Osteoarthritis Pain Management in Dogs and Cats
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Pain management is emerging as one of the hottest topics in veterinary medicine. It is a true mix of the art and science of clinical practice. The science of managing pain has recently entered the veterinary curriculum in two forms: pre-emptive analgesia and multimodal analgesia. Prior to this approach, we were taught that a little pain was beneficial. The thought was that it would keep our patients quiet; therefore, they would be less likely to reinjure themselves. This is no longer the case. We now know that pain is felt in all species, and we can assume that if it hurts us, it will hurt them. The benefits of pain relief go well beyond the reduction of physical pain. It is a complex neuroendocrine response, which if left untreated, can lead to a catabolic state. The response to ongoing pain can result in high heart rates and eventual lowered cardiac output because of increased myocardial oxygen consumption and insufficient time for refill. We are quite fortunate to be able to attack pain from numerous pathways through a multimodal approach.

Osteoarthritis (OA) is one of the most common causes of chronic pain in dogs. It is estimated to affect up to 20% of dogs and cats. Another study showed that almost all cats over 10 years of age have radiographic evidence of appendicular degenerative joint disease. Although we cannot cure this progressive disease, we can relieve the pain and discomfort associated with it. As a result, we improve the quality of life in our patients and our clients.

We once thought that the pain of OA was simply from the wear and tear on the joint. We now know that it is the result of a complex set of predisposing factors along with the patient’s inability to repair joint damage. Pain arises from the synovium and subchondral bone, but the stretching of the joint capsule, damage to the ligaments and periarticular muscles also play a major role. Chronic low-grade inflammation within the arthritic joint capsule and changes in hyaluronic acid production lead to loss of synovial fluid viscosity, joint effusion, and collagen deposition in the capsule itself. This results in pain and thickening of the capsule and loss of range of motion. Reduced flexibility leads to increased stress on the joint capsule or may cause the dog to maintain an abnormal posture, perpetuating the vicious cycle of pain and reduced use of the limb.

Constant stimulation of the pain receptors leads to windup of the central nervous system through hypersensitivity of the dorsal horn of the spinal cord. Altered perception of pain leads to hypersensitivity, hyperalgesia, and allodynia, all of which lead to genetic alteration in the transmission of pain. We should think of pain associated with OA similarly as we think of surgical or traumatic pain and control it through a multimodal approach. This approach allows us to use multiple classes of drugs in a synergistic manner, thus reducing the doses necessary to relieve the pain and reduce the potential side effects from the medications themselves.
**NSAIDS:** Nonsteroidal Anti-inflammatory Drugs are the mainstay of OA pain relief. They work quickly and are effective in reducing pain and inflammation. There is a potential for side effects associated with any drug in this class and these side effects should be thoroughly explained to owners. Vomiting, diarrhea, and anorexia are the most common side effects, and clients should be advised to stop the medication immediately if noted.

NSAID’s work by preventing the production of inflammatory mediators from the metabolism of arachidonic acid, specifically the cyclooxygenase 2 (COX-2) generated prostaglandins. Prostaglandins (PG) produced by metabolism of COX-1 are protective to the gastric mucosa, maintain renal blood flow and platelet function. Therefore, we should choose drugs that spare the COX-1 produced PG and inhibit the COX-2 produced PG. Other factors to consider when selecting NSAID’s include drug-specific factors (safety, efficacy, formulation, dosing, palatability), patient specific factors (concurrent medical condition), and client-specific factors (ability to administer the medication, cost).

Successful dispensing of NSAID’s depends on a numerous variables. A risk-benefit analysis should be performed, especially in patients with concurrent disease. Double checking of the correct dose should be done to ensure the patient is not being inadvertently overdosed. Clients should always be questioned as to the use of other meds, especially other NSAID’s (including aspirin) or steroids.

**Chondroprotectants:** Also called disease-modifying OA drugs, this is the only class of FDA-approved drugs labeled to slow the progression of disease. Adequan Canine® contains polysulfated glycosaminoglycan (PSGAG), primarily chondroitin sulfate that has a higher sulfur content to make this molecule more effective in inhibiting enzymatic activity. This agent acts as both an anti-inflammatory and inhibits matrix metalloproteinase and cartilage oligomeric matrix proteins that degrade the glycosaminoglycans and hyaluronic acid within the joint. PSGAG’s encourage cartilage matrix synthesis and improve synovial fluid quality. It’s most beneficial early in the progression of the disease, but is quite useful at all stages.

**Adjunct Therapies:** This component of therapy involves the use of multiple drugs to aid in the management of OA pain. Clinical experience has shown us that NSAID’s may not provide complete pain control in all of our patients. We have access to a variety of drugs that can be used in addition to NSAID’s.

**Tramadol:** An opioid analgesic that also acts on the serotonergic and alpha-adrenergic systems. It works at the level of the CNS to alter pain perception. The analgesic effects result from the complex interactions between the opioid receptors while interfering with the release and reuptake of norepinephrine and serotonin in the descending inhibitory pathways. There is significant controversy on the efficacy of this drug. As a result, many practitioners are moving away from its use. Additionally, tramadol has been classified as a Schedule IV controlled substance effective August 18, 2014.

**Amantadine and Ketamine:** NMDA-receptor antagonists that appear to work by closing the gateway to central sensitization, which may help make chronic pain easier to manage. The NMDA-receptor is located in the dorsal horn of the spinal cord that allows
for propagation of the pain impulse. If blocked, the pain impulse cannot cross the synapse and enter the spinal cord and then be transmitted to the cerebrum, therefore, pain is not perceived.

**Gabapentin**: An antiseizure medication that is also used in neurogenic pain. Gabapentin works by blocking the calcium channels and inhibiting the transmission of the pain impulse at the neuron synapse in the CNS.

*Weight Control, Therapeutic Diets and a variety of other adjunct treatments are available to complement the treatment of canine OA*. It is important to recognize that all components of the multimodal approach to canine OA management may not be used every day for the remainder of the dog’s life. Rather, some elements will be used initially or for a short time, while other components will ideally become part of the dog’s and the owner’s daily routine.

References:


