

## **PHYSICAL THERAPY APPLICATIONS FOR PAIN MANAGEMENT IN SPORTS MEDICINE**

Laurie Edge-Hughes, BScPT, MAnimSt (Animal Physiotherapy), CAFCI, CCRT  
The Canine Fitness Centre Ltd  
Calgary, AB, Canada

Canine athletes can suffer from a wide variety of musculoskeletal injuries, which may impede their ability to compete in their sport until the injury is fully resolved. However, a good number of athletes may suffer from painful chronic musculoskeletal conditions that are not injuries per se, but rather conditions of overuse or aging. One such condition is osteoarthritis, which can be managed with physical therapy applications in order to improve the quality of life of the animal athlete and allow for the continuance of engaging in sporting activities.

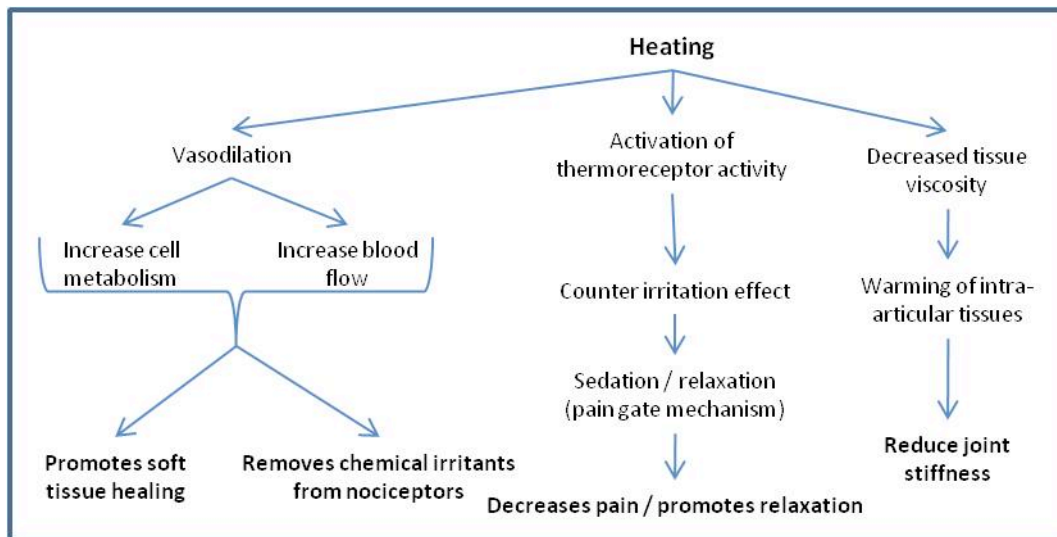
### **OSTEOARTHRITIS**

Osteoarthritis creates both mechanical pain, due to abnormal loading and stress on capsule, ligament and periarticular tissues, and some inflammatory pain, due to chemicals released. Following inflammation, it has been shown there is an increase in responses (within  $A\beta$ ,  $A\delta$ , and type C afferent fibres) to both noxious activity and innocuous joint movement. As well “silent nociceptors” (those that do not normally respond to mechanical stimuli) will respond to innocuous and noxious stimuli, joint movement, and pressure (hence a peripheral sensitization). The peripheral sensitization can subsequently lead to central sensitization of dorsal horn neurons. As well, it has been shown that there is also a loss of inhibitory pain control mechanisms in osteoarthritis. Together, the peripheral and central changes observed with osteoarthritis contribute to the pain and loss of function.

Osteoarthritis (OA) clinically manifests with aching, discomfort, and pain that increases with excessive activity, a reduction in the overall activity level or ability to perform activities, poorer proprioception, joint stiffness and enlargement, effusion, and loss of flexibility and strength.(Edge-Hughes & Nicholson 2007) While dogs with severe cases of OA should likely be retired from sport, there are a number of dogs in competition sports that have mild to moderate OA and still derive a seemingly tremendous amount of joy from these activities. It is this subset of athletes that can benefit from proactive physical therapy pain management strategies.

### **THERMOTHERAPY**

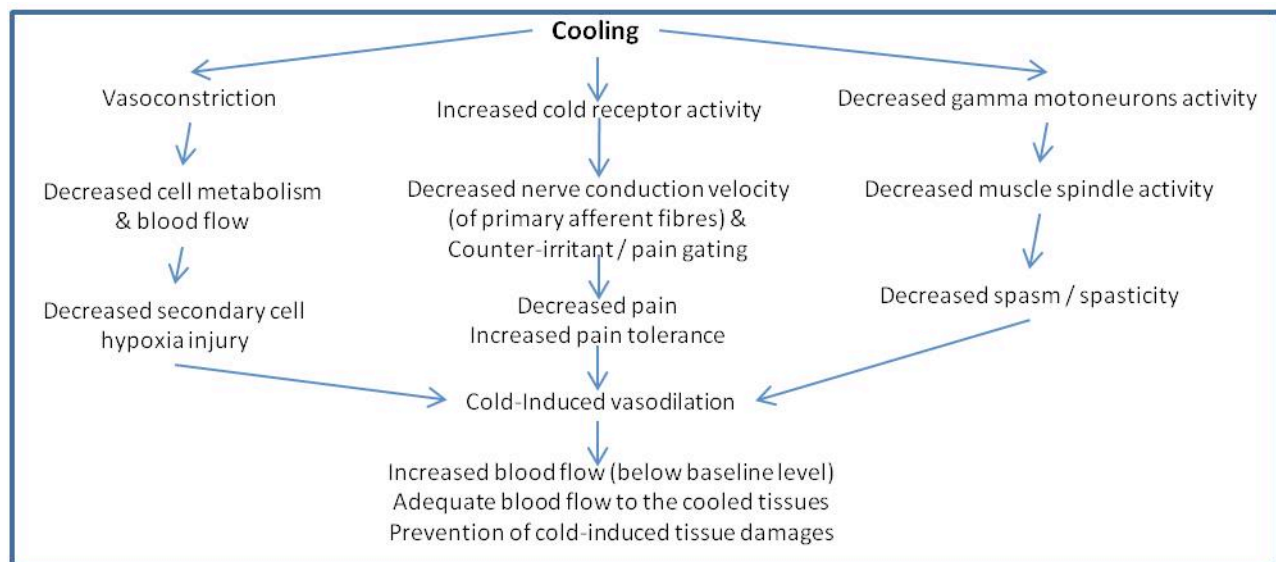
Heat has long been used for symptomatic treatment of arthritis pain and stiffness. Improvement in arthritic stiffness has been shown with thermal therapies. (Wright & Johns 1961; Robinson et al 2002) Additionally superficial heat has demonstrated short-term reductions in pain in cases of acute or subacute low back pain. (French et al 2006) Specific to osteoarthritis, the proposed sequences of physiological and cellular functions with heating are as follows. (Belanger 2004)



Thus heat can be utilized for the above benefits as part of the day to day management of arthritic joints and prior to engaging in physical activities.

### CRYOTHERAPY

Ice is often utilized and well supported by research in cases of acute pain and injury. (Belanger 2004) Reviews have shown that clinically, ice provides benefits in terms of swelling and range of motion in osteoarthritis. (Brosseau et al 2003) The physiologic effects of Cryotherapy as they may relate to osteoarthritis are found in the following chart. (Belanger 2004; Sluka 2009)



Thus icing may be effectively utilized post-sporting activities to aid in pain relief in addition to its use in acute inflammatory conditions or in cases of OA 'flare ups'.

## **THERAPEUTIC LASER**

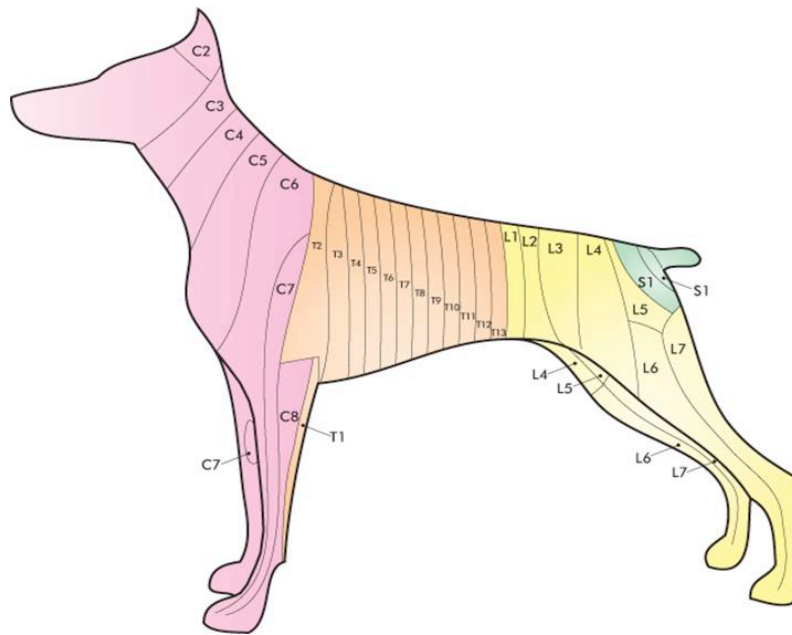
Low-level laser therapy (LLLT) uses laser light to aid tissue repair, relieve pain, and stimulate acupuncture points. (Woodruff et al 2004; Enwemeka et al 2004; Siendentopf et 2002) It's general effectiveness can be attributed to anti-inflammatory mechanisms (which can be similar to pharmacological agents such as celecoxib, meloxicam, diclofenac and dexamethasone), the ability to reduce oxidative stress, improved angiogenesis, augmentation of collagen synthesis, and skeletal fatigues, and inhibition of transmission at the neuromuscular junction. (Bjordal et al 2006; Chow et al 2009) Research into the use of LLLT for pain reduction and tissue repair spans more than 30 years, but only more recently have high quality reviews and meta-analyses been able to elucidate details on laser types and dosages that yield positive results. Chow et al 2009 revealed that for acute and chronic neck pain, the optimum dose per point for an 820-830nm laser was 5.9 Joules with an irradiation time of 39.8 seconds and using a 904nm laser, it was 2.2 Joules delivered with an irradiation time of 238 seconds. The number of repetitions and treatments per week were variable. Data from the reviewed trials suggested that positive effects were immediate and could be maintained for up to 3 months after treatment ended. Bjordal et al 2006 determined that LLLT at high doses (7.5 J/cm<sup>2</sup>) at the target tissue in the first 72 hours (to reduce inflammation) followed by the lower doses (2 J/cm<sup>2</sup>) at target tissues in subsequent days (to promote tissue repair) was most advisable. Each of these two authors noted that studies that reported negative results, also utilized inadequate doses, and poor laser exposure technique. As well, systematic reviews that did not employ procedural assessment of factors, such as dosage and exposure technique, should be disregarded.

## **TRANSCUTANEOUS ELECTRICAL NEUROMUSCULAR STIMULATION (TENS)**

Transcutaneous electrical neuromuscular stimulation (TENS) is a modality utilized for pain relief. The two main mechanisms by which electrostimulation produces pain relief are segmental inhibition through pain-gating mechanisms, and via descending inhibitory mechanisms. Animal models have produced studies that demonstrate that different frequencies of TENS produce analgesia through action on different neurotransmitters and receptors. (Sluka & Walsh 2009) Essentially high frequency / conventional TENS (>60Hz) relies on the selective stimulation of larger diameter fibres in peripheral nerves, which in turn helps to 'block' nociceptive activity in smaller afferents at segmental levels. (Baxter & McDonough 2007) Additionally, high-frequency TENS increases the concentration of B endorphins in the bloodstream and cerebrospinal fluid, and increases methionin-enkephalin in the cerebrospinal fluid, in human subjects. Both animal and human studies show that high-freq TENS also reduces release of the excitatory neurotransmitters glutamate and substance P in the spinal cord dorsal horn in animals with inflammation. (Sluka & Walsh 2009) Low frequency TENS (<10Hz) stimulates a release of endogenous opiates, and is often referred to as acupuncture-like TENS because its mechanism of pain relief is similar to acupuncture. Essentially low frequency TENS primarily affects the relevant spinal segmental level, where opioid, GABA, serotonin, and muscarinic receptors are activated by low-frequency TENS to reduce dorsal horn neuron activity, nociception and the consequent pain. (Baxter & McDonough 2007; Sluka & Walsh 2009) It has also been shown that peripheral opioid receptors are also responsible for low-frequency (but not high-frequency) TENS analgesia. It should also be noted that the proposed mechanisms for pain relief utilizing electroacupuncture at different frequencies is similar (if not the same) to those demonstrated following TENS stimulation at similar frequencies.

TENS application can not only be localized to the lesion but also utilize dermatome & myotome patterns in order to increase the effectiveness of this modality. (Note: you can use other modalities on dermatomal & myotomal nerve roots as well).

<b>Canine Nerves, Nerve Roots, and Muscle Innervation</b>		
<b>Nerves</b>	<b>Root</b>	<b>Muscles</b>
Radial Nerve	C7 – T2	All extensor muscles of the elbow & carpus & digits, supinator, brachioradialis, APL, EPL
Median Nerve	C8 – T2	Pron. Teres, Pron Quad., FCR, SDF, Radial head of DDF
Ulnar Nerve	C8 – T2	FCU, Ulnar & Humeral heads of DDF, Lumbricals, Interossei & elbow jt
Musculocutaneous Nerve	C7 (C8)	Coracobrachialis, biceps, brachialis
Axillary Nerve	C7, (C6 & C8)	Teres major, teres minor, deltoid (and subscapularis)
Subscapular Nerve	C6 – C7	Subscapularis
Suprascapular Nerve	C6, (C7)	Supraspinatus & infraspinatus
Pectoral Nerve	C7, C8	Superficial & deep pectorals
Thoracodorsal Nerve	C8, (C7, T1)	Latissimus dorsi
Femoral Nerve	L4 – L6, (L3)	All of the Quadriceps complex, iliopsoas, Sartorius
Sciatic Nerve Tibial Peroneal Supf & Deep	L6, L7, (S1, S2)	Hamstrings, quadratus femoris, gemelli, obturator internus, gastrocs, popliteus, tibialis posterior, tibialis anterior, digital flexotrs and extensors, Fibularis brevis, EHL, muscles of the foot
Obturator Nerve	L5, L6	Obturator externus, pectineus, adductor, gracilis
Anterior Gluteal Nerve	L7, S1	Glutei, TFL, capsularis
Posterior Gluteal Nerve	S1 – S3	Biceps femoris, Middle & Superficial glutes



### **PULSED ELECTROMAGNETIC FIELD THERAPY**

Pulsed Electromagnetic Field Therapy (PEMF) is a greatly debated therapeutic tool in physical therapy practice. Conflicting results exist for its use in the treatment of osteoarthritis pain. (Hulme et al 2002; McCarthy et al 2006) However Vavken et al (2009) reviewed relevant RCTs and concluded that PEMF improved clinical scores and function in patients with osteoarthritis of the knee.

### **THERAPEUTIC ULTRASOUND**

Ultrasound is a highly utilized modality in human physical therapy practice. Clinically, it has been shown to be of benefit for pain relief and improved function in osteoarthritis. (Srbely 2008; Rutjes et al 2010) The proposed mechanisms for this are either via thermal mechanisms (i.e. continuous ultrasound), whereby the deep heating may improve peripheral nerve function, or via increased blood flow, and/or the creation of a counter-irritant hence activating a spinal pain gating mechanism. (Sluka 2009) Alternately non-thermal/mechanical mechanisms have been proposed whereby acoustic streaming (the movement of fluids across cell membranes) or stable cavitation (the creation of transient gas bubbles in the sound field) causes cell membrane disruption, which may enhance cellular permeability and allow for better local utilization of anti-inflammatory medications. (Belanger 2004)

### **MOBILIZATIONS**

The proposed mechanisms through which mobilization and manipulation affect pain and motion restrictions are recently being broadened. Research has not been able to substantiate a biomechanical effect (e.g. correction of a positional fault). However, the neurological effects of mobilizations are reportedly a reduction in pain and inhibition of reflex muscle contractions. (Zusman 1986; Katavich 1998; Björnsdóttir & Kumar 1997; Zelle et al 2005) The achievement of neurophysiological effects requires repetitive (oscillatory) or sustained manual stimulation which results in a hysteresis effect. The hysteresis effect involves inhibition of low threshold mechanoreceptors (group I & II), inhibition of high threshold nociceptors (group III & IV), both

of which result in a reduction of intra-articular pressure and peripheral afferent discharge. (Zusman 1986; Katavich 1998; Conroy & Hayes 1998; Sterling et al 2001)

Two recent articles review the literature and proposed mechanisms for pain relief. (Schmid et al 2008 & Bialosky et al 2009) The following comprehensive model has been suggested to account for all the mechanisms of action of manual therapy.

1. Mechanical stimulus: Biomechanical effects are associated with manual therapy as motion has been quantified with joint-biased and nerve-biased manual therapy. However, there are inconsistencies associated with the purported biomechanical mechanism of action. Subsequently, it has been suggested that a mechanical force is necessary to initiate a chain of neurophysiological responses which produce the outcomes associated with manual therapy.
2. Neurophysiological mechanism: This proposed model accounts for the complex integration of both the peripheral and central nervous system based on associated neurophysiological responses which indirectly implicate specific mechanisms. Suggested mechanisms include actions mediated by the periaqueductal gray and mechanisms mediated by the dorsal horn of the spinal cord.
3. Peripheral mechanisms: Manual therapy may directly affect inflammatory mediators and peripheral nociceptors. It has been observed that following spinal manipulation, there is a significant reduction of blood and serum level cytokines. As well, changes of blood levels of  $\beta$ -endorphin, anandamide, N-palmitoyl-lethanolamide, serotonin and endogenous cannabinoids have been observed following manual therapy.
4. Spinal mechanisms: Manual therapy has been theorized to act as a counter irritant to modulate pain and/or to “bombard the CNS with sensory input from the muscle proprioceptors”. There is indirect evidence for a spinal cord mediated mechanism of action in that manual therapy is associated with hypoalgesia, afferent discharge, motoneuron pool activity, and changes in muscle activity.
5. Supraspinal mechanisms: Supraspinal structures include the anterior cingulate cortex, amygdala, periaqueductal gray, and rostral ventromedial medulla. There is direct support for supraspinal mechanisms for pain modulation with manual therapy (as shown on MRI). There is indirect support for autonomic responses, and opioid responses. Additionally, placebo, expectation, and psychosocial factors may be pertinent in the mechanisms of manual therapy.

## **MASSAGE**

Massage can have neurophysiological effects, neuropsychological effects, and mechanical effects. From a pain perspective, massage has been shown to increase plasma concentrations of  $\beta$ -endorphins, decrease systolic blood pressure, and reduce cortisol levels. (Holst et al 2005; Olney 2005; Field 2005) Not only is there a pain reduction, but an increase in pain tolerance that is present with repeated treatments and lasts for as long as a regular massage regimen is continued. (Plews-Ogan et al 2005; Aourel et al 2005) Massage-like-stroking has also been found to optimize digestion and improve nutrient assimilation. (Holst et al 2005) The neuropsychological benefits of massage may have a direct impact on pain. Massage has been shown to improve mood, reduce anxiety and depression, and improve sleep. (Field 2005; Corbin 2005; Bastard & Tiran 2006)

## **STRETCHING**

Stretching as it relates to osteoarthritis pain has not been studied. One study found that it may increase the effectiveness of isokinetic exercise in terms of functional improvement in patients with knee osteoarthritis. (Wend et al 2009) Crook et al (2007) found that Labrador Retrievers demonstrated a goniometric increase in osteoarthritic joint range of motion utilizing daily passive stretching. It is unknown if these functional improvements may indirectly contribute to pain management.

## **EXERCISE THERAPY**

Systematic review show that exercise is beneficial for a variety of pain conditions including neck pain, chronic low back pain, pelvic pain, osteoarthritis, patellofemoral pain, intermittent claudication, fibromyalgia, rheumatoid arthritis, and tendonitis. (Bement 2009) Strengthening exercises, specifically, have been shown to be efficacious for hip osteoarthritis. (Hernandez-Molina et al 2008) Thus in addition to allowing the animal to maintain participation in sports, it may also be wise to prescribe exercises specific to the affected joint in order to further improve function (and subsequently reduce pain).

## **CONCLUSION**

Mild and moderate osteoarthritis is often a common condition in sporting animals. With an evidence informed approach to pain management for this condition, physical therapies can be part of a multimodal treatment regimen for the canine athlete that can enable the animal to enjoy participation in sports as he or she ages.

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