of medication. These need to be done with sterile technique for obvious reasons.

Options for intraarticular therapies include corticosteroids, hyaluronic acid and regenerative medicine (platelet rich plasma or stem cell therapy). Corticosteroids can have deleterious effects to cartilage, but these are often used in palliative care, or if other options are not financially available. Methylprednisolone acetate has historically been used in these cases for both dogs and horses. A 20mg dose is used for most medium to large breed dogs. I prefer to use triamcinolone which has been shown to have less damage to equine cartilage than methylprednisolone. Additionally, if any systemic side effects happen from corticosteroids, triamcinolone’s effects will be over faster than methylpred. This is especially important in patients with severe mobility issues (less chance of urine scald, accidents in the house) and potentially other metabolic diseases (renal, hepatic impairment).

Hyaluronic acid increases viscosity of joint fluid, is anti-inflammatory, analgesic and they induce production of synovial fluid. Many different options are available, and molecular weight is the primary variable among them. Cost can be variable as well, and some products are not currently available. In my practice Legend (10mg/ml) is used. Usually 20mg for large breed dogs, 10mg per joint for small breeds. It can be combined with a steroid if needed for both a short term and long term effect. Repeating injections in a few weeks to months may be needed in some cases.

Regenerative medicine is an emerging field for both animals and humans. There are many variables with products and product preparation. In clinic and outside laboratory preparations are available. Each has their own pros/cons to be considered by the clinician. Currently either requires patient donation or baseline cells. An over-the-counter synthetic product is not yet available.

Platelet Rich Plasma has positive effects on angiogenesis and extracellular matrix remodeling, provides fibrin for matrix, a potent source of growth factors, cell proliferation and differentiation and stem cell recruitment and chemotaxis.

Stem cells can be of bone marrow or adipose tissue origin. Research is not clear as to which is superior at this time. Stem cells are reported to contribute to generating new tissue, chemotactic for progenitor cells, supply growth factors, make extracellular matrix, angiogenesis, anti-apoptosis, anti-inflammatory and are anti-fibrotic.

ESWT stands for extracorporeal shock wave therapy. Short duration acoustic waves at low frequency and high pressure. 100x atmospheric pressure is reached in microseconds. Mechanisms lacking – but reported is reduced inflammation, improved vascularity, neovascularization, increase bone formation, realignment of tendon fibers and enhanced wound healing. Improved weight bearing and comfortable ROM similar to NSAIDs. Heavy sedation or anesthesia usually required, and repeating treatment q 2-3 weeks for 3-4 treatments, and no concurrent NSAIDS during treatment.

PEMF is pulsed electromagnetic field therapy. This delivers a micro current that targets the body’s own natural anti-inflammatory process. The electrical signal is deposited to the tissue that mimics physiological stimulus that occurs in healthy tissue, thus stimulating tissue repair. It enhances nitric oxide production to help speed healing of tissues. It can be used by the owner at home in between rehab sessions, or as an alternative if other modalities are not available.

TCVM or Traditional Chinese Veterinary Medicine has been available for thousands of years. While herbal and food therapy are options, the most research is available for acupuncture. Acupuncture, the placement of small sterile needles at points along meridians has been shown to have a pain relieving effect. Dry needling, aqua puncture (the placement of small amount of a fluid), gold bead therapy and electro acupuncture are all options for stimulating the acupoints. Acupuncture has been shown to release the body’s natural endorphins and this is the method of analgesia.

Kinesiology taping is an emerging modality in people and animals. This tape lifts on the fascia and can improve blood flow, lymphatic return and pain relief. The proposed mechanism of analgesia is working along the gate theory, stimulation of nerve fibers by non-painful stimulation allows the body to “close the gate” to those nerve fibers transmitting pain from noxious stimuli.

A trial and error approach is sometimes needed to find the right combination of methods to control the pain of osteoarthritis. Having comfort with numerous, but not necessarily all, of these techniques can help the clinician and the patient.

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NSAIDs in Dogs and Cats: The Basics and Beyond
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Non-Steroidal Anti-Inflammatory Drugs (NSAIDS) have historically been used for mild and chronic pain. In humans they are associated with gastrointestinal side effects. Hippocrates showed their benefit when he would prescribe willow tree bark which contains NSAIDS. In 1971 aspirin was the first drug first classified with NSAID actions.

They are a potent anti-inflammatory with central and peripheral effects. They can provide analgesic and anti-pyretic effects as well with a lack of developing dependence on them or the other unwanted side effects of opioids. Being non-steroidal, they still have a glucocorticoid-like end effect, in that they lead to decreased prostaglandin production.

In veterinary medicine their history starts with phenylbutazone, flunixin meglamine and have evolved into carprofen, meloxicam and deracoxib (to name a few). Some generalizations are that: 1) rapidly absorbed by the GI, 2) Highly protein bound, 3) Weak acids – accumulate in inflamed tissue, 4) Metabolized in the liver +/- enterohepatic cycling, 5) Half-lives vary from 2-72 hours and these effects differ in half life and route of administration.

Their method of action runs through the arachidonic acid pathway. By doing this they will have effects by 1) decreasing production of prostaglandin through cyclooxygenase (COX) inhibition, 2) neutrophil inhibition, 3) glycosaminoglycan synthesis exacerbation and 4) metalloproteinase inhibition. Their effects are seen on the pain pathway in transduction, modulation and perception.

Which patients in your clinic can benefit from NSAIDS? Any of those going through osteoarthritis, orthopedic or soft tissue surgeries, dental extractions, biopsy procedures, soft tissue injuries, polyarthritis, uveitis, panostitis, cystitis, otitis. If it ends in -itis then in the author’s opinion it would benefit from an anti-itis.

Osteoarthritis is the number one cause of chronic pain in dogs, and 20% of dogs in the US have osteoarthritis. This can lead to a decrease in the quality of life of the patient and client. It can also lead to increased euthanasia due to patients that become non-responsive to treatment.

The cartilage damage (from any cause) initiates osteoarthritis. Initial trauma = fibrillation of cartilage surface, damage to chondrocytes, release of: 1) Cartilage degradation products (CDPS), 2) Matrix metalloproteinases (MMPs), 3) Nitric oxide (NO) and inflammatory cytokines.

MMPs/CDPS are engulfed by synoviocytes, which release: Inflammatory mediators: PGE2, TNF, IL1β and MMPs. All contribute to cycle of inflammation, degradation and pain of osteoarthritis.

This triggers pain by: 1) thickening of joint capsule, 2) remodeling of subchondral bone during sclerosis 3) osteophyte formation: a) synoviocytes release bone morphogenetic proteins (BMPs), b) periosteum mesenchymal stem cell to differentiate into chondrocytes; initiate osteophyte formation joint mouse or impinge on periosteum. PGE2 is also released in dorsal horn and contributes to the pain signal.

Bottom line: OA becomes a vicious cycle and the joint is less able to bear stress and forces which lead to further joint damage and clinical signs (pain, weakness, decreased activity, etc.).

The cyclooxygenase (COX) pathway is the focus for NSAIDS. Cyclooxygenase – prostaglandin synthase, its job is to place oxygen into arachidonic acid (AA), which metabolizes AA, and produces mediators of function AND pathology.

COX-1 pathway has the following properties: Constitutive to 1) Platelets 2) Kidneys 3) Stomach 4) Repro tract. It maintains homeostasis and therefore renal blood flow and gastric mucus secretions. COX-2 is thought to be the “bad” pathway, since it is inducible for actions of the 1) Fibroblasts 2) Endothelial cells 3) Chondrocytes and 4) Macrophages. However, it is constitutive CNS and kidneys. COX-2 is the main mediator of inflammation and pain.

There are some thoughts on a COX-3 variant. “COX-1 variant or isoform expressed in high amounts in brain and heart.” Acetaminophen poorly inhibits COX-1 and COX-2 and greatly inhibits COX-3. This has also been called partial COX-1 or POCOX-1, and these proteins are abundant in canine brain.

There are different classes of NSAIDS. Examples are provided for each class.
1. Nonselective - Inhibit both COX-1 and COX-2 (aspirin, flunixin)
2. Preferential - Inhibit COX-1 and COX-2, but less COX-1 (carprofen, meloxicam)
3. Dual acting - Inhibit both Cox-1 and COX-2 and lipooxygenase (Tepoxalin/Zubrin)
4. Selective - Inhibit COX-2 only (deracoxib)

However, we cannot think of COX-2 as purely bad. COX-2 can be good in some tissues: 1) Damaged gastric epithelium 2) Hypovolemic and developing renal tissues 3) Brain and 4) Endometrium. Although COX-2 is mainly “bad” it may also modulate (help decrease

<table>
<thead>
<tr>
<th>Drug</th>
<th>Drug Name</th>
<th>COX-2 to COX-1 Ratio</th>
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<tbody>
<tr>
<td>Aspirin</td>
<td>Acetylsalicylic acid</td>
<td>0.28</td>
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<tr>
<td>Bimadyl</td>
<td>Carprofen</td>
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<td>Deramax</td>
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<td>Metacam</td>
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<tr>
<td>Previcox</td>
<td>Firocoxib</td>
<td>384 – 427</td>
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Source: Lester Mandelker, DVM, FAWVF, (Pharmacology/Toxicology), VIN contributor
inflammation long term. Determining a ratio of COX-2 to COX-1 can help to further understand an individual NSAID’s impact on tissue. The higher the ratio, the less SPARING to COX-1.

The problem with COX ratios are: 1) Assays were done in vitro, not in vivo 2) Recombinant enzymes or whole cell cultures? 3) Selectivity for isoform may be lost at greater concentrations (clinical concentration) of drug 4) Individual isoform variability 5) Length of study and 6) Species differences. Why is this not as simple as we would like? 1) Genetic expression of isoform differs from individual to individual 2) Enzymes which metabolize NSAIDs differ from one individual to another and 3) At clinically effective doses, some NSAIDs lose their preference for one COX isoform.

Do the risks outweigh the benefits? Benefits and risk of any medication is assessed on a case by case basis. Understanding the different types of adverse events: 1) Allows veterinarians to minimize the risk 2) Better communicate the actual risks to pet owner and 3) Appropriate patient selection is key to maximize the benefits of NSAID use.

Adverse drug reactions fall into 1 of 2 categories: inducible or idiosyncratic. Inducible reactions are dose-related, attributable to the mechanism of action of the drug and are predictable. Idiosyncratic reactions are: not dose related, not attributable to the mechanism of action, unpredictable, rare and may be serious.

The most common seen with NSAIDs are inducible, and include GI irritation, and typically occur early in treatment. Idiosyncratic reactions occur infrequently (<1 in 10,000) and idiosyncratic reactions are most likely to occur in the first 90 days of treatment. Although individual response may vary, benefits of NSAID use outweigh the risks or the majority of dogs.

The FDA The Label Language: Lowest effective dose for the shortest duration consistent with individual response. Duration of treatment should be determined by the patient’s response. Start at lowest FDA Label Dose; some NSAIDs have dose range. If considering a reduction in dose monitor to ensure that the lower dose is effective. For idiosyncratic reactions, reducing the dose does not decrease the risk. Decision for dose and duration of treatment is based on patient and Benefit/Risk analysis. Frequency is low for most adverse events and resolve with discontinuation and/or supportive treatment. With appropriate patient selection and monitoring can maximize the benefits and minimize the risks.

The adverse effects of NSAIDs can be broken down by body system and include: GI, renal, platelet, respiratory, CNS, repro and cardiac.

GI side effects are related to inhibition of COX-1. COX-1 regulates numerous functions: Motility, Mucosal blood flow and Prostaglandin cytoprotection. When a GI ulcer forms, COX-2 needed for repair and even COX-2 inhibitors will delay healing. Patients with overt GI upset should not be given NSAIDs and patients with pre-existing GI ulcers, NSAIDs, (especially COX-2 selective) should not be used. For NSAIDs and pancreatitis: Bang UC, et al. World J Gastroenterol. 2008 May 21;14(19):2968-76. Pharmacological approach to acute pancreatitis. “The NSAID indomethacin and diclofenac have in randomized studies showed potential as prophylaxis again pancreatitis”. And Otsuka, T. et al. J Gastroenterol. 2012 Aug;47(8):912-7. Low dose rectal diclofenac for prevention of post-endoscopic retrograde cholangiopancreatography pancreatitis: a randomized controlled trial. “Pain was significantly more frequent in the control group than in the diclofenac group (37.7 vs. 7.8%). There were no adverse effects related to diclofenac. Low dose rectal diclofenac can prevent PEP”.


Tramadol may not be a good option to add to a NSAID. Hill T, Blilslager A. Co-Administration of NSAIDs and Tramadol Decreases Gastric Mucosal Barrier Function. ACVIM 2011. Recovery of gastric barrier function after acid injury was inhibited by co-administration of tramadol and indomethacin but not by tramadol or indomethacin alone. And Torring ML, et al. Perforated peptic ulcer and short-term mortality among tramadol users. Br J Clin Pharmacol. 2008 Apr;65(4):565-72. “Among patients hospitalized for perforated peptic ulcer, tramadol appears to increase mortality at a level comparable to NSAIDs.”

Aggregation of platelets is dependent on thromboxane A2. Which is produced by COX-1. Problems related to NSAIDs inhibiting COX-1 → no platelet aggregation. Aspirin irreversibly inhibits COX-1. COX-2 inhibitors (selective and preferential) preserve platelet function. Mullins KB et al. Effects of carprofen, meloxicam and deracoxib on platelet function in dogs. Vet Anes/Anal 2012 39(2) 206-17. “Individual assessment of platelet FUNCTION is advised when administering these drugs prior to surgery, particularly in the presence of other risk factors for bleeding.” This can be accomplished via Pt/Ptt, BMBT.

Other NSAID side effects include: COX inhibition can cause bronchospasm. COX-2 inhibition can result in increased neuron activity in seizure patients. Human studies: risk of thrombosis in patients with pre-existing disease increases with COX-2 inhibition. Chronic blockade of COX-1 and COX-2 may aggravate hypertension in humans. COX-2 in reproduction: Prolonged labor, decreased myometrial contraction and decreased embryo implantation can all be caused by NSAIDS.

Cats and NSAIDS: Most NSAIDs are metabolized via glucuronidation. Cats are deficient in glucuronyl transferase enzymes. Toxic metabolites increase rapidly with repeat dosing of certain NSAIDs. Subclinical renal impairment likely in this species. Operative monitoring of perfusion is very tough, hence most operative problems common, let alone those with NSAIDs. Acetaminophen toxicity causes impaired gluconate conjugation and toxic metabolites overwhelm glutathione reductase.

So how do we be safe about our NSAIDS? Patient selection and careful monitoring. All dogs should undergo a thorough history and physical examination before initiating NSAID therapy.

Appropriate hematological and serum baseline data is recommended prior to and periodically during administration. Avoid in dogs with a history of renal disease. NSAIDs are not recommended for dogs with bleeding disorder. Dogs that have adverse reactions from other NSAIDs, may have adverse reactions with other NSAIDs. Dogs at greatest risk are dehydrated or on concomitant diuretic therapy, or dogs with renal failure, cardiovascular and or hepatic dysfunction

Concomitant use of NSAIDs with other anti-inflammatory drugs such as corticosteroids and other NSAIDs should be avoided. Pet owners may not disclose that they are treating dogs with aspirin, 7% veterinarians recommend aspirin to treat canine osteoarthritis, 28% of pet owner indicated that they use aspirin to treat their dog’s osteoarthritis.

Studies to determine the activity of NSAIDs when administered concomitantly with other protein-bound or similarly metabolized drugs have not been conducted. Drug compatibility should be monitored closely in patients requiring cardiac, anticonvulsant and behavioral medications.

Always provide a Client Information Sheet with prescription. Pet owners should be informed regarding potential adverse events, advised to discontinue NSAID therapy if side effects occur and contact their veterinarian. Store palatable formulations out of reach of dogs, in a secured location. Severe adverse reactions may occur if large quantities of tablets are ingested.

NSAIDs should not be recommended when hyperbilirubinemia, elevated ALT, AST and GGT are present. If any of these are elevated alone or in combination, with or without signs of hepatic disease Albumin decreased; recommend workup for renal, GI or hepatic dysfunction, clotting disorder, elevated ALP with clinical signs of liver or Cushing’s disease.

But if ALP is elevated and no signs of Cushing’s it could be benign nodular hyperplasia, which is fairly common in older dogs. ALP can be 2.5x to >10x normal. Ultrasound and Bile Acids (pre and post prandial) to rule out other disease. Additional diagnostics as needed. Consider NSAIDs if no other underlying disease detected. Monitor to ensure no further elevation or other abnormalities (within 10–30 days, then periodically). Any further increases in hepatic enzymes warrants further evaluations.

NSAIDs in general are safe and efficacious. Start treatment using preferred NSAID Monitor efficacy, safety and pet owner compliance.

With appropriate patient selection and monitoring, benefits will outweigh the risk for most dogs. Consider consequences of not treating. Change in NSAID may be required based on patient’s response. One drug may be more effective than another drug, one drug may be better tolerated, pharmacogenetics may play a role in the variability.

Selected references
Are noted in the text above.
Nutraceuticals for Canine Athletes
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Glen Falls, NY

The first step is to define what a nutritional supplement is. The United States Food and Drug Administration (FDA) defines it as “a product intended for ingestion that contains a dietary ingredient to add further nutritional value to the diet.” Said ingredient may be any one or a combo of the following: vitamin, mineral, herb/botanical, or amino acid.

Well that is pretty broad and nonspecific. How about we look at what a nutraceutical is. That is defined by the North American Veterinary Nutraceutical Council as “a non-drug substance that is produced in a purified or extracted form and administered orally to provide compounds required for normal body structure and function with the intent of improving health and well-being.” So we are looking for something that can help the body. That narrows it down (sarcasm font) since it is not a drug it is NOT regulated by the Food and Drug Administration. This is important and will come up later in our discussion.

Since we do not have specifics, we need to think critically. It is important to know the difference between evidence and claims.

Evidence – These are established facts. Examples are
  • Double blind placebo controlled study
  • Third Party Research
  • A bibliography and sources referenced to back their statements
  • A prospective study
  • It appears in a peer-reviewed publication
  • It utilizes the scientific method

Claims – This is a statement that something is the case, often without proof. Examples are
  • Catchy terms on the label
  • Copying one company’s research and using it as your own
  • Proprietary blend --- don’t keep secrets. Tell us what’s in it
  • Self-published
  • Used for marketing
  • “Organic”
  • “Natural”
  • “Guaranteed”

I put those last 3 in quotes because we see those used freely and there are no regulatory methods for them. While those words sounds straightforward, they can be very misleading. On the other hand, I don’t want you to be scared by big words. Dihydrogenmonoxide sounds scary right? But you need this every day. 65% of you is made of this. What is it? WATER!

So why should we use a supplement? I think of it in “big picture” terms. We can potentially reduce or eliminate the need for medications with supplements. We want to use it as part of a whole body approach. This can include acupuncture, rehabilitation, surgery, exercise, diet (a well-balanced, appropriate feed with the CORRECT amount of calories), and use it with medications as well. We do need to use it SAFELY though. Over 22,000 trips to human emergency rooms occur EACH YEAR due to issues with supplements. So remember, you can have too much of a “good thing”.

Supplements for joint health
Glucosamine
This is an amino sugar found naturally in the body. It is a building block for the cartilage matrix. It stimulates cartilage cells positively. It is readily available and relatively cheap in the marketplace. Even though it sounds like glucose, it is NOT a factor in diabetic pets. There are studies (EVIDENCE) that show the sulfate form may be absorbed better than the hydrochloride. Perhaps because the hydrochloride is found in the joint. We do need a loading dose, which is 2x maintenance for 4-6 weeks to build up levels in the joints. What is maintenance? For a 75-pound (34kg) dog it’s 500mg/day.

Many dog foods on the market will supplement with glucosamine. This needs to be carefully looked at. If the average 34kg Labrador Retriever eats 1400kcal/day and the food is 4.0 kcal/gram, then they eat 350 grams of food per day. Well if that food has 475ppm (parts per million) of glucosamine that equals 475mg per kg of food. That same food may have 250ppm chondroitin sulfate (foreshadowing!) that equals 250 mg/kg of food.

Our Labrador Retriever is only eating 350 grams (1/3 kg). So, it’s only getting 160mg of glucosamine and 85 mg of chondroitin. It SHOULD be getting 1000mg of each for 4-6 weeks and then 500mg daily. So we still need to supplement. We also don’t know if it is absorbed the same when it is built into the food. So check your inclusion rates and do some math. Do not just trust the bag. It may contain glucosamine but not nearly enough to be a benefit.
**Chondroitin sulfate**

It is also naturally occurring in the body. It helps to form the matrix that cartilage forms to support joints. Chondroitin inhibits cartilage-destroying enzymes. It can be difficult to get a reliable source and extract into an edible form. Therefore it will cost more than glucosamine. Again, we need a loading dose. Both glucosamine and chondroitin are inherently safe. Some dogs that eat too much of it can get mild gastrointestinal (GI) reaction (vomiting, diarrhea). If this happens, you should stop the supplement and immediately consult your veterinarian.

There is good evidence that glucosamine and chondroitin work together. In fact, the two have a synergistic effect. That means that 1+1>2 in this case. They have been shown to lessen inflammation and lameness when given BEFORE a joint injury. Remember that these are chondroprotectants. That means they will PROTECT cartilage from damage. They can still help cartilage after an injury but work more effectively as a pre-emptive agent. They help to maintain healthy cartilage metabolism.

Now to talk about a claim with regards to these products. I was recently on a checkout line at a pet store (Yes, I go to pet stores—I have fish.) and saw a box of dog treats that said “contains glucosamine”. Having a minute as I waited, I flipped the box over and started to do the math. Calculating how much was in each treat and how many treats they would need to reach MAINTENANCE levels per day. Our 75 pound Labrador Retriever would have to eat TWO BOXES of treats PER DAY to get the 500mg glucosamine it would need for its joints. Not only is that not financially smart, it is BAD due to the amount of calories that dog would be eating. Obesity and joint health is a whole topic for another day. So be smart in the stores and READ.

**Avocado and soybean unsaponifiables – (AS)**

The ASUs are extracted out of the oils of the plants. These extracts have been shown to inhibit inflammation in the joint. They also complement the effects of glucosamine and chondroitin (1+1+1>3). Adding ASU decreases the amount of chondroitin needed which saves money in the long run for the consumer.

ASU supports cartilage matrix production and helps protect against cartilage damage. The safety has been widely demonstrated and there are even studies that show Dasuquin (made by Nutramax), which contains all 3 products, may be SIMILAR to the effect of non steroidal anti-inflammatoryy drugs (NSAID) in dogs for joint problems.

**Omega-3 fatty acids**

Eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). These are found in oil from wild caught COLD water fish (anchovies, sardines, salmon). The farm raised has low levels of omega-3 and high levels of omega-6. Omega-3’s have been shown to support heart health, joint health, skin and coats, the nervous system, the kidneys, and the immune system. GREAT! What’s the catch, you ask? They work in all those different systems at DIFFERENT DOSES.

Let’s look at some evidence for them. Studies in dogs with arthritis fed a diet with high levels of EPA and DHA had improved weight bearing on a force plate. They also had subjective reports from owners with regards to being able to walk and play more. Omega-3’s can be used in the diet or as a supplement. They are safe and well tolerated. However, like anything in life, we can have too much of a good thing. When we reach 500mg/kg/day we can run the risk platelet dysfunction. And before you ask: NO. Flax oil or coconut oil will not provide the same effects. In fact coconut oil has been shown to decrease the dog’s ability to SMELL. So for you working dogs out there lay off the coconuts.

It’s been a bit so let’s do some math again. (I know. You love it as much as I do.) Let’s use some round numbers to make it easier. A 100-pound (50kg) dog has arthritis. For arthritis we know that dogs need 80-120 mg/kg/day of Omega-3’s. Let’s go with the median dosage and use 100mg/kg/day which means our 50kg dog will need 5000 mg/day. (100x50). That equals 3 pumps of Welactin twice a day. 3 cups of Hill’s J/D twice a day or up to TWENTY-FIVE omega-3 capsules TWICE a day. So, be careful with those over the counter gel caps. Are you really saving money? And as we will learn later, not all supplements have in them what they say they do.

**Methylsulfonylmethane**

(MSM) This is a sulfur-containing compound found in small quantities in the body as well as fruits, veggies, and grains. It is a byproduct of the breakdown of DMSO (dimethylsulfoxide). DMSO for those of you who haven’t had the pleasure, smells like onions and garlic. It can be used in horses and dogs for neurological conditions and people for interstitial cystitis. (And before you ask, yes that means if your urinary bladder is inflamed we can make your pee smell like onions. Isn’t medicine great?)

Thankfully, MSM doesn’t have that special trait. There’s good evidence with two studies in humans with knee arthritis that MSMs helps them. There’s no published research in dogs yet. So this means we aren’t sure on the dosage needed. But the safety in dogs has been shown. And like other supplements, we can have a bit of GI issues in some cases. But otherwise, no reported problems.

**Eggshell membrane**

Moving on, we have eggshell membrane. (Anybody else getting hungry with all these supplements coming from food?) This is a new supplement. It’s been shown to be of benefit to people. I’m contributing to research on it for dogs. A water soluble, (hydrolized) powder of the eggshell has been shown to contain elastin, collage, desmosine, and isodesmosine. And yes, we are talking about the thin tissue paper like layer on the inside of the hard eggshell. It also contains glucosamine and chondroitin. So, if we can prove it beneficial in dogs, this could become another available supplement.
**Hyaluronic Acid**

Hyaluronic acid is our next joint supplement. It is naturally found in the body as a component of joint fluid. It can be given in the joint or in the vein in horses and dogs. It is given in the joint directly in people. There are some thoughts that it can be orally administered but the one case report in German Shepherds shows that it had benefit, but those dogs were also on other joint supplements. Therefore we are not sure about it for oral routes at this time. However, it is one of my preferred therapies for injecting directly into problem joints.

**Green-lipped mussels**

OK, back to food. Green-lipped mussels. These are a New Zealand shellfish. They are also known as *Perna canaliculus*. There are studies in dogs that suggest benefits, but the results are not consistent. They may contain vitamins C and E, minerals, Omega-3’s, and even chondroitin. So, we are not sure how they help. More research is needed.

**Boswellia serrata extract**

*Boswellia serrata* extract. You may also know this as frankincense. The bark of a tree found in India. It is a mix of boswellia acids that have an anti-inflammatory effect. The benefits have been seen in human arthritis patients. A study in dogs showed improved clinical signs with problems such as lameness, pain, stiff gaits. Is it good enough as a stand-alone therapy? No. But it can help.

**Curcumin extract**

OK, back to food. (Can you tell what drives me?) Curcumin extract. This is the active part of turmeric. We know turmeric from good cooking. The extract has antioxidant and anti-inflammatory effects. One study in dogs showed improvement in clinical scores. The standard extracts are not well absorbed by dogs, but particular ones are. And before you go “wait, I can just flavor my dog food with turmeric” don’t bother, it will not work. Dogs can’t process pure turmeric.

**Pilose antler peptide**

Let’s talk about some antlers. The velvet parts of elk or deer antlers specifically. This is a Traditional Chinese Veterinary Medicine (TCVM) modality. It is from the antlers in a fast growing stage when the antler is CARTILAGE. We can use farmed deer or elk, but we have to consider the ethics of collecting this. What is the active ingredient you ask? Good question. It is pilose antler peptide which is a good source of chondroitin sulfate, minerals, amino acids, collagen, and Omega-3 and Omega-6’s.

Is there any evidence? Another good question. In dogs some gait parameters and owner assessments were good. The same dogs graded by veterinarians at the same time did not show any improvement. We have potential safety concerns with this too. Antlers can be chewed down and get stuck in the mouth, throat, or the GI tract. Use with caution and always under adult supervision.

**Cetylmyrestolate**

Cetylmyrestolate is a fatty acid found in Swiss albino mice. The mechanism of action is decreasing inflammation and joint lubrication. However there are NO STUDIES in dogs and we do not know how or why this MIGHT work.

**Hyperimmune milk factor**

We will end with (shocking) another food product. Hyperimmune milk factor is a natural component of milk. We can use it to manage inflammation. The cows that are immunized with intestinal bacterial agents grow the factor for us. The high molecular weight IgG and low molecular weight anti-inflammatory components aid in reducing inflammation by blocking the entry of neutrophil attachment to the endothelial wall of cells. The concentrated version of this factor is called MicroLactin and it is the active ingredient in Duralactin.

A study of 8000 people with arthritis was done and 80% reported considerable improvement in joint pain and 72% reported less stiffness in the morning. There are also studies in dogs that pet owners felt the dog moved better. These are subjective and more studies are needed but it remains a promising product.

**Nutritional supplements for muscle recovery**

Moving on to nutritional supplements for muscle recovery and strengthening. While these are very popular for people we need to be very critical of them in dogs. Many of the muscle supplements will contain creatine, whey protein isolate, or specific amino acids; they can also contain chromium, dimethylglycine, or lecithin. Let us look at these individually.

**Creatine**

Creatine is an amino acid derivative in the body. It is found in skeletal muscle, heart, brain, and other organs. It is made via the anaerobic formation of ATP (Adenosine Triphosphate). Human studies show it may help performance and strength in repeated bouts of MAXIMAL exercise of short duration (<30 seconds): weight lifting, sprinting, cycling, etc. There are no studies in dogs at all. The biggest side effect is weight gain. This would not be helpful for dogs with arthritis. It would be borderline helpful in dogs trying to become pulling and working dogs to build muscle. I think there are better ways to do this (like rehab and conditioning and a proper diet plan).

**Whey protein isolate**

Whey protein isolate (Yep, curds and whey, just like in the nursery rhyme). This is made from milk. In human studies that looked at resistance training they had mixed results for muscle recovery, muscle mass, and strength. Again, we have no doses in dogs and, as I’ve mentioned earlier, we can’t just offer them milk.
Chromium
Chromium is an essential trace mineral found in many foods: meat, cheese, whole grain foods, fresh fruits, fresh veggies, and brewer’s yeast. It is needed for fat and carbohydrate metabolism. It can be helpful for people with diabetes but is not of benefit to diabetic dogs. The supplement is promoted for building muscle. There is absolutely no research that proves this claim in dogs or people.

Dimethylglycine
Dimethylglycine is an antioxidant that naturally occurs in the body, in cereal grains, and beans. It is marketed to support performance by decreasing lactic acid build up. Once again, we have absolutely NO research to back this up. There is conflicting research that it MAY support immune function.

Lecithin
Lecithin is a fat in the body that is also found in egg yolks and soybeans. It’s an emulsifying agent, which is a substance that helps an emulsion become more stable. An emulsion is usually a mixture of two products such as oil and water that do not mix together or that are also referred to as immiscible. An emulsifying agent is added to an emulsion to prevent the coalescence of the globules of the dispersed phase. Lecithin may support the gut barrier and it is marketed to support athletic performance, but yet again, there’s absolutely no data to support this.

Conclusion
Well that is a lot of options right? How do we implement a plan? We need to look at the breed, job, goals, and medical record of each individual canine athlete. From there, we can identify certain risks and where certain supplements may be of benefit. Remember, many of these take time to have an effect and are not a quick fix. In fact, they work better as preventatives and therefore they will need to be on them consistently (read: DAILY) for weeks to months to have a benefit for your pet.

Another important fact is that the manufacturers are NOT held to the same standard as pharmaceuticals. We therefore have to question the accuracy of information on the label, and the purity and source of the material used. Are they free from contaminants? Do we have any PROOF or do we have CLAIMS?

Selected references
Pain is an unpleasant sensory and emotional experience associated with an actual or potential tissue damage or described in such terms as damage. We as a veterinary community have an obligation to advocate on behalf of beings who cannot advocate for themselves. Every single patient treated by veterinary healthcare teams will experience pain at some point in its life. It truly is our obligation to be as aware and active in our pursuit of pain as possible.

Before we begin, a quick review of some important terminology:

- **Analgesia** - Insensibility to perceive pain WITHOUT lack of consciousness. Pain reliever.
- **Anesthesia** - Loss of feeling with the use of drugs
- **Sedation** - Relaxed or sleepy condition from the use of drugs
- **Multimodal analgesia** - Simultaneous use of more than one drug with different actions. Potential for dose reduction
- **Pre-emptive analgesia** - Done to stop an unwanted act of pain.
- **Hyperesthesia** - Pathologically oversensitive to a painful stimulus
- **Alloodynia** - Pain response from a non-painful stimulus

What are some signs of pain?

Hiding, biting, scratching, chewing, whimpering, limping, grunting, grinding teeth, restless, reluctant to move, sleeps more, growling, ears pinned back, hunched, howling, mydriasis, vomiting, diarrhea. Hissing, trembling, accidents in house, panting, decreased activity (less jumping) decreased appetite, withdrawn. Tachycardia, tachypnea, elevated BP. LOTS of signs. Some are obvious. Some are subtle. Muscle atrophy, decreased elbow flexion, decreased hip extension, check the ears (otitis), open the jaw. A good complete physical exam will reveal the potential disease processes and painful conditions that a pet can be hiding.

What is the cause of pain?

DJD, neoplasia, pancreatitis, parvo, GDV, pneumonia, HBC, ileus, thrombus, IVDD, scalpel blade, fractures, blocked (cats and dogs), whelping/queening, otitis, dental disease, glaucoma, amputation (overuse of other limbs), spondylitis, LS, diabetes (neuropathy), blood draws, catheters. We didn’t plan, for the potential for pain is everywhere.

Pain is the fourth vital sign at the author’s practice. In vet med its after TPR. In humans BP gets #4, pain #5. AAHA requires pain scoring as part of its accreditation standards, and is part of their Pain Management Guidelines.

Many different pain scoring scales are available. Some are: Visual Analog Scale (VAS), Numerical rating scale (NRS), Simple Descriptive Scale (SDS), Glasgow Pain System, CSU Canine and Feline Acute and Chronic Scores. Pick one for your practice and implement it by putting a copy in every medical record. Also have laminated copies in the ICU, OR, etc.

Some pain questions

Who grades pain? Everybody - Vets, LVT’s, assistants, OWNERS. What gets assessed? The patient.


How do we treat pain?

- Drugs - Opioids, NSAIDs, other
- Different routes of administration - IV, IM, epidural, oral, topical (EMLA cream).
- Environmental (hospital) - cage size, blankets, lighting, sound
- Environmental (home) – bedding, stairs, flooring, harnesses

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Review of the pain pathway from transduction, transmission, modulation and perception.

A brief moment here for discussing windup. Sustained stimulation of receptors causes dramatic changes in the second order neurons – Central sensitization (or wind-up). N-methyl-D-aspartate receptors in spinal cord are activated by glutamate. Increased activity in the dorsal horn leads to exaggerated responses to normal stimuli (hyperesthesia) and an increase in the size of the receptive field (recruitment).

Opioids
- The backbone of most analgesic plans (acute pain, surgery)
- Premed – lowers the amount of other drugs needed.
- Great for pre-emptive analgesia
- Butorphanol, Buprenorphine, Morphine, Hydro, Fentanyl
- Rapid onset, reversible (Narcan), IV, IM, epidural, PO, SL, CRI, transdermal
- Simbadol – (buprenorphine, inj, Abbott, SID) FDA approved for cats for pain control

Local anesthetics
- Lidocaine 2-minute onset, 90-minute duration
- Bupivacaine 5-7-minute onset, 4 hours duration
- Combo the two? Slightly longer onset than L alone, won’t last as long as B.
- 1cc/10lbs for dogs, ½ that for cats.
- Infiltration (line blocks, splash blocks, dental, testicular, pedicle), soaker catheters
- IV (lidocaine only, with opioid, ketamine)
- Ring blocks (declaws), regional infiltration
- Intra-articular? – Chondrocyte damage – so not recommended
- How? Blockade of the message to the dorsal horn.

NOCITA is a long-acting local anesthetic that gives you the control of extended-release bupivacaine, providing up to 72 hours of post-operative pain relief with one dose. Extended duration of action assists in preventing analgesia gaps in the first 72 hours post-surgery. Single treatment administered during cranial cruciate ligament surgery closure into the tissues for post-operative pain control. The extended-release bupivacaine technology used in NOCITA consists of multivesicular liposomes composed of hundreds to thousands of chambers per particle, encapsulating aqueous bupivacaine.

NMDA – Unwinding the windup. Options here are: Amantadine: Oral, SID (1-4mg/kg), Dogs and cats. Ketamine - Microdose, (sub anesthetic) -0.6-1.2ml in a one-liter bag, Balanced anesthesia/post-op analgesia, not a standalone (multimodal)

Alpha 2 Agonists - Anesthesia and or analgesia. Dexdomitor, Xylazine. Heart rate will be low. What’s the blood pressure? Quality over quantity with regards to HR. Decrease dose when potentiate with opioid (HR won’t be as low). Reversible – Yohimbine or Atipamazole.


Butorphanol – Agonist/antagonist – short acting. Good for post-op. Use an Alpha-2! – Get both effects. (control the pain and the dysphoria).

**How I dose Alpha-2**
- Observe them first. Scared? Stressed? Aggressive? I always aim for IV dosing
- Big dogs – 3-5 micrograms/kg
- Little dogs – 5-10 micrograms/kg
- Cats – 10-20 micrograms/kg
- Standard – 0.5mg/ml = 500 micrograms/ml
- New 0.1mg/ml = 100 micrograms/ml
- My “rescue” – 0.3ml of standard in a sterile glass tube with 2.7ml saline. Now 50 micrograms/ml. 0.1ml/10lb intra-op or post-op

**NSAIDS** - Non-Steroidal Anti Inflammatory Drugs. Treats the pain AND its SOURCE. K9- Rimadyl, Deramaxx, Metacam, Previcox, others. Feline – ONSior, Metacam (inj only). My first choice in arthritis (anti-“itis”). Watch renal and hepatic function (Prior, 2-4 weeks, then q 6-12 months). GI side effect potential, bleeding potential. Long term therapy may be needed.

Always provide a Client Information Sheet with prescription. Pet owners should be informed regarding potential adverse events, advised to discontinue NSAID therapy if side effects occur and contact their veterinarian. Store palatable formulations out of reach of dogs, in a secured location. Severe adverse reactions may occur if large quantities of tablets are ingested.

NSAIDS should not be recommended when hyperbilirubinemia, elevated ALT, AST and GGT are present. If any of these are elevated alone or in combination, with or without signs of hepatic disease Albumin decreased, recommend workup for renal, GI or hepatic dysfunction, clotting disorder, elevated ALP with clinical signs of liver or Cushing’s disease.

If ALP is elevated and no signs of Cushing’s it could be benign nodular hyperplasia, which is fairly common in older dogs. ALP can be 2.5x to >10x normal. Ultrasound and Bile Acids (pre and post prandial) to rule out other disease. Additional diagnostics as needed. Consider NSAIDs if no other underlying disease detected. Monitor to ensure no further elevation or other abnormalities (within 10-30 days, then periodically). Any further increases in hepatic enzymes warrants further evaluations.

What is galliprant? GALLIPRANT is a first-in-class non-cyclooxygenase (COX) inhibiting, non-steroidal anti-inflammatory drug (NSAID) in the piprant class. Pipfrants are a newly recognized drug class, established and defined by the World Health Organization in 2013 as prostaglandin receptor antagonists (PRA). Antagonizing the prostaglandin E2 (PGE2) EP4 receptor. PGE2 its physiologic effects through binding of four different receptors, EP1, EP2, EP3 and EP4. 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.

Tramadol - Opioid in people (M1 metabolite), also an opioid in cats (good luck giving it). Can be either a M1 or SSRI in dogs! Controlled in NY and other states. Cats 1-2mg/kg BID. **Dogs 3-7mg/kg QID and can take 2-4 weeks to take effect if SSRI method.**

Gabapentin - Neurontin – anticonvulsant. Pre-operative analgesia? Maybe. Postoperative analgesia and chronic pain (DJD? Neuropathy, IVDD/FCE). Dosing: 5-10-20mg/kg BID to TID. Start low, increase every 7-10 days (night time dose first – worst side effect is sleepiness) Titrate coming OFF of it as well – seizures potentially? Pediatric suspension can have xylitol. (Still ok for felines). There is a human suspension without xylitol.

**Other options for pain**
Lyrica – Pregabalin. Compound? $$$, Amitriptyline 1-2mg/kg BID, Acetaminophen 10-15mg/kg TID (dogs only, rescue), Fluoxetine 0.5-2mg/kg SID-BID (Based off Cymbalta (duloxetine) FDA approval for OA pain). Aspirin/dog aspirin? No proven benefits. Proven ulcers. Prednisone/Dex – anti-inflammatory. Side effects, cartilage damage, muscle loss.

**What if this isn’t working?**
- Look again – repeat PE, measurements, talk to owner (what changed at home), radiographs, MSK ultrasound, CT.
- Second opinion – co-worker, VIN, IVAPM forum, call me. No seriously, call me (email if not urgent). More drugs? Oxycodeone, Vicodin can also be used in some patients.
- Need a break? Admit it. Then admit the pet. (your clinic or mine). Other modalities that you may not have at your clinic. Admitting a patient – gives owners a break (paralyzed dog, old dog with incontinence, peace of mind). Also allows for a CRI, IV therapy, rehab technician care.
Other options include but are not limited to: food change (JM, J/D) Supplement change (DASUQUIN, Omega-3.) Traditional Chinese Veterinary Medicine (TCVM): acupuncture, herbal, food therapy. Chiropractic care, massage therapy (you and your team may need one also), LASER therapy, ESWT (Extracorporeal ShockWave Therapy). Stem Cell Therapy, PRP (platelet rich plasma) – DJD, sporting injuries. Adequan Canine (PSGAG’s) – I use this in cats too, SQ, same dose as dogs. Intra-articular injections (Hyaluronic Acid, LEGEND), Diet (huge for obesity) Hill’s, Purina programs for DVM, LVT, Nutritionist Consult, Cerenia – MAC sparing, presumed analgesic effects (GI, bladder in FLUTD cases). Methocarbamol (Robaxin) – muscle relaxant, not true pain reliever but can help (K9 10-15mg/kg TID). Myofascial Trigger Point Therapy, Rehabilitation (Physiotherapy)

Selected references
International Veterinary Academy of Pain Management (www.IVAPM.org)
Canine Rehabilitation & Physical Therapy - Millis, Levine
BSAVA Manual of Canine and Feline Rehabilitation, Supportive and Palliative Care - Lindley and Watson
Handbook of Veterinary Pain Management – James Gaynor
http://www.vasg.org - Veterinary Anesthesia and Analgesia Support Group
Abstract: Rehabilitation is becoming a standard of care in veterinary medicine. As this field grows and owners’ expectations for compassionate care and return to function and activity expand, there is a necessity to offer better and faster options. Laser therapy is a vital and practical cog in the wheel of rehabilitative options. Laser therapy can be utilized for a wide variety of cases, making it a feasible and profitable part of practice. Injuries of many types will benefit from laser treatments. With very few exceptions, cases with injuries can be treated with photobiomodulation therapy as opposed to other modalities that may be limited by a patient’s other medical conditions. This nonpharmaceutical options for treatment can be an asset in veterinary medicine.

**LASER: Light amplification by stimulated emission of radiation**

The lasers used in rehabilitation help to modulate cellular functions. This process is known as photobiomodulation and is defined as non-thermal interaction of monochromatic radiation with a target site. Although the physiologic interaction of this type of energy application on tissue is still not completely understood, low-energy lasers have been reported to modulate various biologic processes, such as mitochondrial respiration and adenosine triphosphate (ATP) synthesis, to accelerate wound and joint healing, and to promote muscle regeneration. In addition, acute and chronic pain control has been reported using this type of low-energy photon therapy.

Treatment of chronic and acute edema, neurologic conditions, and postoperative care are some other popular conditions treated with laser therapy.

Before applying laser therapy to a patient, there are two fundamental attributes that must be established. First, the type of laser must be known, as well as the wavelength. The wavelengths of most lasers used for laser treatment are typically in the infrared or near-infrared range of 600-1000 nm. Wavelengths of low-power lasers commonly used are 632.8 nm (HeNe, gas) in the visible light range, 810 nm (GaAlAs, diode), and 904 nm (GaAs, diode) in the infrared region of the light spectrum. The wavelength is the prime determinant of tissue penetration. Lasers that do not penetrate as deeply (630 to 740 nm) are suitable for acupuncture point stimulation and wound healing but have not proved their clinical effectiveness with deep-seated musculoskeletal conditions. Infrared lasers (750 to 1500 nm) penetrate more deeply and are used to treat trigger points, ligaments, joint capsules, and intraarticular structures. The output power (watts or milliwatts) must also be known. Based on this, and the condition to be treated, the dose of laser light \( \text{J/cm}^2 \) is determined. For most conditions, a dose of 1 to 8 \( \text{J/cm}^2 \) is typically applied. The time the laser must be applied to an area to deliver the dose must be calculated.

For example, if a 904-nm laser with a maximum output power of 250 mW is used, it will take 4 seconds to deliver 1 J.

\[
0.250 \text{ W} = 1 \text{ J/}X \text{ seconds (0.250 W) (X seconds) = 1 J} // X \text{ seconds} = 1 \text{ J}/0.250 \text{ W X} = 4 \text{ seconds}
\]

With this particular laser, it will be necessary to hold the laser on one point for 4 seconds to deliver 1 J of energy. With a 1-W laser, this takes 1 second. It is crucial to always understand what the particular laser unit possesses with regard to power and wavelengths, and the dose, in \( \text{J/cm}^2 \), a condition requires. From there, it should be easy to determine how much time is needed for laser application. Some laser units compute this with preprogrammed software, whereas others require the therapist to calculate how many joules are needed and how many joules are emitted per second. Some units may take 1 second or less to deliver 1 J, whereas other units may take up to 20 seconds to deliver 1 J.

The greater the power density and longer the wavelength, the deeper the penetration is through tissues. Unfortunately, the optimal wavelengths, intensities, and dosages have not been adequately studied in dogs, and information in people is difficult to interpret because of different conditions and treatment regimens. More laser dosage is not necessarily better, because overdosing may retard the desired effect. A common mistake in using laser therapy, especially in wound healing, is to experience a positive result and automatically assume more will be better. The healing process can only progress at a certain rate.

Laser therapy is generally administered with a hand-held probe, with a small beam area that is useful to treat small surfaces; other lasers have several beam areas in the same unit to treat larger areas. Laser energy may be applied with the laser probe in contact with the skin, which eliminates reflection from the skin and minimizes beam divergence, or with the probe not held in contact. With the noncontact method, it is necessary to hold the probe perpendicular to the treatment area to minimize wave reflection and beam divergence. Noncontact application is recommended for wound treatment. The appropriate dosage may be applied to larger areas by administering the calculated dose to each individual site in a grid fashion, or by slowly moving the probe over the entire surface, being certain to evenly distribute the energy to each site. In any case, the probe should be held perpendicular to the skin. A coupling medium is not necessary, as in ultrasound, because the laser beam is not attenuated by air. Lasers are safe but proper integration into the practice is essential. Most companies will provide laser safety training. Goggles (for all those in the room, including the patient) should be worn when using laser therapy. The goggles must be specific to the wavelength of laser light being used.
Veterinary rehabilitation does not focus on one specific problem, but rather sees the patient as a whole. One of the main goals of rehabilitation is a return to normal or near normal function. The main effects of lasers in rehabilitation can be summed up as: reducing pain, reducing inflammation and promoting healing. The benefits of laser therapy for rehabilitation patients focus on two main areas: increasing healing times through faster, stronger tissue repair, and the analgesic effect of the laser. These combine to allow the patient as a whole to feel better and lead to the ability to exercise efficiently (when indicated), to gain strength, and to return to function with an improved quality of life.

**Conditions treated with laser**

Any area that is inflamed or painful can benefit from laser (aside from pregnancy or neoplasia).

**Dermal**

Open wounds, post-surgical wounds, otitis externa and lick granulomas are all conditions that could be treated with laser in a veterinary setting.

**Musculoskeletal**

Treatment with laser therapy may be implemented as part of a multimodal treatment program. The specific goals of laser therapy often include decreased pain, inflammation, and improved circulation. Arthritic joints should be thoroughly treated along the joint lines and surrounding area. Recommendations for arthritic joints have ranged from 4 J/cm² up to 30 J/cm², but more appropriate doses may be 8-10 J/cm². The apparatus may either be held directly over the area for the designated number of joules, or used in a sweeping fashion depending on the unit.

Biceps tenosynovitis, supraspinatus tendonitis, patellar tendonitis, and other in inflammatory conditions involving tendons may be treated with laser therapy. The length of the superficial aspect of the tendon should be treated along with the surrounding soft tissues. The biceps tendon communicates with the shoulder joint capsule; therefore, the shoulder joint capsule should be treated as well.

**Neurologic**

A variety of diseases can affect this system including congenital malformation, genetic diseases, metabolic conditions (diabetes mellitus, hypothyroidism), toxicities (botulism, tick paralysis), autoimmune and inflammatory diseases (acute polyradiculoneuritis, myasthenia gravis), neoplasia, trauma (brachial plexus avulsion), and vascular conditions (feline aortic thromboembolism).

Additionally, traumatic peripheral nerve injuries can also be treated.

Injuries to the spinal cord include those affecting the brain and herniated intervertebral discs. Common conditions affecting the spinal cord include trauma, intervertebral disk disease (IVDD), fibrocartilagineous emboli (FCE), vascular events, cervical spondylomyelopathy (wobbler’s disease), lumbosacral stenosis, discospondylitis, spondylisis deformans, syringomyelia, degenerative myelopathy, and trauma. Trauma, IVDD, and FCE can have acute onset where the spinal cord may have better healing potential than from the more chronic conditions (lumbosacral stenosis, spondylisis deformans, degenerative myelopathy, etc.) Proper diagnosis will aid in determining the prognosis and course of treatment.

**Musculoskeletal evidence**

Osteoarthritis studies with laser therapy in people; a meta-analysis review was conducted on the efficacy of laser therapy on OA in people. Seven trials were included, with 184 patients randomized to laser, and 161 patients to placebo groups, using a variety of lasers and treatment protocols. Treatment duration ranged from 4 to 12 weeks. Pain was assessed in four trials. The pooled estimate of three trials showed no effect on pain measured using a scale, and two demonstrated very beneficial effects with laser. In another trial, with no scale-based pain outcome, significantly more patients reported pain relief (yes/no) with laser. One study found knee ROM was significantly increased. Other outcomes of joint tenderness and strength were not significant. Lower dosages of laser were found to be as effective as higher dosages for reducing pain and improving knee ROM. The authors concluded that for OA, the results are conflicting in different studies and may depend on the method of application and other features of laser application, including wavelength, treatment duration, dosage, and site of application over nerves instead of joints.

Most of the research on the effects of laser therapy on tendon and ligament conditions is done either in experimental models in laboratory animals such as rats or in people. More evidence from studies in dogs, cats, and other animals is needed. Historically, laser therapy has been recommended for tendon and ligament conditions but the clinical efficacy remained controversial. Recent research proves that laser therapy is appropriate for these types of injuries.

One study looked at seven people with bilateral Achilles tendinitis to see if laser therapy has an anti-inflammatory effect. In placebo versus laser treatment the PGE2 levels were reduced for 75-105 minutes after laser therapy, and pain pressure threshold values increased after laser therapy. The authors concluded that laser therapy reduces pain and inflammation in people with acute Achilles tendinitis. A recent review of 25 articles (13 in vitro and 12 animal studies) was done to evaluate the influence of laser therapy on bone healing. All animal studies showed improved bone healing in sites irradiated with laser. It was concluded that laser could accelerate bone healing in extraction sites, bone fracture defects, and distraction osteogenesis.
Evidence for laser therapy in the neurological system

A study by Morries et al. (2015) looked at using laser therapy to treat traumatic brain injuries. In ten patients with chronic traumatic brain injury given ten treatments over the course of 2 months, using an 810 or a 980 nm laser, symptoms of headache, sleep disturbance, cognition, mood dysregulation, anxiety, and irritability improved. Depression scales and a novel patient diary system specifically designed for this study monitored symptoms. They concluded that laser immunomodulates the response to brain damage.

A research group has studied two spinal cord injury models to show 810 nm laser therapy was effective for transected or contused rat spinal cords (Wu et al. 2009). Laser was applied transcutaneously at the lesion site immediately after injury and daily for 14 consecutive days. The daily dosage at the surface of the skin overlying the lesion was 1589 J/cm² (150 mW, 0.3 cm² spot area, 2997 seconds). Mini-ruby was used to label corticospinal tract axons, which were counted and measured from the lesion site distally. Functional recovery was assessed by footprint test for the hemisection model and open field test for the contusion model. The average length of axonal re-growth in the rats in the treated group with hemisection and contusion injuries was significantly longer than the comparable untreated control groups. The total axon number in the treated groups was significantly higher compared to the untreated groups for both injury models. For contusion model rats there was a significant functional recovery in the laser treated groups compared to control.

A pilot study (Rochkind et al. 2007) investigated the effectiveness of 780 nm laser light in the treatment of patients suffering from incomplete peripheral nerve and brachial plexus injuries for 6 months up to several years. It was randomized, double blind, and placebo controlled using 18 patients. Twenty-one consecutive daily sessions of laser or placebo were applied transcutaneously for 3 hours to the injured peripheral nerve and for 2 hours to the corresponding segments of the spinal cord. Clinical and electrophysiological assessments were done at baseline, 21 days, and 3 and 6 months. The laser and placebo groups were in similar clinical conditions at the start of the study. The analysis of motor function during the 6 month follow up period compared to baseline showed significant improvement in the laser treated group compared to the placebo. No significant difference was found in sensory function. Electrophysiological analysis showed statistically significant improvement in recruitment of voluntary muscle activity in the laser treated group.

Role of laser in injury rehabilitation

Based on research, case reviews, and anecdotal reports, it can be concluded that it is indicated to use laser therapy in a clinical setting. A practical approach is also needed, accounting for many factors including patient need, patient cooperation, cost, efficacy, and frequency and duration of treatment. Some clients may allow their pet to be treated with the laser daily; some may only be able to so 2-3 times a week. It is critically important for the veterinarian to prescribe a practical and realistic treatment plan for each individual patient. Laser therapy represents only one element of comprehensive rehabilitation. While laser treatment can be safely and effectively combined with other modalities and integrated easily with other treatment approaches, it is important to prioritize the sequence of application with other modalities.

Laser therapy as a stand-alone therapy

Examples of diagnoses that could benefit from laser treatment as a sole treatment modality include surgical incisions, wounds, lick granulomas, osteoarthritis, and tail pull injuries. Application of the laser to every surgical incision at the end of the anesthetic period can reduce post-operative pain and swelling. This can be provided either bundled or as an option for all appropriate surgeries in a practice. Wounds can benefit from laser therapy in the late inflammatory or early proliferative phase, and laser therapy provides continued benefit in chronic or slow healing wounds as healing progresses. Lick granulomas can arise from many reasons and become cycles of healing and reoccurrence. Addressing pain relief, improved circulation, and antimicrobial pathways via laser therapy can provide improvement where other treatment modalities have either failed or only address a single potential cause of the granuloma.

Palliative management of chronic conditions can be achieved with laser. End stage otitis externa in cases that are not candidates for surgical resection will benefit from reducing bacterial load and reducing inflammation and pain. In some cases of osteoarthritis, the author has managed patients with stand-alone laser therapy. It is also a useful agent if having to discontinue other osteoarthritis management modalities. An example is the severely arthritic patient on an NSAID that develops renal, hepatic, or gastrointestinal disease, and NSAID therapy has to be rapidly discontinued.

The therapeutic laser can be used to stimulate acupuncture points along meridians for those clinicians who practice traditional Chinese veterinary medicine. The author has found this helpful for anxious patients that may not sit for 15-20 minutes or more with acupuncture needles in place.

Neurological patients often need a comprehensive rehabilitation program that includes laser therapy to achieve favorable results. But, tail pull injuries provide a straightforward injury where laser treatment can be the only modality that is available, practical, and easy to achieve. Additionally, patients suffering chronic pain from tail docking will also benefit from laser therapy.
Laser therapy in conjunction with a rehabilitation program

Proper rehabilitation starts with pain management. Regardless of the injury, no patient will be able to return to function through strength training, and maintain that outcome, if they are painful. Laser therapy is, in the author’s opinion, an extremely valuable modality to achieve the goals of the clinician, the client, and the patient. The release of endogenous opioids stimulated by laser therapy has applications throughout both acute and chronic conditions in injury rehabilitation (Hagiwara et al. 2008). Applying laser therapy to painful muscles, tendons, ligaments, or joints before (and sometimes after) having the patient do therapeutic exercises, such as underwater treadmill workouts, cavaletti rails, or other core exercises, makes sense.

It is important for the clinician to incorporate laser therapy as one part of a complete rehabilitation program. An example is incorporating laser therapy during the first few weeks of rehabilitation for a dog with biceps tendonitis. This aids in reducing pain and stimulating cytokines and growth factors to achieve better tendon tissue healing. Laser therapy may be phased out of the rehabilitation program once initial goals have been achieved, and the patient progresses to strength training and maintenance.

Osteoarthritis may initially require a high frequency of treatment (multiple times per week) through an induction phase. Then, as pain and inflammation are reduced, the frequency of treatments can be reduced through a transition phase, until a maintenance phase protocol is achieved. If an acute on chronic flare up occurs, the induction phase can be easily repeated keeping the patient functional.

Neurological patients, such as those with brachial plexus avulsion or intervertebral disc disease, may benefit from laser therapy throughout their entire rehabilitation process. Early it may be used for management of pain at the insult site or in inflamed muscles. It can be used to attempt to achieve return of neurological function throughout the recovery, and can be used either to prevent or manage neuropathic pain that is a potential outcome in these cases.

Reassessing response to laser therapy

Based on the patient, diagnosis, prognosis, and plan of each case, reassessments should be scheduled and documented appropriately by the clinician. Veterinary professionals delivering the treatment should be looking for both subjective and objective indicators of how the patient is doing. Repeated physical examinations and photographs can be helpful in assessment of patient function. Additionally, goniometry, digital thermography, and stance analyzer measurements can be useful for providing objective data in a clinical setting.

Conclusion

Vetinary rehabilitation is an expanding field that requires both a practical approach and staying on the cutting edge of available treatment options. While injuries can occur in many body systems and from many different causes, laser therapy is a vital tool in the veterinary professional’s armament. Its variability, ease of administration, and proven benefits allow many types of patients to recover faster and more effectively. Further research is needed to find other types of injuries that may benefit from laser therapy, as well as further verifying treatment settings and timeframes.

Selected references


